PREFACE

Heart failure is a complex clinical syndrome manifested by dyspnea, fatigue, fluid retention, and decreased exercise tolerance. Heart failure may result from disorders of the pericardium, the myocardium, the endocardium, valvular structures, and the great vessels of the heart or from rhythm disturbances. Nearly 5 million Americans have heart failure today, an incidence approaching 10 per 1000 population after the age of 65 years. Heart failure is the reason for at least 20% of all hospital admissions in persons above age 65; hospitalizations for heart failure have increased by 159%. The prevalence of patients with heart failure has grown markedly as a result of the aging population and the number of patients who have survived heart attacks, heart valve surgery, and other cardiac procedures as a result of improvements in adjunctive medical therapies and surgical techniques. Thus, almost any practicing clinician will encounter a patient with the heart failure syndrome on a regular basis.

Heart Failure: A Clinician's Guide to Ambulatory Diagnosis and Treatment reviews all aspects of heart failure diagnosis and management, with a particular emphasis on office-based/ambulatory care. The volume discusses diagnostic and therapeutic options for clinicians in evaluating patients with dyspnea, fatigue, or edema. The recommendations contained herein are specific and directed at targeted symptoms. The many diagnostic-imaging modalities discussed focus on the practical utility of the tests. *Heart Failure: A Clinician's Guide to Ambulatory Diagnosis and Treatment* reviews the state-of-the-art pharmacologic, device, and surgical options for heart failure management, with care algorithms that are usually supervised by a nurse or nurse specialist.

Heart Failure: A Clinician's Guide to Ambulatory Diagnosis and Treatment is intended for generalists and internists, nurse practitioners, physician assistants, and general cardiologists who practice in the community setting. The epidemic of heart failure that faces our country necessitates a coordinated effort at prevention and optimal treatment of the disease. Unfortunately, all of the solutions to this enormous problem have yet to be elucidated, but additional efforts to educate and inform our busy clinicians will be important to keep up to date with the evolutions in heart failure care available today. We hope *Heart Failure: A Clinician's Guide to Ambulatory Diagnosis and Treatment* will serve as a platform for a systematic approach to the care of patients with heart failure for all clinicians.

We are very grateful for the excellent contributions from our authors, all of whom practice or trained at the University of Pennsylvania Health System. This work represents our approach to the plague of heart failure, a northeastern United States approach certainly, but a concerted and devoted one nevertheless.

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INTRODUCTION

Identification of the etiology of heart failure early in the course of the disease will allow the physician to institute pharmacologic therapies. These therapies may not only effectively ameliorate the symptoms of heart failure, but may also favorably alter the natural history of this disease process by addressing the neurohormonal influences that perpetuate injury in the failing heart. Furthermore, the identification of the pathophysiologic processes that have resulted in myocardial injury or dysfunction may allow the institution of therapies specifically directed at alleviating or reconciling those disorders. The identification of myocardial ischemia (e.g., contributing to myocardial hibernation) may direct the physician to provide therapies directed at improving myocar-

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dial blood flow and thereby improve cardiac function. Similarly, the identification of valvular lesions producing either pressure or volume overload may be reconciled by valve replacement or repair. A history of excessive alcohol consumption, a common cause of myocardial dysfunction, and subsequent avoidance of alcohol use, often results in improved cardiac function.

In addition to the identification of the pathophysiologic processes initiating myocardial injury, the exacerbation of heart failure in patients with known cardiac dysfunction can often be avoided or reconciled if the physician is astute in taking the history that identifies alterations in dietary sodium intake, or the concurrent use of drugs negatively impacting on fluid retention or cardiac function.

PRESENTING SYMPTOMS OF THE HEART FAILURE PATIENT

Frequently, patients present complaints of fatigue, effort-related dyspnea, orthopnea, paroxysmal nocturnal dyspnea, edema, and occasionally, palpitations or embolic events as a result of intramyocardial thrombi. These symptoms often trigger noninvasive cardiovascular testing, which effectively identifies the presence of both diastolic and systolic ventricular dysfunction.

