Cardiac Rehabilitation Manual

Second Edition

Josef Niebauer
Editor

Springer

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Preface

Secondary Prevention: A Second Chance to Make Inevitable Lifestyle Changes

Cardiac diseases are not only the leading causes of death in industrialized countries but also on the rise in many emerging countries worldwide. They also induce considerable harm to survivors and often lead to severe and irreversible physical and neurological disabilities. Despite the fact that there is no cure, a lot can be done to prevent coronary artery disease, i.e., primary prevention, or to slow the progression of the disease, i.e., secondary prevention. Both can be achieved by tackling the panoply of modifiable risk factors, which have been identified to be amenable to lifestyle changes.

As a matter of fact, according to current guidelines, a long list of risk factors ought to be treated first by lifestyle changes before medical therapy is considered or initiated. These risk factors include:

- Physical inactivity
- Smoking
- Hypercholesterolemia
- Hypertriglyceridemia
- Low HDL cholesterol
- Arterial hypertension
- Hyperglycemia

As an example, physical inactivity has been recognized to be among the strongest predictors of morbidity and mortality for both otherwise healthy persons and already affected patients. Often, however, medical therapy has to be initiated concomitantly to avoid further vascular damage and thus to halt or slow the progression of atherosclerosis. All doctors have received excellent training in choosing the right medication for their patients. We even have sales representatives from companies approach us on a regular basis who further try to provide us with up-to-date – albeit not always unbiased – information. The only effective treatment that no one is offering us or our patients is exercise training. Neither do we receive information on dieting. We, thus, have to set out to try and find current and reliable information ourselves: an obvious deficit that this book is trying to reduce.

At the time that we start medical therapy, we also tell our patients to change their lifestyle.

But what exactly does this mean?

What are the lifestyle changes that they are now being expected to make?

And above all, can we provide our patients with an infrastructure that really helps them to deviate from current unhealthy behavior?

Now it not only becomes very demanding for our patients but also for us, which is why many doctors, as shown in the EUROASPIRE trials, do not even recommend lifestyle changes; many because they lack detailed knowledge on how to implement it. Such training however is a prerequisite in order to convince a patient to say goodbye to many of his or her unhealthy habits. Indeed, the vast majority has been leading an unhealthy lifestyle all their lives and may not wish to change this. Usually, there is a narrow window right after a cardiac event during which patients are amenable to our advice. This is the time to initiate changes that nobody can afford to miss. At the same time, these changes have to be agreed on with a patient who we see as partner on a lifelong journey of lifestyle changes, since otherwise patients may not necessarily stick to their good intentions in the long run. They need encouragement but also an infrastructure that ought to be available to them to actually modify their lifestyle. Indeed, all our countries lack out-patient cardiac rehabilitation facilities that would provide a convenient and adequate infrastructure for our patients to not only initiate but also to provide a base for lifelong compliance with current guidelines. Such facilities have to be close to home, since otherwise it is not possible to attend exercise, nutritional, psychological, and other classes several times a week for an extended period of time. Only then, however, can long-lasting lifestyle changes be introduced into our patients' daily lives. Such facilities are especially warranted for those who want to return to work and wish to be on sick leave for as little as possible.

The network of institutions of ambulatory cardiac rehabilitation facilities has to increase, but also general hospitals have to start to establish ambulatory rehabilitation programs, so that patients get a fair chance to actually change their lifestyle. It is not enough if hospitals only concentrate on revascularizing patients, but do little or nothing to ensure optimal reduction in morbidity and mortality thereafter.

If we fail to do this, then we are in a situation that can be compared to prescribing drugs in a place where there are no drug stores.

But even if we were to get better infrastructure, even then we doctors have to improve our skills. Unfortunately, too few physicians have experience in cardiac rehabilitation, which comes as no surprise as it has never been taught in medical school, internist, or subspecialty training. It is only those of us who have chosen to work in cardiac rehabilitation centers or hospitals who know what to recommend and how to prescribe exercise training and other healthy treatment choices. I am no exception to this rule and had to learn the hard way by initiating training groups in various medical centers, what is best for our patients. Also several of the coauthors not only pursued a career in cardiac rehabilitation but got to where they are by trial and error. It is with this background and understanding that we hope to provide knowledge and advice to those who would like to learn more about cardiac rehabilitation.

After all, it becomes obvious that cardiac rehabilitation has not only come to stay but will become increasingly important, since it is a cost-effective treatment option. As a matter of fact, the number and quality of cardiac rehabilitation programs have to increase, which in turn will require an increasing number of skilled staff. More doctors have to be trained adequately to receive the skills that are required to effectively recommend appropriate measures to patients, let alone to actually guide or accompany them on this lifelong journey. It is thus the aim of this book to provide doctors with in-depth but still hands-on information to quickly grasp the leading problems of our patients and to design or recommend appropriate programs.

In this book, we have refrained from presenting exciting and exotic cases, but rather concentrate on the vast majority of our everyday patients in ambulatory or in-hospital cardiac rehabilitation.

All authors were or still are members of the nucleus of the working group on cardiac prevention and rehabilitation of the European Society of Cardiology. Their expertise not only spans the whole spectrum of cardiac diseases but also contributes various aspects of challenges in cardiac rehabilitation from centers throughout Europe. It is our wish to make a little but significant contribution to further excel the knowledge of our readers by writing this book which at first addresses general issues of cardiac rehabilitation, until it then teaches how to treat patients by focusing on individual patients with specific but very common cardiac conditions.

At first, this book will cover general principals of exercise testing and training as well as nutritional and psychological support. After these fundamentals of cardiac rehabilitation have been laid out in appropriate depths, chapters follow on the most common cardiac diseases. Cases include symptomatic coronary artery disease with or without diabetes, myocardial infarction or revascularization, and cases of heart failure in rather stable conditions, with or without cardiac devices. Our book will then be wrapped up with cardiac rehabilitation in patients with congenital cardiovascular diseases, valvular surgery, and peripheral arterial disease with claudication.

