ANSWERS TO CHAPTER REVIEW QUESTIONS

1. c Patients often think fluid restriction may be part of diuretic therapy, but that can lead to dehydration.
2. a Beta agonists are used for asthma, and beta blockers are used for hypertension.
3. a This sounds like a trick question. Frequently, questions in patient care matters are too vague to answer unless you can ask more questions and find out more details. For example, is this question asking about central or peripheral sympatholytics? Increased heart rate or increased total peripheral resistance probably wouldn't lower blood pressure but would increase blood pressure.
4. e Pharmacology questions are rarely black and white, and they are hard to ask using a multiple-choice format. Actually, Answer a is also correct. Most diuretics cause hypokalemia, except the K-sparing class ex spironolactone. Maxzide (thiazide/triamterene) is a combination product with a K-sparing and K-losing diuretic together. Diuretics have a "ceiling" effect that is dose related. When you reach that magic dose, the blood pressure doesn't really lower more, but increased side effects show up. Frequently, a diuretic is used with another antihypertensive.
5. a They can cause hyperkalemia and can be used to treat HF. They work at a step in the angiotensin system before the angiotensin II blockers.
6. BP = TPR × CO (blood pressure = total peripheral resistance × cardiac output). Anything that affects CO or TPR could change the blood pressure. Different people have different etiologies for their hypertension. Different drugs work on TPR and CO. Cardiac output is related to heart rate and stroke volume.
7. Concurrent diseases and drugs influence the choice of antihypertensive. Side effects, frequency of dosing, and cost can also affect which drug is used. Diuretics are some of the least expensive medications that have been proven beneficial for morbidity and mortality.
8. Anything that damages the blood vessels can cause platelet aggregation and release of thromboplastin. Antiplatelet agents work to prevent platelet activation. They do this by interfering with platelet aggregation induced by adenosine diphosphate (ADP) or interfering with the synthesis of thromboxane. Thromboplastin, thrombin, and fibrinogen activation result in a clot.
9. Anticoagulants—Prevent clot formation

   Antiplatelets—Prevent formation of the platelet plug and aggregation

   Thrombolytics—Dissolve emboli and thrombi

   A patient may be on each of these agents concurrently for an acute episode such as stroke.
10. You would not want to make a treatment decision based on one blood pressure reading. *Mild* is kind of an outdated term because it is likely to sound nonthreatening to the patient and may result in no action. You would need to know what risk factors the patient has and what target organ disease is present before you could make a lifestyle modification or drug therapy.
11. Pelvic surgery is a risk factor for deep vein thrombosis. Prevention of deep vein thrombosis with low-dose heparin may be indicated. Additional risk factors to look for are age and obesity. Low-dose therapy doesn't require PTT monitoring. Because she has a history of pulmonary embolism, more aggressive prophylaxis may be needed. A PE would be treated with heparin at a dose to prolong the PTT to 1.5 or 2 times the lab control value. She would then be transitioned to warfarin and discharged on warfarin. Warfarin precautions include bleeding, bruising, watching for clot recurrence, such as warm swelling, and shortness of breath. She needs to be cautioned about warfarin drug interactions and the importance of INR lab testing.