Naturopathic Management of Infections of the Heart and their Sequelae

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Introduction

Acute infections of the heart present infrequently to the alternative medical care setting, but their acute manifestations and chronic sequelae are important and demand the attention and consideration of the integrative medicine practitioner. Moreover, chronic infections of any type are a risk to cardiovascular health. They can result in many of the same devastating sequelae and chronic heart pathologies as the better-appreciated, acute infections of the cardiovascular system.

Acute cardiovascular infections often present first to the emergency department setting, to hospitals, or to other conventional medical settings; and that is for the best. The management of acute, life-threatening conditions is where conventional medicine excels, and many of these conditions demand heroic management.

Conversely, the downstream manifestations and chronic sequelae of past cardiovascular infections often present to the integrative/alternative care setting. Management of chronic disease is where alternative and integrative medicines excel. Chronic conditions often demand physiologic management and the rebuilding of the body starting at the cellular level.

Heart failure is a condition that is rampant in the US, with a prevalence of over 5 million cases and a lifetime risk of 20%, or 1 in 5 people.[1,2] Heart failure results from many initial causes, acute and chronic infections of the heart among them.

In the first part of our discussion, we will turn our attention to the recognition of some of the more common types of acute and chronic infectious heart diseases. We will then discuss the manner in which these conditions lead to chronic heart failure.

The latter part of our discussion will focus on the treatment of all types of chronic heart failure and cardiomyopathies using natural medicine and illustrate the treatments with which we have had success in real patient cases.

Acute Rheumatic Fever

Acute rheumatic fever (ARF) is an inflammatory consequence of a recent past infection with group A streptococcal (GAS) bacteria. It is an immune-mediated syndrome that we believe results from the fact that certain antigens on the heart valves, myocardium, and synovium of the joints are similar to antigens on GAS, a phenomenon referred to as "molecular mimicry."[3] Thus, the immune system attacks both the bug and the body.

Acute rheumatic fever (ARF) remains the leading cause of cardiovascular-related death during the first five decades of life worldwide. It is largely a disease of youth, with nearly all cases occurring in children between ages 5 and 15 years. ARF results from GAS pharyngitis only and not from other GAS infections such as scarlet fever, cellulitis, impetigo, or toxic shock. It can occur in up to 3% of cases of untreated strep throat and up to 50% of strep throat cases in someone who has previously had ARF.[4–6]

The initial episode of ARF manifests during the period two to four weeks after a GAS throat infection and most commonly presents with migratory arthritis of the large joints. Inflammation of the heart and heart valves is common and is an important indicator of severity, prognosis, and risk of future reactivation and sequelae.[6] Initial heart involvement can be severe enough to cause acute heart failure. When a patient presents after a recent history of strep throat, the astute clinician always auscultates the heart to rule out valvular sequelae. Other classic manifestations such as chorea, subcutaneous nodules, and the skin rash erythema marginatum are much less common. The diagnosis of ARF is made by confirming a recent past GAS pharyngitis infection and by the presence of positive Jones criteria (Table 1, p. 58).[5]

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Table 1
Diagnosing Acute Rheumatic Fever, "Jones Criteria," 1992 Update

Major manifestations	Carditis, polyarthritis, chorea, erythema marginatum, subcutaneous nodules
Minor manifestations	Arthralgias, fever, elevated erythrocyte sedimentation rate, elevated C-reactive protein, prolonged PR interval on EKG
Evidence of antecedent group A strep infection	Recent past positive throat culture or rapid antigen test, elevated or rising anti-streptolysin O, or anti-DNase B antibody titers.

Reference: Guidelines for the diagnosis of rheumatic fever. Jones Criteria, 1992 update. Special Writing Group of the Committee on Rheumatic Fever, Endocarditis, and Kawasaki Disease of the Council on Cardiovascular Disease in the Young of American Heart Association. *JAMA*. 1992;21;268(15):2069–2073.

