

Congestive Heart Failure — Living on Borrowed Time

Dave awoke from a fitful sleep, restless, confused, his body bathed in sweat. His pulse shallow and heart pounding, he lay listening to the tell-tale crackle of each laboured breath — and Dave knew that, this time, it was different.

He'd seen the signs many times before: a gnawing fatigue after just the slightest effort; the inability to climb the stairs he'd once bounded up; and the incessant swelling in his legs and feet. The symptoms had come on slowly, almost imperceptibly, and, as usual, Dave had simply put it down to overwork. "Certainly nothing a little 'down-time' won't fix," he had convinced himself.

The breathy crackle became more pronounced and with it the rising dread that something was seriously wrong. "Dammit!" Dave declared silently, with his usual defiance. "This can't be happening to me! Four more years — that's all I need — but how am I going to enjoy my retirement when I can hardly get out of bed any more?"

For Dave, and millions like him, the dream of retirement may remain forever just that — a dream. Congestive heart failure (CHF) strikes an estimated 400,000 Americans each year. Regardless of treatment, one-fifth of those diagnosed with CHF will die within one year and one-half will be dead within five years. Only about 20 percent survive much longer than 8 to 12 years — a prognosis worse than most cancers. For those that do survive, quality of life is often severely compromised.

The American Heart Association estimates that nearly five million Americans are currently living with CHF. Although it is most common in the elderly, CHF can strike at any age and does not discriminate by gender or race, although African Americans are almost twice as likely to die of CHF than Caucasians.

According to figures released by the National Heart, Lung and Blood Institute, the annual number of deaths from CHF climbed from 10,000 in 1968 to 42,000 in 1993, a rise of over 320 percent. Visits to physicians' offices for CHF increased from 1.7 million in 1980 to 2.9 million in 1993. It is predicted that another 15 to 20 million people may develop CHF in the next five years. In fact, the affliction is becoming so common that the National Heart, Lung and Blood Institute refers to CHF as a new epidemic.

Dave's prognosis is not good. There is no cure for CHF and, if not treated aggressively, it is unlikely he will live to enjoy his retirement.

The Heart of the Matter

Congestive heart failure is a term used to describe any condition in which the heart is unable to adequately pump blood throughout the body or is unable to prevent blood from backing up into the lungs. Chronic CHF is most often due to the effects of high blood pressure, previous myocardial infarction (heart attack), disorders of the heart muscle or the valves of the heart, and chronic lung diseases such as asthma or emphysema.

While not a itself a disease, the onset of CHF involves a constellation of pathological changes that can involve several organ systems. The condition is now recognized as the end stage of cardiac disease, the confluence of physiological changes caused by one or more underlying conditions that affect the contractile (pumping) ability of the heart.

Risk factors for CHF include: high blood pressure (hypertension); previous heart attacks (myocardial infarction); arrhythmia (irregular heartbeat); coronary heart disease; dysfunctional heart valves; cardiomyopathy (weakening of the heart muscle); chronic lung disease; congenital heart disease; and diabetes. Certain chemotherapy drugs and the abuse of alcohol and street drugs, such as amphetamines and cocaine, can also increase the risk of CHF. Other contributing factors include smoking, obesity, lack of exercise, high salt intake and emotional distress.

