

Effects of short-term moderate exercise training on sexual function in male patients with chronic stable heart failure

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Received 6 December 2003; received in revised form 13 March 2004; accepted 5 May 2004

Available online 9 September 2004

Abstract

Background: Patients with chronic heart failure (CHF) have sexual dysfunction that impairs quality of life. Recent trials have demonstrated that exercise training (ET) improves quality of life (QOL) of CHF patients, but it is not established whether this benefit may be associated with an improvement in sexual dysfunction.

Objective: To determine whether ET can improve sexual dysfunction in patients with CHF.

Methods: We prospectively studied 59 male patients (57 ± 9 years) with stable CHF in sinus rhythm and without prostatic disease. Patients were randomized into two groups. A group (T, $n=30$) underwent supervised cycle ergometer ET at 60% of peak VO_2 , three times a week, 60 min each session, for 8 weeks. A group (NT, $n=29$) was not exercised. Medications were not changed during the study. On study entry and at 8 weeks all patients underwent a symptom-limited cardiopulmonary exercise testing, brachial artery endothelium-dependent (ED) and endothelium-independent (EI) vasomotor responses, QOL and sexual activity profile assessment (SAP) by questionnaire.

Results: At 8 weeks, no changes were observed in control patients. In trained patients, however, peak VO_2 improved by 18% ($P<0.005$) and was correlated with QOL ($r=0.80$; $P<0.001$). Flow-mediated dilation improved in trained patients (from $2.29 \pm 1.13\%$ to $5.04 \pm 1.7\%$, $P=0.0001$), while EI dilation (after 0.3 mg sublingual NTG) did not. In group T, all three domains (i.e. Domain 1=relationship with the partner; Domain 2=quality of penile erection; Domain 3=personal wellness) were significantly improved from baseline (total score patients: from 3.49 ± 3.4 to 6.17 ± 3.2 , $P<0.001$; partners: from 2.47 ± 2.7 to 4.87 ± 2.5 , $P<0.001$). Pre-post training change in SAP total score was correlated with changes in coronary risk profile ($r=-0.49$; $P=0.01$), peak VO_2 ($r=0.67$; $P<0.001$) and QOL ($r=0.73$; $P=0.01$). Multivariate analysis selected the improvement in ED-vasomotor response as the strongest independent predictor of SAP improvement ($r=0.63$, $P<0.001$).

Conclusions: In stable CHF, cycle ergometer ET significantly improves brachial artery endothelial dysfunction, suggesting a systemic effect of leg exercise. This benefit was correlated with improvements in sexual activity.

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Keywords: Sexual activity; Chronic heart failure; Exercise training; Endothelium-dependent relaxation

1. Introduction

Sexual dysfunction frequently occurs in cardiac patients and its pathophysiological mechanism is not fully understood [1–4]. A vascular or neurological impairment for penile erection is frequently coupled with psychological and relational problems [5–7]. Perception of well being and self-

esteem are generally depressed and contribute to worsen quality of life (QOL).

One hypothesis is that a depressed sexual activity is caused by endothelial dysfunction. As a matter of fact, erectile dysfunction frequently occurs in patients with one or more coronary risk factors who do not have yet clinical manifestations of coronary artery disease [8–12]. It is well known that hypercholesterolemia, hypertension, cigarette smoking and diabetes are not only established risk factors for atherosclerosis and premature coronary artery disease, but they are also known to impair endothelial function [13–15].

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Thus, there are similarities between risk factors for coronary artery disease and risk factors for erectile dysfunction, suggesting a common pathophysiological background [16]. Nitric oxide activity may be a possible common link between the two abnormalities, and therapeutic strategies aiming to improve endothelial dysfunction may positively affect penile erection. There is evidence that an increased nitric oxide synthase activity generally translates into enhanced erectile function [17–23]. However, information is lacking about sexual activity in patients with chronic heart failure (CHF) and whether an improvement in endothelial function may be associated with a more active sexual behaviour.

Chronic heart failure is a complex pathophysiological syndrome characterized by reduced exercise tolerance which is partly related to endothelial dysfunction [24]. Recent evidence is mounting that moderate aerobic exercise training (ET) attenuates endothelial dysfunction in clinically stable chronic heart failure patients, and this effect is correlated with improved functional capacity [25,26]. This benefit is in part related to improvement in coronary risk profile. However, it may also be the consequence of a direct effect of exercise training on the nitric oxide pathway [27]. Thus, if endothelial dysfunction plays an important role in both chronic heart failure and erectile dysfunction, it is conceivable that an improvement in the endothelium-dependent (ED) vasorelaxation can exert positive effects in both conditions. Therefore, we hypothesize that exercise training, by improving endothelial function, may enhance not only functional capacity, but also sexual activity and quality of life in men with stable chronic heart failure.

