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Hemodynamics during active and passive recovery from a single bout of supramaximal exercise

Abstract The aim of this work was to study the differences in cardiovascular response during two modes of recovery [active (AR): pedalling at 40 W; and passive (PR): complete rest seated] from a single bout of supramaximal exercise. Eight male amateur soccer players underwent two supramaximal cycle-ergometer tests, each consisting of pedalling against a resistance equivalent to 150% of the maximum workload achieved in a previous incremental test, followed by randomly assigned AR or PR. Cardiodynamic variables were obtained using an impedance cardiograph. Subjects were also connected to a sphygmomanometer, for systolic and diastolic blood pressure, and to a metabolimeter for oxygen uptake ($\dot{V}O_2$) assessments. We measured: heart rate (HR), stroke volume (SV), cardiac output (CO), the inverse of myocardial contractility calculated as pre-ejection period/left ventricular ejection time ratio (PEP/LVET), mean blood pressure (MBP), thoracic electrical impedance (Z_0) as an index of central blood volume, and arterio−venous oxygen difference (A−V O_2 Diff.). PR caused a lower CO compared to AR [mean (SE): 7 (0.7) vs. 10.4 (0.6) l·min^{-1} at the 5th min of recovery] due to lower HR [106.2 (3.6) vs. 121.8 (4.5) bpm at the 5th min of recovery], SV [67.1 (5) vs. 86.1 (4.8) ml at the 5th min of recovery], and PEP/VET values [0.44 (0.007) vs. 0.39 (0.015) at the 5th min of recovery]. No differences were found in MBP and Z_0 between PR and AR [95.1 (1.9) vs.

92.3 (2.7) mmHg and 26.2 (1.1) vs. 26.6 (1) Ω respectively at the 5th min of recovery], while A−V O_2 Diff. values were higher during AR than during PR [108.8 (4.3) vs. 75.2 (5.4) ml·l^{-1} at the 5th min of recovery]. Thus, although after a single bout of supramaximal exercise SV and CO are lower during PR than during AR, these differences are not due to an impairment of cardiovascular function, but are fully explained by the lesser muscular engagement that leads to a reduction in stimuli deriving from mechanoreceptors and central commands, thus causing a faster return of myocardial contractility and HR to resting values.

Keywords Blood pressure · Cardiac output · Myocardial contractility · Stroke volume · Transthoracic electrical impedance

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Introduction

The period following strenuous exercise is thought to be particularly critical for the cardiovascular apparatus. In fact, on stopping exercise, sudden changes in cardiac pre-load, after-load and contractility occur (Miles et al. 1984; Plotnick et al. 1986). These changes, together with the reduction in metabolic needs, the changes in blood electrolyte concentration, the decrease in circulating catecholamines, and the vagal rebound (Arai et al. 1989; Paterson 1996), may alter cardiovascular homeostasis, thus explaining the relatively high incidence of post-exercise accidents such as arrhythmias, hypotension, and syncope (Crisafulli at al. 2000; Fleg and Lakatta 1986). To diminish these risks some authors have suggested the use of active or supine recovery to facilitate venous blood flow to the heart (Carter et al. 1999; Goldberg and Shepard 1980; Johnson et al. 1990; Takahashi et al. 2000). In particular, the use of active recovery is based upon the fact that rhythmic muscle contractions generate intra-muscular pressure oscillations which, by facilitating venous blood flow to the heart, help in

maintaining stroke volume (SV) and consequently cardiac output (CO).

To study the influence of different modes of recovery (passive up, active up, or supine) most authors have investigated cardiodynamics following moderate or maximal aerobic activity, i.e. after a rectangular or progressive bicycle work test(s) (Carter et al. 1999; Johnson et al. 1990; Plotnick et al. 1986; Takahashi and Miyamoto 1998; Takahashi et al. 2000). However, there are few data on cardiodynamic responses following supramaximal dynamic exercise (Kilgour et al. 1995). This work intensity "is of particular interest, since for most individuals, it covers more than 70% of the range of intensities from rest to maximal effort, and it occurs frequently in many sports" (Bangsbo 1996). For example, in soccer it was reported that an interval between 11.2% and 18.8% of the total distance run was covered by players at their highest individual speeds (Ekblom 1986).

The purpose of this report is to study the differences in cardiovascular response during two modes of recovery from a single bout of supramaximal exercise. In particular, since the cessation of leg movement is known to impair venous return so reducing SV and CO, we tested whether passive, as compared to active recovery, could affect these cardiovascular variables.

Methods

Subjects

Eight volunteers (adult male) amateur soccer players [age 29.2 (0.9) years; height 175.5 (1.81) cm; mass 70.4 (1.96) kg] were studied. All subjects were free of any known cardiovascular disease and none reported suffering from post-exercise intolerance or hypotension. Each subject gave his informed written consent to participate in this study which was approved by the local ethics committee.

