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Patterns of systolic blood pressure response at the end of exercise and mortality and morbidity in patients referred for exercise testing

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ABSTRACT

Objectives Peak exercise systolic blood pressure (SBP) is associated with future cardiovascular disease (CVD) and mortality. We aimed to evaluate the predictive value of different SBP patterns at the end of exercise with these outcomes.

Methods We studied 6329 adults (45% women) referred for exercise testing, with test duration of 6–14 min, maximal effort and valid SBP measurements at the end of exercise. The two last SBPs were indexed to work rate (mmHg/Watt), defining responses as: *drop* (negative change), *plateau* (no change), *slow* (lower tertile of increase), *intermediate* (middle tertile) and *steep* (upper tertile). Data were cross-linked with nationwide disease and mortality registries. Associations with all-cause mortality and incident CVD were analysed using Cox proportional hazards regression (hazard ratio (HR), 95% confidence interval), using slow SBP increase as reference, adjusted for sex, age, body mass index, baseline CVD (mortality analysis only), beta-blockers and exercise capacity (peak Watt).

Results The prevalence of SBP responses at the end of exercise were drop (1.1%), plateau (15.0%), slow (30.4%), intermediate (25.2%) and steep increase (28.3%). Follow-up was 8.8±3.4 years. Compared with a slow increase, the adjusted all-cause mortality risks were not statistically different for a drop (HR 1.16 (0.50–2.65)), plateau (HR 1.19 (0.85–1.66)), intermediate (HR 1.24 (0.93–1.66)) or steep SBP increase (HR 1.16 (0.89–1.52)). CVD risk was increased in those with a SBP drop (HR 3.10 (1.85–5.19), but not significantly for plateau (HR 1.17 (0.92–1.48)), intermediate or steep SBP increases (HRs 0.99–1.00).

Conclusion Subjects with a slow SBP increase at the end of exercise tended to have the lowest mortality risk, although no SBP response pattern predicted all-cause mortality independently. CVD risk was strongly increased in patients with a drop in SBP and tended to be increased (non-significantly) also in patients with a plateau in SBP at the end of exercise, in comparison with increasing SBP.

WHAT IS ALREADY KNOWN ON THIS TOPIC

⇒ It is known that the peak systolic blood pressure (SBP) during exercise testing is associated with future cardiovascular disease (CVD) and mortality. Furthermore, while it has already been shown that a decrease in SBP at the end of an incremental exercise test is indicative of severe cardiac disease, we now also examine the predictive value of other SBP response patterns at the end of an exercise test.

WHAT THIS STUDY ADDS

⇒ When taking CV risk factors into account, subjects with a slow SBP increase at the end of exercise tended to have the lowest mortality risk, although not statistically different from other SBP response patterns. Regarding the CVD risk, any increase in the SBP at the end of exercise (slow, intermediate or steep) was associated with similar risks, while an SBP plateau tended to confer a slightly (non-significant) higher risk, and drop in SBP was independently associated with CVD.

HOW THIS STUDY MIGHT AFFECT RESEARCH, PRACTICE OR POLICY

⇒ This study adds new knowledge on the SBP response to exercise, particularly at the end of the exercise test, related to all-cause mortality and incident CVD. Hereby, it may support clinicians in interpreting the SBP response during exercise testing, beyond evaluation of only peak SBP.

INTRODUCTION

During exercise testing, systolic blood pressure (SBP) is routinely measured throughout the test.[1] SBP is dependent on cardiac output (CO) and total peripheral resistance (TPR) and is expected to rise during exercise as a result of increasing CO.[2] The increase in SBP during exercise is also known to be different across age and sex.[3 4]

The peak SBP (SBPpeak) reached during exercise testing has been shown to be associated with future outcome. While a marked SBP response to exercise is associated with hypertension,[5–8] an impaired SBP response (low SBPpeak) is associated with cardiovascular

disease (CVD) and mortality.[9–13] Apart from the absolute SBPpeak, other aspects of the SBP response to exercise may also have a predictive value, in particular the SBP response in relation to work rate, which can be obtained by indexing the SBP increase to the increase in Watts (W),[3] oxygen uptake (VO_2)[14] or metabolic equivalents of task.[15] Recent research has underscored the importance of taking exercise capacity into account in the SBP assessment,[16 17] and considering the SBP relative to exercise intensity when interpreting the SBP response has been proposed.[18]

It is widely known that a drop in SBP during progressive exercise is a negative prognostic marker and a contraindication for continuation of the exercise test.[19] However, less is known about the implications of other SBP response patterns at the end of exercise. To our knowledge, there are no reports studying other SBP response types, such as a plateau or minimal increase in SBP at the end of a test.

