EMERGENCY MEDICINE REPORTS

Practical, Evidence-Based Reviews in Emergency Care

JULY 1, 2017 VOL. 38, NO. 13

AUTHORS

Leslie Anne Juarbe Rivera, MD, MPH, Emergency Medicine Surgical Critical Care Fellowship, R Adams Cowley Shock Trauma Center, University of Maryland, Baltimore.

Jay Menaker, MD, Associate Professor of Surgery and Emergency Medicine, University of Maryland School of Medicine; Medical Director, Lung Rescue Unit; Medical Director, Critical Care Resuscitation Unit, R Adams Cowley Shock Trauma Center, Baltimore, MD.

PEER REVIEWER

Steven M. Winograd, MD, FACEP, Assistant Professor of Clinical Emergency Medicine, New York Institute of Technology College of Osteopathic Medicine, Old Westbury, NY; St. John's Riverside, Yonkers, NY; CityMD, New York City.

STATEMENT OF FINANCIAL DISCLOSURE

To reveal any potential bias in this publication, and in accordance with Accreditation Council for Continuing Medical Education guidelines, we disclose that Dr. Farel (CME question reviewer) owns stock in Johnson & Johnson. Ms. Light (nurse planner) reports that she serves as a consultant for Bard Medical. Brian Hocum, PharmD, (pharmacist reviewer) reports that he is an employee of United Therapeutics. Dr. Schneider (editor), Dr. Stapczynski (editor), Dr. Juarbe Rivera (author), Dr. Menaker (author), Dr. Winograd (peer reviewer), Ms. Mark (executive editor), Ms. Coplin (executive editor), and Ms. Hatcher (AHC Media editorial group manager) report no financial relationships with companies related to the field of study covered by this CME activity.

AHC Media

A RELIAS LEARNING COMPANY

Systolic and Diastolic Heart Failure

Introduction

Acute decompensated heart failure is a serious condition that presents in the emergency department and the intensive care unit. It is associated with mortality rates of 4% to 11% during hospitalization and 20% to 36% during the first year after discharge.[1,2] The causes of heart failure are multifactorial, making it, at times, difficult to diagnose and treat. However, with modern technological advancements, clinicians are becoming more efficient at identifying and treating this disease.

The American Heart Association (AHA)/American College of Cardiology (ACC) defines heart failure as "a complex clinical syndrome that can result from any structural or functional cardiac disorder that impairs the ability of the ventricle to fill or eject blood."[3] This definition emphasizes that the diagnosis is largely a clinical one that is based on a careful history and physical examination, which can make the condition a challenge to identify.[3] Symptoms and signs of heart failure develop when the heart no longer has the ability to pump the amount of blood needed to satisfy the demands of the various organ systems. Once heart failure is diagnosed, patients require medication for the rest of their life to improve quality of life and survival.[4]

Heart failure can be classified into different categories. These include acute vs. chronic, left vs. right sided, high output vs. low output, and systolic vs. diastolic heart failure. This article will focus on systolic and diastolic heart failure classification, which is the most commonly used.

Epidemiology

Heart failure continues to be one of the most common reasons for hospital admissions in the United States, especially among the elderly.[5,6] It currently has a prevalence of more than 5.8 million in the United States and more than 23 million worldwide.[7] In the United States alone, more than 500,000 new cases of heart failure are reported each year.[8] It is estimated that nearly 300,000 people die annually from heart failure in the United States.[8]

EXECUTIVE SUMMARY

- Diastolic heart failure is more common than systolic heart failure in elderly patients.
- The primary risk factor for heart failure is hypertension.
- The diagnosis of heart failure is primarily through clinical assessment, supplemented by ancillary testing.
- Patients with diastolic heart failure are sensitive to volume changes, and over-diuresis will reduce stroke volume and decrease renal function.
- The emergent treatment of acute heart failure is guided by the blood pressure.

The epidemiology of diastolic heart failure differs somewhat from systolic heart failure. In various retrospective studies, the reported incidence of diastolic heart failure varies between 20-40%.[9,10] On the other hand, all the studies have shown that the incidence increases with age, and more so in elderly women. Why this disease is seen more in the elderly population and in elderly women is still unanswered. A five-year mortality of 50% was observed in those with diastolic heart failure, which is similar to that of primary systolic heart failure.[9,11] Every time patients with either systolic or diastolic heart failure are re-hospitalized, they have a 20-22% increase in their mortality.[12]

Minority groups, such as blacks, hispanics, and Native Americans, have a higher incidence and prevalence of heart failure. Presumably, this is because these groups also have the highest incidence and prevalence of hypertension and type 2 diabetes.[13]

Risk Factors

The two major risk factors for developing heart failure, specifically for the elderly, are, first, hypertension, and second, coronary artery disease (CAD).[8] Understandably there can be other etiologies, such as diabetes mellitus, valvular heart disease (especially aortic stenosis and mitral regurgitation), and nonischemic cardiomyopathies.[8] These are all diseases that affect the coronary microcirculation, causing chronic coronary insufficiency that leads to ischemic cardiomyopathy and ventricular remodeling.[14]

Lifestyle choices also can increase risk factors for heart failure, especially if one has any of the above-mentioned diseases. Unhealthy choices — such as smoking; eating foods high in fat, cholesterol, and sodium; not exercising enough; and being obese — are modifiable factors that increase the risk for heart disease.

Pathophysiology of Systolic and Diastolic Heart Failure

Systolic heart failure is defined as "a clinical syndrome associated with congestive symptoms and/or symptoms of low cardiac output due to impaired ventricular pump function (reduced EF)." By general consensus, impaired ventricular pump function in systolic heart failure is defined as an ejection fracture approximately < 45%. Diastolic heart failure occurs when there is increased resistance to the diastolic filling of part or all of the heart but systolic function (ejection fraction > 45%) is still preserved.[6]

It is not uncommon to see patients with diastolic heart failure who also suffer from hypertension and left ventricular hypertrophy (LVH).[6] The proportion of heart failure that is primarily diastolic increases with age, from about 45% in patients younger than 45 years to almost 60% in patients older than 85 years.[14] Studies have shown that 50% of older patients with heart failure may have isolated diastolic dysfunction.[16] It also is important to remember that the left ventricle becomes stiffer with age.

There are several issues involved in the pathophysiology of diastolic heart failure. One is impaired relaxation that affects diastolic filling of the ventricle. Of interest, impaired diastolic relaxation is the first manifestation of myocardial ischemia, seen before contractile abnormalities in left ventricular wall motion are present. The sarcoplasmic reticulum calcium ATPase pump (SERCA) is responsible for relaxation. Thus, decreased SERCA leads to impaired relaxation. This can be seen in patients who suffer from LVH secondary to hypertension or aortic stenosis. Both the levels of SERCA and diastolic function decrease with age.[6] Impaired relaxation also can be seen in patients with myocyte hypertrophy cardiomyopathies and hypothyroidism.