Experimental design

Prior to entering the study each subject performed a preliminary 20 W·min^{-1} incremental test, starting from 20 W at a pedalling frequency of 60 rpm on an electromagnetically braked cycle-ergometer (Tunturi EL-400, Finland) to assess the maximum workload achievable (W_{max}). Exhaustion was taken as the point when the subject was unable to maintain a pedalling rate of at least 50 rpm. Mean values (SE) of maximum heart rate (HR), maximal oxygen consumption ($\dot{V}O_{2max}$), and W_{max} during this preliminary test were 175.9 (1.12) bpm, 3.12 (0.14) l·min^{-1} and 191.4 (15.9) W respectively. The relatively low values of $\dot{V}O_{2max}$ we found (i.e. 44.3 ml/kg) are consistent with previous findings in amateur soccer players, who are probably engaged in inadequate aerobic training (Ekblom 1986). On a separate day (the interval from the preliminary test was at least 3 days) each subject performed two supramaximal tests, identical in the dynamic phases but different in recovery. The interval between these tests was at least 1 h. In this period the subject sat on a chair and waited for the return of cardiorespiratory variables to the pre-exercise level. Recovery was considered complete when HR was not higher than 10 bpm compared with the pre-exercise level and when the respiratory ratio, calculated as the carbon dioxide/oxygen uptake ratio, was less than 0.9. The dynamic phases consisted of: a period of 3 min in an upright seated position on the cycle-ergometer in order to obtain hemodynamic and metabolic data utilized as baseline; a warm-up period of 3 min pedalling at 40 W followed by the supramaximal test, which consisted of pedalling at the maximum voluntary speed in the upright position against a resistance equivalent to 150% of W_{max} until exhaustion. Recovery was evaluated either actively (AR; pedalling at 40 W at a pedalling rate of 50 rpm) or passively (PR; complete rest seated on the cycle), assigned randomly. We chose to perform active recovery at 50 rpm in order to minimize the impedance artefacts due to exercise movements (see Hemodynamic and metabolic measurements and Technical and protocol limitations of this work). The power of 40 W was chosen because we noticed that when pedalling at lighter workloads athletes tended to pass the suggested rate of 50 rpm. All experiments were conducted between 9 a.m. and 12 noon. Subjects had a light meal at least 2 h before the exercise tests. In every case recovery was studied for 5 min.

Hemodynamic and metabolic measurements

Cardiodynamic variables during all cycle-ergometer tests were continually measured using an impedance cardiograph (NCCOM 3, BoMed, Irvine, Calif., USA), a device commonly utilized to assess hemodynamics either in resting or exercising subjects (Belardinelli et al. 1996; Concu and Marcello 1993; Crisafulli et al. 2001; Northridge et al. 1990; Pianosi and Garros 1996; Richard et al. 2001). NCCOM 3 was connected to the subject with eight spot electrodes. Dual lower thoracic voltage-sensing electrodes were placed perpendicularly to the longitudinal plane of the sternum, laterally to the xiphoid process in the mid-axillary line. Two cervical voltage-sensing electrodes were placed as close as possible to the clavicles at the lateral aspect of the base of the neck. The current-injecting electrodes (2.5 mA, 70 kHz) were placed 5 cm above the cervical sensing electrodes and below the thoracic sensing electrodes. NCCOM 3 analog traces of electrocardiogram (ECG), thorax impedance (Z_0), and the maximal Z_0 first derivative (dZ/dt) were stored using a digital chart recorder (ADInstruments, PowerLab 8sp, Castle Hill, Australia). We made SV calculation afterwards, excluding signals in which obvious error signals were evident, using the following Sramek–Bernstein equation (Bernstein 1986):

$$SV = (VEPT \cdot Z_0^{-1}) \cdot (dZ/dt)_{max} \cdot (LVET)$$

All traces were analysed by the same operator. We directly measured all the variables constituting the Sramek–Bernstein equation, except for VEPT (volume of electrical participating tissues), which was calculated from a nomogram knowing individual sex, height and mass values (Bernstein 1986). In particular, LVET (left ventricular ejection time) was obtained as the interval between points B and C in dZ/dt, while dZ/dt_{max} was the distance in the dZ/dt trace between the beginning of the widest deflection and point D, i.e. the deflection peak (Fig. 1a). We also measured pre-ejection period (PEP) identified as the time interval between the onset of the electrocardiogram Q wave (point A in Fig. 1) and the beginning of the widest deflection occurring in the dZ/dt (point B). As it was found that PEP and LVET vary inversely with HR and to allow a proper interpretation of the deviation of these systolic time intervals, we corrected absolute values by using standard equations that minimize variability due to HR inter-individual differences (Lewis et al. 1977). By dividing corrected values of PEP and LVET we obtain the PEP/LVET ratio, which is inversely correlated to myocardial contractility (Lewis et al. 1977).

