

Clinical Diagnosis of Heart Failure

Muralikrishna Gopal, MBBS

Bernard Karnath, MD

Hear failure is a common clinical syndrome that results from the impaired ability of the ventricle to fill with or eject blood. The term *heart failure* is preferred over the older term *congestive heart failure* because not all patients with heart failure have volume overload. Heart failure results from multiple causes, including coronary artery disease, hypertension, valvular heart disease, and idiopathic dilated cardiomyopathy. The cardinal symptoms of heart failure include dyspnea and fatigue that can occur at rest in severe cases and with exertion in milder cases. The diagnosis of heart failure was traditionally made at the bedside based on clinical evaluation that combined characteristic symptoms from the history with various signs on physical examination. Other than the obvious need to determine whether a patient has heart failure, it is also important to determine what type of heart failure is present. Patients with heart failure need a comprehensive workup that begins with the history and physical examination.

Medical education and residency training have undergone many changes in the last few decades, partly because of the advent of modern technologies. Training increasingly is focused on use of technologies, namely echocardiography in the diagnosis of cardiac conditions. With the increased focus on technology, less emphasis has been placed on the physical examination. A recent study demonstrated the poor cardiovascular examination skills of residents and pointed out that cardiac examination skills do not improve after the third year of medical school.¹ This article reviews the approach to diagnosing heart failure, with a focus on the signs and symptoms. A brief discussion of laboratory evaluation is also included.

EPIDEMIOLOGY

Heart failure is a relatively common condition encountered in clinical practice. Although the incidence of heart failure has been stable over the past 2 decades, it remains a substantial health burden.^{2,3} Heart failure is the most frequent cause of hospitalization in persons

EVALUATION OF HEART FAILURE

- Coronary artery disease is believed to be the underlying cause of heart failure in approximately two thirds of patients with heart failure.
- The cardinal symptoms of heart failure include dyspnea and fatigue that can occur at rest in severe cases and with exertion in milder cases.
- For the dyspneic patient in the acute care setting, a history, physical examination, chest radiograph, and electrocardiogram should be performed.
- The presence of a third heart sound is a strong predictor of heart failure, with a specificity of 99%.
- If heart failure is suspected, measurement of serum brain natriuretic peptide may help exclude heart failure.

aged 65 years or older.² Other negative outcomes associated with heart failure include a poor quality of life, frequent hospitalizations, medication side effects, and an increase in morbidity and mortality. The mortality rate associated with heart failure may be underestimated, but it is approximately 50% at 2 years and 70% at 3 years.⁴ However, over the past 50 years survival after the onset of heart failure has improved, probably due to more effective treatment of hypertension, coronary heart disease, and valvular disease and increasing use of pharmacologic therapies.⁵

CLASSIFICATION SYSTEMS

The most commonly cited heart failure classification system is the New York Heart Association (NYHA) classification (Table 1).⁶ The NYHA classification system is

Dr. Gopal is a resident, and Dr. Karnath is an associate professor of medicine, Division of General Internal Medicine, University of Texas Medical Branch, Galveston, TX.

Table 1. New York Heart Association Classification for Stages of Heart Failure

Class	Symptoms
Class I (mild)	Ordinary physical activity does not cause symptoms
Class II (mild)	Comfortable at rest but ordinary physical activity results in symptoms
Class III (moderate)	Comfortable at rest but less than ordinary activity causes symptoms
Class IV (severe)	Symptoms at rest

based on the patient's symptoms obtained only by the history and does not rely on objective measures. The NYHA classification also predicts mortality. According to one study, the estimated 1-year mortality among patients with NYHA heart failure classes II, III, and IV was 7%, 15%, and 28%, respectively.⁷ A newer approach to the classification of heart failure was presented by the American College of Cardiology and the American Heart Association (**Table 2**).^{8,9} Unlike the NYHA classification system, this newer approach takes into account objective measures, risk factors, and subjective symptoms.

SYMPTOMS

Dyspnea is defined as an uncomfortable awareness of breathing.¹⁰ It is a subjective sensation for which there is no accurate objective measurement. Dyspnea is usually the result of a cardiac or pulmonary disease process, although healthy individuals may experience dyspnea with exercise. The most common pathological causes of dyspnea include asthma, congestive heart failure, chronic obstructive pulmonary disease, pneumonia, and cardiac ischemia.¹¹ The mechanisms of dyspnea are not well understood.

