HEART FAILURE

Heart failure (HF) occurs as a result of structural or functional impairment of ventricular filling or ejection of blood. Diagnosed primarily on clinical grounds, HF typically manifests as fluid retention, dyspnea, and fatigue, which may progress to overt congestion and pulmonary edema.[1]

In recent years, great strides have been made in the prevention and treatment of cardiovascular disease overall, but those advances have not extended to the setting of HF where, once established, the disease follows a predictable path towards worsened morbidity and death. The statistics speak for themselves—hospitalization rates have been dropping, but the same cannot be said for mortality rates. In 2010, 1 in 9 death certificates (279,098 deaths) in the United States mentioned heart failure. This was up from 1 in 8.6 (282,754 deaths) in 2006.[2] The number of any-mention deaths attributable to heart failure was nearly as high in 1995 (287,000) as it was in 2010 (279,000), and hospital discharges for HF remained stable from 2000 to 2010.

Clearly, the best way to treat HF is to prevent its development in the first place. Lifestyle and dietary measures promoting heart health should be practiced early and throughout a person’s life. Careful lifelong surveillance for and aggressive treatment of hypertension, diabetes, obesity, and coronary artery disease (CAD) is likewise essential.

The pathophysiology of heart failure is complex and multifactorial, involving a variety of initially positive neurohormonal compensatory mechanisms that become maladaptive over time. Conventional medical therapy represents the centerpiece of treatment for people with symptomatic heart failure, with complementary treatments playing an adjunctive role. General treatment goals include:

- Preventing progressive cardiovascular deterioration
- Minimizing symptoms and enhancing quality of life
- Increasing survival

Physiologic aims of treatment in the setting of significant left ventricular dysfunction (left ventricular ejection fraction [LVEF] less than 45%) are:

- Reducing preload and afterload
- Maintaining stable left ventricular function
- Limiting activation of the renin-angiotensin-aldosterone system
- Inhibiting release of neurohormonal factors

Pharmacotherapy considered standard towards achieving these goals includes the use of:

- Beta blockers. Outside of the United States consideration may be given to adding ivabradine (Procoralan), a selective If current inhibitor, for patients with decreased LVEF who are in sinus rhythm with a heart rate above 70 beats per minute.
- Angiotensin converting enzyme (ACE) inhibitors or angiotensin-2 receptor blockers (ARBs) for people who are ACE-intolerant.
- Aldosterone antagonists. Use has been expanded to those with less severe disease
- Diuretics
- For African American patients with HF not responding well to standard therapy, strong consideration should be given to adding hydralazine-nitrate combination therapy

It was not long ago that digitalis glycosides (digoxin) were considered an important part of HF management, especially in the setting of HF with atrial fibrillation. Recent data, however, cast significant doubt on the clinical utility, even safety, of using digoxin for those with HF. In one community-based study, introduction of digoxin therapy in patients with new systolic HF was associated with a 72% increase in all-cause mortality regardless of whether patients were also taking beta-blockers.[3] It is believed that digoxin may increase the risk of cardiac arrhythmias.

Cardiac resynchronization therapy (CRT) appears to reduce the risk of HF exacerbations by improving left ventricular structure and function. This type of treatment had previously been reserved for those with advanced disease, but may now be considered appropriate for those with even moderate HF.

**SELF-CARE**

The following information highlights the importance of being proactive about self-care to prevent heart failure. [4]

Assistance with tobacco and alcohol cessation, as well as healthy weight management, should be available to all patients.

**FOOD AND DRINK**

Adhering to a Mediterranean or otherwise anti-inflammatory diet may help prevent development of HF as well as slow progression of established disease. People with Class C disease or higher (volume overload) may benefit from sodium restriction.

**MOVING THE BODY**

The cardioprotective effects of regular aerobic exercise are well-established, and having HF is not in and of itself a contraindication to participation in fitness activities. Regular exercise may also improve depressive symptoms in people with HF. Tai chi, a form of low impact aerobic exercise, as well as a martial art, has been shown to improve quality of life and mood in patients with HF.
POWER OF THE MIND

Attention to mood can be of critical importance, as negative emotional states may induce autonomic imbalance by promoting sympathetic overdrive and parasympathetic withdrawal. Psychosocial stress is one of the most important triggers for HF exacerbation and decompensation. Depression is an independent risk factor for HF and is extremely common among those with established disease. Depression-specific activation of inflammatory cytokines occurs in people with HF and may lead to worsened morbidity and mortality.

Only a few mind/body therapies have been studied in the setting of HF, but they suggest promise for improving both emotional and physical health, at least in the short-term, and include:[5]

- Biofeedback
- Meditation (mindfulness and transcendental)
- Relaxation response training
- Slow breathing exercises (as clinically appropriate)

SPIRIT AND SOUL

Regular participation in spiritual practices may help promote optimal overall health. Of course, once HF is present, many patients struggle with their spirituality, adding to an already stressful situation. Appropriate attention to the spiritual needs of a person with HF may help them with coping, as well as finding peace with their circumstances.

