



Darcy Lamb

In my master's of pharmacy program, I used Saskatchewan Health Data to analyze medications that treat heart failure. I felt there was a need for a basic review of heart failure geared towards pharmacists.

Durant ma maîtrise en pharmacie, j'ai utilisé les données sur la santé de la Saskatchewan pour analyser les médicaments employés dans le traitement de l'insuffisance cardiaque. Il m'a alors semblé qu'il y aurait lieu de faire une révision de base du traitement de l'insuffisance cardiaque à l'intention des pharmaciens.

Heart failure: Back to basics for pharmacists

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Abstract

Heart failure is a clinical syndrome associated with poor quality of life, frequent hospital admissions, and high mortality rates. Since the number of people with heart failure is continuing to rise, it is important to understand the mechanisms that result in disease progression and the medications used to treat it. Clinical trials involving medications that block the neurohormonal systems involved in the disease have shown

significant improvements in both hospitalization and mortality rates. Pharmacists who are not experts in heart failure can contribute to the successful management of these patients by understanding key characteristics and drug therapy goals and by communicating regularly with the heart failure patient. *Can Pharm J* 2008;141:48-57.

Heat failure is a progressive clinical syndrome that impairs the ability of the heart to pump enough blood to meet the metabolic demands of the body.¹⁻³ This "heart malfunction" may be due to a problem with ventricular contraction (systolic heart failure), ventricular relaxation and filling (diastolic heart failure), or a combination of the two. From the initial diagnosis of heart failure, over half of all patients will die within 5 years.⁴⁻⁶

The incidence of heart failure has not increased over the past decade;^{5,6} however, the absolute number of people with a new diagnosis of heart failure continues to rise. Since the mean age of the population is increasing, it is projected that by the year 2025 the number of new heart failure patients will have doubled.⁷ As the number of people with heart

failure continues to grow, so does the cost to the health care system. Currently, the economic impact of heart failure is enormous in the developed world, accounting for 1% to 2% of global health care budgets.⁸

The aim of this paper is to provide a basic review of the major concepts surrounding the development and management of heart failure for the nonspecialist pharmacist (see Box 1 for a glossary of terms). Although there is an enormous body of literature dedicated to the management of this common syndrome, it is not necessary to be an expert to help patients optimize their treatment. By understanding the unique characteristics of heart failure patients, pharmacists may be able to educate and assist patients in managing their condition.

Clinical presentation

The clinical syndrome of heart failure is characterized by signs and symptoms that can include fatigue, pulmonary and peripheral edema, bibasilar rales, pleural effusion, dyspnea, orthopnea, cough, wheezing, and decreased exercise tolerance. In other words, patients exhibit difficulty breathing and can become very short of breath after minimal activity or even when lying down.^{2,3,9} Based on symptoms, a patient's condition can be categorized into 1 of 4 functional classes developed by the New York Heart Association (NYHA), as shown in Table 1.

Systolic versus diastolic heart failure

Simply put, systolic heart failure results from the inability of the heart to contract appropriately, whereas diastolic heart failure results from the heart's inability to adequately fill with blood. Of the 2 categories, it was generally thought that patients with a preserved ejection fraction (diastolic heart failure) had a better prognosis. However, recent data suggest that diastolic heart failure carries a similar risk of death as systolic failure.¹⁰ Because there is little evidence available on the optimal treatment of heart failure with preserved systolic function (indicated by a normal ejection fraction), the following review of medication use in heart failure is based on evidence from trials of patients with reduced ejection fraction, termed systolic heart failure.

