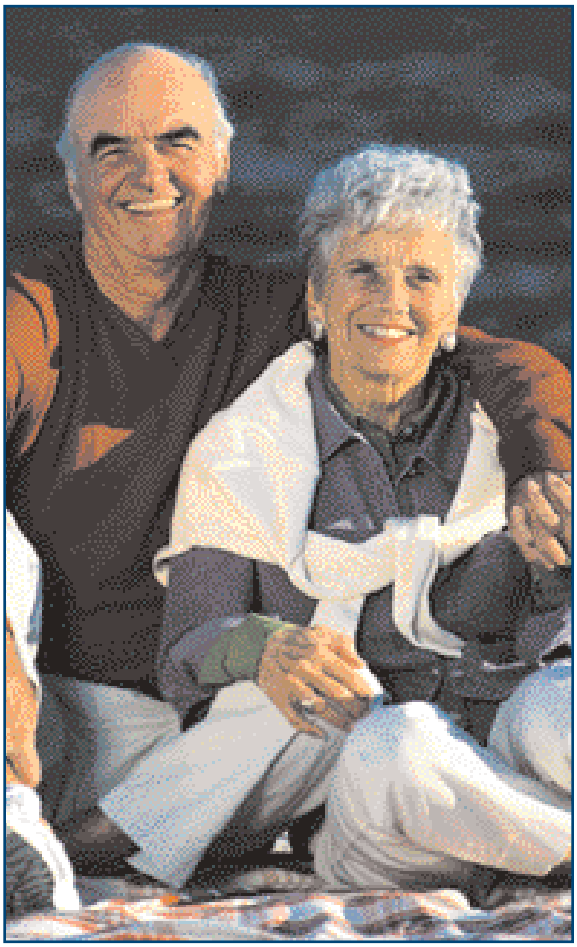
Heart Failure:

Getting to the Heart of the Problem



With a prevalence of 1% to 2% among the general population, heart failure has become a major problem, both in the clinical setting and in the public-health sector. Approximately 300,000 Canadians suffer from congestive heart failure, and the number is escalating.

By Anique Ducharme, MD, MSc, FRCP(C)

Introduction

The increase in cases of congestive heart failure (CHF) is primarily a result of the fact that we are treating acute infarction more aggressively, with means such as thrombolysis, emergency dilation and angiotensin-converting enzyme (ACE) inhibitors. Our aging population (and the fact that the prevalence of CHF increases with age) and under-use of drugs that have proven effective, such as beta-blockers and ACE inhibitors, also play a role.

Despite recent strides in our understanding of the physiopathology of CHF and the development of new drugs, the prognosis remains poor. The five-year mortality rate is 50%, which represents a life expectancy below that of victims of many forms of cancer. Nonetheless, through early diagnosis and effective treatment of CHF, we can help improve patients' quality of life and reduce the incidence of hospitalization and the mortality rate.

About the author...

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Table 1
Causes of LV Systolic and Diastolic Dysfunction

Causes of Systolic Dysfunction

- Atherosclerotic heart disease*
- Arterial hypertension*
- Valvulopathy*: aortic insufficiency, mitral insufficiency
- Idiopathic dilated cardiomyopathy
- Infiltrative* and electrolytic* disorders (Na, K, PO_4, Ca), nutritional deficiencies (protein, thiamin, selenium)
- Infectious or toxic agents (heroin, cocaine, alcohol,* adriamycine, cyclophosphamide, sulfonamides, glycol ethylene)
- Collagenosis
- Endocrine disorders (diabetes, hypo/hyperthyroid acromegaly, pheochromocytoma,* etc.)
- Systolic dysfunction induced by tachycardia*: incessant supraventricular tachycardia or atrial fibrillation with rapid ventricular response

Causes of Diastolic Dysfunction

- Atherosclerotic heart disease (ischemia)*
- Arterial hypertension*
- LV hypertrophy (hypertrophic cardiopathy or aortic stenosis)
- Normal aging
- Diabetes
- LV systolic dysfunction
- Constrictive/infiltrative pericarditis*

