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Original Article

Exercise Training May Enhance Functional Capacity and Improve Heart Rate Impairment in Coronary Artery Disease Patients with Chronotropic Incompetence

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This study investigated whether coronary artery disease patients with chronotropic incompetence (CI) can significantly improve their functional capacity after exercise training. Forty-two men (mean age: 55.3±9.3yr) underwent coronary revascularization and without beta-blockade participated in a three-month outpatient exercise training program. Patients were separated into a CI group (n=18) and a non-CI group (n=24) based on whether they could reach 85% of the age-predicted peak heart rate (HR). Chronotropic incompetence was defined as failure to achieve 85% of the age-predicted maximal HR. Bicycle exercise workouts were conducted three times weekly. The exercise duration was 30 minutes per session included 5 minutes warm up and 5 minutes cool down, and the exercise intensity was adjusted to the subject's ventilatory threshold (VeT). Graded exercise tests with gas analysis were conducted before and after training to evaluate changes in cardiorespiratory function. Before training, the CI group displayed lower peak oxygen uptake ($\dot{V}O_{2peak}$), HR and work rate than the non-CI group ($p<0.01$). After training, the $\dot{V}O_{2peak}$ increased from 19.5±3.4 to 25.1±5.5 $mL \cdot kg^{-1} \cdot min^{-1}$ in the CI group, while it increased from 22.0±3.9 to 25.4±5.4 $mL \cdot kg^{-1} \cdot min^{-1}$ in the non-CI group. At VeT, the CI group also displayed greater increase in $\dot{V}O_2$ than the non-CI group. In conclusion, although pre-training $\dot{V}O_{2peak}$ was lower for the CI group than the non-CI group, both groups can achieve a similar level of functional capacity. CI thus appears not to be a limiting factor for improvement during cardiac rehabilitation. (Tw J Phys Med Rehabil 2007; 35(4): 189 - 196)

Key Words: coronary artery disease, cardiac rehabilitation, maximal oxygen uptake, exercise

INTRODUCTION

Chronotropic incompetence (CI), an attenuated heart

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rate response at peak exercise, has been shown to predict mortality and coronary artery disease (CAD) risk.[1-4] Although CI is easily observed during exercise testing, it is even a stronger independent predictor of death than angiographic severity of CAD.[5] In a follow-up study of patients undergoing angiography, McNeer et al.[6] found those with a maximal HR below 120 bpm displayed a 40% mortality rate at 4 years compared to 10% for those with maximal HR exceeding 160 bpm. Additionally, Lauer et al.[7] reported that CI was a strong predictor for cardiac events (death, myocardial infarction, unstable angina, or revascularization), with the relative risk for patients with CI, was 2.47 compared to patients without CI.

Patients with CI have lower exercise capacity than those without CI,[8] and low exercise capacity is a strong predictor of mortality among patients with known or suspected coronary disease.[9] Further, exercise training can enhance physical capacity and reduce the risk of death. Kavanagh et al.[10] reported that a 1 $mL \cdot kg^{-1} \cdot min^{-1}$ increase in $\dot{V}O_{2peak}$ was associated with a 9% improvement in survival. To our knowledge, few studies have reported the impact of CI on the effect of exercise training in coronary patients. If training can reverse HR impairment and increase $\dot{V}O_{2peak}$, it not only indicates that exercise improves functional capacity, but also implies that exercise may reduce mortality among patients with CI. Accordingly, the objective of this study was to investigate whether patients with CI can achieve sufficient functional recovery during cardiac rehabilitation.

MATERIALS AND METHODS

Patients

Patients after successful coronary artery bypass graft surgery (CABG) or percutaneous transluminal coronary angioplasty (PTCA) were enrolled in this study. The exclusion criteria included 1) beta-blockade, 2) postoperative angina, 3) congestive heart failure, 4) significant myocardial ischemia, 5) unstable dysrhythmias, and 6) uncontrolled diabetes and/or hypertension. Subjects participated in a three-month outpatient rehabilitation program. Graded exercise tests with gas analysis were conducted before and after training to evaluate the changes of cardiorespiratory function. Patients were separated into a CI group and a non-CI group according to the result of baseline exercise test, and CI was defined as failure to achieve 85% of the age-predicted maximal HR. An institutional committee for the protection of human subjects approved the study protocol, and informed consents were obtained from all subjects.

