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“Part 2: Angina Pectoris: Review & Update”

October 2018



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PART 2: ANGINA PECTORIS: REVIEW & UPDATE. Angina pectoris is the term for chest pain or discomfort due to coronary heart disease. It is a symptom of a condition called myocardial ischemia. In this lesson (Part 2), and the previous one, we discuss a number of factors related to treatment of angina pectoris. Background descriptions and classification of the disease were discussed in the previous lesson. In this lesson, we provide a detailed synopsis of organic nitrate therapy.

In this lesson (“Part 2: Angina Pectoris—Review & Update”), we also provide explanations and information regarding the other 3 classes of angina medications, along with summaries of therapeutic treatment rationale.

This lesson is intended for pharmacists & technicians in all practice settings. The program ID # for this lesson is 0798-0000-18-226-H01-P for pharmacists, and 0798-0000-18-226-H01-T for technicians.

This lesson furnishes 1.25 (0.125 CEUs) contact hours of credit.

Participants completing this lesson by September 30, 2021 may receive full credit. Release date for this lesson is October 1, 2018. This is knowledge-based continuing pharmacy education.

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The objectives of this lesson are such that upon completion participants will be able to:

For Pharmacists:

- 1. Describe angina treatment using beta-receptor blockers, calcium channel blockers and ranolazine.
2. Discuss pharmacotherapeutic approaches to angina therapy.
3. List the rationale for using non-pharmacological treatments, SL nitroglycerin, and dual platelet therapy for angina.
4. Describe an approach for treating unstable angina.

For Technicians:

- 1. Describe angina treatment using beta-receptor blockers, calcium channel blockers and ranolazine.
2. Discuss pharmacotherapeutic approaches to angina therapy.
3. List the rationale for using non-pharmacological treatments, SL nitroglycerin, and dual platelet therapy for angina.
4. Describe an approach for treating unstable angina.

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REVIEW FROM PREVIOUS LESSON

Ischemic Heart Disease (IHD) is a form of heart disease that results from the narrowing of one or more of the major coronary arteries supplying the heart. This results from an imbalance between myocardial oxygen supply or oxygen demand.

Angina pectoris is the most common symptom of IHD. It is a clinical sign resulting from transient myocardial ischemia (lack of blood supply to the heart muscle). The typical episode lasts 3-5 minutes and is brought on usually by physical exertion or emotional stress. Other signs and symptoms may include: shortness of breath, weakness, abdominal fullness, sweating, peripheral vasoconstriction, and palpitation.

The **pain of angina** is due to the inability of the sclerotic or stenosed coronary arteries to provide adequate amounts of oxygen through adequate blood flow to the myocardium during time of increased oxygen demand. The pain is a dull or heavy feeling in the middle of the chest, which may move to either arm (usually the left), or up through the throat, into the jaw, and may radiate to the back. Precipitating factors for typical angina pectoris may be: strenuous physical exercise, emotional stress, drugs which increase the workload and oxygen demand on the heart, heavy meals or, possibly, exposure to rapid changes in temperature (hot & cold). The pain is usually relieved by rest or by stopping (eliminating) causative factors.

Atypical angina, also called **variant angina** or **Prinzmetal's angina**, is not induced by the commonly known predisposing factors. It may occur at rest and is not relieved by the common methods that will be discussed. This type of myocardial ischemia is thought to be due to coronary artery vasospasm (quick constriction of a vessel in a particular segment).

APPROACHES TO THE THERAPY OF ANGINA

1. Acute
 - Rest,
 - Nitroglycerin and,
 - Possibly, oxygen. if hospitalized.
2. Chronic
 - Modification of lifestyle (diet, smoking),
 - Treatment of associated underlying diseases (MI, diabetes, HTN {hypertension}, etc.).
 - Drug therapy
 - Nitrates,
 - Beta-blockers,
 - Calcium-channel blockers, or
 - Ranolazine

In this lesson, and the next, we will discuss a number of factors related to treatment of angina pectoris.

Primarily, we are seeking to (our goals are):

1. Describe the specific families of medications:
 - a. Organic nitrates
 - b. Beta – receptor blockers
 - c. Calcium – channel blockers
 - d. Ranolazine
2. Discuss therapeutic rationale

Background descriptions and classification of the disease were discussed in the previous lesson (**“Part 1: Angina Pectoris---Review & Update”**). **Additionally, in this lesson, we provide a detailed synopsis of organic nitrate therapy.**

In this lesson (**“Part 2: Angina Pectoris---Review & Update”**), we provide explanations and information regarding the other 3 classes of angina medications, along with summaries of therapeutic treatment rationale.