Pathophysiology: Neurohormonal activation in heart failure

The first step in the development of heart failure may be due to some kind of injury to the myocardium. Myocardial infarction, hypertension, coronary artery disease, and heart valve disorders are common sources of injury, but many other factors may also be responsible.^{3,9,11-13} In approximately 17% of heart failure patients, a traditional cause cannot be identified because the myocardial damage

is the result of genetic and/or autoimmune processes on the myocardium. This type of heart failure is commonly termed "idiopathic dilated cardiomyopathy."¹⁴⁻¹⁶ Regardless, injury or changes in the myocardium can affect the heart's ability to either contract fully, relax enough to fill with blood, or both, resulting in a prolonged decrease in both cardiac output and function.^{3,17} In response to the reduction in cardiac output, certain neurohormonal systems are activated, primarily the sympathetic nervous system and the renin-angiotensin-aldosterone system (RAAS), which provide short-term maintenance of normal cardiac function.¹⁷ Over time, constant activation of these systems can cause progression of heart failure through sodium and water retention, vasoconstriction, and constant stimulation, which eventually become toxic to the myocardium.^{9,17-19} These factors also promote cardiac remodelling, resulting in a dilated ventricle and a more spherical shape to the heart. These changes can increase myocardial wall stress, cause mitral regurgitation, and increase the risk of arrhythmia.^{19,20}

Effects of sustained RAAS output

In heart failure patients, the adverse effects of RAAS are mostly driven by angiotensin II and aldosterone.^{17,18} Activation of the angiotensin II receptor results in vasoconstriction and release of aldosterone, catecholamines, and vasopressin, leading to sodium and water retention and increased sympathetic stimulation of the heart.¹⁷ Angiotensin II itself can activate growth genes leading to hypertrophy of the myocardium and can cause alterations in the collagen makeup of the heart.¹⁷ Aldosterone increases sodium and water retention and also stimulates collagen production, which results in myocardial fibrosis. Myocardial fibrosis is the process of replacing damaged heart muscle with scar tissue, which seriously impairs the ability of the heart to relax and contract. Aldosterone also increases potassium and magnesium excretion and may prevent uptake of norepinephrine by the heart, thus promoting arrhythmia.¹⁷

Effects of sustained sympathetic output

It has been shown that there is both an increased and sustained sympathetic stimulation of cardiac tissue in patients with heart failure. Over the short

Key points

- The prevalence of heart failure is continuing to increase.
- Certain hormonal systems contribute to the progression of heart failure.
- Drug therapy that blocks these neurohormonal systems has been shown to decrease mortality and hospitalization.
- By improving drug use and optimizing therapy, heart failure patients will have increased quality of life, fewer hospitalizations, and a lower risk of death.

TABLE 1 NYHA functional classification

I	No symptoms
II	Symptoms with ordinary activity
III	Symptoms with less than ordinary activity
IV	Symptoms at rest or with any minimal activity

NYHA = New York Heart Association

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BOX 1 Glossary of terms

Apoptosis	Cell death, sometimes referred to as “cell-suicide.”
Bibasilar rales	A “crackle” sound that physicians can hear when listening to the chest of a heart failure patient.
Diastolic heart failure	Heart failure due to a problem with ventricular relaxation (also called HF with preserved ejection fraction).
Dyspnea	Difficulty breathing.
Ejection fraction	The percentage of blood that is ejected with each contraction of the heart. Patients with systolic heart failure exhibit ejection fractions less than 40%.
Hyperkalemia	Increased potassium levels in the blood. If not detected or left untreated, it can promote the formation of serious cardiac arrhythmias and even death.
Mitral regurgitation	Refers to the backward flow of blood through one of the heart’s major valves, the mitral valve. Mitral regurgitation can adversely affect the amount of blood that can be pumped out with every beat.
Myocyte	Heart muscle cell
Orthopnea	Difficulty breathing when lying down.
Pleural effusion	Fluid that has accumulated just outside the outer lining of the lungs.
Pulmonary edema	When excess fluid has accumulated in lung tissue. This problem often makes breathing more difficult.
RAAS	Renin angiotensin aldosterone system: A pathway of hormones that promote the formation of angiotensin II and aldosterone. These hormones may have beneficial effects early, but eventually become toxic if they remain elevated for long periods of time.
Sympathetic	Refers to the “sympathetic nervous system,” which is responsible for releasing adrenalin-like hormones in the body. Sympathetic nervous system hormones are well known for their ability to increase heart rate, contractility, and blood pressure.
Systolic heart failure	Heart failure due to a problem with ventricular contraction.