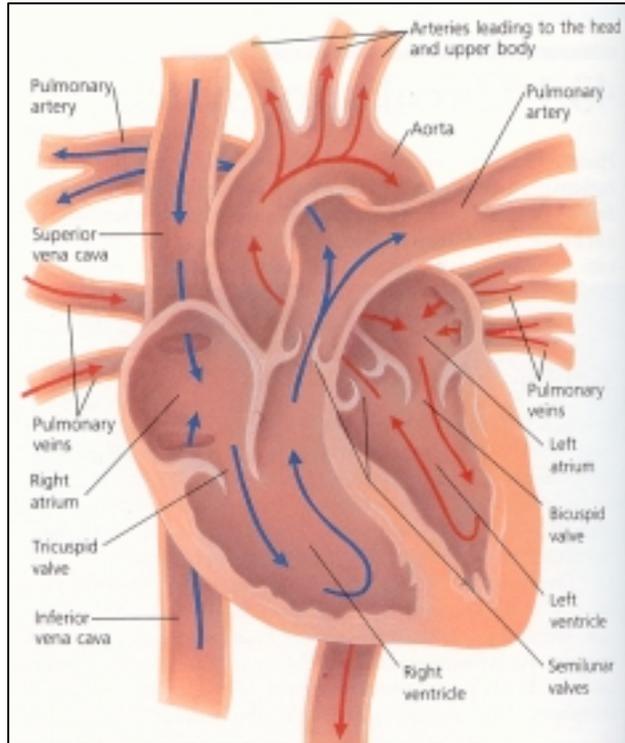
CHF, left unchecked, involves a progressive and ultimately lethal deterioration of the heart muscle. In struggling to compensate for a shortcoming in function (a consequence of a specific disease state) the heart is forced to work harder. Over time, changes to its architecture lead to irreversible damage, including hypertrophy (enlargement) of the heart muscle, tachycardia (rapid heart beat) and kidney malfunction. The longer the heart is forced to overwork, the more its pumping ability will be impaired and the more likely catastrophic failure will occur.

Damage to the heart leads to pulmonary congestion, as blood leaks back and pools in the lung tissue. This gives rise to a range of symptoms including: shortness of breath (dyspnea), fatigue, weakness, and swelling (edema) of the legs and abdomen. As a result of these changes, vital organs no longer receive enough oxygen and nutrients and toxic wastes begin to accumulate. Eventually, vital systems will begin to break down.

A Two-Stage Pump

The heart is a two-staged pump composed of separate pumping systems, one on the right side and the other on the left, separated by a thick muscular wall called the septum. Each stage has two chambers, an atrium (where blood returning to the heart is collected) and a ventricle (where blood is pumped from the heart to the body).

Oxygen-poor blood returning from the body passes into the right atrium and oxygen-rich blood returning from the lungs enters the left atrium. During systole (resting phase), the heart is relaxed and the atrioventricular (A/V) valves, which regulate blood flow to the ventricles, are open. Blood accumulating in the atria passively flows through these one-way valves and into the right and left ventricles, accordingly. Contractions of the atria force the A/V valves closed, pumping the remaining blood into the ventricles. This is followed by powerful



contractions (systole) which force the blood from the right ventricle toward the lungs and from the left ventricle into the body.

A healthy heart will beat for a lifetime; but, once damaged, this amazing biological pump will eventually wear out and fail. The symptomatic presentation of CHF, the end-game of such failure, consequently depends on whether the damage occurs to the left or to the right side of the heart:

Left Ventricular Dysfunction: When the left ventricle, the heart's main pumping chamber, cannot adequately push blood into the body, or when one or more of the valves becomes leaky or stenotic (sticky), blood pools in the lungs and causes the alveoli (air sacs) to become engorged with fluid. Breathing becomes difficult and the transport of oxygen from the lungs into the bloodstream is impaired. This results in shortness of breath (dyspnea), coughing and pulmonary congestion. Individuals with left ventricular CHF frequently waken at night, struggling for breath due to fluid build-up in the lungs that is worsened while lying prone.

Right Ventricular Dysfunction: The right ventricle is responsible for pumping oxygen-poor blood, that has returned from the body, into the lungs for re-oxygenation. If the right ventricle does not work properly this return is impeded, causing fluid retention in the lower extremities. Swelling (edema) occurs in the feet, legs, and ankles, which sometimes spreads to the lungs, liver

and stomach. This build-up of fluids also affects the ability of the kidneys to dispose of wastes, which can lead to kidney failure.

Contractile Phase Dysfunction: CHF is also identified by which contractile phase is most affected. Systolic CHF is the most common and occurs when the left ventricle of the heart is unable to eject adequate amounts of blood during its contractile phase (systole). This leads to lung congestion and edema in the lower extremities. Diastolic CHF, or right-sided heart failure, occurs when the heart is unable to relax during diastole, the resting period following contraction. Fluid entering the heart backs up, causing the veins in the body and tissues surrounding them to swell. Although much less common than systolic CHF, the symptoms are essentially identical.

