High Blood Pressure & Cardiovascular Prevention
https://doi.org/10.1007/s40292-021-00491-5

ORIGINAL ARTICLE

Relationship Between Cardiorespiratory Fitness, Baseline Blood Pressure and Hypertensive Response to Exercise in the Ferrari Corporate Population

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Received: 3 November 2021 / Accepted: 15 November 2021

Abstract

Aim To evaluate the incidence and clinical significance of impaired cardiorespiratory fitness (CRF) and the association with baseline blood pressure (BP) levels and hypertensive response to exercise (HRE).

Methods A cross-sectional study was conducted on a total sample of 2058 individuals with a mean age of 38 ± 9 years, enrolled for the first time at the Ferrari corporate wellness program "*Formula Benessere*", including a maximal exercise stress testing (EST). BP and heart rate (HR) values were obtained from EST at rest, during exercise and recovery time. CRF was arbitrarily classified according to estimated $VO_{2\ max}$ in optimal, normal, mildly and moderately reduced.

Results One-hundred and thirty-nine individuals of 2058 (6.7%) showed a moderate CRF reduction assessed by EST. Subjects with elevated resting and/or exercise BP showed a worse CRF than those with normal BP levels, also after the adjustment for age, sex, body mass index, smoking habits, peak SBP and DBP. Seventy-seven individuals (3.7%) showed an HRE during EST, with normal baseline BP levels.

Conclusion About 7% of a corporate population showed a significantly reduced CRF, assessed by EST. Individuals with lower levels of CRF have higher resting and/or peak exercising BP values after adjusting for co-variables. This study expands the role of EST outside of traditional ischemic CVD evaluation, towards the assessment of reduced CRF and HRE in the general population, as a possible not evaluated CV risk factor.

Keywords Hypertension · Hypertensive response to exercise · Corporate wellness · Cardiorespiratory fitness · Exercise stress testing

1 Introduction

Cardiorespiratory fitness (CRF) has been recently added to the traditional risk factors to improve lifetime risk prediction validity [1–4]. Among the common risk factors for cardiovascular (CV) disease, indeed, CRF has consistently shown to be one of the strongest prognosticators [5]. Importantly, for every 1 estimated MET increase, all-cause and CV disease mortality were reduced by 15 and 19%, respectively [6]. Considering the highly prognostic nature of CRF and its representation of the whole-body physiological function, its assessment has been used as the primary end point also in non-exercise training intervention (ie. pharmacological) in heart failure patients [7]. Exercise stress testing (EST), evaluating exercise tolerability, may represent an essential and simple tool for assessing CRF and its clinical relevance in the general population, particularly in the context of CV primary prevention initiatives, such as corporate wellness projects [8]. EST may also help to identify apparently healthy individuals who present hypertensive response to exercise (HRE), with normal baseline blood pressure (BP) levels, and a consequent increased risk of developing hypertension in the future.

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Published online: 25 November 2021

The aim of our study was to evaluate the incidence and the clinical significance of impaired CRF, assessed by EST, in a company healthy population and its relationship with baseline BP and exercise hemodynamic responses, matched to other characteristics of the subjects (age, body mass index, body fat percentage, baseline heart rate—HR and BP).

2 Methods

From January 2017 to December 2019 our study group included a total sample of 2058 consecutive individuals, 1784 males (87%) and 274 females (13%), with a mean age of 38 ± 9 years. The subjects were selected among those at the first entrance of the Ferrari "*Formula Benessere*" corporate wellness program, organized by the *Med-Ex* company on a voluntary basis at the Ferrari factory in Maranello (Modena, Italy) and accounted for about a half of the entire company employee population. This project, already elsewhere described [9], consists in an onsite yearly medical screening, including complete CV evaluation (family and personal history, physical examination, BP measurements, resting and EST electrocardiogram and taking pharmacological therapy), glucose measurement and lipid profile (triglycerides, high-density lipoprotein, low-density lipoprotein and total cholesterol) and nutritional and exercise prescription (to be practiced in a company gym). Pressure levels consisted of a mean of three BP manual measurements, performed 2 min apart. Anthropometric measures (including height and weight), body mass index and body composition were also calculated by impedenziometry (Tanita MC 780 MA, Amsterdam, Netherlands). According to 2018 European Guidelines, BP was categorized as normal (SBP < 130 mmHg and DBP < 85 mmHg), high-normal (SBP 130–139 mmHg or DBP 85–89 mmHg), grade 1 hypertension (SBP 140–159 mmHg or DBP 90–89 mmHg), grade 2 hypertension (SBP 160–179 mmHg or DBP 100–109 mmHg) and grade 3 hypertension (SBP ≥ 180 mmHg or DBP ≥ 110 mmHg) [10].