BETA-RECEPTOR BLOCKERS

Beta-receptor blockers (i.e., beta-blockers) that lack intrinsic sympathetic activity (ISA) are useful in the pharmacotherapy of angina by decreasing myocardial oxygen demand through reducing heart rate and contractility. Pure beta-blockers do reduce coronary blood flow because their pharmacologic activity can induce coronary vasoconstriction. This latter effect explains why beta-blockers, unless they also have the ability to block alpha-receptors such as labetalol and carvedilol, should be avoided in variant angina. Beta-blockers with ISA can actually increase heart rate since they are partial beta-receptor agonists and stimulate beta-receptors to some extent, therefore increasing myocardial oxygen demand; and thus, their use is undesirable to prevent stable angina in most instances.

When dosing beta-blockers to prevent angina, the dose can be increased until they are effective in prevention of angina without lowering the heart rate or blood pressure excessively. A reasonable goal for the lowering of heart rate is to achieve a rate that is at least in the lower 60's and ideally in the high 50's, assuming the patient can tolerate heart rates this low. Adverse effects associated with beta-blocker use include bronchospasm, worsening peripheral vascular disease and Raynaud disease, sexual dysfunction, and CNS disturbances. Many patients with COPD and some patients with asthma do tolerate being on a beta-blocker without experiencing any complications. Beta-blockers may be used in patients with diabetes mellitus, but the patient needs to be made aware that beta-blockers can mask the sympathomimetic response to hypoglycemia. Except for those proven to enhance the survival of patients with systolic heart failure (such as carvedilol and metoprolol succinate), beta-blockers should be avoided in patients with systolic heart failure. When used to treat cardiovascular disease, abrupt discontinuation of beta-blockers has precipitated angina and, in some instances, a myocardial infarction.

A listing of the more commonly-used beta-blockers and their pharmacological properties and general dosing are available in **Table 1**.

Table 1. Beta-blocker Pharmacological Properties and Typical Dosing

Product	β1-Receptor Selective	α-Receptor Antagonism	ISAa	Typical Dose (mg)	Dosing Frequency	Predominate route of elimination
Propranolol	No	No	No	20-40 ^b	BID-QID ^b	Hepatic
Metoprolol	Yes	No	No	25-50 ^c	BID ^c	Hepatic
Nadolol	No	No	No	40-80	QD	Renal
Atenolol	Yes	No	No	50-100	QD	Renal
Labetalol	No	Yes	No	200-300	BID	Hepatic
Carvedilol	No	Yes	No	12.5-25 ^d	BID ^d	Hepatic
Nebivolol ^e	Yes	No	Yes	10-20	QD	Hepatic
Pindolol ^f	No	No	Yes	5-20	BID	Hepatic

^a ISA = Intrinsic sympathomimetic activity (see text for explanation).

^b Available in a long-acting once-a-day preparation with a typical dose being 80-160 mg.

^c This information relates to immediate-release metoprolol tartrate; an extended-release once-a-day metoprolol succinate product is also available with a typical dose being 50-100 mg.

^d Available in a constant-release once-a-day preparation with a typical dose being 40-80 mg.

^e Also causes peripheral vasodilation by stimulating nitric oxide release.

^f The presence of ISA properties makes pindolol undesirable to use in the pharmacotherapy of angina in most instances.

Adapted from: Lexicomp Drug Information Handbook, 26th edition. Wolters Kluwer:2017

In the past, atenolol was a very commonly-used beta-blocker because it was relatively long-acting and beta1-selective. Beta1-selective agents are less apt to induce bronchospasm and peripheral vasoconstriction, albeit, selectivity is lost as the beta-blocker dose is increased. However, atenolol is renally eliminated. Patients' renal function tends to deteriorate with aging and this can lead to an accumulation of atenolol in the body and place patients at risk for experiencing symptomatic sinus bradycardia. In addition, a retrospective analysis has suggested that atenolol enhances morbidity and mortality.⁶ For these reasons, metoprolol use has become even more common, especially with the availability of the once-a-day extended-release succinate salt. Recall that metoprolol tartrate is given twice a day.

