Problems in Family Practice

Congestive Heart Failure

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Congestive heart failure in adults should be conceptualized and dealt with as a clinical syndrome. It is neither a diagnosis nor a disease as such. Patients with heart failure present with clusters of symptoms that define sets of systemic congestion, pulmonary congestion, and inadequate cardiac output. Some have potentially correctable anatomic or metabolic defects, others have myocardial failure, and some have both as underlying causes of the syndrome. A careful analysis of the symptom clusters may provide important clues to the underlying etiologic diagnosis, which should be established before initiating therapy for heart failure.

Recent therapeutic developments, including the widespread use of vasodilators and open heart surgery, have increased the need for a systematic approach to the recognition and analysis of the common condition known as congestive heart failure. This paper outlines such an approach, designed for application to adults, emphasizing the need to recognize specifically treatable causes.

Definition

No totally satisfactory definition of congestive heart failure exists. Exceptions can be found to most of the definitions used in standard texts. Take, for example, the one used in Hurst's *The Heart*: "that condition in which the heart is no longer able to pump an adequate supply of blood in relation to the venous return and in relation to the metabolic needs of the tissues of the body at that particular moment."[1] According to this definition a victim of ventricular fibrillation resulting from electrical shock would be suffering from heart failure. Braunwald et al[2] define heart failure as "the pathologic state in which an abnormality of myocardial function is responsible for the failure of the heart to pump blood at a rate commensurate with the requirements of the metabolizing tissues." This definition excludes the shock victim, but suffers from the implication that myocardial failure (in addition to overwhelming valvular insufficiency, for example) must always be present.

In this article, the terms *heart failure* and *congestive heart failure* will be used interchangeably and are defined as follows: the clinical syndrome that results from a protracted inability of the heart to pump blood at a rate commensurate with metabolic requirements at rest or during normal activity.

Diagnostic Considerations

Heart failure is not a disease, and it should not be considered a diagnosis, since establishing a diagnosis carries with it the implication that there is a specific treatment applicable to that diagnosis. Correct treatment of adults presenting with the syndrome depends more upon the specific etiologic diagnosis than on the mere presence or absence of the syndrome itself. Figure 1 characterizes the syndrome of heart failure as a Venn diagram[3] consisting of three overlapping sets of symptoms: those of systemic congestion, of pulmonary congestion, and of inadequate cardiac output. The specific manifestations of each set, listed in Table 1, are best thought of as clues to or

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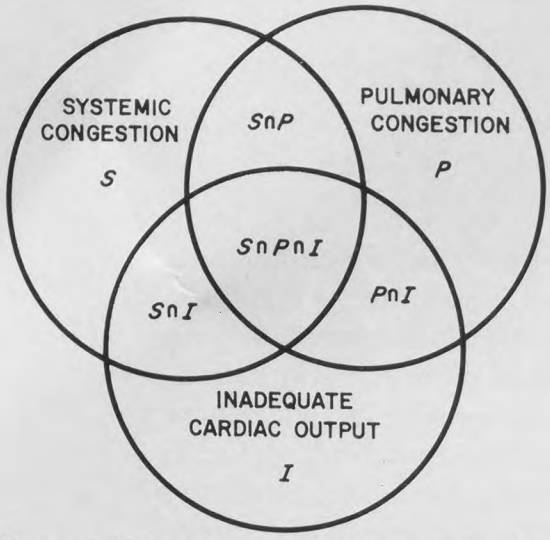

Figure 1. Venn diagram showing overlapping sets of systemic congestion, pulmonary congestion, and inadequate cardiac output. Individual subsets are indicated by Boolean notation

Table 1. Manifestations of Sets of Heart Failure

Systemic Congestion
Jugular venous engorgement
Hepatic congestion (enlargement, tenderness)
Pitting edema
Hepatojugular reflux
Pulmonary Congestion
Exertional dyspnea
Orthopnea
Cough
Paroxysmal nocturnal dyspnea
Rales
Inadequate Cardiac Output
Fatigue
Oliguria
Nocturia
Cutaneous vasoconstriction

"diagnostic tests" for the presence of the syndrome. Importantly, each clue or "test" has limited sensitivity and specificity; no single clue is pathognomonic. Clues to the presence of subtle heart failure, for example dyspnea and failure, are particularly nonspecific and commonly result from many other conditions.