2. Methods

We longitudinally studied 59 male patients with chronic heart failure in stable condition in the last 3 months. (Table 1) The majority of the population study had an ischemic cardiomyopathy with depressed left ventricular function and multivessel coronary artery disease. Inclusion criteria were stable chronic heart failure, absence of prostatic disease, a woman partner, ability to exercise. Exclusion criteria were unstable angina, recent acute myocardial infarction, decompensated heart failure, hemodynamically significant valvular heart disease, uncontrolled hypertension, renal insufficiency (serum creatinine >2.5 mg/dl), urologic disorders, and any orthopedic or neurologic illness limiting the ability to exercise. Patients were randomized into two groups. A group was exercised for 8 weeks (group T, $n=30$), and a group (NT, $n=29$) was not exercised and recommended to avoid regular exercise. On entry and at 8 weeks all patients underwent a cardiopulmonary exercise testing, a brachial artery vasomotor function study, quality of life and sexual activity profile assessments (SAP). Medications were given at standard doses, were similarly distributed in the two groups, and were not changed during the study. As shown in

Table 1

Clinical characteristics of study population

| | Group T | Group NT |
|----------------------------------|---------------------------------|----------------------------------|
| Number | 30 | 29 |
| Age, years (mean±S.D.) | 55.9±15 | 58±12 |
| Diagnosis, n | | |
| Ischemic | 24 | 24 |
| Idiopathic | 6 | 5 |
| Hypercholesterolemia, n (%) | 11 (36.7) | 12 (41.4) |
| Hypertension, n (%) | 8 (26.7) | 8 (27.6) |
| Smoke, n (%) | 10 (36.7) | 9 (27.6) |
| Diabetes mellitus, n (%) | 2 (6.7) | 3 (10) |
| No risk factors, n (%) | 10 (33.3) | 9 (31) |
| One risk factor, n (%) | 9 (30) | 9 (31) |
| Two risk factors, n (%) | 7 (23.3) | 8 (27.6) |
| Three risk factors, n (%) | 4 (13.3) | 3 (10.3) |
| Peak VO_2 , ml/kg/min | 16.8±3.7 | 15.9±1.5 |
| NYHA functional class, n | 18II, 12III | 15II, 14III |
| Coronary lesion score | 1.9±0.9 | 1.8±0.9 |
| LV ejection fraction, % | 29.3±6 | 28.1±5 |
| Medications, n | D19, DIU30, C16, N17, ACE127 | D17, DIU28, C17 N18, ACE1, 26 |

Peak VO_2 =oxygen uptake at peak exercise; LV=left ventricular; D=digitalis; DIU=diuretics; C=carvedilol; N=nitrates; ACEI=angiotensin converting inhibitors.

Table 2, coronary risk factors were present in 41 out of 59 patients (69.5%) and were similar in the two groups.

The protocol was approved by the local Ethical Committee. Patients and their partners were informed and signed a written consent form.

2.1. Cardiopulmonary exercise testing

After a familiarization test 48–72 h before the initial evaluation, an exercise stress testing with gas exchange and ventilation breath-by-breath analysis was performed on an electronically braked cycle ergometer connected with a metabolic cart (Sensormedics 2900 Z, Yorba Linda, CA). Blood pressure and 12-lead ECG were taken every minute. Work rate was smoothly increased (10 or 15 W/min, ramp) in order to reach exhaustion after 8–12 min. Other criteria for stopping the test were ischemic changes on ECG tracings with or without angina, blood pressure reduction >10 mm Hg with work rate increase, frequent ventricular premature contractions, or tachyarrhythmias. Peak oxygen uptake was calculated as the average of single measurements during the last 15 s of exercise [44].

2.2. Brachial artery vasomotor function

All studies were performed in air-conditioned room with a constant temperature (~23 °C), barometric pressure (~760 mbar), and humidity (~50%). Patients were evaluated in the morning in fasting condition. After 5 min of relaxation in supine position, a 7.5 MHz ultrasound probe was positioned over the dominant arm to detect good quality brachial artery

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