If hypertension is a concurrent concern with the angina, carvedilol or labetalol may be preferred since they have alpha-receptor blocking activity in addition to their beta-receptor blocking activity. As discussed above, since beta-blockers with ISA can increase myocardial oxygen demand because they increase heart rate, they are generally not considered in the pharmacotherapy of angina. Nebivolol is a beta-blocker that also can induce vasodilation. It does so by stimulating nitric oxide production, not by blocking alpha-receptors. Any clinical advantage that nebivolol has over traditional beta-blockers has yet to be clearly delineated.

CALCIUM CHANNEL BLOCKERS

Calcium channel blockers are also effective in the preventive treatment of angina. Typically, calcium channel blockers are divided into three categories: verapamil, diltiazem, and the dihydropyridines. Two examples of commonly-used dihydropyridines are nifedipine and amlodipine. The pharmacological properties of the different classes of calcium channel blockers (as well as beta-blockers and organic nitrates) and their effects on myocardial oxygen supply and demand are available in **Table 2**. All three categories improve coronary artery blood flow by dilating coronary arteries. All categories dilate peripheral arteries and reduce afterload with dihydropyridines doing so to the greatest extent. None of them dilate veins and, as a result, have no impact on reducing preload. Verapamil and diltiazem directly reduce heart rate while dihydropyridines reflexively increase heart rate in response to their ability to dilate arteries. Therefore, one should be cautious with using a dihydropyridine in the absence of a

beta-blocker since an increase in heart rate increases myocardial oxygen demand. Nearly all the calcium channel blockers reduce myocardial contractility to some extent with verapamil doing so to the greatest extent. Two calcium channel blockers that do not reduce contractility are amlodipine and felodipine. These two agents can be considered in the pharmacotherapy of angina in patients with systolic heart failure whereas the other calcium channel blockers should be avoided in such patients. Although not germane to the prevention of angina, verapamil and diltiazem block conduction within the AV node. A listing of commonly-used calcium channel blockers, their availability as immediate-release or sustained-release preparations, and their typical dosing are available in **Table 3**.

Calcium channel blockers can be used in stable, unstable, and variant angina. In fact, they are the drug of choice in the treatment of variant angina. The actual calcium channel blocker to use in a patient can be influenced by the patient's other medical conditions. If a patient has atrial fibrillation, verapamil or diltiazem are often preferred because of their AV nodal effects. If a patient is already receiving a beta-blocker or has a low heart rate, a dihydropyridine is often preferred. Due to its long half-life and once-a-day dosing, amlodipine is the dihydropyridine most frequently used. Due to their association with enhanced mortality, immediate-release nifedipine use in patients with coronary artery disease should be avoided.⁷ All dihydropyridines are typically avoided in patients with hypotension.

Adverse effects of calcium channel blockers vary amongst the different agents. Constipation is most frequently associated with verapamil use. Dihydropyridines are associated with flushing, gingival hyperplasia, and leg edema. All calcium channel blockers have been implicated in causing gastroesophageal reflux and precipitating eczema.

With respect to drug interactions, diltiazem and verapamil can inhibit CYP 3A4 and can reduce the clearance of medications metabolized by this hepatic enzyme and, on occasion, have been associated with increasing digoxin serum concentrations by reducing the clearance of digoxin and/or increase digoxin bioavailability by inhibiting para-glycoprotein efflux pump activity.

Table 2. Pharmacological Comparison of Calcium Channel Blockers and Other Antianginals.

Hemodynamic Parameter	Diltiazem	Dihydropyridines	Verapamil	Beta-blockers	Organic nitrates
Coronary blood flow	↑↑↑	↑↑↑	↑↑↑	↓	↑↑
Afterload	↓	↓↓↓	↓↓	sl↑	↓
-Normative					
-Hypertensive	↓↓↓	↓↓↓	↓↓↓	sl↑	↓↓
Preload	0	0	0	↑	↓↓↓
Heartrate	↓↓	↑ ^a	↓↓	↓↓↓	↑ ^a
AV node conduction	↓↓	0	↓↓	↓	0
Contractility	↓	↓ ^b	↓↓	↓↓↓	↑ ^a

^aThis increase is a reflexive reaction in response to peripheral arterial vasodilation.

^bNearly all dihydropyridines (the exceptions being felodipine and amlodipine) intrinsically reduce contractility; in theory, dihydropyridines may reflexively increase contractility in response to peripheral arterial vasodilation but this has not been demonstrated to be beneficial in the clinical setting.

Adapted from:

Schroeder JS. Calcium and beta-blockers in ischemic heart disease: when to use which. *Modern Medicine*, 1982;7(11):13-26.

