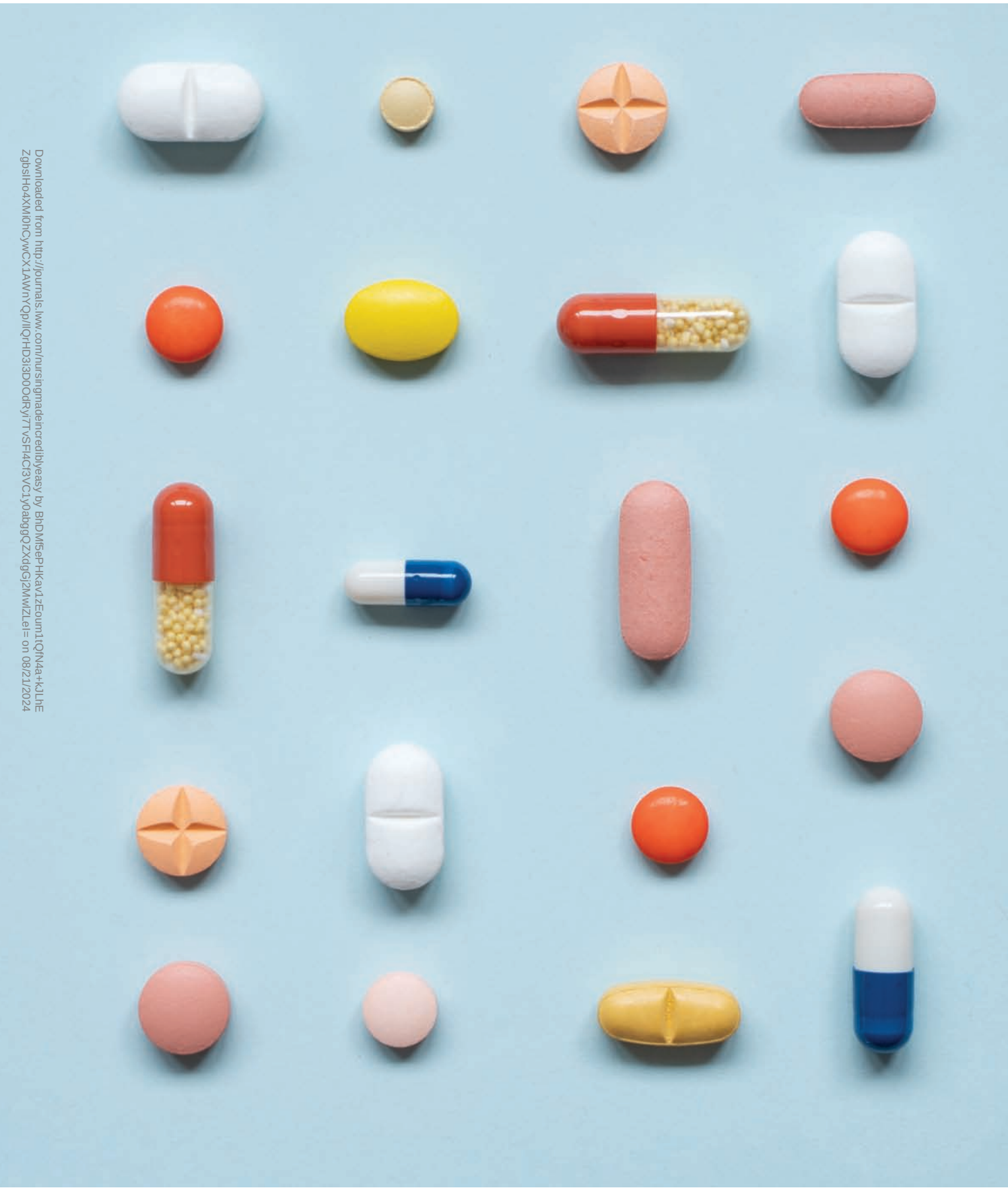

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Beta-blockers and calcium channel blockers: What's the difference?

Nurses must know the difference between beta-blockers and calcium channel blockers, including understanding their mechanisms, indications, adverse reactions, and interactions, and how to monitor patient needs to promote patient care.

