Does Aerobic Exercise have a Role in the Treatment Plan of a Patient with Heart Failure

R Andreuzzi

Citation

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Abstract

Heart failure (HF) prevention is an urgent public health need with coronary artery disease, hypertension, dilated cardiomyopathy, and a growing elderly population increasing the prevalence of HF. Management of HF involves multiple medications, lifestyle modification, and control of risk factors. Aerobic exercise (AE) has become more accepted in HF treatment as information regarding its safety and benefits has emerged. AE enhances peak VO2, improves muscle oxygen utilization, increases exercise duration, reduces sympathetic and increases vagal tone at rest, improves endothelial function, reduces plasma levels of proinflammatory cytokines, improves symptoms of dyspnea and fatigue, and improves quality of life in persons with HF. As a universal exercise prescription for HF patients does not exist, an individualized approach is recommended allowing for adjustments in the exercise program as necessary. Though there is need for further study, AE is more accepted and utilized in the treatment of persons with HF and should be viewed as a complement to rather than replacement of the lifestyle modification, pharmacological therapy, and physician follow-up.

INTRODUCTION

The prevention of heart failure (HF) has emerged as an urgent public health need with national and global implications with an estimated 550,000 new cases diagnosed each year and over 5 million Americans with HF (1). HF is a lethal condition that has emerged as a leading cause of hospitalizations with 6.5 million hospital days each year, over $33 billion spent on its treatment in 2007, and an estimated annual mortality of 21% in men and 17% in women (1,2). One factor increasing the prevalence and incidence of HF is the growth in the elderly population (>65 years of age) which is expected to grow to 70.3 million in 2030 (1). The management of HF typically involves multiple medications, lifestyle modification, and control of risk factors that increase the prevalence or worsen the course of HF.

In the past, patients with HF were advised to avoid physical exertion in the belief that resting would minimize symptoms and exertion would accelerate the progression of left ventricular dysfunction (2). As information has become available regarding the safety and benefits of aerobic exercise (AE) in persons with HF, AE has become more widely accepted as a valuable component in the treatment of persons with HF (2,3). AE has been shown to enhance peak VO2, improve muscle oxygen utilization, increase exercise duration, reduce sympathetic and increase vagal tone at rest, improve endothelial function, reduce plasma levels of proinflammatory cytokines, improve symptoms of dyspnea and fatigue, and improve quality of life (QOL) in persons with HF (2,3). This EBM paper will evaluate if aerobic exercise has a role in the treatment of a patient with heart failure.

BACKGROUND

HF is a complex syndrome that may result from any structural or functional cardiac disorder that impairs the ability of the ventricle to fill with or eject blood with the cardinal symptoms being fatigue and shortness of breath. HF can also manifest as limited exercise capacity or fluid retention that may lead to pulmonary congestion, peripheral edema, and shortened life expectancy. HF was thought to arise as a result of depressed left ventricular ejection fraction (EF); however, studies have shown that up to half of patients with HF have a normal or preserved EF (4). Therefore, patients with HF may be categorized as having either systolic heart failure (a depressed ejection fraction) or diastolic heart failure (a preserved ejection fraction).

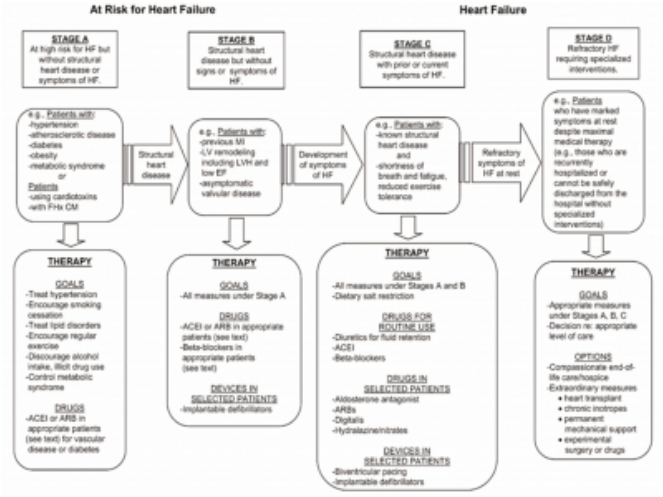
Left ventricular dysfunction is a progressive process that

begins with an injury or stress to the myocardium resulting in a change in the geometry and structure of the left ventricle causing the chamber to dilate and/or hypertrophy and become more spherical. Patients with left ventricular dysfunction may remain asymptomatic for months or years due to several compensatory mechanisms being able to sustain or modulate left ventricular function. The compensatory mechanisms include: activation of the renin-angiotensin-aldosterone and adrenergic systems which maintain cardiac output through increased retention of water and salt; increased myocardial contractility; and vasodilatory molecules that offset the peripheral vascular vasoconstriction including atrial and brain natriuretic peptides, prostaglandins, and nitric oxide.

