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Position statement

Exercise & Sports Science Australia Position Statement on exercise training and chronic heart failure

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Abstract

Chronic heart failure (CHF) is a complex syndrome characterised by progressive decline in left ventricular function, low exercise tolerance and raised mortality and morbidity. Regular exercise participation has been shown to be a safe and effective treatment modality in the majority of CHF patients, partially reversing some of the maladaptations evident in myocardial and skeletal muscle function, and resulting in improvements in physical fitness and quality of life, and perhaps reduced mortality. The volume and intensity of exercise that is recommended depends on the syndrome severity, however in most patients it should consist of a combination of low-to-moderate intensity aerobic (endurance) exercise on most days of the week and individually prescribed low-to-moderate intensity resistance (strength) training at least twice per week. Additionally, all patients should be closely monitored prior to and during exercise for contraindications by an appropriately trained health professional. The purpose of this statement is to inform and guide exercise practitioners and health professionals in the safe and effective prescription and supervision of exercise for patients with CHF.

Keywords: Heart failure; Exercise prescription; Physical function; Quality of life

1. Background

This Position Statement provides evidence-based guidelines for exercise interventions in individuals with stable chronic heart failure (CHF).

CHF is a complex clinical syndrome in which the ability of the left ventricle to fill with or eject blood[1] is impaired. The diagnosis requires a combination of specific clinical features linked with an objective demonstration of abnormal left ventricular function. The overall prevalence of heart failure in general Australian population is approximately 2.5% in people aged 55–64 to 8.2% in those aged 75 years and over.[2] It is one of the most common reasons for hospital admission and it is estimated that in Australia CHF accounts for a total of 100,000 hospitalisations per annum with 1.4 million days of hospital stay at a cost of >$1 billion annually.[3] CHF is also extremely common in general (or family medicine) practice in Australia with a prevalence of 13.2% in consecutive consultations with patients over the age of 60 years.[4]

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Table 1
NYHA classification.

Class I	No symptoms at rest, symptoms only at levels of exertion that would limit healthy individual
Class II	No symptoms at rest or mild exertion, symptoms on moderate exertion
Class III	No symptoms at rest, symptoms at mild exertion
Class IV	Symptoms at rest

The prognosis for patients with CHF remains poor in spite of improved medical treatments.[5] The two main causes of heart failure are hypertension and coronary artery disease, where long-term exposure to raised pressure, or acute insult to the cardiac muscle, result in ventricular remodelling and dysfunction.[6] The hallmark symptoms of CHF are fatigue[7] and dyspnea,[8] accompanied by decreased exercise tolerance,[9] and strength.[9,10] The severity of CHF symptoms are commonly classified under the New York Heart Association (NYHA) classification system (Table 1).[1]

Although reduced cardiac output defines the syndrome of CHF, it is widely accepted that functional capacity and cardiac function are poorly correlated and that peripheral factors including impaired blood flow[11] and skeletal muscle dysfunction contribute to the exercise intolerance evident in this group. Functional capacity, ideally measured as peak cardiopulmonary oxygen uptake ($\dot{V}O_2$ peak), is more important than ejection fraction and pulmonary artery wedge pressure as a prognostic indicator in advanced HF, and $\dot{V}O_2$ peak predicts survival better than changes in these haemodynamic indices.[12,13]

2. Role of exercise for management of CHF

Physical inactivity is a risk factor for heart disease.[14] The importance of adding exercise to the medical management of patients with CHF has been demonstrated in many studies. There is an inverse relationship between physical fitness and mortality.[15,16] Even moderate improvements in physical fitness can improve health status,[16] functional capacity and quality of life and decrease hospitalisation and mortality.[17,18] The recently completed HF-ACTION trial,[19] the largest exercise study in CHF to date, randomised 2331 heart failure subjects (NYHA II–IV, EF < 35%) to either usual care plus 12 weeks of supervised and ongoing home based aerobic training or usual care alone and reported no change in all-cause mortality and CHF hospitalisations at a median follow up of 30 months. However, after adjustment for predetermined prognostic factors, exercise training was associated with a statistically significant 15% decrease in all-cause mortality and CHF hospitalisations. It is important to acknowledge that this study yielded only modest overall gains in $\dot{V}O_2$ peak, and small increases in 6-min walk test (6MWT) distance. Although in the opinion of the authors of this statement the exercise intervention was sound as it was comparable to other smaller studies that have reported improvements in exercise tolerance,[20–22] it is possible that adherence levels were lower than intended and this may account for the lack of effect on the primary endpoints. However, there are many smaller randomised clinical trials showing clinically and functionally significant improvements in $\dot{V}O_2$ peak,[20–22] which has been closely linked to cardiovascular mortality in heart failure patients.[23,24] Alternatively, VE/VCO_2 slope has also been used as a prognostic marker. It has been recommended that all data for an incremental exercise test from the initiation of exercise to maximal exertion be included to calculate the VE/VCO_2 slope.[25] Arena et al.[25] reported that 24 of 26 studies found that the VE/VCO_2 relationship was superior to $\dot{V}O_2$ peak as a prognostic marker. It seems that $\dot{V}O_2$ peak and VE/VCO_2 slope provide independent and complementary information for the study of interventions in HF, with the latter perhaps better reflecting the overall, multi-system pathophysiology associated with HF.

Low aerobic power ($\dot{V}O_2$ peak) and exercise intolerance are common in CHF patients[26,27] with substantial evidence linking low $\dot{V}O_2$ peak with poor prognosis.[23,24] Aerobic training improves exercise capacity and $\dot{V}O_2$ peak in patients with CHF.[14,28,29] It is estimated that over 50% of patients with heart failure have preserved systolic function.[30] Published exercise training data suggest that patients with preserved systolic function will improve $\dot{V}O_2$ peak at least as much as HF patients with reduced systolic function.[31] Following exercise training HF patients may expect an improvement in $\dot{V}O_2$ peak in the order of 15%,[18] however not all patients respond to this extent. Possible reasons for low exercise training responses are low resting cardiac output,[32] poor adherence and autonomic neuropathy.[33] As many patients are sedentary and debilitated, average gains in aerobic power of 20% are achievable in as little as 3–4 weeks[34,35] and have important implications for physical function and quality of life. Concerns regarding the potential for adverse myocardial remodelling in response to exercise training have been allayed by evidence that aerobic training results in small but significant improvements (falls) in end-diastolic volume (EDV) and end-systolic volume (ESV),[21] while other work has demonstrated benefits in diastolic function,[36] indicating modest exercise-induced improvements in cardiac function. Exercise training in patients with CHF is generally safe, with no deaths and only 1 adverse event in 60,000 patient hours of supervised training documented in published trials, predominantly in subjects with NYHA Class ≤ III symptoms.[18] Interval training has been trialled as a means for heart failure patients to be able to exercise at relatively high intensities.[20,37] This may offer an effective alternative to moderate intensity continuous training. $\dot{V}O_2$ peak improved by 46% following high intensity interval training, compared to 14% for moderate intensity continuous training,[20] with no reported adverse events and even small improvements in left ventricular end-diastolic volumes and stroke volume.

CHF is associated with maladaptations in skeletal muscle structure and function. Resistance training is considered the most effective exercise modality to improve muscle mass,