We aimed to investigate the predictive value, in terms of all-cause mortality and incident CVD, of different SBP response patterns at the end of incremental bicycle exercise, in patients referred for exercise testing. Apart from a drop in SBP being a negative prognostic marker, as previously shown, we hypothesised that a plateau of the SBP response could be a marker of impaired cardiac performance, associated with worse clinical outcomes than increasing SBP at the end of exercise.

METHODS

Study cohort

From a clinical database of consecutive cycle ergometer exercise tests in 13 763 patients, we included subjects aged ≥18 years with maximal tests (rating of perceived exertion (RPE) ≥17 on the Borg scale) and exercise duration of 6–14 min. Tests with no SBP recordings during the last two minutes of exercise and fewer than two SBP readings during the last third of the test were excluded. We also excluded subjects with a body mass index (BMI) >40 kg/m^2 or arrhythmia during the test, where reliable SBP measurements could be difficult to obtain, as well as those with an SBP at rest <80 or >180 mm Hg (figure 1).

The data were cross-linked with nationwide registries on mortality, in- and outpatient hospital diagnoses, and

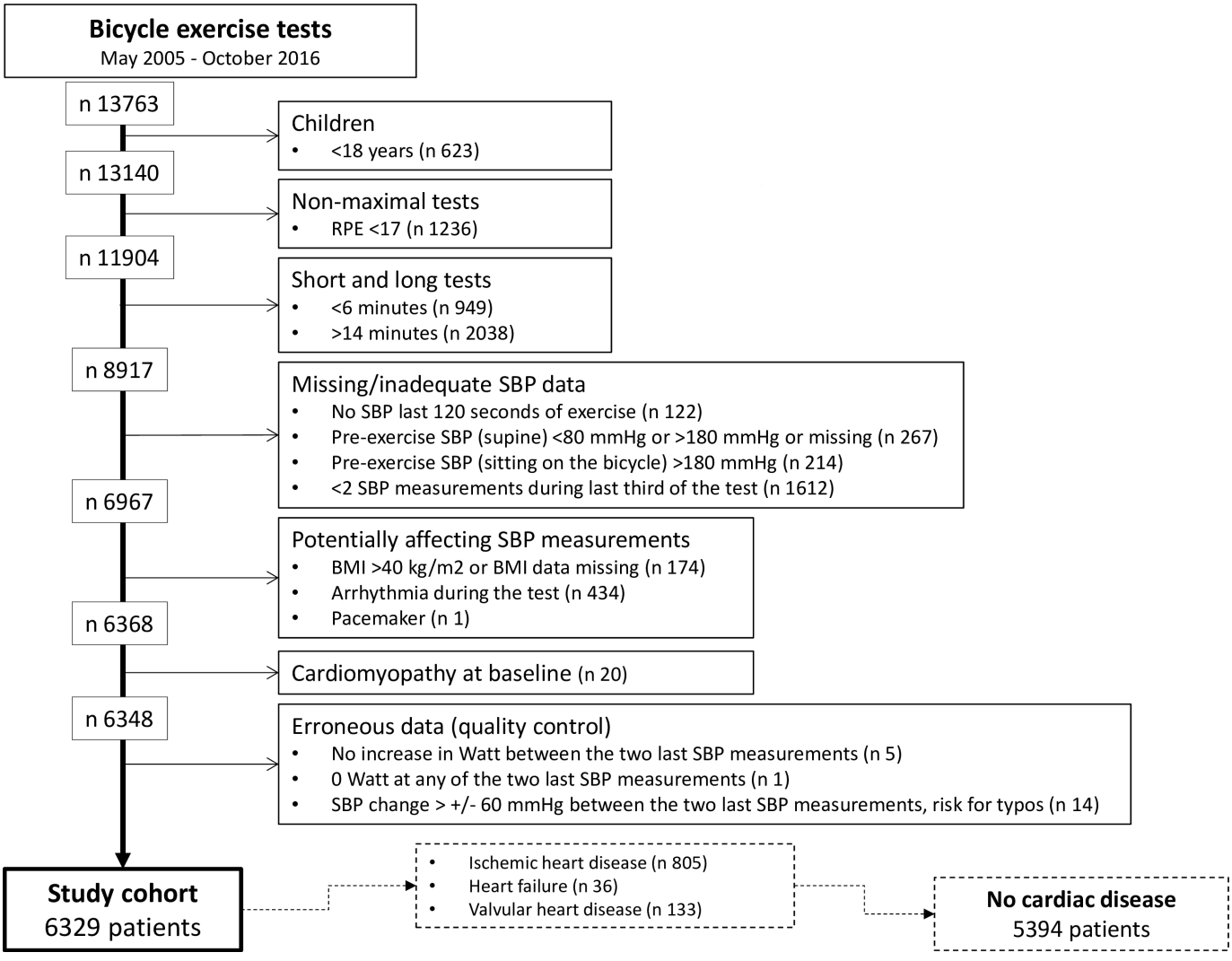

Figure 1 Study flow chart. BMI, body mass index; RPE, rate of perceived exertion; SBP, systolic blood pressure.

current medications, as detailed elsewhere,[3 20] used for cohort characterisation and outcomes during follow-up.