Dyspnea, especially with exertion, is one of the most common symptoms of heart failure, and it frequently appears early in the disease. In the early stages of heart failure, dyspnea occurs with severe exertion, but as the heart failure worsens the amount of exertion required to produce dyspnea progressively decreases. According to a study that assessed the features of heart failure, the absence of dyspnea on exertion essentially ruled out the presence of heart failure due to left ventricular dysfunction.¹² Dyspnea on exertion has a sensitivity of 84% to 100% in the diagnosis of heart failure, but the specificity is much lower, ranging from 17% to 34%.^{12,13}

Dyspnea presents in other forms, including orthopnea and paroxysmal nocturnal dyspnea (PND). Orthopnea is the worsening of dyspnea on assuming

Table 2. American College of Cardiology/American Heart Association Stages of Heart Failure

Stage	Description
A	Patients at high risk for developing heart failure due to underlying conditions such as systemic hypertension, coronary artery disease, diabetes mellitus, or family history of cardiomyopathy
B	Patients have structural heart disease such as left ventricular hypertrophy or valvular heart disease but show no symptoms of heart failure
C	Patients have current or prior symptoms of heart failure associated with underlying structural heart disease that includes left ventricular systolic dysfunction
D	Patients with advanced structural heart disease and marked symptoms of heart failure at rest despite maximal medical therapy

Adapted from the ACC/AHA Guidelines for the Evaluation and Management of Chronic Heart Failure in the Adult: Executive Summary A Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee to Revise the 1995 Guidelines for the Evaluation and Management of Heart Failure). *Circulation* 2001;104:2996–3007.

the supine position. Orthopnea has a reported sensitivity of 22% to 50% and a specificity of 74% to 77% for heart failure.^{12,13} Patients with heart failure require increased numbers of pillows to sleep at night in order to avoid symptoms of orthopnea. The number of pillows required can be used to gauge the severity of heart failure. PND refers to attacks of shortness of breath that occur at night and may awaken the patient from sleeping. The sensitivity and specificity of PND for heart failure range from 39% to 41% and from 80% to 84%, respectively.^{12,13}

Advanced or end-stage heart failure can present with an abnormal pattern of breathing characterized by periods of hyperpnea and apnea called Cheyne-Stokes respiration. Several factors contribute to Cheyne-Stokes respiration, including hyperventilation from pulmonary congestion and hypoxia; hyperventilation leads to a low arterial CO₂ level, which triggers a central apnea once the apneic threshold is reached. This cycle further stresses the failing heart.¹⁴ In addition, patients with heart failure have a prolonged circulation time, leading to delays in transmitting changes in arterial blood gas measurements to the carotid body chemoreceptors. This circulatory delay influences the length of hyperpnea.¹⁴ Cheyne-Stokes respiration is a marker of poor prognosis in patients with heart failure, especially if it occurs during the daytime.¹⁵

Fatigue and weakness are common but nonspecific symptoms of heart failure. Severe heart failure may lead

to symptoms of anorexia and can be severe enough to cause cardiac cachexia, a syndrome characterized by end-stage heart failure and severe weight loss and malnutrition.¹⁶ Cachexia is a sign of very poor prognosis. The term cachexia is of Greek origin derived from the word *kakos* (bad) and *hexis* (condition).¹⁶ The exact mechanism of anorexia in cardiac cachexia is not known, but it may be related to a protein-losing enteropathy caused by intestinal edema.¹⁶ Anorexia could also be a side effect of medications used in the treatment of heart failure, such as digitalis.

HISTORY

The presence of comorbidities such as hypertension, hyperlipidemia, diabetes mellitus, and obstructive sleep apnea increase the likelihood of developing heart failure.^{8,9} Elements of the social history including smoking, heavy alcohol use, and illicit drug use are also important risk factors. A family history of coronary artery disease, dilated cardiomyopathy, and sudden cardiac death can provide important information regarding the etiology of heart failure. A detailed family history is also helpful in identifying whether there is a familial predisposition to atherosclerotic disease. Coronary artery disease is believed to be the underlying cause of heart failure in approximately two thirds of patients with heart failure.⁹

Heart failure is characterized by specific symptoms in the medical history. The history should focus on the following questions:

- When did the symptoms start?
- Are symptoms exertional or do they occur at rest?
- Are there associated symptoms such as chest pain?
- Is orthopnea or PND present?
- Is lower extremity swelling present?

