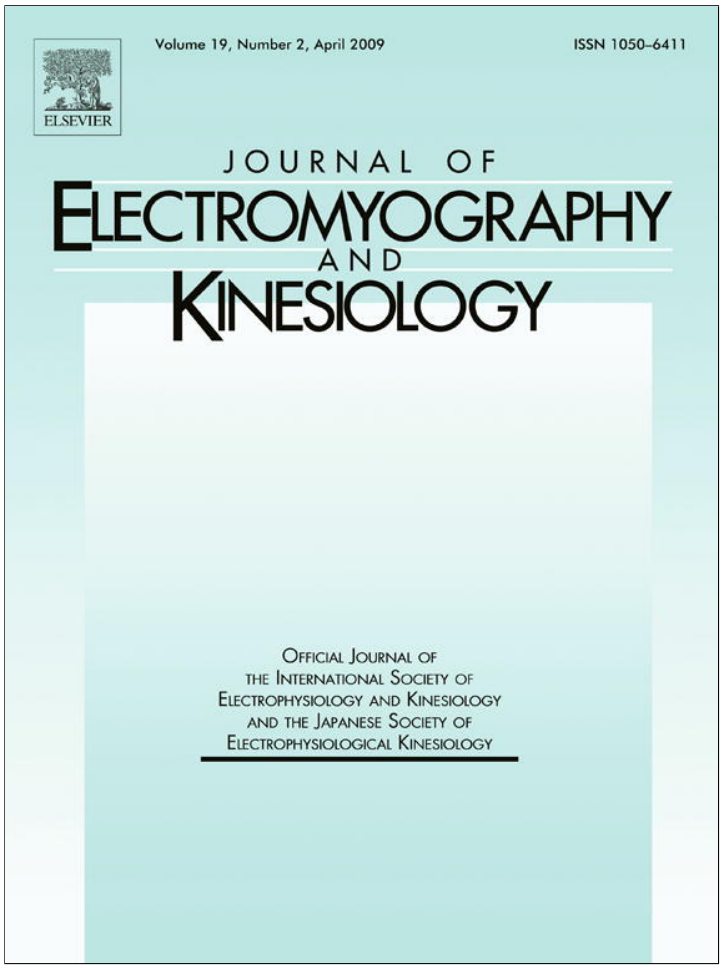

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Effects of exercise training modality on skeletal muscle fatigue in men with coronary heart disease

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Abstract

Cardiopulmonary and skeletal muscle effects of combined aerobic and resistance training vs. aerobic training were studied in men with coronary heart disease. Sixteen men with coronary heart disease underwent a cardiopulmonary exercise testing and a quadriceps skeletal muscle fatigue assessment. Patients were divided into two groups and trained in a combined aerobic and resistance or aerobic training group during 7 weeks. Maximal voluntary contraction and isometric endurance time were measured with electromyographic signals recorded from vastus lateralis (VL), rectus femoris (RF) and vastus medialis (VM) during isometric endurance time. Exercise tolerance increased only in the combined group ($p < 0.05$). Maximal voluntary contraction and isometric endurance time did not change after training in either group but was performed at 5.8% higher force output for the combined group. After training, median frequency values were higher for the VL and VM ($p < 0.001$) in the aerobic group and also higher for the VL, RF ($p < 0.001$) and VM ($p < 0.05$) in the combined group. Combined aerobic and resistance training was more effective to improve exercise tolerance, decrease skeletal muscle fatigue and correct neuromuscular alterations in men with coronary heart disease.

Keywords: Coronary heart disease; Exercise training; Modality; Skeletal muscle fatigue; Electromyography

1. Introduction

Impaired exercise tolerance represents a major problem in some patients with coronary heart disease (CHD) and often results in functional disabilities (Ades, 2001). Myocardial ischaemia in patients with CHD can lead to exertional dyspnea, which may limit exercise capacity (Ades, 2001). Patients with CHD limit their physical activities because of exercise intolerance. This results in a cycle of inactivity and physical deconditioning, consequently daily activities and quality of life are reduced (Ades, 2001). Positive effects of aerobic exercise training have been well documented in CHD patients with benefits on clinical outcomes, exercise capacity, cardiac function, coronary risks factors, endothelial function, quality of life and psychological parameters (Ades, 2001; Fletcher et al., 2001). Resistance training is recommended as a complement to aerobic training, with additional benefits on body composition, bone density, strength and glucose metabolism in this population (Fletcher et al., 2001; Pollock et al., 2000).

