Heart failure (HF) is a complex, progressive disorder in which the heart is unable to pump sufficient blood to meet the needs of the body. Its cardinal symptoms are dyspnea, fatigue, and fluid retention. HF is due to an impaired ability of the heart to adequately fill with and/or eject blood. It is often accompanied by abnormal increases in blood volume and interstitial fluid. Underlying causes of HF include arteriosclerotic heart disease, myocardial infarction, hypertensive heart disease, valvular heart disease, dilated cardiomyopathy, and congenital heart disease.

Role of physiologic compensatory mechanisms in the progression of HF

Chronic activation of the sympathetic nervous system and the renin–angiotensin–aldosterone system is associated with remodeling of cardiac tissue, loss of myocytes, hypertrophy, and fibrosis. This prompts additional neurohormonal activation, creating a vicious cycle that, if left untreated, leads to death.

Compensatory physiological responses in HF

The failing heart evokes three major compensatory mechanisms to enhance cardiac output. Although initially beneficial, these alterations ultimately result in further deterioration of cardiac function.

1. Increased sympathetic activity: Baroreceptors sense a decrease in blood pressure and activate the sympathetic nervous system. In an attempt to sustain tissue perfusion, this stimulation of β-adrenergic receptors results in an increased heart rate and a greater force of contraction of the heart muscle. In addition, vasoconstriction enhances venous return and increases cardiac preload.

An increase in preload (stretch on the heart) increases stroke volume, which, in turn, increases cardiac output. These compensatory responses increase the work of the heart, which, in the long term, contributes to further decline in cardiac function.

2. Activation of the renin–angiotensin–aldosterone system:

A fall in cardiac output decreases blood flow to the kidney, prompting the release of renin, and resulting in increased formation of angiotensin II and release of aldosterone. This results in increased peripheral resistance (afterload) and retention of sodium and water. Blood volume increases, and more blood is returned to the heart. If the heart is unable to pump this extra volume, venous pressure increases and peripheral and pulmonary edema occur. Again, these

compensatory responses increase the work of the heart, contributing to further decline in cardiac function.

3. Myocardial hypertrophy: The heart increases in size, and the chambers dilate and become more globular. Initially, stretching of the heart muscle leads to a stronger contraction of the heart However, excessive elongation of the fibers results in weaker contractions, and the geometry diminishes the ability to eject blood. This type of failure is termed "systolic failure" or HF with reduced ejection fraction (HFrEF) and is the result of the ventricle being unable to pump effectively. Less commonly, patients with HF may have "diastolic dysfunction," a term applied when the ability of the ventricles to relax and accept blood is impaired by structural changes such as hypertrophy.

The thickening of the ventricular wall and subsequent decrease in ventricular volume decrease the ability of heart muscle to relax. In this case, the ventricle does not fill adequately, and the inadequacy of cardiac output is termed "diastolic HF" or HF with preserved ejection fraction. Diastolic dysfunction, in its pure form, is characterized by signs and symptoms of HF in the presence of a normal functioning left ventricle. However, both systolic and diastolic dysfunction commonly coexist in HF.

If the adaptive mechanisms adequately restore cardiac output, HF is said to be compensated. If the adaptive mechanisms fail to maintain cardiac output, HF is decompensated and the patient develops worsening HF signs and symptoms. Typical HF signs and symptoms include dyspnea on exertion, orthopnea, paroxysmal nocturnal dyspnea, fatigue, and peripheral edema.

TREATMENT OF CHRONIC HEART FAILURE

Desired Therapeutic Outcomes

There is no cure for HF. The general therapeutic management goals for chronic HF include preventing the onset of clinical symptoms or reducing symptoms, preventing or reducing hospitalizations, slowing progression of the disease, improving quality of life, and prolonging survival.

Nonpharmacologic Interventions

Non pharmacologic treatment involves dietary modifications such as sodium and fluid restriction, risk factor reduction including smoking cessation, and supervised regular physical activity.

- Patient education regarding monitoring symptoms, dietary and medication adherence, exercise and physical fitness, risk factor reduction are important for the prevention of AHF exacerbations. Home monitoring should include daily assessment of weight and exercise tolerance. Daily weights should be done first

thing in the morning upon arising and before any food intake to maintain consistency.