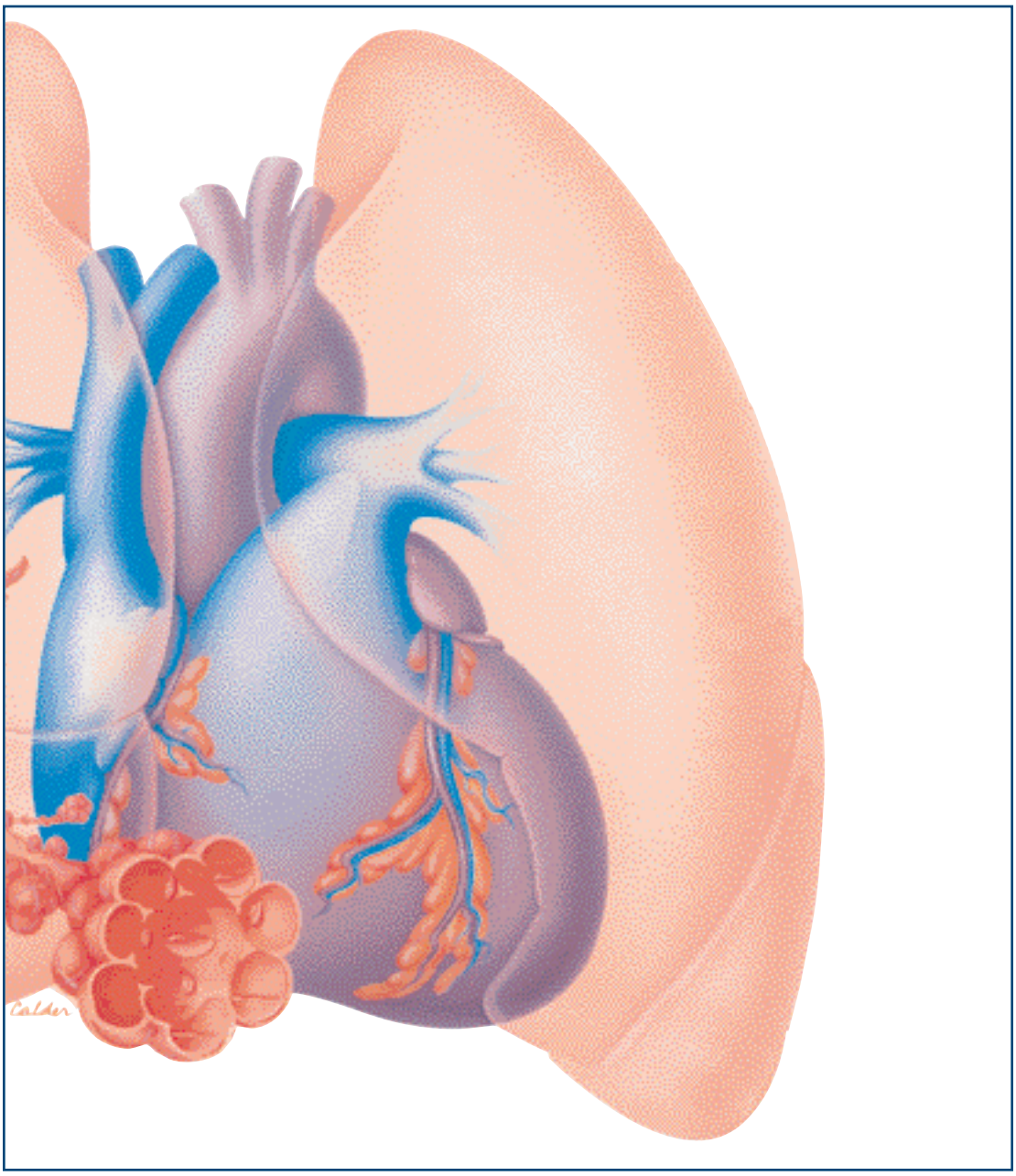
*Potentially reversible
Na = sodium; K = potassium; PO_4 = phosphorus; Ca = calcium

Defining heart failure

Heart failure is a clinical syndrome in which the heart is unable to pump sufficient blood to meet the metabolic needs of tissues, or can do so only with extreme pumping effort.

Systolic dysfunction is generally caused by problems in myocardial contractility. Coronary disease and arterial hypertension are at the root of 60% to 70% of CHF cases. In some cases, CHF is caused by disorders that make sudden demands on a normal heart, exceeding its capacity.[1]

In approximately one-third of patients suffering from CHF, "diastolic dysfunction" predominates and is characterized by pul-



monary or systemic venous congestion in the presence of normal or near-normal systolic function. "Diastolic dysfunction" results from problems of left ventricular (LV) filling—a consequence of impaired LV relaxation or a structural abnormality that causes a decrease in ventricular compliance. Myocardial ischemia, hypertrophy and fibrosis are generally the underlying pathological processes in cases of diastolic dysfunction. An increase in filling pressure is generally sufficient to maintain a normal ejection fraction and normal cardiac output. The ejection fraction and cardiac output may be compromised, however, in situations that require increased cardiac output

Table 2
Assessing Systolic Dysfunction

Recommended diagnostic tests

- Complete blood count and urinalysis
- Biochemistry profile: electrolytes, urea, creatinine, glucose, phosphorus, magnesium, calcium and albumin
- Thyroid-stimulating hormone (TSH) concentration (especially in cases of atrial fibrillation)
- Chest X-rays and ECG
- Echocardiogram
- Non-invasive assessment of ischemia (if angina and major risk factors are present)
- Coronary angiography (if angina or ischemia are present or if non-invasive assess ment reveals myocardial hibernation)
- Assessment of ischemia (all patients suffering from heart failure)

Potentially useful diagnostic tests

- Serum iron and ferritin
- Coronary angiography
- Biopsy
- TSH concentration (if sinus rhythm is present)

(*e.g.*, exercise). The most frequent causes of "diastolic" and systolic dysfunction are listed in Table 1. It should be noted that most cases of heart failure involve elements of both systolic and diastolic dysfunction.