Skeletal muscle fatigue occurs sooner in CHD patients than in normal patients and is associated with exercise intolerance (Gayda et al., 2005, 2003) and surface electromyography (SEMG) with spectral analysis of motor unit

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function provides an objective measure of skeletal muscle fatigue (Buonocore et al., 1998; Gabriel et al., 2001; Gayda et al., 2005; Giannesini et al., 2003; Portero et al., 2001). Skeletal muscle fatigue is associated with an increase in the SEMG signal amplitude (as the root mean square) and with a shift of the SEMG spectrum (as the median frequency) towards lower frequencies (Buonocore et al., 1998; Gayda et al., 2005; Giannesini et al., 2003; Portero et al., 2001). Using SEMG, skeletal muscle fatigue has been studied in chronic heart failure (CHF) patients. CHF patients show the early development of muscle fatigue and impaired myoelectrical activity in the large (Minotti et al., 1992; Wilson et al., 1992) and small (Buonocore et al., 1998) muscle groups. Recently, SEMG activity was correlated with skeletal muscle fatigability in CHF patients (Schulze et al., 2004).

Impaired skeletal muscle function in CHD patients has been in association with a reduced aerobic capacity compared with healthy controls (Gayda et al., 2003). We also previously demonstrated impaired skeletal muscle endurance with neuromuscular abnormalities in men with CHD compared to healthy control subjects (Gayda et al., 2005). Skeletal muscle fatigue in CHD patients may have important consequences for quality of life and the ability to complete daily activities (Ades, 2001; Gayda et al., 2005). Additionally, skeletal muscle function and fatigue is clinically very important because, in CHD patients, improvement of exercise tolerance is often mediated by improvement of skeletal muscle metabolism and function (Ades et al., 1996; Cottin et al., 1996; Fletcher et al., 2001).

During cardiac rehabilitation, exercise training is an important part of therapeutics interventions, resistance training is recommended to be added to aerobic exercise to optimize exercise training benefits. To our knowledge, in CHD patients, there is no previous data on the effects of exercise training on skeletal muscle fatigue and it is not known if exercise training could modify neuromuscular alterations previously reported in CHD patients (Gayda et al., 2005). Our hypothesis is that, combined aerobic and resistance training would be superior to aerobic training in the improvement of neuromuscular alterations and muscle fatigue in CHD patients. The purpose of this study is to compare the cardiopulmonary and skeletal muscle effects of combined aerobic and resistance training versus aerobic training alone in men with coronary heart disease.

2. Materials and methods

Sixteen male patients with CHD took part in this study (age: 55 ± 8 years, weight: 86 ± 11 kg, and height: 172 ± 6 cm). The clinical characteristics of the male CHD patients are given in Table 1, and they were included in the study one month after their cardiac event (phase II cardiac rehabilitation program). Medication was checked on patient's medical chart and remained unchanged during the cardiac rehabilitation program (Table 1). Inclusion criteria for CHD men consisted of the presence of documented CHD (prior myocardial infarction, prior coronary angioplasty, coronary bypass surgery, or documented myocardial ischemia on myocardial scintigraphy). Patients were excluded from the study if the resting left ventricular ejection fraction (LVEF) was $\leqslant 35\%$, if they had documented history of heart failure, severe exertional ischaemia (>3 mm ST-segment depression), severe exertional arrhythmias, or an exercise limitation due to a non-cardiopulmonary cause (e.g., arthritis). The resting LVEF was evaluated using the angioventriculography technique and obtained from medical records.

Table 1
Clinical characteristics of the 16 male patients with CHD

	Number of patients
Diagnosis	
MI	4
CABS	7
Angioplasty	5
NYHA classification	
I	9
II	7
LVEF (%)	52 ± 9
Treatment	
Calcium channel blockers	11
ACE inhibitors	5
Aspirin	10
Amiodarone	1
Statins	10
Diuretics	1
Oral antidiabetics	1
β-Blockers	11

The numbers in the right part of the table indicated how many patients are concerned (MI: myocardial infarction; CABS: coronary artery bypass surgery; NYHA: New York Heart Association; LVEF: left ventricular ejection fraction).