Along with impaired relaxation, one also can have increased stiffening of the heart or passive stiffness. Passive stiffness can be seen in patients with postinfarction scarring, myocyte hypertrophy, and infiltrative cardiomyopathies, such as amyloidosis. Diffuse fibrosis seems to play a part in this, as pathology studies have documented increased serum markers of collagen turnover linked to passive stiffness.[6] Another reason for heart failure includes processes that cause increased resistance to diastolic filling to all or part of the heart, such as those found in endocardial and pericardial disorders including mitral stenosis and tamponade.[6]

Microvascular flow and its effect on extravascular compression also can lead to a pathological process that can increase left ventricle diastolic pressures. Elevated left ventricle diastolic pressure will act mainly on capillaries and small resistance coronary vessels, possibly affecting the autoregulation and the ability to vasodilate. Also, myocardial turgor, which results from engorgement of the microvasculature with blood, can cause an increase in diastolic stiffness.[6]

The final physiologic process to consider is the neurohormonal regulation, specifically regarding the renin-angiotensin system. This system contributes to the development of diastolic heart failure by promoting hypertension and by diminishing myocardial relaxation.

Common Signs and Symptoms

Patients with heart failure can present with a variety of signs and symptoms, such as fatigue, dyspnea on exertion, paroxysmal nocturnal dyspnea, orthopnea, jugular venous distention, rales, tachycardia, third or fourth heart sounds, hepatomegaly, and edema.[16]

The cardinal symptom of heart failure is shortness of breath, initially manifested when increased cardiac output is needed to provide oxygen to active muscles, as with exertion. As heart failure progresses, dyspnea occurs with less stress, as provoked by fluid shifts when recumbent, and in the final stages, shortness of breath is seen at rest.

There is an imprecise correlation with myocardial function and clinical features. In general, a larger number of findings with greater severity are found in patients with severely impaired myocardial function, but also, population-based echocardiograph studies have found that as many as half of patients with depressed left ventricular ejection fraction (< 35-40%) have no definitive signs or symptoms of heart failure.[17] Any of the signs mentioned above also can be accompanied by chest pain or pressure and palpitations, which are the more traditional cardiac symptoms.

Classes and Stages of Heart Failure

The New York Heart Association (NYHA) classification is used to categorize the stages of congestive heart failure based on the patient's functional capacity, from asymptomatic to limited activity secondary to dyspnea at rest. The classifications are:

- Class I — Asymptomatic on ordinary physical activity;
- Class II — Symptomatic on ordinary physical activity;
- Class III — Symptomatic on less than ordinary physical activity;
- Class IV — Symptomatic at rest.[14]

The American College of Cardiology/American Heart Association (ACC/AHA) created guidelines for the evaluation and management of heart failure based on four stages of the disease.[8] The ACC/AHA stages are:

- Stage A — High risk for heart failure, without structural heart disease or symptoms;
- Stage B — Has structural heart disease related to the development of heart failure but has never shown symptoms or signs of heart failure;
- Stage C — Current or prior symptoms of heart failure associated with structural heart disease;
- Stage D — Advanced structural heart disease and marked symptoms of heart failure at rest despite maximal medical therapy and requires specialized interventions.[8]

Diagnosis of Diastolic and Systolic Heart Failure

Diagnosis of heart failure is ultimately a clinical one, but objective evidence can be obtained via echocardiography or cardiac catheterization. While systolic heart failure has a measurable value that characterizes impaired left ventribular systolic function — an ejection fraction < 35-40% — there is no correspoinding ejection fraction value or criteria to diagnose diastolic heart failure. To make matters more interesting, diastolic heart failure usually accompanies systolic heart failure, so a depressed ejection fraction cannot exclude diastolic heart failure.

The gold standard for diagnosing diastolic heart failure is cardiac catheterization, which shows increased ventricular diastolic pressure with preserved systolic function and normal ventricular volumes.[6] If during cardiac catheterization micromanometer catheters are placed in the left ventricle, impaired LV diastolic relation can be assessed by determining the peak negative change in intracavitary presssure (dP/dt) and the time constant of LV relaxation (tau).

Cardiac catheterization continues to be an invasive procedure that is not without risk. That is why echocardiography is a more attractive approach to assist in the diagnosis of systolic heart failure while simultaneously being able to evaluate blood flow across the valves and ventricular and valvular functions.

The brain natriuretic peptide (BNP) is a test that can help differentiate between heart failure and other causes of acute dyspnea, such as chronic obstructive lung disease.[18] It is important to emphasize that elevated levels of BNP do not distinguish between diastolic and systolic heart failure. Instead they have predictive value specifically regarding increased mortality and readmission to the hospital, in addition to having a sensitivity and specificity in the diagnosis of heart failure of about 85%.[19] For most clinical purposes, diastolic heart failure is defined as clinical signs and symptoms of heart failure in the presence of preserved left ventricular systolic function (ejection fraction > 45%).[9,20]

During an episode of acute heart failure decompensation, most interventions focus only on the precipitating factors, and usually the prognosis is much better compared to the decompensation of a chronic heart failure patient.[14]

Some argue that the diagnosis of acute heart failure decompensation is one that is mostly clinical, and, therefore, a thorough physical examination is more reliable than ancillary tests. It has been publicized that the clinical impression of heart failure has a specificity of 0.86 but has limited sensitivity of 0.61.[21] The same can be said about the use of chest radiograph findings, such as pulmonary venous congestion and interstitial edema, although they are more specific.[21]

Heart failure has some traditional signs and symptoms, which include peripheral edema, elevated jugular venous pressure, an S3 gallop, rales, dyspnea, and orthopnea.[21,22,23] Although all patients are different, most clinicians look for the classic sign of dyspnea and the patient being "warm and wet" upon arrival to the emergency department. The signs and symptoms with the greatest specificity (0.92-0.99) but poor sensitivity (0.13-0.39) are a third heart sound, hepatojugular reflux, and elevated jugular venous pulse.[21,24]

Chronic obstructive pulmonary disease (COPD), with a prevalence of 20-30% in patients with heart failure, can conceal the recognition of heart failure.[14] This is because of the chronic increase in pulmonary arterial pressure and the changes (hypertrophy and dilation) to the right ventricle as a form of compensation.

It is important to consider other causes that may contribute to fluid retention or mimic symptoms of heart failure, such as severe anemia or kidney

Figure 1. B Lines (arrow) on Lung Ultrasound

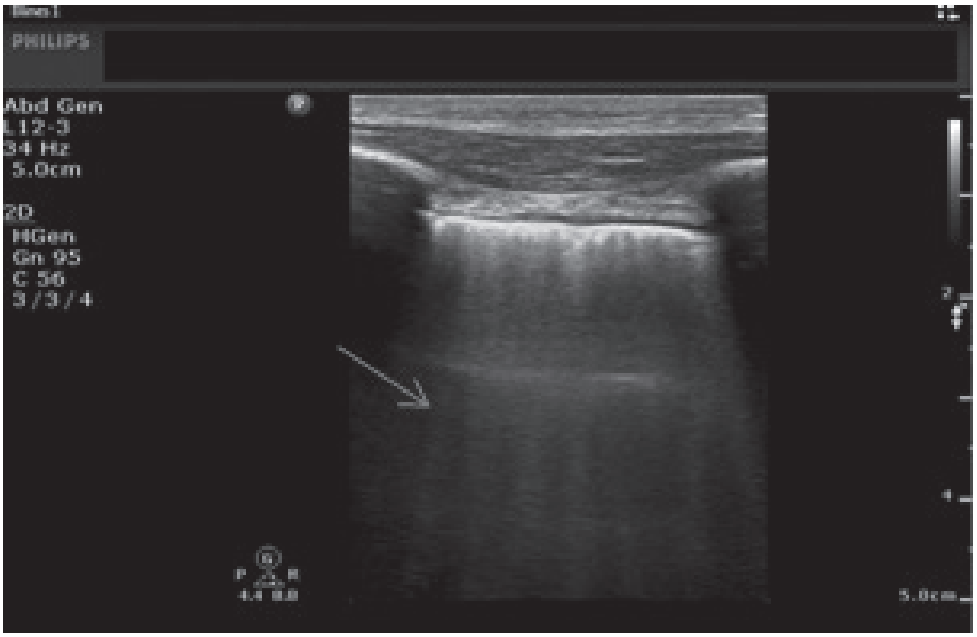


Image courtesy of Daniel Haase, MD, RDCS, RDMS, Assistant Professor of Emergency Medicine, Program in Trauma/Surgical Critical Care, Shock Trauma Critical Care Ultrasonography, University of Maryland School of Medicine and R Adams Cowley Shock Trauma Center.

failure. This can be achieved by conducting some initial routine laboratory tests such as a complete blood count, a complete metabolic panel, and a hepatic panel.