Exercise Protocol

All patients participated in an outpatient cardiac rehabilitation 6-8 weeks after discharge. Exercise sessions were performed three times a week for three months. The exercise included 5 minutes warm up, 20 minutes of bicycle exercise combined with treadmill walking, and 5 minutes of cool down. The exercise intensity was prescribed according to the ventilatory threshold (51-59% of maximal heart rate reserve) and the Borg's Scale[11] rating of perceived exertion 11 to 13 (fairly light to somewhat hard). The exercise session was supervised by a physical therapist, and the heart rate and blood pressure were monitored during the exercise. A home-based walking program with similar intensity of the outpatient program was also recommended to all patients.

Equipment and Measurement

An exercise testing was performed 4-6 weeks after PTCA or CABG. Symptom-limited, graded exercise testing was conducted no sooner than two hours after breakfast. Continuous electrocardiographic monitoring and breath-by-breath analysis of expired gas were performed during the exercise testing. Before exercising, blood pressure was measured by the standard cuff method in each subject after sitting quietly for five minutes. Afterwards, the subjects used a cycle ergometer (Ergotests, Erich Jaeger, Wuerzburg, Germany) to perform a symptom-limited exercise test. Exercise was performed with the patient in the upright position, and continued until the appearance of symptoms (i.e. fatigue, angina, undue dyspnea, claudication and cerebral symptoms) or signs (2 mm ST depression over resting, significant ectopic activity, inappropriate BP response). Inappropriate BP responses included the following criteria: 1) a drop in systolic BP of 20 mmHg or more, 2) systolic BP drop below the value obtained before testing, 3) an increase in systolic BP to 260 mmHg or an increase in diastolic BP to 115 mmHg.[12] The workload was 10 watts for the first three minutes, and then increased in 10-watt increments every minute. The

pedaling cadence was maintained at 60±10 rpm. Expired gas was analyzed by an automated system (System 2001, Medical Graphics Corporation, St Paul, MN). Exercise cardiorespiratory parameters included heart rate (HR), oxygen uptake ($\dot{V}O_2$), carbon dioxide production ($\dot{V}CO_2$), oxygen pulse, minute ventilation ($\dot{V}_E$), respiratory exchange ratio (RER), ventilatory equivalent for O_2 ($\dot{V}_E/\dot{V}O_2$), ventilatory equivalent for CO_2 ($\dot{V}_E/\dot{V}CO_2$), end tidal PO_2 ($P_{ET}O_2$), end tidal PCO_2 ($P_{ET}CO_2$), and rate pressure product (heart rate times systolic blood pressure; RPP). A blood sample was drawn from the antecubital vein five minutes after the termination of exercise for quantitation of lactate.

The ventilatory threshold (VeT) was determined by at least two of the following criteria: 1) the $\dot{V}_E/\dot{V}O_2$ began to increase systematically without a corresponding increase in the $\dot{V}_E/\dot{V}CO_2$,[13] 2) the $P_{ET}O_2$ began to increase without a decrease in the $P_{ET}CO_2$,[13] 3) departure from linearity for minute ventilation.[14] Two independent observers with experience in cardiopulmonary exercise testing determined the VeT.

Data Analysis

An unpaired *t*-test was performed to analyze the between-group differences of the pre-training physiological data. Meanwhile, post-training differences between groups were also analyzed by the unpaired *t*-test. The paired *t*-test was used to compare the cardiorespiratory variables of the pre-training and the post-training measurements within each exercise group. A two-sided p value of less than 0.05 was considered statistically significant for all tests. All data are presented as means ± standard deviations.

RESULTS

This study enrolled 42 subjects who had undergone revascularization and completed a three-month exercise program (Table 1). Patients were selected by convenience, and were separated into a CI group or a non-CI group based on their peak HR at the exercise testing just before training. The cut-point for patient assignment was set at 85% of the age-predicted maximal HR. The CI group included 18 patients (8 PTCA, 10 CABG); the Non-CI patients included 24 patients (11 PTCA, 13 CABG). Table 1 lists the baseline data of both groups. Age, body height, weight, resting ejection fraction, and days of training were similar for both groups. No major cardiovascular complications occurred in either group during training, and no subjects received repeated revascularization in this period.