Contents is not presented in textbook style, but rather taught on representative clinical cases. Each chapter focuses on a particular patient and discusses pros and cons of the most appropriate diagnostic tools and treatment options. It is thus designed to be a practical guide for doctors and geared to help them guide their patients. Medical therapy, which most doctors will be very familiar with, has been addressed from the perspective of primary or secondary prevention and is of course in line with current guidelines of our national and international medical societies and associations. A therapeutic option that has long and that still is terribly neglected will receive the attention that it deserves – physical exercise training. Data on reduction of morbidity and mortality but also on improvement in quality of life are so striking that neither we nor our patients can afford to not use this poly-pill. Most of the modifiable risk factors of cardiovascular diseases can be treated by these lifestyle changes. Nevertheless, in the real world, treatment strategies concentrate almost solely on pharmaceutical interventions, neglecting the beneficial effects of heart-healthy diets and exercise training programs. For managing both long- and short-term risk, lifestyle changes are the first-line interventions to reduce the metabolic risk factors. Indeed, the importance of physical activity and heart healthy nutrition

cannot be overestimated. This will be highlighted in several chapters. Primary and secondary prevention of cardiovascular diseases need to focus on all modifiable risk factors and implement pharmaceutical therapy wherever appropriate.

Exercise training has to become an integral part of it. It is inacceptable that it is only integrated into the daily routine by a minority of patients. Further cardiac rehabilitation programs have to be installed and doctors need to be trained to be able to refer and treat patients at this stage in their disease history appropriately. We strongly believe that this book will add to the knowledge of our readers and that it will enable them to better guide their patients on a lifelong journey of primary and secondary prevention.

Salzburg, Austria
January 2017

Josef Niebauer MD, PhD, MBA

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Part I

Introduction to Cardiac Rehabilitation

General Principles of Exercise Testing in Cardiac Rehabilitation

1

Miguel Mendes

1.1 Introduction

Before the admission into a cardiac rehabilitation program (CRP), every patient is submitted to a clinical assessment which must include a medical consultation, an evaluation of LV function (usually by echocardiography), a maximal exercise test (ET) limited by symptoms, and blood tests to evaluate the CVD risk factor profile. In special cases, after the clinical assessment, the patients need further diagnostic tests like a 24 h Holter monitoring, an imaging technique to study perfusion or coronary anatomy or bypass grafting [1–5].

The ET is a very important part of this clinical assessment performed before admission and repeated at the end of the CRP phase, because it gives indispensable data regarding functional capacity and information regarding the hemodynamic adaptation to maximal and submaximal levels of exercise (HR and BP), residual myocardial ischemia, and cardiac arrhythmias induced or worsened by exercise and permits the identification of the training heart rate (THR) for the aerobic training [2–4].

Besides the objective parameters mentioned above, the ET is very important from the psychological point of view for many patients and partners, because they realize that the patient usually has a better functional capacity than they could predict. In the follow-up period, the ET is very useful to detect or confirm eventual clinical status changes which occurred during the program, update exercise prescription intensity, measure the gains obtained after the CRP, and perform global prognostic assessment.

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1.2 What Kind of Exercise Test

Cardiopulmonary exercise test (CPX) is the ideal ET to be used in all kinds of patients in the setting of a CRP [6]. Although it is almost mandatory to use it in heart failure patients [3], due to its higher cost, more complicate delivery, and interpretation, it is usually replaced in many CR centers, mainly in CAD patients with normal or near-normal LV function, by the standard ET which is more widely available and familiar to most cardiologists.

During the CPX, peak VO_2, ventilatory thresholds, VE/VCO_2 slope, and O_2 kinetics are measured beyond all the parameters recorded in the standard ET, like maximal load reached, HR and BP changes from rest to maximal exercise, and, during recovery, the eventual arousal of symptoms like angina pectoris or ECG abnormalities (ST changes or arrhythmias) [7].

Considering the parameters obtained from the CPX, peak VO_2 is the most important because it is the gold standard for functional capacity and it was identified as the strongest prognostic parameter in CVD [8–13]. Peak VO_2 is also very important to prescribe exercise intensity since continuous moderate aerobic training, the classical modality, is performed at a percentage of peak VO_2 ranging from 50 to 70 % [16].

The first and second ventilatory thresholds (VT1 and VT2), which are expected to occur at submaximal level during the CPX, are independent of motivation, contrary to peak VO_2. Due to this fact, they are considered good indicators of the training effect of the program, if they occur at a higher percentage of VO_2 max. The determination of the ventilatory thresholds is also useful to calculate the training intensity for moderate continuous aerobic training, which must start at the HR attained at the level of VT1 and increased to HR reached at the VT2 moment [14, 15].

1.3 How to Perform an ET in the Setting of a CRP

A fully equipped exercise lab test, including at least one ergometer (bike or treadmill), an ECG system with several exercise protocol options, and an emergency cart, together with a well-trained and experienced staff (cardiologist and technician), must be available to perform the exercise tests in the setting of a CRP [7, 17, 18].

After the team welcome of the patient immediately before the ET, he must be asked about his usual exercise tolerance, in order to estimate his maximal functional capacity and to choose the test protocol. The test must be programmed in a way that the patient's physical exhaustion or high-grade fatigue will be attained around 10±2 min of exercise [19]. In case of test interruption before 8 min, a less intense protocol must be used and a steeper one in the reverse situation to evaluate correctly functional capacity.

It's important to rule out a possible recent worsening of clinical status, which may oblige to postpone the test and the interruption of the regular cardiovascular medication before the test. The exercise tests integrated in a CRP must be performed under the patient's usual medication, and one must try to schedule the test for a moment of the day similar to the foreseen moment of the CRP sessions.

The respect of these two issues, medication and moment of the day, will prevent that the drug effect and consequently the patient's protection will be different during

ET and the exercise sessions of the CRP. This is very important, for example, in patients under beta-blockers which are usually taken in the morning and whose effect can decrease during the day, namely, in the afternoon. If the THR was calculated after a test performed at a certain time of the day, it may happen that it will be difficult to reach if the test was performed late in the day and the session is in early morning, where the beta-blocker effect is more intense or will be easily surpassed in late-afternoon exercise sessions if the ET took place in the morning.