Systemic congestion is commonly referred to as right ventricular or right heart failure, and pulmonary congestion as left ventricular or left heart failure. This terminology is counterproductive. Equating these symptom clusters with failure of one of the ventricles may lead to inaccurate diagnostic analysis of the cause or causes of the manifestations. For example, if the cluster of dyspnea, orthopnea, paroxysmal nocturnal dyspnea, and rales is considered evidence for failure of the left ventricle, mitral stenosis (which simply prevents normal left ventricular filling) may be considered only if the characteristic murmur is obvious. Similarly, believing that a right ventricle is failing in a patient who presents with manifestations of marked systemic congestion may result in the omission of constrictive pericarditis from the list of possible causes of this symptom cluster.

Physicians readily recognize the syndrome of heart failure when it presents as the overlap of all three sets ($S \cap P \cap I$, Figure 1). If most manifestations in Table 1 are present, the predictive accuracy, or likelihood that the condition is indeed present given the presence of the manifestations, is nearly perfect. Combinations of common noncardiac conditions, however, may result in the simultaneous presence of several manifestations from the three sets. This situation may closely mimic heart failure. For example, chronic lung disease plus venous insufficiency plus depression can cause dyspnea plus edema plus fatigue. Patients with conditions other than heart failure usually present with only a few of the individual manifestations, thus providing an important clue to the masquerade. If a patient presents with pulmonary congestion alone (particularly if paroxysmal nocturnal dyspnea is absent), the problem may not be heart failure at all, even though there is a history of an old myocardial infarction or an impressive heart murmur. Rales of chronic bronchitis are often indistinguishable from those of true pulmonary venous congestion, and chronic lung disease often masquerades as heart failure when there is other evidence of some cardiac abnormality.[4]

True heart failure, on the other hand, need not present with all of the individual manifestations either. Patients commonly present without obvious signs of systemic congestion or inadequate cardiac output, ie, subsets P, $P \cap I$, and $S \cap I$. Likewise, early in the course of an illness that will eventually induce the full-blown syndrome, patients may manifest only evidence of inadequate cardiac output; sufficient time has not yet passed for retention of salt and water to result in the con-

gestive manifestations of the other two sets. A typical example is the patient with a large acute myocardial infarction and profound depression of left ventricular performance who (unless hypotension or shock develops) shows little evidence of inadequate cardiac function clinically (ie, heart failure) for some time.

Cluster or subset analysis may also provide important clues to the specific disease process responsible for heart failure in a specific case. Symptoms within subsets S and $S \cap I$ provide particularly important clues to possible underlying etiologies. Absence of clear evidence for pulmonary congestion should suggest that the problem lies in the right heart (eg, tricuspid valve disease, the murmurs of which may not be evident) or in the pericardium (eg, constrictive pericarditis).

Causes of Heart Failure

Two other important sets overlap the heart failure symptom sets. Causes of the syndrome can be divided into two general classes: potentially correctable anatomic or metabolic defects, and myocardial failure, for which only palliative therapy can be offered. The symptomatic manifestations of these two general causes of heart failure are often clinically indistinguishable. Myocardial failure need not be present for the syndrome to exist; many patients have both correctable causes and an element of myocardial failure as well. Physicians who, upon recognizing the syndrome of congestive heart failure, initiate treatment (digitalis, diuretics, etc) without further analysis are behaving as though recognition of the syndrome were synonymous with diagnosing myocardial failure.