By Tyler Sturdivant, DNP, RN, AGCNS-BC, SCRN, COI; Katrina Allen-Thomas, MSN, RN; and Kristin Allen-Tew, PharmD, BS

Beta-blockers were first marketed decades ago for the treatment of angina, better known as chest pain.[1] Since their introduction, beta-blockers have been widely used in patients with hypertension, heart failure, myocardial infarction, tachycardia, cardiac arrhythmia, coronary artery disease, aortic dissection, and portal hypertension. Beta-blockers are also used, although less commonly, in patients with hyperthyroidism, anxiety, essential tremors, migraines, glaucoma, and hypertrophic obstructive cardiomyopathy. With the generic name of most beta-blockers using the suffix "-lol," medications in this class are similar in action and have like adverse reactions.[2] Used alone or in combination with other medications to treat disease processes or to prevent complications of diseases, beta-blockers are commonly prescribed today in the healthcare environment.

After the introduction of beta-blockers, calcium channel blockers were developed to help promote coronary dilation, and also treat angina or chest pain.[3] Since their introduction, calcium channel blockers have been commonly used in patients with hypertension, angina, supraventricular arrhythmia, coronary spasms, pulmonary hypertension, and hypertrophic cardiomyopathy. Less commonly, calcium channel blockers are also prescribed for Raynaud phenomenon, migraines, and subarachnoid hemorrhage. Like beta-blockers, most calcium channel blockers end in a special suffix, "-pine," and share actions and adverse reactions among the medications in the class.[4]

Nurses and nursing students are likely to administer beta-blockers and calcium channel blockers in a variety of healthcare settings. Basic knowledge of current uses, mechanisms of action, adverse reactions, and nursing implications is necessary. Understanding the similarities and differences between beta-blockers and calcium channel blockers will allow you to safely administer and educate your patients on these medications. Throughout this article, we'll describe beta-blockers and calcium channel blockers to solidify your knowledge on these common medications.

Understanding beta receptors

Let's start by understanding the normal functions of beta receptors. Beta receptors exist in three different forms: beta-1, beta-2, and beta-3.[2] Under normal conditions, these receptors function within the sympathetic nervous system by responding to the catecholamines of epinephrine and norepinephrine. Beta-1 receptors are primarily located in the heart and kidneys. When beta-1 receptors are activated in the heart, myocardial contractility, automaticity, and heart rate increase. When beta-1 receptors are activated in the kidneys, smooth muscle cells activate the renin-angiotensin-aldosterone system, increasing blood volume and BP.[5] Most notably, beta-1 works in the heart to improve myocardial function.

When beta-2 is activated, smooth muscle cells are relaxed in some organs, including the lungs, heart, and uterus. Although present in these organs, beta-2 receptors are predominantly found in the smooth muscles of the lungs. With beta-2 activation, bronchodilation occurs, increasing oxygenation.[6] Beta-3 receptors promote lipolysis, or the breakdown of fat cells, but are less clinically relevant than beta-1 and beta-2 function.[3]

Mechanism of action: Beta-blockers

Beta-blockers may work on only beta-1 receptors, known as selective beta-blockers, or can work on all beta receptors, known as nonselective beta-blockers (see *Common beta-blockers*). Through the effects on just beta-1, selective beta-blockers inhibit the sympathetic nervous system's influence on the heart. This blockade reduces the heart's chronotropic (rate) effect, lowering the heart rate. Also, selective beta-blockers reduce the heart's inotropic (contractility) effect, lowering overall cardiac output. With the beta-1 receptors' blockade in the kidneys, renin secretion is diminished, reducing blood volume and BP. Through the negative chronotropic and inotropic actions, oxygenation to myocardial tissue improves secondary to reduced oxygen demand.[2]

Nonselective beta-blockers work on all beta receptors, primarily beta-1 and beta-2. Along with the cardiac effects seen with selective beta-blockers, nonselective beta-blockers also inhibit smooth muscle relaxation, mostly in the lungs. Inhibition of smooth muscle relaxation in the lungs causes bronchoconstriction, reducing air movement and the potential for oxygenation.[2] To remember the actions of beta-1 receptors and beta-2 receptors, think that we have one heart and two lungs in our body. Beta-1 receptors work on the heart, and beta-2 receptors work on the lungs (see *Beta-blocker actions*).

