Effects of Cardiac Rehabilitation on Atrial Wave in Patients After Myocardial Infarction

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Cardiac rehabilitation (CR) can improve cardiac hemodynamic performance in patients after myocardial infarction (MI). Little evidence is provided concerning the consequences of CR on atrial wave duration, and less is known about the link between pre-arrhythmogenic patterns and the cardiovascular performance improvement in these subjects. Twenty-six patients, post-MI 0 to 7 days, underwent a complete CR cycle and a signal-averaged electrocardiogram (SAECG) for the evaluation of atrial activation parameters (group 1) to appreciate if physical training can promote parallel improvement in cardiovascular and intra-atrial conduction parameters. A control group of 24 well-matched nonischemic subjects (group 2) was chosen for data comparison. Resting heart rate ($p<0.01$) and resting double product ($p<0.01$) decreased after CR in groups 1 and 2, while diastolic blood pressure at maximal stress was decreased in group 1 ($p<0.01$) with a parallel increase in the time of physical training ($p<0.05$). SAECG parameters of atrial activation were unchanged in group 1 after the comparison and only total atrial duration activation (dA) reached statistical significance (113.3 ±17.2 msec vs 120.8 ±14.2 msec, subjects after CR vs before CR, $p<0.01$). CR could improve intra-atrial activation in subjects after MI, but the consequences of hemodynamic adjustment of the trained heart must undergo a more accurate evaluation to verify if CR can prevent adverse arrhythmogenic complications of MI through cardiovascular performance improvement.

Introduction

Hemodynamic and arrhythmic consequences of ischemic heart disease after myocardial infarction (MI) are clearly demonstrated in the literature.[1-3] Pharmacologic therapy can improve prognosis in these ischemic subjects,[4-6] and physical training can produce better myocardial perfusion through different mechanisms, as demonstrated by several classic observations.[7-10] Another suggestive result can be obtained by the association between improved diastolic function, decreased left ventricular telediastolic pressure and volume in the trained heart; this phenomenon is commonly observed at the end of complete cycles of cardiac rehabilitation (CR).[11] Thus, patients with coronary artery diseases after MI can surely exhibit better cardiovascular performance if submitted to complete physical training; simple and complex arrhythmias are less common in the trained heart.[12,13] On the contrary, little is known about atrial activation change and its link with cardiac hemodynamic profile in the ischemic myocardial

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tissue. A more accurate study of the supraventricular activation pattern can provide useful data for the assessment of a correct prediction of supraventricular arrhythmias in patients after MI.[14-16] A full evaluation of these subjects cannot exclude validated signal-averaged electrocardiography (SAECG) parameters such as late ventricular potentials, useful in predicting ventricular arrhythmias[17,18]; an accurate risk stratification can be obtained with other SAECG measurements, also useful in the evaluation of supraventricular arrhythmogenic patterns. Prolonged atrial activation is actually considered as a significant predictor of supraventricular arrhythmias that are commonly observed in the post-infarction period.[19,20] With regard to ventricular arrhythmias, previous studies clearly demonstrated a reduction in frequency after CR, perhaps due to a modulation of sympathetic response during exercise.[21,22]

The aim of this study was to evaluate the relationship between cardiac hemodynamic performance and atrial wave duration in 26 subjects after myocardial infarction to furnish a useful evaluation of the pre-arrhythmogenic pattern changes after a CR cycle.

Methods

Patients were enrolled from ambulatory and clinical settings with the characteristics shown in Table I. Ischemic patients after MI (group 1) underwent a complete CR cycle (Table II) after an ECG stress test (Bruce symptom-limited protocol) with cycle-ergometry. Heart rate at baseline was controlled at 65% to 70% of maximum heart rate recorded during ECG stress test to 80% to 85% in full-trained subjects. Pharmacologic treatment was started following clinical criteria; aspirin, ace-inhibitors, calcium antagonists, β blockers, diuretics, and nitrates were given to all the patients. Exclusion criteria were left bundle branch block, dangerous ventricular arrhythmias, left ventricular systolic function less than 20%, congestive heart failure (III and IV NYHA classes), unstable angina pectoris, severe aortic stenosis, recent embolic diseases, recent thrombophlebitis, acute infective diseases, serum electrolyte concentration exceeding reference range, and diastolic blood pressure greater than 115 mm Hg without antihypertensive drug therapy. A control group of 24 healthy subjects was selected from our database

Table I. Clinical and anthropometric characteristics of the study population.