Exercise testing

All exercise tests were performed on an electrically braked bicycle (Rodby Inc, Karlskoga, Sweden). The testing procedure has been described elsewhere.[20] In brief, a ramp protocol was used, in most cases starting at 30 W in women and 50 W in men, followed by a continuous and consistent increase by 10 W/min in women and 15 W/min in men until maximal exertion, aiming at a total exercise time of 8–12 min.[1] The peak work rate in W (Wpeak) was recorded for each individual and was compared with normative values (Wpeak%pred), based on age, sex and height.[21]

Blood pressure (BP) measurements

Resting SBP and diastolic blood pressure (DBP) were measured in the supine position, before exercise after a few minutes of rest, with a stethoscope over the brachial artery and manual cuff inflation/deflation. Sitting on the bicycle, SBP was measured in the right arm using a Doppler probe over the radial artery, with manual cuff inflation/deflation, before the start of exercise and every 2–3 min during cycling. The highest SBP measured during exercise was defined as SBPpeak. We also calculated the SBP/W-slope over the entire exercise phase, by dividing the difference between SBP at supine rest and at peak exercise (last SBP measurement), by the increase in W between the same time points.[5]

SBP response patterns

The SBP response at the end of exercise was calculated as the difference between the two last SBP measurements, standardised to the difference in W between the same time points, mm Hg/W (figure 2). If there was less than 1 min between the two last SBP measurements, the third-to-last SBP measurement was used instead of the second-to-last. The SBP response patterns at the end of exercise were characterised as: *drop* (negative change), *plateau* (no change in SBP) or increase (positive change) in SBP. Any SBP increase was further divided into tertiles labelled *slow*, *intermediate* and *steep increase* (figure 2).

Ethics

The study complied with the Declaration of Helsinki and was approved by the regional Ethical Review Board in Linköping, Sweden (2018/141-31, 2019–05739). Informed consent was waived for this analysis of already collected data.

Statistics

The data were analysed using IBM SPSS Statistics (V.29). Comparisons of continuous data between multiple groups were performed using one-way analysis of variance. Where significant differences were found across groups, Tukey post hoc tests were performed to analyse differences compared with the reference group (slow SBP increase). Proportions were compared between groups using Pearson's χ^2 tests. Associations between SBP response pattern and outcomes were analysed using Cox proportional hazards regression. Hazard ratios (HRs) were presented with 95% confidence interval (CIs). Model 1 was unadjusted. Model 2 was adjusted for age, sex, BMI, hypertension, diabetes mellitus, history of CVD (heart failure (HF), ischaemic heart disease (IHD) or cerebrovascular disease) and medication with beta-blockers. In model 3, additional adjustment was made for exercise capacity (Wpeak). The SBP response at the end of exercise was analysed as a continuous variable and as the difference in risk between the SBP response categories, using a slow increase as the reference.

The primary outcome was all-cause mortality. The secondary outcome was incident CVD, defined as a new diagnosis of IHD (diagnoses I20–I25 according to the International Classification of Diseases (ICD)-10),[22] HF (ICD-10 diagnose I50) or CV death (any circulatory