Patients with HF are typically classified according to the New York Heart Association functional classification as class I to IV (2). Class I is asymptomatic; class II are considered symptomatic with ordinary physical activity; class III are symptomatic with less than ordinary activity; and class IV are symptomatic at rest. This classification system has several limitations in that it primarily gauges the severity of symptoms in patients, is highly subjective, and patient symptoms may vary from day to day. The American College of Cardiology (ACC) and the American Heart Association (AHA) have developed a new approach to the classification of HF emphasizing the development and progression of the disease, with the new classification system designed to complement but not replace the New York Heart Association functional classification (2). Figure 1 shows the new ACC/AHA classification system for HF.

Figure 1

Figure 1. ACC/AHA Classification System for Heart Failure (2)

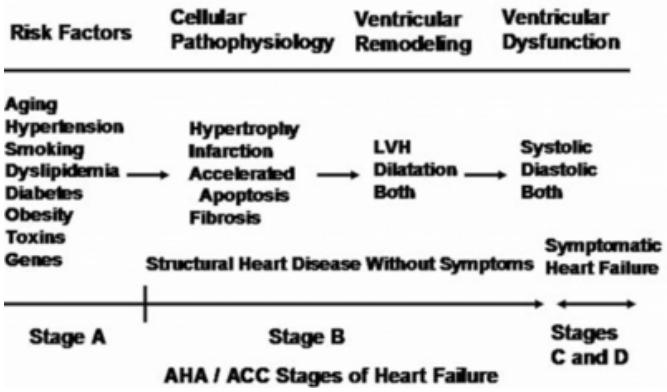


The new ACC/AHA classification system recognizes that HF has established risk factors and structural prerequisites, that HF has symptomatic and asymptomatic phases, and specific treatments at each stage can reduce morbidity and mortality. Figure 2 shows the evolution of HF along with the new ACC/AHA classification system for the diagnosis and management of HF clinical stages.

The new ACC/AHA classification system recognizes that HF has established risk factors and structural prerequisites, that HF has symptomatic and asymptomatic phases, and specific treatments at each stage can reduce morbidity and mortality. Figure 2 shows the evolution of HF along with the new ACC/AHA classification system for the diagnosis and management of HF clinical stages.

Figure 2

Figure 2. Evolution of Heart Failure With the New ACC/AHA Classification System for the Diagnosis and Management of HF Clinical Stages (1)



Most patients with HF are managed with a combination of 3 types of drugs: a diuretic, an angiotensin converting enzyme inhibitor (ACE) or an angiotensin receptor blocker (ARB), and/or a beta-blocker. Patients with evidence of fluid retention should take a diuretic until a euvolemic state is achieved with the diuretic therapy continued to prevent the recurrence of fluid retention. An ACE inhibitor and a beta-blocker should be initiated and maintained, even if there is a favorable response to the diuretic, as these drugs have been shown to favorably influence the long-term prognosis of HF. In addition, digoxin may be initiated at any time to reduce symptoms, prevent hospitalization, control rhythm, and enhance exercise tolerance.

In addition to the pharmacological therapy, lifestyle changes including diet and sodium restriction with daily weight (to allow for adjustment of the diuretic dose as necessary) and participation in an AE training program (because restriction

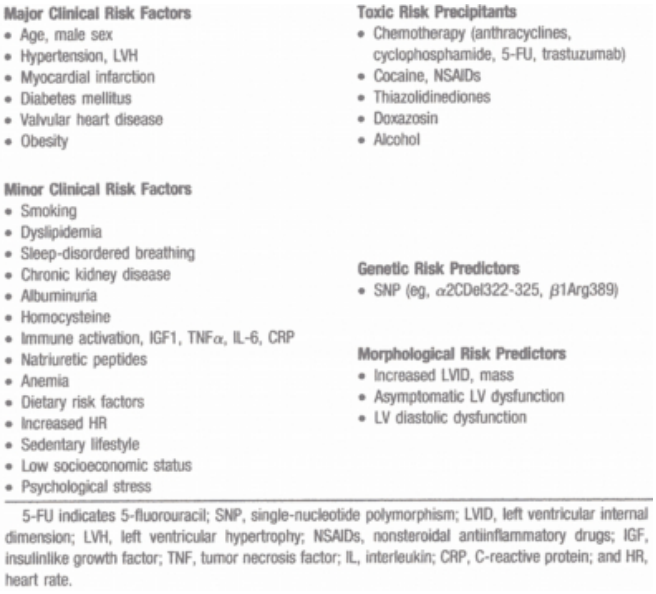
of activity promotes physical deconditioning which affects clinical status and contributes to the exercise intolerance) should be utilized in patients with HF. Possibly the most effective, yet least utilized in the treatment of patients with HF, is close attention and follow-up as nonadherence with diet and medications can affect the patient's clinical status with increases in body weight and development of symptoms leading to major clinical episodes requiring emergency care or hospitalization.