However, on other occasions, myocardial dysfunction is identified incidentally during echocardiographic testing in patients with heart murmurs or nondescript symptoms. Once such dysfunction is found, the physician must seek to identify the etiology of myocardial injury.

WHEN TO SUSPECT ISCHEMIC HEART DISEASE IN THE PATIENT WITH HEART FAILURE

Ischemic heart disease is currently the most common etiology of congestive heart failure (CHF) in the United States. Patients often provide a history of prior myocardial infarction or classical angina pectoris. Noninvasive testing provides a tool to assess the severity of myocardial ischemia and quantitate to the extent of the myocardial scar. However, patients frequently do not report classical angina pectoris, but instead complain of dyspnea that represents their ischemic equivalent. The presence of cardiac risk factors contributing to the development of coronary disease, such as tobacco abuse, diabetes mellitus, dyslipidemia, hypertension, a family history of premature coronary disease, male gender, as well as increasing age, make it more likely that the patient's symptomatology may well be a result of coronary atherosclerosis. However, some patients are entirely asymptomatic despite the presence of substantial

coronary disease *(1)*. In particular, diabetics seem to be more likely to have "silent ischemia" and therefore may not complain of angina. Some of these patients may have sustained silent myocardial infarctions or have significant myocardial hibernation from severe ischemia in the absence of classical anginal symptomatology. The occurrence of silent or symptomatic myocardial infarction will often initiate or exacerbate heart failure. New Q waves on the electrocardiogram or exercise imaging studies will often confirm the diagnosis.

Valvular Heart Disease

Valvular heart disease may also produce heart failure or asymptomatic ventricular dysfunction. Aortic and mitral regurgitation may over time produce left-ventricular volume overload, resulting in systolic ventricular dysfunction. The presence of a heart murmur has often been recorded during prior examinations. Other patients with stenotic lesions of the aortic or mitral valves, or patients with congenital pulmonic stenosis, or tricuspid valvular disease, may also develop symptomatic heart failure. Echocardiography testing defines the magnitude of the valvular lesion, as well as its impact on ventricular function.

Occasionally, a patient with trivial pre-existent valvular disease may develop bacterial endocarditis that may produce an acute or subacute alteration in valvular incompetence and new volume overload, resulting in CHF *(2)*. A febrile illness after dental work, urologic manipulation, or skin- or soft-tissue infection may raise suspicion for the presence of either acute or subacute bacterial endocarditis. When the infection is subacute, clinical deterioration is of more gradual onset and is often associated with malaise, anorexia, weight loss, myalgias, and systemic emboli. Acute endocarditis, especially with *Staphylococcus aureus*, may present a septic syndrome with acute CHF. At times, the presenting complaint may result from an embolic event to the brain or extremities.

Congenital Heart Disease

Most patients with significant congenital heart disease have already undergone cardiologic evaluation. These lesions may produce CHF in adulthood. Occasionally, patients with patent ductus arteriosus or atrial septal defect may be undiagnosed until they develop heart failure later in life. Noninvasive imaging and cardiac catheterization will define these lesions.

Hypertension

Long-standing hypertension, especially when poorly controlled, not only contributes to the presence of coronary atherosclerosis, but also

may produce myocardial dysfunction as a result of long-standing pressure overload. A history of poorly controlled or severe hypertension is often obtained.

Diabetes Mellitus

Although most patients with diabetes mellitus will develop myocardial dysfunction from concomitant coronary disease, myocardial dysfunction from diabetes, particularly in the setting of hypertension, is well recognized *(3)*. This diagnosis is made by excluding the presence of coronary disease or other known etiologies of myocardial dysfunction in the diabetic, hypertensive patient.

Myocardial Toxins

Excessive alcohol intake is a frequent cause of myocardial dysfunction in our society *(4)*. An accurate history assessing the magnitude and duration of alcohol consumption must be obtained in any patient with heart failure. Patients will often underreport the amount of alcohol they consume. A more accurate history may be forthcoming from a spouse or other family member. Quantitation of alcohol intake is very relevant because abstinence from alcohol in these patients may produce substantial improvement in myocardial function. Concomitant, excessive alcohol use in the setting of other etiologies of ventricular dysfunction will potentiate the severity of heart failure. Furthermore, alcohol may trigger supraventricular and ventricular arrhythmias in these patients.