Common beta-blockers

Selective beta-blockers	
• Atenolol	• Esmolol
• Betaxolol	• Acebutolol
• Bisoprolol	• Metoprolol
• Nebivolol	
Nonselective beta-blockers	
• Propranolol	• Nadolol
• Labetalol	• Pindolol
• Penbutolol	• Sotalol
• Carvedilol	• Timolol

Understanding calcium channels

Voltage-gated calcium channels are located throughout the entire body. Calcium ions help regulate muscle contraction; neurotransmitter secretion; cell growth and migration; and the activity of several proteins including enzymes, ion channels, and transporters.[7] In the cardiovascular system, calcium influx plays an important role in increasing the excitation and contraction of the heart and in vascular smooth muscles. This calcium influx helps control atrioventricular conduction, heart rate, pacemaker activity, and vascular tone, impacting heart rhythm and BP.[8]

Mechanism of action: Calcium channel blockers

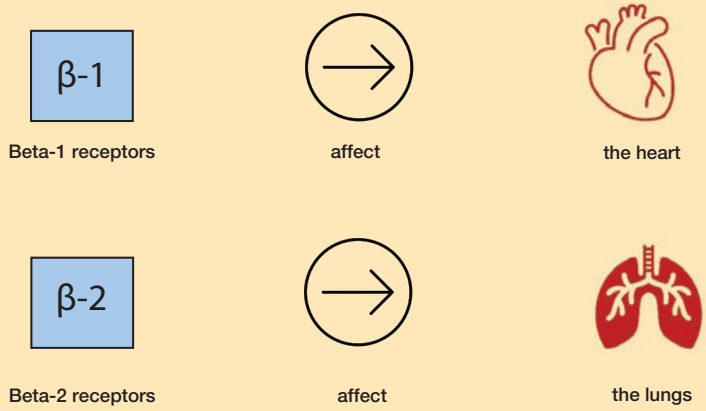
Calcium channel blockers inhibit the movement of calcium ions through the calcium channels in the heart, smooth muscle, and pancreas. There are two main types of calcium channel blockers based on how they function: nondihydropyridines and dihydropyridines (see *Common calcium channel blockers*). Nondihydropyridine calcium channel blockers impact the sinoatrial and atrioventricular nodes through a reduction in contractility and cardiac conduction. Dihydropyridine calcium channel blockers have a limited effect on the actual heart tissue, but cause vasodilation in the vascular smooth muscle through calcium blockade.[4] Dihydropyridines follow the naming scheme with the suffix "-pine," whereas nondihydropyridines don't use a consistent naming structure.

As nondihydropyridines lower cardiac contractility, automaticity, and conduction, a reduction in heart rate and myocardial oxygen demand is noted. Because of these actions, these types of calcium channel blockers are useful in tachyarrhythmias and angina. Dihydropyridines inhibit vascular constriction, both in the coronaries and peripheral vasculature, lowering BP and cardiac afterload, all improving oxygenation. Because of these actions, these types of calcium channel blockers are also useful in treating hypertension and angina.

Adverse reactions: Beta-blockers

One of the most common adverse reactions of beta-blockers is bradycardia.[2] As beta-1 receptors work primarily on the heart, both selective and nonselective beta-blockers can cause a reduction in heart rate. This reduction in heart rate may lead to decreased cardiac output and altered cardiac contractility. With beta-1 blockade after the administration of beta-blockers, the renin-angiotensin-aldosterone system is also inhibited. This blockade may lead to a decrease in systolic and diastolic BP. For patients with a history of systolic heart failure, cardiac arrhythmias, hypotension, and peripheral vascular disease, beta-blockers should be used with caution secondary to the potential for diminished perfusion. The patient may report excessive fatigue, dizziness, and depression, especially if reduced perfusion is noted in the brain.[8]

Beta-blocker actions



Source: Tyler Sturdivant, DNP, RN, AGCNS-BC, SCRN, COI; Katrina Allen-Thomas, MSN, RN; Kristin Allen-Tew, PharmD, BS

Keep in mind that under normal circumstances, beta receptors are part of the sympathetic nervous system in response to the catecholamines epinephrine and norepinephrine. If a patient's blood glucose level drops, it's a natural compensatory response to release catecholamines to alert the patient of the reduction in blood glucose and attempt to raise the glucose levels in the blood. This normal sympathetic response would increase the heart rate and BP, improving perfusion and circulation of glucose in the event of hypoglycemia.[9]

Beta blockade from beta-blockers prevents the sympathetic response that typically occurs when blood glucose levels drop. Patients with a reduction in blood glucose while concurrently taking beta-blockers may not experience the tachycardia and hypertension associated with an expected sympathetic response;

hence, beta-blockers mask the symptoms of hypoglycemia. It's important to note that although beta-blockers can have minor effects on either raising or reducing blood glucose levels, the primary effect is on the masking of hypoglycemia symptoms.[8]