There are 3 classes of drugs that should be avoided in most patients with HF as they can exacerbate the HF syndrome: antiarrhythmic agents, calcium channel blockers, and nonsteroidal anti-inflammatories (2). Antiarrhythmic agents can cause cardiodepressant and proarrhythmic effects. Calcium channel blockers can lead to worsening HF and have been associated with an increased risk of cardiovascular events. Nonsteroidal anti-inflammatory drugs can cause sodium retention and peripheral vasoconstriction and can attenuate the efficacy and enhance the toxicity of diuretics and ACE inhibitors.

Identifying and preventing the risk factors that lead to the development or progression of this debilitating and potentially fatal syndrome should be paramount among the approaches to prevent HF. Coronary artery disease (CAD), hypertension (HTN), and dilated cardiomyopathy (DCM) are leading causes of HF in a large number of patients. Other clinical risk factors that may contribute to the development or progression of HF include aging, smoking, dyslipidemia, diabetes, obesity, toxins, and genetics. Established and hypothesized risk factors for the development of HF are listed in Table 1.

Table 1. Established and Hypothesized Risk Factors for the Development Heart Failure (1)

Figure 3



5-FU indicates 5-fluorouracil; SNP, single-nucleotide polymorphism; LVID, left ventricular internal dimension; LVH, left ventricular hypertrophy; NSAIDs, nonsteroidal antiinflammatory drugs; IGF, insulinlike growth factor; TNF, tumor necrosis factor; IL, interleukin; CRP, C-reactive protein; and HR, heart rate.

The prevention of CAD is key to maintaining functional myocytes and preventing left ventricular dysfunction with ischemic heart disease being the leading cause of HF in Western countries (1). The contribution of CAD to HF is not limited to the initial ischemic insult as the progressive nature of CAD contributes to recurrent cardiovascular events, sudden death, and the progression to HF. In patients having a myocardial infarction (MI), there is a decrease in the functional myocyte reserve, myocardial fibrosis, and development of left ventricular remodeling that results in chamber dilation and neurohormonal activation causing a progressive deterioration of the remaining myocardium resulting in a 2- to 3-fold increased risk of developing HF (1). In addition, the chronic ischemia seen in patients with CAD superimposed on a damaged myocardium may result in "hibernation" causing further decline in ventricular function (1). ACE inhibitors, beta-blockers, antiplatelet agents, statin therapy, and lifestyle changes including diet and exercise can be used in patients with CAD to help prevent the progression to symptomatic HF.

The prevention and control of HTN provides the earliest opportunity to prevent HF as HTN increases the risk of developing structural heart disease and HF due to myocyte hypertrophy (secondary to increased afterload), myocardial fibrosis (increased collagen synthesis and decreased degradation), and loss of myocardial contractile tissue (through increased incidence of MI), all of which lead to a reduction in functional myocyte reserve resulting in a 2- to 3-fold increased risk of developing HF (1). Elevated levels of diastolic and especially systolic blood pressure are major

risk factors for the development of HF, and long-term treatment of both systolic and diastolic HTN has been shown to reduce the risk of developing HF (2). The typical blood pressure response to AE is a gradual increase in systolic blood pressure (SBP) and a decrease in diastolic blood pressure (DBP) with expected peak AE SBP of 180-210 mmHg and DBP of 60-85 mmHg (5). There are several possible mechanisms by which AE may lower blood pressure including lowering both cardiac output and peripheral vascular resistance at rest and any given level of work, reducing serum catecholamines, reducing plasma renin activity, reducing plasma norepinephrine, and decreasing heart rate from increased vagal tone (5). ACE inhibitors, beta-blockers, diuretics, and lifestyle changes including diet and exercise can be used to prevent the development or progression of HTN helping to prevent the development of HF.

Although no cause for its development is apparent in many cases, DCM is either familial or the end result of myocardial damage from a variety of infectious, metabolic, or toxic agents (6). Up to one-third of patients' with DCM have familial forms with mutations in genes that are transmitted in an autosomal dominant fashion with the most common mutations being in genes that encode for sarcomeric proteins resulting in contractile dysfunction by impairing the production and/or transmission of force (6). Most patients' who develop DCM have a downhill course with the majority, especially those >55 years of age, dieing within 4 years of the onset of symptoms due to either progressive HF or ventricular tachy- or bradyarrhythmia (6). Treatment of the patient with DCM should include ACE inhibitors, beta-blockers, ARBs, diuretics, lifestyle changes including diet and exercise, chronic anticoagulation due to increased risk of systemic embolization, cardiac resynchronization therapy with insertion of an implantable cardioverter defibrillator, and in refractory cases, cardiac transplantation, with the avoidance of alcohol, calcium channel blockers, nonsteroidal anti-inflammatories and antiarrhythmic agents.