Lung ultrasound, for B-line evaluation, is an evolving practice that is being used for identification of extravascular lung water.[25] (*See Figure 1.*) Detection of B-line on a lung ultrasound can identify acute decompensated heart failure with high sensitivity and specificity in an emergency setting.[25] (*See Figure 2.*)

Treatment for Systolic Heart Failure

The ideal outpatient treatment of systolic heart failure has three goals that should be followed simultaneously: control of risk factors for the development and progression of heart failure, treatment of heart failure symptoms, and education of patients.[26]

Control of risk factors involves treating hypertension, diabetes, and coronary artery disease, and eliminating the use of alcohol and tobacco. Symptoms of volume overload may be controlled with the use of diuretics and restricted dietary sodium. Diuretics, specifically furosemide, still continue to be the first-line drug in the treatment of heart failure and volume overload.[8] While diuretics decrease venous return, reduce ventricular filling pressures, increase fluid loss, and decrease symptoms of pulmonary and systemic congestion and edema, no decrease in mortality has been observed with the use of these drugs.[8]

All patients with heart failure should be taking an angiotensin-converting enzyme (ACE) inhibitor or angiotensin-receptor blocker (ARB). ACE inhibitors work by decreasing both afterload and preload. In addition to decreasing systemic vascular resistance and myocardial oxygen consumption, ACE inhibitors also increase cardiac output. Significant evidence shows that ACE inhibitors reduce morbidity and mortality when used as a first-line therapy in patients who have heart failure with reduced ejection fraction.[27]

ACC/AHA guidelines recommend that ARBs be used in patients with heart failure if they develop an intolerance to ACE inhibitors. Some examples of ACE inhibitor side effects are cough, worsening kidney function, angioedema, or increasing blood potassium levels. Randomized, controlled trials have shown that ARBs can reduce morbidity and mortality, especially in patients who do not tolerate ACE inhibitors.[27]

Beta-blockers should be considered by physicians as an adjuvant to ACE-inhibitor therapy. Beta-blockers not only decrease the heart muscle's oxygen requirement but also can help slow the progression of the disease.[26] It is important to remember that beta-blockers are not a rescue drug for patients who have acute decompensated heart failure. Patients with heart failure and a history of prior myocardial infarction with or without asymptomatic low LV ejection fraction can benefit from a treatment regimen that includes both ACE inhibitors and beta-blockers.[8,28] The reason behind this is that the combination of both medications can help patients reduce the incidence of new coronary events and heart failure.[8]

In patients with severe heart failure, spironolactone can be added to the treatment regimen. Digoxin comes

Figure 2. B Lines (arrow) and Comet Tail on Lung Ultrasound

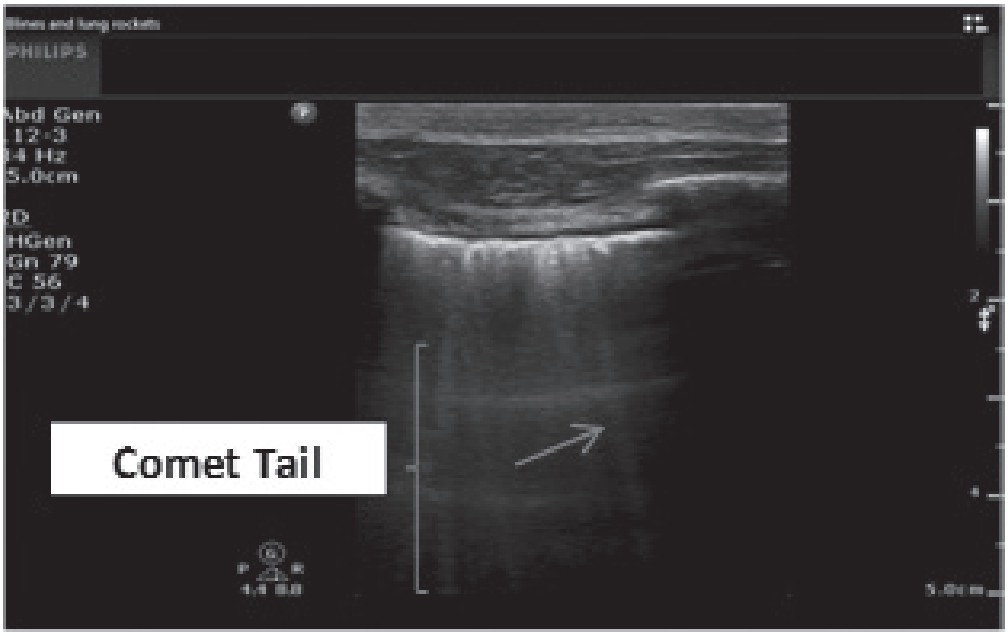


Image courtesy of Daniel Haase, MD, RDCS, RDMS, Assistant Professor of Emergency Medicine, Program in Trauma/Surgical Critical Care, Shock Trauma Critical Care Ultrasonography, University of Maryland School of Medicine and R Adams Cowley Shock Trauma Center.

into play when a patient continues to have symptoms despite already being treated with diuretics, ACE inhibitors, and beta-blockers.[29] Digoxin also can be used with patients who have dyspnea at rest or a recent history of dyspnea at rest.[29] It is important to state that digoxin therapy may reduce the probability of hospitalization but does not reduce mortality.[30] One of the drawbacks of digoxin is that it must be monitored closely, with a target dosage level of 0.5 to 1.1 ng per mL.[26]

In patients with moderate to severe heart failure, a low left ventricular ejection fraction, and a QRS duration on the resting ECG of 120 ms or more, cardiac resynchronization therapy (CRT) has been shown to provide significant clinical improvement.[8,31,32,33]

The ultimate option for patients who have exhausted all medical management and/or device therapy is a heart transplant. Unfortunately, there are more patients with end-stage heart failure that are eligible for heart transplant than the current available number of donor hearts. In this circumstance, ventricular assist devices (VADs) can be used as a bridge pathway while the patient waits to make a decision, awaits recovery, or waits to be eligible.[34] The main advantage of VADs and/or other mechanical circulatory support (MCS) is the improvement of organ perfusion and function, which in turns leads to better quality of life and increased survival. However, despite the benefits, VAD implantation is not without risk, and it is associated with the risk of serious complications, such as bleeding, infection, arrhythmias, blood clots, right ventricular failure, and cardiovascular events.