Information about pharmacological therapy and particularly about antihypertensive treatment were collected. One-hundred and nineteen subjects (5.8%) were already in antihypertensive treatment: the majority of patients received angiotensin converting enzyme inhibitors (ACEi-33%) or angiotensin receptor blockers (ARB-31%), 28% had calcium channel blockers (CCB), 16% beta-blockers, 9% diuretics and 2% alpha-blockers. Combination therapy with two or more drugs was prescribed in 38.5% of treated patients. Beta-blockers were withdrawn 72 h before performing EST, due to their influence on HR response and on exercise performance.

Each participant performed a maximal EST at cycloergometer (Daum Ergometer Premium 8i, Daum Electronic Gmbh, Fürth, Germany). The protocol consisted of 2 min of unloaded cycling, followed by increments of 50W for men and 30W for women every 2 min. In the absence of ECG changes and/or other events that imposed early termination, the test continued until volitional exhaustion (at least Borg scale level 17) [8]. Twelve-lead ECG was recorded at rest, during the exercise (with maximal HR calculation) and recovery time. Similarly, SBP and DBP were monitored at rest, during each phase of EST and during the first 5 min of recovery. The highest values of peak exercise BP were manually measured to the stretched patient right arm, detached from the handlebar. All tests were performed under the supervision of a cardiologist or a sports medicine physician trained in cardiology, in compliance with the exercise standards for testing [11]. To determine the exercise intensity, oxygen uptake (pVO_2) was indirectly calculated from the maximum workload measured by the ergometer software during EST, according to the published guidelines, and adjusted for body weight (pVO_2/kg) [12]. Circulatory power (CP) was obtained by the product of pVO_2/kg and peak SBP. Rate-pressure product was calculated multiplying peak SBP and peak HR. The predicted pVO_2 was determined by using the gender-, age-, and weight-adjusted Hansen/Wasserman equations [13]. As set by convention, one metabolic equivalent of task (MET) has been considered as 3.5 mL of oxygen per kilogram per minute.

The definition of hypertensive response to exercise was derived by previous studies, consisting in SBP ≥ 210 mmHg for men and ≥ 190mmHg for women.

Adhesion to this program was totally free and based on personal voluntary agreement. Written informed consent was waived for all subjects undergoing a standard evaluation pursuant to Italian law. All clinical data assembled from individuals are kept in the institution database and individual privacy was fully respected.

2.1 Statistical Analysis

Continuous variables were expressed as mean ± standard deviation (SD), while dichotomous variables were expressed as proportions or frequencies among either overall population or specific subgroups. Differences between continuous variables were assessed using one-way analysis of variance. Categorical variables were compared among groups by the chi-square test, applying Bonferroni correction. The Pearson correlation coefficient was used to measure the strength of the linear relationship between variables. All tests were two-sided, and a P value of <0.05 was considered statistically significant. All calculations were generated using SPSS version 20.0 (SPSS Inc, Chicago, IL).

3 Results

3.1 Anthropometric and Basal Results

The study group included a total sample of 2058 individuals, 1784 males (87%), 274 females (13%), with a mean age of 38 ± 9 years.

With regards on EST parameters, mean peak SBP and DPB was 172 ± 14 and 83 ± 10 mmHg, respectively. Mean peak heart rate was 167 ± 14 bpm. No EST was terminated prematurely due to medical reasons. Mean Borg scale level was 18 ± 1, all the subjects reaching at least a value of 17.