Diabetes is an independent risk factor for the development of HF and may act synergistically to increase the risk of HF 2- to 5-fold by accelerating the development of atherosclerosis, myocardial infarction, and ischemic HF (1). Diabetes may predispose to HF by promoting atherogenic risk traits, obesity, left ventricular hypertrophy, disease of the coronary microvasculature, endothelial dysfunction, autonomic dysfunction, and metabolic abnormalities (1). Therefore, strategies aimed at preventing or delaying the onset of type 2 diabetes would be an effective means to reduce the incidence of HF (1). AE in type I diabetes results in improvements in insulin sensitivity, glucose metabolism, and CAD risk factors while AE in type II diabetes results in improved insulin sensitivity, increased insulin receptor affinity, improved daily blood glucose control, and a decreased glycosylated hemoglobin (7). Glucose lowering drugs, lifestyle changes including diet and exercise, can be used to prevent or delay the onset of type 2 diabetes with ACE inhibitors and beta-blockers exerting a cardioprotective effect in the diabetic patient with HTN.

Dyslipidemia is an independent risk factor for CAD and is linked to the development of HF. Although elevated total cholesterol is not a strong predictor of new-onset HF, an increased ratio of total cholesterol to high-density lipoprotein cholesterol is associated with an elevated HF risk (1). Dyslipidemia may predispose to HF as high cholesterol levels, low high-density lipoprotein cholesterol levels, and high triglyceride levels correlate with greater left ventricular mass and impaired diastolic function especially in hypertensive patients (1). AE training (of sufficient intensity, duration, and longevity) will affect lipoprotein metabolism resulting in decreased triglycerides and increased high-density lipoprotein cholesterol; however, the decrease in total and low-density lipoprotein cholesterol seen with AE is less consistent and may be better linked to weight loss (8). Untested in terms of its impact on the incidence of HF, it is reasonable to hypothesize that dietary and lipid-lowering strategies will prevent HF with an emphasis on both intensity of therapy and the need for adherence (1).

Obesity is associated with an increased risk for CAD with excess body weight being an independent risk factor for the development of HF and contributing to other HF risk factors including HTN, dyslipidemia, and type 2 diabetes (1). Obesity may predispose to HF by contributing to atherogenic risk factors and increasing preload and afterload, as well as through neurohormonal upregulation (by natriuretic peptide inadequacy), and an association with sleep-disordered breathing or chronic kidney disease (1). Lifestyle changes with modest weight loss through reduced caloric intake and increased physical activity can be used to prevent the development or progression of obesity and its CAD related risk factors helping to prevent the development of HF.

Tobacco use is the single largest preventable cause of disease and premature death with approximately 440,000 Americans dieing each year of smoking related illness with

smoking independently associated with a 47% increased risk of developing HF (1). Tobacco use may predispose to HF by promoting insulin resistance, dyslipidemia, diabetes, endothelial dysfunction, coronary vasospasm, oxidative stress, and may induce direct toxic effects on myocytes (1). Patients should be questioned about their tobacco use with active smokers counseled to quit with referral to a formal cessation program and/or pharmacological therapy to increase success rates as those who quit smoking have a 50% lower risk of death from CAD compared to those who continue to smoke (1).

Several other substances classified as toxic risk precipitants have been shown to increase the risk of developing HF. Excessive alcohol intake may increase the risk of HF by 45% by increasing blood pressure or direct myocardial toxicity while the use of cocaine may precipitate acute MI and subsequent HF (1). Chemotherapeutic agents such as doxorubicin, cyclophosphamide, and 5-fluorouracil are associated with myocardial damage resulting in left ventricular dysfunction, HF, and death. In diabetic patients using insulin sensitizers, troglitazone which has been withdrawn from the market due to issues regarding precipitation of HF and the thiazolinedione agents (Avandia and Actos) may increase the incidence of HF by 50 % (1).

Less thought of causes for the development of HF include sleep-disordered breathing and chronic kidney disease. Obstructive sleep apnea is associated with a 2.4-fold relative risk of HF independent of other known risk factors (1). Although there is no direct evidence that treating obstructive sleep apnea prevents incident HF, treatment of established left ventricular dysfunction with continuous positive airway pressure has been shown to improve left ventricular structure and function in patients with either obstructive or central sleep apnea syndrome (1). Activation of the renin-angiotensin-aldosterone system and sympathetic nervous system plays an important pathophysiological role in the initiation and progression of both renal disease and HF. Renal insufficiency increases the risk of HF shown by increasing levels of serum creatinine with even mild insufficiency associated with a progression of asymptomatic left ventricular systolic dysfunction to overt HF (1). Chronic renal insufficiency may predispose to HF by promoting anemia (erythropoietin deficiency), worsening of HTN, arterial stiffening, hypervolemia (water and sodium retention), neuroendocrine activation, hypercoagulability, endothelial dysfunction, increased proinflammatory cytokines, and increased homocysteine levels.

Physical inactivity has been recognized as an important risk factor in the development of CAD and HF with epidemiological studies documenting a reduced incidence of CAD, stroke and HF in the more physically active and fit individuals (1). Exercise intolerance is the reduced ability to perform activities that involve dynamic movements of large skeletal muscles because of symptoms of dyspnea or fatigue making the inability to perform exercise without discomfort one of the first symptoms experienced by patients with HF inextricably linking exercise intolerance to the HF diagnosis (3).