Body composition data (fat mass: FM, lean body mass: LBM, total volume of the lower limb: TVLL, muscle volume of lower limb: MVLL) were carried out before and after training with measurement of skin folds and lower limb circumferences (Gayda et al., 2005, 2003). All subjects gave their written consent to participate in the study. The experimental procedures complied with the ethical standards of the 1975 Helsinki Declaration. All subjects were assigned to a maximal exercise testing session on cycle ergometer and an evaluation of the skeletal muscle function on an isokinetic apparatus with SEMG measurements. A medical doctor was present during cardiopulmonary exercise testing and skeletal muscle function assessment. Patients with CHD were then randomly divided into a combined aerobic–resistance (CAR, $n = 8$) or an aerobic training group (AT, $n = 8$), and trained 1 h, three times per week during 7 weeks. Aerobic training intensity was individualized according to ventilatory threshold (VT) power.

2.1. Cardiopulmonary exercise testing

Cardiopulmonary exercise testing was performed on a cycle ergometer (Ergoline 800 S, Ergoline®, Germany) using an incremental protocol. The initial power was fixed at 50 W and increased by 15 W every minute; the pedaling speed was fixed at 60 rpm during the entire test. Gas exchange was measured using an open circuit technique with the CPX system (CPX system, Medical Graphics®, USA) as previously described (Gayda et al., 2003). ECG was continuously measured during testing with a blood pressure measurement every minute. The test lasted until subjects attained a gas exchange ration (VCO_2/VO_2) > 1.1, or

until exhaustion (i.e. inability to maintain a pedaling frequency greater than 60 rpm) despite encouragement. Standard verbal encouragement was used for all subjects. Exercise tolerance was assessed with peak VO_2 (highest VO_2 reached during exercise; gas analysis data were averaged every 15 s) and ventilatory threshold (VT) values using Beaver's method (Beaver et al., 1986). At the end of the test, each patient had a 5-min passive recovery.

2.2. Skeletal muscle function assessment

Skeletal muscle fatigue was measured for the quadriceps muscle on an isokinetic apparatus (Cybex Norm II, Cybex®, USA) as previously described (Gayda et al., 2005, 2003). Subjects were seated and positioned at a knee angulation of 60°, with a hip angulation of 120° and were strapped at chest and knee levels. Skeletal muscle function was evaluated with the measurement of the maximal voluntary contraction (MVC) and the isometric endurance time (IET) at an intensity level of 50% of the MVC. The three best values (from three sets of five repetitions) were retained, averaged and considered as the MVC. During IET, subjects were asked to maintain an isometric contraction at 50% of the MVC until exhaustion with a visual feedback.

2.3. Aerobic training

For both groups, aerobic exercise training was performed three times per week, on non-consecutive days for 7 weeks (20 sessions). Each session was performed in the morning, and consisted of 30 min of callisthenics movements, stretching and respiratory exercises with a physical therapist. Then, patients exercised during 30 min on an cycle ergometer and training intensity was individualized according to ventilatory threshold (VT) power (W). Exercise training intensity (VT power) were controlled and applied during training by a computer system connected with the training ergocycles. As part of each training session, patients were also advised to take an outdoor walk of 1-h duration in the afternoon on the same day.

2.4. Resistance training

Resistance training was only performed in the CAR group ($n = 8$), three times per week for 7 weeks. Resistance training was performed on special resistance apparatus (Banc de Koch, Génin Médical®, France) (Koch et al., 1992) that allows successive local muscle resistance training. Because use of this apparatus is very time consuming and requires a physical therapist, two major movements were chosen: quadriceps leg extension and lateral pull down. Exercise intensity was set at 40% of the maximal voluntary contraction measured on the apparatus dynamometer. Patients performed three sets of 10 repetitions for each movement (quadriceps leg extension and lateral pull down). Exercise time for one movement repetition was around 30 s. Exercising time for quadriceps leg extension was 4 min and 30 s, with a total exercising time including lateral pull down around 9 min. Each session was performed in the afternoon, the same day than aerobic training which was performed in the morning.