Parameter	Group 1	Group 2
Total number	26	24
Males/females	21/5	16/8
Mean age (yr)	57.1 ±9.2	55.3 ±8.5
Body mass index (kg/m^2)	26.3 ±3.1	25.9 ±4.6
Body surface area (dm^2)	177.5 ±8.6	168.6 ±7.6
Q-wave infarction	24	NA
Non-Q-wave infarction	2	NA
Anterior infarction	12	NA
Inferior infarction	10	NA
Lateral, Inferior-lateral infarction	4	NA

NA: not applicable.

Table II. Cardiac rehabilitation protocol.

Maximum number of sessions	20
Maximum time of exercise training (min)	30
Week frequency of sessions	3
Mean load (W)	10
Basal load (W)	35 (0.7 kg/m at 50 Hz)

(group 2). No patients had a history of atrial fibrillation or echocardiographic evidence of atrial dilation. All the patients gave written informed consent and the study was approved by the local ethical committee. Arterial blood pressure (WHO guidelines) and heart rate were measured at baseline and at the end of each CR step. Oxygen consumption, ventilation, and respiratory quotient were monitored during CR[23] (Tables III, IV). Double product was also calculated from the previous data to provide an indirect index of oxygen consumption. SAECG was performed at enrollment and after CR by MAC 1 Marquette connected with a microcomputer for signal processing and analysis; the following SAECG parameters were calculated[15] (Table V, Figure 1): (dA) total atrial activation duration (msec); (dAP+) interval between the beginning of atrial activation and maximum positive peak (msec); (dAP–) interval between the beginning of atrial activation and maximum negative peak (msec); (dB) interval between the beginning of atrial activation and the midpoint between maximum positive and maximum negative peak (msec); (dAP+P–) interval between maximum positive and maximum negative peak (msec); (aPP) peak-to-peak amplitude (mV); (aP+) maximum positive peak amplitude (mV); and (aP–) maximum negative peak amplitude (mV). A frequency band of 100 to 300 Hz at 1 mm/0.5 μV amplification and mean noise level of 0.2 μV was considered. Values are expressed as mean ± standard deviation (SD) as an index of dispersion. Statistical computation was performed with Student's *t* test and the linear regression test when appropriate.

Results

Hemodynamic performance and the variation in oxygen consumption are shown in Table III for the studied subjects. Heart rate at rest (84.4 ±16.5 beats/minute before CR vs 71.4 ±12.0 beats/minute after CR), indirect (double product: 11746.4 ±2383.8 before CR vs 9449 ±1812.5 after CR) and direct (oxygen consumption: 37.4 ±6.2 before CR vs 28.1 ±5.5 after CR, mL/kg/min^{-1} $p < 0.001$) indexes of aerobic capacity are decreased after CR (see Table III). Ventilation and respiratory quotient decreased after physical conditioning but they did not reach statistical significance (see Tables III, IV). Systolic and diastolic blood pressure after controlled stress were decreased in the ischemic subjects (without statistical significance) with a appreciable increase in the time of exercise training (see Table III). Intermediate parameters of atrial activation (see Table V) did not reach statistical significance except for total atrial duration of activation (dA) which was decreased after CR (120.8 ±14.2 before CR vs 113.3 ±17.2 msec after CR).

Limitations of the Study

A relative limitation was represented by the small sample size, but the selection criteria were very strict to reduce possible confounding factors. Hemodynamic measurements were executed in few patients who gave the written informed consent, so the statistical strength of this sample was too low to be helpful. Also myocardial reperfusion was obtained in few subjects. Another possi-

Table III. Hemodynamic and oxygen consumption parameters during ECG stress test before and after CR (group 1).