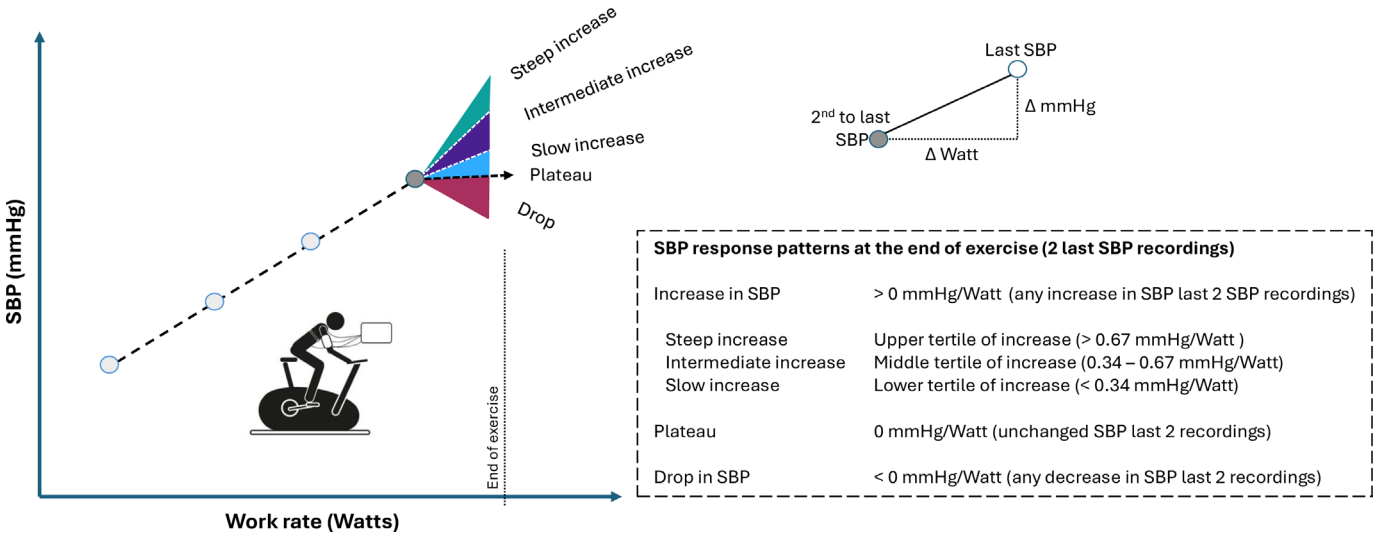

Figure 2 Categorisation of the SBP response patterns at the end of exercise. SBP, systolic blood pressure.

disease coded as the underlying cause of death in the mortality registry). For analyses of the secondary outcome, patients with a baseline diagnosis of IHD, HF or valvular heart disease were excluded (figure 2).

A two-sided significance level of 0.05 was used.

Equity, diversity and inclusion statement

The author group consisted of junior and senior researchers from three different countries. The first author is a woman, while the rest of the team consists of men. The study cohort included real-world patients referred for exercise testing with both sexes represented in similar proportions and a wide age range. We did not examine the effects of ethnicity or socioeconomic status.

Patient and public involvement statement

In this study, including retrospectively collected data, patients and the public were not involved in the planning of the research.

RESULTS

In this retrospective cohort study, 6329 patients (45% women) were included and followed for an average of 8.8±3.4 years.

Systolic blood pressure (SBP) at the end of exercise

The last SBP measurement was recorded on average 14.2±22.4 s before the end of the work phase, with a mean time difference between the two last SBP recordings of 100.5±36.5 s.

In 5311 patients (83.9%), there was an increase in SBP between the two last measurements, whereas in 950 patients (15.0%), there was a plateau, and in 68 patients (1.1%), there was a drop (table 1).

Compared with patients with a slow SBP increase at the end of exercise, resting SBP before exercise was higher in patients with a drop, plateau or a steep SBP increase, while SBPpeak was higher in patients with a steep SBP increase. The exercise capacity (Wpeak) was highest in patients with a slow SBP increase, although % of predicted Wpeak was similar in patients with a slow increase and a plateau (table 1). Online supplemental table 1 shows baseline comorbidities in the whole sample.

Systolic blood pressure (SBP) response pattern and mortality

There were 402 (6.4%) deaths during follow-up, with an incidence rate of 7.3 per 1000 person-years. There was a positive association between SBP increase at the end of exercise and mortality in the unadjusted model but no significant association in the fully adjusted model (HRadjusted 1.06 (95% CI 0.85 to 1.32) per 1 mm Hg/W increase) (online supplemental table 2). In a sensitivity analysis including only patients on beta-blockers, the risk estimates were further attenuated (online supplemental table 3).

In the unadjusted model, steeper SBP increases at the end of exercise were associated with increased mortality risks, as compared with a slow SBP increase. Similarly, after adjustment for demographics and baseline comorbidity (model 2), intermediate and steep SBP increases were associated with all-cause mortality (HRs 1.36–1.39, p <0.05), while after additional adjustment for exercise capacity, there were no significant differences in mortality risks between any of the SBP response categories (table 2).

Systolic blood pressure (SBP) response pattern and incident cardiovascular disease

In the 5394 patients with no cardiac disease at baseline (figure 1), there were 715 incident cases of IHD, HF or CV death during follow-up of 8.0±3.7 years, distributed between the SBP response categories as follows: drop n=16 (34.0%), plateau n=111 (13.3%), slow increase n=179 (10.8%), intermediate increase n=168 (12.3%) and steep increase n=241 (16.2%).

There was a positive association between SBP increase at the end of exercise and incident CVD in the unadjusted model, but no significant association in the fully adjusted model (HRadjusted 0.88 (0.74–1.05) per 1 mm Hg/W increase) (online supplemental table 2).