The physiopathology of heart failure

CHF is associated with major cardiac and peripheral changes. The initial incidence of heart failure usually entails a loss of myocytes. An impairment of LV function and a drop in cardiac output become apparent, causing fatigue, along with an increase in filling pressure, which in turn causes congestion and dyspnea.

An increase in peripheral vascular resistance and LV wall stress occurs, resulting in decreased peripheral infusion and neurohormonal activation—factors that especially affect the sympathetic nervous system, angiotensin II, aldosterone and endothelin. These vasoconstricting hormones increase stimulation of renal sodium and water retention, and increase preload and afterload. At the same time, they have direct toxic effects on the myocardium, thereby provoking an additional decrease in cardiac function. This is how the vicious circle of CHF takes hold.

Assessment

All patients suspected of suffering from CHF must undergo an assessment (see Table 2), which should initially be limited to tests that enable the physician to do the following:

- Confirm the diagnosis;
- Determine the predominant type of ventricular dysfunction;
- Determine one or more underlying noncardiac disorders;
- Discover any reversible etiological factors;
- Determine the prognosis; and
- Determine the course of treatment.

Even if the signs and symptoms of CHF are, in and of themselves, inadequate for making a diagnosis, patients generally present with a set of signs and symptoms that, considered together, point to a diagnosis of CHF. It is important to diagnose CHF accurately and rule out other causes that may produce similar symptoms, such as obstructive lung disease or severe anemia.

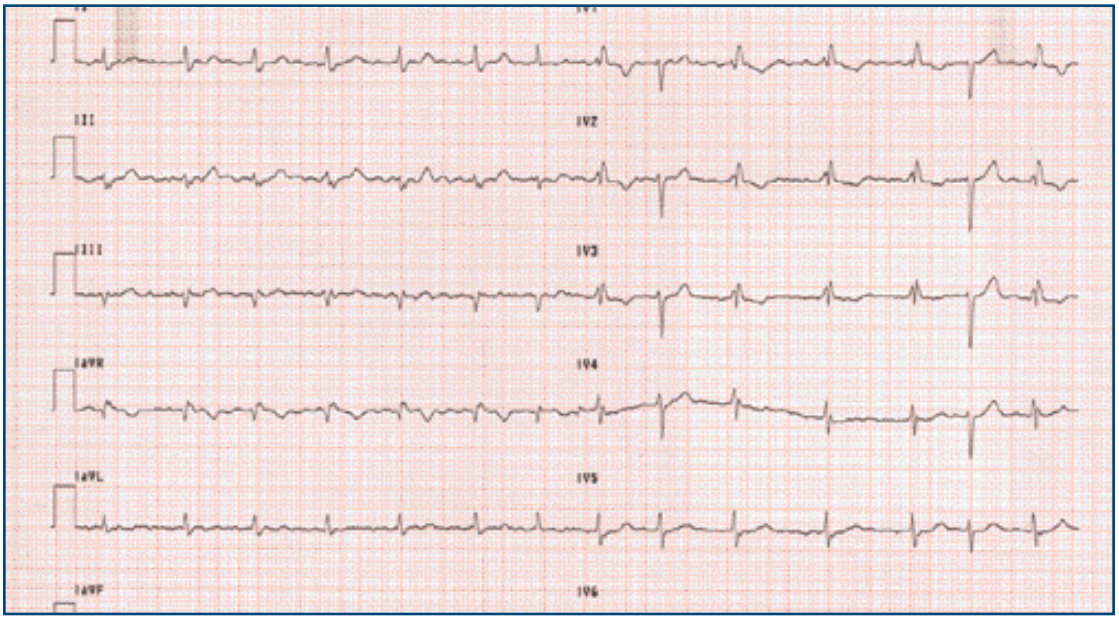
Anamnesis. A history of myocardial infarction, symptoms of orthopnea, paroxysmal nocturnal dyspnea, and progressive dyspnea on exertion are more likely linked to heart failure than symptoms such as edema, reduced exercise tolerance, and fatigue, especially when the latter occur in isolation. The patient's functional classification on the New York Heart Association (NYHA) scale should also be determined; this is important in establishing a prognosis and allows for objective longitudinal follow-up (see Table 3).

Physical examination. The sensitivity of a physical examination is limited in diagnosing CHF in patients who are obese or suffer from chronic lung disease. The discovery of high jugular vein pressure, a third heart sound, a wide apex beat, or laterally displaced apex beat is specific enough for a diagnosis of systolic heart failure. The detection of pulmonary rales and peripheral edema, however, is not specific enough for a diagnosis of CHF. Edema of the lower limbs is not likely caused by CHF if the jugular vein pressure is normal. Moreover, a history of hypertension, a predominant "A" wave in jugular pulse tracing and an audible fourth heart sound more likely suggest a diastolic component. Since both types of dysfunctions frequently co-exist in a patient, paraclinical examinations are required to establish an accurate diagnosis.