DIETARY SUPPLEMENTS

Note: Please refer to the Passport to Whole Health, Chapter 15 on Dietary Supplements for more information about how to determine whether or not a specific supplement is appropriate for a given individual. Supplements are not regulated with the same degree of oversight as medications, and it is important that clinicians keep this in mind. Products vary greatly in terms of accuracy of labeling, presence of adulterants, and the legitimacy of claims made by the manufacturer.

HAWTHORN (CRATAEGUS SP.)

Hawthorn is used to make a slow-acting cardiac tonic that has long been favored in Europe. Its active constituents include flavonoids and oligomeric proanthocyanidins. Hawthorn possesses slight hypotensive activity and may offer some ACE-inhibition. Old data reported both subjective and objective improvement in patients with mild forms of heart failure (New York Heart Association [NYHA] classes I and II), but many of these studies were completed before the advent of pharmacotherapy now considered a standard part of HF management, such as ACE inhibitors and beta blockers.
More recent HF studies employing hawthorn in combination with current standard medical therapy report less success, and one retrospective safety analysis in NYHA class II-III HF patients revealed an increased risk for early progression; hospitalization rates were higher, and death rates were slightly higher compared with placebo. Prior to this, hawthorn had generally been considered safe, with some caution over its use in combination with digitalis glycosides. It had been suggested that hawthorn could enhance the activity of digitalis glycosides. Enthusiasm for the use of hawthorn in HF patients has waned significantly.

When used, hawthorn is usually standardized to its content of flavonoids (2.2%) or oligomeric proanthocyanidins (18.75%). The recommended daily dosage ranges from 160-1800 milligrams daily, but it appears to be more effective at daily doses ranging from 600-1800 milligrams.

**COENZYME Q10 (COQ10)**

CoQ10 is a naturally occurring, vitamin-like substance present in small amounts in most diets. It is produced within the body from tyrosine, partially through a common pathway shared with cholesterol synthesis. CoQ10 is found in highest concentrations within the mitochondrial membranes of organs that have significant energy requirements, especially the heart, where it is intricately involved with the electron transport chain and energy production. It also possesses antioxidant and membrane stabilizing effects. Plasma and myocardial CoQ10 concentrations are lower in people with HF regardless of etiology. Many trials of CoQ10 in the setting of HF have been published, but they are of variable quality, and many were performed before the widespread use of ACE inhibitors, beta blockers, and aldosterone antagonists. Nonetheless, the majority suggest a supportive role for CoQ10 with beneficial effects on ejection fraction, end-diastolic volume index, and symptoms. There has even been suggestion of a survival benefit when CoQ10 is added to a conventional therapeutic regimen. More recent studies, however, failed to show any clinical benefit, so the jury remains out.

The optimum CoQ10 dosage for HF is still undetermined, but most practitioners initially prescribe 100 to 200 milligrams daily. Caution is advised for those taking anticoagulation therapy because of possible procoagulant activity, likely as a result of CoQ10’s structural similarity to vitamin K. Patients taking statins may benefit from CoQ10 supplementation, particularly in terms of statin-related muscle pain.

**L-CARNITINE**

Another vitamin-like substance, L-carnitine acts as a specific carrier of fatty acids required for energy production. Myocardial carnitine is most highly concentrated within the left ventricle, and levels are low in patients with HF. L-carnitine may improve muscle metabolism, ventricular performance and exercise tolerance, but a recent investigation suggests that high carnitine concentrations (from the eating of red meat) may promote atherosclerosis through the gut-mediated generation of trimethylamine N-oxide (TMAO).[6] L-carnitine has also been reported to cause an unpleasant body odor in
extremely high doses. When used, the typical dosage is 2 grams daily, with a dose range of 1-3 grams daily.

**ADDITIONAL SUPPLEMENTS TO CONSIDER**

- B vitamins, especially thiamine
- Magnesium. Hypomagnesemia may increase the risk for arrhythmias and sudden cardiac death
- Fish oils. Research findings have been contradictory, but fish oil may reduce incidence of sudden cardiac death due to arrhythmia. Some studies suggest proarrhythmic effects with HF; however, dietary omega-3s are believed to be antithrombotic, antiatherogenic and anti-inflammatory
- L-arginine. This essential amino acid possesses vasodilatory effects; clinicians should use it with caution in elderly patients. One study suggested increased mortality, and it may increase potassium levels when combined with potassium-sparing agents
- D-ribose is a possible metabolic substrate providing additional energy to myocytes

**COMPLEMENTARY APPROACHES**

**ACUPUNCTURE**

A paucity of data exists, but there is some suggestion of improvement in exercise tolerance in the setting of HF.

**SAUNA TREATMENT**

In a preliminary study, repeated sauna treatment (Waon therapy) was shown to improve cardiac and endothelial function, as well as exercise tolerance in patients with HF.

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“Heart Failure” was written by Russell H. Greenfield, MD (2014).

This Whole Health tool was made possible through a collaborative effort between the University of Wisconsin Integrative Health Program, VA Office of Patient Centered Care and Cultural Transformation, and Pacific Institute for Research and Evaluation.

**REFERENCES**