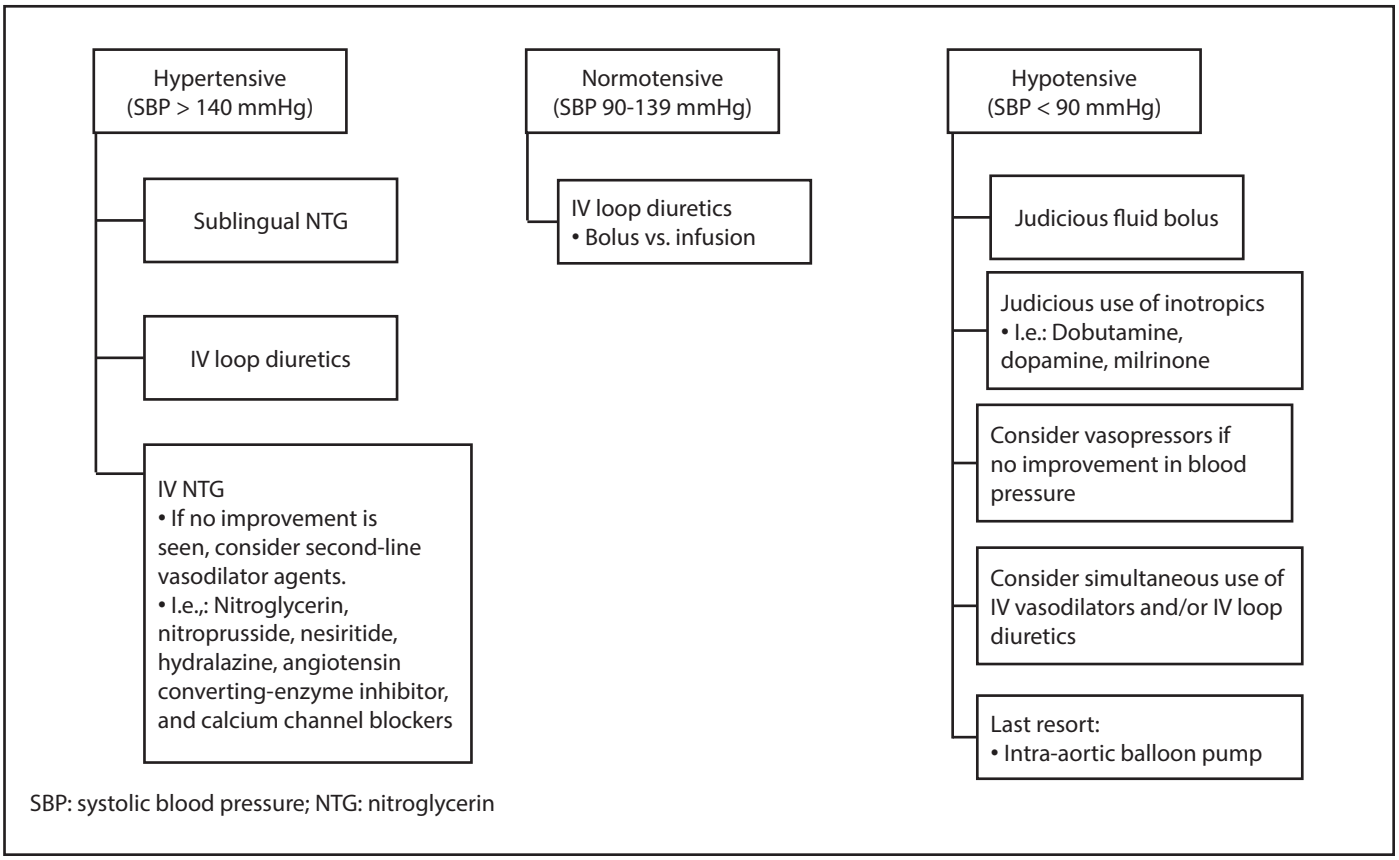
Treatment for Diastolic Heart Failure

The treatment of diastolic heart failure is less well defined than the treatment of systolic heart failure. When pharmacologic treatments of both diastolic and systolic heart failure are compared superficially, the impression is that they do not vary significantly. However, current recommendations for diastolic heart failure treatment are based on the disease pathophysiology, knowledge about other aspects of cardiovascular disease, data from small studies, and expert opinion. Unfortunately, to this author's knowledge, none of the treatment recommendations have been validated by prospective randomized, controlled trials.

Currently, the best available treatment for diastolic decompensated heart failure is built around the concept of modifying the disease pathophysiology, such as relieving volume overload, decreasing heart rate, prolonging diastolic filling time, restoring atrial contraction, blocking the renin-angiotensin system, blocking aldosterone, controlling hypertension, and relieving ischemia.[6]

Diuretics and vasodilators often are used to reduce volume overload that presents as pulmonary congestion. However, aggressive diuresis can be dangerous; over-diuresis of the patient with decompensated heart failure will decrease the patient's preload and stroke volume and impair renal function. Most patients with diastolic dysfunction are highly sensitive to volume changes and preload.

Figure 3. General Outline for Treatment of Acute Heart Failure



SBP: systolic blood pressure; NTG: nitroglycerin

Beta-blockers are another option, with their ability to decrease heart rate, increase diastolic filling time, decrease oxygen consumption, lower blood pressure, and cause reversion of left ventricular hypertrophy. ACE inhibitors have some similarities to the benefits of betablockers, such as the ability to decrease blood pressure and cause regression of left ventricular hypertrophy. While ACE inhibitors are potentially beneficial, there have been few studies of patients with diastolic heart failure who use ACE inhibitors as part of their treatment regimen.[35]

It is theorized that calcium channel blockers may be more beneficial because these medications can also decrease oxygen demand (similar to beta-blockers) and dilate coronary arteries, in addition to decreasing blood pressure. However, there are not enough data available that measure patient-centric outcomes such as morbidity and mortality. Thus, calcium channel blockers should be used with caution in patients with both systolic and diastolic heart failure.

Management of Acute Heart Failure in the Emergency Department

The treatment for acute heart failure has not changed meaningfully in the last four decades. Medications such as loop diuretics and vasodilators still are used as the primary treatment for acute heart failure. However, one new addition to the management of heart failure is the use of noninvasive ventilation in cardiogenic pulmonary edema. Studies have shown that early use in these patients can prevent the need for endotracheal intubation.[21,36]

Initial treatment of acute heart failure is most influenced by the blood pressure. (*See Figure 3.*) About 50% or more of patients who arrive at the emergency department with acute heart failure have an elevated blood pressure, about 40% have a normal blood pressure, and only 5% have a low blood pressure.[21,37,38] For intial treatment decisions, the three primary blood pressure divisions are: hypertensive (SBP > 140 mmHg), normotensive (SBP 139-90 mmHg), and hypotensive (SBP < 90 mmHg).[21,39] It is important to stress that these cutoff points are intended as guidelines and are not absolute thresholds.[21]

Patients who are hypertensive should be treated with the goal of vasodilation instead of concentrating on fluid removal. The symptoms of patients with hypertension are most likely related to volume re-distribution, as opposed to volume overload.[21,40,41] Examples of medications used for initial vasodilation are nitroglycerin (most commonly used), nitroprusside, nesiritide, hydralazine, ACE inhibitors, and calcium channel blockers.[21] (*See Table 1.*)