Figure 1b shows how artefacts can affect impedance traces, making it difficult to clearly recognize the reference points. We excluded beats affected by artefacts and performed calculations using only traces in which at least the 20% of the total beats were artefact-free in 1 min of recording time. NCCOM 3 automatically eliminated the effects of respiration using a reset synchronous with the P wave of the ECG, which allowed us to erase the breath component on derivative impedance traces (Ciuti et al. 1994).
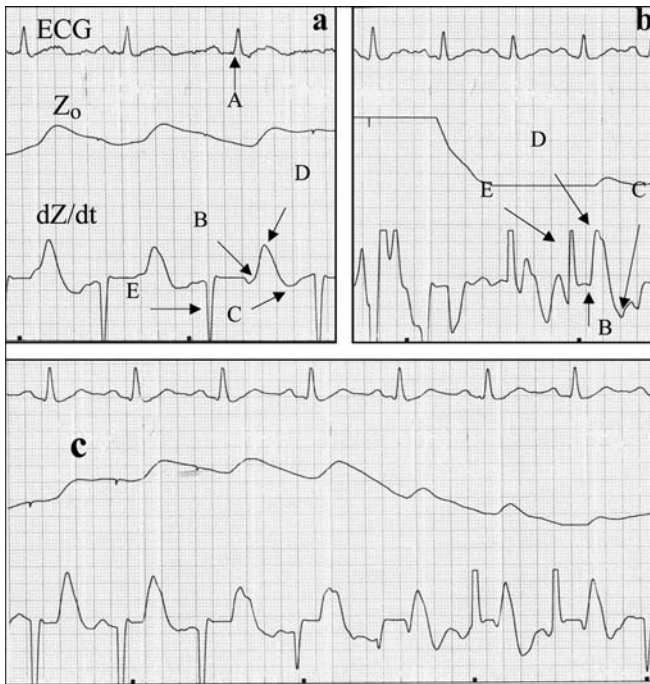
Fig. 1a–c Traces of ECG, thoracic electrical impedance (Z_0) and dZ/dt as measured by an impedance cardiograph (NCCOM 3) are shown in each panel. **a** Traces obtained at rest: the critical points for determining hemodynamic variables are distinguished. *A* the beginning of the QRS complex, *B* the beginning of the widest deflection of dZ/dt, *C* the inverse deflection in the dZ/dt trace, *D* peak of dZ/dt (dZ/dt_{max}), *E* automatic reset synchronous with the P wave of the ECG. **b** The same traces as in **a** obtained during high-intensity exercise. Critical points are readable at the fourth complex, while the previous three are affected by artefacts. **c** dZ/dt trace is stable notwithstanding the presence of the respiratory component in Z_0. Paper speed 50 mm/s

Therefore, derivative impedance depended only on the mechanical events of the heart (Fig. 1c). HR was calculated as the reciprocal of the R-R interval in the ECG trace and CO was obtained by the product SV·HR. As it was found that, during exercise recovery, a decrease in thoracic blood volume is associated with an increase in Z_0 (Carter et al. 1999), we used changes in Z_0 as an indirect measurement of changes in central blood volume. We calculated diastolic time (DT) subtracting the sum of PEP and LVET from the total period of the cardiac cycle. We obtained the ventricular filling rate by dividing SV by DT (SV/DT) (Crisafulli et al. 2000; Gledhill et al. 1994). Subjects were also connected to a standard sphygmomanometer for arterial systolic (SBP) and diastolic blood pressure (DBP) measurements, which were made in the left arm by the same physician for all tests. By dividing mean arterial blood pressure [MBP, obtained as DBP+(SBP−DBP)/3] by CO and multiplying by 80 we calculated systemic vascular resistance (SVR). According to Fleg and Lakatta (1986), we defined post-exercise hypotension as a decline in SBP of at least 20 mmHg below sitting values before exercise to an absolute level of 90 mmHg or less. Measurements of $\dot{V}O_2$ were made using a mass spectrometer (MedGraphics Breeze, St. Paul, Minn., USA) calibrated immediately before each exercise test. By dividing $\dot{V}O_2$ values by CO we obtained arterio-venous oxygen difference (A−V O_2 Diff.).

Data analysis

The mean values (SE) of the above variables at rest and during the five recovery minutes from the two modes of recovery are shown. Comparisons were performed with two-way ANOVA for repeated measures (factors: recovery modes and time) followed by Newman-Keuls post hoc when appropriate. Statistical significance was set at a *P* value of < 0.05 in all cases.