Cocaine abuse, a common problem in urban societies, is a common cause of myocardial injury, resulting in cardiomyopathy along with coronary vasospasm and acute myocardial infarction. A history regarding illicit drug use must be obtained in any patient with heart failure. In addition to the direct toxicity associated with the use of illicit drugs, these patients are often at risk for developing HIV, which may result in clinical AIDS and cardiomyopathy associated with severe AIDS involvement.

Chemotherapeutic agents used to treat neoplastic disease may likewise produce myocardial injury. The anthracycline derivatives are the most frequent agents associated with myocardial toxicity. Although oncologists attempt to limit the exposure of their patients to these agents in order to prevent cardiotoxicity, idiosyncratic injury occurs at times, particularly when associated with other myocardial disease or concomitant radiation therapy in which the heart is included in the radiation field *(5)*. Radiation itself may produce myocardial injury and initiate premature atherosclerotic and valvular disease.

Nutritional deficiencies that lead to severe anemia, such as beri beri or other chronic anemic states, may produce high-output CHF. Recon-

ciliation of the anemia and/or nutritional deficiency may reverse myocardial dysfunction.

TACHYCARDIA-INDUCED MYOCARDIAL DYSFUNCTION

It is also well recognized that chronic tachycardia, as a result of incessant tachyarrhythmias or poorly controlled atrial fibrillation, can produce reversible myocardial dysfunction *(6)*. It is now recognized that patients with atrial fibrillation often require the addition of β-blockers or calcium channel blockers to modulate their ventricular response. Treadmill testing or ambulatory electrocardiographic monitoring will often provide the physician with evidence of excessive heart rates. In these patients, the heart rate may exceed 110–140 beats per minute for much of the day, or if the heart rate increases to 120–140 beats per minute within the first 2 minutes of exercise, tachycardia-related cardiomyopathy should be considered.

Sleep Apnea

Some patients with right-sided CHF may suffer from obstructive sleep apnea. In these patients, chronic pulmonary hypertension and hypoxemia produce right-ventricular dysfunction along with systemic hypertension. Such patients are often obese and snore heavily, reporting daytime somnolence, early morning headache, or developing nocturnal tachyarrhythmias that bring them to the care of the cardiologist. Treatment for sleep apnea may ameliorate heart failure in these patients.

Other Causes of Ventricular Dysfunction

In some patients who develop myocardial dysfunction and CHF, no apparent etiology can be identified. Occasionally, patients may report a viral syndrome in the weeks to months before the onset of their symptomatology, suggesting a postviral myocardiopathy. Some patients may have a fulminant course of acute myocarditis resulting in severe and rapid clinical deterioration. Myocardial biopsy may be required to identify these individuals.

Some patients may suffer from congenital cardiomyopathy. There is often a family history of sudden cardiac death or early myocardial dysfunction. Hemochromatosis, hypertrophic cardiomyopathy, and the muscular dystrophies are heritable disorders that lead to significant cardiac involvement. Family histories of cardiac death or heart failure can often be obtained.

Unexplained heart failure in women postpartum was described in the 1930s and has become a well-recognized form of cardiomyopathy.

Peripartum cardiomyopathy should be suspected in patients who develop signs or symptoms of CHF in the third trimester of pregnancy or postpartum. The prognosis in these patients is excellent in comparison to other patients with cardiomyopathy, with one-third recovering to normal function and one-third stabilizing for prolonged periods of time *(7)*. Because the disease often recurs with subsequent pregnancies, the recognition of this syndrome is essential to prevent the patient from being exposed to additional myocardial injury.

CHRONIC HEART FAILURE ASSESSMENT

Fluid Retention: Why Is it Occurring?

In addition to identifying potential etiology of the myocardial dysfunction, the patient history will often assist in identifying factors contributing to a deterioration in the patient's clinical status.