Treatment administered without careful analysis of specific causes in an individual patient often results in symptomatic improvement, even though specifically correctable processes such as anemia, mild hyperthyroidism, or a surgically curable lesion have not been recognized or appropriately attended to. Thus, inappropriate therapy often receives positive reinforcement. Although effective in many patients with heart failure, this reflex approach to treatment may cause serious problems. Digitalis, for example, is a leading cause of drug-induced problems in the elderly, in whom specifically correctable causes may be particularly occult.[5] Furthermore, this approach virtually guarantees that some unusual curable causes of congestive failure such as atrial septal defect (the murmur, and other physical manifestations that can be quite obscure) will be missed.

Table 2. Physiological Classification of Heart Failure

Pump Overload
Volume overload
Intrinsic
Valvular regurgitation*
Intracardiac shunt*
Ventricular aneurysm*
Extrinsic
Arteriovenous communication*
Metabolic high-output states*
Pressure overload
Intrinsic
Ventricular outflow obstruction*
Extrinsic
Systemic hypertension*
Pulmonary hypertension (primary, embolic,* asphyxial*)
Pump Insufficiency
Myocardial
Intrinsic
Myocardial infarction
Ischemic cardiomyopathy/fibrosis
Other cardiomyopathies
Extrinsic
Myocardial depression (eg, drugs,* alcohol*)
Extramyocardial
Dysrhythmias*
Inadequate ventricular filling
Pericardial constriction*/tamponade*
Atrioventricular valve stenosis*/atrial tumors*
Hypovolemic states*

*Often surgically or medically curable

An Approach to Specific Diagnosis

Once the syndrome of heart failure is recognized, a thoughtful history and physical examination and review of the chest roentgenogram and electrocardiogram will usually identify the specific cause or causes. Table 2 outlines a physiologic classification of the causes of heart failure. Note that the majority of categories of true heart failure are potentially surgically or medically "curable." More than one lesion may be present in a given patient, for example, valvular regurgitation plus myocardial insufficiency. In such cases, clinicians

should quantify the relative contribution of each lesion. Quantification can sometimes be done on clinical grounds, but most often requires referral for noninvasive or invasive studies.

The diagnosis of myocardial insufficiency should be based on positive criteria; it cannot safely be made simply by exclusion of other apparent causes. The ubiquitous arteriosclerotic heart disease (ischemic cardiomyopathy) is often assumed to be the cause of heart failure in individuals who are in the arteriosclerotic age group and who seem to present no other ready explanation for the presence of heart failure. When there are no positive criteria for ischemic heart disease, such as clear-cut angina pectoris or evidence of old or recent myocardial infarction, particular consideration should be given to certain notoriously occult conditions such as the restrictive cardiomyopathy associated with cardiac amyloidosis, alcoholic cardiomyopathy, silent mitral stenosis, atrial septal defect, and constrictive pericarditis. These last three are particularly important, since they may be amenable to operative correction. Even if evidence of an old myocardial infarction is present, the possibility of a surgically correctable problem must be considered. Left ventricular aneurysm is not always apparent on routine physical examination, but it should be carefully considered in any such patient who may be an operative candidate. Cor pulmonale, alone or in combination with other common causes of heart failure, such as hypertensive heart disease, is another important etiologic consideration that is easily overlooked in the older patient. This oversight can have potentially disastrous consequences if efforts are made to increase conventional digitalis and diuretic therapy for refractory dyspnea and edema without correcting the hypoxemia and hypercarbia responsible for the pulmonary vasoconstriction, which plays a major contributing role in the genesis of heart failure in such cases.

Table 3 lists causes of "pseudo heart failure." This re-emphasizes that all patients whose symptom clusters suggest the syndrome of congestive heart failure do not necessarily have heart disease. Recognition of pseudo heart failure may be particularly tricky in patients with other suggestive evidence of heart disease, such as the click or murmur of mitral valve prolapse or chest pain of noncardiac origin. Failure to notice the absence of objective signs of heart failure and lack of evidence of a sufficient degree of underlying heart disease to cause failure in such cases can be highly unfortunate if it causes or reinforces a cardiac neurosis.

