Evaluation of suspected left ventricular systolic dysfunction

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KEY POINTS FOR CLINICIANS

- Heart failure is an increasingly common problem in primary care, with a mortality rate higher than that of most cancers.
- The absence of dyspnea on exertion or a normal electrocardiogram (ECG) result indicates that heart failure is unlikely; a gallop rhythm or laterally displaced apical rhythm is strong evidence in favor of heart failure.
- The history and physical examination and ECG alone are usually inadequate to confirm diagnosis of left ventricular systolic dysfunction, and echocardiography remains the gold standard to confirm the diagnosis.

Heart failure is increasing in incidence and prevalence; it currently affects 0.4% to 2% of the general population and 8% to 10% of the elderly.[1,2] In the United States, heart failure is the second most common cardiovascular reason for an outpatient visit in the ambulatory care setting and remains the most common cause for hospitalization among patients older than 65 years.[3] The total cost for heart failure management in 1999 was estimated to approach $56 billion.[4] Those suffering with this illness experience high levels of morbidity and mortality[5] that are reflected in the workloads of both primary and secondary care. Heart failure admission rates are rising, and the prognosis of heart failure has been compared with that of malignancy, with a 6-year mortality rate of 84% in men and 77% in women.[6,7]

A number of heart failure guidelines[8-14] provide direction regarding "best practice" with regard to diagnosis and management. These guidelines have all been produced by expert panels and base their evidence on systematic critical reviews of the literature, plus expert consensus opinion. The evidence underlying the development of these guidelines ranges from well-conducted randomized controlled trials to expert opinion. These guidelines all emphasize the ways in which approaches to the diagnosis and management of heart failure have altered substantially in recent years and are continuing to change rapidly. The need to detect heart failure at an early stage to slow the progression of left ventricular systolic dysfunction (LVSD) is now well accepted.[15]

The following provides an overview of the current recommended approaches to diagnosis, focusing specifically on LVSD, the most common type of heart failure and also the usual focus of most guidelines. Accurate diagnosis of LVSD is the single most important step in management.[16] An adequate diagnosis should establish the existence of heart failure, differentiate systolic from diastolic dysfunction, and identify the main underlying cause and any subsidiary diagnoses that may exacerbate heart failure. The etiology of heart failure and the presence of exacerbating factors or other diseases need to be carefully considered in all patients. Coronary artery disease remains the most common potentially reversible etiologic factor in heart failure.[8]

Using the history and physical examination

The major symptoms of heart failure are fatigue, exercise intolerance, exertional dyspnea, orthopnea, paroxysmal nocturnal dyspnea, and dependent edema. However, such symptoms are similar to those of many other diseases, particularly pulmonary diseases. For example, exertional dypsnea is a common symptom in heart failure but can be due to a wide range of other causes, such as chronic obstructive pulmonary disease, interstitial lung disease, asthma, respiratory infection, deconditioning, or obesity. Many patients with impaired left ventricular function may have no obvious symptoms.[17] This highlights the importance of exploring past medical and medication history as these contribute to the overall clinical assessment.

Physical findings that may support a diagnosis of heart failure include raised jugular venous pressure, peripheral edema not due to venous insufficiency, presence of a third heart sound, gallop rhythm, laterally displaced apical impulse, tachycardia, and

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TABLE 1

The use of clinical symptoms and signs to diagnose heart failure, by study

Sign or symptom	N	Setting*	Study quality (1a-5)†	Sensitivity (%)	Specificity (%)	LR+	LR-	PV+ (%)	PV- (%)
Previous myocardial infarction									
Davie, 1997[20]	259	R	2b	59	86	4.1	0.48	44	92
Morgan, 1999[28]	817	P	2b	39	91	4.3	0.67		
Dyspnea on exertion									
Davie, 1997[20]	259	R	2b	100	17	1.20	0.06	18	100
Morgan, 1999[28]	817	P	2b	15	97	5.4	0.88		
Orthopnea									
Davie, 1997[20]	259	R	2b	22	74	0.85	1.05	14	83
Paroxysmal nocturnal dyspnea									
Davie, 1997[20]	259	R	2b	39	80	1.95	0.76	27	87
History of peripheral edema									
Davie, 1997[20]	259	R	2b	49	47	0.92	1.09	15	83
Tachycardia									
Davie, 1997[20]	259	R	2b	22	92	2.75	0.85	33	86
Elevated JVP									
Davie, 1997[20]	259	R	2b	17	98	8.95	0.84	64	86
Morgan, 1999[28]	817	R	2b	11	97	3.6	0.92		
Gallop rhythm									
Davie, 1997[20]	259	P	2b	24	99	24.0	0.77	77	87
3rd heart sound									
Rihal, 1995[24]	554	H	2b	9	97	3.00	0.94	54	78
Laterally displaced apical impulse									
Davie, 1997[20]	259	R	2b	66	96	16.4	0.35	75	94
Pulmonary rales									
Davie, 1997[20]	259	R	2b	29	77	1.26	0.92	19	85
Morgan, 1999[28]	817	P	2b	44	82	2.4	0.68		
Peripheral edema on examination									
Davie, 1997[20]	259	R	2b	20	86	1.43	0.93	21	85
Morgan, 1999[28]	817	P	2b	18	91	2.0	0.90		