It is now understood that reductions in physical activity (either by the symptoms of HF or by the physician treating HF) leads to a state of physical deconditioning that contributes to the symptoms and exercise intolerance seen in patients with HF. Limitations of activity not only impair exercise capacity but may produce adverse psychological effects and impair peripheral vasodilatory responses leading to the hypothesis that AE training might improve the clinical status of patients with HF (2).

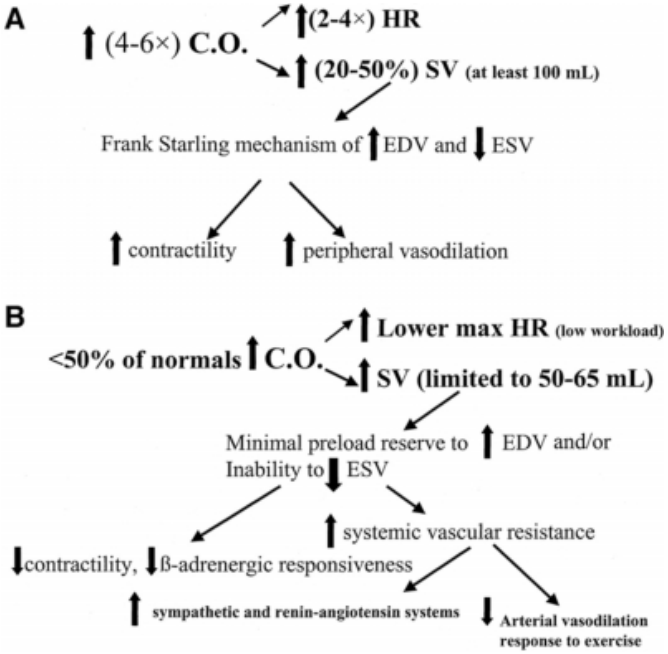
Several studies have shown that AE training can lessen symptoms, increase exercise capacity, and improve the QOL in patients with HF (2). These improvements were comparable to those achieved with pharmacological interventions, and were associated with an enhancement of endothelium-dependent peripheral vasodilation and skeletal muscle metabolism (2).

The capacity for performing AE depends on the ability of the heart to augment its output to the exercising muscles and the ability of these muscles to utilize the oxygen from the delivered blood. The increase in cardiac output during AE in healthy patients is typically 4- to 6-fold being accomplished by a 2- to 4-fold increase in heart rate and a 20% to 50% augmentation of stroke volume while cardiovascular disease, age, sex, and conditioning status may affect the ability of cardiac output to increase during AE (3). A hallmark of HF is a reduced ability to perform AE mediated by inadequate blood flow to active skeletal muscles secondary to impaired cardiac output, achieving <50% of the maximal cardiac output attained by healthy individuals, and a modest rise in stroke volume to a peak of 50 to 65 mL compared to >100 mL in healthy individuals (3). Failure to increase left ventricular systolic emptying and thus augment left ventricular ejection fraction, comes from impaired intrinsic contractility, reduced B-adrenergic responsiveness, elevated systemic vascular resistance due to increased activity of the sympathetic and renin-angiotensin systems, and a blunted

peripheral arterial vasodilator response to AE (3). In patients with CAD, stroke volume may fall during AE if myocardial ischemia develops due to excessive myocardial oxygen demand making cardioacceleration the primary means to augment cardiac output in patients with HF (3). Figure 3 shows the mechanisms that augment cardiac output in patients with and without HF.

Figure 4

Figure 3. Mechanisms that Augment Cardiac Output in Patients With (B) and Without (A) HF (3).



Because abnormalities in central hemodynamic function are not sufficient in themselves, multiple peripheral factors are used to help explain the exercise intolerance seen in patients with HF including abnormalities of endothelial function, ergoreflex activation, vasodilatory capacity, and distribution of cardiac output. The failure of muscle blood flow to increase normally during AE in patients with HF is due not just to a reduction in cardiac output but to abnormalities in peripheral vasodilation due to excessive sympathetic stimulation causing vasoconstriction, activation of the renin-angiotensin system, higher-than-normal levels of endothelin, and vascular stiffness secondary to increased vascular sodium content.

The vascular endothelium releases vasoactive substances including nitric oxide, endothelins, and prostaglandins in response to various chemical, pharmacological, mechanical, and exercise stimuli that play an important regulatory role in peripheral vasomotor tone with impaired endothelial function contributing to the reduced vasodilatory capacity seen in patients with HF. Studies evaluating the role of exercise training on endothelium-dependent vasodilation have shown that exercise training might improve flow-dependent relaxation of peripheral arteries and that this beneficial effect can translate into an increased blood flow to the skeletal muscles (3). In addition, exercise training has been shown to improve endothelial nitric oxide formation and endothelial-dependent vasodilation of the skeletal muscle vasculature (3).

Increased plasma catecholamines have been associated with a poor prognosis in patients with HF; however, the results of studies investigating if AE training causes a decrease in catecholamines have been variable (3). The variability in these findings may be related to the severity of disease, etiology and duration of the HF syndrome, intensity and duration of exercise training, and the presence of sympathetic activity-modulating drugs (ACE inhibitors or beta-blockers).