2.5. SEMG measurements and analysis

During IET, SEMG activities were recorded from the vastus lateralis (VL), rectus femoris (RF) and vastus medialis (VM). Bipolar Ag–AgCl surface electrodes with a disc shape (model E224A, In vivo Metric®, Healdsburg, USA; sensor diameter: 8 mm; inter-electrode distance: 20 mm) were employed and placed according to previous published methodology (Gayda et al., 2005). For VL, electrodes were located on quarter distance proximal to the lateral tibial condyle on a line connecting this and the anterior–superior-iliac-spine (ASIS). For RF, electrodes were placed at mid-distance along the line connecting the ASIS to the superior aspect of the patella. The electrodes were located over VM at a position approximately 20% of the distance along a line connecting the medial gap of the knee to the ASIS. Before electrodes application, the skin was cleaned by abrasion and sponging with an alcohol–ether–acetone mixture to reduce inter-electrode impedance below 2 kΩ, electrodes cavities were filled with saline electrode gel (Signa Gel®, In vivo Metric®, Healdsburg, USA). Electrode positions were carefully marked on the skin with a permanent marker to ensure accurate repositioning after training. The signals of VL, RF and VM were stored on the computer after isolated differential amplification (bandwidth 1 Hz–1 kHz, gain = 1000, Gould 6600 amplifier). SEMG signals were analyzed online using acquisition and spectral analysis software (Calvise, Divergent®, Compiègne, France) and data computing software (Spatol, Divergent®, Compiègne, France) (Gayda et al., 2005; Portero et al., 2001). The SEMG signals were sampled at 1024 Hz. Each spectrum was calculated from 0.5 s time windows and was defined by 256 points, on a 0–512-frequency band. The root mean square (RMS) and median frequency (MF) were calculated in real-time by the computer. RMS and MF were averaged for each muscle and normalized in percentage of initial values (Gayda et al., 2005; Portero et al., 2001).

2.6. Statistical analysis

Anthropometric, skeletal muscle, SEMG and exercise testing data of CHD patients were compared before and after training for each group (CAR group: $n = 8$ and AT group: $n = 8$) using an analysis of variance (statview software 5.0, SAS institute, USA). A p-value $\leqslant 0.05$ was considered statistically significant and a post hoc test (Scheffé F-test) was used to localize the differences. Normalized SEMG data were analysed and compared between groups by using the common isometric endurance time (60 s) (Gayda et al., 2005).

3. Results

3.1. Body composition and skeletal muscle function measurements

Body composition and skeletal muscle function data were not significantly modified in CHD patients after training (Table 2). There was a non-significant increase (+11.6%, $p = 0.2$) for MVC in the CAR group, and IET was performed at a 5.8% higher force output after training.

3.2. Cardiopulmonary exercise testing data

After training, the CAR training group improved VO_2, VCO_2 and power at VT and peak VE, VCO_2 and power at maximal effort (Table 3). There was a tendency to an increase ($p = 0.08$) for peak VO_2 in the CAR group. No training effect was noted for the AT training group (Table 3).

Table 2
Body composition and skeletal muscle data in men with CHD in both training groups (means ± SD, CAR: combined aerobic and resistance; AT: aerobic training; ns: not significant)

Parameter	CAR group ($n = 8$)		AT group ($n = 8$)		p-Values
	Pre	Post	Pre	Post	
Weight (kg)	86 ± 8	84 ± 7	85 ± 15	84 ± 14	ns
Fat mass (FM, in %)	27 ± 2	28 ± 3	28 ± 3	30 ± 4	ns
Lean body mass (LBM, kg)	62 ± 5	61 ± 6	61 ± 8	59 ± 10	ns
Muscular volume of lower limb (MVLL, L)	8.8 ± 0.5	9.4 ± 1.3	8.1 ± 1.5	8.1 ± 1.5	ns
Maximal voluntary contraction (MVC, N m^{-1})	249 ± 53	278 ± 45	208 ± 45	214 ± 56	ns
Isometric endurance time (IET, s)	69 ± 23	66 ± 12	58 ± 6	59 ± 3	ns

NB: Isometric force output is 5.8% higher for the CAR group during IET after training. a: CAR group; ns: not significant.