- Nonadherence is an important issue because it relates to acute exacerbations of HF. Ensuring an understanding of the importance of each medication used to treat HF, proper administration, and potential adverse effects may improve adherence.
- Dietary modifications in HF consist of initiation of an AHA step II diet as part of cardiac risk factor reduction, sodium restriction, and sometimes fluid restriction. Exercise, although discouraged when the patient is acutely decompensated and is recommended when patients are stable.
- Modification of classic risk factors, such as tobacco alcohol consumption, is important to minimize the potential for further aggravation of heart function. Patients with HF should be counseled to receive yearly influenza vaccinations. Additionally, a pneumococcal vaccine is recommended.

Pharmacologic Treatment

Diuretics

Diuretics have been the mainstay for HF symptom management for many years. Diuretics are used for relief of acute symptoms of congestion and maintenance of euvolemia. In mild HF, diuretics may be used on an as-needed basis. However, once the development of edema is persistent, regularly scheduled doses will be required. Two types of diuretics are used for volume management in HF: thiazides and loop diuretics. Thiazide diuretics such as hydrochlorothiazide, chlorthalidone, and metolazone block sodium and chloride reabsorption in the distal convoluted tubule. Thiazides are weaker than loop diuretics in effecting an increase in urine output and therefore are not utilized frequently as monotherapy in HF. They are optimally suited for patients with hypertension who have mild congestion. Additionally, the action of thiazides is limited in patients with renal insufficiency (creatinine clearance less than 30 mL/min) due to reduced secretion into their site of action. An exception is metolazone, which retains its potent action in patients with renal dysfunction. Metolazone is often used in combination with loop diuretics when patients exhibit diuretic resistance, defined as edema unresponsive to loop diuretics alone. Oral torsemide can be considered an alternative to the IV route of administration for patients who do not respond to oral furosemide in the setting of profound edema.

Once diuretic therapy is initiated, dosage adjustments are based on symptomatic improvement and daily body weight. Because body weight changes are a sensitive marker of fluid retention or loss, patients should continue to weigh themselves daily. Once a patient reaches a euvolemic state, diuretics may be cautiously tapered and then withdrawn in appropriate patients.

The maximal response to diuretics is reduced in HF leading to diuretic resistance which is due to a compensatory increase in sodium reabsorption in the distal tubules, which decreases the effect of blocking sodium reabsorption in the loop of Henle. Apart from increasing diuretic doses, strategies to improve diuretic efficacy include increasing the

frequency of dosing to two or three times daily, utilizing a continuous infusion of a loop diuretic, and/or combining a loop diuretic with a thiazide diuretic. The latter strategy theoretically prevents sodium and water reabsorption at both the loop of Henle and the compensating distal convoluted tubule. Metolazone is used most often for this purpose because it retains its activity in settings of a low creatinine clearance. Metolazone can be dosed daily or as little as once weekly.

Diuretics side effects

Diuretics cause numerous adverse effects and metabolic abnormalities, with severity linked to diuretic potency. A particularly worrisome adverse effect in the setting of HF is hypokalemia which predispose patients to arrhythmias and sudden death. Hypomagnesemia often occurs concomitantly with diuretic-induced hypokalemia, and therefore both should be assessed and replaced in patients needing correction of hypokalemia. Magnesium is an essential cofactor for movement of potassium intracellularly to restore body stores. Patients taking diuretics are also at risk for renal insufficiency due to over diuresis and reflex activation of the renin-angiotensin system.

ACE inhibitors

Angiotensin-converting enzyme (ACE) inhibitors are a part of standard pharmacotherapy in HF. These drugs block the enzyme that cleaves angiotensin I to form the potent vasoconstrictor angiotensin II. They also diminish the inactivation of bradykinin. Vasodilation occurs as a result of decreased levels of the vasoconstrictor angiotensin II and increased levels of bradykinin (a potent vasodilator). By reducing angiotensin II levels, ACE inhibitors also decrease the secretion of aldosterone.

Actions on the heart: ACE inhibitors decrease vascular resistance (afterload) and venous tone (preload), resulting in increased cardiac output. ACE inhibitors also blunt the usual angiotensin II–mediated increase in epinephrine and aldosterone seen in HF. ACE inhibitors improve clinical signs and symptoms of HF and have been shown to significantly improve patient survival in HF.

Numerous clinical studies show ACE inhibitor therapy is associated with improvements in clinical symptoms, exercise tolerance, LV size and function, and quality of life as compared with placebo. ACE inhibitors significantly reduce hospitalization rates and mortality regardless of underlying disease severity or etiology.