Severity of CHF

The New York Heart Association has categorized the severity of CHF into four classes according to the limitation of physical activity imposed by the condition. Class I CHF involves no limitation of physical activity; individuals with Class I CHF generally do not experience any shortness of breath, fatigue or heart palpitations with ordinary physical activity. Class II CHF is identified by a shortness of breath, fatigue or heart palpitations with ordinary physical activity. Class III CHF involves a marked limitation of activity; shortness of breath, fatigue or heart palpitations are experienced with minimal physical activity, but patients are comfortable at rest. At the far end of the scale, Class IV CHF imposes complete limitation of activity due to shortness of breath, fatigue or heart palpitations with any physical exertion. Individuals with Class IV CHF can experience these symptoms even at rest.

To help determine the severity of CHF, physicians use a calculation called an ejection fraction, which is the percent of the blood pumped out during each heart beat. An ejection fraction of 50 to 75 percent is normal. In systolic dysfunction the ejection fraction falls below 40 percent, and in severe failure it may drop as low as five percent. In diastolic dysfunction the ejection fraction is paradoxically normal or even high. Because of the interconnectedness of the chambers of the heart, systolic failure may ultimately precipitate diastolic failure.

Treatment Protocols

There is no cure for CHF. It can, however, be effectively treated and relieved with appropriate therapies. It is essential to tackle the underlying causes in order to prevent progression of the condition. Early intervention is crucial — treating heart failure as early as possible offers the best chance for a longer and higher-quality life. The first step in management is to identify and mitigate the primary conditions causing or complicating CHF. These typically include coronary artery disease, abnormalities in the valves of the heart, high blood pressure,

arrhythmia, anemia, and thyroid dysfunction. Early intervention will minimize the uncorrectable damage that can occur if CHF is allowed to progress.

Conventional Treatment

There are a variety of conventional medications used to treat heart failure, each with a specific function: diuretics reduce edema, the build-up of fluids in the lung and body tissues; ACE (angiotensin-converting enzyme) inhibitors open blood vessels to enhance circulation; beta blockers slow the heart rate, reducing the workload on the weakened heart muscles; digoxin increases the heart's ability to contract; and vasodilators open blood vessels to reduce circulatory resistance to blood flow.

While diuretics and ACE inhibitors have the best record for patients with CHF, Carvedilol, a new pharmaceutical medication recently approved by the FDA, has been found to be of significant value to individuals suffering from mild to moderate CHF. Used in conjunction with diuretics, ACE inhibitors and digoxin, clinical trials demonstrate that hospitalization time, patient morbidity and mortality are significantly reduced.

Depending upon the severity of the damage, interventional procedures may also be necessary. These include: balloon angioplasty (a surgical procedure in which a balloon is used to open narrowed or blocked blood vessels of the heart (coronary arteries); coronary stenting (a stent is a small, self-expanding, stainless steel mesh tube that is placed within a coronary artery to keep the vessel open); coronary artery bypass surgery; heart valve surgery; pacemaker insertion; and heart transplantation.

Complementary Treatment

The naturopathic approach to the treatment of heart failure focuses on nutritional and lifestyle modifications designed to reduce the working load on the heart muscle and improve myocardial energy production and contractile function. Consequently, successful treatment of CHF using complementary methods requires a purpose that incorporates those measures needed to mitigate or correct the patho-physiological changes that have occurred during progression of the condition. This includes: the restoration of neuro-hormonal and metabolic integrity; improving myocardial energy and contractile strength; decreasing oxidative stress; improving mineral balance; decreasing vascular resistance; reducing the risk of thrombosis; and preventing arrhythmia.

The keys to enhancing cardiovascular health also include effective stress management, strengthening of the systemic circulatory system and removal of calcified deposits through chelation therapy. Reduction of common risk factors is a prerequisite and should include the avoidance of excessive alcohol consumption and the limitation of salt intake (which increases

both blood volume and pressure). In more severe CHF cases, sodium intake should be limited to 2 g/day and ingestion of water to 1.5-2 L/day.

A detoxification program is beneficial, and the establishment of good dietary habits is essential, since a major cause of heart disease is a poor diet high in refined carbohydrates and saturated fats. As well, a combination of physical activity and stress reduction, along with a healthy diet, can help reduce and maintain optimal body weight, thereby significantly decreasing the daily workload on the heart.