Parameter	Before	After	p
Heart rate at rest (beats/min)	84.4 ±16.5	71.4 ±12.0	<0.01
Systolic blood pressure at rest (mm Hg)	138.5 ±12.1	132.1 ±13.5	NS
Diastolic blood pressure at rest (mm Hg)	86.4 ±6.9	84.2 ±6.0	NS
Double product at rest (mm Hg × beats/min)	11746.4 ±2383.9	9449 ±1912.5	<0.01
Maximal heart rate (beats/min)	141.4 ±19.3	141.2 ±20.9	NS
Maximal systolic blood pressure (mm Hg)	201.4 ±23.4	199.2 ±27.0	NS
Maximal diastolic blood pressure (mm Hg)	92.8 ±6.9	88.5 ±6.9	<0.01
Oxygen consumption (mL/kg/min^{-1})	37.4 ±6.2	28.1 ±5.5	<0.001
Ventilation (L/min^{-1})	95.34 ±15.6	97.33 ±13.4	NS
Respiratory quotient (VCO_2/VO_2)	1.20 ±0.2	1.15 ±0.3	NS
Maximal double product (mm Hg × beats/min)	28165.7 ±4270.2	28906.4 ±6025.5	NS
Time of exercise training (min)	6.1 ±2.3	8.0 ±3.2	<0.05

NS: not significant.

Table IV. Hemodynamic and oxygen consumption parameters during ECG stress test before and after CR (group 2).

Parameter	Before	After	p
Heart rate at rest (beats/min)	82.2 ±6.3	66.4 ±10.0	<0.01
Systolic blood pressure at rest (mm Hg)	118.5 ±11.1	111.1 ±9.5	NS
Diastolic blood pressure at rest (mm Hg)	80.8 ±2.9	77.2 ±7.9	NS
Double product at rest (mm Hg × beats/min)	10231.4 ±1123.3	9122 ±917.1	<0.01
Maximal heart rate (beats/min)	134.4 ±12.3	130.2 ±17.9	NS
Maximal systolic blood pressure (mm Hg)	179.4 ±19.4	177.2 ±25.0	NS
Maximal diastolic blood pressure (mm Hg)	88.8 ±3.9	86.5 ±7.7	<0.01
Oxygen consumption (mL/kg/min^{-1})	35.9 ±4.2	29.2 ±4.5	<0.001
Ventilation (L/min^{-1})	97.22 ±13.6	96.88 ±12.4	NS
Respiratory quotient (VCO_2/VO_2)	1.18 ±0.3	1.16 ±0.2	NS
Maximal double product (mm Hg × beats/min)	26188.7 ±3870.2	25606.4 ±5127.3	NS
Time of exercise training (min)	6.3 ±2.5	8.8 ±1.2	<0.05

NS: not significant.

Table V. Atrial activation parameters after a CR complete cycle.

Parameter	Group 1			Group 2		
	Before	After	p	Before	After	p
dAP+ (msec)	54.7 ±13.1	68.1 ±11.2	NS	53.9 ±12.5	63.3 ±10.1	NS
aP+ (mV)	10.5 ±2.1	10.2 ±3.1	NS	11.8 ±1.1	10.0 ±2.2	NS
dAP– (msec)	53.6 ±29.8	65.7 ±32.3	NS	51.9 ±21.1	62.8 ±28.7	NS
aP– (mV)	9.0 ±2.6	7.0 ±3.4	NS	7.5 ±2.6	7.0 ±3.4	NS
aPP (mV)	19.5 ±4.0	17.2 ±6.6	NS	18.8 ±3.0	17.5 ±4.6	NS
dPP (msec)	16.6 ±9.1	21.1 ±13.4	NS	13.9 ±7.1	18.8 ±12.7	NS
dA (msec)	120.8 ±14.2	113.3 ±17.2	<0.01	115.2 ±12.6	114.9 ±11.8	NS
dB (msec)	53.9 ±20	67.4 ±20	NS	55.7 ±18.1	65.9 ±17.3	NS

NS: not significant.

ble limitation of the study consisted of the lack of a third control group of MI patients with no CR, but the hemodynamic improvement of the atrial wave duration obtained with medical treatment was not the primary focus of this study.