The decision to stop exercise during the ET is crucial to quantify exercise tolerance accurately. If no medical contraindications to continue the effort are present, like major ST changes, serious arrhythmias, blood pressure (BP) drop, or hypertensive response, and if the patient seems to be relatively comfortable, the exercise period must be interrupted only upon patient request, based on the perception that he reached his maximal exercise capacity or feels a major discomfort, like claudication, eventually related to peripheral arteriopathy or orthopedic disease [17, 18].

The exercise period must never be stopped based on the attainment of any level of predicted maximal HR, due to the large variability of peak HR among subjects. This procedure, followed in some centers, prevents an accurate quantification of exercise tolerance and maximal HR. When THR is calculated based on the chronotropic reserve, it is crucial to not stop the test without reaching maximal HR because the peak HR may not be the maximal attainable HR during the ET.

After confirming that the patient is in the desired situation in terms of clinical status and medication, it's time to choose the ergometer and the protocol to be used.

If both of the usual possibilities are available (bicycle or treadmill), the choice must be made or performed taking into consideration which ergometer will be more used for aerobic training, the patient's preference, and the clinical staff familiarity.

Regarding the protocol choice, two issues must be considered:

1. The patient's predicted exercise tolerance
2. Type of protocol (ramp type or with small or large increments between stages)

Considering the type of increments of the ET protocol, there is a preference for ramp or short increment (around 1 MET) protocols [20, 21], because the error of the functional capacity estimation will be lower, in the case that respiratory gas analysis will not be performed. The ergometer must also be taken into consideration because the load is more accurately determined on a bike than with the treadmill, since the treadmill calibration is more difficult and especially if the patient grasps the handrails during effort, diminishing the oxygen demand needed to perform the test (Tables 1.1 and 1.2) [7, 17, 18].

Table 1.1 Stationary bike most used protocols [22, 23]

	Load (watts)			Duration (min)		
Designation	Start	Increase	Peak	Stage	Total	Peak estimated METs
Balke (men)	50	25	175	2	12	9.5
Balke (women)	25	25	150	2	12	8.3
Astrand	25	25	150	3	18	8.3

Table 1.2 Treadmill most used protocols [24–26]

Designation	Estimated METs		
	At 8 min	At 9 min	At 12 min
Naughton	4	NA	6
Balke-Ware[a]	5	NA	8
Modified Bruce	NA	7	10
Bruce	NA	10	13

NA not applicable

[a]Usually not acceptable for old people and frail patients, because it has a constant speed (5.47 km/h), which is not tolerated by most patients

1.4 When to Do It

The ET must be performed at the admission of the CRP in the majority of program participants, sometimes in the middle of a phase when it seems that the patient's clinical status changed or THR is inadequate due to the acquisition of a better exercise tolerance as a consequence of exercise training and at the end of each phase to measure the final functional capacity [2, 4, 16].

Patients recently submitted to cardiac surgery are usually admitted in the CRP, without performing an ET, because they may face physical limitations that advise to postpone the test for 2–4 weeks. During these early weeks, the patients are involved in respiratory and global physiotherapy and may even start exercising in a stationary bike or on the treadmill, below a THR of 100 or 120, respectively, if they are or not under β-blocker medication, till they reach a satisfactory exercise tolerance that enables them to be submitted to the ET, after what an individualized THR will be calculated [2].

1.5 How to Report the ET in the Setting of a CRP

A standard ET must be reported not only in terms of the presence or absence of myocardial ischemia but also about enlightening the global prognosis, as it is shown in the Table 1.3.

The test must be reported not only in terms of myocardial ischemia but also on functional capacity, chronotropic index, HR recovery, BP, and ventricular or supraventricular arrhythmias.

Despite having informed the patient at the beginning of the ET about the need to spontaneously report the occurrence of any unexpected symptom, namely, angina or a disproportionate grade of dyspnea or fatigue, it's also advised to ask periodically, for example, at the end of each stage and at the moment of ST depression occurrence, if the patient is experiencing angina and what is his perception of exercise intensity (Borg scale). During the exercise period, it is also recommended to record every minute a full ECG in order to define accurately the eventual moment after which ST segment depression reaches 1 mm and 60 or 80 ms after the J point, the so-called ischemic threshold.

Table 1.3 Parameters to describe in the ET report in the CR setting [18, 27]

1. *Exercise capacity*
(a) Test duration and reason to stop the exercise
(b) In a classical ET, estimate exercise tolerance, as ratio between the achieved and the predicted METs, calculated by the following equations:
(i) Men: Predicted METs = 14.7–0.11 × age
(ii) Women: Predicted METs = 14.7–0.13 × age
(c) In a CPX, measure exercise tolerance and use Weber classification and percentage of predicted VO_2 max
Classify functional capacity below normal if lower than 85 % of the predicted value
2. *Heart rate*
HR at rest, at the end of each stage, at the moment of the ischemic threshold, ventricular or supraventricular arrhythmias starting, abnormal BP (drop or hypertensive response) at peak exercise and in recovery at 1, 3, and 6 min
Classify chronotropic evolution during exercise as:
Normal, if peak HR value is above 85 % of the predicted value (220 bpm minus age), for individuals not under β-blocker or above 62 % under β-blocker
Abnormal, if below the mentioned values
Classify chronotropic evolution during recovery as
Normal, if HR difference between peak exercise and min 1 > 12 on protocols where there is an active recovery (slow walking or pedaling) or >18 bpm, if exercise is immediately stopped at peak effort
Abnormal, if below the mentioned values
3. *Blood pressure*
Classify blood pressure evolution as
Normal, if SBP increases ~10 mmHg per MET and there is no change or a small drop is found in DBP. It's acceptable to find a drop <15 mmHg at peak exercise
Hypertensive, if SBP reaches values >250 or >DBP 120 mmHg
Insufficient, if SBP increases <30 mmHg
4. *Ischemia*
Classify the test as *negative, positive, equivocal*, or *inconclusive* for myocardial ischemia, taking into consideration the presence or absence of angina or ST depression/elevation induced during the test, in the exercise or the recovery period, according to the criteria defined in the guidelines
Use the ST/HR index, the ST rate-recovery loops, and/or the ST/HR slope to increase the accuracy of the diagnosis of ischemia
Grade ischemia as severe, moderate, or low level, taking into consideration the precocity of appearance, magnitude of ST changes, time until normalization in the recovery period, association with limiting angina, BP fall, chronotropic deficit, or ventricular arrhythmias
Identify clearly the HR of the ischemic threshold, because the THR to be observed during the exercise sessions must be 10 bpm below this value for safety reasons
5. *Prognosis*
Assess globally the prognosis, considering functional capacity, ST/HR index, chronotropic response, HR recovery, ventricular ectopy during recovery, and ST/HR slope, which are implicated in global and cardiovascular mortality and events
6. *Aerobic training intensity*
Classically, THR is calculated as the HR at (50), 60–70 % of HR reserve or (50), or the HR reached at 60–70 % of VO_2 reserve or at HR of the VAT level, respectively, if the patient was submitted to a standard ET or to a CPX [16]