Skeletal muscle characteristics have important ramifications on substrate and oxygen utilization during AE with patients with HF having decreased oxidative type I fibers and increased glycolytic type IIb fibers causing anaerobic metabolism to occur early during AE and is a cause of exercise intolerance. Patients with HF who have compromised AE capacities have a maladaptive angiogenic response to exercise increasing capillary density before skeletal muscle changes while levels of glycolytic enzymes appear to be unchanged, levels of oxidative enzymes are decreased in patients with HF. These findings suggest that alterations in skeletal muscle may contribute to abnormal oxygen extraction or substrate delivery/utilization and may further limit exercise tolerance in HF (3).

Exercise tolerance depends not only on the capacity of the cardiopulmonary system to deliver oxygen to the working muscle but on regional flow and in patients with HF vascular resistance in the muscle fails to decrease normally during exercise while flow to the non-exercising tissues may be preferentially maintained causing hypoperfusion in the exercising muscle. Afferent fibers present in the skeletal muscle are sensitive to metabolic changes related to muscular work (ergoreceptors) and are abnormally enhanced in patients with HF causing abnormal hemodynamic, autonomic, and ventilatory responses to exercise. The result of this enhanced ergoreflex response, which may be attenuated with AE, is hyperventilation and heightened sympathetic outflow, causing an increase in peripheral resistance and a decrease in muscle perfusion.

Ventilatory symptoms in patients with HF are related to excessive increases in blood lactate during low level exercise, reductions in VO2 at peak exercise, and disproportionate increases in ventilation at submaximal and peak workloads leading to rapid and shallow breathing during exercise. The reduced ventilatory response to AE is largely through delayed blood lactate accumulation, although better ventilation/perfusion matching in the lung and attenuation of ergoreflex activation may also contribute. Studies have shown favorable effects of AE on the ventilatory abnormalities seen in patients with HF with the changes primarily through peripheral mechanisms with little or no effect on resting left ventricular function (3). In addition, respiratory muscle training has been shown to improve ventilatory muscle endurance, decrease perceived dyspnea during volitional isocapnic hyperpnea, and increase maximal exercise capacity (3).

Studies evaluating QOL in patients with HF who have participated in AE training are limited; however, the majority of the existing data support an improvement in QOL after training in these patients (3).

Patients with HF have greater overall morbidity and mortality compared to healthy individuals and those with other forms of heart disease (3). Many factors affect the risk of participating in an exercise program in patients with HF with three of the most important being age, presence of heart disease, and intensity of exercise (3). In HF patients where functional class often changes, it is difficult to determine whether worsening HF is related to the frequent variability of symptoms or to the AE program (3).

AE is recommended for patients with stable HF (who are not having signs or symptoms of active HF) and because agreement on a universal exercise prescription for patients with HF does not exist, an individualized approach is recommended (3). The most common events seen with AE participation in patients with HF are postexercise hypotension, atrial and ventricular arrhythmias, and worsening HF symptoms (3). Most studies have utilized a training frequency of 3 to 5 days per week with patients who develop exhaustion after training encouraged to take a day of rest prior to the next training session (3). Training intensity does not seem to directly influence the magnitude of the increase in exercise tolerance in patients with HF; however, it is essential that progression be built into the exercise prescription to allow for adjustment in the exercise intensity as the patient becomes better conditioned (3). The most frequently used exercise intensity is 70% to 80% of peak VO2 while 60% to 65% of peak VO2 or lower may need to be used in patients with more severe HF. Use of the heart rate as a means of determining exercise intensity in patients with HF may be inaccurate or impractical due to the chronotropic reserve being limited and the concomitant use of beta-blockers. The Borg scale is another means of prescribing exercise intensity, especially in those taking beta-blockers, with a rating of perceived exertion (RPE) of 12 to 13 being tolerated in patients with HF (3).

The type (mode) of exercise prescribed in patients with HF should include low-impact exercises such as walking, treadmill, cycling, rowing, stair climbing, and arm/leg ergometry with the avoidance of high-impact exercises to help with injury prevention and the promotion of long-term exercise compliance. The time (duration) of the exercise session will depend upon the patient but should be able to be increased over time as the patient's conditioning improves. The exercise session should consist of a warm-up, conditioning phase, and cool-down. The warm-up period should usually be 10 to 15 minutes or longer in more debilitated patients with HF; a conditioning phase duration of 20 to 30 minutes at the desired intensity is usually advised, and a cool-down period of sufficient duration dependent upon the intensity and duration of the exercise session.