NOTE: Pretest probability = 50%.
*P denotes cross-sectional primary care population; R, primary care patients referred for suspected heart failure; H, hospitalized patients undergoing angiography.
†Level 1a is the most rigorous; level 5 is the least rigorous.
LR+ denotes positive likelihood ratio; LR-, negative likelihood ratio; PV+, positive predictive value; PV-, negative predictive value.

pulmonary rales that do not clear with coughing. Although clinical findings are particularly useful in acute severe heart failure at the time of hospitalization,[18] it is difficult to accurately diagnose mild heart failure in the community on the basis of clinical grounds alone.[2,19] The value of different symptoms and aspects of the medical history and use of medications in the evaluation of potential heart failure patients have been examined by researchers.[18, 20-23] Similarly, the utility of physical examination has also undergone investigation.[18,20,22-28] Table 1 summarizes the study findings with regard to clinical symptoms and signs.

Davie and colleagues[20] assessed the value of symptoms, past history, medications, and signs in the evaluation of patients who may have LVSD. No one clinical feature predicted LVSD, as assessed by echocardiography with sensitivity, specificity, and a high positive and negative predictive value. Absence of dyspnea on exertion essentially ruled out heart failure (negative likelihood ratio [LR-] = 0.06), while gallop rhythm (positive likelihood ratio [LR+] = 24.0), laterally displaced apical impulse (LR+ 16.4), and elevated jugular venous pulsation (LR+ = 8.9) are strong evidence in favor of the diagnosis. Furthermore, the combination of history of myocardial infarction and displaced apex on physical examination, although not particularly sensitive (39% sensitivity) was very specific (99% specificity) with high positive (89%) and negative (89%) predictive values. The authors also suggest that a breathless patient with a past history of myocardial infarction and a dis-

TABLE 2

Key investigations used for the diagnosis of left ventricular systolic dysfunction

Test	N	Setting*	Study quality†	Sensitivity (%)	Specificity (%)	LR+	LR-	PV+ (%)	PV- (%)
Electrocardiogram									
Davie, 1997[29]	534	R	1c	94	61	2.43	0.10	35	98
Lindsay, 2000[43]	416	R	1c	90	65	2.59	2.76	43	90
Mosterd, 1997[32]	1980	R	1c	54	79	2.55	0.58	7	98
Electrocardiogram (patient older than 70 years)									
Mosterd, 1997[32]	1980	R	1c	67	64	1.88	0.52	7	98
Talreja, 2000[30]	330	H	1c	65	98	38.2	0.36	98	64
Chest x-ray									
Badgett, 1996[34]	29 studies		2a	51	79	2.43	0.62	71	62
Rihal, 1995[24]	554	H	2b	20	89	1.82	0.90	34	79
Echocardiogram									
Erbel, 1984[35]	110	H	1c	80	100	80.0	0.20	100	85
N-terminal ANP > 4.4 ng/mL									
McClure, 1998[40]	134	M	2b	—	—	1.08	0.96	52	51
N-terminal pro-BNP > 275 fmol/mL									
Talwar, 1999[42]	249	R	2b	94	55	2.09	011	58	93
BNP > 75 pg/mL									
Maisel, 2001[37]	200	R	1c	86	98	43.0	0.14	98	89
Dao, 2001[44]	250	U	1b	98	92	12.2	0.02	92	98
BNP > 46 pg/mL									
McClure, 1998[40]	134	M	2b	—	—	2.25	0.83	69	55
BNP > 17.9 pg/mL									
McDonagh, 1998[41]	1653	P	2b	76	87	5.85	0.28	16	97

NOTE: Pretest probability = 50%.