(continued)

Table 1.3 (continued)

More recently, important changes in terms of determination of THR occurred, due to the adoption of new modalities of exercise training like high-intensity exercise training (HIIT) and to the change of concept of the submaximal exercise thresholds. Today, the concept of only one threshold, previously called ventilatory anaerobic threshold (VAT), was abandoned, and it was adopted the concept of two submaximal thresholds, the first and the second ventilatory thresholds identified, respectively, by the nadir of the curves of the O_2 and VCO_2 equivalents [14, 15]
In the case of continuous moderate aerobic training, exercise training must start at the level of HR attained by the subject at the level of the first ventilatory threshold (LVT1) and move till the HR attained at the level of the second ventilatory (LVT2)
After the seminal paper of Wisloff [28] and coworkers, a new paradigm emerged: HIIT. In this case, exercise intensity is prescribed at up to 95 % of HR attained during the ET for periods of 4 min, intercalated by 3 min periods of not so intense training at 50–75 % of peak exercise

Ischemia is diagnosed by the occurrence of angina and/or definitive ST changes on the exercise or in the recovery period. In order to increase the diagnostic accuracy of the ET, ST changes must be interpreted considering ST/HR index, which must be superior to 1.6 μV/bpm, and rate-recovery loops that are suggestive of myocardial ischemia if there is a counterclockwise rate-recovery loop.

Functional capacity is probably the most important finding after an ET as it is the best parameter to predict all-cause mortality. When peak VO_2 is not measured, it can be estimated by the ratio between the estimated METs achieved at the last stage of the ET and the predicted value given by the following formula: Predicted METs = 14.7–0.11 × age or 14.7–0.13 × age, respectively, for men and women. To allocate the estimated METs of a stage, the patients must exercise at least 1 min at that stage. If he was not able to do it, his maximal METs attained will be the ones estimated for the previous completed stage.

The Duke score tries to put together the presence/absence of ischemia and functional capacity and classifies the patients in low, intermediate, and high categories of risk, according to the value of the score.

Chronotropic index, HR recovery, and ventricular arrhythmias predict increased/decreased risk of death if they are negative or positive.

1.6 How to Assess Exercise Training with a Standard ET or a CPX

At the end of a CRP phase, the ET or the CPX must be repeated to be compared with the test performed at the phase start, in order to document eventual gains provided by the program.

These gains must be observed in terms of maximal and submaximal functional capacity, ischemic threshold, exercise-induced or exercise-worsened arrhythmias, heart rate, and blood pressure evolution during the exercise and the recovery periods [2, 6, 16].

To make a correct comparison between both tests, they must be performed under the same medication, at the same time of the day, and using the same ergometer and

protocol. If any revascularization procedure, like a PCI, is performed, the medication is changed between the tests, or if the ergometer or the protocols are also different, a direct comparison of both tests is impossible.

1.6.1 Standard ET

If exercise training is successful, the standard ET will usually show in the second test:

(a) Higher duration/load attained
(b) Lower levels of HR and BP at each stage and an early normalization of HR during recovery
(c) Starting of ischemia later during the test, although at the same or higher double product
(d) Lower frequency and complexity of ventricular arrhythmias, in the exercise or recovery periods

Functional capacity can be estimated for each patient in terms of METs (metabolic units of oxygen consumption: 1 MET = 3.5 ml/kg/min) considering the oxygen consumption previously known to be inherent to the highest stage attained at peak exercise, if the patient was able to keep this stage more than 1 min. If the test was stopped before staying 1 min or more in the last stage, the attributed estimated METS must be those predicted for the previous completed, since usually it takes, at least, 1 min to stabilize oxygen consumption in each exercise protocol stage.

Functional capacity must also be classified regarding the predicted values for the same age, gender, and physical activity status, provided by several equations.

The maximal load reached by the patients can also be considered as a measure of functional capacity, especially when a stationary bike is used. In a treadmill, due to the body weight dislocation effect and the walking, the peak load values are less accurate.

The estimation of aerobic capacity by the standard ET is not very accurate, since it usually overestimates the load, namely, in the case of patients and old people and when treadmill protocols with high increment protocols are used, like the Bruce protocol.

1.6.2 Cardiopulmonary Exercise Test

The CPX allows the best identification of maximal aerobic capacity because peak VO_2, the gold standard for exercise capacity, is directly measured "breath by breath" during the entire test. Due to some variability, the values should be determined by calculating the rolling average of each period of 20–30 s [29].

Peak VO_2 is the most used parameter to evaluate the CRP benefit. In case of doubt that the CPX is a maximal test, one must specially look at VO_2, HR, and

Table 1.4 How to assess the training effect with an exercise test [7, 17, 18]

Standard ET	Cardiopulmonary exercise test
Test duration, maximal load, and estimated METs	The same parameters as in standard ET, plus:
Presence or absence of ischemia	Peak VO_2
HR at rest, at each stage, at peak exercise, and on recovery	VO2 and HR at VAT
Blood pressure at rest, at each stage, at peak exercise, and on recovery	O_2 kinetics in the recovery period
Ischemic threshold: HR, double product, and load	Peak RER
Grade of myocardial ischemia, in terms of ST normalization, ST depression morphology	VE and breathing reserve
Ventricular arrhythmias	VE/VCO_2 slope

respiratory exchange ratio (RER) and RPE at peak exercise level. VO_2 and/or HR must fail to increase significantly despite load further increments; RER and RPE must be, respectively, equal or over 1.10 and 8/10 at peak [30].