Blood work, biochemistry profile and thyroid function tests. A basic assessment should include a blood work-up and a biochemistry profile, as well as thyroid function tests.[2] These tests provide a means of finding non-cardiac explanations for the symptoms present (*e.g.*, hypoproteinemia, renal failure or anemia). These tests can also help the physician make therapeutic decisions if systolic dysfunction is subsequently confirmed.

Table 3
NYHA Functional Classification of Signs of Heart Disease

Class I
No symptoms
Class II
Symptoms present during ordinary physical activity
Class III
Symptoms present during less than ordinary physical activity
Class IV
Symptoms present at rest



An echocardiogram is especially useful in assessing patients suffering from CHF.

For instance, one might have to take into consideration the effects of uremia or hyperkalemia on the use of ACE inhibitors. If systolic dysfunction is confirmed, additional investigation may be required in order to find potentially reversible causes (Table 1). The choice of tests will vary from one patient to another, according to the information obtained from the anamnesis and clinical examination.

Chest X-rays. An increased cardiothoracic index revealed by a chest X-ray strongly suggests systolic dysfunction, especially when it is accompanied by pulmonary venous congestion. A normal chest X-ray does not necessarily rule out this diagnosis, however.

Electrocardiogram. Normal electrocardiogram (ECG) results are of little use diagnostically. The presence of LV hypertrophy, however, especially when associated with left atrial repolarization or dilation, may suggest diastolic dysfunction. The presence of an infarction (Q waves) points more to systolic or combined dysfunction.

Assessing left ventricular ejection fraction. The left ventricular ejection fraction (LVEF) should be assessed in all cases where a diagnosis of heart failure is suspected.

Echocardiogram. An echocardiogram is especially useful in assessing patients suffering from CHF, specifically for determining ventricular mass, the size of the cardiac chambers as well as systolic and diastolic functions. It is also useful for determining causes that require specific treatment, such as abnormalities in segmental contractility (which suggest an atherosclerotic cardiac disease), intracardiac tumors, or valvular or pericardial disorders. The possibility that the patient may be suffering from constrictive pericarditis—a condition that can be treated surgically—must be ruled out in patients whose diastolic dysfunction seems to be the result of restrictive physiology.

Radionuclide ventriculography is a means of accurately assessing the left and right ventricular ejection fractions. The results are less

Table 4
Precipitating Factors Potentially Leading to Hospitalization for Heart Failure

Inadequate reduction in treatment	The most frequent cause.
Arrhythmia	Supraventricular (supraventricular tachycardia or atrial fibrillation) or ventricular: can provoke decompensation in a previously stable patient.
Infection	Patients suffering from heart failure are particularly vulnerable to lung infections. Any infection, however, can precipitate deterioration.
Pulmonary embolism	Pulmonary embolism can cause elevated right ventricular pressure, as well as tachycardia and a secondary rise in temperature, which may not only cause dyspnea, but also decompensation.
Excessive physical exertion (extraordinary physical exercise), extreme environmental factor (temperature) or emotional factor (major stress)	Relatively frequent causes.
Development of a disorder unrelated to heart failure	Renal or hepatic disorder, transfusion, etc.
Administration of cardiomyodepressant drugs	Alcohol, beta-blockers, most antiarrhythmic drugs, first-generation calcium inhibitors, and antineoplastic drugs (especially doxorubicin and cyclophosphamide).
Administration of drugs that foster sodium retention	Estrogens, androgens, glucocorticoids, and nonsteroid anti-inflammatories (NSAIDs) can all cause sodium and water retention.

dependent on the operator than on the echocardiogram. The diagnostic tool must be selected according to the resources at hand and the degree of clinical uncertainty.

Non-invasive assessment of ischemia and myocardial viability. Patients with ischemia in association with systolic dysfunction generally have a poor prognosis and stand to benefit the most from revascularization.

Myocardial revascularization may even bring about an improvement in the clinical condition and LVEF of patients with severe LV dysfunction. A non-invasive assessment to detect ischemia and myocardial viability is, therefore, required in most cases—exercise or dipyridamole thallium, or stress-echocardiography (treadmill or pharmacological).

Stress test. Although a stress test is not required for diagnosis, it can help assess the patient's functional capacity or the effect of treatment on exercise tolerance. A six-minute walk test can be used as an alternative in severe cases. Holter monitoring is not systematically recommended in assessing arrhythmia.

Table 5
Contraindications for the Use of ACE Inhibitors

- History of intolerance or serious side effects, such as angioedema, pronounced renal failure, or absence of concomitant hypovolemia. Cough symptoms are also a contra-indication.
- Persistent hyperkalemia (potassium levels less than or equal to 5.5 mEq/L).
- Symptomatic hypotension. Asymptomatic patients with systolic blood pressure under 90 mmHg may take ACE inhibitors, but must be very closely monitored.
- Serum creatinine levels exceeding 265 mmol/L or a creatinine clearance under 30 mL/min are relative contra-indications.