Nonselective beta-blockers have effects on beta-2 receptors. Recall that beta-2 receptors affect the lungs. Inhibition of beta-2 receptors can cause bronchoconstriction; therefore, nonselective beta-blockers should be used cautiously in patients who have restrictive airway disease, including asthma, chronic bronchitis, and chronic obstructive pulmonary disease. Patients who experience bronchoconstriction with the administration of nonselective beta-blockers may experience wheezing and shortness of breath. The healthcare provider may consider prescribing nonselective beta-blockers to patients with a history of restrictive airway disease as patient-specific benefits may outweigh the risks.[8]

Adverse reactions: Calcium channel blockers

Dihydropyridine calcium channel blockers are the most commonly prescribed type of calcium channel blockers. With the effects of dihydropyridines on smooth muscle, hypotension is one of the most common adverse reactions of this class of medication.[10] Remember, calcium influx in the vascular smooth muscle normally causes constriction, increasing BP.

After the administration of calcium channel blockers, calcium channel blockade inhibits the calcium influx into vascular smooth muscle, thereby reducing BP. As vascular smooth muscle tone is reduced, peripheral edema may occur secondary to limited venous return. With additional smooth muscle relaxation, constipation from reduced peristalsis, reflex tachycardia, and bradycardia can also occur. Nondihydropyridines have some effect on smooth muscle tone but primarily work on the conduction and contractility of the heart.[9] Palpitations from arrhythmias, dizziness, reflex tachycardia, and bradycardia are the most common adverse reactions of nondihydropyridines. Of note, although dihydropyridines can have both vascular and cardiac effects, most adverse reactions are vascular. Nondihydropyridines can also have both vascular and cardiac effects; however, most adverse reactions are seen related to heart rate and rhythm.[11]

Common calcium channel blockers

Nondihydropyridine	
• Verapamil	• Diltiazem
Dihydropyridine	
• Amlodipine	• Nicardipine
• Clevidipine	• Nifedipine
• Nimodipine	• Felodipine
• Isradipine	• Nisoldipine

Nursing implications: Beta-blockers

The nurse should monitor for bradycardia while administering beta-blockers. Remember to assess the radial or apical pulse before administering the medication. Notify the healthcare provider if the patient's pulse falls below 60 beats/minute (this number may not apply to every patient depending on diagnosis; refer to the provider's prescription), or if the patient displays symptoms of decreased perfusion. Assess level of consciousness, capillary refill, peripheral pulses, and skin color to determine if decreased perfusion is present. With the potential for bradycardia, advocate for the use of continuous telemetry monitoring while administering beta-blockers, especially during initial treatment. For the home environment, educate patients and family members to measure and record the heart rate before administration.

As beta-blockers have the potential to mask the symptoms of hypoglycemia, inform patients that the most reliable method for determining a hypoglycemic episode is performing a blood glucose check.

Dizziness, headache, fatigue, and measurable hypotension are common adverse reactions due to diminished perfusion from the administration of beta-blockers. Educate patients that feelings of fatigue typically improve after several weeks of treatment. Patients are at risk for falls secondary to reduced perfusion to the brain. Perform a standardized fall risk assessment and implement fall precautions per your facility's protocol. Monitor BP readings and notify the healthcare provider if the systolic BP falls below 100 mm Hg (this number may not apply to every patient depending on diagnosis; refer to the provider's prescription), or if the patient is symptomatic. Encourage patients and family members to measure and record BP readings at home. Orthostatic BP measurements may be indicated, especially during the initiation of treatment.

As beta-blockers have the potential to mask the symptoms of hypoglycemia, inform patients that the most reliable method for determining a hypoglycemic episode is performing a blood glucose check. Educate patients that typical sympathetic symptoms (such as tachycardia and sweating) of a hypoglycemic episode may not be present while taking beta-blockers. Encourage patients to keep hard candy or glucose-rich snacks near at all times in the event of a hypoglycemic episode. Glucagon is often administered in the event of a severe hypoglycemia episode and with beta-blocker toxicity or overdose.

With nonselective beta-blockers, the risk of bronchoconstriction in the lungs is present. Assess the patient's history for a respiratory disorder, such as asthma or chronic obstructive pulmonary disease, before administering a nonselective beta-blocker. During treatment, assess lung sounds for wheezing, and determine the patient's baseline respiratory rate. Reports of shortness of breath and noted tachypnea may indicate respiratory compromise secondary to bronchoconstriction from the nonselective beta-blocker. Although the benefits of administering a nonselective beta-blocker to patients with a history of restrictive airway disease may outweigh the risks, notify the healthcare provider if adverse respiratory events occur.

