Exercise Therapy for Heart Failure Patients in Canada

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KEYWORDS

• Functional capacity • Disability • Aerobic capacity • Muscular strength • Muscular endurance

KEY POINTS

- Contemporary heart failure (HF) pharmacologic therapies are intended to improve ventricular function and reduce ventricular afterload.
- HF patients have significant central and peripheral deconditioning.
- Incidence of HF in Canada is similar to other industrialized countries.
- HF patients require carefully tailored, individualized exercise programs.
- Exercise therapy for HF patients, both aerobic and resistance training, should start with ultrashort episodes of exercise activity, gradually increasing the frequency and duration of the exercise episodes.

INTRODUCTION

Time was when a diagnosis of congestive heart failure (HF) gave rise to the treatment of patients with digoxin and diuretics. Although these therapies almost invariably acutely improved patient's symptoms, mostly through a reduction in volume overload, decades later they were shown to probably hasten mortality, possibly through increased sympathetic activation.[1] In contemporary cardiology practice, beta blockers for severe left ventricular (LV) systolic dysfunction, once contraindicated; ace inhibitors or angiotensin receptor blockers; aldosterone inhibitors; and judicious use of diuretics, often on an as-needed basis based on weight and symptoms, have become the mainstays of pharmacologic therapy.[2] Despite these significant and substantial advances in medical therapy, with subsequent significant and substantial reductions in mortality, many patients remain significantly debilitated from a functional capacity perspective.

What has not been generally appreciated in the medical community, even the cardiology community or the wider associated health care professions, is the massive deconditioning influence of systolic HF on skeletal muscle aerobic function, along with reductions in capillary density, and thus aerobic capacity.[3] Many individuals, despite optimal pharmacologic therapy, continue to have significant functional impairment with concomitant shortness of breath during exertion. The general assumption is that these individuals are short of breath secondary to reduced cardiac output from their LV systolic dysfunction, even in the absence of volume overload or elevations in b-type natriuretic peptide (BNP) levels. Decades of medical dogma dictated that if patients can improve their LV function, they could reduce their symptoms of exertional breathlessness. Like most dogma, it

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was based on personal and professional beliefs rather than hard science.

In reality, patients with significant LV systolic dysfunction are often deconditioned from an aerobic capacity perspective, because of a significant reduction in oxidative enzymes within skeletal muscle. With reductions in forward blood flow, as a consequence of reduced cardiac output from LV systolic function, there is less tissue oxygenation of skeletal muscle (Fig. 1).[3,4] At a very simplistic level, the reduction in tissue oxygenation eventually translates into a reduction in oxidative enzymes within skeletal muscle. Interestingly, in the same manner that deprivation of myocardial blood flow can produce a reduction in myocyte function and subsequent hibernating myocardium, the same deprivation of oxygen at the skeletal muscle level may result in the reduced formation of oxidative enzymes in order to protect skeletal muscle from oxidative injury.[5]

From a patient perspective, it makes little difference whether their exertional shortness of breath and physical incapacitation are secondary to diminished cardiac output or secondary to diminished oxidative enzymes and energy production within the working skeletal muscles. Patients understand that within hours, even days, after a major cardiac insult that reduces cardiac output, anything other than minimal exertion causes significant shortness of breath. In hyperacute and acute situations, this is almost certainly a manifestation of poor cardiac output. Very quickly, however, the reduced cardiac output translates into reduced oxidative enzymes, reduced capillary density, and reduced energy production at the level of the skeletal muscle.

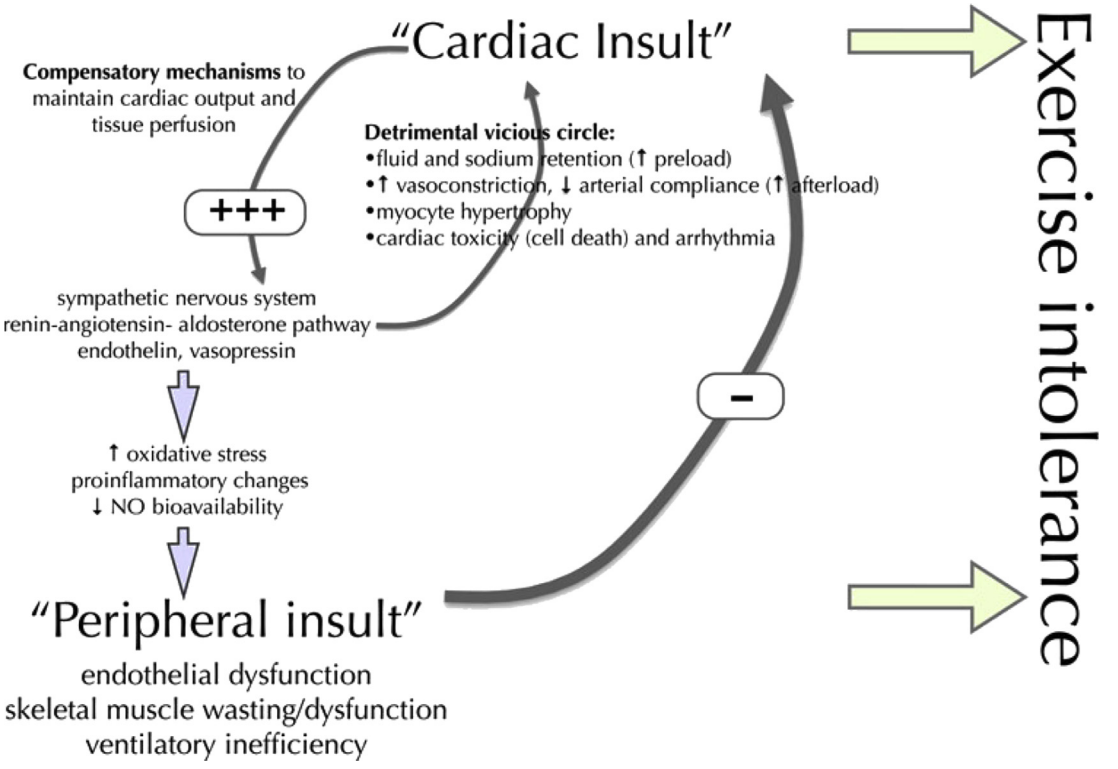

Fig. 1. Determinants of exercise intolerance in patients with chronic heart failure. Reduced cardiac performance will trigger compensatory neuro-hormonal mechanisms to preserve cardiac output, blood pressure and organ perfusion; these include the renin-angiotensinaldosterone pathway, the sympathetic nervous system, increased levels of endothelin and vasopressin. However, as these compensatory systems become chronically stimulated, they initiate a vicious circle and cause cardiotoxicity, myocyte hypertrophy/death, changes in peripheral and coronary artery compliance and excessive fluid retention. Progressively, other detrimental pathways are turned on, leading to oxidative stress, a pro-inflammatory status and reduced nitric oxide (NO) bioavailability, which initiate peripheral maladaptations, such as peripheral endothelial dysfunction, skeletal muscle wasting and ventilator inefficiency occur. Both central cardiac and hemodynamic changes, as well as these peripheral abnormalities will determine the heart failure phenotype and will culminate in symptoms of exercise intolerance. (*From* Conraads VM, Van Craenenbroeck EM, De Maeyer C, et al. Unraveling new mechanisms of exercise intolerance in chronic heart failure. Role of exercise training. Heart Fail Rev 2013;18:66; with permission.)