Recently, the terminology of the events observed at submaximal level during a CPX was changed. Now, two thresholds are recognized instead of the only one, the formerly designed ventilatory anaerobic threshold (VAT), presently called the first VT (VT1). Also, the formerly designated respiratory compensation point is called now the second ventilatory threshold. These thresholds are defined, respectively, as the nadir points of the curves of the O_2 and CO_2 equivalents, which have a U shape form during the exercise period. These equivalents are, respectively, the ratios of O_2 and CO_2/ventilation [14, 15].

VT1, formerly designated by VAT, can also be calculated by the V slope method and defines the end of the period where exercise intensity is not perceived by the individual as difficult to be performed. Between VT1 and VT2, exercise intensity is perceived as moderate and after surpassing VT2 as very intense and difficult to maintain for a few minutes.

To overcome the limitations of peak VO_2, the VO_2 attained at the VT1 can be used to evaluate the training effect, because it is independent of patient motivation and expresses better the patient capacity to perform daily life activities.

In cardiac patients, peak VO_2 and VO_2 at the VT1 increase between 7 and 54 % after a period of some weeks of exercise training, although the average increase is usually around 20–30 % [31–33].

VE/VCO_2 slope, which evaluates ventilatory efficiency, one of the most important parameters for prognosis assessment in CHF, is also expected to decrease as a demonstration of a favorable exercise training period (Table 1.4) [34, 36].

Compare pre- and post-exercise tests, performed at the same time of the day, under the same medication and protocol.

1.7 Clinical Cases

Case #1

Male, 41 years old

Apparently healthy till 20th of April 2009 when he suffered an anterior myocardial infarction. Tobacco smoking, obesity, and psychological stress were identified as risk factors for CVD in this case: he smoked one pack a day during 25 years and has a BMI of 30.6 (99 kg of weight and 180 cm height). His BP, blood cholesterol, and glucose levels were normal.

He was submitted to primary PCI of LAD (middle portion) that was totally occluded by a thrombus. The PCI was performed within 2 h of symptoms and was very successful, with the exception of the occurrence of a right thigh hematoma related to the femoral puncture, which obliged him to rest in bed for a week. No other lesions were found in the coronary arteries, and LV function was near normal.

He was discharged from the hospital on the fifth day after ACS under ASA, clopidogrel, ramipril (2.5 mg, od), bisoprolol (2.5 mg, od), and pravastatin (40 mg, od).

When he started to go out of bed and to move around 1 week after hospital discharge, he felt dizziness, nausea, and a thoracic discomfort, different from the one that arose during the ACS, which stopped immediately when he lay down. No pericardial effusion was found on echocardiography. After this he took the initiative to contact our CRP 3 weeks after the ACS.

After a medical consultation and physical examination, where everything seemed to be OK, he was submitted to an ET (Table 1.5; Fig. 1.1).

Table 1.5 Exercise test parameters (case #1)

Stage	Speed km/h	Grade %	METs	HR bpm	SBP mmHg	DBP mmHg	Symptoms	ECG
Rest	0	0	1	75	130	80	No	T wave inversion V1-V4
I	2.7	10	4.6	98	150	80	No	Almost normal
II	4.0	12	7.0	117	175	90	Mild fatigue	Normal
III	5.4	14	10.0	138	200	100	Moderate fatigue	Normal
IV	6.7	16	12.5	150	210	100	Severe fatigue	Normal
Exercise duration: 10 min 20 s								
Rec. 1’	1.5	0		132	200	90	No	Normal
Rec. 3’	0	0		104	190	90	No	Normal
Rec. 6’	0	0		95	170	85	No	Normal

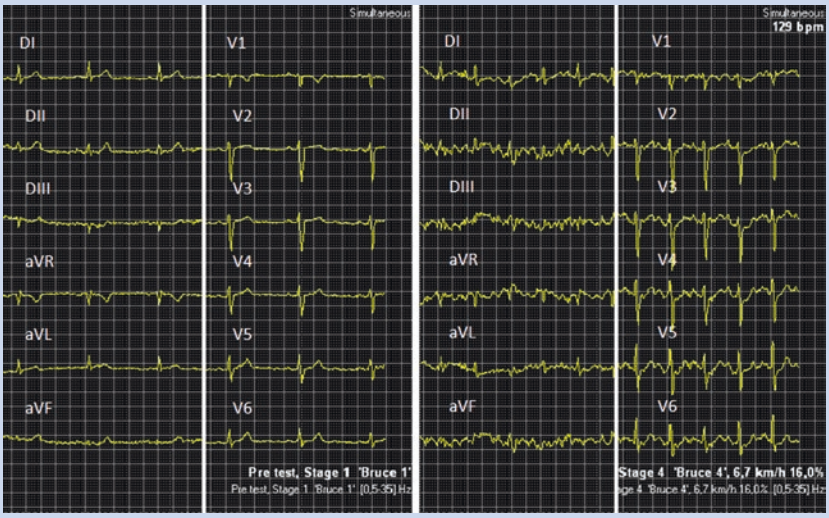

Fig. 1.1 Rest and peak exercise 12-lead ECG (case #1)

Comments ET#1

Confronting the findings of this ET with what is supposed to be found in a normal ET, this patient shows:

1. Good exercise tolerance: 10–20 min exercise duration on the Bruce protocol ~12.5 METs (122 % of the predicted).
2. Normal evolution of HR: from 75 to 150 bpm at peak effort and a drop of 18 bpm on the first minute of an active recovery.
3. Normal increase of SBP: from 130/80 at rest to 210/100 at peak exercise.
4. Hypertensive pattern on DBP: increase from 80 to 100 mmHg
5. No arrhythmias, ST changes, and angina were found.
6. The normalization during the exercise period of the T wave previously present in the rest ECG suggests the presence of stunned myocardium.