Rheumatic Heart Disease

The most common and severe long-term consequence of ARF is rheumatic heart disease (RHD). Rheumatic heart disease is the most common cause of acquired heart disease in the world and occurs in 50% of those patients who suffered cardiac involvement at the time of acute rheumatic fever. Symptoms of RHD may not arise for decades after the rheumatic fever episode. The mitral valve of the heart is most commonly involved. The aortic valve can be involved in addition to or instead of the mitral valve. The tricuspid valve is more rarely involved. The valvular disease resulting from RHD may cause subsequent heart failure and may ultimately require valve replacement.[7]

Infective Endocarditis

Infective endocarditis (IE) is caused by the population of the endothelial layer of the heart valves or structures with any type of microorganism. The lining of the heart and valves is naturally resistant to infection, so the onset of infective endocarditis typically requires two conditions: first, that previous damage to the endothelial layer of the heart has occurred and allowed a nidus for the infection; second, that infectious agents are present in the blood. Some highly virulent organisms can cause IE without previous endothelial damage, but in the majority of cases, the infection begins at the site of previously sterile vegetation.

Sterile vegetations are collections of fibrin and platelets that have accumulated at sites where the endothelium has been damaged. When the endothelium is damaged, tissue factor is released to initiate healing. If the healing process is overly vigorous, it can result in accumulations of tissue, vegetations. These vegetations can later become a place for infective organisms in the blood to collect and create colonizations.

Bicuspid aortic valve (BAV) is the most common congenital heart defect in the US, affecting between 1% and 2% of people.[8] It is often asymptomatic and is just one of the conditions that can result in vegetations. Other conditions that can create vegetations include mitral valve prolapse, prosethetic heart valves, other congenital heart defects, rheumatic heart disease, and cardiomyopathies.[9]

The demographics of those affected by infective endocarditis are changing. While the predominant number of cases still occur in males, female cases have risen more that fourfold over the last 35 years. More than half of IE cases occur in those over 60 years of age. Risk factors for IE include intravenous drug use (IVDU), but also include the presence of prosethetic heart valves, structural heart disease, hemodialysis, ulcerative lesions of the colon (such as inflammatory bowel disease and colon cancer), organ transplants, immunocompromised states, including pregnancy, and previous bouts of infective endocarditis.[9]

Bacterial endocarditis is the most common form of IE and can be either acute or subacute. Staphylococcus bacteria, especially *S. aureus*, are the most common cause of the acute form, while Streptococcus bacteria, especially *S. virdans* from the gingiva of the mouth, and Enterococci from the gut are the most common causes of subacute IE.[9] Other than the microbial etiology, the primary difference between the two conditions is in their pathological pace.

Heart murmurs and fevers are often the first symptoms that come to mind when we think of IE. While these two symptoms will become present almost universally over time, they may be present in less than 15% of cases of subacute IE cases and in less than 50% of cases of acute IE at the time of initial presentation. When murmurs develop, they are typically regurgitant murmurs of the aortic or mitral valves, though IV drug users have a greatly increased risk of right-sided involvement and may present with murmurs of the right-sided valves. Up to 35% of cases may present with central nervous system involvement, including transient ischemic attacks, strokes, brain abscesses, and encephalopathy.[3]

It is important to scrutinize any potential IE patient for signs of septic emboli and immunologic involvement in the form of Roth spots, petechiae of the conjunctiva or buccal mucosa, Osler nodes, Janeway lesions, and splinter hemorrhages. Diagnosis of IE is made according to the Modified Duke Criteria. IE is 100% fatal unless treated. Typically, 2 to 8 weeks of IV antibiotics are required.[9–12]

Antibiotic Prophylaxis and Prevention of IE

In 2007, the consensus committee on prevention of IE, comprising experts from the American College of Cardiology, the American Heart Association, and others from various disciplines, including infectious disease experts, made significant updates to the 1997 recommendations regarding the use of preprocedural antibiotics for the prevention of IE. A full discussion of those recommendations goes beyond the scope of this article, but it is sufficient to say that prophylaxis is now recommended for a more limited number of people. There are also a more limited number of procedures that require prophylaxsis, even in high-risk individuals. Furthermore, the committee has made several new confirmations that certain procedures (such as vaginal birth and tattooing) do not require prophylaxis.[12]

