Heart transplantation is currently the preferred treatment for patients with end stage heart failure, with a median survival rate of 11 years for all patients and 14 years for patients who survive the early post-implantation period [1]. A limited number of donor organs and more people living with serious heart failure have increased the importance for alternative options. Ventricular assist devices are being successfully used as bridge-to-transplant or as destination therapy when heart transplantation cannot be considered, for example due to inoperability or non-compliance. Implantation of a VAD has shown to improve exercise capacity, quality of life and mortality in patients with severe heart failure, although exercise capacity after VAD therapy remains below that reported for age-matched controls after heart transplantation [2]. Exercise and improved peak oxygen uptake (VO2peak) are related to improved quality of life and prognosis in patients with severe heart failure [3]. Therefore, improving VO2 peak is an important goal with multifaceted clinical relevance in patients on VAD therapy.

The few studies investigating exercise in patients on VAD therapy have observed little to no effect on VO2 peak [4–6]. However, studies applying high intensity exercise in heart failure patients have observed feasibility and significantly higher increases in VO2 peak compared to moderate continuous exercise [7]. Therefore we applied this mode of exercise in one of our BiVAD patients to assess feasibility.

We present a case of a 61-year-old male with dilated cardiomyopathy (DCM) who volunteered to receive exercise therapy ten months after successful implantation of ventricular assist devices (HeartWare HVAD; HeartWare International Inc, Framingham, MA, USA) in both the left and right ventricles. Post-implantation, the patient presented with severely reduced left ventricular ejection fraction (EF = 15%) and NYHA functional classes II–III under optimal medical treatment. He reported regular walking of approximately 30–60 min per day, limited by dyspnea and fatigue due to the weight of the devices. The patient was 96 kg and had a BMI of 29.0 kg/m2 at baseline. Resting heart rate and mean arterial pressure were 85 bpm and 90 mm Hg, respectively. The patient reported no symptoms except for slight discomfort at the insertion-points of the VAD tubes and dyspnea upon exercising.

At baseline cardiopulmonary exercise testing (CPX), he reached a maximal power output of 62 W (0.64 W/kg) and VO2 peak of 0.9 l/min (9.7 ml/kg/min). The VE/VCO2-slope, an independent predictor of mortality in patients with reduced LVEF, was 51.

The patient performed supervised moderate exercise for six weeks at an intensity of 70%–80% of baseline maximal heart rate (HRmax) for 20–25 min, during which the exercise load increased from 49% to 55% of maximal (31 to 35 W). This was followed by six weeks of submaximal intensity interval training (IT). During IT, the patient began with 5 min of moderate exercise on a cycle ergometer after which he performed four submaximal-intensity intervals at 80%–90% of baseline HRmax (75%–80% VO2 peak) separated by three minute active recovery intervals at 60%–70% HRmax (50–60% VO2 peak).

After 12 weeks and a total of 36 exercise sessions, the patient reduced resting heart rate from 85 to 72 bpm. Maximum heart rate and heart rate reserve (HRR; HRmax–HRrest) both increased from 102 to 129 bpm and from 18 to 51 bpm, respectively. Exercise testing showed an increase in HRmax from 103 to 126 bpm and an increase in VO2 peak from 0.9 l/min (9.7 ml/kg/min) at baseline to 1.6 l/min (15.3 l/kg/min). Maximal power output increased from 62 W (0.64 W/kg) at baseline to 102 W (1.06 W/kg) and VE/VCO2-slope decreased from 51 to 44 (see Fig. 1).

This is the first patient with bilateral ventricular assist devices to have performed IT as supplemental to pharmaceutical and device therapy. The patient markedly improved clinically important CPX prognostic parameters [8–10], including heart rate response to exercise (HRrest, HRmax and HRR), VO2 peak, maximal power output and VE/VCO2-slope.

There are very few data on exercise in heart failure with VAD therapy. A recent review concluded that exercise seems to be safe and has a positive effect on exercise capacity in patients on VAD therapy [4]. The studies that have been conducted have reported very promising enrolment and compliance, but have been ambiguous on exercise...
performance [4–6]. Hayes et al. reported a non-significant increase in VO2 peak of 3.0 ml/kg/min after eight weeks of mobilization (walking) plus moderate intensity endurance and resistance exercise (N = 7) compared to a control group (N = 7) who performed mobilization therapy alone [5]. More recently, a study investigated the effects of 18 sessions of moderate intensity continuous exercise (60–80% of heart rate reserve) on 26 patients with VAD [6]. In this study, patients improved VO2 peak 10%, but this was not significant compared to usual care controls. The failure of these studies to observe significant effects may be due to small sample sizes and relatively low exercise intensities. In the current case, the patient performed submaximal exercise of up to 90% of baseline HRmax over 12 weeks, a higher intensity and volume than the aforementioned studies. This preliminary report is in agreement with interval training studies in exercise in heart failure without VAD therapy, having observed improvements in VO2 peak of up to 40% more than moderate continuous exercise and 50% more than usual care [7].

This case demonstrates that higher intensity exercise may be feasible in heart failure patients with VAD or BiVAD therapy. The improvements in clinically significant CPX prognostic indicators warrant further research into higher-intensity exercise therapy for heart failure patients with VAD and BiVAD.

**Conflict of interest**

The authors report no relationships that could be construed as a conflict of interest.

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**Fig. 1.** A–D: Results of CPX at baseline and after 12 weeks of endurance exercise training. A. Chronotropic incompetence is defined as reaching >80% of either predicted maximum heart rate (127 bpm; dark broken line) or predicted heart rate reserve (63 bpm; light broken line) [10]. B. Maximal power output. C. Peak oxygen uptake. Broken line represents threshold for one year cardiac mortality prognosis (VE/VCO2 = 34) [8,9]. D. Ventilatory equivalent ratio for CO2. Broken line represents threshold for one year cardiac mortality prognosis (14.0 ml/kg/min) [8,9].