Discussion

Controlled physical training can increase myocardial vascular distribution through a higher capillary density and increased coronary lumen diameters. In experimental settings, physical training increased the number of collateral coronary branches in pigs with MI.[24] Ferguson and coworkers[25] and Connor and colleagues[26] demonstrated a significant increase in coronary collateral branches evaluated by coronary arteriography in subjects with coronary artery disease who were submitted to CR programs. Indirect information is provided about the possible role of controlled physical training in the maximal coronary flow improvement and better oxygen distribution in the ischemic myocardial muscle. Other authors observed a higher double product and ischemic

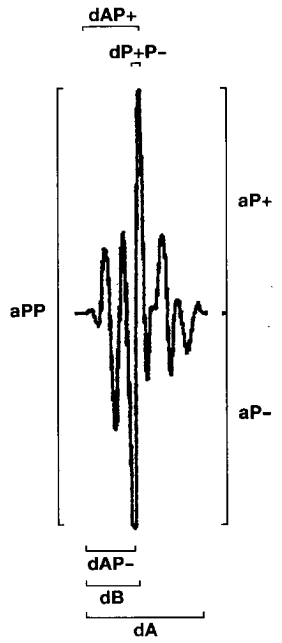

Figure 1.
Atriogram wave.

signs disappeared at the same or at higher double product value after CR in ischemic patients.[27] Myers and co-workers noted a significant improvement in ischemic score at myocardial scintigraphy with 210-Thallium in a heterogeneous group of subjects after 1 year of CR.[28] All these preliminary experimental evidences suggest that a personalized and controlled CR program can increase myocardial perfusion and reduce myocardial oxygen demand.[29] Furthermore, recent reports show that exercise rehabilitation after MI can influence heart rate variability parameters through an improvement of parasympathetic drive and a reduction of sympathetic activity in hypertension and normotension.[30] This effect on sympathetic and parasympathetic balance can significantly influence the arrhythmogenic status of ischemic patients. Also from our preliminary study, data indicative of better myocardial perfusion (lower double product at rest and after controlled effort, lower heart rate at rest and after ergometric load) seem to emerge from the ischemic subjects; but the potential link with atrial activation can be considered only indirectly. Furthermore our evidence seems to suggest a substantial positive effect of CR on the reduction of atrial activation duration. Only dA reached statistical significance after the computation; the other intermediate measurements of atrial activation were decreased after CR, showing a tendency to decrease the atriogram wave amplitude (see Figure 1). The better oxygen utilization and the improvement of cardiovascular performance, shown by direct and indirect indexes, are commonly observed after CR cycles but further investigations are needed to link morphologic changes in atrial dimensions with hemodynamic improvements and electrical consequences in the ischemic trained heart. Previous observations[31] pointed out that chronic ischemic heart disease after MI can lead to a prolonged atrial activation duration in ischemic patients in comparison with normal controls. Transient nonspecific alterations of atrial activation during angina pectoris were described in our studies[32]; in acute MI[33] or during transient ischemic heart disease[34]; other similar modifications were checked as a possible consequence of myocardial perfusion alterations. In fact atrial activation occurs in a thin myocardial wall, hence minimal hemodynamic variations can produce electrophysiologic changes[35] that 12-lead standard electrocardiography can reveal only casually. Furthermore, left ventricular diastolic function is closely linked to reduced myocardial nutrition,[36,37] so a better myocardial perfusion, induced by controlled physical training in ischemic patients, could determine a normalization of diastolic function and also could provide a significant improvement in intraatrial pressures and volumes. These hemodynamic parameters are important in the evaluation of left ventricular diastolic function, but the indirect link between atrial activation detected by SAECG and increased left ventricular hemodynamic performance after CR requires further investigation in both experimental and clinical settings.

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