The NYHA functional classification system has limitations depending on the questions used to ascertain functional capacity. Patients with heart failure are commonly asked to estimate the distance walked before the onset of dyspnea. Estimated walking distance is not as accurate as pointed questions.¹⁶ The following pointed questions are helpful in distinguishing between patients belonging to class II and class III heart failure.¹⁷

- Can you get dressed without feeling breathless?
- Do you feel breathless when showering?
- Can you walk up one flight of stairs without stopping?

A study by Mueller and colleagues presented odds ratios for the likelihood of heart failure in the following symptoms for acutely dyspneic patients in the acute

care setting: weight gain (3.6), lower extremity edema (2.8), and paroxysmal nocturnal dyspnea (2.4). In this study of 452 consecutive patients presenting with acute dyspnea to the emergency department, heart failure was the cause in 48% of cases.¹⁸

In patients presenting in the acute care setting with dyspnea, how useful are the medical history, physical examination, and rapid diagnostic tests in heart failure? A medical history and clinical examination alone in the evaluation of the dyspneic patient predicts the final diagnosis in 70% to 80% of cases.^{19,20} A recent study evaluating clinical examination revealed that 77% of patients with heart failure were diagnosed by history alone.²¹

PHYSICAL EXAMINATION

Mueller and colleagues also presented odds ratios for the likelihood of heart failure in the following clinical signs for the acutely dyspneic patients in the acute care setting: elevated jugular venous pressure (4.3), inspiratory crackles (3.1), lower extremity edema (2.8), hepatojugular reflux (2.7), and wheezing (0.4).¹⁸ The following sections discuss the evidence-based evaluation of each clinical sign in heart failure.

Pulse and Blood Pressure

Blood pressure changes occur with heart failure, with a diminished pulse pressure that reflects a reduction of stroke volume. A decrease in cardiac output should be suspected when the pulse pressure is low (eg, pulse pressure of ≤ 20 mm Hg in a blood pressure reading such as 90/70 mm Hg). Pulsus alternans, the beat to beat variation in the amplitude of the peripheral pulse, if present, is virtually pathognomonic of severe left ventricular failure. Pulsus alternans is attributed to an alternation in stroke volume with each cardiac cycle and is typically seen in patients with severe advanced heart failure.

Neck Exam

The jugular venous pulse is best observed in the right internal jugular vein with the patient's head turned away from the examiner. Jugular venous pressure reflects right ventricular filling pressure, and it remains the most reliable noninvasive means of estimating central venous pressure (CVP).²² In adults, normal CVP is typically in the range of 5 to 9 cm H₂O. In patients with left-sided heart failure, elevated left-sided pressures are transmitted through the pulmonary circulation and right ventricle to the jugular veins, resulting in elevated jugular venous pressure. As such, it is a poor prognostic indicator for patients with heart failure.²³

The abdominojugular (hepatojugular) reflux sign is useful in diagnosing right ventricular failure.²⁴ The hepatojugular reflux sign is elicited by applying slow, steady abdominal pressure to the middle of the abdomen for 15 seconds.²⁵ It is not necessary to apply pressure to the liver, as the name might imply. Midabdominal pressure may be preferred as pressure on the liver may cause pain. A positive result is defined by an increase in jugular venous pressure of more than 3 cm H₂O that is sustained for longer than 15 seconds. A positive abdominojugular reflux sign suggests reduced right ventricular compliance in that the right ventricle cannot accommodate an increase in venous return.²⁴ Other conditions such as constrictive pericarditis, right ventricular infarction, and restrictive cardiomyopathy can cause a positive sign. Left ventricular failure will cause a positive sign only when the right ventricular pressures are elevated.²⁴

Elevation in jugular venous pressure in patients with asymptomatic left ventricular dysfunction was also found to correlate positively with future risk of progression to heart failure.²⁶ In patients with heart failure, increased jugular venous pressure has been shown to be an adverse prognostic indicator, predicting rates of hospitalization and correlating with risk of death from heart failure.²³ More recent data show that jugular venous pressures can be estimated in the sitting position with comparable sensitivity and specificity.²⁷