term, these “adrenalin-like” hormones help to maintain cardiac performance, but long-term sympathetic activation is harmful to the cardiac muscle.^{17,21,22} This long-term activation leads to many alterations in the heart’s signaling system, one of which is down-regulation of beta receptors. As a result of these and other changes to the heart, routine contraction becomes suboptimal. In addition to contractile dysfunction, chronic elevated sympathetic activity also leads to myocardial necrosis and induction of apoptosis (cell death).^{20,23} Indeed, elevated norepinephrine levels are associated with a higher risk of death in heart failure patients.²⁴

Drug therapy in systolic heart failure

Historically, medical management of heart failure focused on increasing cardiac output. As a result, digoxin was considered a drug of choice for many years because of its positive inotropic effects.³ Digoxin can provide symptom relief and decrease the risk of hospitalization; thus, it is used for

patients with persistent symptoms despite optimized therapy with angiotensin-converting enzyme (ACE) inhibitors and beta blockers. However, digoxin does not prolong the life of heart failure patients, and it is not recommended in asymptomatic individuals unless another condition warrants its use (e.g., atrial fibrillation).^{3,25}

Diuretics have long been valuable medications to control symptoms of heart failure and are used regularly in the vast majority of patients. Fluid accumulation in the lungs is a major problem because it contributes to classic heart failure symptoms such as dyspnea, orthopnea, cough, and decreased exercise tolerance. Consequently, regulation of fluid volume by diuretics is an effective way to control symptoms. The most common diuretic used for heart failure is furosemide, which can provide rapid symptom improvement within hours to days of starting therapy. Although diuretics do not block neurohormonal systems and there are no studies to demonstrate an effect on survival,

TABLE 2 ACE inhibitor heart failure trials²⁶⁻³¹

Trial	CONSENSUS	SOLVD	ATLAS*	AIRE	TRACE	SAVE
Sample size (ACE inhibitor group)	253 (127)	2569 (1285)	3164 (1568 HD, 1596 LD)	1986 (1004)	1749 (876)	2231 (1115)
Description of patients [†]	Chronic HF; NYHA IV	Chronic HF; NYHA II-III	Chronic HF; mostly NYHA III	Post-MI HF	Post-MI HF	Post-MI HF
Drug	Enalapril	Enalapril	Lisinopril	Ramipril	Trandolapril	Captopril
Mean/median follow-up	6.2 months	41.4 months	45.7 months	15 months	Not reported	42 months
Total mortality [‡]	39 vs 54 RRR = 27%	35 vs 40 RRR = 16%	43 vs 45 NS	17 vs 23 RRR = 27%	35 vs 42 RRR = 22%	20 vs 25 RRR = 19%
Hospitalizations [‡]	—	69 vs 74 RRR = 7%	RRR = 13% (for HD)	—	—	—

HD = high dose; LD = low dose; HF = heart failure; NYHA = New York Heart Association classification of heart failure severity; MI = myocardial infarction; RRR = relative-risk reduction; NS = not significant.

*There was no placebo control in this trial.

[†]Chronic HF refers to subjects who were diagnosed with heart failure for a significant period of time prior to initiating ACE inhibitor therapy. Post-MI HF refers to subjects who developed heart failure after a sudden (often severe) myocardial infarction. In this setting, ACE inhibitors were started as soon as possible after heart failure was diagnosed (i.e., in the hospital).

[‡]Percentage of events in active treatment group versus placebo group.

they are considered standard therapy in the management of heart failure patients because of their obvious clinical benefits.¹⁻³

In addition to diuretics, beta blockers, ACE inhibitors, angiotensin receptor blockers (ARBs), and aldosterone antagonists have become the mainstay of drug therapy in heart failure. These medications block the neurohormonal systems most responsible for the progression and worsening of heart failure and can prolong the life of many patients. In addition, they have been shown to improve symptoms over time and increase patient quality of life.¹⁻³ For information on dosing of heart failure therapies, you may refer to Table 2 in “The Canadian Cardiovascular Society consensus conference recommendations on heart failure update 2007: A summary for pharmacists,” available in the online version of the January/February 2008 issue of *CPJ* (www.pharmacists.ca/cpj or www.cpjjournal.ca).