The distribution ranges for peak SBP and DBP varies from 150 to 210 mmHg and from 60 to 110 mmHg, respectively. One-hundred and twenty-five subjects (6%) showed peak BP values above these cut offs, showing a hypertensive response to exercise (SBP ≥ 210 mmHg for men, ≥ 190 mmHg for women). Out of these 125, 77 subjects (3.7% of the total population and 62% of the individuals with exaggerated BP) had normal resting BP values. Only 28 individuals (23%) of this subpopulation received antihypertensive therapy. Also for peak BP levels (as already showed for resting BP values), there were significant differences among groups, with an age-induced increase both for peak SBP and peak DBP. Inversely, Table 1 shows a general decrease of all exercise performance parameters with age (peak workload in Watt, peak workload in W/kg, estimated peak $VO_{2\ max}$ in mL/kg/min and circulatory power (mL/min/kg).

3.2 Relationship with Cardiorespiratory Fitness

We arbitrarily divided our population in groups related to CRF values (Table 1). CRF (n = 1183) was defined as optimal for pVO_2 > 100% of predicted, normal (n = 613) for pVO_2 85–99%, mildly reduced (n = 115) for pVO_2 70–84%, moderately reduced (n = 147) for pVO_2 69–55% and severely reduced (n = 0) for pVO_2 < 55%. Two hundred and fifty-four subjects (12%) had mildly (5.3%) or moderately reduced (6.7%) CRF. CRF showed a significant negative correlation with age and sex (female). A significant negative correlation was also documented with baseline and peak SBP, with baseline DBP and with resting HR. A parallel decrease both in CRF and all hemodynamic responses was documented.

Table 1 Classification of cardiorespiratory fitness (CRF) according to different exercise performance parameters in 2058 employees

	Optimal CRF (n = 1183)	Normal CRF (n = 613)	Mildly reduced CRF (n = 115)	Moderately reduced CRF (n = 147)	P value
General characteristics					
Age (years)	$40 \pm 9^{\#,\$}$	$35 \pm 8^{*,\$,\&}$	$35 \pm 9^{*,\#,\&}$	$39 \pm 10^{\#,\$}$	< 0.001
Male sex, n (%)	$948\ (80\%)^{\#,\$,\&}$	$594(97\%)^{*,\&}$	$113\ (98\%)^{*,\&}$	$34\ (88\%)^{*,\#,\$}$	< 0.001
BMI (kg/m^2)	24.8 ± 3.5	24.9 ± 3.5	24.9 ± 4.7	25.5 ± 4.8	NS
Body fat (%)	21 ± 14	19 ± 6	19 ± 8	20 ± 7	NS
Rest SBP (mmHg)	$119 \pm 9^{\#,\$,\&}$	$121 \pm 7^{*,\$,\&}$	$122 \pm 10^{*,\#,\&}$	$123 \pm 15^{*,\#,\$}$	< 0.001
Rest DBP (mmHg)	$77 \pm 7^{\#,\$,\&}$	$78 \pm 6^{*,\&}$	$80 \pm 8^{*,\&}$	$80 \pm 10^{*,\#}$	< 0.001
Rest HR (bpm)	$77 \pm 12^{\#,\$,\&}$	$82 \pm 11^{*,\$,\&}$	$86 \pm 13^{*,\#,\&}$	$87 \pm 15^{*,\#,\$}$	< 0.001
EST data					
Peak SBP, mmHg	$164 \pm 16^{\#,\$}$	$161 \pm 12^{*,\$,\&}$	$156 \pm 13^{*,\#,\&}$	$163 \pm 14^{\#,\$}$	< 0.001
Peak DBP, mmHg	83 ± 25	83 ± 8	83 ± 8	83 ± 8	NS
Peak HR, % of predicted	86 ± 12	85 ± 8	84 ± 11	84 ± 10	NS
Peak workload, W	$218 \pm 47^{\#,\$,\&}$	$189 \pm 23^{*,\$,\&}$	$162 \pm 21^{*,\#,\&}$	$161 \pm 25^{*,\#,\$}$	< 0.001
Peak workload/kg, W/kg	$2.93 \pm 0.59^{\#,\$,\&}$	$2.50 \pm 0.43^{*,\$,\&}$	$2.15 \pm 0.45^{*,\#,\&}$	$1.56 \pm 0.51^{*,\#,\$}$	< 0.001
Estimated Peak VO_2, mL/min	$2921 \pm 524^{\#,\$,\&}$	$2594 \pm 260^{*,\$,\&}$	$2289 \pm 237^{*,\#,\&}$	$1657 \pm 279^{*,\#,\$}$	< 0.001
Estimated Peak VO_2/kg, mL/min/kg	$39.4 \pm 7.2^{\#,\$,\&}$	$34.4 \pm 5.5^{*,\$,\&}$	$30.4 \pm 5.9^{*,\#,\&}$	$26.3 \pm 5.8^{*,\#,\$}$	< 0.001
Estimated Peak VO_2, % of predicted	$117 \pm 15^{\#,\$,\&}$	$93 \pm 4^{*,\$,\&}$	$81 \pm 3^{*,\#,\&}$	$66 \pm 3^{*,\#,\$}$	< 0.001
METs	$11.3 \pm 2.0^{\#,\$,\&}$	$9.8 \pm 1.6^{*,\$,\&}$	$8.7 \pm 1.7^{*,\#,\&}$	$6.2 \pm 1.7^{*,\#,\$}$	< 0.001
CP, mL/min/kg *mmHg	$6485 \pm 1441^{\#,\$,\&}$	$5522 \pm 981^{*,\$,\&}$	$4679 \pm 989^{*,\#,\&}$	$3359 \pm 960^{*,\#,\$}$	< 0.001