Pericarditis

Pericarditis is an inflammatory condition of the tissue layers that surround and enclose the heart. There are many kinds of pericarditis with many potential causes. For our purposes, we will discuss only the types of pericarditis caused by infections. Any organism can infect the pericardium. Most cases of uncomplicated acute pericarditis are caused by viruses or are idiopathic. Acute pericarditis affects from 2% to 6% of the US population annually; it affects more men than women and more adults than children.[13]

It has long been thought that Coxsackie viruses A and B and echovirus were the most common viral causes of pericarditis. This may still be true in pediatric populations; however, adult cases are now considered to be more frequently caused by the herpes family of viruses and HIV. The herpes family includes not only herpes simplex viruses 1 and 2 and varicella-zoster virus, but also cytomegalovirus, Epstein-Barr virus, human herpesvirus 6 (the cause of the childhood illness roseola infantum), and human herpesvirus 8 (the cause of Kaposi's sarcoma).[14,15] Bacteria, fungi, and atypical organisms can also cause pericarditis.

Acute pericarditis typically presents with mild to severe substernal chest pain that is sharp and stabbing, though it can also be dull. The pain can radiate anywhere into the chest, back, jaw, arms, and shoulders and may convincingly mimic a heart attack. Unlike a heart attack, the pain of pericarditis is typically worse with deep breathing, changing positions, or lying recumbent, and better leaning forward. Other signs such as tachycardia, fever, and general findings consistent with infection may be present.[16]

The sine qua non of pericarditis is the pericardial friction rub, which can be appreciated while listening to the chest with the diaphragm of the stethoscope along the left sternal border. The rub may have one, two, or three components and is a highly specific finding for pericarditis, though it may only be present in 35% to 85% of cases.[16,17] A highly specific set of electrocardiographic findings are also present in this illness depending on its stage.

Most cases of acute, uncomplicated pericarditis are self-limited and require treatments that are supportive, anti-inflammatory, and aimed at the suspected etiology of the illness.[18–20] It is important to monitor these patients for complications, including pericardial effusion, tamponade, and hemodynamic compromise. Complicated or recalcitrant cases or those with severe effusion can require more intensive treatment considerations and hospitalization.

Myocarditis

Myocarditis is the infection or inflammation of the heart muscle itself. It can occur separately from or in combination with pericarditis. Viral infections are far and away the most common causes of myocarditis and can progress quickly to acute heart failure or smolder slowly for decades, resulting in structural derangement and chronic heart failure.[21]

Coxsackie B virus, echovirus, and adenovirus were long considered the most common causes of myocarditis. Influenza viruses, parvovirus B19, herpes viruses, and HIV were considered to be less common causes. More recently, however, genomic investigations have revealed that parvovirus B19 (the cause of the childhood exanthem fifth disease), and human herpesvirus 6 (the cause of the childhood exanthem roseola infantum) are the most commonly isolated organisms in cases of suspected viral myocarditis.[22-29]

The epidemiology of viral myocarditis is difficult to accurately ascertain, but estimates indicate that the prevalence may be as high as 5% to 6%.[29–32] Moreover, the incidence of dilated cardiomyopathy caused by viral myocarditis may be as high as 10%.[33] There are many proposed mechanisms by which viral infection of the heart may lead to the derangement and dysfunction of the heart muscle, called cardiomyopathy. No one mechanism has been demonstrated to be the primary cause of cardiomyopathy resulting from viral myocarditis, but the association between the two has long been appreciated as clinically important.[34]

Cardiomyopathy

Cardiomyopathies are conditions that affect the heart muscle, derange its structure, and disrupt its appropriate function. Cardiomyopathies are classified into several categories. A full discussion of classifications and consequences of the various types of cardiomyopathies is the subject of another complete article. For our purposes, we will confine our discussion to dilated cardiomyopathy (DCM) because it is most closely associated with infections of the heart, while other cardiomyopathies are more often genetic, infiltrative, or idiopathic in etiology.

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Dilated Cardiomyopathy

DCM is characterized by enlargement of the ventricular space and contractile dysfunction. There are many potential causes of DCM, but gene mutations and viral infections are among the most common. Secondary causes such as ischemia, hypertension, and valve disease can also lead to ventricular dilatation and dysfunction.