Comments

This is a typical case of a low-risk patient for CR, with normal LV ejection fraction, no residual ischemia, no arrhythmias, good exercise tolerance, and a normal adaptation of hemodynamic parameters to maximal exercise.

He was admitted to a formal CRP under medical supervision during some weeks, since he was wishing to start an exercise program and he didn't had any previous physical activity habits.

A THR of 120 bpm was calculated using the Karvonen formula, adding 60 % of his HR reserve [(150 − 75)*0.60 = 45 bpm] to his rest HR (75 bpm): 45 + 75 = 120 bpm [31–34].

Case #2

Male, 54 years old

CVD risk factors: Type 2 diabetes and hypertension.

Assessment performed before admission to CRP on the 4th of October 2004, following a noncomplicated CABG on the 11th of July 2004 and a previous inferior myocardial infarction in an indeterminate date.

He was submitted to complete revascularization, by a triple CABG with LIMA to LAD and single saphenous grafts to the second diagonal and posterior descendent arteries. Three months after surgery, a nuclear perfusion scan requested for routine clinical assessment and identified residual silent ischemia in the inferior wall (Fig. 1.2).

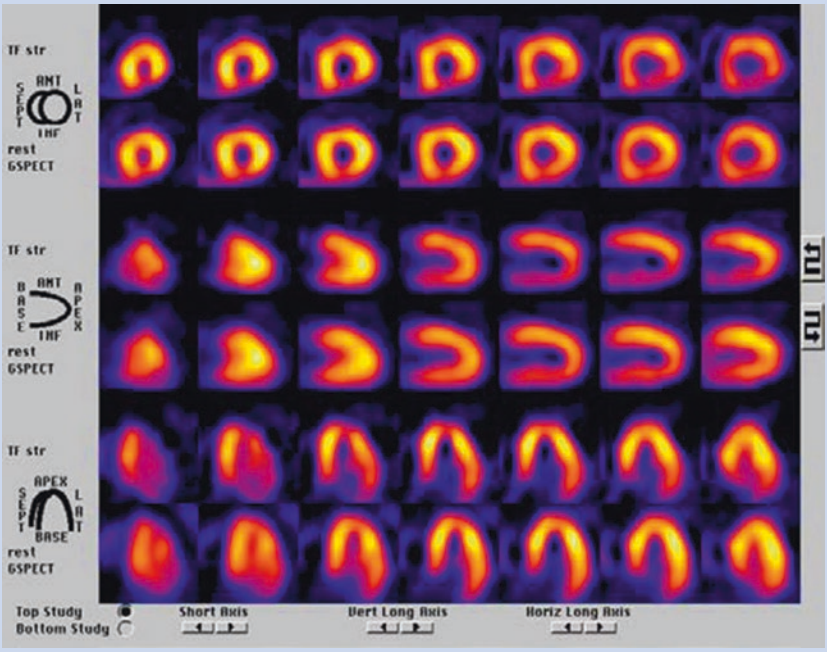

Fig. 1.2 Myocardial nuclear perfusion scan (case #2)

After this test, he was re-submitted to coronary angiography, and it was found that the graft to the posterior descendent artery was occluded and the artery was not amenable to PCI. He had good collateral circulation from the left coronary artery, and the other bypasses were patent, with normal flow. His attending cardiologist decided to keep him on medical therapy and send him to CR.

Before the CRP, he was submitted to an ET, under his usual medication: bisoprolol (5 mg, od), IMN (50 mg, od), losartan (50 mg, od), enalapril (20 mg, od), HCTZ

Table 1.6 Admission exercise test (case #2)

Stage	Speed Km/h	Grade %	METs	HR bpm	SBP mmHg	DBP mmHg	Symptoms	ECG
Rest	0	0	1	59	130	80	No	Q waves on DII, DIII, and aVF
I	2.7	10	4.6	113	150	80	No	No change
II	4.0	12	7.0	131	170	80	Intense fatigue	ST downslope of 1 mm in V5-V6
Exercise duration, 6 min 00 sec; onset of ischemia, at 4 min 00 s with 123 bpm								
Rec. 1'	1.5	0	1	112	140	80	No	ST downslope of 1 mm in V5-V6
Rec. 3'	0	0	1	90	130	80	No	ST downslope of 1 mm in V5-V6
Rec. 6'	0	0	1	82	120	75	No	ST downslope of 1 mm in V5-V6
Rec. 9'	0	0	1	79	120	80	No	Equal to rest ECG

Table 1.7 End of CRP exercise test (case #2)

Stage	Speed Km/h	Grade %	METs	HR bpm	SBP mmHg	DBP mmHg	Symptoms	ECG
Rest	0	0	1	68	120	90	No	Inferior Q waves
I	2.7	10	4.6	91	160	80	No	No change
II	4.0	12	7.0	103	170	80	No	No change
III	5.4	14	10.0	125	190	80	Mild fatigue	ST downslope of 1 mm in V5-V6
IV	6.7	16	12.5	142	190	80	Intense fatigue	ST downslope of 1 mm in V5-V6
Exercise duration, 10 min 00 s; onset of ischemia, at 10 min 00 s of exercise with 123 bpm								
Rec. 1'	1.5	0	1	123	190	80	No	ST downslope of 1 mm in V5-V6
Rec. 3'	0	0	1	95	180	80	No	ST downslope of 1 mm in V5-V6
Rec. 6'	0	0	1	88	130	80	No	ST downslope of 1 mm in V5-V6
Rec. 9'	0	0	1	80	120	75	No	Equal to the rest ECG

(12.5 mg, od), simvastatin (20 mg, od), ASA (100 mg, od), and two oral antidiabetic drugs (Table 1.6).

After 12 weeks of CRP, he was reassessed by a new ET, under the same protocol (Bruce) and medication (Table 1.7).