Table 2 lists the cardiorespiratory parameters before and after training. On pre-training examination, the CI group showed a 12.8% higher in $\dot{V}O_{2peak}$ than the CI group (22.0±3.9 vs. 19.5±3.4 $mL \cdot kg^{-1} \cdot min^{-1}$, $p < .05$). Additionally, the non-CI group also had a higher HR and rate pressure product (RPP). At the VeT, the non-CI group also displayed a significantly higher $\dot{V}O_2$ and HR than the CI group ($p < .05$).

In the post-training examination, the non-CI and CI groups did not differ significantly at peak exercise in terms of $\dot{V}O_2$, RPP and work rate (Figure 1). The only differences between the two groups were in HR and oxygen pulse ($p < .05$). After training, the between-group difference in peak HR was reduced, and 11 patients of the CI group (61%) exceeded 85% of the age-predicted maximal HR at peak exercise. At the VeT, the non-CI group also displayed lower oxygen pulse than the CI group ($p < .05$), but post-training $\dot{V}O_2$ was similar for both groups.

After three months of training, both groups displayed significant improvements over parameters of gas exchanges (Table 2). In the peak exercise, the non-CI group displayed a 15.5% increase in $\dot{V}O_{2peak}$, from 22.0±3.9 to 25.4±5.4 $mL \cdot kg^{-1} \cdot min^{-1}$. Peak work rate also increased from 113±22 to 133±27 watt. Meanwhile, the CI group achieved a 28.7% increase in $\dot{V}O_{2peak}$, from 19.5±3.4 to 25.1±5.5 $mL \cdot kg^{-1} \cdot min^{-1}$. Peak work rate also increased from 107±16 to 139±23 watt. Peak HR significantly increased in the CI group but not in the non-CI group. Peak oxygen pulse also significantly improved in both the CI and non-CI groups, by 1.3 and 1.5 $mL \cdot beat^{-1}$, respectively. Figure 1 displayed the changes of cardiorespiratory parameters over time in both groups.

At the VeT, the CI group showed greater increases in $\dot{V}O_{2peak}$ and work rate than the non-CI group. Oxygen pulse at VeT also increased significantly in both groups. Although peak HR failed to reach the age-predicted maximal HR in the CI group, the mean peak respiratory exchange ratios reached 1.15 in both tests, indicating of maximal or near-maximal effort.