Nitroglycerin has a rapid onset and clearance, in addition to being inexpensive. It can be used both sublingually and intravenously. Nitroglycerin can be used in higher dosages and still be both safe and effective.[21,42,43] Furthermore, lower rates of intubation and myocardial

Table 1. Drugs Used to Treat Acute Heart Failure

Agent	Mechanism	Dosing	Comments
Nitroglycerin SL	Direct vasodilator	0.4 to 1.2 mg SL every 5 minutes	Can be initiated while IV access is being obtained
Nitroglycerin IV	Direct vasodilator	Start 10 mcg/min IV, increase by 5 mcg/min every 3 to 5 minutes until dyspnea improves or dose is 20 mcg/min, then increase in increments of 10 to 20 mcg/min Max dose 200 mcg/min	Typical dose is 100 mcg/min for resolution of dyspnea and control of blood pressure
Nitroprusside	Vasodilator	Start 10 mcg/min IV, increase by 10 mcg every 10 minutes according to clinical response	Typical dose is 200 to 300 mcg/min IV for control of dyspnea and blood pressure
Nesiritide	Recombinant form of BNP; vasodilator	2 mcg/kg IV loading dose, followed by 0.01 mcg/kg per min IV infusion, increase by 0.005 mcg/kg per min increments every 3 hours to 0.03 mcg/kg per min	Stop if hypotension develops
Furosemide	Loop diuretic	40 mg IV bolus or 40 mg/h infusion over 1 hour	May repeat in 1 hour
Enalaprilat	ACE inhibitor vasodilator	0.625 to 1.25 mg IV over 5 minutes every 6 hours	Consensus guidelines do not encourage early use in acute heart failure
Hydralazine	Direct vasodilator	10 to 20 mg IV	May repeat in 2 hours
Dobutamine	Beta-agonist inotropic	Start infusion at 1 to 2 mcg/kg per min IV and increase according to clinical response Max dose 20 mcg/kg per min	Fewer adverse side effects than milrinone
Milrinone	Type III phosphodiesterase inhibitor; vasodilator	50 mcg/kg IV loading dose, followed by 0.375 mcg/kg per min IV infusion, increase according to clinical response Max dose 0.75 mcg/kg per min	Consider in cases of acute heart failure associated with severe pulmonary hypertension

infarction, as well as improvement in heart rate, respiratory rate, and oxygen saturation have been seen with high-dose nitrates when compared to lower doses.[21,42,43]

The normotensive patient is usually the one that has chronic heart failure with reduced ejection fraction that decompensates; however, the symptoms might not be as obvious as in the patient with hypertensive acute heart failure. Generally, these patients are maxed out on their outpatient oral diuretic therapy when they present with exacerbations. The goal for these patients should be to focus on decongestion through fluid removal via IV loop diuretics, which is the first-line treatment. There are debates regarding the most effective way to administer loop diuretics in acute heart failure exacerbation, either as a bolus or continuous infusion.[21,44]

Less common (< 5%) is the hypotensive patient. It is important to remember that patients without any previous history of heart failure who present with cardiogenic shock most likely have a life-threatening etiology, such as a myocardial infarction.[21] In addition, it is important to assess the patient's volume status. The most important goal in the treatment of this type of patient is to improve perfusion, not simply just to raise their blood pressure. Current guidelines recommend the use of inotropes such as dobutamine and milrinone only for those patients who truly require them, meaning patients who present with hypotension and acute heart failure. Inotropic therapy can help in the short term but is associated with worsening morbidity and mortality.[21,45,46]

Lifestyle Modification

Lifestyle modifications are important in the long-term management of patients with heart failure. And physicians should provide advice and encourage patients to modify their lifestyle in addition to prescribing appropriate medications. In some patients, lifestyle modifications and weight loss may be so effective as to enable reduction or even complete elimination of daily medication. This is why all patients with stable heart failure should be encouraged to begin and maintain a regular exercise program. Specifically, to patients classified at stage A of heart failure, according to the ACC/AHA Classification.[5,8] In the 1970s, patients who complained of fatigue and/or shortness of breath related to exertion were not encouraged to exercise. This also applied to patients with heart failure or those who had suffered from a heart attack. However, in the late 1980s, it was understood that these patients suffered from deconditioning and muscle atrophy from the lack of activity.

Exercise in patients with heart failure

has multiple benefits that include improvement in the muscle tolerance to exertion, neuro-hormonal changes that favor cardiac hemodynamics, and overall improvement of symptoms of heart failure.[47] Moreover, it is proven that exercise not only improves symptoms but also decreases the number of admissions to the hospital and improves survival in patients with heart failure.[48]

Patients can control their fluid status and, consequently, their heart failure symptoms by simply restricting sodium intake to 2 g or less per day.[26] There are no studies available regarding the effect in morbidity and mortality of dietary sodium restriction, either alone or in combination with pharmacological treatment. Moderate exercise (i.e., at 60% of maximum exercise capacity on a stationary bicycle for two to three hours per week) improves quality of life, decreases mortality, and decreases hospital readmissions for heart failure in patients with stable chronic heart failure.[49]

Conclusion

Heart failure is a major health issue and will continue to be so because of the growing elderly population. Diastolic heart failure can be diagnosed in a patient with clinical symptoms of heart failure and preserved systolic function, regardless of the absence of a laboratory and echocardiographic findings. The BNP measurement can increase the accuracy of diagnosis. Further randomized therapeutic studies are needed regarding the treatment of diastolic heart failure. Currenlty, physicians should focus on treating aspects of the pathophysiology that are modifiable, such as reducing volume overload, decreasing the heart rate, controlling hypertension, and relieving myocardial ischemia.[20] Patients need to be educated on the importance of making good lifestyle choices, such as healthy eating and medication compliance.