The goals of treatment

There are three goals in treating CHF:

1. Enhance quality of life by increasing exercise tolerance and reducing the incidence of hospitalization and episodes of symptomatic heart failure.
2. Alter the natural evolution of the disease by attempting to reduce the LV mass and limit dilation of the cardiac chambers.
3. Improve chances of survival.

It should be noted that pharmacotherapy will not necessarily act on all three levels. Certain drugs relieve symptoms without increasing the chances of survival; they may, in fact, even decrease them.[1]

Treating systolic dysfunction

Education and exercise. Patients and their families must understand the illness and its treatment. It is also important for them to know the signs of deterioration in the patient's clinical condition in order to take appropriate action if the condition should worsen. The importance of compliance with pharmacotherapy and diet must be emphasized. Patients should weigh themselves daily and see their physicians immediately if they gain more than 1.5 kg to 2.5 kg in two or three days. Moreover, they should be encouraged to participate in a physical activity program, as this approach has proven effective in increasing exercise tolerance and, therefore, the quality of life in patients who fall into classes II and III of the NYHA classification (see Table 3).

Determining precipitating factors. Precipitating factors can be identified in more than 90% of patients admitted to a general hospital for CHF (see Table 4). Since they can result in a refractory condition if they remain unidentified, such factors must be sought systematically.

Diet and limited water, sodium and alcohol consumption. Diet is important, and restricted sodium intake (2 g to 4 g per day) is indispensable. An energy supplement may be required for patients suffering from cardiac cachexia. It is essential that patients with severe congestive symptoms (class III or IV on the NYHA scale), and especially those with a tendency towards hyponatremia, watch their fluid intake. All liquids ingested (including soups, Jello-O®–type gelatins, etc.) must be counted and limited to 1 to 1.5 L per day. Lastly, since alcohol is a myocardial depressant, it should be avoided.

Table 6
Effective Vasodilator Dosages in the Treatment of Heart Failure

Vasodilator	Initial Dose	Objective
Captopril*	6.25 mg tid	50 mg tid
Enalapril*	2.5 mg bid	10 mg bid (20 mg bid if Class IV in the NYHA classification)
Fosinopril	5 mg die	20 mg die
Lisinopril*	2.5 mg die	10 mg die
Perindorpril	2 mg die	4 mg die
Quinapril	10 mg die	40 mg die
Ramipril*	1.25 mg bid	5 mg bid
Hydralazine/isosorbide dinatrate*	10 mg qid/5 mg tid	75 mg qid/40 mg tid
Carvedilol*	3.125 mg bid	25 mg bid
Metoprolol*	12.5 mg bid	50 mg bid (or SR 100 mg die)
Losartan	25 mg die	50 mg die
Amlodipine	5 mg die	10 mg die

* Proven effective in mortality studies.
tid = 3 times per day; bid = twice per day; NYHA = New York Heart Association; die = daily; qid = 4 times per day; SR = slow-release.

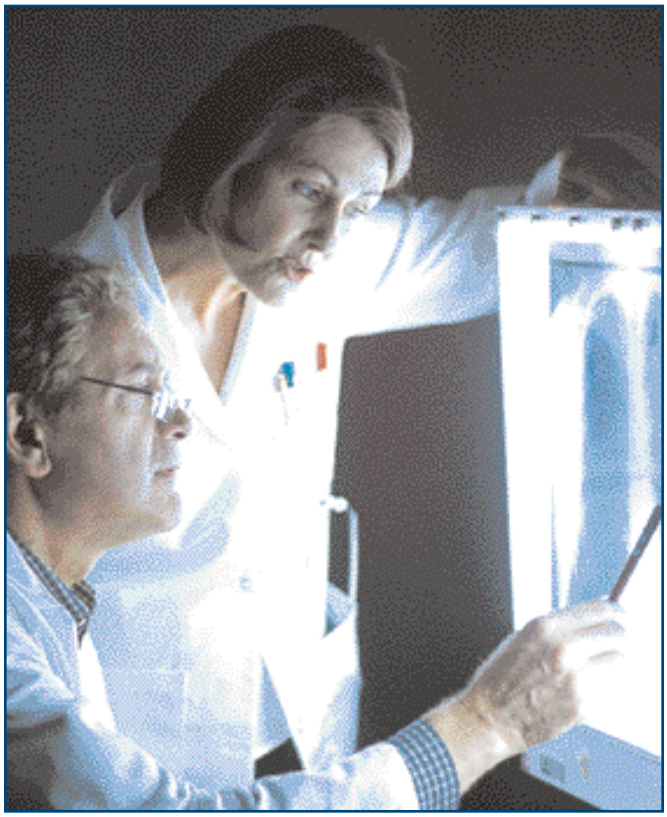
Pharmacotherapy. Loop diuretics are essential if generalized or lung congestion is present. Chronic use of such medications, however, has no positive effect on survival. Minimum dosages should be used whenever possible in order to maintain a stable weight. The use of excessive doses of diuretics may, in fact, activate neurohormonal mechanisms that can contribute to a progressive deterioration of the condition. The concomitant use of a thiazidic diuretic (2.5 mg to 5 mg metolazone 30 to 60 minutes before furosemide) may be necessary in advanced and refractory cases, as the combination may substantially increase diuresis. Nevertheless, there is a potential danger of excessive diuresis and hypotension, and patients taking a combination of diuretics must be monitored closely.