ACE inhibitors are effective in preventing HF development in high-risk patients. Studies in acute MI patients show a reduction in new-onset HF and death with ACE inhibitors whether they are initiated early (within 36 hours) or started later. In addition, ACE inhibition decreases the risk of HF hospitalization and death in patients with asymptomatic LV dysfunction. All patients with documented LV systolic dysfunction, regardless of existing HF symptoms, should receive ACE inhibitors unless a contraindication or intolerance is present.

Contraindications

Absolute contraindications include a history of angioedema, bilateral renal artery stenosis, and pregnancy.

Relative contraindications include unilateral renal artery stenosis, renal insufficiency, hypotension, hyperkalemia, and cough. Relative contraindications provide a warning that close monitoring is required, but they do not necessarily preclude their use.

Side effects:

Hypotension occurs commonly at the initiation of therapy or with dosage increases but may happen anytime. Therefore, in euvolemic patients, diuretic doses may be decreased or withheld during ACE inhibitor dose titration. Initiating at a low dose and titrating slowly can also minimize hypotension. It may be advisable to initiate therapy with a short-acting ACE inhibitor, such as captopril, and subsequently switch to a longer-acting agent, such as lisinopril or enalapril, once the patient is stabilized.

It can be challenging to distinguish an ACE inhibitor– induced cough from cough caused by pulmonary congestion. A productive or wet cough usually signifies congestion, whereas a dry, hacking cough is more indicative of a drug related etiology. If a cough is determined to be ACE inhibitor– induced, its severity should be evaluated before deciding on a course of action. If the cough is truly bothersome, a trial with a different ACE inhibitor or switching to an ARB is warranted. Hyperkalemia is also caused by ACEI so potassium levels must be monitored, particularly with concurrent use of potassium supplements, potassium-sparing diuretics, or aldosterone antagonists due to risk of hyperkalemia.

Angiotensin Receptor Blockers

Angiotensin receptor blockers are considered an equally effective replacement for ACE inhibitors in patients who are intolerant or have a contraindication to an ACE inhibitor. The addition of an ARB to ACE inhibitor therapy can be considered in patients with evidence of disease progression despite optimal ACE inhibitor therapy. Many of the other considerations for the use of ARBs are similar to those of ACE inhibitors, including the need for monitoring renal function, blood pressure, and potassium. Contraindications are similar to those of ACE inhibitors. In patients truly intolerant or contraindicated to ACE inhibitors or ARBs, the combination of hydralazine and isosorbide dinitrate should be considered.

Hydralazine and Isosorbide Dinitrate

The combination of hydralazine and isosorbide dinitrate was the first therapy shown to improve long-term survival in patients with systolic HF, but it has largely been supplanted by angiotensin II antagonist therapy (ACE inhibitors and ARBs). Therefore, until recently, this combination therapy was reserved for patient intolerant to ACE inhibitors or ARBs secondary to renal impairment, angioedema, or hyperkalemia.

β-Adrenergic Antagonists

Chronic β-blockade reduces ventricular mass, improves ventricular shape, and reduces LV end-systolic and diastolic volumes. β-Blockers also exhibit antiarrhythmic effects, slow or reverse catecholamine-induced ventricular remodeling and decrease myocyte

death from catecholamine-induced necrosis or apoptosis. Consequently, β-blockers improve EF, reduce all-cause and HF-related hospitalizations, and decrease all-cause mortality in patients with systolic HF.

It is important that the β-blocker be initiated when a patient is clinically stable and euvolemic. Volume overload at the time of β-blocker initiation increases the risk for worsening symptoms. β-Blockade should begin with the lowest possible dose after which the dose may be doubled every 2 to 4 weeks depending on patient tolerability.

Side effects

β-Blockers may cause an acute decrease in left ventricular ejection fraction (LVEF) and short-term worsening of HF symptoms upon initiation and at each dosage titration. After each dose titration, if the patient experiences symptomatic hypotension, bradycardia, or worsening symptoms, further increases in dose should be withheld until the patient stabilizes. After stabilization, attempts to increase the dose should be reinstituted. If mild congestion ensues as a result of the β-blocker, an increase in diuretic dose may be warranted. If moderate or severe symptoms of congestion occur, a reduction in β-blocker dose should be considered along with an increase in diuretic dose.