When initiating an AE training program in a patient with HF, whether this should be a supervised or home based program has not been well studied. The current recommendation of the American Association for Cardiovascular and Pulmonary Rehabilitation is that the initial training sessions should be done under direct supervision with the inclusion of telemetry monitoring (3). Having the initial AE training sessions supervised allows for instruction on proper exercise technique, monitoring for arrhythmias or symptoms that may contraindicate participating in an exercise program, education to include recognition of symptoms, when/how to make adjustments in the exercise program, nutrition guidelines, the disease process, and the importance of compliance with medications, exercise, and physician follow-up. Transitioning to a home-based exercise program should follow the supervised program and will vary from patient to patient depending on their level of deconditioning and disease stability.

METHODS

This paper asked the question "Does Aerobic Exercise have a Role in the Treatment Plan of a Patient with Heart Failure"

which is a therapy and/or prevention question. This type of question is best answered by randomized double-blinded, placebo controlled trial, meta-analysis, and/or systemic review, which are all evidence level A/I. A computerized literature search for relevant studies was performed in Academic Search Premier (EBSCO), CINAHL with full text, and MEDLINE with full text databases. The following MeSH terms or textwords in various combinations were used: "heart failure" and "aerobic exercise". Several online sites were also searched while at Lehigh Valley Hospital in Allentown, Pennsylvania including www.uptodate.com, www.acc.org, and www.americanheart.org.

Non-English papers were excluded because translations were not available. The restriction to English language studies is unlikely to cause any bias, as a recent assessment reported that non-English papers are likely to be of low quality and could introduce bias into a review. The most up-to-date information was used so articles no older than 2003 were used in this paper. Only papers that involved humans and that were peer-reviewed were included.

DISCUSSION

The article entitled "Prevention of Heart Failure: A Scientific Statement from the American Heart Association Councils on Epidemiology and Prevention, Clinical Cardiology, Cardiovascular Nursing, and High Blood Pressure Research: Quality of Care and Outcomes Research Interdisciplinary Working Group; and Functional Genomics and Translational Biology Interdisciplinary Working Group" is a consensus statement of the available literature regarding the prevention of HF (1). This original statement from the American Heart Association was published in 2008. Two hundred and seventy one studies included in this original scientific statement were studies done as far back as 1970; however, most studies for this original scientific statement were from the late 1990's through 2007.

There was no discussion on how the studies were performed or designed, the sample size of the studies, if there were any concerns in study's design, or how the studies were chosen for inclusion in this original scientific statement. This scientific statement was published in a reputable journal that is subject to peer review and strict publication guidelines. All reviewers for this original scientific statement were required to complete and submit a disclosure questionnaire revealing any relationships that could be perceived as actual or potential conflicts of interest.

This article is a consensus statement of the available literature regarding the prevention of HF. This original scientific statement tries to be thorough by giving a basic foundation for the prevention of HF by including the global burden of HF, discussion of the major and minor clinical risk factors for the development of HF, summary of efforts to prevent HF due to atherosclerotic CAD, advances in prevention of hypertensive heart disease, special considerations in HTN, other risk factors and the prevention of HF, and future directions for HF prevention.

DISCUSSION

The article entitled "ACC/AHA 2005 Guideline Update for the Diagnosis and Management of Chronic Heart Failure in the Adult: A Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to Update the 2001 Guidelines for the Evaluation and Management of Heart Failure)" is a consensus statement of the available literature regarding the diagnosis and management of HF in adults (2). The original practice guideline from the American College of Cardiology/American Heart Association was published in 1995 with revised guidelines in 2001 and the most recent in 2005. Six hundred and ninety four studies included in the current scientific statement were studies done as far back as the 1964; however, most studies for the current scientific statement were from the late 1990's through 2005.

There was no discussion on how the studies were performed or designed, the sample size of the studies, if there were any concerns in study's design, or how the studies were chosen for use in the current practice guideline. The recommendations listed in the practice guidelines are evidence based whenever possible. This practice guideline was published in a reputable journal that is subject to peer review and strict publication guidelines. All reviewers for the current scientific statement were required to complete and submit a disclosure questionnaire revealing any relationships that could be perceived as actual or potential conflicts of interest.

This practice guideline is a consensus of the available literature regarding the diagnosis and management of HF in adults with normal or low left ventricular ejection fraction. The current practice guideline specifically did not consider acute HF, which might merit a separate set of guidelines and is addressed in part in the ACC/AHA Guidelines for the Management of Patients with ST-Elevation Myocardial Infarction and the ACC/AHA 2003 Update of the Guidelines for the Management of Unstable Angina and Non-ST

Elevation Myocardial Infarction. The practice guideline is comprised of the following sections: introduction, characterization of HF as a clinical syndrome, initial and serial clinical assessment of patients, therapy, treatment of special populations, patients with HF who have concomitant disorders, end-of-life considerations, and implementation of practice guidelines.