The prevalence of DCM is estimated at 36 cases per 100,000, but this may be a vast underestimate due to the presence of a significant percentage of asymptomatic cases. There are approximately 10,000 deaths and 46,000 hospitalizations annually in the US resulting from DCM, and it is the number one indication for cardiac transplant in the US.[33,35] DCM may be completely asymptomatic even in moderate to severe cases. DCM may or may not lead to overt heart failure. It may result in problems with ventricular pumping or filling (systolic or diastolic dysfunction, respectively) and it is an important cause of heart failure.[36,37]

Infectious Heart Disease as a Prelude to Heart Failure

We have arrived at the hub of our discussion, heart failure. Each of the conditions that we have discussed until now have the potential to cause heart failure: many of them can do so during their acute phases; all of them can do so in their chronic sequelae. Heart failure has many potential causes, many causes which are more common than those that we have discussed thus far. For instance, ischemic heart disease is the primary cause of heart failure in the US, responsible for 42% of cases. [36,37]

Regardless of etiology, heart failure is one of the most common cardiac conditions in our country, affecting 1 in 5 people (20%) at some point in their lives.[1,2] While acute heart failure often requires hospital management, it may be amenable to outpatient management in certain circumstances. Even in the outpatient setting, acute heart failure demands the attention of a medical professional with a great deal of expertise in managing the condition. Many patients who experience acute heart failure will remain in chronic failure even after acute resolution; a large number will battle chronic heart failure for the remainder of their lives. Chronic heart failure is extremely amenable to alternative and adjunctive management.

Epidemiology and Types of Heart Failure

There are about 6 million cases of ongoing heart failure in the US.[1,2] Additionally, we know that the prevalence of left ventricular systolic (pumping) dysfunction in men and women over 50 years of age may be as high as 14%.[38,39] Even with normal pumping function, 5% to 6% of the population may have moderate to severe diastolic (filling) dysfunction.[40]

There are many ways to classify heart failure: systolic versus diastolic, forward versus backward, right-sided versus left-sided, and so on. Regardless of etiology, classification, and duration, stable chronic heart failure is a condition that benefits substantially from alternative medicine interventions. While we could certainly spend an entire weeklong seminar discussing all of the available alternative medicine treatments for heart failure and their bases of evidence, we will confine our discussion here to the six most reliable, important, and evidence-based. We do not have the space necessary to discuss all of the evidence, so we will discuss the largest and most important pieces of evidence and hopefully pick up the discussion in greater depth another time.

Conventional Management of Chronic Stable Heart Failure

Before we discuss the management of heart failure by alternative methods, it is important to make at least a brief mention of the conventional management of heart failure. It is important to understand the goals and management strategies of conventional care when providing adjunct care, as well as to be familiar with the conventional therapeutics that are most commonly used in that management.

Beta Blockers, ACE Inhibitors, and Angiotensin Blockers

Conventional management of chronic heart failure will, at minimum, seek to control the workload placed on the heart and to control the fluid burden on the body resulting from the decreased cardiac work. Angiotensin converting enzyme inhibitor (ACEI), angiotensin II receptor blocker (ARB), and beta blocker (BB) drugs are commonly used to decrease the cardiac workload.[41]

Beta blocker drugs primarily decrease the amount of filling work (preload) that the heart must do and protect the heart from adrenergic stimulus. Carvedilol is a special type of beta blocker that has particular application in heart failure. It is a beta-1 adrenergic receptor blocker, but also acts on alpha-1 adrenergic receptors on the muscular layer of the arteries, causing them to dilate and therefore lessening the pumping work of the heart (afterload) as well as the filling work.[41] Clinical trials of carvedilol have shown its use to decrease hospitalizations and mortality resulting from heart failure.