Selection of B blockers

Apart from possible clinical differences between the β-blockers approved for HF, selection of a β-blocker may also be affected by pharmacologic differences. **Carvedilol** exhibits a more pronounced blood pressure lowering effect, and thus causes more frequent dizziness and hypotension as a consequence of its β1 and α1-receptor blocking activities.

Therefore, in patients predisposed to symptomatic hypotension, such as those with advanced LV dysfunction (LVEF less than 20% [0.20]) who normally exhibit low systolic blood pressures, **metoprolol succinate** may be the more desirable first-line β-blocker. In patients with uncontrolled hypertension, carvedilol may provide additional antihypertensive efficacy.

A selective β1-blocker such as metoprolol is a reasonable option for patients with reactive airway disease. The risk versus benefit of using any β-blocker in peripheral vascular disease must be weighed based on the severity of the peripheral disease, and a selective β1-blocker is preferred.

Aldosterone Antagonists

Currently, the aldosterone antagonists available are spironolactone and eplerenone. Each agent (spironolactone and eplerenone) has been studied in a defined population of patients with HF. Both Effective in reducing HF hospitalizations, sudden cardiac death, and improving all-cause mortality.

The major risk related to aldosterone antagonists is hyperkalemia. Before and within 1 week of initiating therapy, two parameters must be assessed: serum potassium and creatinine clearance (or serum creatinine).

Aldosterone antagonists should not be initiated in patients with potassium concentrations greater than 5.5 mEq/L (5.5 mmol/L). Likewise, these agents should not

be given when creatinine clearance is less than 30 mL/minute or serum creatinine is greater than 2.5 mg/dL (221 μmol/L).

In patients without contraindications, spironolactone is initiated at a dose of 12.5 to 25 mg daily, or occasionally on alternate days for patients with baseline renal insufficiency.

Eplerenone is used at a dose of 25 mg daily, with the option to titrate up to 50 mg daily. Doses should be halved or switched to alternate-day dosing if creatinine clearance falls below 50 mL/min.

Potassium supplementation is often decreased or stopped after aldosterone antagonists are initiated, and patients should be counseled to avoid high potassium foods. At any time after initiation of therapy, if potassium concentrations exceed 5.5 mEq/L (5.5 mmol/L), the dose of the aldosterone antagonist should be reduced or discontinued. In addition, worsening renal function dictates consideration for stopping the aldosterone antagonist.

Adverse effects

Other adverse effects observed mainly with spironolactone include gynecomastia for men and breast tenderness and menstrual irregularities for women. Gynecomastia leads to discontine in up to 10% of patients on spironolactone.

Digoxin

The exact role of digoxin in therapy remains controversial largely due to disagreement on the risk versus benefit of routinely using this drug in patients with systolic HF. Digoxin was shown to decrease HF-related hospitalizations but did not decrease HF progression or improve survival. Moreover, digoxin was associated with an increased risk for concentration-related toxicity and numerous adverse effects.

Current recommendations are for the addition of digoxin for patients who remain symptomatic despite an optimal HF regimen consisting of an ACE inhibitor or ARB, β-blocker, and diuretic. In patients with concomitant atrial fibrillation, digoxin may be added to slow ventricular rate regardless of HF symptoms.

Dosing

Digoxin is initiated at a dose of 0.125 mg to 0.25 mg daily depending on age, renal function, weight, and risk for toxicity. The lower dose should be used if the patient satisfies any of the following criteria: older than 65 years, creatinine clearance less than 60 mL/min (1.0 mL/s), or ideal body weight less than 70 kg (154 lb). The 0.125-mg daily dose is adequate in most patients.

Antiplatelets and Anticoagulation

Aspirin is generally used in HF patients with an underlying ischemic etiology, a history of ischemic heart disease, or other compelling indications such as history of embolic stroke. If aspirin is indicated, the preference is to use a low dose (81 mg daily).

Current consensus recommendations support the use of warfarin in patients with reduced LV systolic dysfunction and a compelling indication such as atrial fibrillation or prosthetic heart valves. In addition, warfarin is empirically used in patients with

evidence of a mural thrombus or severely depressed (LVEF less than 20%) LV function.

Heart Failure with Preserved Left Ventricular Ejection Fraction

Treatment goal

(a) Correction or control of underlying etiologies (including optimal treatment of hypertension and CAD and maintenance of normal sinus rhythm).

(b) Reduction of cardiac filling pressures at rest and during exertion.

(c) Increased diastolic filling time. Diuretics are frequently used to control congestion.