Results

The mean (SE) power output during supramaximal exercise tests was 291.4 (23.3) W, i.e. 150% of W_{max}. No difference was found between the average duration of these tests [86.2 (8.5) s and 92.4 (7.3) s for the AR and PR respectively; $P = 0.59$]. Table 1 shows the mean (SE) of some cardio-metabolic variables recorded during the tests. Data are from six subjects because we were unable to record reliable (at least 20%) thoracic impedance waveforms during the exercise in two of the eight subjects, due to movement and respiratory fake signals. No statistical differences were found between variables; therefore, it can be stated that the tests were similar for cardiovascular and metabolic engagement. During recovery none of the subjects studied experienced hypotension, as defined above, and no abnormalities were noticed in the ECG traces.

Figure 2 illustrates the dynamic profiles of HR, SV, and CO (panels a, b and c respectively). As expected, exercise increased HR ($p < 0.01$ vs. rest in AR and PR), which was restored towards resting values in both tests during recovery. But, starting from the 2nd min, HR was significantly lower during PR than during AR ($p < 0.05$) and this trend continued until the 5th min. It is worth noting that HR did not recover completely during AR or PR. SV was increased similarly by exercise in both AR and PR tests. In particular, during the 1st min of recovery, SV was near double compared to pre-exercise values. However, during the following few minutes of recovery, SV showed a clear trend to decrease towards resting values, but this drop was sharper during PR than during AR, reaching significance at the 5th min of recovery ($p < 0.05$). ANOVA revealed significant differences between recovery modes in terms of CO ($p < 0.05$). In fact CO was lower during PR compared to AR, starting from the 2nd min of recovery and remained higher than rest in both modes of recovery ($p < 0.05$) (Fig. 2c).

Table 1 Mean values (SE) of hemodynamic and metabolic data during the supramaximal tests followed by active recovery ($Test_{AR}$) and by passive recovery ($Test_{PR}$) respectively ($n = 6$)

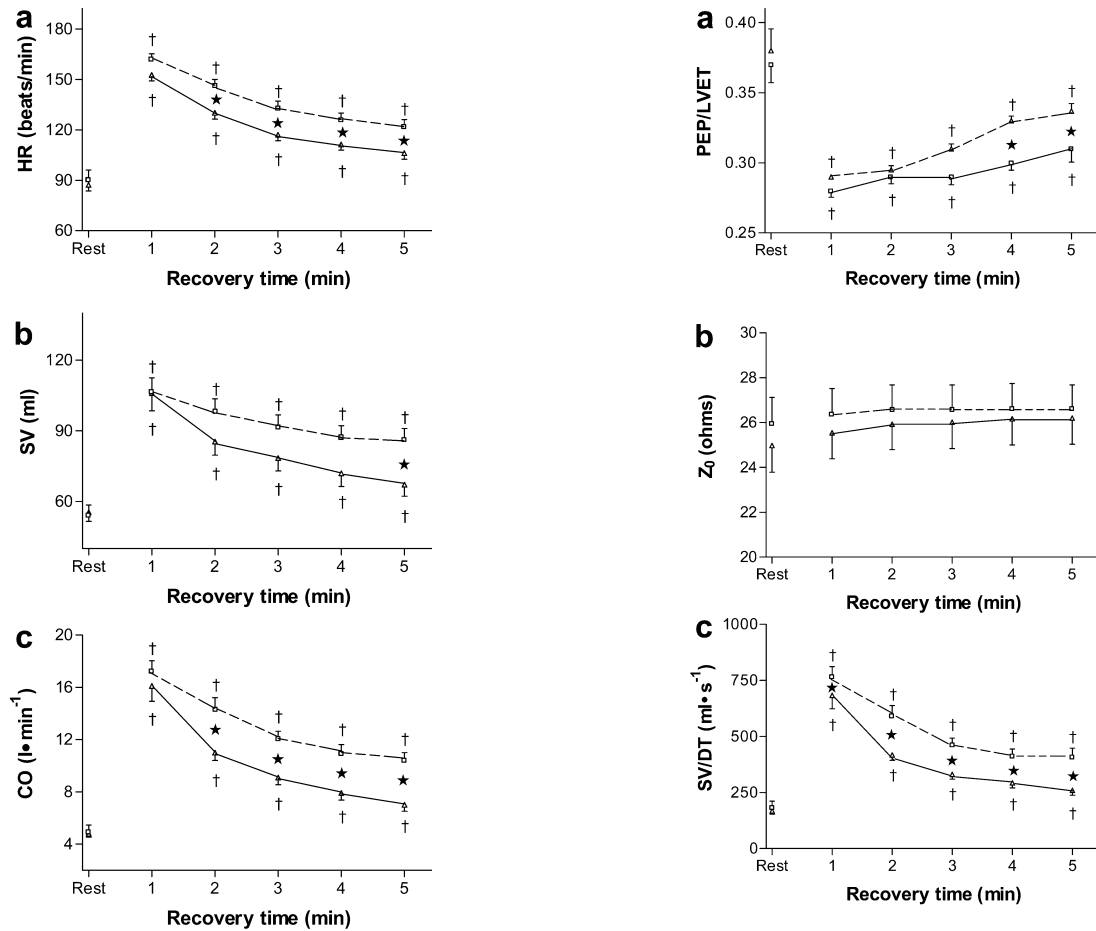| Variable	$Test_{AR}$		$Test_{PR}$		P values
	Mean	SEM	Mean	SEM	
HR (beats/min)	154.9	(3.2)	151.6	(3.3)	0.7
SV (ml)	102.5	(7.6)	113.7	(8.4)	0.72
CO (l·min^{-1})	15.8	(1.2)	17.4	(1.3)	0.7
PEP/LVET	0.37	(0.015)	0.36	(0.011)	0.8
Z_0 (Ω)	25.3	(1)	24.4	(1)	0.8
SV/DT (ml·s^{-1})	665.1	(167.9)	777.2	(197.5)	0.3
$\dot{V}O_2$ (l·min^{-1})	2326.3	(145.8)	2384.4	(235.6)	0.9
A–V O_2 Diff. (ml·l^{-1})	146.6	(5.1)	131.5	(6.4)	0.5