A drop in SBP at the end of exercise was associated with CVD (HRadjusted 3.10 (95% CI 1.85–5.19)). There was a trend towards a risk increase for increasing (intermediate or steep) SBP at the end of exercise and incident CVD, as well as for a SBP plateau, but in the adjusted models, there were no significant differences in risks for incident CVD between a plateau, intermediate or steep SBP increase, when compared with a slow SBP increase (table 2).

Survival curves and the unadjusted and adjusted risks of mortality and CVD for the different SBP response categories are seen in figure 3.

DISCUSSION

In patients referred to exercise testing, we found no independent prognostic values in the SBP pattern at the end of exercise, when taking both CV risk factors and exercise capacity into account. However, there was a trend towards a lower mortality risk with a slow SBP increase. A drop in SBP at the end of exercise was independently associated with incident CVD, while any type of SBP increase had similar CVD risks, and an SBP plateau tended to increase the risk of CVD when compared with a slow increase.

Slow systolic blood pressure (SBP) increase at the end of exercise

During exercise, CO must increase to meet the greater metabolic demands of the working muscles. The augmentation in CO is normally generated by a combined increase of stroke volume (SV) and heart rate (HR). At the same time, there is an active vasodilation in the arteries of the working skeletal muscles, generating a minor reduction in TPR. We anticipated that a lack of SBP increase during the final phase of incremental exercise, manifesting as either a drop, a plateau or perhaps even a slow SBP increase, would indicate inadequate increase of the CO

Table 1 Baseline characteristics and exercise test data by SBP response pattern at the end of a maximal exercise test

	SBP response at the end of exercise					
	Drop n=68	Plateau n=950	Slow increase n=1927 (reference)	Intermediate increase n=1592	Steep increase n=1792	P across all groups
Women, %	49.3%	45.8%	29.6%	53.6%	52.2%	<0.001
Age, years	58.8±13.1 **	55.4±14.6 ***	52.9±14.6	56.5±12.9 ***	59.5±12.0 ***	<0.001
BMI, kg/m^2	26.4±4.5	26.4±3.9	26.5±3.7	26.9±4.2 *	27.2±4.2 ***	<0.001
Prevalent disease or risk factors at baseline, n (%)						
IHD	13 (19.1%)	101 (10.6%)	222 (11.5%)	197 (12.4%)	272 (15.2%)	<0.001
Hypertension	17 (25.0%)	227 (23.9%)	438 (22.7%)	426 (26.8%)	586 (32.7%)	<0.001
Heart failure	3 (4.4%)	4 (0.4%)	10 (0.5%)	6 (0.4%)	13 (0.7%)	<0.001
Valvulopathy	7 (10.3%)	15 (1.6%)	49 (2.5%)	28 (1.8%)	34 (1.9%)	<0.001
Diabetes mellitus	1 (1.5%)	48 (5.1%)	83 (4.3%)	99 (6.2%)	153 (8.5%)	<0.001
Hyperlipidaemia	14 (20.6%)	131 (13.8%)	245 (12.7%)	243 (15.3%)	333 (18.6%)	<0.001
Atrial fibrillation	4 (5.9%)	21 (2.2%)	49 (2.5%)	44 (2.8%)	63 (3.5%)	0.13
COPD	1 (1.5%)	5 (0.5%)	9 (0.5%)	10 (0.6%)	22 (1.2%)	0.06
Cardiac medication at baseline, n (%)						
Antihypertensive†	17 (25.0%)	219 (23.1%)	414 (21.5%)	401 (25.2%)	550 (30.7%)	<0.001
Beta-blocker	17 (25.0%)	165 (17.4%)	297 (15.4%)	307 (19.3%)	393 (21.9%)	<0.001
Loop diuretics	1 (1.5%)	21 (2.2%)	24 (1.2%)	39 (2.4%)	50 (2.8%)	0.18
Exercise test data						
SBP_{rest}, mm Hg	141.4±20.1 *	137.9±18.0 ***	134.7±17.0	135.4±17.7	136.7±18.1 **	<0.001
DBP_{rest}, mm Hg	80.5±9.5	78.7±9.5	78.4±9.9	78.6±9.3	79.0±9.7	0.17
SBP_{peak}, mm Hg	185.1±30.1 *	193.2±25.7	195.1±23.9	195.5±25.1	201.6±25.7 ***	<0.001
SBP/Watt-slope (rest to peak)	0.21±0.21 ***	0.33±0.15	0.33±0.12	0.38±0.14 ***	0.45±0.16 ***	<0.001
HR_{peak}, 1/min	150.4±24.2 **	158.3±22.3	159.7±20.7	153.9±20.0 ***	148.8±19.4 ***	<0.001
HR_{peak}, % pred	93.3±12.9	96.1±10.7	95.6±9.7	94.1±10.0 ***	92.7±10.2 ***	<0.001
$Watt_{peak}$	158.5±40.0 ***	178.6±58.0 ***	195.6±55.6	165.2±49.9 ***	155.0±43.8 ***	<0.001
$Watt_{peak}$, % pred	91.0±14.0	95.8±14.7	95.0±14.6	93.3±14.3 **	90.5±13.6 ***	<0.001
Events during follow-up						
Deaths, n (%)	6 (8.8%)	57 (6.0%)	88 (4.6%)	101 (6.3%)	150 (8.4%)	<0.001