ACE inhibitors

ACE inhibitors have been part of the drug regimen of heart failure for the last 2 decades and should be considered for all heart failure patients.³ They work

by inhibiting the enzyme responsible for the conversion of angiotensin I to angiotensin II and also prevent the breakdown of bradykinin, a vasodilatory hormone. Through this action, ACE inhibitors cause vasodilation and help to attenuate the negative effects of angiotensin II, such as remodelling of the heart.¹⁷ Prevention of this effect is very important because remodelling is associated with further decline in ventricular function and progression of the disease. Various ACE inhibitors have been studied in both chronic heart failure and in left ventricular dysfunction following a myocardial infarction (Table 2). They have been found not only to improve the signs and symptoms of heart failure over time, but also to decrease mortality and hospitalization rates. By using an ACE inhibitor, patients can decrease their risk of death by approximately 23%.²⁶⁻³¹

Although most heart failure patients can take an ACE inhibitor without any serious problems, certain patients may experience an excessive elevation of potassium levels, an unacceptable decrease in blood pressure or kidney function, or a cough that may not allow them to tolerate the medication. If

TABLE 3 Beta blocker heart failure trials^{34-38,40*}

Trial	US Carvedilol HF study	MERIT-HF	CIBIS-II	COPERNICUS	CAPRICORN	COMET†
Sample size (beta blocker group)	1094 (696)	3991 (1990)	2647 (1327)	2289 (1156)	1959 (975)	3029 (1511 carv., 1518 metop.)
Description of patients	NYHA II-III; EF ~ 23%	NYHA II-III; EF ~ 28%	Mostly NYHA III; EF ~ 27%	NYHA IV; EF ~ 20%	Post-MI HF‡; EF ~ 33%	NYHA II-III; EF ~ 26%
Drug	Carvedilol	Metoprolol CR	Bisoprolol	Carvedilol	Carvedilol	Carvedilol vs metoprolol
Mean/median follow-up	6.5 months	12 months	16 months	10.4 months	12 months	58 months
Total mortality [§]	3.2 vs 7.8 RRR = 65%	7.2 vs 11 RRR = 34%	12 vs 17 RRR = 34%	11.2 vs 16.7 RRR = 35%	12 vs 15 RRR = 23%	34 vs 40 RRR = 17%
Hospitalizations [§] (total)	14.1 vs 19.6 RRR = 27%	—	33 vs 39 RRR = 20%	—	—	—

*HF = heart failure; NYHA = New York Heart Association classification of heart failure severity; EF = ejection fraction; CR = controlled release; RRR = relative risk reduction.

†COMET was a comparison trial of carvedilol versus short-acting metoprolol. There was no placebo control.

‡Post-MI HF refers to subjects who developed heart failure after a sudden (often large) myocardial infarction. In this setting, beta blockers were started as soon as possible after heart failure was diagnosed (i.e., in the hospital). All other beta blocker trials shown here were in patients who were diagnosed with heart failure for a significant period of time prior to initiating beta blocker therapy.

§Percentage of events in active treatment group versus placebo group.

possible, these patients should be maintained on an ACE inhibitor, even at lower doses, so as to retain some benefit. Alternatives to using an ACE inhibitor include an ARB (which carries many of the same risks) or a combination of hydralazine and isosorbide dinitrate.^{3,12}