References

1. Ahmed A, Allman RM, Fonarow GC, et al. Incident heart failure hospitalization and subsequent mortality in chronic heart failure: A propensity-matched study. *J Card Fail* 2008;14:211-218.
2. Solomon SD, Dobson J, Pocock S, et al. Influence of nonfatal hospitalization for heart failure on subsequent mortality in patients with chronic heart failure. *Circulation* 2007;116:1482-1487.
3. Hunt SA, Abraham WT, Chin MH, et al. ACC/AHA 2005 Guideline Update for the Diagnosis and Management of Chronic Heart Failure in the Adult: A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to Update the 2001 Guidelines for the Evaluation and Management of Heart Failure): Developed in collaboration with the American College of Chest Physicians and the International Society for Heart and Lung Transplantation: Endorsed by the Heart Rhythm Society. *Circulation* 2005;112:e154.
4. American Heart Association. Heart Disease and Stroke Facts, 2006 Update. Dallas, Texas: AHA, 2006.
5. Azad N, Lemay G. Management of chronic heart failure in the older population. *J Geriatr Cardiol* 2014;11:329-337.
6. Angeja BG, Grossman W. Evaluation and management of diastolic heart failure. *Circulation* 2003;107:659-663.
7. Roger VL. Epidemiology of heart failure. *Circulation Research* 2013;113:646-659.
8. Aronow WS. Epidemiology, pathophysiology, prognosis, and treatment of systolic and diastolic heart failure. *Cardiol Rev* 2006;14:108-124.
9. Chatterjee K. Primary diastolic heart failure. *Am J Geriatric Cardiol* 2002;11:178-189.
10. Dauterman KW, Massie BM, Gheorghiade M. Heart failure associated with preserved systolic function: A common and costly clinical entity. *Am Heart J* 1998;135:S310-S319.
11. Senni M, Tribouilloy CM, Rodeheffer RJ, et al. Congestive heart failure in the community: A study of all incident cases in Olmsted County, Minnesota, in 1991. *Circulation* 1998;98:2282-2289.
12. Roger VL, Go AS, Lloyd-Jones DM, et al, for the American Heart Association Statistics Committee and Stroke Statistics Subcommittee. Heart disease and stroke statistics—2011 update: A report from the American Heart Association. *Circulation* 2011;123:e18-e209.
13. Dumitru I. Heart failure. Medscape. Jan. 11, 2016. Available at: http://emedicine.medscape.com/article/163062-overview#a5. Accessed May 1, 2017.
14. O'Brien JF, Hunter CL. Chapter 81: Heart failure. In: Marx J, Hockberger R, Walls R, eds. *Rosen's Emergency Medicine – Concepts and Clinical Practice*, 8th ed. Philadelphia, PA: Elsevier Saunders; 2013:1075.
15. Chapter 53. Acute heart failure. In: Tintinalli JE, Stapczynski JS, Ma OJ, eds. *Tintinalli's Emergency Medicine: A Comprehensive Study Guide*, 7th ed. New York, NY: McGraw-Hill Education: 2011.
16. Satpathy C, Mishra TK. Diagnosis and management of diastolic dysfunction and heart failure. *Am Fam Physisican* 2006;73:841-846.
17. Mosterd A, Hoes AW. Clinical epidemiology of heart failure. *Heart* 2007;93:1137-1146.
18. Morrison LK, Harrison A, Krishnaswamy P, et al. Utility of a rapid B-natriuretic peptide assay in differentiating congestive heart failure from lung disease in patients presenting with dyspnea. *J Am Coll Cardiol* 2002;39:202-209.
19. Lubien E, DeMaria A, Krishnaswamy P, et al. Utility of B-natriuretic peptide in detecting diastolic dysfunction: Comparison with Doppler velocity recordings. *Circulation* 2002;105:595-601.
20. Askoxylakis V, Thieke C, Pleger ST, et al. Long-term survival of cancer patients compared to heart failure and stroke: A systematic review. *BMC Cancer* 2010;10:105.

21. Pang PS, Levy P, Shah SJ. Treatment of acute heart failure in the emergency department. *Expert Rev Cardiovasc Ther* 2013;11: 1195-1209.
22. Gheorghiade M, Filippatos G, De Luca L, Burnett J. Congestion in acute heart failure syndromes: An essential target of evaluation and treatment. *Am J Med* 2006;119(12Suppl1):S3-S10.
23. Gheorghiade M, Pang PS. Acute heart failure syndromes. *J Am Coll Cardiol* 2009;53:557-573.
24. Zehtabchi S, Brandler ES. Evidence-based emergency medicine/rational clinical examination abstract. Does this patient have congestive heart failure? *Ann Emerg Med* 2008;51:87-90.
25. Aggarwal M, Gupta M, Vijan V, et al. Use of lung ultrasound for diagnosing acute heart failure in emergency department of southern India. *J Clin Diagn Res* 2016;10: TC05–TC08.
26. McConaghy JR, Smith SR. Outpatient treatment of systolic heart failure. *Am Fam Physician* 2004;70:2157-2164.
27. Yancy CW, Jessup M, Bozkurt B, et al. 2013 ACCF/AHA guideline for the management of heart failure: A report of the American College of Cardiology Foundation/American Heart Association task force on practice guidelines. *J Am Coll Cardiol* 2013;62:e147-e239.
28. Brophy JM, Joseph L, Rouleau JL. Beta-blockers in congestive heart failure. A Bayesian meta-analysis. *Ann Intern Med* 2001;134:550.
29. Chavey WE 2nd, Blaum CS, Bleske BE, et al. Guideline for the management of heart failure caused by systolic dysfunction: Part II. Treatment. *Am Fam Physician* 2001;64: 1045-1054.
30. Ahmed A. Digoxin and reduction in mortality and hospitalization in geriatric heart failure: Importance of low doses and low serum concentrations. *J Gerontol A Biol Sci Med Sci* 2007;62:323-329.
31. Aronow WS. CRT plus ICD in congestive heart failure. Use of cardiac resynchronization therapy and an implantable cardioverter–defibrillator in heart failure patients with abnormal left ventricular dysfunction. *Geriatrics* 2005;60:24-28.
32. Young JB, Abraham WT, Smith AL, et al. Combined cardiac resynchronization and implantable cardioversion defibrillation in advanced chronic heart failure: The MIRACLE ICD Trial. *JAMA* 2003;289:2685-2694.
33. Bristow MR, Saxon LA, Boehmer J, et al. Cardiac-resynchronization therapy with or without an implantable defibrillator in advanced chronic heart failure. *N Engl J Med* 2004;350:2140-2150.
34. Szyguła-Jurkiewicz B, Szczurek W, Suliga K, et al. Mechanical circulatory support in heart failure. *Kardiochir Torakochiururgia Pol* 2016;13:130-134.
35. Chavey WE 2nd, Blaum CS, Bleske BE, et al. Guideline for the management of heart failure caused by systolic dysfunction: Part II. Treatment. *Am Fam Physician* 2001;64: 1045-1055.
36. Vital FM, Saconato H, Ladeira MT, et al. Non-invasive positive pressure ventilation (CPAP or bilevel NPPV) for cardiogenic pulmonary edema. *Cochrane Database Syst Rev* 2008;(3):CD005351.
37. Adams KF Jr, Fonarow GC, Emerman CL, et al. Characteristics and outcomes of patients hospitalized for heart failure in the United States: Rationale, design, and preliminary observations from the first 100,000 cases in the Acute Decompensated Heart Failure National Registry (ADHERE). *Am Heart J* 2005;149:209-216.
38. Gheorghiade M, Abraham WT, Albert NM, et al. Systolic blood pressure at admission, clinical characteristics, and outcomes in patients hospitalized with acute heart failure. *JAMA* 2006;296:2217-2226.
39. Collins S, Storrow AB, Kirk JD, et al. Beyond pulmonary edema: Diagnostic, risk stratification, and treatment challenges of acute heart failure management in the emergency department. *Ann Emerg Med* 2008;51:45-57.
40. Cotter G, Felker GM, Adams KF, et al. The pathophysiology of acute heart failure — is it all about fluid accumulation? *Am Heart J* 2008;155:9-18.
41. Cotter G, Metra M, Milo-Cotter O, et al. Fluid overload in acute heart failure — re-distribution and other mechanisms beyond fluid accumulation. *Eur J Heart Fail* 2008;10: 165-169.
42. Cotter G, Metzkor E, Kaluski E, et al. Randomised trial of high-dose isosorbide dinitrate plus low-dose furosemide versus high-dose furosemide plus low-dose isosorbide dinitrate in severe pulmonary oedema. *Lancet* 1998;351:389-393.
43. Breidthardt T, Noveanu M, Potocki M, et al. Impact of a high-dose nitrate strategy on cardiac stress in acute heart failure: A pilot study. *J Intern Med* 2010;267:322-330.
44. Felker GM, O'Connor CM, Braunwald E, et al.; Heart Failure Clinical Research Network Investigators. Loop diuretics in acute decompensated heart failure: Necessary? Evil? A necessary evil? *Circ Heart Fail* 2009;2:56-62.
45. Hunt SA, Abraham WT, Chin MH, et al. 2009 Focused update incorporated into the ACC/AHA 2005 Guidelines for the diagnosis and management of heart failure in adults: A report of the American College of Cardiology Foundation/ American Heart Association task force on practice guidelines developed in collaboration with the International Society for Heart and Lung Transplantation. *J Am Coll Cardiol* 2009;53:e1-e90.
46. Felker GM, Benza RL, Chandler AB, et al. Heart failure etiology and response to milrinone in decompensated heart failure: Results from the OPTIME-CHF study. *J Am Coll Cardiol* 2003;41:997-1003.