Precordial Exam

The term apical impulse traditionally refers to the left ventricular impulse. It is normally located within the left midclavicular line in the fifth intercostal space. Inspection and palpation of the chest can reveal the presence of cardiac enlargement by displacement of the apical impulse past the midclavicular line. This sign is very sensitive but not specific for the diagnosis of heart failure.²⁸

An apical impulse of greater than 3 cm may be an accurate indicator of left ventricular enlargement with a sensitivity of 92% and a specificity of 91%.²⁸ A hypokinetic apical impulse, which is of low amplitude, occurs as a result of a reduction in left ventricular stroke volume. This pattern is usually a sign of severe left ventricular dysfunction. Palpation of the precordium in the setting of dilated cardiomyopathy reveals a displaced left ventricular impulse that is down and to the left of its normal position. The left ventricular impulse is also enlarged and sustained.

Cardiac Auscultation

A third heart sound (S₃) producing a galloped

rhythm can be heard in some patients with heart failure and has been shown to be associated with increased left ventricular end-diastolic pressures. Clinical studies have shown that the sign has a sensitivity of 30% to 50% and a specificity of 80% to 90%.^{29,30} The presence of a left ventricular S₃ has also been shown to correlate with risk of hospitalization and death from heart failure and progression to heart failure in asymptomatic individuals.^{23,26} Although the presence of an S₃ is a strong predictor of heart failure, with a specificity of 99%, it may be difficult to hear an S₃ in a noisy acute care setting.¹³

The S₃ gallop (also called a ventricular gallop) is frequently a sign of left ventricular failure. It is a low-pitched sound that is heard best at the apex with the bell of the stethoscope in early diastole. It is believed the S₃ occurs during deceleration of rapid ventricular filling. When the blood flow through the mitral valve decelerates in the left ventricle, it causes vibrations of the ventricular wall. These vibrations can be heard as an S₃.³¹ Although agreement between observers in the correct identification of an S₃ is very poor,³² it is increased among experienced clinicians.³³ In a prospective observational study of 100 patients undergoing left-sided heart catheterization, patients with an S₃ had significantly higher brain natriuretic peptide (BNP) levels and a lower left ventricular ejection fraction than those without a detectable S₃.³³

Lung Examination

Patients with heart failure can present with inspiratory crackles on pulmonary auscultation; these crackles are coarse and especially appreciated over the lung bases. The crackles typically occur late in inspiration. Dullness to percussion and diminished breath sounds over the lung bases may be caused by pleural effusions.

The term *cardiac asthma* is used to describe a patient with heart failure and associated wheezing.³³ This finding is more common in elderly patients, who have the highest prevalence of heart failure. Although wheezing is classically associated with asthma, asthma is a rare cause of new-onset wheezing in elderly patients. Cardiac asthma is often missed in elderly patients.³⁴ The classic explanation for wheezing in heart failure is that it is caused by pulmonary edema and accompanying bronchial wall edema.³⁵ However, it is now thought to be caused by bronchial hyperreactivity.³⁶ It can be difficult to differentiate between heart failure and chronic obstructive pulmonary disease in many cases because both can have features of wheezing, dyspnea, orthopnea, chest pain, and nocturnal symptoms.³⁷

Other Findings

Patients with advanced heart failure can present with bilateral, pitting edema of the lower extremities. Weight gain can also occur in patients with heart failure.¹⁷ Congestive tender hepatomegaly and ascites are manifestations of patients with advanced heart failure, and these can cause right upper quadrant abdominal pain and early satiety.

A combination of signs and symptoms can lead the clinician to the diagnosis of heart failure. According to the Framingham criteria, the diagnosis of heart failure requires the presence of 2 major criteria or 1 major criterion and 2 minor criteria, provided that the minor criteria are not attributed to another medical condition (Table 3).^{38,39}

LABORATORY EVALUATION

The advent of echocardiography revolutionized the diagnosis of heart failure with its high sensitivity and specificity and the ability to differentiate systolic from diastolic failure. Two-dimensional echocardiography with Doppler is performed to assess LVEF, left ventricular size, wall thickness, and valve function. It also has the ability to quantify the severity of heart failure and is used as a prognostic marker to guide therapy.