Slow, intermediate and steep increase represent tertiles of any SBP increase at the end of exercise.
Continuous data was presented as mean ± standard deviation. Results of the post-hoc analyses of continuous variables, when compared to a slow SBP increase at the end of exercise, were indicated as *, ** or ***, when significant.
* p<0.05
** p<0.01
*** p<0.001
† Angiotensin converting enzyme inhibitors, angiotensin receptor blockers, thiazide diuretics and calcium channel blockers were included as anti-hypertensive drugs.
COPD, chronic obstructive pulmonary disease; IHD, ischaemic heart disease; SBP, systolic blood pressure.

and inability to 'fill' the dilated circulation during exercise, which could imply risk of future CVD or death.

Rather, given the low mortality rate in patients with a slow SBP increase, as well as the higher absolute exercise capacity and the lower mean age as compared with patients with an SBP plateau, it could be argued that a slow SBP increase at the end of exercise reflects a healthy vascular response. By being able to appropriately reduce TPR, a healthy vascular system could accommodate the increase in BP generated by increasing CO. In the

Table 2 SBP response at the end of exercise and association to all-cause mortality and incident cardiovascular disease

SBP response at the end of exercise (patients/events)	Model 1, unadjusted HR (95% CI)	P	Model 2 HR (95% CI)	P	Model 3 HR (95% CI)	P
All-cause mortality						
Drop (68/6)	1.68 (0.74 to 3.84)	0.22	1.36 (0.59 to 3.11)	0.47	1.16 (0.50 to 2.65)	0.73
Plateau (950/57)	1.33 (0.95 to 1.86)	0.09	1.25 (0.90 to 1.75)	0.19	1.19 (0.85 to 1.66)	0.32
Slow increase (1927/88)	Reference		Reference		Reference	
Intermediate increase (1592/101)	1.37 (1.03 to 1.83)	0.029	1.36 (1.02 to 1.82)	0.036	1.24 (0.93 to 1.66)	0.14
Steep increase (1792/150)	1.77 (1.36 to 2.31)	<0.001	1.39 (1.06 to 1.81)	0.017	1.16 (0.89 to 1.52)	0.27
Incident CVD						
Drop (47/16)	3.68 (2.21 to 6.13)	<0.001	3.18 (1.90 to 5.31)	<0.001	3.10 (1.85 to 5.19)	<0.001
Plateau (833/111)	1.27 (1.00 to 1.60)	0.052	1.22 (0.96 to 1.54)	0.11	1.17 (0.92 to 1.48)	0.20
Slow increase (1660/179)	Reference		Reference		Reference	
Intermediate increase (1368/168)	1.14 (0.92 to 1.40)	0.38	1.09 (0.88 to 1.35)	0.42	1.00 (0.81 to 1.24)	0.98
Steep increase (1486/241)	1.51 (1.24 to 1.83)	<0.001	1.14 (0.94 to 1.39)	0.19	0.99 (0.81 to 1.21)	0.93

A drop, a plateau, and an intermediate or steep SBP increase at the end of exercise were analysed with a slow SBP increase at the end of exercise as reference. All-cause mortality risk was analysed in the full sample (N=6329), while incident CVD risk was analysed in the subgroup of patients with no cardiac disease at baseline (n=5394).
Model 2 was adjusted for age, sex, BMI, hypertension, diabetes mellitus, medication with betablockers, and history of CVD (in the analysis of all-cause mortality only). Model 3 was additionally adjusted for exercise capacity (Wpeak).
CI, confidence interval; CVD, cardiovascular disease; HR, hazard ratio; SBP, systolic blood pressure.

current study, patients with a slow SBP increase at the end of exercise tended to have the lowest prevalence of hypertension and hyperlipidaemia, while the prevalence was substantially higher in patients with a steep SBP increase (table 1), supporting a healthier vascular state with a levelling off of the SBP.

Importantly, a slow SBP increase at the end of exercise, as studied here, should be distinguished from a low absolute SBPpeak, which has been found to be associated with increased mortality risk.[11 16] In this study, the absolute SBPpeak did not differ significantly between patients with a plateau, slow or intermediate SBP increase, while patients with a steep SBP increase at the end of exercise had a higher SBPpeak.