47. Working Group on Cardiac Rehabilitation & Exercice Physiology and Working Group on Heart Failure of the European Society of Cardiology. Recommendations for exercise training in chronic heart failure patients. *Eur Heart J* 2001;22: 125-135.
48. McKelvie RS, Teo KK, McCartney W, et al. Effects of exercise training in patients with congestive heart failure: A critical review. *J Am Coll Cardiol* 1995;25:789.
49. Khand A, Gemmel I, Clark AL, Cleland JG. Is the prognosis of heart failure improving? *J Am Coll Cardiol* 2000;36:2284-2286.

CE/CME Questions

1. Which population has the most admissions to the hospital due to heart failure?
   a. Elderly
   b. Men
   c. Women
   d. Children
2. Which factor is most significant in the development of heart failure?
   a. Cardiac valve disease
   b. Peripheral artery disease
   c. Hypertension
   d. Coronary artery disease
3. What is the second most common risk factor for heart failure?
   a. Previous history of heart attack
   b. Peripheral artery disease
   c. Hypertension
   d. Coronary artery disease
4. What ejection fraction is defined as systolic heart failure?
   a. < 35%
   b. < 40%
   c. < 45%
   d. < 50%
5. Echocardiographic population-based studies have observed that about half of individuals with a reduced ejection fraction (< 45%)

EMERGENCY MEDICINE REPORTS

CME/CE Objectives

Upon completion of this educational activity, participants should be able to:

- recognize specific conditions in patients presenting to the emergency department;
- apply state-of-the-art diagnostic and therapeutic techniques to patients with the particular medical problems discussed in the publication;
- discuss the differential diagnosis of the particular medical problems discussed in the publication;
- explain both the likely and rare complications that may be associated with the particular medical problems discussed in the publication.

have no signs or symptoms of heart failure.
a. True
b. False

6. What is the cardinal symptom associated with heart failure?
a. Shortness of breath
b. Leg swelling
c. Tachycardia
d. Weakness

7. Which symptom are diuretics are used to control in the treatment of heart failure?
a. Weakness
b. Bradycardia
c. Tachycardia
d. Fluid retention

8. What is the primary advantage of mechanical circulatory support?
a. Decrease in shortness of breath
b. Improvement of organ perfusion and function
c. Decrease in outpatient medication consumption
d. Permanently eliminates the need for heart transplant

9. To which of the following are patients with diastolic dysfunction particularly sensitive?
a. Volume changes and preload
b. Hypotension
c. Tachycardia
d. Bradycardia

10. Restricting sodium intake to which of the following amounts per day can help in controlling fluid status and heart failure symptoms?
a. 1 g
b. 2 g
c. 3 g
d. 4 g

CME/CE INSTRUCTIONS

To earn credit for this activity, please follow these instructions:

1. Read and study the activity, using the references for further research.
2. Log onto AHCMedia.com and click on My Account. *First-time users must register on the site.*
3. Pass the online tests with a score of 100%; you will be allowed to answer the questions as many times as needed to achieve a score of 100%.
4. After successfully completing the test, a credit letter will be emailed to you instantly.
5. Twice yearly after the test, your browser will be directed to an activity evaluation form, which must be completed to receive your credit letter.

EMERGENCY MEDICINE REPORTS™
(ISSN 0746-2506) is published twice per month by AHC Media, a Relias Learning company, 111 Corning Road, Suite 250, Cary, NC 27518. Telephone: (800) 688-2421.

Executive Editor: Shelly Morrow Mark

Executive Editor: Leslie Coplin

AHC Media Editorial Group Manager: Terrey L. Hatcher

Senior Accreditations Officer: Lee Landenberger

GST Registration No.: R128870672

Periodicals Postage Paid at Atlanta, GA 30304 and at additional mailing offices.

POSTMASTER: Send address changes to ***Emergency Medicine Reports,*** AHC Media, LLC, P.O. Box 74008694 Chicago, IL 60674-8694.

Back issues: $30. Missing issues will be fulfilled by customer service free of charge when contacted within one month of the missing issue's date.

SUBSCRIBER INFORMATION

CUSTOMER SERVICE: (800) 688-2421

Customer Service Email Address:
Customer.Service@AHCMedia.com

Editorial E-Mail Address:
mmark@reliaslearning.com

Online:
AHCMedia.com

SUBSCRIPTION PRICES

1 year with 72 ACEP/72 AMA/36 AAFP Category 1/Prescribed credits: $508

1 year *without* credit: $419
Add $19.99 for shipping & handling

MULTIPLE COPIES:
Discounts are available for group subscriptions, multiple copies, site-licenses, or electronic distribution. For pricing information, please contact our Group Account Managers at Groups@AHCMedia.com or (866) 213-0844.

AHC Media
A RELIAS LEARNING COMPANY

ACCREDITATION

Relias Learning is accredited by the Accreditation Council for Continuing Medical Education (ACCME) to provide continuing medical education for physicians.

Relias Learning designates this enduring material for a maximum of 3.0 *AMA PRA Category 1 Credits™*. Physicians should claim only credit commensurate with the extent of their participation in the activity.

Approved by the American College of Emergency Physicians for a maximum of 3.0 hour(s) of ACEP Category I credit.

This Enduring Material activity, *Emergency Medicine Reports*, has been reviewed and is acceptable for credit by the American Academy of Family Physicians. Term of approval begins 01/01/2017. Term of approval is for one year from this date. Physicians should claim only the credit commensurate with the extent of their participation in the activity. Approved for 1.5 AAFP Prescribed credits.

The American Osteopathic Association has approved this continuing education activity for up to 2.5 AOA Category 2-B credits.

Relias Learning LLC is accredited as a provider of continuing nursing education by the American Nurses Credentialing Center's Commission on Accreditation. Contact hours [3.0] will be awarded to participants who meet the criteria for successful completion. California Board of Registered Nursing, Provider CEP#13791.

This is an educational publication designed to present scientific information and opinion to health professionals, to stimulate thought, and further investigation. It does not provide advice regarding medical diagnosis or treatment for any individual case. It is not intended for use by the layman. Opinions expressed are not necessarily those of this publication. Mention of products or services does not constitute endorsement. Clinical, legal, tax, and other comments are offered for general guidance only; professional counsel should be sought for specific situations.

This CME/CE activity is intended for emergency and family physicians and nurses. It is in effect for 36 months from the date of the publication.