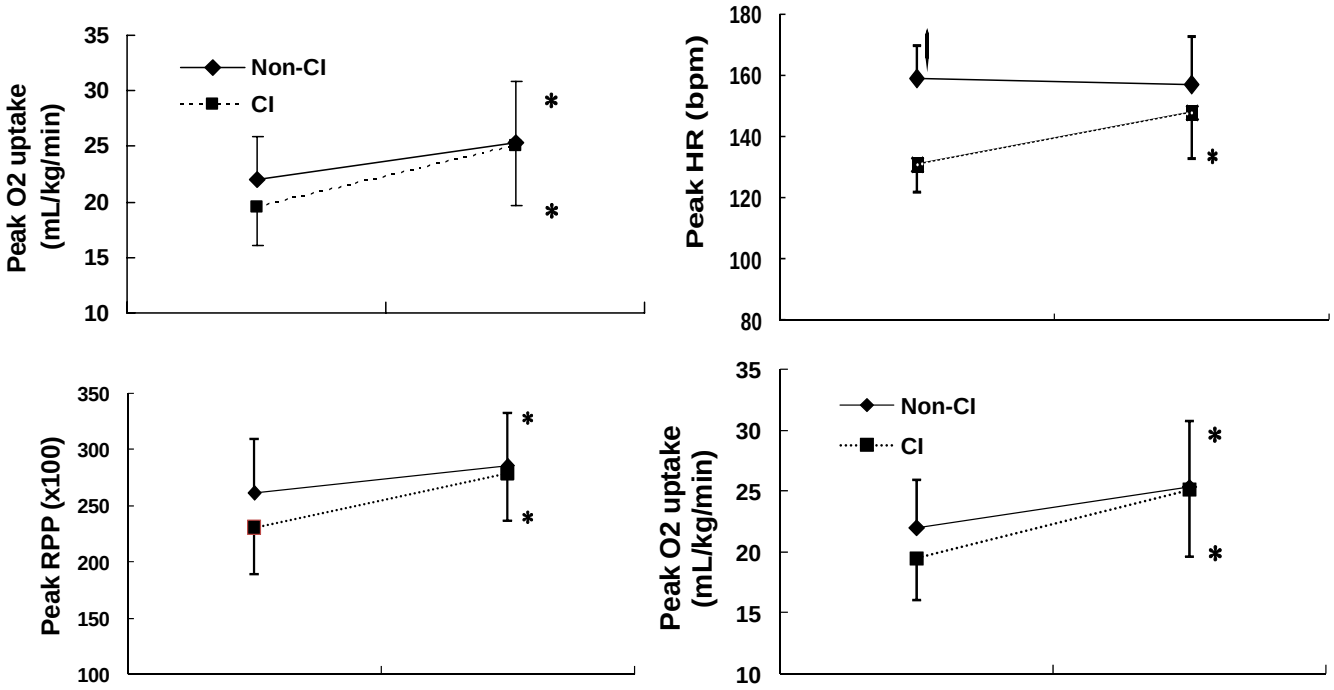

Figure 1. Cardiorespiratory variables at the peak exercise before and after training (◆ non-CI group, ■ CI group, values are mean±SD. * < .05, within-group difference from pre-training to post-training; + p < .05, pre-training between-group difference).

Table 1. Baseline Demographic and Clinical Characteristics of Patients

	CI (*n*=18)	Non-CI (*n*=24)
Age (yr)	55.8±8.5	54.9±9.9
Body Height (cm)	168.4±6.7	167.0±5.6
Body weight (kg)	70.8±9.9	67.6±9.1
Resting systolic BP (mmHg)	120±16	117±15
Resting diastolic BP (mmHg)	77±16	86±9
Resting heart rate (bpm)	71±12	82±12
Resting ejection fraction (%)	58.4±12.7	60.1±13.2
Resting rate pressure product ($\times 10^2$)	97±20	116±25*
Myocardial infarction	12 (66.7%)	15 (62.5%)
Hypertension	10 (55.6%)	14 (58.3%)
Diabetes	2 (11.1%)	3 (12.5%)
Hyperlipidemia	10 (55.6%)	13 (54.1%)
Days of training	105±34	109±31

CI: Patient with chronotropic incompetence

Non-CI: patients without chronotropic incompetence

* P < .05

Table 2. Cardiorespiratory Function of Patients Before and After Training

	CI (*n*=18)			Non-CI (*n*=24)		
	Pre-training	Post-training	% change	Pre-training	Post-training	% change
Peak exercise						
$\dot{V}O_{2peak}$ (mL·kg^{-1}·min^{-1})	19.5±3.4	25.1±5.5*	28.7	22.0±3.9†	25.4±5.4*	15.5
HR_{peak} (beats·min^{-1})	131±9	148±15*	13.0	159±11†	157±16‡	-1.3
Peak O_2 pulse (mL·beat^{-1})	10.5±1.7	11.8±1.6*	12.4	9.2±1.8†	10.7±2.4*‡	16.3
RER	1.16±0.08	1.15±0.06	-0.9	1.16±0.08	1.15±0.07	-0.9
$\dot{V}E_{peak}$ (L·min^{-1})	56.7±11.8	69.4±10.9*	22.4	57.9±12.4	63.9±14.5	10.4
WR_{peak} (watt)	107±16	139±23*	29.9	113±22	133±27*	17.7
RPP (×10^2)	230±41	278±42*	20.9	261±48†	285±47*	9.2
BP(mmHg)	177/95	189/91		167/90	183/93	
Ventilatory Threshold						
$\dot{V}O_2$ (mL·kg^{-1}·min^{-1})	12.7±3.0	15.0±3.6*	18.1	13.3±2.4	15.2±3.3*	14.3
HR (beats·min^{-1})	99±10	102±15	3.0	121±19†	110±15*	-9.0
O_2 pulse (mL·beat^{-1})	8.9±2.3	10.4±2.1*	16.9	7.5±1.7†	9.0±2.1*‡	20.0
$\dot{V}E$ (L·min^{-1})	27.4±6.8	30.9±7.4	12.8	27.1±5.0	28.2±6.0	4.1
WR (watt)	49±13	69±18*	40.8	53±15	63±19*	18.9

CI: Patient with chronotropic incompetence; Non-CI: patients without chronotropic incompetence