Furthermore, although both HR and SV in healthy subjects may continue to increase throughout exercise until reaching VO_2max,[23] at maximal exercise intensity, there is a physiological plateau of the VO_2 despite further increase in intensity. An absent or slow SBP increase at the end of such maximal exercise may therefore in some subjects indicate an ability to continue exercising at, or above, maximal aerobic capacity, with a plateau in both VO_2 and SBP. Studies including cardiopulmonary exercise test data would further elucidate this hypothesis.

It should also be noted that the proportion of women in patients presenting with a slow SBP increase in this study was lower than within the other SBP response categories. It has previously been shown that healthy women have a steeper SBP/W-slope when calculated over the whole exercise phase.[3] Sex-specific cut-offs may be needed in future studies of the SBP response at the end of exercise, to better characterise potential sex differences in SBP response patterns.

Steep systolic blood pressure (SBP) increase at the end of exercise

A steep SBP increase at the end of exercise was associated with mortality and CVD in the unadjusted models and was more often seen in older patients with a higher prevalence of baseline comorbidity, including IHD, hypertension, diabetes and hyperlipidaemia. This is in line with previous studies showing that an exaggerated peak BP is more often seen in subjects with impaired endothelial vasodilator function.[24]

In the current study, patients with a steep SBP increase at the end of exercise also reached the highest absolute SBPpeak among all studied SBP response categories. This, as well as the finding of a low exercise capacity in comparison with the other studied groups, may be explained by both age[3] and CV comorbidity. A recent meta-analysis summarised the higher occurrence of CV risk factors (including arterial structure, lipid, metabolic, inflammatory and kidney function markers) among patients with an exaggerated exercise SBP response compared with normal responders, supporting a hypertensive response to exercise being an indicator of CV risk.[25] Our results suggest that the same may also be true for the SBP trajectory at the end of exercise, where a steep SBP increase reflects the underlying CV risk factor profile.