Beta blockers

Although contraindicated in the past, today it is known that beta blockers provide great benefit in heart failure and should be considered for all heart failure patients.^{3,12} They exert their beneficial properties by blocking the sympathetic nervous system effects of norepinephrine at the beta-adrenergic receptors.^{20,21,23} Blocking these receptors in the heart reduces the negative impact norepinephrine has on cardiac remodelling and myocyte survival. Furthermore, in contrast to traditional concerns that beta blockers may be detrimental because they reduce heart rate and cardiac output, these agents actually improve myocardial function by prolonging ventricular filling time, resulting in a more productive heartbeat.³² Since beta blockers reduce heart rate and cardiac output, worsening of heart failure

symptoms may be noted by patients when they first start the medication or after a dosage increase. Thus, it is important to slowly titrate this medication to target doses as tolerated by the patient.³ Although beta blockers can be difficult to tolerate initially, the long-term benefits outweigh the risks of worsening heart failure while titrating the medication.

Three beta blockers have been extensively studied and have shown a decrease in morbidity and mortality in heart failure patients (Table 3). These are metoprolol and bisoprolol, which are beta-1 specific, and carvedilol, a nonspecific beta blocker with alpha-1-blocking properties.^{3,33-39} In regard to metoprolol, there is a long-acting formulation (metoprolol succinate, not available in Canada) that was shown in the MERIT-HF study to significantly reduce mortality by 34% compared to placebo.³⁴ It is unknown if the short-acting metoprolol (metoprolol tartrate) that is available in Canada provides the same benefit, and thus it is not indicated for the treatment of heart failure in Canada. Interestingly, this short-acting formulation was shown to be inferior to carvedilol in the COMET trial.³³ There is not enough evidence to

assume that other beta blockers aside from the 3 mentioned above provide the same benefits,⁴⁰ so the use of other beta blockers in heart failure should be discouraged. Of note, the benefits of beta blockers were observed in these trials despite widespread use of ACE inhibitors. Therefore, both beta blockers and ACE inhibitors should be used in the management of individual patients.¹⁻³ With the introduction of both these medications into the drug therapy regimen of heart failure patients, great strides have been made in reducing the death rate. One-year mortality can now approach 11.5%, compared to the 17% to 28% seen prior to beta-blocker use.^{5,6,34-38}

Angiotensin receptor blockers

Angiotensin receptor blockers have been studied as alternative agents to ACE inhibitors and also as add-on therapy.⁴¹⁻⁴⁹ Like ACE inhibitors, ARBs cause vasodilation and help to attenuate the effects of angiotensin II, but instead of inhibiting the production of angiotensin II, ARBs block the angiotensin II receptor to inhibit its physiologic effects.³ Because angiotensin II can be produced by other pathways besides the angiotensin-converting enzyme, it had been postulated that ARBs may provide superior protection against the detrimental effects of angiotensin II.^{3,41-43} However, ACE inhibitors also promote the accumulation of bradykinin, which may be responsible for some clinically important benefits (as well as adverse effects such as cough) that are not observed with ARBs.^{3,17,18}

Three ARBs have been studied in heart failure patients, namely losartan, valsartan, and candesartan (Table 4).^{41-46,49} Only valsartan and candesartan have shown significant benefits in mortality and hospitalization and are approved as acceptable agents for heart failure patients.⁴²⁻⁴⁶ Overall, there is not enough evidence to conclude that these ARBs are completely equivalent to ACE inhibitors, but they are good alternatives in patients who cannot tolerate an ACE inhibitor for various reasons.³ In addition, some benefit has been shown when an ARB is added to ACE inhibitor therapy,^{43,45} and this combination may be used in patients who are unable to tolerate a beta blocker or who have continued symptoms while receiving ACE inhibitor and beta-blocker therapy.^{1,3}

Aldosterone antagonists

Spironolactone is an aldosterone antagonist that can promote positive outcomes in heart failure patients. Its benefits are thought to arise from the blockade of aldosterone's negative effects on the heart.⁵⁰ In patients with NYHA class III and IV

heart failure (patients with more severe symptoms), the use of spironolactone for an average of 2 years in addition to usual heart failure medication significantly reduced the death rate by 25% (45.9% for placebo vs 34.5% for spironolactone).⁵⁰