The most frequent cause of fluid overload and a worsening of congestive symptoms is excessive dietary intake of salt. Detailed questioning should be undertaken in regard to a change in diet, such as eating meals outside of the patient's home or the consumption of salt-laden substances. Educated patients may augment their diuretic dosing to prevent such dietary alterations from producing decompensation.

Another common cause of clinical deterioration is the failure to comply with the prescribed medical regimen. The most common medications that are not taken as prescribed are the loop diuretics. Patients often complain that the brisk diuresis that accompanies the administration of these drugs fequently limits their ability to leave their homes and impacts their ability to attend social activities. They usually will not volunteer this information, but will admit to their omission of their prescribed doses with direct questioning. The physician will often find that compliance will improve when diuretics are prescribed at times when the patient has easy access to toilet facilities.

DRUGS IMPACTING ON HEART FAILURE

Many patients with CHF suffer from arthritic infirmities and may be prescribed nonsteroidal anti-inflammatory drugs by other physicians or may obtain them over the counter. These drugs may produce salt retention and blunt the effect of angiotensin-converting enzyme (ACE) inhibitors. The physician must inquire about the use of these drugs in patients with heart failure who decompensate and seek alternative drugs or augment their current regimens. In general, however, these drugs should be avoided in patients with severe heart failure.

Other pharmacologic agents used in the management of cardiovascular disorders may impact adversely on patients with heart failure. Drugs such as the nondihydropyridine calcium channel blockers (e.g., verapamil, diltiazem) used to treat angina, hypertension, or supraventricular dysrhythmias and certain antiarrhythmic drugs such as flecainide or disopyramide, may significantly impair left-ventricular fuction. β-blockers, although now indicated for the long-term treatment of heart failure patients, may produce transient deterioration in ventricular function and promote fluid retention. An occasional patient may decompensate when prescribed eyedrops for glaucoma that contain β-blockers because these may be systemically absorbed through the lacrimal ducts. Alternative therapy for glaucoma should be provided.

MYOCARDIAL ISCHEMIA

Myocardial ischemia or infarction may be superimposed on preexistent heart failure, which may cause acute or subacute decompensation. A history of accelerating angina or prolonged ischemic discomfort may be elicited in these patients, although some may not report classical ischemic symptomatology.

DYSRHYTHMIAS

Palpitations occur commonly in heart failure patients. Ventricular ectopy is nearly ubiquitous in patients with substantial left-ventricular dysfunction, but when extremely frequent or occurring in runs, may reduce cardiac output substantially.

The development of atrial fibrillation in patients with compensated heart failure may produce rapid deterioration as a consequence of the loss of atrial contribution to ventricular filling, as well as rapid heart rates that in and of themselves may cause further deterioration of ventricular function. In the patient with ischemic disease, atrial fibrillation with rapid rates may also produce significant ischemic dysfunction.

Bradyarrhythmias may also produce a reduction in cardiac output that may trigger decompensation. Symptoms of light headedness or worsening fatigue may be reported. Ambulatory echocardiographic monitoring is useful in defining these dysrhythmias.

OTHER FACTORS

Anemia is poorly tolerated by heart failure patients because it requires an increase in cardiac output and promotes fluid retention. Patients with severely impaired left-ventricular function often receive oral antico-

agulants that may promote bleeding. The physician must inquire as to the occurrence of melena or hematochezia.

A decline in renal function and progressive renal insufficiency may also result in fluid overload and diminished diuretic efficacy. Furthermore, the metabolism of drugs excreted via the kidneys will be altered potentially resulting in an accumulation of these drugs, such as β-blockers and digoxin. Accumulation of these drugs may produce bradycardia or heart block along with life-threatening digitalis toxicity.

Hypo- or hyperthyroidism may contribute to CHF decompensation. Amiodarone, a commonly prescribed antiarrhythmic drug in heart failure patients, may produce hyper- or hypothyroidism. Thyroid function studies should be followed closely in patients taking this drug.

Therefore, the history provides insights as to the etiology of the patient's heart failure syndrome, as well as identifying factors contributing to subsequent deterioration in function and quality of life. When possible, the identification of these factors and their reconciliation will substantially assist in the management of these patients.

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