ACEI and ARB drugs decrease both pumping work (afterload) and filling work (preload). By acting on the renin-angiotensin-aldosterone portion of the kidneys and vascular system, these drugs decrease the amount of total peripheral resistance in the vascular system (afterload) and help to decrease venous return to the heart (preload).[41]

ACE inhibitors and beta blockers (more so than ARBs) also carry an

important side benefit: they participate in the ventricular remodeling process. Over a period of dysfunction, the heart structure becomes more and more deranged. That derangement further exacerbates heart dysfunction. Both ACEIs and BBs help the heart prevent deranged remodeling and can even facilitate healthy remodeling. The two used in combination seems to convey benefits that exceed the use of either alone and may even result in the recovery of function over time. There are natural substances that also participate in healthy ventricular remodeling, and we will discuss them.[42-45]

Diuretics

Loop diuretics are the drugs most commonly used in chronic heart failure to control fluid overload, though you may see them used in combination with potassium-sparing diuretics and occasionally alongside thiazide diuretics. Furosemide/Lasix is the loop diuretic drug typically encountered in heart failure patients. In the process of unloading fluid from the body by increasing and maintaining urine output, loop diuretics also cause the wasting of many important nutrients into the urine. It is important to understand the types of diuretics used in a heart failure patient because it guides the rational use of other natural therapeutics.[41]

Naturopathic Medicine in the Management of Heart Failure

The six natural treatments for chronic heart failure which are the most reliable, demonstrate the best evidence, and which we use to greatest effect in our practice are: hawthorn (*Crataegus spp*.), coenzyme Q10, L-carnitine, taurine, omega-3 polyunsaturated fatty acids (PUFAs), and a combination of macro- and micronutrients including calcium, magnesium, potassium, vitamins B1, B2, and B6, selenium, and zinc.

Throughout our discussion of these treatments, we will highlight some of the best available research and evidence on each, but that undertaking is neither complete nor is this article intended to be a systematic review of the evidence on these medications. Several recent reviews of limited scope are available in the conventional medical literature on "CAM" therapies in heart failure used singly or in combinations. A full review of the present evidence for CAM therapies in heart failure would certainly be a worthwhile future undertaking. ➤

Table 2: Evidence-Based Naturopathic Treatments for Cardiomyopathy and Heart Failure

Naturopathic Treatment	Dose	Notes & Cautions
Hawthorn (*Crataegus spp*.)	Solid extract, 4:1 concentration, ¼–½ tsp. (200–1500 mg) b.i.d.–t.i.d., p.o.	Caution in high output heart failure and with several medications, including nitroglycerine and digitalis.
Coenzyme Q10	100–600 mg/day, daily or divided doses, p.o.	Evidence not yet conclusive on ubiquinol over ubiquinone. Interactions with warfarin and beta blockers.
L-carnitine	1–4 grams/day, p.o., divided doses	Interacts with warfarin and thyroid hormone. May increase seizure activity.
Taurine	500 mg three times daily, p.o.	Positive inotrope. Interacts with lithium. May exacerbate bipolar disorder.
Omega-3 polyunsaturated fatty acids	2 grams or more daily, p.o. At high doses consider 1–2 grams of DHA content.	Contraindicated in bleeding disorders, with immune suppressants and with some anticoagulants. Caution advised with concomitant use of many drugs, herbs, nutrients.
Thiamine, riboflavin, pyridoxine	10–100 mg/day, p.o.	Consider a B complex supplement to ensure B vitamin balance.
Calcium	250–1000 mg/day, p.o.	Consider in balance with magnesium as a naturally occurring calcium channel blocker. Evidence is mixed. If taking iron, take calcium at a separate meal.
Magnesium glycinate, gluconate, or amino acid chelate	250–800 mg/day, p.o.	Consider a 1:1 rather than a 2:1 cal/mag ratio. Consider divided doses to enhance calcium channel blocking effects of magnesium. Watch for laxative effect.
Potassium chloride or citrate	2–20 mEq/day, p.o. For potassium, 1 mEq = 39 mg	Scrupulous potassium monitoring required. Monitor sodium, magnesium, and all electrolytes.
Zinc	10–50 mg/day, p.o.	Supplement 1 mg copper for every 15 mg zinc.
Selenium	200–800 mcg/day, p.o.	Signs of toxicity include brittle hair and nails, metallic taste in the mouth, purple skin pigment, and others.