Therapeutic options

Recent studies failed to show significant reductions in mortality or hospitalizations with the use of ARBs. B-Blockers and calcium channel blockers can theoretically improve ventricular relaxation through negative inotropic and chronotropic effects. Unlike in systolic HF, non-dihydropyridine calcium channel blockers (diltiazem and verapamil) may be especially useful in improving diastolic function by limiting the availability of calcium that mediates contractility.

Outcome Evaluation of Chronic Heart Failure

1. If diuretic therapy is warranted, monitor for therapeutic response by assessing weight loss and improvement of fluid retention, as well as exercise tolerance and presence of fatigue.
2. Once therapy for preventing disease progression is initiated, monitoring for symptomatic improvement continues.
3. It is important to keep in mind that patients' symptoms of HF can worsen with β-blockers, and it may take weeks or months before patients notice improvement
4. Monitor blood pressure to evaluate for hypotension caused by drug therapy.

Acute heart failure

Acute heart failure syndromes (AHFS) may be defined as new-onset, gradual, or rapidly worsening HF signs and symptoms that require urgent therapy. These symptoms reflect congestion behind the failing ventricle and/or hypoperfusion. Patients can be categorized into hemodynamic subsets based on assessment of physical signs and symptoms of congestion and/or hypoperfusion. Patients can be categorized depending on volume status, as well as based on adequacy of tissue perfusion.

Patients with AHFS can be further classified as having new-onset or worsening chronic HF. Approximately 80% of patients with AHFS have chronic HF. Patients with advanced HF have low blood pressure (BP), renal impairment, and signs or symptoms refractory to standard medical therapy and represent up to 10% of hospitalized patients with AHFS.

Clinical presentation of acute heart failure

Subset I

• Patients considered well compensated and perfused, without evidence of congestion

• No immediate interventions necessary except optimizing oral medications and monitoring

Subset II

• Patients adequately perfused and display signs and symptoms of congestion

• Main goal is to reduce preload (PCWP) carefully with loop diuretics and vasodilators

Subset III

• Patients are inadequately perfused and not congested

• Hypoperfusion leads to increased mortality, elevating death rates fourfold compared with those who are adequately perfused

• Treatment focuses on increasing CO with positive inotropic agents and/or replacing intravascular fluids

• Fluid replacement must be performed cautiously because patients can rapidly become congested

Subset IV

• Patients are inadequately perfused and congested

• Classified as the most complicated clinical presentation of AHF with the worst prognosis

• Most challenging to treat; therapy targets alleviating signs and symptoms of congestion by increasing CI as well as reducing PCWP while maintaining adequate mean arterial pressure

• Treatment involves a delicate balance between diuretics, vasodilators, and inotropic agents

• Use of vasopressors is sometimes necessary to maintain blood pressure.

Laboratory Assessment

BNP, electrolytes and blood glucose, serum creatinine and blood urea nitrogen to assess renal function. Complete blood cell count is measured to determine if anemia or infection is present. Creatine kinase and/or troponin concentrations are used to diagnose ischemia, and hepatic transaminases are measured to assess hepatic congestion. Thyroid function tests are measured to assess hyperthyroidism or hypothyroidism as causes of AHF. A urinalysis is attained in patients with an unknown history of renal disease to rule out nephrotic syndrome.

TREATMENT OF ACUTE HEART FAILURE

Desired Therapeutic Outcomes

The goals of therapy for AHF are to (a) correct the underlying precipitating factor(s); (b) relieve the patient's symptoms; (c) improve hemodynamics; (d) optimize a chronic oral medication regimen; and (e) educate the patient, reinforcing adherence to lifestyle modifications and the drug regimen.

The ultimate goal for a patient hospitalized for AHF is the return to a compensated HF state and discharge to the outpatient setting on oral medications. Only through

aggressive management to achieve all of these goals will a patient's prognosis be improved and future hospitalizations for acute decompensations be prevented.
Oral agents such as β-blockers, ACE inhibitors or ARBs, and aldosterone antagonists should be initiated as soon as possible during the hospitalization. These chronic oral medications not only improve mortality and prevent readmissions, acutely they also contribute to improvement in hemodynamics. Patient education prior to discharge from the hospital is recommended to assist in minimizing adverse effects and nonadherence.