Used in conjunction with other clinical information, the BNP level in the acute care setting can improve the evaluation of patients with acute dyspnea. If heart failure is suspected, measurement of serum BNP may be helpful in excluding heart failure.¹³ BNP levels are higher in patients with heart failure than in patients with dyspnea from other causes.^{40,41} A BNP exceeding 100 pg/mL has a sensitivity of 90% for heart failure but is limited by a specificity of only 75%.⁴² An elevated BNP level is not specific to left heart failure as it can be increased in patients with chronic pulmonary diseases due to right ventricular strain.¹³

There is still considerable debate on whether serial measurements of BNP are justified for monitoring patients in the ambulatory care setting.⁴³ The utility of monitoring changes in BNP is uncertain because it is not clear what magnitude of change can be considered clinically significant.⁴⁴ Thus, additional study is needed to define the role of routine BNP measurements in the evaluation and management of chronic heart failure. In the acute care setting, BNP can be used to aid in the diagnosis of heart failure, whereas a repeat BNP performed at least 24 hours later has some value in prognosis and can alleviate the need for repeat echocardiography.⁴⁵ However, physicians should never place a higher value on BNP level than on clinical judgment. Higher BNP levels

Table 3. Framingham Criteria for Congestive Heart Failure

	Major	Minor
History		
Paroxysmal nocturnal dyspnea	X	
Orthopnea	X	
Dyspnea on exertion		X
Nocturnal cough		X
Weight loss in response to treatment	X	
Physical examination		
Neck vein distention	X	
Rales	X	
S ₃ gallop	X	
Hepatojugular reflux	X	
Hepatomegaly		X
Bilateral ankle edema		X
Tachycardia		X
Chest radiograph		
Cardiomegaly	X	
Pulmonary edema	X	
Pleural effusion		X
Pulmonary function testing		
Vital capacity decreased one third from maximum		X

Adapted from McKee PA, Castelli WP, McNamara PM, Kannel WB. The natural history of congestive heart failure: the Framingham study. *N Engl J Med* 1971;285:1442; and Mosterd A, Deckers JW, Hoes AW, et al. Classification of heart failure in population based research: an assessment of six heart failure scores. *Eur J Epidemiol* 1997;13:493.

on admission correlate strongly with in-hospital mortality rates with a near linear relationship.⁴⁶

Confounders in the measurement of BNP may play a minor role in its usefulness in evaluation. Obesity affects BNP levels by lowering circulating levels in patients with a higher body mass index; in patients with chronic kidney disease, BNP levels are likely to increase in correspondence with the severity of renal dysfunction. Either way, the BNP remains a valid marker in the evaluation of suspected heart failure in these patients.^{47,48}

EXAMINER RELIABILITY

Some of the limitations of clinical signs in patient evaluation include the interobserver variability (the difference in rate of detection of physical signs among different observers) and intraobserver variability (the difference in the rate of detection of a physical sign by the same observer on repeated observation at different times).^{32,33,49} However, these limitations also apply to diagnostic tools, such as echocardiography.

CONCLUSION

Heart failure is primarily a bedside diagnosis. It is a common progressive clinical syndrome resulting from multiple underlying causes. For the dyspneic patient in the acute care setting, a history and physical examination should be performed, and a chest radiograph and electrocardiogram should be obtained. A BNP level may also help to clarify the diagnosis. A comprehensive history combined with directed physical examination has enormous utility in the diagnosis, management, and prognosis of patients with heart failure. Medical education and residency training must better incorporate the use of clinical skills and evidence-based clinical tools in the evaluation, diagnosis, and management of heart failure.

HP

Corresponding author: Bernard M. Karnath, MD, 301 University Boulevard, Galveston, TX 77555; bm Karnath@utmb.edu.