Exercise is Essential: Traditionally, CHF sufferers were counselled to rest and avoid exercise in order to delay disease progression and to promote diuresis (excretion of urine) induced by bed rest. In the last decade, however, research has demonstrated that exercise offers much gain with little risk to the CHF sufferer. While studies documenting the benefits of exercise for CHF patients are fewer than those demonstrating its benefits for coronary artery disease patients, it has been found that most CHF patients who exercise can significantly improve their functional status and quality of life. Regular exercise, including aerobic and light resistance training, may also reduce the risk of death for CHF patients, as it does for patients who have coronary artery disease.

Proper Diet a Must: Studies confirm that diets rich in animal products and saturated fats raise atherogenic LDL cholesterol levels and increase the prevalence of heart disease. Such diets should be avoided by those at risk for CHF. In contrast, diets rich in complex carbohydrates and fibre, where the principal sources of fats are the monounsaturated fatty acids (MUFAs) such as olive oil, modulate cholesterol levels and are associated with a low incidence of heart disease. The evidence clearly shows that a diet enriched with MUFAs lowers LDL (bad) cholesterol, enhances HDL (good) cholesterol and provides resistance to oxidative damage.

Increased consumption of essential fatty acids is also warranted. Oils high in the omega-3 and omega-6 fatty acids, such as flax seed and fish oils, have been found to offer significant cardiovascular protection through their ability to decrease blood pressure, increase prostaglandins that favour dilation, lower blood cholesterol, and decrease circulating triglyceride levels. Supplementation with at least 400 mg of eicosapentaenoic acid (EPA) and 300 mg of docosahexaenoic acid (DHA) is recommended.

The elimination of high-glycemic, high-fat foods and processed foods with a high sodium content is another important dietary consideration in the treatment of CHF. Consumption of foods with a low-to-moderate glycemic index serves to stabilize insulin levels and reduces the risk of insulin resistance, a major contributing factor to diabetes and heart disease.

Those at risk, or already suffering from CHF, should also curtail their consumption of fatty meats. High protein intake in the absence of micronutrients, particularly vitamin B₆, promotes

the accumulation of homocysteine, a potent oxidant and toxin related to the onset of cardiovascular disease. Recent studies show that high levels of homocysteine are responsible for the development of the fatty cholesterol plaques that lead to atherosclerosis or hardening of the arteries. The 1992 Physician's Health Study showed that even mildly elevated levels of homocysteine are directly related to heart disease. Subsequent trials confirm that high levels of homocysteine place people at significantly increased cardiovascular risk.

The Healing Herbs: Several common herbs have been found to be effective in preventing and mitigating CHF. Garlic (*Allium sativum*) protects against heart disease and stroke by intervening in the process of atherosclerosis. Garlic is known to lower cholesterol and blood pressure and possesses diuretic and expectorant actions. Standardized garlic preparations have also been shown to inhibit platelet aggregation and promote fibrinolysis (the natural dissolving of blood clots), thus significantly decreasing the risk of heart attacks, strokes and other cardiovascular events. Like garlic, the humble onion (*allium cepa*) also demonstrates potent blood pressure and blood sugar lowering actions, substantiating the use of both of these herbs in diabetic and cardiovascular patients alike. A commercial garlic product should deliver a daily dose of at least 10,000 mg of aliin; the dosage for onion should range from 2 to 5 ounces (1/4 to 1 cup, freshly chopped).

The berries and flowers of Hawthorne (*Crataegus sp.*), a spiny shrub native to Europe, have been used as mild heart tonics and diuretics in several heart disorders, including CHF. Numerous clinical studies have found that standardized extracts show promise as adjunctive agents for the treatment of left ventricular dysfunction.⁴⁵⁻⁴⁸ Extracts of the plant have been clinically proven to reduce blood pressure, angina attacks, and serum cholesterol levels, improve exercise tolerance and prevent the deposition of cholesterol in artery walls. Its ability to dilate coronary blood vessels is well established, as is its ability to enhance energy production and improve the mechanical function of the myocardium. Preparations have been found to be very effective in the early stages of CHF. Hawthorn extract demonstrates several other beneficial effects, including antioxidant activity, ACE inhibition, and mild reduction in systemic vascular resistance.^{49,52,53} The recommended dose ranges from 160 to 900 mg per day. The recommended daily dose of an infusion of the dried berries is 3-5 g or 4-5 mL of Hawthorne tincture.