Fig. 2a–c Heart rate (*HR*; **a**), stroke volume (*SV*; **b**), and cardiac output (*CO*; **c**) changes during rest and 5 min of recovery from supramaximal tests. Values are mean (SE), $n = 8$. The two modes of recovery were active (pedalling at 40 W: *squares*, *dashed line*) and passive (complete rest seated on the bicycle: *triangles*, *continuous line*). *$p < 0.05$ vs. corresponding time point. †$p < 0.05$ vs. rest

Fig. 3a–c Myocardial contractility (*PEP/LVET*; **a**), thoracic impedance (Z_0; **b**), and diastolic filling rate (*SV/DT*; **c**) changes during rest and during 5 min of recovery from supramaximal tests ($n = 8$). The two modes of recovery were active (pedalling at 40 W: *squares*, *dashed line*) and passive (complete rest seated on the bicycle: *triangles*, *continuous line*). (*DT* Diastolic time, *LVET* left ventricular ejection time, *PEP* pre-ejection period, *SV* stroke volume.) *$p < 0.05$ vs. corresponding time point. †$p < 0.05$ vs. rest

Figure 3a illustrates the time course of the PEP/LVET ratio, a variable inversely related to myocardial contractility. PEP/LVET tended to be lower during both recovery modes than during rest ($p < 0.05$). Moreover, we also found significant differences between recovery modes ($p < 0.05$) starting from the 4th min of recovery. Thus, it seemed that there was a gradual reduction in myocardial contractility during the entire recovery period and this tendency was more pronounced during PR compared with AR.

Z_0 values were not different between the two recovery modes or compared to rest values (Fig. 3b). In contrast, SV/DT (Fig. 3c) was significantly increased by exercise with respect to rest values. In fact, this parameter was higher than rest during both recovery modes ($p < 0.05$ in both cases). However, during active recovery, SV/DT was higher than during PR for the entire period of recovery ($p < 0.05$).

Exercise increased SBP, which was significantly different during *both* recovery modes compared to rest.

ANOVA for SBP revealed that the groups were not significantly different ($p > 0.05$), but that there were significant differences within time ($p < 0.05$). In fact, in Fig. 4a it can be seen that SBP significantly increased compared with rest values during the 1st min of PR and AR ($p < 0.05$). Then SBP gradually decreased during the remaining period. As concerns DBP, statistics did not reveal significant differences between groups ($p > 0.05$), but a significant difference within time ($p < 0.05$). Indeed, in both tests, exercise lowered DBP, which decreased compared to rest values during the first 3 min of PR and the first 2 min of AR ($p < 0.05$ in both cases), and then gradually increased throughout the remaining recovery (Fig. 4b). As a consequence of the time courses of SBP and DBP, MBP values dropped slightly during the 1st min of recovery in comparison with rest values, followed by a prompt rise (Fig. 4c) to levels not different than resting. Two-way ANOVA demonstrated that MBP was not significantly different between recovery modes ($p > 0.05$), but was significantly different within time
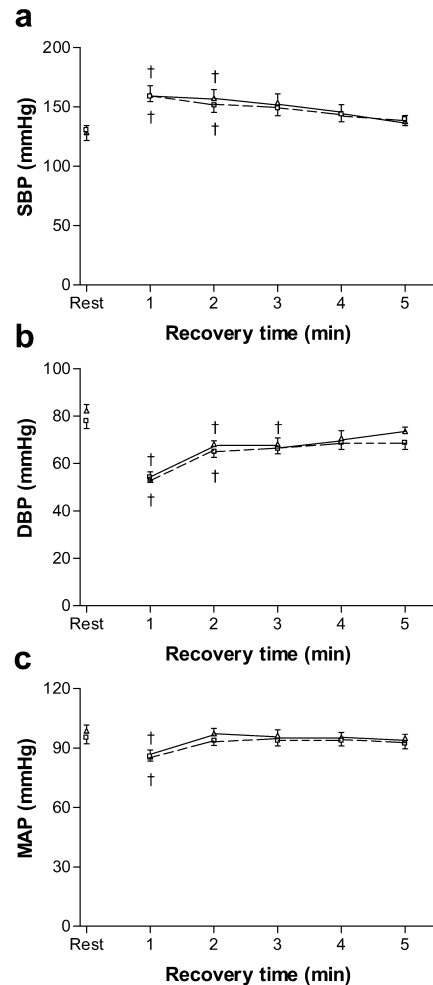
Fig. 4a–c Systolic blood pressure (*SBP*; **a**), diastolic blood pressure (*DBP*; **b**), and mean blood pressure (*MAP*; **c**) during rest and during 5 min of recovery from supramaximal tests (*n* = 8). The two modes of recovery were active (pedalling at 40 W: *squares, dashed line*) and passive (complete rest seated on the bicycle: *triangles, continuous line*). †*p* < 0.05 vs. rest