Although spironolactone is another useful agent to block the effects of neurohormonal overdrive in heart failure, it (along with the other commonly used therapies) can also contribute to hyperkalemia. Therefore, the addition of spironolactone to existing therapies may not be suitable for all patients because the risk of hyperkalemia may outweigh the benefits in some cases.⁵¹ Thus, patients should only receive spironolactone in combination with their existing drug regimen if careful, continued monitoring of potassium levels can be carried out and the risks are perceived to be low.³

Isosorbide dinitrate and hydralazine

Although used frequently in the past for symptom relief due to their vasodilating action, the use of isosorbide dinitrate and hydralazine has been virtually eliminated and replaced by ACE inhibitors.³ However, a recent study (A-HeFT) tested the addition of isosorbide dinitrate and hydralazine to standard heart failure therapy in black patients with class III or IV heart failure.⁵² The use of this combination significantly reduced the death rate by 39%, first hospitalization for heart failure was significantly reduced by 32%, and quality of life was significantly increased despite the use of existing therapies. As a result, this combination may be used in addition to existing therapies in black patients or patients intolerant of ACE inhibitors and ARBs.^{3,12}

Points clés

- La prévalence de l'insuffisance cardiaque ne cesse d'augmenter.
- Certains systèmes hormonaux contribuent à la progression de l'insuffisance cardiaque.
- Il a été démontré que la pharmacothérapie qui bloque ces systèmes neurohormonaux réduit la mortalité et l'hospitalisation.
- En améliorant l'usage de médicaments et en optimisant le traitement, les patients atteints d'insuffisance cardiaque bénéficieront d'une meilleure qualité de vie; ceci permettra en outre de réduire le risque de décès et l'incidence des hospitalisations.

Web Resources

- American Heart Association — www.americanheart.org/presenter.jhtml?identifier=1486
- Canadian Heart and Stroke Foundation — www.heartandstroke.ca/Page.asp?PageID=24 under the congestive heart failure section
- Canadian Cardiovascular Society — www.ccs.ca/home/index_e.aspx
- The Canadian Cardiovascular Society consensus conference recommendations on heart failure update 2007: A summary for pharmacists — www.pharmacists.ca/cpj or www.cpjjournal.ca (January/February 2008)
- Heart Failure Society of America — www.hfsa.org

TABLE 4 Angiotensin receptor blocker heart failure trials^{41-46,49*}

Trial	ELITE II	ValHeFT	OPTIMAAL	CHARM Overall
Sample size (ARB group)	3152 (1578)	5010 (2511)	5477 (2744)	7599 (3803)
Description of patients	NYHA II-III EF ~ 31%	NYHA II-III EF ~ 27%	Post-MI HF [†]	NYHA II-III EF ~ 39%
Drug	Losartan vs captopril	Valsartan vs placebo	Losartan vs captopril	Candesartan vs placebo
Mean/median follow-up	18 months	23 months	32.4 months	37.7 months
Total mortality [‡]	17.7 vs 15.9 NS	19.7 vs 19.4 NS	18.2 vs 16.4 NS	23 vs 25 RRR = 10%
Hospitalizations [‡] (total)	41.8 vs 40.5 NS	— [§]	65.8 vs 64.9 NS	— [§]

*NYHA = New York Heart Association classification of heart failure severity; EF = ejection fraction; MI = myocardial infarction; HF = heart failure; NS = not significant; RRR = relative risk reduction.

[†]Post-MI HF refers to subjects who developed heart failure after a sudden (often large) myocardial infarction. In this setting, ARBs were started as soon as possible after heart failure was diagnosed (i.e., in the hospital). All other ARB trials shown here were in patients who were diagnosed with heart failure for a significant period of time prior to initiating ARB therapy.

[‡]Percentage of events in ARB group versus the comparator group.

[§]Total hospitalizations were not reported; however, an approximate RRR of 23% was seen for admission for heart failure.