Opie LH. Calcium-channel blockers (calcium antagonists). In: Opie LH, Gersh BJ. *Drugs for the heart*, 7th ed. Philadelphia:Saunders Elsevier: 2009:59-87.

Shub C, Vlietstra RE, McGoon MD. Selection of optimal drug therapy for the patient with angina pectoris. *May Clin Proc*. 1985;60:539-48.

Table 3. Typical Dosing of Commonly-used Calcium Channel Blockers

Medication	Immediate-Release (IR)?	Typical IR dosing (mg)	Sustained-release (SR)?	Typical SR dosing (mg)
Diltiazem	Yes	30-90 TID-QID	Yes ^a	120-360 QD or 60-180 BID
Verapamil	Yes	80-120 TID	Yes	240-360 QD
Dihydropyridines Nifedipine	Yes	___ ^b	Yes	30-90 QD
Nicardipine	Yes	20-40 TID	Yes	30-60 BID
Amlodipine	Yes	5-10 QD	No	
Felodipine	No		Yes	5-10 QD

^aAvailable as both a once-a-day preparation and a twice-a-day preparation; assure the correct preparation prescribed is being dispensed.

^bThe use of immediate-release nifedipine should be avoided in patients with coronary artery disease.

Adapted from: Lexicomp Drug Information Handbook, 26th edition. Wolters Kluwer:2017 Furberg CD, Psaty BM, Meyer JV. Nifedipine. Dose-related increase in mortality in patients with coronary heart disease. *Circulation*. 1995;92:1326-31.

RANOLAZINE

The newest FDA-approved medication in the pharmacotherapy of angina is ranolazine, even though, it was released more than 10 years ago. The exact mechanism by which ranolazine prevents angina is not fully understood. It is known that, in contrast to organic nitrates, beta-blockers, and calcium channel blockers, ranolazine does not treat angina by reducing heart rate or blood pressure. One thought is that ranolazine impedes the “late sodium current” and thus prevents the enhanced influx of sodium into myocardial cells that can occur during ischemia. With reduced intracellular sodium within ischemic cells, there is a reduced amount of sodium leaving the ischemic myocardial cells, and can allow this for calcium to enter ischemic myocardial cells. Calcium entering ischemic myocardial cells is harmful for several reasons, including disruption of myocardial relaxation and reduced coronary blood flow. Therefore, by reducing the influx of sodium, ranolazine ultimately reduces the influx of “harmful” calcium.

Ranolazine has been demonstrated to reduce angina episodes and prolong exertional activity duration in patients with stable angina. Ranolazine has not been demonstrated to be useful in the treatment of variant angina. Ranolazine comes as an extended-release tablet. Dosing is started at 500 mg BID and may be increased, if needed, to 1 gram BID.

Ranolazine does increase the QT interval and should be used cautiously, if at all, with other medications known to prolong the QT interval. A prolonged QT interval places a patient at risk of having a unique sinusoidal-shaped ventricular dysrhythmia known as torsade de pointes. Other adverse effects of ranolazine include headache, dizziness, GI upset, and constipation. Ranolazine is metabolized to a great extent by CYP 3A4 and to a small extent by CYP 2D6. It is contraindicated to use ranolazine with ketoconazole, itraconazole, clarithromycin, nefazodone, nelfinavir, ritonavir, indinavir, and saquinavir or enzyme inducers such as phenytoin, phenobarbital, carbamazepine, and rifampin. The dose of ranolazine should not exceed 500 mg BID in patients also receiving diltiazem, verapamil, erythromycin, or fluconazole. Ranolazine

does reduce DIGOXIN CLEARANCE.

PHARMACOTHERAPEUTIC APPROACHES

Acute Attacks

Sublingual nitroglycerin products are useful in the acute treatment of all three types of angina: stable, unstable, and variant. With respect to stable angina, acute attacks can be treated with a sublingual nitroglycerin product, giving a dose every 5 minutes for as many as three doses. As long as the pain is improving and has been relieved by the end of 5 minutes after the third dose of nitroglycerin, there is generally no need to seek medical attention. However, if the patient's stable angina pain stops improving or gets worse, medical attention should be sought immediately. Also, if the initial angina pain is not the patient's typical stable angina pain, medical attention should be sought immediately.

It should also be noted that the first time a patient experiences angina pain, medical attention should be sought immediately. Such an episode is considered unstable angina and the patient most likely has yet to be prescribed a SL nitroglycerin product. Also of note, chewing 325 mg of aspirin may be of benefit to the patient in such an instance.