REFERENCES

- Vukanovic-Criley JM, Criley S, Warde CM, et al. Competency in cardiac examination skills in medical students, trainees, physicians, and faculty: a multicenter study. *Arch Intern Med* 2006;166:610-6.
- Roger VL, Weston SA, Redfield MM, et al. Trends in heart failure incidence and survival in a community-based population. *JAMA* 2004;292:344-50.
- Senni M, Tribouilloy CM, Rodeheffer RJ. Congestive heart failure in the community. Trends in incidence and survival in a 10-year period. *Arch Intern Med* 1999;159:29-34.
- Ho KK, Anderson KM, Kannel WB, et al. Survival after the onset of congestive heart failure in Framingham Heart Study subjects. *Circulation* 1993;88:107-15.
- Levy D, Kenchaiah S, Larson MG, et al. Long-term trends in the incidence of and survival with heart failure. *N Engl J Med* 2002;347:1397-402.
- Criteria Committee of the New York Heart Association. Nomenclature and criteria for diagnosis of diseases of the heart and great vessels, 9th ed. Boston: Little, Brown & Co; 1994.
- Muntwyler J, Abetel G, Gruner C, Follath F. One-year mortality among unselected outpatients with heart failure. *Eur Heart J* 2002;23:1861-6.
- Hunt SA, Baker DW, Chin MH, et al; American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee to Revise the 1995 Guidelines for the Evaluation and Management of Heart Failure); International Society for Heart and Lung Transplantation; Heart Failure Society of America. ACC/AHA Guidelines for the Evaluation and Management of Chronic Heart Failure in the Adult: Executive Summary A Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee to Revise the 1995 Guidelines for the Evaluation and Management of Heart Failure): Developed in Collaboration With the International Society for Heart and Lung Transplantation; Endorsed by the Heart Failure Society of America. *Circulation* 2001;104:2996-3007.
- 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:e154-235.
- Manning HL, Schwartzstein RM. Pathophysiology of dyspnea. *N Engl J Med* 1995;333:1547-53.
- Michelson E, Hollrah S. Evaluation of the patient with shortness of breath: an evidence-based approach. *Emerg Med Clin North Am* 1999;17:221-37.
- Davie AP, Francis CM, Caruana L, et al. Assessing diagnosis in heart failure: which features are any use? *QJM* 1997;90:335-9.
- Wang CS, FitzGerald JM, Schulzer M, et al. Does this dyspneic patient in the emergency department have congestive heart failure? *JAMA* 2005;294:1944-56.
- Lorenzi-Filho G, Genta PR, Figueiredo AC, Inoue D. Cheyne-Stokes respiration in patients with congestive heart failure: causes and consequences. *Clinics* 2005;60:333-44.
- Andreas S, Hagenah G, Moller C, et al. Cheyne-Stokes respiration and prognosis in congestive heart failure. *Am J Cardiol* 1996;78:1260-4.
- Anker SD, Coats AJ. Cardiac cachexia. A syndrome with impaired survival and immune and neuroendocrine activation. *Chest* 1999;115:836-47.
- Raphael C, Brisco C, Davies J, et al. Limitations of the New York Heart Association functional classification system and self reported walking distances in chronic heart failure. *Heart* 2007;93:476-82.
- Mueller C, Frana B, Rodriguez D, et al. Emergency diagnosis of congestive heart failure: impact of signs and symptoms. *Can J Cardiol* 2005;21:921-4.
- Schmitt BP, Kushner MS, Weiner SL. The diagnostic usefulness of the history of the patient with dyspnea. *J Gen Intern Med* 1986;1:386-3. [Bob should this be 93?]
- Mulrow CD, Lucey CR, Farnett LE. Discriminating causes of dyspnea through clinical examination. *J Gen Intern Med* 1993;8:383-2. [Bob should this be 92?]
- Rovai D, Morales MA, Di Bella G, et al. Clinical diagnosis of left ventricular dilatation and dysfunction in the age of technology. *Eur J Heart Fail* 2007;9:723-9.
- Economides E, Stevenson LW. The jugular veins: knowing enough to look. *Am Heart J* 1998;136:6-9.
- Drazner MH, Rame JE, Stevenson LW, Dries DL. Prognostic importance of elevated jugular venous pressure and a third heart sound in patients with heart failure. *N Engl J Med* 2001;345:574-81.
- Wiese J. The abdominojugular reflux sign. *Am J Med* 2000;109:59-61.
- Ewy GA. The abdominojugular test: technique and hemodynamic correlates. *Ann Intern Med* 1988;109:456-60.
- Drazner MH, Rame JE, Dries DL. Third heart sound and elevated jugular venous pressure as markers of the subsequent development of heart failure in patients with asymptomatic left ventricular dysfunction. *Am J Med* 2003;114:431-7.
- Sinisalo J, Rapola J, Rossinen J, Kupari M. Simplifying the estimation of jugular venous pressure. *Am J Cardiol* 2007;100:1779-81.
- Eilen SD, Crawford MH, O'Rourke RA. Accuracy of precordial palpation for detecting increased left ventricular volume. *Ann Intern Med* 1983;99:628-30.
- Marcus GM, Gerber IL, McKeown BH, et al. Association between phonocardiographic third and fourth heart sounds and objective measures of left ventricular function. *JAMA* 2005;293:2238-44.
- Patel R, Bushnell DL, Sobotka PA. Implications of an audible third heart sound in evaluating cardiac function. *West J Med* 1993;158:606-9.
- Manson AL, Nudelman SP, Hagley MT, et al. Relationship of the third heart sound to transmitral flow velocity deceleration. *Circulation* 1995;92:388-94.
- Lok CE, Morgan CD, Ranganathan N. The accuracy and interobserver agreement in detecting the "gallop sounds" by cardiac auscultation. *Chest* 1998;114:1283-8.
- Marcus GM, Vessey J, Jordan MV, et al. Relationship between accurate auscultation of a clinically useful third heart sound and level of experience. *Arch Intern Med* 2006;166:617-22.
- Braman SS, Davis SM. Wheezing in the elderly. Asthma and other causes. *Clin Geriatr Med* 1986;2:269-83.
- Krieger BP. When wheezing may not mean asthma. Other common and uncommon causes to consider. *Postgrad Med* 2002;112:101-11.
- Cabanes LR, Weber SN, Matran R, et al. Bronchial hyperresponsiveness to methacholine in patients with impaired left ventricular function. *N Engl J Med* 1989;320:1317-22.
- Cheng TO. Shortness of breath: COPD or CHF? *Int J Cardiol* 2005;105:349-50.
- McKee PA, Castelli WP, McNamara PM, Kannel WB. The natural history of congestive heart failure: the Framingham study. *N Engl J Med* 1971;285:1441-6.
- Mosterd A, Deckers JW, Hoes AW, et al. Classification of heart failure in population based research: an assessment of six heart failure scores. *Eur J Epidemiol* 1997;13:491-502.
- Mueller C, Scholer A, Laule-Kilian K, et al. Use of B-type natriuretic peptide in the evaluation and management of acute dyspnea. *N Engl J Med* 2004;350:647-54.
- McCullough PA, Nowak RM, McCord J, et al. B-type natriuretic peptide and clinical judgment in emergency diagnosis of heart failure: analysis from Breathing Not Properly (BNP) Multinational Study. *Circulation* 2002;106:416-22.