The use of diuretics can result in electrolyte abnormalities (primarily hypokalemia and hypomagnesemia), thereby causing cardiac arrhythmia. Serum potassium levels should be evaluated every three to five days during initial administration, titration or modification of a diuretic treatment. It has recently been demonstrated that the combination of low doses of spironolactone with ACE inhibitors is risk-free and can reduce the mortality rate of patients suffering from severe CHF (class III or IV on the NYHA scale) by 27%.[3]

ACE inhibitors. Unless there are specific contraindications, patients with systolic dysfunction, whether symptomatic or not, should be prescribed an ACE inhibitor (see Table 5). ACE inhibitors have been shown to be effec-

tive in patients with various degrees of CHF (classes II to IV on the NYHA scale). They reduce mortality, enhance the patient's functional category and reduce the incidence of hospitalization associated with CHF. Moreover, they slow down the progression of ventricular dilation. Asymptomatic patients (classes I to IV on the NYHA scale) with a diminished LVEF (i.e., under 30%), or in the post-infarction phase, (LVEF under 35%) can also benefit from ACE inhibitors.

ACE inhibitors are beneficial as they allow the neurohormonal mechanisms involved in the physiopathology of heart failure to operate more efficiently, fostering the role of vasodilatory kinins. The primary side-effects of ACE inhibitors include hypotension, renal failure and hyperkalemia, though the incidence of such complications is low.



It is important to take a chest x-ray beforehand to ensure that uncontrolled heart failure is not involved.

As soon as a diagnosis of systolic heart failure is confirmed in an euvolemic patient, a low-dose ACE inhibitor should be prescribed. Since ACE inhibitors provoke potassium retention, potassium supplements should be reduced or discontinued, and electrolytes and renal function should be closely monitored. Patients suffering from heart failure often have low blood pressure, usually asymptomatic. If there are no symptoms of hypotension, treatment with ACE inhibitors should be pursued, regardless of the pre-established blood pressure limits. In the case of symptomatic hypotension, it is preferable to reduce or discontinue the use of diuretics, rather than modify ACE-inhibitor use.

There is no clinically significant difference between the various ACE inhibitors, although they have not all been evaluated in the context of mortality studies. The choice of ACE inhibitor should be based on considerations that foster compliance, such as cost and single-dose treatments. It is, nevertheless, important to prescribe these drugs at dosages that have proven effective, as it has been demonstrated that low doses are less effective than high ones (see Table 6). Patients must be reassessed weekly whenever the dosage is increased, and blood pressure must be taken in both prostrate and standing positions in order to detect orthostatic hypotension. An increase in creatinine may be tolerated as

Patients should be encouraged to participate in a physical activity program, as this approach has proven effective in increasing exercise tolerance and, therefore, quality of life.

long as it is closely monitored and levels off. If a patient cannot tolerate an ACE inhibitor because of a deterioration in renal function or development of a cough (it is important to take a chest X-ray beforehand to ensure that uncontrolled heart failure is not involved), the only viable substitute—though less effective—is hydralazine in association with a nitrate derivative. The use of angiotensin II receptor antagonists (ARAs), however, is emerging.

While ACE inhibitors are not necessarily first-line agents in the treatment of purely diastolic heart failure, they can be used to control hypertension, thereby promoting the regression of LV hypertrophy.

Digoxin is recommended in the treatment of patients who present with systolic heart failure and atrial fibrillation.[4] Its efficacy in controlling the working heart rate, however, is limited. Its mechanism involves the vagal system, which is relatively inactive in such instances. Beta-blockers should also be administered in small doses and/or an attempt should be made to restore sinus rhythm.

If sinus rhythm is present, treatment with digoxin should be initiated or continued in patients who present with systolic dysfunction and remain symptomatic, despite treatment with an ACE inhibitor and diuretics. This treatment is intended to relieve symptoms, increase exercise tolerance and reduce

the incidence of hospitalization. The dosage of digoxin should be adjusted according to the patient's weight and renal function, and the serum level should be measured (at least eight hours after dose is administered) only if toxicity is suspected.