Table 3
Cardiopulmonary exercise testing data at VT and maximal effort in men with CHD in both training groups (mean values ± SD)

Parameter	CAR group ($n = 8$)		AT group ($n = 8$)		p-Values
	Pre	Post	Pre	Post	
Values at VT					
VO_2 (mL min^{-1} kg^{-1})	15.1 ± 6.3	21.6 ± 3.0	14.8 ± 4.8	15.9 ± 4.5	a*
VE (L min^{-1})	37.2 ± 11.2	45.1 ± 4.7	33 ± 10	38 ± 10	ns
HR (beats min^{-1})	88 ± 14	97 ± 10	93 ± 9	96 ± 19	ns
VCO_2 (mL min^{-1})	1215 ± 491	1684 ± 175	948 ± 261	1219 ± 391	a*
Power (W)	88 ± 41	129 ± 21	67 ± 18	85 ± 20	a*
Peak values					
VO_2 (mL min^{-1} kg^{-1})	23.4 ± 8.9	30.9 ± 3.6	20.9 ± 6.9	23.9 ± 5.9	a: $p = 0.08$
VE (L min^{-1})	64.9 ± 18.1	89.2 ± 20.1	59 ± 23	73 ± 21	a*
HR (beats min^{-1})	113 ± 13	125 ± 11	109 ± 14	120 ± 24	ns
VCO_2 (mL min^{-1})	2082 ± 743	2805 ± 376	1728 ± 628	2077 ± 512	a*
Power (W)	136 ± 50	190 ± 23	111 ± 34	137 ± 30	a*

VT: Ventilatory threshold; CAR: combined aerobic and resistance; AT: aerobic training; a: CAR group; *: $p < 0.05$; ns: not significant.

3.3. SEMG measurements

3.3.1. Root mean square

After training, normalized RMS values increased for the VL in the CAR group (CAR pre vs. post, $p < 0.001$; Table 4). Additionally, RMS values were lower for the RF in the AT group (AT group pre vs. post, $p < 0.001$; Table 4).

3.4. Median frequency

After training, normalized MF increased significantly in both the CAR and AT groups for the VL (CAR group pre vs. post: $p < 0.001$; AT group pre vs. post: $p < 0.001$) and for the VM (CAR group pre vs. post: $p < 0.05$; AT group pre vs. post: $p < 0.001$) (Table 4). Normalized MF

Table 4
Mean SEMG data (normalized in % of initial value) in men with CHD during IET (mean values and standard deviation)

Parameter	CAR group ($n = 8$)		AT group ($n = 8$)		p-Values
	Pre (%)	Post (%)	Pre (%)	Post (%)	
Root mean square (% of initial value)					
Vastus lateralis	100.36 ± 3.35	109.33 ± 15.06	101.65 ± 9.11	101.34 ± 8.59	a: ‡, b: ns
Rectus femoris	97.25 ± 2.59	93.98 ± 4.97	116.92 ± 11.77	101.25 ± 3.78	a: ns, b: ‡
Vastus medialis	103.05 ± 2.23	101.28 ± 3.91	97.46 ± 3.88	101.21 ± 11.39	a: ns, b: ns
Median frequency (% of initial value)					
Vastus lateralis	81.93 ± 15.06	98.58 ± 0.95	78.92 ± 11.46	101.57 ± 8.60	a: ‡, b: ‡
Rectus femoris	91.60 ± 4.14	98.34 ± 6.66	89.79 ± 5.28	92.09 ± 4.67	a: ‡, b: ns
Vastus medialis	93.36 ± 7.82	98.41 ± 0.99	75.16 ± 10.21	95.11 ± 1.27	a: *, b: ‡

SEMG: Surface electromyography; CHD: coronary heart disease; IET: isometric endurance time; CAR: combined aerobic and resistance; AT: aerobic training. a: CAR training pre vs. post; b: AT group pre vs. post; *: $p < 0.05$; ‡: $p < 0.001$; ns: not significant. NB: Isometric force output is 5.8% higher for the CAR group during IET after training.