Data are expressed as mean ± SD

$^*p < 0.05$ versus optimal CRF; $^{\#}p < 0.05$ versus normal CRF; $^{\$}p < 0.05$ versus mildly reduced CRF; $^{\&}p < 0.05$ versus moderately reduced CRF. See text for abbreviations

3.3 Relationship Between CRF and BP Parameters

Table 2 showed that of 2058 individuals, 1577 (77%) are normotensive, 261 (12.5%) have high-normal BP, 125 (6%) have HRE, 99 (4.5%) have grade 1 and 41 (2%) have grade 2 hypertension. Of 140 subjects with grade 1 and 2 hypertension, 119 (85%) were already in pharmacological treatment, while 21 subjects (15%; 13 with grade 1 and 8 with grade 2 hypertension) had a new diagnosis during the program work-up and received antihypertensive agents later. No subject with high-normal BP received antihypertensive drugs, while only 28 of 125 subjects (23%) with HRE had treatment. Individuals with elevated resting BP ≥ 130/85 mmHg (n = 401), including those with high-normal BP, grade 1 and grade 2 hypertension (no one had baseline grade 3 hypertension) showed a worse CRF and reduced hemodynamic responses than those with normal resting BP levels (< 130/85 mmHg) (Table 2), also after the adjustment for age, sex, BMI, smoking habits, antihypertensive treatment, peak SBP and DBP. A significant difference in exercise parameters was also observed between individuals with high-normal and grade 1 hypertension. Furthermore, the percentage of subjects who presented a reduced CRF increased in parallel with BP values, mild and moderate CRF accounting for 17 and 30% of grade 2 hypertensive subjects, respectively (Fig. 1). Of note, 92 subjects with normal BP (6%), 13 with high-normal BP (5%) and 9 with HRE (7%) showed a moderately reduced CRF.

4 Discussion

In this study we have evaluated more than two-thousand apparently healthy individuals at the first entrance of a corporate wellness project, and this aspect gave us the unique opportunity to verify the clinical status of the general population in the territory and the real and current situation regarding the control of CV risk factors, including reduced CRF and hypertension. Young populations such as those of the companies, indeed, are usually unmonitored on the presence of CV risk factors at the population level and this reduces the possibility to apply the effective primary CV prevention at early stage [9].

The use of mass EST screening in these apparently healthy company populations may assume a novel clinical significance with a favorable cost-benefit approach [8]. This study observes that EST may also be useful in the measurement and classification of CRF, which is considered, when insufficient, as an additional CV risk factor [2, 3, 14, 15]. In this study, we documented a moderate reduction of CRF in young hypertensive individuals (some of them newly diagnosed with the project—15% among those with grade 1 and 2 hypertension), and surprisingly also in a small percentage of subjects with normal, high-normal BP and HRE (from 5 to 7%). Although previous studies defined cut-off values for HRE only in normotensive subjects, we used these thresholds also for hypertensive individuals since our aim was to evaluate the prevalence of impaired CRF according to BP levels at exercise peak.