Beta-blockers can have a beneficial effect on patients suffering from heart failure (classes II to IV on the NYHA scale). Their routine use is, therefore, recommended for all patients. Beta-blockers can, nevertheless, cause dizziness, hypotension and a temporary deterioration of the patient's cardiac condition. It is important to try to bring the temporary deterioration under control by increasing the dosage of diuretics, rather than reducing beta-blockers. Treatment with beta-blockers should begin with very low doses and should be titrated carefully. In certain cases, the administration of beta-blockers can be left to specialists who are used to administering these drugs to a vulnerable category of patients (classes III and IV on the NYHA scale). Contrary to ACE inhibitors, beta-blockers do not all seem to be interchangeable, and only metoprolol, bisoprolol (now available in Canada) and carvedilol have proven effective.[5,6] Lastly, drugs with intrinsic sympathomimetic activity, such as bucindolol, do not seem to be effective in the treatment of CHF.

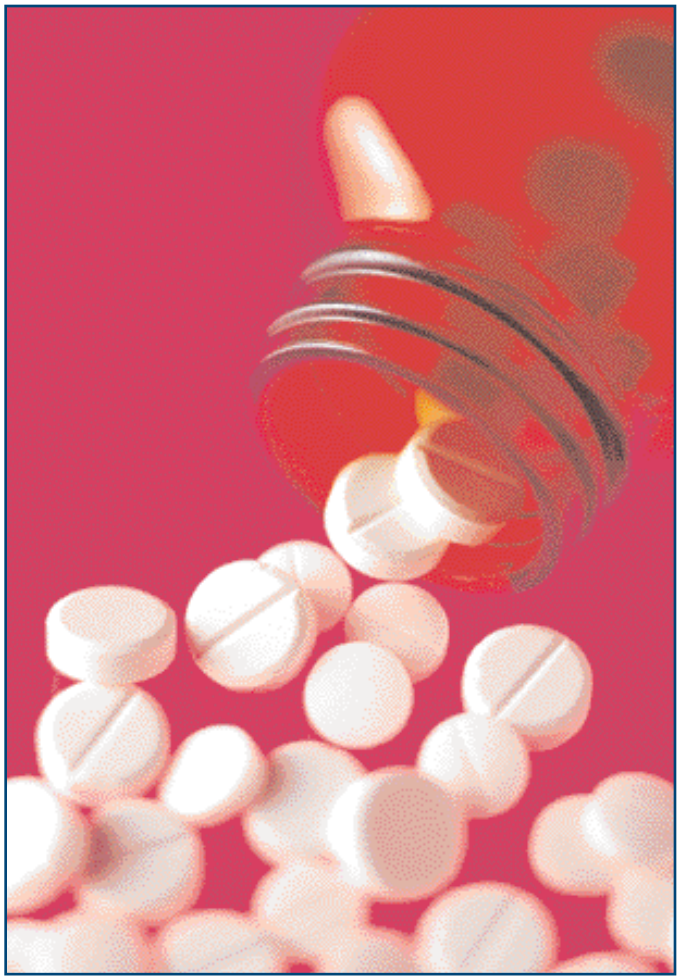
First-generation calcium inhibitors (verapamil, diltiazem and nifedipine) are negative inotropic agents. They cause a rise in mortality and in the incidence of heart failure, resulting in hospitalization. They must be avoided if LV systolic dysfunction is present.

Second-generation calcium inhibitors involve fewer risks, but according to the findings of the Prospective Randomized Amlodipine Survival Evaluation II (PRAISE II) study,[7] they provide no benefits with respect to the mortality rate, incidence of hospitalization, etc. They may be used to treat angina (in patients already receiving optimal doses of beta-blockers), persistent hypertension (despite maximum doses of ACE inhibitors), and as a first-line treatment for pure diastolic dysfunction.

Prolonged-action nitrate derivatives are useful when taken before bed in the form of a skin patch, in order to reduce symptoms of orthopnea and paroxysmal nocturnal dyspnea, as well as ischemic coronary symptoms.

Hydralazine in association with isosorbide dinatrate. Since the introduction of ACE inhibitors and ARAs, an effective drug combination seems to have been overlooked: hydralazine/isosorbide dinatrate, the benefits of which have been demonstrated in blind trials with respect to survival and exercise tolerance. This combination is nevertheless inferior to ACE inhibitors, such as enalapril, where reduction of mortality rate is concerned.

ARAs. The ELITE II study[8] demonstrated losartan is less effective than captopril in reducing mortality. Losartan should only be used as an alternative to hydralazine/ isosorbide dinatrate combinations with patients who are intolerant to ACE inhibitors. Nevertheless, Losartan provokes less coughing than ACE inhibitors. With respect to deterioration in renal function, ARAs have no advantages over ACE inhibitors.



Treatment of CHF resulting from diastolic dysfunction requires treatment of the underlying cardiovascular disorder and the precipitating factors.

Non-digitalic positive inotropic agents. Patients who present with excessive refractory volume (despite restricted sodium intake and the use of high doses of diuretics) may require an intravenous diuretic or an inotropic adjuvant. Only one major study has assessed the effects of intermittent intravenous administration of a positive inotropic agent (milrinone). The results were neutral, showing neither benefits nor harmful effects. The data suggest this practice could also have an adverse effect on mortality. Nevertheless, infusion of positive inotropic agents is commonly used in cases of severe CHF and is valid in refractory cases in which symptoms are limited.