At the beginning of each section are recommendations for the diagnosis, management, and treatment of the particular topic discussed with that section categorized as class I, II, IIa, IIb, or III and level of evidence A, B, or C. Class I is defined as conditions for which there is evidence and/or general agreement that a given procedure or treatment is beneficial, useful, and effective. Class II is defined as conditions for which there is conflicting evidence and/or a divergence of opinion about the usefulness/efficacy of a procedure or treatment. Class IIa is defined as weight of evidence/opinion is in favor of usefulness/efficacy. Class IIb is defined as usefulness/efficacy is less well established by evidence/opinion. Class III is defined as conditions for which there is evidence and/or general agreement that a procedure/treatment is not useful/effective and in some cases may be harmful. Level of evidence A is defined as data derived from multiple randomized clinical trials or meta-analysis. Level of evidence B is defined as data derived from a single randomized trial, or nonrandomized studies. Level of evidence C is defined as only consensus opinion of experts, case studies, or standard-of-care.

The information used from the practice guideline was extremely limited and only what was necessary for the EBM including definition of HF; heart failure as a progressive disorder; Figure 1 ACC/AHA classification system for heart failure; treatment of HTN; general measures for treating HF; drugs recommended for routine use; and exercise training in HF.

The article entitled “Exercise and Heart Failure: A Statement from the American Heart Association Committee on Exercise, Rehabilitation, and Prevention” is a consensus statement of the available literature regarding the effects of exercise in people with HF (3). This original statement from the American Heart Association was published in 2003. One hundred and seventy for studies included in this original scientific statement were studies done as far back as 1959; however, most studies for this original scientific statement were from the 1990’s through 2001.

There was no discussion on how the studies were performed or designed, the sample size of the studies, if there were any concerns in study’s design, or how the studies were chosen for inclusion in this original scientific statement. This scientific statement was published in a reputable journal that is subject to peer review and strict publication guidelines. All reviewers for this original scientific statement were required to complete and submit a disclosure questionnaire revealing any relationships that could be perceived as actual or potential conflicts of interest.

This article is a consensus statement of the available literature regarding the effects of exercise in people with HF. This original scientific statement tries to be thorough by giving a basic foundation for the use of exercise training in patients with HF by including an introduction, factors affecting exercise tolerance, role of exercise training in HF, exercise training recommendations, and areas for future research.

DISCUSSION FOR REFERENCE # 5,7,8

The book entitled “ACSM’s Resource Manual for Guidelines for Exercise Testing and Prescription. 4th ed.” is a consensus of the available literature compiled as a concise reference for those in the health and fitness industry (5,7,8). The first edition was published in 1988 with subsequent editions in 1993, 1998, and the most recent in 2001. The book is comprised of eighty chapters divided into the following sections: lifestyle and health, anatomy, biomechanics, exercise physiology, coronary artery disease, chronic disease, other chronic diseases, health and fitness assessment, electrocardiography, exercise programming, human development, modifications of health behavior, and program management.

There was no discussion on how the studies were performed or designed, the sample size of the studies, if there were any concerns in study’s design, or how the studies were chosen for inclusion in the current edition. The current edition includes the most recent information for each section with all the information within the resource manual being referenced. This resource manual was published by Lippincott, Williams & Wilkins in conjunction with a reputable organization, the American College of Sports Medicine, with all information contained within the resource manual subject to peer review and strict publication guidelines.

The current resource manual tries to be thorough by giving a foundation of information for each section. The information used from the resource manual was extremely limited and

only what was necessary for the EBM including: the expected peak AE SBP of 180-210 mmHg and DBP of 60-85 mmHg and the mechanisms by which AE may lower blood pressure; how AE affects type I and type II diabetes; and how AE affects triglycerides, total, high-density, and low-density lipoprotein cholesterol.

CONCLUSION

The prevention of HF has emerged as an urgent public health need with national and global implications. The management of HF typically involves multiple medications, lifestyle modification including diet and sodium restriction, and identifying and preventing the risk factors that lead to the development or progression of HF. An earlier and more aggressive treatment approach based on the new ACC/AHA classification system for HF (Figure 1) can help reduce the morbidity and mortality associated with HF (2). In addition, research has shown in patients with HF that AE cannot only have a favorable effect on the risk factors that promote the development or progression of this potentially fatal disease, but can lessen symptoms, increase exercise capacity, and improve QOL (2).

Because a universal exercise prescription for patients with HF does not exist, an individualized approach is recommended. This will allow for flexibility and adjustments to be made in the exercise program for when the patient is stable and when they are having signs or symptoms of active HF. Whenever possible, the initial training sessions should be done under direct supervision with telemetry monitoring. Having these initial sessions supervised will allow for instruction on proper exercise technique, monitoring for arrhythmias or symptoms that may contraindicate participation in an exercise program, and patient education. Transitioning to a home-based exercise program will vary from patient to patient depending on their level of deconditioning and disease stability.

Even though there is a need for further study, the benefits of AE training in modulating the CAD and HF risk factors and abnormal central hemodynamic and peripheral factors, AE is becoming more accepted and utilized in the treatment plan of persons with HF and should be viewed as a complement to rather than replacement of the pharmacological therapy, lifestyle modification including diet and sodium restriction, and physician follow-up.

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Author Information

Richard Andreuzzi, M.S., PA-S
King's College Department of PA Studies St. Wilkes Barre, Pennsylvania