Pharmacist's role

The body of evidence concerning heart failure therapies is expanding rapidly and it can be a challenge to keep up, even for specialist pharmacists. However, pharmacists need not be specialists in the area to support patients in achieving optimal therapy. In fact, appreciation of a few key points will go a long way in helping heart failure patients.

1. Drugs can prolong life in heart failure patients

As mentioned above, several medications work additively to improve quality of life, prevent emergency hospitalizations, and actually prolong life. Thus, patients should be taking multiple medications concurrently, and pharmacists are in a position to ensure that these patients are taking medications proven to provide a benefit. Also, organization of these medications (for example, through the use of dosettes or bubble packing) and education about their purpose (to prolong life and reduce symptoms) is essential to promote long-term adherence. Patients need to be on the right medications and take them regularly. Try to remind them of the benefits they are receiving from these

medications and congratulate them every once in a while for keeping their heart failure in check.

2. Heart failure patients are susceptible to side effects

These patients are often elderly and may not have high blood pressure; regardless, they are supposed to receive at least 2 medications (ACE inhibitor and beta blocker) for their neurohormonal benefits and a diuretic to prevent fluid retention, all of which have the effect of lowering blood pressure. In addition, in order for these medications to provide maximal benefit, they need to be titrated as close to target doses as possible. Therefore, it is critical that they are started at low doses and titrated slowly with close attention to signs of low blood pressure, worsening heart failure symptoms, and electrolyte disturbances. This is especially important for beta blockers, as there may be a transient worsening of heart failure symptoms with their introduction and titration. Pharmacists should pay close attention during dosage titration to ensure patients are free from side effects before doses are increased. When heart failure patients return for refills, inquire

about how long it's been since their dose was last increased. This simple question might open some important dialogue for further reinforcement.

3. Heart failure patients are at risk of fluid overload

These patients have an inability to excrete excess fluid, resulting in pulmonary edema that can seriously impair breathing and add more stress to an already overworked heart. Noncompliance with dietary recommendations is one of the leading causes of worsening heart failure, and, therefore, excess dietary salt (>2500 mg) and excess fluids must be avoided.^{3,12} Keep in mind that anything that can cause fluid retention, including drugs such as glitazones and nonsteroidal anti-inflammatory drugs (NSAIDs), should also be avoided. Taking simple precautions to prevent this fluid retention from occurring might be enough to avert an emergency room visit. Ask patients if they have ever experienced worsening symptoms from eating too much salt. You can gain useful experience by listening to their stories. You may wish to direct them to information about salt restriction in heart failure, such as that provided on the American Heart Association website (www.americanheart.org/presenter.jhtml?identifier=1486).

4. Body weight may signal a deterioration of condition

Patients should be instructed to weigh themselves frequently as rapid weight gain may precede the first signs of fluid retention that could result in a heart failure exacerbation and hospitalization.

Patients should weigh themselves every morning and watch out for worsening symptoms. Any weight gain of more than 2 pounds (1 kg) over 2 days or more than 5 pounds (3 kg) in 1 week is a sign of fluid retention. Some physicians may have a prespecified plan with their patients to increase the dose of diuretic for a few days if their weight does increase. Whether or not there is a prespecified plan in place, patients should not ignore rapid increases in body weight and should seek medical attention if it occurs.

Conclusion

Heart failure is a debilitating disease that affects many elderly people and has a prevalence that is continuing to increase.^{3,7,53,54} Drug therapy that blocks the neurohormonal systems has been shown to decrease mortality and hospitalization rates.³ Standard drug therapy for heart failure patients is comprised of beta blockers and ACE inhibitors, along with diuretics to decrease fluid volume and improve symptoms. Other neurohormonal blockers (spironolactone, ARBs) as well as digoxin and the combination of isosorbide dinitrate and hydralazine also have a place in treating this condition in select patients.¹⁻³ By improving drug use and optimizing therapy, heart failure patients will have increased quality of life, fewer hospitalizations, and a lower risk of death. Simple education and support can help facilitate successful management of these complex patients, and pharmacists should ensure patients are aware of the simple precautions outlined above.¹⁻³ ■

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