Coleus forskoli, a small perennial member of the mint family, has been shown to increase the activation of the enzyme adenylate cyclase, which regulates the production of cyclic AMP. Its basic cardiovascular actions improve the contractility of the heart and lower lower blood pressure. According to Murray (1995), the most useful clinical applications of the herb include the treatment of hypertension, CHF and angina. The current recommended dose is 50 mg 2-3 times per day for an extract standardized to contain 18 percent forskolin.

Ginger (*Zingiber officinale*) possesses an impressive range of pharmacologic properties, including the inhibition of inflammatory prostaglandins and platelet aggregation, cholesterol-

lowering actions and potent antioxidant effects. It also has demonstrated impressive cardio-tonic activity, a consequence of its ability to enhance the uptake of calcium in the myocardium. While there remain many questions as to the best form and dosage of ginger for clinical applications, its complete lack of toxicity provides for considerable range. It is commonly used in India at dosages of 8 to 10 g per day.

Gugulipid is derived from the myrrh tree (*Commiphora mukul*), native to Arabia and India. The pharmacology of gugulipid focuses on its impressive ability to lower triglycerides and cholesterol. The cholesterol-lowering action is particularly beneficial as the herb lowers very low density (VLDL) cholesterol and low density cholesterol (LDL) while simultaneously increasing the high density (HDL) lipoprotein. The herb is also known to improve the metabolism of the heart and protect the myocardium from oxidative stress. Clinical studies show that 25 mg of guggulsterone three times a day is an effective treatment for elevated cholesterol and elevated triglycerides.

Panax ginseng, also known as Korean ginseng has been shown to reduce total serum cholesterol, triglyceride and fatty acid levels while increasing HDL (good) cholesterol levels. Platelet adhesiveness is also reduced. Its effect on blood pressure is paradoxical, as it appears to act as a hypertensive at low doses; administered at higher doses, however, it acts as a hypotensive. This fact must be kept in mind in any clinical application involving hypertension. The standardized dose for a high quality ginseng root is 4 to 6 g per day.

Turmeric (*Curcuma longa*), a perennial herb of the ginger family, contains curcumin, an effective antioxidant, antiinflammatory and cardioprotective agent. Long used as an anti-inflammatory and analgesic in Ayurvedic (East Indian) and traditional Chinese (TCM) systems of medicine, curcumin is known to lower cholesterol levels and inhibits platelet aggregation. Turmeric has a noted lack of toxicity and should be consumed liberally in the diet.

The Critical Role of Micronutrients

The scientific evidence that micronutrients play a central role in cardiac health is no longer disputed. A comprehensive restoration of adequate myocyte (heart cell) nutrition is important to any therapeutic strategy designed to benefit patients suffering from CHF.^{15,16} In this regard, the efficacy of nutritional supplementation is well established.

Supplementation with essential nutrients is critical for individuals suffering from CHF. Vitamins and other cofactors serve as bio-energy carriers to millions of heart muscle cells, enhancing myocardial energy production. Research shows that the most common symptoms of heart failure improve when patients supplement with vitamins. Other clinical studies demonstrate the value of vitamins, minerals and other nutrients in the alleviation of CHF symptoms, including shortness of breath, fatigue and edema.

Nevertheless, traditional practitioners frequently disregard the effective use of nutritional supplementation in restoring and maintaining proper cardiac function. Also, frequently overlooked is the synergistic relationship between several vitamins, minerals and antioxidants that can have a profound effect on general cardiac function.

Most symptomatic patients with CHF are significantly malnourished. Several specific metabolic deficiencies have been found in the failing myocardium, including reduced levels of l-carnitine, coenzyme Q₁₀, creatine and thiamine. These nutrients and cofactors are important for proper myocardial energy production. Individuals suffering from CHF also present with a deficiency of taurine (an amino acid integral to intracellular calcium homeostasis), evidence of excessive myocardial oxidative stress, and impaired cellular antioxidant status. Deficiencies of carnitine and taurine are well documented and result in dilated cardiomyopathy (thinning and weakening of the heart muscle) in animals and humans.