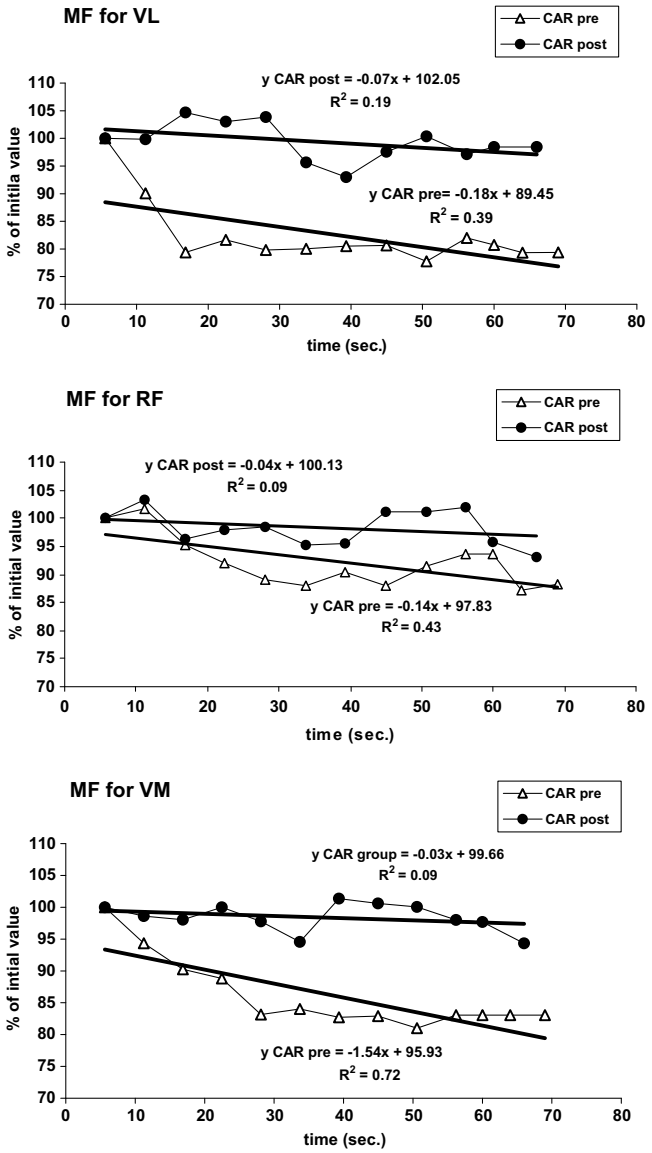

Fig. 1. MF modifications for the vastus lateralis, rectus femoris and vastus medialis in the CAR group after training (vs. pre values). Mean values (in % of initial value) fitted out with a linear regression. NB: Isometric force output is 5.8% higher for the CAR group after training. MF = median frequency and CAR = combined aerobic and resistance.

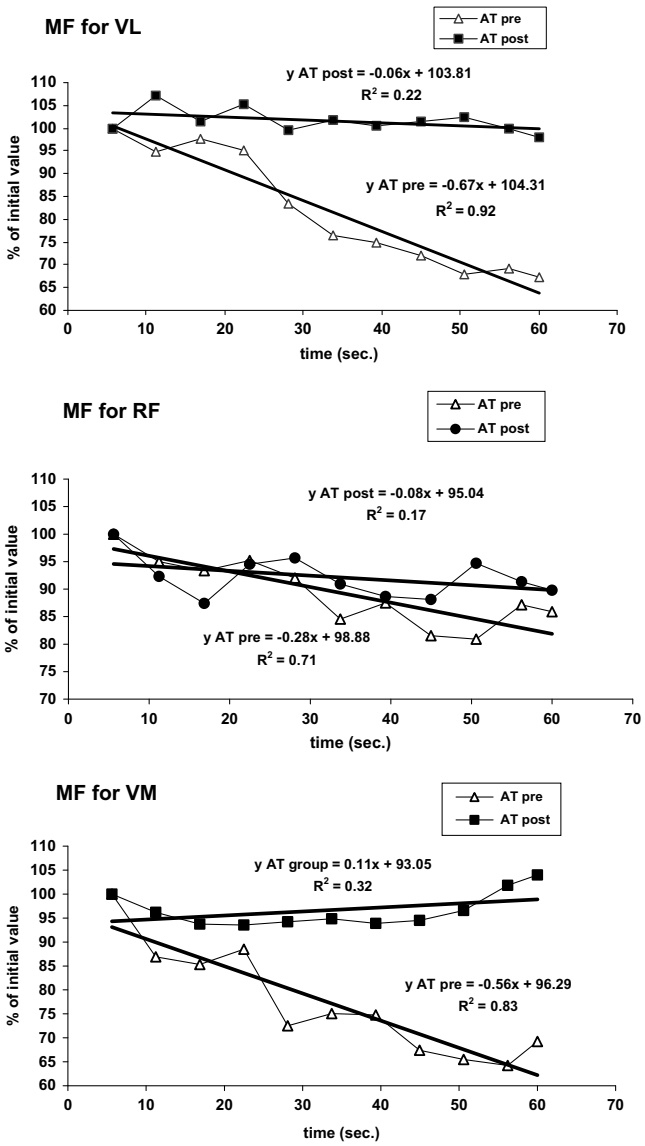

Fig. 2. MF modifications for the vastus lateralis, rectus femoris and vastus medialis in the AT group after training (vs. pre values). Mean values (in % of initial value) fitted out with a linear regression. MF = median frequency and AT = aerobic training.

increased significantly in the CAR group for the RF (CAR group pre vs. post: $p < 0.001$). MF kinetics modifications (MF decrease rate) after training for the CAR and AT groups are illustrated in Figs. 1 and 2.