EMERGENCY MEDICINE **REPORTS**

Systolic and Diastolic Heart Failure

B Lines (arrow) on Lung Ultrasound

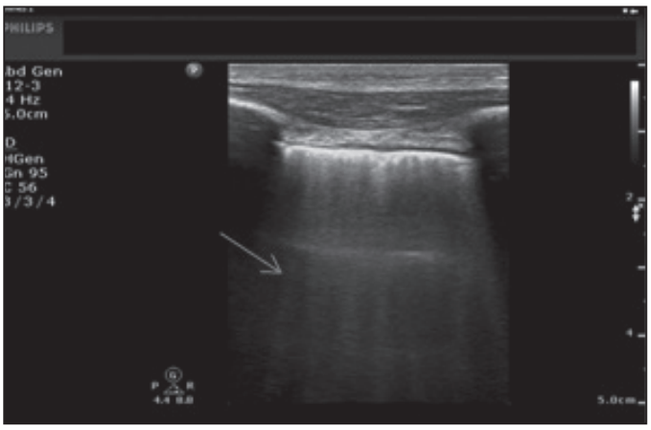


Image courtesy of Daniel Haase, MD, RDCS, RDMS, Assistant Professor of Emergency Medicine, Program in Trauma/Surgical Critical Care, Shock Trauma Critical Care Ultrasonography, University of Maryland School of Medicine and R Adams Cowley Shock Trauma Center.

B Lines (arrow) and Comet Tail on Lung Ultrasound

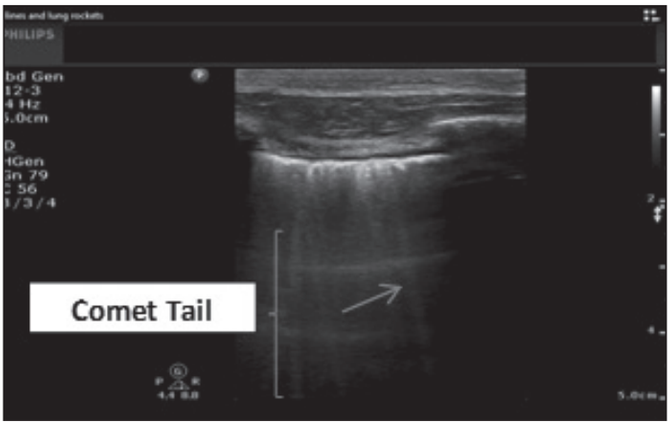
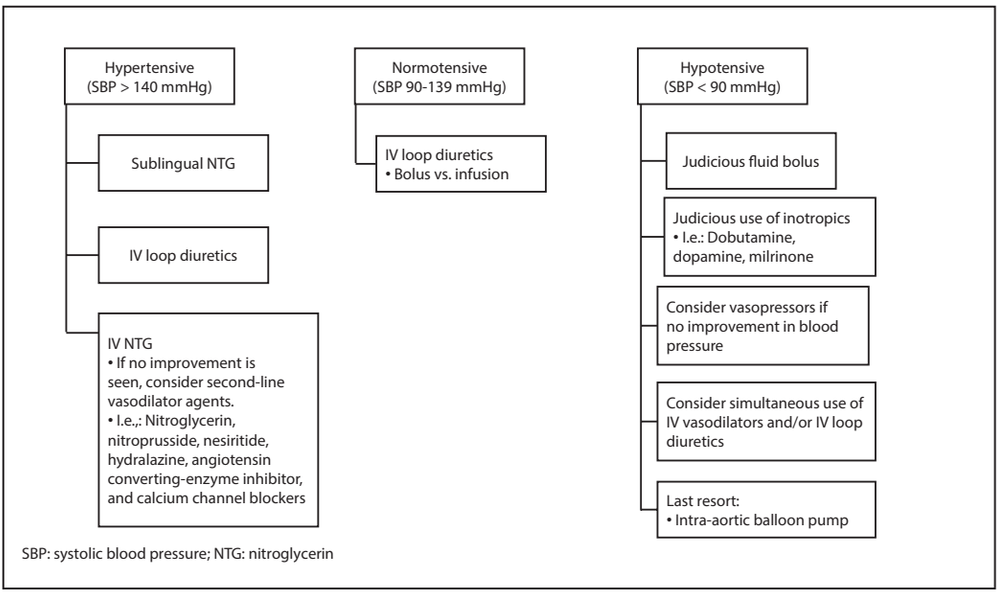


Image courtesy of Daniel Haase, MD, RDCS, RDMS, Assistant Professor of Emergency Medicine, Program in Trauma/Surgical Critical Care, Shock Trauma Critical Care Ultrasonography, University of Maryland School of Medicine and R Adams Cowley Shock Trauma Center.

General Outline for Treatment of Acute Heart Failure



SBP: systolic blood pressure; NTG: nitroglycerin

Drugs Used to Treat Acute Heart Failure

Agent	Mechanism	Dosing	Comments
Nitroglycerin SL	Direct vasodilator	0.4 to 1.2 mg SL every 5 minutes	Can be initiated while IV access is being obtained
Nitroglycerin IV	Direct vasodilator	Start 10 mcg/min IV, increase by 5 mcg/min every 3 to 5 minutes until dyspnea improves or dose is 20 mcg/min, then increase in increments of 10 to 20 mcg/min Max dose 200 mcg/min	Typical dose is 100 mcg/min for resolution of dyspnea and control of blood pressure
Nitroprusside	Vasodilator	Start 10 mcg/min IV, increase by 10 mcg every 10 minutes according to clinical response	Typical dose is 200 to 300 mcg/min IV for control of dyspnea and blood pressure
Nesiritide	Recombinant form of BNP; vasodilator	2 mcg/kg IV loading dose, followed by 0.01 mcg/kg per min IV infusion, increase by 0.005 mcg/kg per min increments every 3 hours to 0.03 mcg/kg per min	Stop if hypotension develops
Furosemide	Loop diuretic	40 mg IV bolus or 40 mg/h infusion over 1 hour	May repeat in 1 hour
Enalaprilat	ACE inhibitor vasodilator	0.625 to 1.25 mg IV over 5 minutes every 6 hours	Consensus guidelines do not encourage early use in acute heart failure
Hydralazine	Direct vasodilator	10 to 20 mg IV	May repeat in 2 hours
Dobutamine	Beta-agonist inotropic	Start infusion at 1 to 2 mcg/kg per min IV and increase according to clinical response Max dose 20 mcg/kg per min	Fewer adverse side effects than milrinone
Milrinone	Type III phosphodiesterase inhibitor; vasodilator	50 mcg/kg IV loading dose, followed by 0.375 mcg/kg per min IV infusion, increase according to clinical response Max dose 0.75 mcg/kg per min	Consider in cases of acute heart failure associated with severe pulmonary hypertension

Supplement to *Emergency Medicine Reports*, July 1, 2017: "Systolic and Diastolic Heart Failure." Authors: Leslie Anne Juarbe Rivera, MD, MPH, Emergency Medicine Surgical Critical Care Fellowship, R Adams Cowley Shock Trauma Center, University of Maryland, Baltimore; Jay Menaker, MD, Associate Professor of Surgery and Emergency Medicine, University of Maryland School of Medicine; Medical Director, Lung Rescue Unit; Medical Director, Critical Care Resuscitation Unit, R Adams Cowley Shock Trauma Center, Baltimore, MD.