Chronic Prevention

For the prevention of stable angina, beta-blockers are advocated by expert guidelines as the first pharmacotherapeutic option, especially if the patient has a history of a myocardial infarction or systolic heart failure.⁴ The beta-blocker selected should lack ISA (intrinsic sympathomimetic activity). The dose of beta-blocker can be increased as needed as long as the dose does not induce symptomatic bradycardia, hypotension, or other beta-blocker-associated adverse effects described earlier. If hypertension is a concurrent problem, a beta-blocker with alpha-blocking properties may be considered. If a patient is unable to receive a beta-blocker because of a contraindication or intolerance, consideration can be given to a non-dihydropyridine calcium channel blocker, assuming the patient does not have a reduced ejection fraction, since these agents can also lower heart rate as well as dilate coronary arteries. Recall that using a dihydropyridine in the absence of a beta-blocker may result in an increase in heart rate, an effect that is not desired in the treatment of angina.

A reasonable second drug to add to someone receiving a beta-blocker is a calcium channel blocker or an organic nitrate. Both can lower a patient's blood pressure, but an organic nitrate has less propensity to do so. With respect to what calcium channel blocker to use, assuming the patient's heart rate is already low due to maximizing the patient's beta-blocker dose, a dihydropyridine such as amlodipine would be reasonable. With respect to what organic nitrate to use, the use of once-a-day sustained-release isosorbide mononitrate is a very worthy option. The dose of whichever medication is selected can be increased as needed for as much as the blood pressure will allow and/or the patient's ability to tolerate the medication. If a low blood pressure prevents one from adding either medication, then ranolazine becomes a worthy consideration since it has minimal impact on the hemodynamic parameters. Ranolazine has traditionally been reserved as a latter selection due to its monthly cost relative to the other antianginal agents.

If a patient was originally on a calcium channel blocker because a beta-blocker could not be tolerated, adding an organic nitrate as the second agent is reasonable. Again, if hemodynamic issues prevent this, then ranolazine can be considered.

In some instances, a patient may be started on two medications initially since no one medication addresses all of the hemodynamic parameters in a positive manner that leads to

prevention of angina. Beta-blockers with an organic nitrate or with a calcium channel blocker may be considered here.

If a patient still has frequent attacks of angina despite therapeutic doses of two antianginals, a third antianginal can be added. If a patient is on a beta-blocker and an organic nitrate, adding a calcium channel blocker is reasonable but, again, one must realize that if the beta-blocker is dosed to achieve a low heart rate, the calcium channel blocker being added needs to lack the propensity to further lower the heart rate. Amlodipine is a very attractive option here. If hemodynamics limit the use of adding on a calcium channel blocker, ranolazine is a worthy consideration.

If angina continues to be an issue despite three medications, assuming all can be tolerated, all four medications may be used. That said, make sure the patient can tolerate the medications, the blood pressure and heart rate are not excessively low, and, if verapamil or diltiazem are being used, assure the dose of ranolazine does not exceed 500 mg BID.

OTHER PHARMACOTHERAPEUTIC CONSIDERATIONS

Non-Pharmacological Treatments of Angina

Non-pharmacological treatment of stable angina include coronary artery bypass surgery, percutaneous transluminal angioplasty with or without the insertion of an intracoronary stent, transmyocardial revascularization, and the use of enhanced external counter pulsation cuffs. It is beyond the scope of this lesson to further discuss these treatments.

Prophylactic Use of SL Nitroglycerin

If a patient knows an activity will precipitate angina, the patient may elect to use a SL nitroglycerin product in a prophylactic manner. The patient should be instructed to take a dose at least 5 minutes prior to initiation of the activity.

Dual Antiplatelet Therapy

If a patient receives coronary artery angioplasty followed by insertion of an intracoronary stent, the patient needs to be on both aspirin and a P2Y₁₂ inhibitor such as clopidogrel, prasugrel, or ticagrelor for a period of time. This use of dual antiplatelet therapy protects the patient from thrombosis related to the exposed metal struts of the stent until this metal can be endothelialized. The duration of dual antiplatelet use is constantly being re-evaluated. At the time of this writing, for patients receiving a drug-eluting stent, it would be rare that a course of dual antiplatelet therapy would be less than 6 months in duration and, if the stent was inserted related to unstable angina, preferably be continued for at least 12 months. It should be noted that if a patient received a bare metal stent, dual antiplatelet therapy is needed for only a month in the absence of unstable angina but dual antiplatelet therapy for 12 months would also be preferred if the stent was inserted due to an episode of unstable angina. Patients who experience unstable angina and have angioplasty performed but no stent inserted or do not have angioplasty performed would also benefit from dual antiplatelet therapy for 12 months.⁸ In addition to the antiplatelet therapy, these patients should also receive daily aspirin and a statin and be considered for an ACE-inhibitor or ARB (angiotensin II receptor blocker). In theory, chronic antianginal pharmacotherapy would not be needed if the procedure was totally successful, but consideration would be given to antianginal pharmacotherapy if chest pain returned despite the procedure.