Table 2 Exercise performance parameters according to blood pressure levels at resting and during exercise in 2058 employees

	Normal BP (n = 1657)	High-normal BP (n = 261)	HRE (n = 40)	Grade 1 hypertension (n = 99)	Grade 2 hypertension (n = 41)	P value
EST data						
Peak SBP, mmHg	$160 \pm 14^{\#,\$,\&}$	$171 \pm 12^{*,\$,\&}$	213 ± 14	$174 \pm 13^{*,\#,\&}$	$177 \pm 17^{*,\#,\$}$	< 0.001
Peak DBP, mmHg	$82 \pm 22^{\#,\$,\&}$	$87 \pm 7^{*,\&}$	116 ± 10	$93 \pm 9^{*,\&}$	$102 \pm 10^{*,\#,\$}$	< 0.001
Peak HR, % of predicted	86 ± 12	88 ± 11	86 ± 9	88 ± 14	85 ± 12	NS
Peak workload, W	$204 \pm 47^{\&}$	$210 \pm 39^{\&}$	204 ± 45	196 ± 39	$181 \pm 27^{*,\#}$	0.002
Peak workload/kg, W/kg	$2.67 \pm 0.84^{\#,\$,\&}$	$2.49 \pm 0.73^{*,\$,\&}$	2.53 ± 0.93	$2.21 \pm 0.83^{*,\#,\&}$	$1.50 \pm 0.99^{*,\#,\$}$	< 0.001
Estimated Peak VO_2, mL/min	$2641 \pm 727^{\&,\$}$	$2610 \pm 626^{\&}$	2630 ± 725	$2513 \pm 729^{*,\&}$	$1950 \pm 964^{*,\#,\$}$	< 0.001
Estimated Peak VO_2/kg, mL/min/kg	$36.5 \pm 9.5^{\#,\$,\&}$	$33.7 \pm 8.8^{*,\$,\&}$	34.1 ± 11.1	$30.3 \pm 10.3^{*,\#,\&}$	$22.5 \pm 11.1^{*,\#,\$}$	< 0.001
Estimated Peak VO_2, % of predicted	$102 \pm 24^{\&}$	$99 \pm 24^{\&}$	101 ± 27	$98 \pm 27^{\&}$	$76 \pm 24^{*,\#,\$}$	< 0.001
METs	$10.4 \pm 2.8^{\#,\$,\&}$	$9.7 \pm 2.5^{*,\$,\&}$	9.8 ± 3.1	$8.6 \pm 2.9^{*,\#,\&}$	$6.3 \pm 3.4^{*,\#,\$}$	< 0.001
CP, mL/min/kg *mm Hg	$5800 \pm 1871^{\$,\&}$	$5794 \pm 1653^{\$,\&}$	5710 ± 1868	$5251 \pm 1910^{*,\#,\&}$	$3720 \pm 2455^{*,\#,\$}$	< 0.001

Data are expressed as mean ± SD

*p < 0.05 versus normal BP; #p < 0.05 versus high-normal BP; $p < 0.05 versus grade 1 hypertension; &p < 0.05 versus grade 2 hypertension. See text for abbreviations and for definitions of hypertension