Table 3. Causes of "Pseudo Heart Failure"

Combinations of Organic (Noncardiac) Mimics
(eg, chronic pulmonary disease plus venous insufficiency)
Psychogenic
Neurocirculatory asthenia
Cardiac neurosis
Combined Psychogenic Problems
(eg, anxiety [hyperventilation] plus depression [fatigue])

When the physician has completed analysis of the possible causes of heart failure in a specific case, he should either (1) have established with reasonable confidence, based on positive criteria, one or more specific causes, or (2) having failed to identify the cause, request either further laboratory data or consultation before proceeding with treatment.

Precipitating Factors

Having established the etiologic diagnosis, the physician's diagnostic job is only partly complete. He must now identify why the patient presents with this particular episode of congestive heart failure at this time. Table 4 lists possible precipitating factors. The presence or absence of most of these factors should be readily apparent from the history and physical examination. Progression of the underlying heart disease is a rather infrequent explanation for abrupt worsening of heart failure; evidence of such disease progression should be actively sought.[6] If none is found, and no other explanation seems apparent, particular consideration should be given to the following possibilities, the evidence for which may be particularly subtle: repeated pulmonary emboli that may not manifest with typical symptoms of pleuritic chest pain and hemoptysis, infective endocarditis (with or without worsening of the primary valvular lesion), rupture of a chorda tendineae, and silent myocardial infarction.

In the patient with chronic obstructive lung disease with or without previously recognized cor pulmonale, sudden worsening of congestive heart

Table 4. Precipitating Factors in Heart Failure

- *Increased Workload*
  - Physical, dietary, and emotional excess
    - Increased activity
    - Increased salt intake
    - Increased environmental temperature
    - Emotional stress
    - Increased obesity
  - Intercurrent conditions
    - Infection
    - Anemia
    - Hypertension
    - Thyrotoxicosis
    - Pregnancy
- *Decreased Cardiac Capacity*
  - Exogenous
    - Omitted digitalis
    - Acute myocardial depression (drugs such as disopyramide, quinidine, alcohol)
  - Endogenous
    - Dysrhythmias
- *Progression of Primary Lesion(s)*
- *New Primary Lesion*
  - Related to basic disease
    - Myocardial infarction (arteriosclerotic heart disease, hypertension)
    - Infective endocarditis with ruptured cusp (rheumatic heart disease, etc)
    - Ruptured chorda tendineae
    - Reactivation of rheumatic carditis
  - Unrelated to basic disease
    - Myocardial infarction (rheumatic heart disease, etc)
    - Pulmonary emboli

failure provides virtually conclusive evidence of recent deterioration of the underlying ventilatory problem.[7] This deterioration is almost always due to bronchial infection, although evidence of altered sputum, fever, new rales, and pulmonary infiltrate may be totally lacking. Failure to recognize and treat cryptic bronchitis often contributes to heart failure in such patients.[8] In contrast, appropriate improvement of alveolar ventilation, cautious administration of oxygen, and antibiotics are far more effective than the standard treatment of heart failure and much less hazardous.

Illustrative Cases

The following two cases illustrate the utility of these approaches.

Case 1

A 62-year-old man with no previous cardiac history presented with shortness of breath on exertion, paroxysmal nocturnal dyspnea, and orthopnea of two months' duration. There was no prior history suggesting heart disease. He had never experienced chest pain. He had been treated for the ten years previous for hypertension with fair control of his blood pressure.

On physical examination the man was generally healthy but appeared somewhat elderly. The blood pressure was 160/90 mmHg, the temperature 98°F, and the pulse 80 beats/min and regular. The neck veins were not distended, and there was no hepatojugular reflux. Bilateral basilar rales were heard in the chest. The cardiac examination revealed a normal precordium; the first and second heart sounds were normal, and there were no extra sounds or murmurs audible with the patient supine, squatting, sitting forward, and lying in the left lateral decubitus position. No peripheral edema, cyanosis, or clubbing of the extremities was evident. There was no hepatomegaly or ascites. The chest roentgenogram was interpreted as showing "increased density in the lower lung fields in an alveolar pattern and the heart is borderline with question [of] left ventricular enlargement. Both are consistent with congestive heart failure." The electrocardiogram suggested left atrial enlargement.