PHARMACOTHERAPY OF UNSTABLE ANGINA

A calcium channel blocker is the therapy of choice to treat variant angina. If this is insufficient, an organic nitrate may be added but not a beta-blocker since they can induce coronary vasospasm. If additional therapy is needed beyond an initial calcium channel blocker and an organic nitrate, a second calcium channel blocker may be added, preferably one that allows the patient to be ultimately on a dihydropyridine and either verapamil or diltiazem.

Footnotes

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I am a Pharmacist

I am a Technician

CPEMonitor ID _____ **Birthdate (MM/DD)** _____

ARE YOU LICENSED IN FLORIDA? IF YES, FL LIC # _____

EMAIL Address (REQUIRED) _____

LESSON EVALUATION

Please fill out this section as a means of evaluating this lesson. The information will aid us in improving future efforts. Either circle the appropriate evaluation answer, or rate the item from 1 to 7 (1 is the lowest rating; 7 is the highest).

1. Does this lesson meet the learning objectives? (Circle your choice).

- | | | |
|---|-----|----|
| Describe angina treatment | YES | NO |
| Discuss drug therapy approaches to treat angina | YES | NO |
| List rationale for other treatments for angina | YES | NO |
| Describe an approach for treating unstable angina | YES | NO |

2. Was the program independent & non-commercial? YES NO

3. Relevance of topic

	Low Relevance					Very Relevant
	1	2	3	4	5	6 7

4. What did you like **MOST** about this lesson? _____

5. What did you like **LEAST** about this lesson? _____

6. How would you improve this lesson? _____

Please Mark the Correct Answer(s)

1. **Beta-blockers are useful in the treatment of stable angina by:**

- a. Decreasing myocardial contractility
- b. Increasing wall tension
- c. Dilating peripheral arteries
- d. Increasing heart rate

2. **Ranolazine:**

- a. Increases blood pressure
- b. Enhances sodium influx into myocardial cells
- c. Reduces heart rate
- d. Prolongs the QT interval

3. **In general, the initial pharmacotherapy to prevent stable angina should be:**

- a. Pindolol
- b. Nifedipine
- c. Metoprolol
- d. Isosorbide mononitrate

4. **Constipation is a common adverse effect of:**
 - a. Verapamil
 - b. Atenolol
 - c. Ranolazine
 - d. Amlodipine
5. **A patient with stable angina is on metoprolol 50 mg BID and sustained-release isosorbide mononitrate 90 mg daily. The patient's heart rate averages 58 bpm and blood pressure averages 102/72 but has no symptoms and feels fine; however, the patient still experiences exertional angina when gardening. Which do you suggest for chronic preventive treatment?**
 - a. Add amlodipine
 - b. Add verapamil
 - c. Add ranolazine
 - d. Increase the dosing of the sustained-release isosorbide mononitrate to BID
6. **Metoprolol tartrate should NOT be used in patients with:**
 - a. Stable angina
 - b. Unstable angina
 - c. Variant angina
 - d. None of these
7. **In addition to antianginal medications, what medication from the list below should a patient with stable angina typically be receiving?**
 - a. Warfarin
 - b. Simvastatin
 - c. Sumatriptan
 - d. Ibuprofen
8. **A reasonable goal for the lowering of heart rate is to achieve a rate that is at least in the lower 60's and ideally in the high 50's, assuming the patient can tolerate heart rates this low.**
 - a. True
 - b. False
9. **The newest FDA approved medication for treating angina is:**
 - a. Verapamil
 - b. Tocix
 - c. Ranolazine
 - d. Cresaline
 - e. Mictriacine
10. **Which of these have been used for prevention of angina:**
 - a. Beta blockers
 - b. Calcium channel blockers
 - c. Organic nitrates
 - d. All of these
 - e. None of these