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*Hawthorn (*Crataegus spp.*)*

The evidence for the use of hawthorn (*Crataegus spp.*) in the treatment of heart failure is some of the best evidence for any treatment in "alternative" medicine. The *Cochrane Database of Systematic Reviews* is widely regarded as the bastion of evidence-based medicine. Rarely is there sufficient evidence around the use of any particular natural therapy for Cochrane to issue a review of it at all, let alone a positive review. However, a 2008 *Cochrane Review* concluded, "…[T]here is a significant benefit in symptom control and physiologic outcomes from hawthorn extract as an adjunctive treatment for chronic heart failure." Studies on the use of hawthorn in heart failure have revealed that maximum work load, exercise tolerance, and cardiac oxygen consumption all significantly improved. Significant symptomatic improvements in shortness of breath and fatigue were also evident.[46]

In our practice, we typically use a certified organic hawthorn solid extract product which has a minimum herb strength ratio of 4:1. Several vendors make a good product and we have used them interchangeably without problem. However, some vendors assay their hawthorn for procyanidin or flavonoid content. These constituents may be reduced in the heating process of creating a solid extraction. High procyanidin content may enhance efficacy. We typically dose ¼ to ½ teaspoon, 2 or 3 times daily; see Table 2 (p. 61) for further guidelines.[47]

Consider treatments other than hawthorn in high-output cardiac failure states such as atrial fibrillation, anemia, and hyperthyroid, as hawthorn increases cardiac output and can therefore exacerbate these types of failure. Hawthorn interacts to some degree with many common medications, especially cardiac medications such as nitroglycerine and digoxin, so be sure to double-check your patient's medications and your recommendations for potential interactions.[48]

Coenzyme Q10

Coenzyme Q10 (CoQ10) is one of the most important natural medications available for the treatment of heart failure. The published evidence on its use in systolic dysfunction has been routinely impressive. A 2006 systematic review published in the *Journal of Cardiac Failure* concluded: "CoQ10 enhances systolic function in chronic heart failure, but its effectiveness may be reduced with concomitant use of current standard therapies."

In this study, a significant improvement in left ventricular ejection fraction was demonstrated to be 6.7% in those *not* on ACE-inhibitors (ACEI) and 3.4% for those on ACEIs, with use of CoQ10 60 to 200 mg/day. A trend toward increased cardiac output was also demonstrated.[49]

Data from various small trials on CoQ10 have consistently shown inverse correlations between heart failure severity and CoQ10 concentrations in the blood plasma and myocytes.[50–52] A 2009 study from New Zealand demonstrated that plasma CoQ10 levels may be an independent predictor of survival in heart failure.[53]

There is also evidence for the importance of CoQ10 in diastolic dysfunction. A small study (n = 46) of hypertrophic cardiomyopathy patients using CoQ10 at 200 mg/day to improve diastolic dysfunction demonstrated significant improvements in New York Heart Association (NYHA) failure class, quality of life, and six-minute walk test. That research also demonstrated improved septal wall thickness, which decreased by 22% ($p < 0.005$), and posterior wall thickness, which decreased by 23% ($p < 0.005$).[54]

We typically use 100 to 600 mg/day of CoQ10 in the treatment of heart failure. Available data show that CoQ10 is extremely safe, even at doses over 2 grams daily, and it is extremely well tolerated.[55] CoQ10 has few drug interactions, but warfarin and beta blockers are among them, so always double-check and monitor for potential interactions.[56]

L-Carnitine

Carnitine is an amino acid responsible for facilitating the transfer of fatty acids into the mitochondria for energy production. Randomized trials of carnitine in heart failure have shown evidence of improved diastolic parameters.

A randomized, controlled trial (RCT) of carnitine in post–heart attack patients demonstrated attenuation of left ventricular dilatation similar to the effect expected for ACEIs and beta blockers (the ventricular remodeling effect).[57] A significant mortality benefit was demonstrated in NYHA class II, IV heart failure patients with lower rates of death from all causes in patients on carnitine at a dose of 2 grams daily.[58] Other studies of carnitine in heart failure have shown significant improvements in exercise capacity, maximum exercise time, peak heart rate, peak oxygen consumption, and improved hemodynamic and echocardiographic parameters.[59,60]

In heart failure, we use L-carnitine at 1 to 4 grams/day, orally, in divided doses. L-carnitine inhibits peripheral thyroid hormone conversion (T4 to T3) and is therefore indicated in hyperthyroid conditions and relatively contraindicated in hypothyroid conditions.[61]

L-carnitine can cause an increase in seizure activity and is therefore contraindicated in seizure disorders. L-carnitine does have drug interactions with warfarin, acenocoumarol/Sintrom, and thyroid hormone.[61]

Taurine

Taurine is a "semiessential" amino acid. It comprises up to ¼ of the amino acid pool in the heart, it functions as an antioxidant, and it regulates calcium homeostasis.