42. Knudsen CW, Omland T, Clopton P, et al. Diagnostic value of B-type natriuretic peptide and chest radiographic findings in patients with acute dyspnea. *Am J Med* 2004;116:363-8.
43. Sanderson JE. BNP or echocardiography for monitoring heart failure? *Eur Heart J* 2004;25:1763-4.
44. Packer M. Should B-type natriuretic peptide be measured routinely to guide the diagnosis and management of chronic heart failure? *Circulation* 2003;108:2950-3.
45. Gackowski A, Isnard R, Golmard JL, et al. Comparison of echocardiography and plasma B-type natriuretic peptide for monitoring the response to treatment in acute heart failure. *Eur Heart J* 2004;25:1788-96.
46. Fonarow GC, Peacock WF, Phillips CO, et al. Admission B-type natriuretic peptide levels and in-hospital mortality in acute decompensated heart failure. *J Am Coll Cardiol* 2007;49:1943-50.
47. Horwich TB, Hamilton MA, Fonarow GC. B-type natriuretic peptide levels in obese patients with advanced heart failure. *J Am Coll Cardiol* 2006;47:85-90.
48. Mark PB, Petrie CJ, Jardine AG. Diagnostic, prognostic, and therapeutic implications of brain natriuretic peptide in dialysis and nondialysis-dependent chronic renal failure. *Semin Dial* 2007;20:40-9.
49. Ishmail AA, Wing S, Ferguson J, et al. Interobserver agreement by auscultation in the presence of a third heart sound in patients with congestive heart failure. *Chest* 1987;91:870-3.

Copyright 2009 by Turner White Communications Inc., Wayne, PA. All rights reserved.