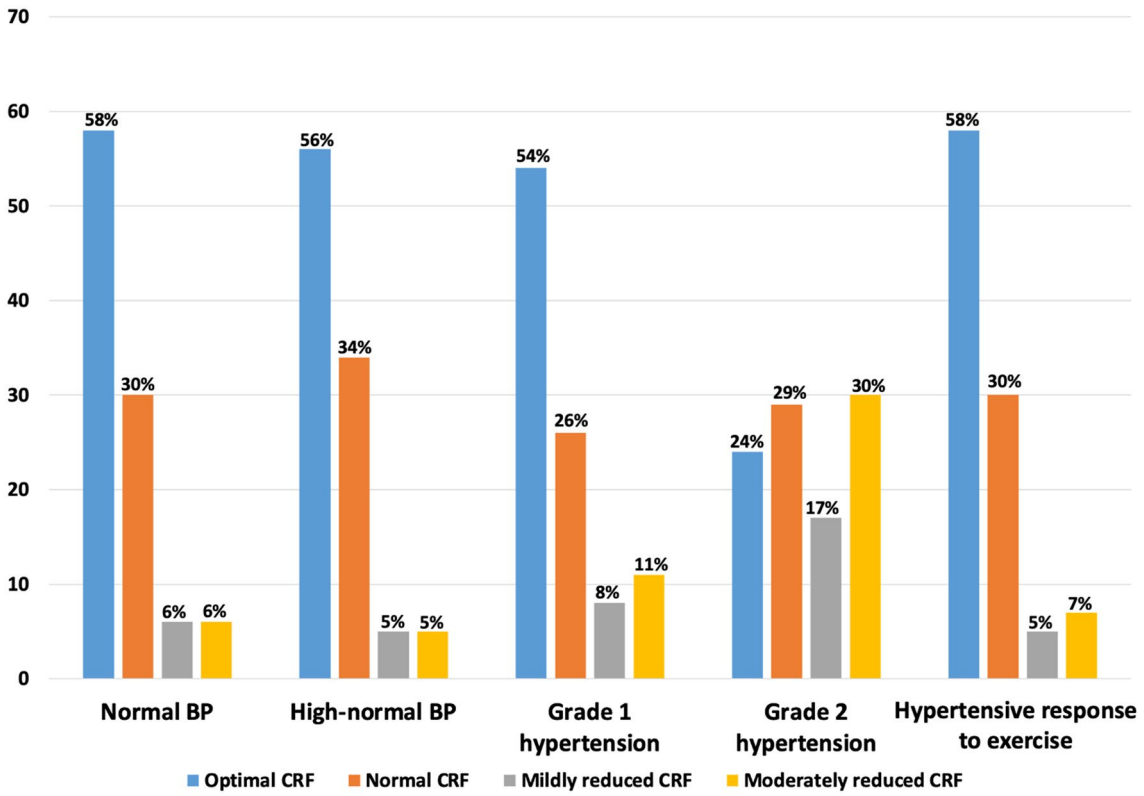

Fig. 1 Cardiorespiratory fitness distribution according to baseline and exercise BP response. The figure shows the distribution of cardiorespiratory fitness among the different values of BP response. Subjects with grade 1 and grade 2 hypertension have the lower values of cardiorespiratory fitness (columns in grey and yellow) than subjects with normal, high-normal and hypertensive response to exercise. However, these latter groups show from 5 to 7% of reduced cardiorespiratory fitness. Definitions of CRF and of hypertension are reported in the text

Although the clinical value of CRF impairment is not yet well defined, it deserves the utmost consideration. A growing body of evidence has showed that reduced CRF is associated with a higher incidence of CV disease, all-cause and CV mortality, increasing the predictive power of well-established risk factors, such as smoking, hypertension, diabetes and dyslipidemia [14, 15]. Each 1-MET increase in exercise capacity corresponded to a 21% decrease in the risk of CV mortality independently from age and sex [16]. Healthy subjects who achieved an exercise performance < 5 METs had a 4-fold increased risk of all-cause mortality compared with most fit individuals [17]. The main pathophysiological mechanisms proposed to explain this inverse association between CRF and mortality include a more cardio-protective CV risk profile in fit subjects, a reduced arrhythmogenic and thrombotic risk and a more favorable autonomic tone [5–7]. However, further studies are necessary to clarify the reasons why an optimal CRF reduces CV mortality and why, as demonstrated in this study, a small percentage of apparently normal and asymptomatic individuals showed a significantly reduced CRF. It is relevant, therefore, to familiarize in quantifying CRF in the general population and in studying its clinical correlation, as was done for the most common risk factors. Even though it may be plausible that sedentary individuals are more prone to hypertension and at the same time have a lower exercise tolerance, the reduction of CRF documented in this study in hypertensive patients may represent an example by which a subclinical pathology, such as hypertension, can early influence the hemodynamic response, aggravating its inherent risk. This finding can explain why low CRF level and physical inactivity have been associated to a higher incidence of hypertension at 5-year follow up [18], suggesting that increased fitness may attenuate an abnormal rise in BP [19]. This assumption particularly recommends the practice of physical activity in those hypertensive patients with lowest levels of CRF, as a further preventive measure. The physio-pathological response of a reduced CRF is the final result of general sympathetic over-activity, of increased vascular resistance, of decreased venous compliance, of impaired skeletal muscle vasodilatation and glucose uptake with a consequent corrupted oxygen delivery [20–25].