Coenzyme Q₁₀ : As its name implies, ubiquinone, or coenzyme Q₁₀ (CoQ₁₀), plays an important role in the energy production of all cells, but is particularly prevalent in heart cells. It has been used for decades as a nutritional supplement for cardiovascular disease¹⁸ and is found in high concentrations within the mitochondrial membranes of organs that have significant energy requirements, especially the heart.¹⁹⁻²² A powerful antioxidant, coenzyme Q₁₀ also exerts membrane stabilizing²⁴ effects, protecting the membranes of the mitochondria from oxidative damage. The first clinical application of coenzyme Q₁₀ in cardiovascular disease was reported in 1967.²⁵ Since that time, numerous studies have evaluated the efficacy coenzyme Q₁₀ in the treatment of CHF.

CoQ₁₀ plays an important role in the terminal stages of cellular respiration carried out within the mitochondria. Because of the high energy demand of heart muscle cells, the presence of CoQ₁₀ is critical in maintaining proper heart muscle function. Research demonstrates the beneficial effects of the coenzyme on improvement of ejection fractions (a measure of the percent of the blood pumped out during each heart beat)⁶⁻²⁸ and end-diastolic volume (a measure of cardiac filling between beats, related to diastolic function).^{27,29} Supplementation with coenzyme Q₁₀, taurine and carnitine is associated with a reduction in left ventricular end-diastolic volume in patients with left ventricular dysfunction.¹⁷

CoQ₁₀ supplementation has also been found to reduce the symptoms of pulmonary edema,^{30,31,32} whereas depletion of the coenzyme results in a significant decline in cardiac function and the clinical manifestation of associated symptoms.³³ Other studies suggest an improvement in survival rates when coenzyme Q₁₀ is added to conventional therapies.^{34,35} While the optimal dose of coenzyme Q₁₀ for CHF is not yet defined, studies have utilized dosages ranging from 30 to 600 mg daily. Most practitioners prescribe 100-200 mg daily.

Magnesium: The primary function of magnesium is enzyme activation; consequently, the mineral plays a central role in energy metabolism. Cells with the highest magnesium concentration are those that are the most metabolically active, such as the heart. Magnesium plays an important role in the functioning of the entire cardiovascular system. Its efficacy in preventing heart disease and strokes is now widely accepted. There is now a wide body of knowledge heralding its importance in the treatment of a wide range of cardiovascular disorders, including acute myocardial infarction, angina, cardiac arrhythmias, cardiomyopathy, high blood pressure and congestive heart failure.

Low magnesium status has been linked with tachyarrhythmia (a fast irregular heart beat) and increased mortality in patients with CHF.⁵⁴ The research shows that the use of magnesium supplements in these situations may be beneficial for treating and preventing life-threatening conditions. Magnesium supplements can be administered safely either orally or by other means depending on the situation.^{55,56} While many nutritional experts recommend an optimal daily intake of magnesium based on body weight, oral supplementation with magnesium citrate at 300 mg/daily for 30 days has been shown to achieve substantial improvement in intracellular magnesium status.⁵⁵

Potassium: Potassium is an electrolyte, a mineral that conducts electricity when dissolved in water. Consequently, it plays a pivotal role in muscle and nerve cell function, including the contractile function of the heart. Potassium deficiencies are characterized by weakness, fatigue, confusion and heart disturbances — symptoms characteristic of CHF. Many studies confirm that increasing potassium intake can significantly lower both systolic and diastolic pressure, particularly in hypertensive subjects. Recent evidence suggests that potassium supplementation may be particularly useful in lowering blood pressure in people over 65 years of age who, as a group, tend to not respond well to hypotensive drug therapy. Typically, such studies use dosages ranging from 2.5 to 5.0 grams per day.

There are several other minerals and vitamins that may play an important role in the prevention and treatment of CHF. Calcium shows promise in the reduction of blood pressure, particularly in elderly hypertensives. Epidemiological evidence supports a link between calcium supplementation and reduction of hypertension; however, the results are not as persuasive as those for potassium and magnesium. Studies show that 1.4 to 2.0 grams per day of elemental calcium can significantly reduce diastolic and systolic pressure in human subjects.