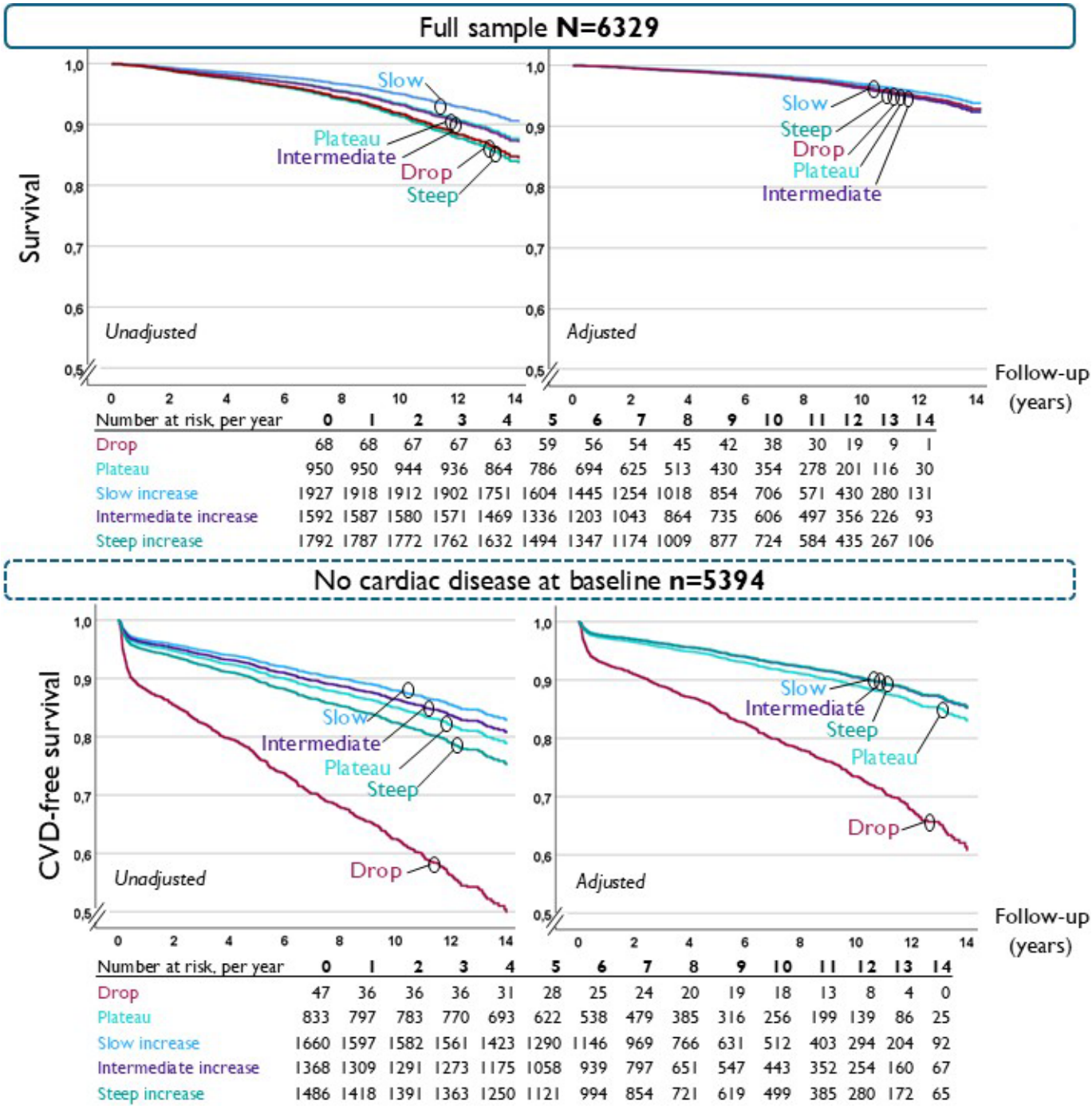

Figure 3 Cox regression survival curves for survival (upper panels) and CVD-free survival (lower panels), per category of SBP response at the end of a maximal bicycle exercise test. The SBP categories were based on the two last SBP measurements during exercise and were defined as drop (negative change), plateau (no change), slow SBP increase (lower tertile of increase), intermediate SBP increase (middle tertile) and steep SBP increase (upper tertile). A slow SBP increase was used as reference. Adjustments (in upper and lower right panels) included age, sex, BMI, hypertension, diabetes, medication with beta-blockers, exercise capacity (Wpeak) as well as history of CVD (upper right panel only). SBP, systolic blood pressure. CVD, cardiovascular disease. BMI, body mass index. W, Watt.

Systolic blood pressure (SBP) drop at the end of exercise

A drop in SBP at the end of a maximal exercise test was infrequent (1%) but independently associated with CVD. Hence, our results are consistent with previous assumptions that a drop in SBP is a strongly negative marker that may indicate pathology such as severe ischaemia[26 27] and in line with guidelines advocating discontinuation of an incremental exercise test in case the SBP drops >10 mmHg.[19] We found no significant association between a SBP drop and mortality, yet the absolute number of patients and events in that group was low and the CIs wide. Of note, this study included only maximal exercise

tests (RPE ≥17 and a duration of ≥6 min). Consequently, patients with a drop in SBP at submaximal exercise were excluded per protocol.

A plateau in systolic blood pressure (SBP) at the end of exercise

Our initial assumption was that a 'flat response' could be a milder manifestation of the same mechanisms underlying an SBP drop. The results were somewhat in line with this hypothesis, since a trend towards increased mortality and CVD risk was observed in patients with a SBP plateau in comparison with patients showing a slow SBP increase, yet the associations were not statistically significant in the fully adjusted models (table 2). This trend suggests that there might be a small-effect size association and that the a priori hypothesis merits further research in other cohorts to confirm or contrast these findings.

Clinical implications

SBP is routinely measured during clinical exercise testing, and an increased understanding of how to interpret different SBP responses is of great relevance to clinicians and exercise physiologists. Our findings support that only a drop in SBP had a strong and robust association with an increased risk of incident CVD, beyond that of traditional CV risk factors and exercise capacity. Further, we observed a mildly and non-significantly increased risk in patients experiencing a plateau in SBP at the end of the exercise test, which should be further investigated. We believe that this study contributes to the conceptual understanding of different SBP responses during exercise, beyond evaluation of the SBPpeak and their relevance in a clinical exercise testing context.

Limitations

We lack data on smoking habits in the current cohort, which may have an impact on both the SBP response and the outcome. Furthermore, the cut-offs for a slow and a steep SBP increase at the end of exercise are derived from the current clinical population of patients referred to exercise testing, which limits their generalisability. As the prevalence of women in the group with slow SBP increase at the end of exercise was low, the findings related to a slow SBP increase should be interpreted with caution in women. To further address this, sex specific cut-offs may be needed in future studies.

Conclusion

In this study of different trajectories of the SBP during the final phase of a maximal exercise test in patients referred to exercise testing, a drop in SBP was the only pattern independently and strongly associated with incident CVD. There was a trend towards higher mortality and CVD risk also for a plateau in the SBP response, although the effect size was small and did not reach statistical significance in the adjusted models. No statistically significant differences were observed for mortality risk among the different SBP trajectories, yet the lowest risk estimate was observed in those with slow SBP increase. Our findings illustrate the importance of interpreting the SBP response in the context of patient characteristics including comorbidities, age, sex and exercise capacity.

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