*P denotes cross-sectional primary care population; R, primary care patients referred for suspected heart failure; H, hospitalized patients undergoing angiography; U, urgent care center; M, long-term myocardial infarction survivors recalled by their family physician.

† Level 1a is the most rigorous; level 5 is the least rigorous.

ANP, atrial natriuretic peptide; BNP, B-type natriuretic peptide; LR+, positive likelihood ratio; LR-, negative likelihood ratio; PV+, positive predictive value; PV-, negative predictive value.

placed apex beat on physical examination will almost certainly have heart failure and, if resources are limited, may not need echocardiography to confirm the diagnosis. However, less than 50% of breathless patients will have this combination, and the other half would therefore need echocardiography as the gold standard diagnostic tool for LVSD.

Morgan and coworkers[28] assessed the prevalence and clinical characteristics of LVSD among elderly patients (those aged 70 years to 84 years) in a primary care setting by echocardiographic assessment of ventricular function. They found that no single clinical symptom or sign was both sensitive and specific, and concluded that diagnosis should not be based on clinical history and examination alone. They found that a substantial number of elderly individuals had asymptomatic or misdiagnosed LVSD, and suggested this might be due to the extremely limited sensitivity and specificity of clinical history taking and examination. For example, only 11% of patients with LVSD had a raised jugular venous pressure, and bilateral ankle edema was common but nonspecific. Researchers have therefore concluded that although these clinical findings are useful in acute severe heart failure, they have only a small role in detecting LVSD in the community.[18]

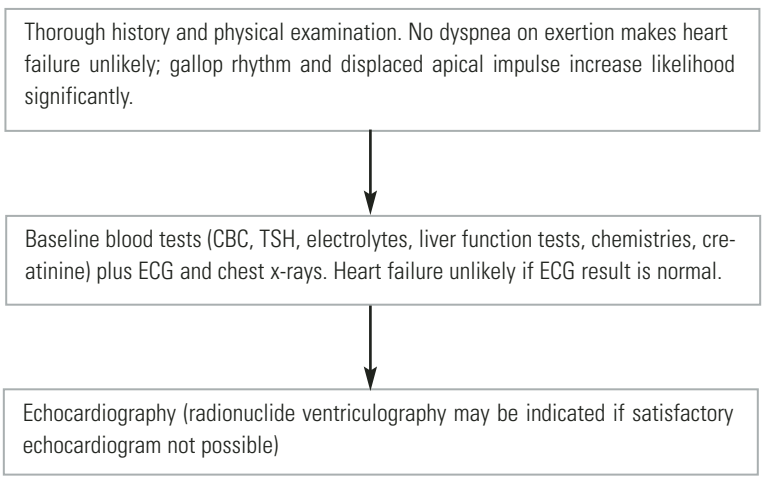
Laboratory and imaging evaluation

Although an important and valuable part of the evaluation, the history and physical examination alone are insufficient to confirm a diagnosis in most cases. Recommended initial tests for patients with signs or symptoms of heart failure include complete blood count (CBC), serum electrolytes, serum creatinine, serum albumin, liver function tests, urinalysis, electrocardiogram, and chest x-ray (Figure).

Blood tests. For those older than 65 years or with atrial fibrillation or evidence of thyroid disease, thyroid function tests should also be performed because heart failure due to thyrotoxicosis is frequently associated with rapid atrial fibrillation and hypothyroidism may also present as heart failure.[8,10]

FIGURE

Steps in the assessment of the patient with suspected heart failure



CBC denotes complete blood count; ECG, electrocardiogram; TSH, thyroid-stimulating hormone.