The initial assessment was congestive heart failure, etiology not established. Hypertensive heart disease was considered, but no clear evidence of myocardial insufficiency was present. Left atrial enlargement on the electrocardiogram, a rather common and nonspecific finding that could have been attributed to the history of hypertension, suggested the possibility of "silent" mitral stenosis, however, and an echocardiogram was ordered. The study revealed mitral stenosis and normal left ventricular size and function. Subsequent cardiac catheterization confirmed the findings of the echocardiogram, revealing significant mitral stenosis, normal coronary arteries, and normal left ventricular wall motion. When symptoms proved refractory to diuretic therapy, the patient was referred for cardiac surgery. Mitral commissurotomy relieved the stenosis, and the patient's symptoms were markedly improved.

This case illustrates that symptoms and signs of pulmonary congestion are not necessarily those of

myocardial failure. This patient's symptoms could have easily been ascribed to "left heart failure" caused by hypertension and coronary atherosclerosis. The auscultatory signs of mitral stenosis may not be audible, particularly in older patients with hyperinflated lungs, even when specific efforts are made to hear them, as in this case. Although diuretics reduced the congestive symptoms in this case, the resultant reduction in left atrial pressure further impaired cardiac output. Digitalis would have had little effect (until atrial fibrillation intervened), and aggressive treatment with afterload-reducing vasodilators could have caused serious problems with hypotension because of the mechanical obstruction to blood flow across the mitral valve that was in this case the principal problem. The correct treatment was surgical relief of that obstruction.

Case 2

A 39-year-old woman complained of dyspnea on exertion and ankle edema for six weeks. She noted mild abdominal swelling and fatigue, but reported no orthopnea, paroxysmal nocturnal dyspnea, or chest pain. The past history was unremarkable except for a brief hospitalization after an automobile accident 11 months earlier. She was observed for 24 hours and released after experiencing no apparent sequelae.

Physical examination revealed an ill-appearing middle-aged woman with distended neck veins but no dyspnea. The blood pressure was 120/82 mmHg, pulse 88 beats/min, and the temperature 99.2°F. The liver was enlarged and ascites and moderate peripheral edema were present. The lungs were clear to percussion and auscultation. Heart sounds were normal, and there were no extra sounds or murmurs. The chest roentgenogram was interpreted to be normal. The electrocardiogram revealed low QRS voltage and T wave flattening.

The initial assessment was that the patient had evidence of congestive heart failure with disproportionate systemic congestion, suggesting a differential diagnosis that included constrictive pericarditis. Repeat examination showed a pulsus paradoxus of 12 mmHg, but no pericardial knock could be heard. Further questioning revealed that the prior automobile accident had caused blunt trauma (steering wheel), but there had been no apparent chest wall or internal injury at the time.

An echocardiogram was thought to show two separate echos representing visceral and parietal pericardium and decreased posterior left ventricular wall motion. Cardiac catheterization revealed hemodynamic findings consistent with constrictive pericarditis, the patient was referred for pericardectomy, and her symptoms were markedly decreased postoperatively.

This case again illustrates the utility of viewing heart failure as a syndrome not necessarily synonymous with myocardial failure. Despite the disproportionately marked systemic congestion ("right heart failure"), the patient's associated dyspnea could have been interpreted as evidence of "left heart failure" and inappropriate drug therapy begun. This patient was not suffering from myocardial failure; rather, she had constrictive pericarditis probably resulting from trauma occurring months before the onset of heart failure symptoms.

Summary

Optimal diagnosis of a patient with heart failure results from a rigorous analysis of the symptom clusters that define systemic congestion, pulmonary congestion, and inadequate cardiac output. This method helps to identify potentially correctable anatomic and metabolic defects by requiring that myocardial failure be diagnosed by positive criteria rather than by exclusion. Meticulous use of this method of analysis will ensure prompt, accurate diagnosis of remediable causes of heart failure and appropriate therapy for the remainder.

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