$\dot{V}O_2$: O_2 consumption; RER: respiratory exchange ratio; $\dot{V}E$:minute ventilation; WR: work rate; RPP: rate pressure product

* $p < .05$, within-group difference from pre-training to post-training

† $p < .05$, pre-training between-group difference

‡ $p < .05$, post-training between-group difference

DISCUSSION

Chronotropic incompetence is common for CAD patients who underwent exercise stress testing.[15] The patients who had a low peak HR response during exercise thereby decreasing the oxygen uptake, either at peak exercise or at the VeT.[8] However, Keteyian et al.[16] reported that 6 months of exercise training could induce a significant increase in peak HR and $\dot{V}O_{2peak}$ among heart failure patients with CI. Our data also showed that a 3-mon exercise program significantly enhanced peak HR and $\dot{V}O_{2peak}$ in CAD patients in spite of CI.

To our knowledge, this is the first study comparing the effect of exercise training on revascularized patients with CI and without CI. In our earlier work, the $\dot{V}O_{2peak}$ of normal sedentary men with a similar age was 26.3±4.4 mL·kg^{-1}·min^{-1}.[17] Compared with the normal data, the CI group thus displayed 6.8 mL·kg^{-1}·min^{-1} decrease in $\dot{V}O_{2peak}$ ($p<.01$), while the non-CI group only displayed 4.3 mL·kg^{-1}·min^{-1} decrease in $\dot{V}O_{2peak}$ ($p<.01$). Because both groups had similar ventricular function in terms of the left ejection fraction at rest, the difference in their fitness level might be partially attributable to the lower peak HR in the CI group. The greater decrease of $\dot{V}O_{2peak}$ in patient with CI may influence to their adaptation in daily activities. Furthermore, lower $\dot{V}O_{2peak}$ may predict higher mortality in patients with CAD. According to Kavanagh et al. study,[10] $\dot{V}O_{2peak}$ values < 15, 15-22, and >22 mL·kg^{-1}·min^{-1} yielded multivariate adjusted hazard ratios of 1.00, 0.62, and 0.39 for cardiac deaths, respectively. Given the apparent effectiveness of exercise training in reversing HR impairment, it is important to encourage patients with CI to participate in rehabilitation programs.

After three months of cardiac rehabilitation, patients with CI displayed a mean increase of 17±5 bpm in peak HR. An increase in peak HR may contribute to an in-

crease in peak cardiac output and an increase in $\dot{V}O_{2peak}$. Therefore, the CI group showed a greater increase in $\dot{V}O_{2peak}$ than the non-CI group (5.6 vs. 3.4 $mL \cdot kg^{-1} \cdot min^{-1}$), and exercise training allowed both groups to achieve a similar level of $\dot{V}O_{2peak}$. Although pre-training $\dot{V}O_{2peak}$ was lower in the CI group, the potential of improvement through training was to be greater. Furthermore, the peak RPP increased significantly in both groups, but RPP tended to increase more in the CI group. Because RPP is closely related to myocardial oxygen uptake and coronary blood flow,[18] this result reflected that exercise training might increase more myocardial oxygen uptake in patients with CI at peak exercise. Although improvement of $\dot{V}O_{2peak}$ should not be interpreted as a surrogate end points for mortality rate, reversing CI and increasing both peak HR and $\dot{V}O_{2peak}$ are favorable physiological responses and have potential benefits for long-term outcomes.[16]

VeT was used initially to assess endurance during exercise at submaximal work rates in individuals with CAD.[19] VeT is an objectively detectable breakpoint at an individually moderate exercise level that can be used to characterize CI. At VeT, a physiological HR response was approximately 220 −age −50 bpm, and a deviation exceeding 10 bpm below this physiological value characterized CI.[8] Using this equation, the age-predicted HR at VeT in our subjects should be 115±14 bpm, but the mean HR at VeT was significantly lower in the CI group than the non-CI group (99±10 vs. 121±19 bpm). When assessing endurance, VeT is considered a sensitive indicator of physical performance. Our data showed that the pre-training $\dot{V}O_2$ at VeT was 12.7 $mL \cdot kg^{-1} \cdot min^{-1}$ (3.6 METs) for the CI group and 13.3 $mL \cdot kg^{-1} \cdot min^{-1}$ (3.8 METs) for the non-CI group. Meanwhile, our earlier study found that $\dot{V}O_2$ at VeT was 14 $mL \cdot kg^{-1} \cdot min^{-1}$ (4 METs) in normal sedentary men of a similar age.[17] After three months of training, Both groups displayed a significant increase in $\dot{V}O_2$ at VeT, and attained a similar value approximated 4.3 MET. Notably, even a small increase in $\dot{V}O_2$ at VeT is beneficial to cardiac patients, because it can improve endurance in daily activities at submaximal work rates.