The B-complex vitamins, including vitamin B₁ (thiamin), vitamin B₂ (riboflavin), vitamin B₃, (niacin), vitamin B₅ (pantothenic acid) and vitamin B₆, are all involved with energy production in the cell. As such, supplementation with these nutrients can help improve cardiac function, contractile performance and physical endurance. Evidence of the synergistic role of vitamins B₆, B₁₂ and folic acid in reducing blood homocysteine levels and cardiovascular risk is substantive. Ubbink and co-workers (1993) found that supplementation with folic acid will lower

homocysteine levels only if adequate blood levels of vitamins B₆ and B₁₂ are also present. This apparent synergy between the vitamins B₆, B₁₂ and folic acid in reducing cardiovascular risk has been reported in several recent studies. Because high circulating homocysteine levels are a primary risk factor for atherosclerotic disease, supplementation with all three B-vitamins is warranted. This is particularly so for the elderly, who demonstrate an age-related decline of vitamin B₁₂. Recommended median daily dosages for these nutrients are: 50 mg of vitamin B₁, 43 mg of vitamin B₂, 75 mg of vitamin B₃, 75 mg of vitamin B₅, 63 mg of vitamin B₆ and 300 µg of vitamin B₁₂ (MacWilliam, 2003).

The antioxidant triad of vitamins C, E and beta-carotene has also proven effective in the reduction and prevention of heart disease. The relationship between dietary intake of vitamin C, vitamin E, beta-carotene and the reduction in risk of coronary mortality has been shown to be significant for both sexes. In a study relating antioxidant intake to the prevalence of coronary artery disease it was found that blood levels of vitamins C, E and beta-carotene were significantly lower and oxidized lipids significantly higher in coronary disease patients, compared to the normal population.

Other studies provide more evidence of the preventive role of vitamins C, E and beta-carotene in the reduction of the risk of ischemic heart disease and myocardial infarction. The evidence for the mitigation of risk is so strong that some investigators conclude a major determinant for heart attack may, in fact, be vitamin deficiency.

Other Nutritional Factors

Another vitamin-like substance is l-carnitine. This compound is essential in the transport of fatty acids into the mitochondria for energy production. Carnitine appears to have beneficial effects on CHF.³⁶ Several double-blind clinical studies have shown that l-carnitine is able to improve cardiac function in CHF patients.³⁷⁻³⁹ After six months of use the ejection fractions were shown to increase up to 13.6 percent.^{38,39} What's more, the longer l-carnitine was used, the more dramatic was the improvement. Long-term administration of l-carnitine has been shown to improve ventricular function, reduce systemic vascular resistance, and increase exercise tolerance.^{38,40} The dosage used in most studies is 1-3 grams.

Taurine is an important nutrient found in very high concentrations in excitable tissue; its deficiency is a frequent cause of heart failure. Patients diagnosed with CHF respond favorably to taurine therapy: several double-blind studies confirm that supplementation with the amino acid significantly reduces symptoms of CHF.^{41,42} Taurine promotes natriuresis (sodium excretion) and diuresis (water excretion), thus minimizing many of the adverse actions of angiotensin II, a regulatory protein in the blood that causes profound vasoconstriction with a consequent increase in blood pressure. Since the ACE (angiotensin-converting enzyme) inhibitors are the mainstay in the allopathic treatment of CHF, this action of taurine is highly important.⁴³ The dosage for taurine supplementation is 2-3 grams daily.

Alpha lipoic acid, also known as thioctic acid, is a vitamin-like antioxidant that plays an important role in aerobic metabolism. It is involved, along with thiamin and niacin, in two vital energy-producing reactions in the production of ATP (the body's storage form of energy). Supplementation with lipoic acid may improve energy metabolism in individuals with lower than normal levels, such as those with CHF. The active form of lipoic acid, dihydrolipoic acid (DHLA), is also a powerful antioxidant and, because it is water soluble, is effective against a broader range of free radicals than either vitamin C or vitamin E. Recent evidence suggests that lipoic acid can, in fact, mimic the effect of these important antioxidants. In addition, lipoic acid and DHLA regenerate and synergize the activity of several antioxidant molecules, including vitamin C, glutathione, coenzyme Q₁₀, and alpha tocopherol (vitamin E). Several clinical studies confirm that lipoic acid has a wide range of therapeutic uses related to its efficacy as a cellular energy booster and antioxidant. While maintenance doses of 20-50 mg per day are recommended (Murray, 1995), therapeutic doses of up to several hundred mg per day have been used.