4.1 Exercise Stress Testing and HRE

The measurement of exercise BP by EST can provide additional information to the early identification of a subgroup of subjects with HRE, more likely to develop hypertension later in time [26]. Indeed, a positive association between HRE and incidence of hypertension has been previously demonstrated, consisting in a 3- to 4-fold greater risk for developing hypertension [27, 28]. Consistently, Caselli and colleagues showed that in young normotensive athletes, an abnormally high systolic and/or diastolic BP response to EST independently predicted the incidence of hypertension during an average follow-up of almost 7 years (hazard ratio 3.6 times compared with subjects normal BP levels) [29]. The relationship between an HRE and the development of hypertension may be explained by different mechanisms, such as increased angiotensin II levels, rise of catecholamines, aldosterone, and plasma renin activity, endothelial dysfunction and the presence of unrecognized masked hypertension [26, 30–32]. In such a context, it has been suggested that patients with normal BP levels at rest, but HRE, should undergo an out-of-office BP monitoring [33]. However, due to the lack of methodological standardization [34], univocal cut-offs for elevated peak SBP and particularly DBP response during EST have not yet been clearly established. In our population, we identified distribution ranges for peak SBP and DBP from 150 to 210 mmHg and from 60 to 110 mmHg, respectively. Our results are in agreement with those obtained in other studies, which have identified cut-off values for increased exercise SBP of 190 mmHg in women and 210 mmHg in men and of 115 mmHg for DBP [35, 36]. We found also a positive correlation between both resting and peak SBP and DBP, suggesting that hypertensive subjects are more likely to develop HRE. As previously documented [37–40], we also observed a linear increase in peak SBP and DBP with age, as a consequence of elevated aortic stiffness and impaired vasodilatation response to exercise. Furthermore, we found that peak oxygen uptake had a positive correlation with peak SBP and a negative correlation with peak DBP. This finding is concordant to a larger study conducted in 20,726 patients, in which subjects with an abnormal DBP response during exercise have a lower functional aerobic capacity and cardiorespiratory fitness compared to those with normal and borderline DBP [41]. Therefore, elevated peak DBP is a consequence of impaired peripheral vascular resistances, which also cause a reduction in peripheral oxygen extraction and in peak oxygen uptake, according to Fick law [42].

Some potential limitations of our study should be acknowledged. First of all, oxygen uptake was not directly measured during the exercise, but it was calculated by the ergometer software. Estimation of CRF from maximal exercise testing is typically obtained from the peak attained cycle ergometer workload using established prediction equations. However, it should be underlined that, although direct measure of VO_2 is more objective and precise, estimated CRF can be obtained also by other performance parameters easier to detect, such as METs and W/kg (as shown in Table 1), particularly in epidemiological studies involving large populations of apparently healthy individuals.

Since we did not perform a cardiopulmonary exercise testing, EST was self-terminated by the subjects when they claimed that they had achieved maximal effort and we did not use respiratory exchange ratio and duration as criteria of truly maximal exercise. Other limitations are related to the possible error in DBP measurement during exercise, its accuracy being sometimes questionable due to methodological difficulties. Also, those subjects usually treated with beta-blockers, who stopped treatment 72 h before EST, could show peak BP values that could have been higher due to the interruption of antihypertensive therapy. Moreover, we did not evaluate the relationship between ECG and echocardiographic parameters, exercise BP response and cardiorespiratory fitness. Finally, since our study has based on a single examination, we are not able to provide information about the development of hypertension during follow-up. Although we are conscious that routine mass EST cannot be feasible as a screening test, it should be performed and encouraged at least in more selected settings, such as occupational medicine and corporate wellness programs.

5 Conclusions

Corporate wellness projects represent an unmissable context in identifying asymptomatic individuals with an inadequate control of CV risk factors, such as hypertension, HRE and reduced CRF, anticipating therapeutic strategies and reducing clinical inertia. The inclusion of mass EST in the protocol helped to identify the relationship between early abnormal values of resting and/or exercise BP parameters and reduced cardiorespiratory fitness. This study expands the role of EST towards the assessment of reduced CRF and HRE, as still not evaluated CV risk factor in the general population.

Funding, grant/award info None.

Declarations

Conflict of interest None.

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