Much of what has been termed CI is related to early termination of exercise owing to angina pectoris. In this study, no patient displayed angina during baseline exercise testing, thus the reason of CI might be attributed to myocardial dysfunction, deconditioning and muscle weakness. After training, 61% of patients with CI could increase their peak HR to a normal range (> 85% of the age-predicted maximal HR). Consequently, CI in patients after revascularization appears partially reversible, and those patients could obtain physiological benefits from exercise training as their non-CI counterpart. In patients remained chronotropic incompetent after training, a small but significant increase in maximal oxygen uptake was observed.

This study has several limitations. First, a precise definition of CI is lacking, and the age-predicted HR is also debating. Chronotropic incompetence is generally defined as the inability of heart rate to increase in proportion to exercise and metabolic requirements, or to achieve 80-90% of age-predicted maximal HR. However, most authors take 85% of the age-predicted maximal HR as a cut-off point.[5,7,15,20] Second, subject's motivation may be an important confounding factor during maximal exercise testing. To confirm that the subjects were highly motivated to reach maximal volitional level, we checked that the respiratory exchange ratio (RER) > 1.10 and rate of perceived exertion (RPE) 17-19 (hard to very hard) at peak exercise. The average RER achieved 1.16 in both groups, and it implied that CI did not hinder subjects to attain peak exercise. Finally, the study population was limited to men owning to difficulties in enrolling female patients, future studies should enroll female participants.

In conclusion, this study has shown that a three-month exercise program could favorably enhance cardiorespiratory function in CAD patients, regardless of the presence of HR impairment during exercise. Both groups could achieve a similar fitness level as normal sedentary men, both in maximal exercise and ventilatory threshold. Despite having lower baseline exercise capacity, the CI group displayed greater improvement of $\dot{V}O_{2peak}$ compared to the non-CI group. It appears that CI is not a limiting factor for exercise training in CAD patients after revascularization.

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運動訓練可促進節律性不足之冠狀動脈疾病患者的體能並改善運動時的心跳率

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本研究探討心臟復健是否可增進節律性不足的冠狀動脈疾病患者之體能。42 位接受過冠狀動脈繞道術或氣球擴張術後的男性患者（平均年齡 55.3±9.3 歲，且沒有服用乙型阻斷劑），參與三個月的心臟復健。根據在運動訓練時的心跳反應，將患者分爲節律性不足組（18 人）及節律性正常組（24 人）。節律性不足的定義是患者在運動時無法達到年齡預估最大心跳率的 85%。復健訓練每週進行三次，每次 30 分鐘，包括暖身及緩和運動各 5 鐘，運動強度則以換氣閾值爲準。在訓練計劃前後，各作一次進階最大運動測驗，並做氣體分析，以評估患者心肺功能的變化。在訓練開始前，節律性不足組的最大攝氧量較節律性正常組爲低（$p<0.01$）。訓練完成後，節律性不足組的最大攝氧量增加了 28.7%，從 19.5±3.4 進步至 25.1±5.5 $mL \cdot kg^{-1} \cdot min^{-1}$（$p<0.01$）；節律性正常組的最大攝氧量則增加了 15.5%，從 22.0±3.9 進步到 25.4±5.4 $mL \cdot kg^{-1} \cdot min^{-1}$（$p<0.01$）。在換氣閾值時，節律性不足組的攝氧量也有增加，且較節律性正常組爲大。本研究發現雖然節律性不足組的最大攝氧量在訓練前較低，訓練後仍可達到節律性正常組的體能水準。因此，節律性不足並非限制心臟復健體能進步的因子。（台灣復健醫誌 2007；35(4)：189 - 196）

關鍵詞：冠狀動脈疾病(coronary artery disease)，心臟復健(cardiac rehabilitation)，最大攝氧量(maximal oxygen uptake)，運動(exercise)

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