Hormonal Control

Life is a balance between anabolic (building up) and catabolic (breaking down) processes. Early in life, anabolic processes, driven by the hormone testosterone, dominate the scene. As we grow older, the balance begins to change and catabolic influences, controlled primarily by the hormone cortisol, begin to predominate. Andropause and menopause herald the natural cessation of reproductive function — changes that also mark the end of natural protection against heart disease. The accumulation of excess cholesterol, impaired carbohydrate metabolism, decreased fibrinolysis, and several other symptoms associated with heart disease become evident at this time.

For these reasons, age is the most powerful risk factor for the onset of heart disease and CHF.

Despite the fact that the prevalence of CHF increases with age, the correlation between increased risk and the reduction in gonadal function has not yet been well explored. The fact remains that CHF is an infrequent event before these age-dependent changes occur. It is therefore plausible that steroid hormone therapy may offer significant cardiovascular protection for those in their later years.

We know that CHF is basically a metabolic disturbance. We also know that hormonal function is closely related to metabolic function — which is usually abnormal in patients with CHF.⁵⁹ Several studies show that CHF patients also have relatively low levels of DHEA, testosterone, estrogen, and IGF-1.⁶⁰⁻⁶⁵

Optimal levels and correct ratios of the steroid hormones pregnenolone, dehydroepiandrosterone (DHEA), progesterone, estrogens, testosterone, and cortisol are necessary for maintaining optimal health in both females and males. It stands to reason that

alteration of these hormones may play a significant role in the onset of CHF. Therefore, correcting age-induced hormonal imbalances may ameliorate metabolic disturbances and thereby serve as an effective preventive therapy for CHF. This rationale is supported by a study in which patients received hormone therapy (HT) with natural hormones, including pregnenolone, DHEA, triestrogen gel, progesterone gel, and testosterone gel, for correction of high levels of cholesterol.⁷¹ One hundred percent of patients responded positively. These results, although not conclusive, suggest that HT could be a very promising adjunctive treatment for CHF.

Testosterone therapy has been proposed as a useful ancillary treatment for men with CHF.⁶⁶ This hormone reduces blood pressure and enhances relaxation of brachial arteries.⁶⁷⁻⁶⁹ Low circulating levels of testosterone may therefore contribute to the generalized increase in vascular tone found in patients with CHF. This noted vasodilator effect could also be important in relieving pulmonary congestion. Testosterone therapy may also improve cardiac function by reducing workload on the heart and by increasing coronary blood flow. It is currently being considered as a therapy for augmenting skeletal muscle strength in patients with CHF.⁷⁰

The use of androgens (male hormones) to redress the catabolic/anabolic imbalance of CHF has not been widely studied; however, androgen replacement therapy could potentially ameliorate CHF symptoms by improving cardiac and vascular function and by increasing strength and endurance. It may also suppress cytokine (small inflammatory proteins) activation which leads to progression of the condition. Unquestionably, the potential benefits of this approach call for further investigation.

Summary

Congestive heart failure is a progressive, debilitating condition that, left unchecked, will terminate with catastrophic failure of the body's primary pump, the heart. For that reason, individuals diagnosed with CHF must seek immediate attention to attenuate the progression of this potentially lethal condition. Intervention with conventional drug therapies can provide effective relief of symptoms, particularly if addressed in the early stages of the affliction; however, patients following the pharmaceutical approach are often confronted with serious and deleterious side-effects from the use of these medications.

A holistic approach to treatment, involving dietary and lifestyle modifications — including a daily regime of aerobic and light resistance exercise, and supplementation with several nutritional factors, including herbal remedies — can have a marked impact on reducing the severity and progression of CHF. As well, recent evidence indicates that neuroendocrine regulation of the anabolic and catabolic processes, involving modulation of the cortisol/DHEA (catabolic/anabolic) ratio and associated testosterone levels, may prove to be a promising avenue for preventive care.