Small RCTs have shown taurine at doses of 500 mg three times daily to improve exercise capacity and systolic function as well as decrease left ventricle end-diastolic pressures (diastolic dysfunction) in heart failure.[62–64]

We dose taurine 500 mg, three times/day, but 2 to 6 grams per day can be used safely. Taurine is extremely well tolerated. Its most notable drug interaction is with lithium and it may exacerbate bipolar disorder.[65]

Omega-3 Polyunsaturated Fatty Acids

A recent editorial published in the *Lancet* concluded: "Supplementation with omega-3 PUFAs [polyunsaturated fatty acids] should join the short list of evidence-based life-prolonging therapies for HF [heart failure]."[66]

GISSI-HF, a large Italian study of 7000 HF patients taking 1 gram/daily of n-3 (omega 3) PUFAs demonstrated a significant reduction in all-cause mortality and reduced hospital admissions. The authors of this study and others have suggested that higher doses than those used in the study are likely necessary to obtain maximum benefit from PUFAs.[67]

In the Atherosclerosis Risk in Community (ARIC) study, a significant inverse relationship between n-3 PUFA intake and incidence of heart failure in women was demonstrated.[68] A small study (n = 18) of HF patients using 5.1 grams/day of EPA and DHA demonstrated decreased levels of the inflammatory cytokines IL-1 and TNF-alpha.[69]

In general, the clinical benefits of n-3 fatty acids in HF are small, but significant. PUFAs are contraindicated in bleeding disorders, and in combination with some anticoagulants and immunosuppressants; caution is advised when they are used in combination with many drugs, herbs, and nutrients.[70] Most of our patients are on at least 2 g/day of omega 3 PUFAs; dosing regimens are typically dictated by numerous factors in any individual case.

Macro- and Micronutrients

The need for multiple vitamin and mineral supplementations in the setting of heart failure has been illustrated in many small studies. The evidence and appreciation for the importance of micronutrient assessment and therapies in acute and chronic heart failure continues to grow. Discussion of this topic is occurring within the conventional medical literature, and it represents an area of great promise for the treatment of patients and for the contributions of natural medicine to the practice of conventional medicine.

Vitamin B1 (Thiamine). The prevalence of deficiency in HF patients may be 13% to 33% and loop diuretics cause thiamine wasting. Small studies have demonstrated modest improvements in ejection fraction (EF) after supplementation. Thiamine deficiency itself can cause of heart failure.[71-74]

Vitamins B2 (Riboflavin) and B6 (Pyridoxine). These vitamins are also wasted by loop diuretics. Small studies have shown deficiencies in up to 27% and 38% of HF patients, respectively.[75]

Most of our patients are on a sublingual B complex that provides thiamine 13 mg, riboflavin 10 mg, pyridoxine 10mg.

Calcium, Magnesium, and Potassium. Calcium (Ca), potassium (K), and magnesium (Mg) are depleted by loop diuretics. Deficiencies in Ca and Mg contribute to a state of secondary hyperparathyroidism, which exacerbates heart failure by slowing renal filtration and increasing fluid retention. Increased fluid retention leads to the use of increased amounts of diuretics, perpetuating a vicious cycle.

In one small study, oral supplementation with a multivitamin mineral complex containing 250 mg Ca, and 150 mg Mg (as well as others) demonstrated significant improvements in EF, end-systolic, and end-diastolic volumes and quality of life.[76]

Serum potassium concentrations are affected more or less (either wasting or sparing) by all diuretics (botanical and pharmaceutical). ACEIs, ARBs, and other drugs commonly used in this population also affect serum potassium concentrations. We test levels frequently, as often as weekly when adjusting acute treatments and not less than every 8 to 12 weeks even in medically stationary cases. We typically use Klor-Con (generic prescription potassium chloride) as our form of potassium supplementation. It is on most of the $4 drug formularies ($4/month). We typically use doses of 10 to 20 mEq/day, orally.