4. Discussion

The present study was performed to assess the effects of combined aerobic and resistance training vs. aerobic training alone on cardiopulmonary function and skeletal muscle fatigue using SEMG in men with CHD. We firstly showed that only combined aerobic and exercise training improved cardiopulmonary function in our CHD men group. More importantly, we showed that exercise training (either combined aerobic resistance or aerobic training alone) could have favourable impact on SEMG fatigue indexes (MF decrease rates) and neuromuscular alterations in CHD men. To our knowledge, there was no previous study in CHD patients on the effects of exercise training interventions on skeletal muscle fatigue and how two different exercise-training modalities would improve neuromuscular alterations. As expected, combined aerobic and resistance training had more impact on skeletal muscle fatigue indexes, with a reduction of MF decrease rates for the three muscles vs. only two muscles in the aerobic group, with a slightly higher isometric force output (+5.8%) performed during isometric endurance time in the combined aerobic and resistance group compared to aerobic group.

Our data indicate that our combined aerobic and resistance training group demonstrated no improvement in the isometric quadriceps strength (+11.6%, $p = 0.2$) and isometric endurance time. However, equivalent isometric endurance time (69 s vs. 66 s) was performed with a 5.8% higher isometric force output in the combined group. Because isometric endurance time is force output-dependent, this may suggest an improvement of muscle endurance in the combined group; unfortunately, we did not test our patients at the pre-training level 50% of the MVC. Leg strength has been shown to increase by 20–56% with combined aerobic and resistance training (Pierson et al., 2001; Santa-Clara et al., 2002; Stewart et al., 1998), a finding superior to our results. However, these differences are probably due to first, the small patients sample ($n = 8$, in each group) and secondly, to differences between force testing (isometric) and resistance training mode (dynamic movement on the Banc de Koch). In the aerobic trained group, our results are consistent with two previous studies showing a lack of effect of aerobic training on leg strength in heart failure patients (Barnard et al., 2000; Kiilavuori et al., 2000), but disagrees with others where leg strength improved after aerobic training in CHD (Pierson et al., 2001; Stewart et al., 1998) and CHF patients (Delagardelle et al., 2002).

In the combined aerobic and resistance group, higher RMS values for the vastus lateralis ($p < 0.001$) was noted with training. This result agrees with previous training studies in healthy subjects (Creer et al., 2004; Lucia et al., 2000) and patients with chronic obstructive pulmonary disease (Gosselin et al., 2003), where RMS values increased for the vastus lateralis after endurance training. An increase in RMS values for other muscle groups has also been demonstrated with isometric, combined aerobic and resistance training or electrical stimulation training in healthy subjects (Gabriel et al., 2001; Hakkinen et al., 2003; Marqueste et al., 2003). We recently documented neuromuscular abnormalities in men with CHD, with lower RMS values for the vastus lateralis compared to healthy controls (Gayda et al., 2005). This result suggests that after combined training, patients were able to recruit additional motor units and/or to synchronize the motor units already active to compensate for muscle fatigue. Additional motor units recruited were presumably mainly composed of slow twitch fibers (Creer et al., 2004; Gosselin et al., 2003; Lucia et al., 2000), and CHD patients may have lower initial motor unit recruitment at the start of isometric contraction. In the aerobic training group, we have noted a significant decreased in RMS for the RF after training. Lower RMS values in the aerobic group can be explained by less activation of the RF during the fatigue test relative to the vastus lateralis and medialis muscles, and/or to alternations strategies in quadriceps agonist muscles (Gayda et al., 2005). A prior study in CHF patients (Kiilavuori et al., 2000) suggest no modification in RMS profiles after aerobic training.