Comments

First test:

The patient had residual ischemia with a moderate compromise of functional capacity (7 estimated METs). He was admitted to the CRP, with a THR of 100 bpm, calculated with the Karvonen formula, adding 60 % of his HRR to the rest HR: [(131–59) × 0.60] = (43+59) = 102 ~100 bpm. If the calculated value for THR would be superior or equal to the HR of the ischemia threshold, the THR would be

assigned to a HR 10 bpm lower than the HR of the ischemic threshold that would be around 110–115 bpm.

He didn't complain about any symptom during the program and he progressed very well.

Second test:

The second test was performed immediately at the end of the program. It shows a very good evolution [35]:

1. Better functional capacity (12.5 vs 7.0 estimated METs).
2. Lower values of HR at each stage of the protocol, with silent ischemia appearing almost at the same HR value, although much later in the ET. Although some references show that ischemic threshold can appear at a higher HR and double product after exercise training, in this case, like in the majority of the published articles, only a delayed appearance of the threshold was found.
3. Higher HR and BP values at peak exercise.

Case #3

Male, 64 years old, asymptomatic, submitted to ET on the 3rd of December 2009, 5 days after inferior STEMI

Risk factors: dyslipidemia, hypertension, smoking, and family history of CVD below 60 years.

Medication: aspirin, clopidogrel, ß-blocker, ACE inhibitor, and statin.

Baseline ECG: sinus rhythm and Q waves on inferior leads.

Coronary angiography: LM lesion <50 %. RCA occlusion at the middle segment with retrograde filling from the left coronary. No significant lesions were found on LAD and circumflex arteries (Table 1.8; Figs. 1.3 and 1.4).

Table 1.8 Admission exercise test (case #3)

Exercise data								
Stage	Speed Km/h	Grade %	METs	HR bpm	SBP mmHg	DBP mmHg	Symptoms	ST-T changes
Rest	0	0	1	75	120	60	None	
I	2.7	10	4.6	111	150	80	Fatigue	ST depression: 1.0 mm
II	4	12	7	113	150	80	Fatigue, not-limiting angina	ST depression: 1.5 mm

Test stopped at 3 min and 33 s of Bruce protocol due to fatigue:					
Stage	HR bpm	SBP mmHg	DBP mmHg	Symptoms	ST-T changes
Rec. 1'	102	150	80	None	ST depression: 1.5 mm
Rec. 3'	90	140	80	None	ST depression: 1.5 mm
Rec. 6'	78	120	70	None	ST depression: 1 mm
Rec. 9'	81	120	70	None	Absent

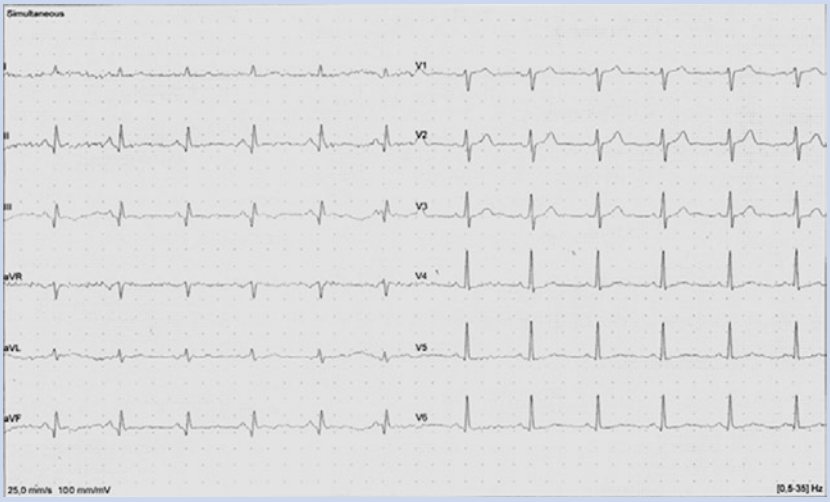

Fig. 1.3 Rest 12-lead ECG (case #3)

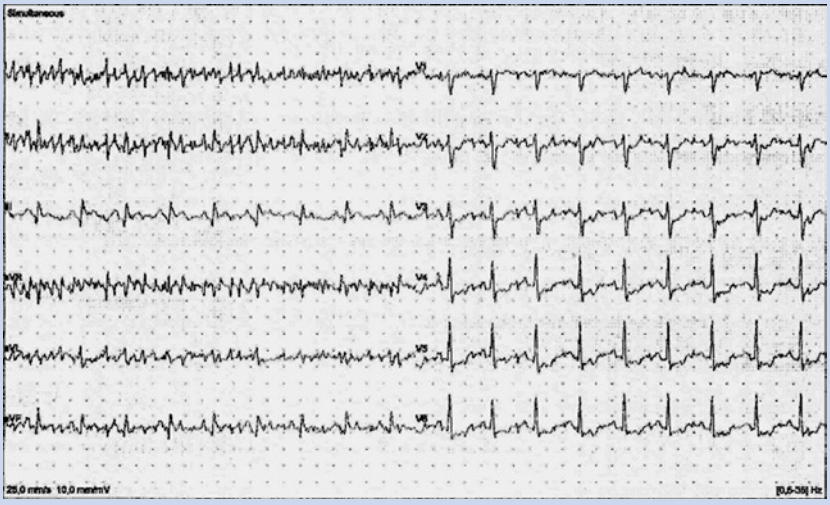

Fig. 1.4 Peak exercise 12-lead ECG (case #3)

1.8 Summary

METS = 4.6 (59 % of the predicted)

Peak HR: 113 ppm = 72 % predicted HR

% HR reserve use: 72.4 % (abnormal if ≤62 %)

HR decay in the first minute of recovery: 11 bpm (abnormal ≤12 ppm)

Peak double product = 16,950

ST changes: Horizontal downslope ST segment depression starting at 3 min of the exercise, with maximal amplitude of 1.5 mm in V4, V5, and V6. ST depression normalized at 9 min of recovery, after sublingual TNG at 6 min

Arrhythmias: absent

Conclusions

1. Moderate exercise tolerance limitation (<5 METs)
2. Myocardial ischemia starting at low exercise level (Table 1.9)

Table 1.9 Risk classification grades of exercise training for cardiovascular patients