The other routine blood tests are important as a way to exclude alternative diagnoses; they also help with the search for predisposing or exacerbating causes of the heart failure. These baseline tests also help guide future therapeutic decision making. For example, electrolyte and renal function results are pertinent when initiating angiotensin-converting enzyme (ACE) inhibitors. Anemia can exacerbate pre-existing heart failure, and measurement of renal function is essential to distinguish fluid overload due to heart failure from renal failure. Liver enzymes may be affected by hepatic congestion. Urinalysis is valuable in the detection of underlying renal disease or diabetes.[8]

Electrocardiography. An electrocardiogram (ECG) is another recommended part of the evaluation of the suspected heart failure patient.[8-14] Considerable attention has been paid to examining the value of this test in the diagnosis of LVSD.[29-32] Davie and colleagues[29] assessed the value of the ECG in identifying patients with possible heart failure by examining referrals for echocardiography by primary care practitioners. A total of 534 patients were referred for echocardiography for possible heart failure, of whom 18% (n = 96) had LVSD. They showed that LVSD was extremely unlikely if the ECG result was normal, but that 1 in 3 patients with an abnormal result had significant LVSD. Thus, a normal ECG result virtually excludes chronic heart failure due to LVSD. However, the ECG is not a substitute for echocardiography, as an abnormal result does not accurately predict the presence of LVSD (Table 2).

Others have confirmed these findings.[30,32] Talreja and colleagues[30] found that of 330 consecutive inpatients referred for echocardiographic assessment of left ventricular function, 124 (41%) had LVSD. Only 2 of 124 patients with LVSD had a normal electrocardiogram result. When the ECG result is normal, the authors suggest that echocardiography is not needed. However, they concede that physicians are unlikely to adhere to this because many may not be as sophisticated in interpreting the ECG and may feel it important to get an accurate measure of ejection fraction. Guidelines published by the European Society of Cardiology[10] state that a normal ECG result in patients with suspected heart failure should lead us to doubt the accuracy of the diagnosis.

Chest x-ray. The chest x-ray is most valuable as a test to exclude pulmonary causes. However, the existing evidence suggests it is not a reliable way to exclude LVSD.[24,26,33,34] Table 2 provides information about the value of radiography in predicting LVSD. A systematic review of the literature concluded that redistribution and cardiomegaly were the best chest radiographic findings for diagnosing increased preload and reduced ejection fraction, respectively.[34] However, neither finding alone could adequately exclude or confirm LVSD. Studies published since that review have confirmed this finding.[33] Although part of the evaluation of the heart failure patient, radiography is only one part of the diagnostic process and cannot be used to provide definitive diagnostic information.

Echocardiography. The most important step in the evaluation of the heart failure patient is the assessment of left ventricular systolic function. Both echocardiography and radionuclide ventriculography have been advocated.[8-14] However, echocardiography is preferred as it is widely available, simple, noninvasive, safe, usually less expensive, and provides more information about valve function and left ventricular hypertrophy. Table 2 demonstrates the high sensitivity and specificity of echocardiography.[35] In view of this, it is recommended as a standard adjunct to the clinical diagnosis of patients with dys-

pnea on exertion and suspected heart failure. Between 8% and 18% of patients will have inadequate echocardiograms, in which case radionuclide ventriculography is advocated.[8]

Neurohormonal markers. In recent years there has been increasing interest in the potential role of neurohormonal markers, such as B-type natriuretic peptide (BNP), atrial natriuretic peptide (ANP), N-terminal pro-ANP (N-ANP), and N-terminal pro-BNP (N-BNP) as indices of LVSD.[36-42] Most of the data relating to these markers are relatively recent; therefore their use is not addressed in any detail in any of the aforementioned guidelines.

Some studies[41,42] suggest that BNP and N-BNP are useful for diagnosing LVSD even when the positive predictive values are low, because of their high negative predictive values. One of the most recent studies[44] examined the utility of BNP in an urgent care setting and suggested that BNP was an extremely reliable indicator of LVSD. In this population of patients with acute dyspnea where 39% had a final diagnosis of heart failure, 90% with a positive BNP had heart failure and 98% of those with a negative BNP did not. Although there appears to be a growing body of evidence supporting the role of these neurohormonal markers in the evaluation of the patient with LVSD, Table 2 illustrates that there have also been conflicting findings. This is partly because of differences in study design, study populations, cut-off points for ANP and BNP, and the definition of LVSD. Most studies agree that assessment of BNP, in particular, may be a cost-effective method for initial screening for LVSD, but should still be followed by an echocardiogram to confirm the diagnosis.

Management follows diagnosis. Making the correct diagnosis is the crucial first step in the management of chronic heart failure. Figure 1 summarizes the steps currently recommended for the evaluation of the patient with LVSD. A confirmation of a diagnosis of LVSD, however, is not the end of the story. Management will then need to include initiation of appropriate therapies and consideration of treatable and reversible etiologies, a subject to be addressed in the June 2002 issue of this journal.

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