Beta-blockers carry a black-box warning for early discontinuation. Educate patients that beta-blockers shouldn't be discontinued abruptly and that doses should be tapered over 1 to 2 weeks. Tapering of beta-blockers prevents rebound tachycardia, hypertension, and coronary artery occlusion that may potentially lead to angina or a myocardial infarction.[8]

Nursing implications: Calcium channel blockers

Calcium channel blockers carry similar risks for bradycardia and hypotension. Monitoring protocols of heart rate, BP, orthostasis, and falls should be followed like beta-blockers. With the potential for bradycardia and arrhythmia, especially with

Consider this

LN, a 72-year-old Black American female presents to the clinic with complaints of shortness of breath, fatigue, and dizziness. She states that her symptoms began 3 days ago but have progressively worsened. She has a past medical history of hypertension, hyperlipidemia, and coronary artery disease. LN's home medication profile includes amlodipine 10 mg P.O. daily, atorvastatin 20 mg P.O. daily at bedtime, aspirin 81 mg P.O. daily, and cetirizine 10 mg P.O. daily. Upon assessment, her oral temperature is 37° C and her respiratory rate is 24 breaths/minute, slightly labored, with no use of accessory muscles. Crackles are noted in the posterior lung fields bilaterally, and her SpO_2 is 94%. Cardiac S_1 and S_2 are noted with an S_3 gallop. Her heart rate is regular at 86 beats/minute, and her BP is 134/86 mm Hg. Bilateral radial and dorsalis pedis artery pulses are 1+ in strength, with 1+ pitting edema noted in the bilateral lower extremities. Her bowel sounds are normoactive, and her abdomen is slightly distended.

LN's healthcare provider orders a comprehensive metabolic panel, complete blood cell count, cardiac enzymes, brain natriuretic peptide (BNP), 12-lead ECG, and chest X-ray. Of note, LN's BNP results were 270 pg/mL and her ECG tracing showed normal sinus rhythm with a rate of 78 beats/minute. The chest X-ray showed scant opacities in the bilateral bases. All other lab values were unremarkable.

In the case of LN, the nurse should determine if her symptoms are related to one of the home medications, or if a new or worsening disease process is occurring. As LN is on a calcium channel blocker, amlodipine, the nurse should recognize that the assessment findings of pitting edema and abdominal distension could be related to the adverse reactions of the medication. The nurse should consider the patient's home medications and the associated adverse reactions in addition to the complaints presented during LN's clinical visit. Is the pitting edema related to the calcium channel blocker, or is the patient experiencing fluid volume overload? Is the abdominal distension related to the adverse reaction of constipation from the calcium channel blocker, or is the patient experiencing ascites? With the resulting BNP value, crackles, and the S_3 gallop, the patient is likely experiencing fluid volume overload secondary to new-onset heart failure. The nurse should communicate the findings with the healthcare provider to discuss this potential diagnosis.

nondihydropyridines, advocate for the use of continuous telemetry monitoring during administration. Concurrent use of calcium channel blockers and beta-blockers may increase the incidence of severe bradycardia and hypotension. Although concurrent use may be medically indicated, notify the healthcare provider if these two classes of medications are used together.

Patients shouldn't administer calcium channel blockers with grapefruit juice, as it may inhibit the metabolism of the drug, increasing the risk of toxicity.[8] In some cases, calcium channel blocker toxicity or overdose is managed with the administration of I.V. calcium. For relief of constipation, encourage fluid intake if not contraindicated, ambulation, and fiber-rich foods, including fresh fruits, vegetables, legumes, and whole grains.

The nurse should conduct routine extremity checks to determine the presence of peripheral edema. Assess for nonpitting or pitting edema in the ankles and feet and document accordingly. Monitor daily fluid status through the completion of daily weight and assessment of fluid intake and output. Excessive peripheral edema or fluid retention may necessitate a change in medication or the addition of pharmacologic treatment. (See *Consider this*.)

Final overview

Beta-blockers and calcium channel blockers are commonly prescribed for a variety of indications and are often seen in nursing practice. Through work on either beta receptors or calcium channels in the heart and vasculature, these medications help lower BP and manage tachycardia

or other arrhythmia. You should be aware of major adverse reactions, including bradycardia and hypotension for both classes and assess for these complications. Most importantly, educating your patients on the adverse reactions specific to each class of medication will help with adherence to these medications that are vital in maintaining cardiac function in certain populations. By understanding the basics of beta-blockers and calcium channel blockers, you can advocate for your patients if they experience unwanted or adverse events from these medications. ■

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