After training, the combined aerobic and resistance group showed higher mean MF values and lower decrease rates in all muscle groups studies relative to pre values (Fig. 1). In the aerobic training group, lower MF decrease rates were shown only for the vastus lateralis and medialis muscles. Those changes were not accompanied by increased quadriceps strength in both groups. However, isometric endurance time was performed at 5.8% higher force output in the combined group. Skeletal muscle fatigue is often associated with a diminution of the MF during submaximal isometric contraction in healthy subjects (Gayda et al., 2005; Marqueste et al., 2003; Portero et al., 2001), and in CHF and CHD patients (Buonocore et al., 1998; Gayda et al., 2005). Our data are consistent with previous studies, where a diminution of MF or mean power frequency was shown during cycle ergometer testing after endurance training in athletes (Creer et al., 2004; Lucia et al., 2000), COPD patients (Gosselin et al., 2003) and after resistance training or electrical stimulation in healthy subjects (Marqueste et al., 2003; Portero et al., 2001). Two major mechanisms have been given to explain this shift: a decrease of the muscle fiber conduction velocity and the changing statistics of the motor units patterns (Gabriel et al., 2001; Giannesini et al., 2003; Marqueste et al., 2003). A decrease in muscle fiber conduction velocity and spectral parameters (e.g., MF) has been associated with an accumulation of anaerobic metabolism by-products including lactate, protons, H_2PO^{4-}, potassium and ammonium (Gayda et al., 2005; Giannesini et al., 2003; Portero et al., 2001). In both trained group, the lower rate of MF decline was probably due to lower and/or later accumulation of metabolites after training (Ades et al., 1996; Cottin et al., 1996; Ohtsubo et al., 1997) but also to modifications of motor units function (motor unit synchronization and later recruitment of fast type II fibers) during isometric contraction. However, a better impact on MF indexes are observed in the combined aerobic and resistance training group.

Exercise tolerance improved only for the combined aerobic and resistance group in agreement with one study (Stewart et al., 1998). However, others studies showed no or equal improvement for ventilatory threshold values, and equivalent peak VO_2 improvement (Pierson et al., 2001; Santa-Clara et al., 2002) for both training modalities. A possible explanation for an improved exercise tolerance in the combined aerobic and resistance group may be the increases in leg strength in this group, allowing patients to reach higher level of oxygen uptake during cycling. Magnetic resonance spectroscopy studies have shown reduced muscle oxidative capacity in CHD and CHF patients during exercise (Adamopoulos et al., 1999; Cottin et al., 1996), and that training improves skeletal oxidative metabolism in these same populations (Ades et al., 1996; Cottin et al., 1996; Ohtsubo et al., 1997). The improvements witnessed in VO_2 at ventilatory threshold (close to significativity for peak VO_2, $p = 0.08$) in the combined aerobic and resistance group may in part be the result of improved skeletal muscle metabolism. The lack of a training effect in the aerobic training group could be explained by several factors

including small sample size, the low training intensity (VT), short training period and a poor responders patient to training (Vanhees et al., 2004).

Our study has several limitations, our study possessed a small trained patients sample size. Nevertheless, we were able to detect significant differences in the variables studied between combined aerobic and resistance and aerobic training groups. Secondly, we did not possess a CHD control group in our study to evaluate the effect of combined aerobic and resistance training relative to usual standard care. Finally, the study included men only and cannot be generalized the female CHD population, additionally, a larger patients sample is needed to generalized our results to CHD population. A difference between testing contraction mode (isometric) and training contraction mode (dynamic) must also been considered as a limitation.

In conclusion, combined aerobic and resistance training was found more effective for improvement of exercise tolerance and skeletal muscle fatigue in men with CHD compared to aerobic training alone. Skeletal muscle fatigue and neuromuscular alterations in CHD patients can be improved with aerobic training, but greater benefits are obtained when resistance is added to training. SEMG is a useful tool to assess the efficacy of cardiac rehabilitation programs on skeletal muscle function. Further studies however are needed in a large CHD population to generalize our results (particularly in CHD women). It would be important to know if clinical improvements (i.e. quality of life, performing daily activities) will be in relationship with the diminution of skeletal muscle fatigue. Additionally, it would necessary to explore neuromuscular adaptations and skeletal muscle fatigue modifications after training but with a testing in dynamic mode (isokinetic contractions) and also in other muscle groups such as upper body muscles. Finally, it would be necessary to explore relationship between skeletal muscle metabolism modifications and neuromuscular modifications after training in CHD population.

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