Risk	Low stable conditions	Moderate	High unstable conditions
Decision regarding patient admission in the CRP	Accept	Decide case by case	Consider to reject or return to referral MD for stabilization
Type of CRP team	Basic experience	Advanced experience	
Individualized exercise prescription	Yes	Yes	Exercise training recommended only in few specific situations
Session supervision	Nonmedical personnel with advanced cardiac life support	Medical and nonmedical with advanced cardiac life support, until safety apparently guaranteed	As in moderate-risk patients
ECG and BP monitoring	6–12 sessions	≥12 sessions	≥12 sessions
NYHA	I or II	III	IV
Exercise capacity	≥7 METs	<5 METs	<5 METs
Myocardial ischemia	Absent or ≥7 METs	<7 METs	<5 METs
Ejection fraction	≥50 %	40–49 %	<40 %
Rise of BP and HR	Appropriate	Appropriate	Fall or non-increase of SBP or HR during exercise
VT at rest or during exercise	Absent		Complex arrhythmias
Self-monitoring	Able	Some difficulties	Unable

Adapted from Refs. [2, 14]

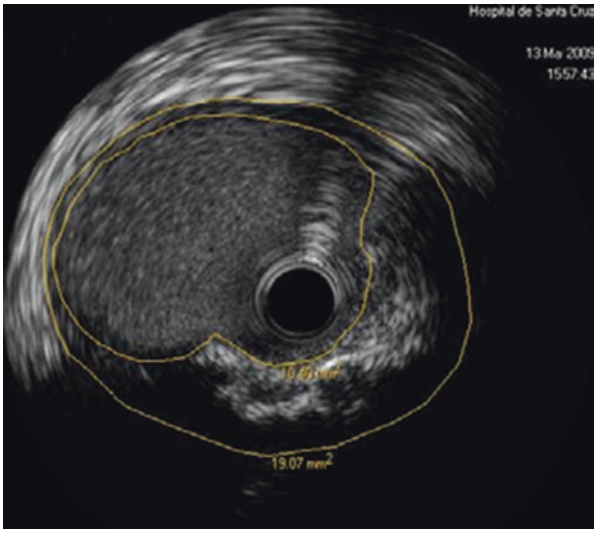

Fig. 1.5 Left main IVUS (case #3)

Comments

The patient was not accepted in the CRP, since he had myocardial ischemia starting below 5 METs, a contraindication for CRP admission.

This patient's myocardial ischemia must be considered serious, since it starts at low level of exercise, is associated with angina (although non-limiting), and is normalized only at 9 min in the recovery period after a sublingual TNG.

He was submitted to a new coronary angiography where IVUS was performed. The LM lesion, with an area of 10.4 mm^2 and a plaque burden of 47 % on IVUS, was considered as nonsignificant (Fig. 1.5), but a lesion of 70 % of the first marginal obtuse was defined as the lesion responsible for the ischemia. This lesion was submitted successfully to PCI with DES implantation.

He was reevaluated and admitted in the CRP 1 week later, after being submitted to a new ET that showed good exercise tolerance (9 min on Bruce protocol) and no residual ischemia.

Case #4

Male, 64 years old

Patient with an ischemic cardiomyopathy (ejection fraction of 28 %) in NYHA class III, after an anterior STEMI occurred 6 years before the clinical assessment for the CRP in February 2016.

He was submitted to primary PCI on his proximal LAD. No residual ischemia was diagnosed in a myocardial perfusion scan although several lesions on the right coronary artery were found. Moderate pulmonary hypertension and mitral and tricuspid regurgitation were present. An ICD was implanted 3 years before based on a primary prevention strategy. Cardiovascular risk factors: type 1 diabetes mellitus, former heavy smoker (quit smoking 30 years ago).

Current medication: bisoprolol, ivabradine, lisinopril, furosemide, simvastatin, ezetimibe, insulin, metformin, sitagliptin, and clopidogrel.

The ECG showed sinus rhythm and slow progression of R waves from V1 to V5.

Before admission he was submitted to a treadmill CPX under a ramp protocol (speed, from 2 to 5 km/h; grade, from 0 to 20 %; increments every 15 s; ECG recording and BP measurement every 2 min during exercise and on min 1 and 3 of the recovery period (Table 1.10).

Table 1.10 CPX parameters of case #4

	Rest	VT1	VT2	End of exercise
Time (min)	01:52	4:20	6:40	9:03
Ex Time (min)	00:00	02:20	4:40	7:03
Vt BTPS (L)	0.86	0.93	1.23	1.62
RR (br/min)	24.5	27.9	27.6	35.7
VE BTPS (L/min)	21.0	26.0	34.0	58.0
VO_2 (mL/kg/min)	7.1	8.7	10.4	11.7
VO_2 (mL/min)	524	641	773	864
VCO_2 (mL/min)	420	541	752	1073
RER	0.80	0.85	0.97	1.24
HR (BPM)	70	79	86	93
VO_2/HR (mL/beat)	7.5	8.1	9.0	9.3
VE/VO_2	34.0	35.4	40.4	62.9
VE/VCO_2	42.4	41.9	41.4	50.6
$P_{ET}CO_2$ (kPa)	4.11	4.17	4.04	3.33

Stage	**HR**	**SBP**	**DBP**	**Borg scale**	**ST-T changes**
Rest	70	85	60	6	Q waves V1 to V5
Min 2	84	90	60	10	
Min 4	84	90	60	14	
Min 6	91	130	80	16	
Min 7:09	90	135	90	18	No ST changes
Exercise duration: 7:09 min					
Rec. 1 min	71	70	40	16	
Rec. 3 min	68	80	55	12	

Exercise duration: 7:03 min	*Stop exercise due to:* exhaustion
Peak HR: 91 bpm (58 % of predicted maximal HR)	*HR decay peak – recovery min 1:* 22 bpm
Chronotropism: limited even for a patient under β-blocker	
Delta SBP = 15 mmHg	*Double product* = 9100
ST-T changes: none	*Arrhythmias*: none

Comments

The patient demonstrated a severe compromise of exercise tolerance: peak VO_2 is 11.7 ml/kg/min (41 % of the predicted value) being classified as Weber class C