Pharmacologic Approaches to Treatment
Treatment of AHF targets relief of congestion and optimization of CO utilizing oral or IV diuretics, IV vasodilators, and, when appropriate, inotropes, based on presenting hemodynamics. Current treatment strategies in AHF target improving hemodynamics while preserving organ function.

Diuretics
Loop diuretics, including furosemide, bumetanide, and torsemide, are the diuretics of choice in the management of AHF. Furosemide is the most commonly used agent. Diuretics decrease preload by functional venodilation within 5 to 15 minutes of administration and subsequently by an increase in sodium and water excretion. This provides rapid improvement in symptoms of pulmonary congestion.
Diuretics is recommended to be administered by intravenous route in AHF. Bolus injection should be administered at a rate not exceeding 4 mg per minute to avoid ototoxicity

Patients who received double their regular oral diuretic dose by intravenous route experienced more weight loss, diuresis and subsequent symptom relief compared to those who receive intravenous doses equivalent to their oral doses. Relatively higher doses of diuretics are needed in patients with renal impairment because they need adequate glomerular filtration to reach their site of action. For example if creatinine clearance is more than 75 ml/min, infusion rate of intravenous furosemide is 10 mg/hour. However, in patients with creatinine clearance less than 25 ml/min, the dosing rate would be 20 mg increased to 40 mg/hour. Lower doses are needed if the patient improves.

Occasionally, patients with HF do not respond to a diuretic, defined as failure to achieve a weight reduction of at least 0.5 kg after several increasing bolus doses can be managed by either increasing the dose of the diuretics, switching to intravenous infusion or addition of oral diuretic with a different mechanism of action such as thiazide (hydrochlorothiazide, bendroflumethiazide) or thiazide like diuretics (metolazone) to counteract diuretic resistance. The addition of these diuretics provides synergistic diuretic effect by preventing sodium uptake from the distal tubule. It should be noted that this co-administration of these diuretics increases the risk of electrolyte abnormalities (hypokalaemia, hyponatremia) and renal dysfunction. Therefore, the renal function and electrolytes should be closely monitored. Combining diuretics should be used with caution due to an increased risk for cardiovascular collapse due to

rapid intravascular volume depletion. Strict monitoring of electrolytes, vital signs, and fluid balance is warranted.

Careful use of diuretics is recommended to avoid over diuresis. Monitoring parameters for diuretics includes: ↓HF symptoms, Signs of volume depletion (Weakness Hypotension, dizziness Orthostatic changes in BP, ↓Urine output ↑BUN), Serum potassium and magnesium (avoid hypokalemia and hypomagnesemia), ↑Uric acid, ↑Glucose, Weight loss.
Finally, poor CO may contribute to diuretic resistance. In these patients, it may become necessary to add vasodilators or inotropes to enhance perfusion to the kidneys. Care must be taken because vasodilators can decrease renal blood flow despite increasing CO through dilation of central and peripheral vascular beds.

Vasodilators

IV vasodilators cause a rapid decrease in arterial tone, resulting in a decrease in SVR and a subsequent increase in SV and CO. Additionally, vasodilators reduce ventricular filling pressures within 24 to 48 hours, reduce myocardial oxygen consumption, and decrease ventricular workload. Vasodilators are commonly used in patients presenting with AHF accompanied by moderate to severe congestion. This class includes nitroglycerin, nitroprusside, and nesiritide. Usual doses and monitoring of commonly used hemodynamic medications: BP, HR, urinary output and kidney function, ECG, extremity perfusion.
IV nitroglycerin is primarily used as a preload reducer for patients exhibiting pulmonary congestion or in combination with inotropes for congested patients with severely reduced CO. Continuous infusions of nitroglycerin should be initiated at a dose of 5 to 10 mcg/min and increased every 5 to 10 minutes until symptomatic or hemodynamic improvement. Effective doses range from 35 to 200 mcg/min.

Inotropic Agents
There are several practical considerations to dobutamine therapy in AHF. First, owing to its vasodilatory potential, monotherapy with dobutamine is reserved for patients with systolic blood pressures greater than 90 mm Hg. However, it is commonly used in combination with vasopressors in patients with lower systolic blood pressures.
In patients on β-blocker therapy, it is recommended that consideration be given to the use of phosphodiesterase inhibitors such as milrinone, which do not depend on β-receptors for effect.
Dopamine is most commonly reserved for patients with low systolic blood pressures and those approaching cardiogenic shock. As with other inotropes, dopamine is associated with a risk for arrhythmias.