

Exercise Therapy for Older Heart Failure Patients

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KEYWORDS

- Heart failure syndrome • Aerobic training • Resistance training • Exercise therapy
- Aerobic capacity

KEY POINTS

- Both the aging process and heart failure (HF) syndrome are characterized by a dramatic reduction of aerobic capacity caused by a combination of cardiac and peripheral factors. Significant decreases in muscle mass and strength are also common to both conditions.
- Although a growing literature has documented that aerobic exercise training (ET) elicits improvement in peak oxygen consumption (V_{O_2}), submaximal exercise measures, and quality of life in younger HF patients, few HF training studies have included meaningful numbers of older individuals, especially those greater than 80 years of age and older women with HF with reduced ejection fraction (HFrEF). Nevertheless, the modest data available suggest similar benefits in older as in younger HF patients as well as excellent safety.
- Resistance training may provide additional benefit in older patients with HF, especially those with substantial muscle wasting.
- Whether ET can reduce mortality, hospitalizations, and overall health care costs in patients with HFpEF must await the outcome of adequately powered multicenter trials in this large subset of the HF population.

REDUCED AEROBIC CAPACITY: A CENTRAL FEATURE OF NORMATIVE AGING AND HEART FAILURE

Reduction in aerobic capacity, best quantified by peak V_{O_2} , is a central feature of both normative aging and chronic HF. Numerous observational studies over the past half-century have documented declines in peak V_{O_2} of approximately 50% across the adult age span in apparently healthy populations (Fig. 1).¹⁻⁴ In men, peak V_{O_2} decreases from approximately 45 mL/kg/min in a healthy 25 year old to approximately 25 mL/kg/min in a 75 year old (see Fig. 1). Comparable numbers in women are approximately 20% lower because of their smaller proportion of muscle

mass and lower hemoglobin levels. A healthy 80-year-old woman typically has a peak V_{O_2} of 15 mL/kg/min to 20 mL/kg/min, a range characteristic of mild HF. Even lower peak V_{O_2} is common in older adults with significant comorbidities, such as pulmonary disease, coronary or peripheral arterial disease, arthritis, and orthopedic or neurologic disorders, that further impair aerobic capacity. Furthermore, recent data suggest that longitudinal age-associated declines in peak V_{O_2} in healthy volunteers accelerate with age, exceeding 20% per decade in the 8th and 9th decades (see Fig. 1).⁵

Peak V_{O_2} is the product of cardiac output and arteriovenous oxygen (AVO_2) difference.

In healthy Baltimore Longitudinal Study of Aging volunteers, declines in peak HR and AVO_2

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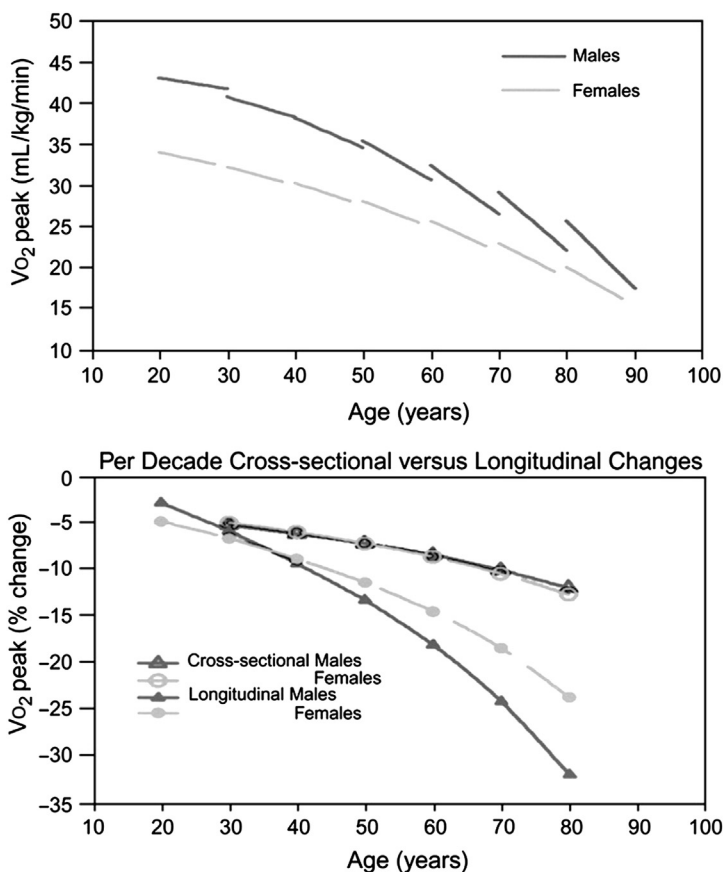


Fig. 1. Cross-sectional and longitudinal changes in peak Vo_2 per weight in kilograms in healthy adults by age decade and gender. (Top panel) The per-decade longitudinal change in peak Vo_2 for age decades from the 20s through the 70s, predicted from a mixed-effects regression model. Peak Vo_2 declines more steeply with successive age decades, especially in men. (Bottom panel) Per-decade percent cross-sectional and longitudinal changes in peak Vo_2 by age decade and gender, derived from the mixed-effects model. From the 50s onward, longitudinal declines in peak Vo_2 substantially exceed cross-sectional declines. (From Fleg JL, Morrell CH, Bos AG, et al. Accelerated longitudinal decline of aerobic capacity in healthy older adults. *Circulation* 2005;12:677; with permission.)

difference make similar contributions to the decline in peak Vo_2 with aging.⁶ In contrast, exercise stroke volume (SV) is not age related among individuals screened for the absence of coronary heart disease by clinical criteria and exercise thallium scintigraphy. In these individuals, enhanced use of the Frank-Starling mechanism augments left ventricular end-diastolic volume (LVEDV), compensating for modest blunting of systolic emptying with age.⁷ Plasma catecholamines are increased with age at peak exercise.⁸ This exercise hemodynamic profile of normal aging resembles that of β -adrenergic blockade of a young adult.⁹

HF, like aging, is characterized by a major reduction in peak Vo_2 , which provides powerful prognostic information regarding risk for hospitalization, mortality, and need for ventricular assist devices or cardiac transplantation.¹⁰⁻¹² The impairment in peak Vo_2 in these patients is attributable to both cardiac and peripheral factors. In patients with systolic HF, also known as HFpEF, peak HR and SV are reduced approximately 20% and 45%, respectively, compared with normal individuals.^{13,14} Peripheral factors contributing

to reduced AVO_2 difference, thence, peak Vo_2 , include reduced muscle mass, decreased mitochondrial density in exercising muscle, and peripheral vasoconstriction because of intrinsic abnormalities of smooth muscle vasodilation and neurohormonal factors.^{15,16} A similar constellation of peripheral abnormalities contributes to the age-associated decrease in AVO_2 difference at peak exercise (Table 1).¹⁷ In patients with HF and preserved systolic function (HFpEF), peak Vo_2 is reduced to nearly the same degree as in patients who have systolic HF.¹⁸ The reduction in peak exercise SV in these individuals is explained primarily by a lower LVEDV.¹⁹

DO OLDER PEOPLE WITHOUT HEART FAILURE RESPOND TO AEROBIC TRAINING LIKE YOUNGER ADULTS?

Although early studies suggested that aerobic capacity could not be augmented by exercise training (ET) in healthy older adults, multiple subsequent investigations have documented 10% to 25% increases in peak Vo_2 in previously sedentary adults through the ninth decade, comparable to

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