Fig. 5a–c Systemic vascular resistance (*SVR*; **a**), oxygen consumption (*VO₂*; **b**), and arterio-venous oxygen difference (*A–V O₂ Diff*.; **c**) during rest and during 5 min of recovery from supramaximal tests (*n* = 8). The two modes of recovery were active (pedalling at 40 W: *squares, dashed line*) and passive (complete rest seated on the bicycle: *triangles, continuous line*). *p < 0.05 vs. corresponding time point. †*p* < 0.05 vs. rest

($p < 0.05$). Figure 5a illustrates the time course of SVR. Statistics showed that SVR was significantly different between recovery modes ($p < 0.05$) and time ($p < 0.05$). Exercise decreased this parameter, which remained low at the 1st min of recovery during both AR and PR in relation to rest values ($p < 0.01$ in both cases). However, during PR, recovery of SVR was quicker than during AR and this difference had *a trend* to increase gradually throughout the remaining minutes, reaching statistical significance at the 3rd min of recovery ($p < 0.05$).

Exercise induced VO_2 to increase several times with respect to rest ($p < 0.05$). The pattern of VO_2 restoration towards rest was similar in the 1st min of recovery in both modes compared with rest values. But, starting from the 2nd min, VO_2 was significantly lower during PR than during AR ($p < 0.05$) (Fig. 5b). Similar behaviour was seen for A–V O₂ Diff. (Fig. 5c). After exercise, this variable was higher than during rest ($p < 0.05$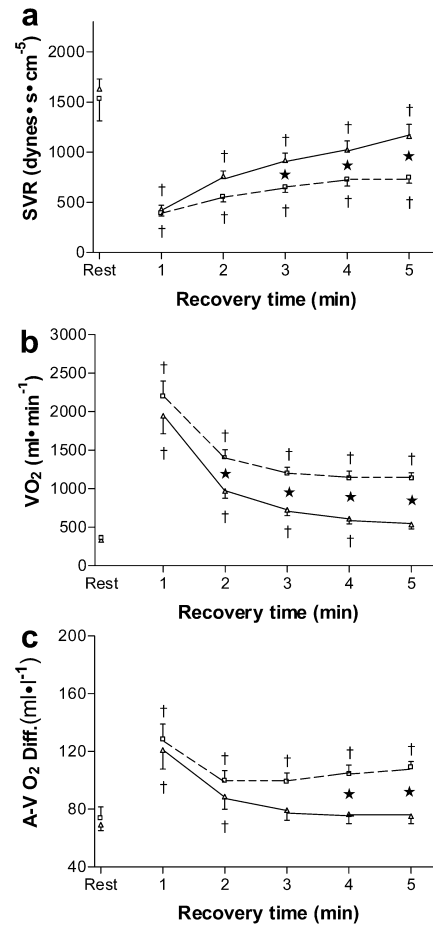). In particular, during the 1st min of recovery both AR and PR reached similar values; then, PR gradually decreased towards rest and seemed to be lower than during AR, reaching statistical significance at the 4th min of recovery ($p < 0.05$).