Selenium and Zinc. Selenium (Se) and zinc (Zn) levels have been noted as low in substantial percentages of cardiomyopathy patients. Low levels contribute to the depletion or dysfunction of endogenous antioxidant enzymes. HF is now appreciated as a pro-inflammatory state that puts large demands on endogenous antioxidant systems. Zinc is wasted by loop diuretics and ACE inhibitors. In the same study mentioned above, Zn and Se were supplemented at 15 mg and 800 mcg/day, respectively.[76]

Conclusion

In our discussion we have only scratched the surface of several topics, including infectious heart disease, cardiomyopathy, heart failure, and the evidence-based natural treatment of heart failure. We could devote a textbook chapter, or in some cases an entire text, to any one of these topics. The depth, breadth, and import of this information and its bearing on clinical practice illustrate the need for the continuing postgraduate education and training of alternative medical practitioners, particularly naturopathic physicians.

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The Case for Postgraduate Training in Preventive and Integrative Cardiology

Mastering the vast amount of information which is left to naturopathic medical students in only four years of medical school is a formidable task. In the modern era, naturopathic physicians in training are called upon to master at least two systems of medicine: naturopathic medicine and conventional primary care medicine.

To truly understand the size of that task, we must understand several other facts:

1. Medical doctors master a single system of medicine over a total of seven years, (four years of medical school followed by three years of postgraduate clinical training).
2. Naturopathic doctors trained at colleges accredited by the CNME are trained in both naturopathic medicine and general/family practice conventional medicine, including the conventional therapeutics of pharmacology and minor surgery.
3. Naturopathic medicine could be viewed as more than one system of medicine itself as it comprises doctoral-level mastery of clinical nutrition, physical medicine, counseling and mind-body medicine, botanical medicine, and homeopathy. Doctoral-level training programs are available in each of these stand-alone disciplines.
4. Many naturopathic medical students are voluntarily attempting to master at least one other medical discipline simultaneously with naturopathic and conventional medicine. Most often, those additional disciplines include one or more of: Chinese medicine, natural obstetrics and midwifery, and medical research.
5. Naturopathic doctors licensed to practice medicine are not yet required to complete residency; therefore, most complete this vast training in only four years. There are not yet enough residency positions available to fill the educational need. Thus students complete almost twice the education in a little over half of the time that medical doctors take to master a single system of medicine.

While many naturopathic medical students choose to complete their curriculum in five years, or more if they have chosen to study multiple disciplines, the overall programmatic timeline is still exceptionally short given the amount of material to be mastered.

Several other considerations inform our point of view regarding the necessity of postgraduate training of licensed naturopathic physicians. For instance, the number and variety of patient conditions treated and managed by naturopathic interns vary widely depending on the rotations that they complete, the settings in which those rotations occur, the types and numbers of preceptors whom an intern has, and where they accumulate their external rotations with those preceptors.

We are extremely proud of and pleased by the robust training completed by licensed naturopathic physicians by the time of their graduation. We are impressed by the ability of each individual to master the mountain of material with which they are presented and in such a short period of time. We are routinely in awe of the high quality of the physicians who leave our training programs. Yet, it can not be denied that in certain areas of medicine the majority find themselves routinely underprepared for the demands of clinical practice.

Naturopathic Cardiology and Its Opportunities

A handful of naturopathic physicians in this country relish and excel in the treatment of cardiovascular conditions. Unfortunately, most naturopathic physicians do not share that confidence and excitement; rather, they feel out of their depth when dealing with the heart. With such wide opportunities to influence the health of the nation, cardiovascular medicine is an area where naturopathic physicians need to excel. There is an opportunity here to promote our profession and to help our conventional colleagues become aware of and enthusiastic about the ability of naturopathic medicine to fill an important niche within the greater medical field. The steps forward in capitalizing on those opportunities are to increase the postgraduate education and training of naturopathic physicians in cardiovascular medicine and other special disciplines and to establish professional organizations to create and uphold standards of practice and training in areas of naturopathic practice focus. A professional board of naturopathic cardiovascular medicine will achieve both of these necessary advances in our profession; it is an idea whose time has come.

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These authors have no financial conflicts of interest to declare.

Notes

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