Other therapeutic approaches. Ventricular arrhythmia and sudden death occur more commonly in patients suffering from CHF. Currently, amiodarone is the only risk-free treatment for heart failure. It is a drug of choice for patients who present with symptomatic arrhythmia. In patients with LV dysfunction and non-sustained ventricular tachycardia, the use of an implantable defibrillator would seem preferable to standard treatments for arrhythmia.

Antiplatelet agents and anticoagulants. Antiplatelet agents are recommended in the treatment of coronary disease, as they reduce post-infarction mortality and the risk of a subsequent infarction. Oral anticoagulants are recommended only after a previous infarction extending to the apex (for three months) or in the presence of a LV thrombus or atrial fibrillation (paroxysmal or chronic), in order to reduce the risk of an embolism from a cardiac source. Patients suffering from heart failure are, nevertheless, at greater risk of thromboembolisms (dilation and stasis of the cardiac chambers, endothelial dysfunction etc).[9] No other definitive clinical studies have been conducted that can help guide physicians in such cases.

Pharmacotherapy for isolated diastolic dysfunction

Treatment of CHF, resulting from diastolic dysfunction, requires treatment of the underlying cardiovascular disorder (arterial hypertension, valvulopathy or coronary disease) and the related precipitating factors.[10] Most patients require diuretics in order to suppress conges-

tive symptoms. Treatment must be closely monitored, however, as a drop in filling pressure (to a level that is inadequate for LV filling) could result in hypotension.

Reduction of ischemia by means of beta-blockers, nitrate derivatives or calcium inhibitors, in conjunction with revascularization, is appropriate for patients whose diastolic dysfunction results from coronary disease. Calcium inhibitors and beta-blockers lessen diastolic dysfunction because of their effects on hypertension, LV hypertrophy and coronary disease, *not* because of an intrinsic effect on the diastolic dysfunction itself.

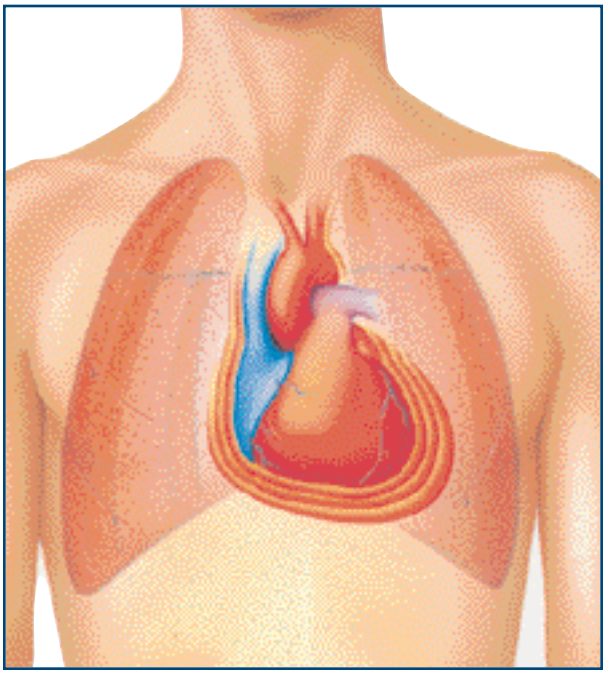
Patient follow-up

The patient's medical history and physical examination will help determine the frequency of follow-up visits. Facts gleaned from the clinical examination may prove more useful in patient follow-up than in the initial diagnosis.

It is generally unnecessary to assess the patient's ejection fraction or exercise tolerance more than once. However, if significant clinical changes are suspected or major changes in the treatment strategy envisaged, these factors should be reassessed.

How should refractory cases be handled?

In cases of refractory heart failure, it is important to review basic assumptions and ensure the diagnosis is correct. Ensure also the patient is taking his/her medication and is limiting sodium and water intake. Try to determine, once again, whether there are any precipitating or aggravating factors. The patient and his/her family may require additional information on diet, strict compliance with pharmacotherapy and changes in risk factors. It may also be necessary to change the patient's medication and monitor the patient more carefully in light of the evolution of the disease.



When should a patient referral be considered?

Patients who remain in a precarious state, despite being treated with the maximum dose of conventional drugs, should be referred to centres that are experienced in treating severe CHF, have access to experimental drug treatments, and have a well-established transplant program in place.

Conclusion

CHF is an increasingly common disease with a poor prognosis. As the population ages, heart failure will continue to progress and put a greater strain on our already overloaded health-care system. Diagnosing CHF depends on a diligent clinical assessment, which should

include paraclinical tests. Once a diagnosis has been made and it has been confirmed that the condition is not reversible, patient and family education are essential if clinical stability is to be maintained. Though effective treatments are available, they are underused at present. Optimal pharmacological treatment may be a means of controlling this quasi-epidemic and enhancing the quality of life of patients suffering from the disease. *PCard*

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