Discussion

The aim of this work was to characterize hemodynamic changes following supramaximal exercise. This work intensity may be frequently reached in a large numbers of sports such as rugby, basketball, soccer, tennis, athletics, etc., which are played by a large number of people. Features of these activities include continuous accelerations lasting several seconds and frequently supramaximal, decelerations, and stops (Bangsbo 1996). As a consequence, a steady state in hemodynamic and metabolic variables is not achieved and body homeostasis is continually perturbed. In spite of the great

importance of these sports, the majority of hemodynamic studies have focused on submaximal or maximal exercises, which normally allow a steady state in cardiovascular and metabolic variables.

The major finding of this study is that PR causes a lower CO compared with AR during the recovery period from supramaximal cycle exercise due to lower HR and SV values. The differences in HR, SV and CO values between PR and AR were not unexpected since the majority of authors who inquired into exercise recovery obtained similar results (Carter et al. 1999; Goldberg and Shephard 1980; Miles et al. 1984; Takahashi and Miyamoto 1998), while only one paper reported a continuous rise in SV after a Wingate Anaerobic Power Test (Kilgour et al. 1995). We cannot account for the marked difference from our work, but the authors of the quoted paper claimed that their result was unexpected.

The lower HR values during PR compared with AR were probably mediated by the cessation of a central command from the cerebral motor cortex and by a reduction in the stimuli from mechanoreceptors (O'Leary 1993). The reduced SV during PR compared to AR was, in our opinion, mainly due to the faster restoration of myocardial contractility towards resting values during PR, confirmed by the steeper increase in the PEP/LVET ratio during motionless recovery; probably the cessation of the same stimuli that reduced HR contributed as well. Another mechanism that may have decreased SV during PR compared to AR was the reduction in cardiac pre-load due to a reduction in venous return caused by the lack of muscle pump activity. Actually, there was a reduction in venous return during PR compared to AR, as shown by the pattern of SV/DT change, a measure of the mean rate of diastolic blood flux (Crisafulli et al. 2000; Gledhill et al. 1994). However, we think that, despite the decrement in SV/DT noticed during PR, cardiac pre-load was kept constant. In fact, even if we were not able to measure the myocardial pre-load directly, we are quite confident that there was no reduction in end-diastolic volume because there was no increase in Z_0 during PR compared with AR. This impedance index was used by Carter and co-workers (1999) to demonstrate a decrease in central blood volume during inactive recovery from cycle-ergometer exercise. These authors reported an increase in Z_0 of about 5 Ω during the 1st min of inactive recovery after dynamic cycle exercise. They attributed this increase in Z_0 to a decrease in venous return to the heart, presumably due to accumulation of blood in the leg veins. In our study we did not observe such a dramatic increase in Z_0, so we think that end-diastolic volume was maintained despite the inactivity of leg muscles. This fact is not surprising since it is known that factors other than muscle pump action contribute to diastolic filling. In particular, the length of the diastolic phase is of primary importance in allowing effective diastolic filling and tachycardia, shortening diastolic time, limits ventricular filling (Higginbotham et al. 1986). Thus, the lower HR that occurred during PR compared to AR may have enhanced ventricular filling by prolonging diastole. Therefore, in our experiment, despite the reduced diastolic filling rate found during PR, it may have been that a relative bradycardia during PR successfully maintained the end-diastolic volume and so Z_0 in the face of the cessation of the central command, the mechanoreflexes and the muscle pump.

Another finding was that MBP, after a small transient fall with respect to pre-exercise levels in both AR and PR during the 1st min of recovery, promptly returned to values similar to those of pre-exercise. This transient descent in MBP was the result, in both cases, of the reduction in DBP, which was not compensated by the increase in SBP. The gradual restoration of DBP, together with SBP values higher than at rest, allowed MBP to be similar to pre-exercise values during the remaining minutes of recovery. No differences were found between MBP values between AR and PR. These results support the light hypotensive effect of an acute bout of exercise in normotensive subjects, well documented in other works (Coats et al. 1989; Kenney and Seals 1993; Piepoli et al. 1993). However, in our study the reduction lasted only for 1 min, meaning that our exercise protocol, in spite of the high intensity, was probably too short to lead to the post-exercise permanent hypotension noted by the aforementioned authors. Moreover, during both AR and PR, none of the subjects studied developed symptoms of hypotension and none had a decline in SBP of more than 20 mmHg compared with pre-exercise, the value that we chose to define post-exercise hypotension. In addition, non-significant differences were found between AR and PR in all pressure measurements, in spite of the lower CO values during PR. This was the consequence of the higher values in SVR during PR in comparison with AR, starting from the 3rd min of recovery. These results appear to suggest that circulatory mechanisms regulate arterial blood pressure to a set point that is kept constant by adjustments of vascular resistances in response to cardiac output changes.

The final consideration regards time courses of $\dot{V}O_2$ and A−V O_2 Diff. During AR, $\dot{V}O_2$ was, as expected, higher than during PR starting from the 2nd min of recovery, because of the metabolic cost of cycling. A−V O_2 Diff. values were not different during AR compared with PR during the first 3 min of recovery and, starting from the 4th min, were higher during AR than during PR. This fact suggests that CO during PR was adequate to fully support the metabolic needs of muscles. Actually, one of the most important responses to diminished CO during exercise is the increase of oxygen utilization in muscles, which causes an increase in the A−V O_2 Diff. (Katz et al. 2000). As a consequence, this study does not support the hypothesis that during PR from a single bout of supramaximal exercise the cardiovascular system suffers from some impairment, since A−V O_2 Diff. was not increased during this condition. This assertion is further strengthened by the fact that arterial blood pressure was very similar between AR and PR during the entire recovery period. The lower values of A−V O_2 Diff. during the last minutes of PR compared with AR

were probably because CO needs more time than $\dot{V}O_2$ to return to pre-exercise values after exercise (Yoshida and Whipp 1994), thus causing a reduction in the $\dot{V}O_2/CO$ ratio.

Technical and protocol limitations of this work

Our use of impedance cardiography to measure hemodynamic variables during exercise is still the subject of a series of criticisms (Warburton et al. 1999). One problem is that respiration and exercise may generate artefacts, especially during maximal and supramaximal exercise. We used visual inspection of recorded signals, performed by a skilled physician, to overcome this problem so as to calculate SV only from the traces in which at least 20% of the impedance waveforms were reliable. This method was time-consuming in processing signals, but the results during rest, exercise and recovery were highly reproducible. In fact, during AR and PR tests we obtained very similar SV values at rest, during exercise and in the 1st min of recovery, when subjects' conditions were comparable. Other parameters derived from the impedance traces (PEP/LVET and Z_0) also showed very similar values during rest, exercise and the 1st min of recovery, so we suggest that the impedance method is sufficiently reproducible when errors in signals are eliminated. However, it is important to note that it was not possible to have a sufficient number of artefact-free traces during the supramaximal exercise in all subjects. In fact, for two of them during both AR and PR tests, the artefact-free traces amounted to less than 20% of the total, the pre-established limit for considering the records suitable for calculating SV. However, the aim of this work was to study recovery periods, not exercise phases. During the recovery phases from the supramaximal tests in all the subjects the artefact-free signals accounted for more than 20% of the total time; consequently all records during recovery were suitable for SV assessment. The 20% threshold of artefact-free signals was chosen to allow a sufficient number of clear cardiac cycles to be used in the calculation of SV during each recovery minute. This threshold was higher than that proposed by Miles and Gotshall (1989) who suggested that, during maximal exercise, the impedance cardiogram should be taken for at least 30 s to obtain four to five suitable cardiac cycles (Miles and Gotshall 1989). Thus, we were quite confident that the number of cardiac cycles analysed during each recovery minute was sufficient to obtain reliable mean SV values. Another criticism inherent in impedance cardiography is the report by some authors that the method tends to underestimate high CO values, as occur during exercise (Warburton et al. 1999). However, the aim of this work was not to study the absolute values of cardiodynamic variables, but to study the relative differences between two modes of recovery. Moreover, in our work, values of A−V O$_2$ Diff. during exercise (Table 1) and recovery (Fig. 5c) were not higher compared with those calculated with other standard methods (Higginbotham et al. 1986), as would be expected if impedance cardiography really underestimates high CO values. This fact provides indirect evidence that the error, if any, connected with the use of impedance cardiography is small. Our data are in accordance with two recent papers which found a good agreement between impedance cardiography and the direct Fick method for evaluating CO during exercise (Charloux et al. 2000; Richard et al. 2001). So, even though the impedance method is not quantitatively accurate and suffers from difficulties in recording reliable traces, we think that it is suitable for use in studies such as ours.

Concerning the protocol, we used a single bout of supramaximal exercise that was necessarily of short duration because of the early fatigue of the subjects. As a consequence, the absence of cases of hypotension or post-exercise intolerance was perhaps due to the fact that exercise duration was too short to stress cardiovascular homeostasis. Moreover, since our subjects were soccer players, it is probable that they were accustomed to efforts such as those used in this study, as during soccer games there are repeated bouts of supramaximal exercise. Consequently, it is possible that to stress the cardiovascular apparatus of our athletes sufficiently to cause homeostatic impairment, we should have used repeated bouts of supramaximal exercise.

In summarizing, our study provides evidence that, although after a single bout of supramaximal exercise SV and CO are lower during PR than during AR, these differences are not due to an impairment of cardiovascular function, but are fully explained by the lesser muscular engagement that leads to a reduction in stimuli deriving from mechanoreceptors and central commands, thus causing a faster return of myocardial contractility and HR to rest. Further research is needed to verify whether repeated bouts of supramaximal exercise cause a more pronounced perturbation in cardiovascular homeostasis than a single bout.

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