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Correlation between blood pressure response to sub maximal exercise and left ventricular hypertrophy

T. Gaprindashvili, R. Agladze, S. Khajalia,
I. Bochorishvili, Z. Pagava

Bokhua Memorial Center, Tbilisi, Georgia

Abstract

There is currently no consensus on the definition of normal BP (blood pressure) increase during physical activity and thus of the exaggerated BP response to exercise. A hypertensive response to exercise (HRE) is frequently observed in individuals without hypertension or other cardiovascular disease. However, mechanisms and clinical implication of HRE is not fully explained. Endothelial dysfunction and increased afterload contribute to development of HRE. From neurohormonal aspects. Studies showed that excess stimulation of sympathetic nervous system and augmented rise of angiotensin II seems to be important mechanism in HRE [1]. However, evidences for efficacy and outcomes of treatment of HRE to submaximal exertion in individuals without hypertension or low grade hypertension is scarce and therefore warrants further studies. Presents small cohort of patients a total of 38 women and 46 men, aged 52±8 years, without evidence of cardiovascular disease, with a mean resting BP of 142±7/75±7 mmHg had their BP measured at rest and during submaximal treadmill exercise. LV mass was measured using trans thoracic echocardiography. For the majority of the subjects on the initial evaluation we discovered echocardiographic evidence of concentric hypertrophy. Among the resting and exercise BP indices, sub maximal SBP was the strongest correlate of LV mass

(r=0.40, P<0.04). In multivariate analysis, maximal SBP was independently associated with LV mass an concentric LV hypertrophy, after adjustment for lean body mass and gender. Subaximal exercise SBP is a modest but still independent predictor of LV remodeling (grade and type of LV geometry) in population with prehypertension, I and II grade hypertension. These results raise the possibility that the SBP response to sub maximal exercise is an early marker of developing LV hypertrophy.

KEYWORDS: Hypertension; Exercise; Left ventricular hypertrophy

Introduction

As cardiac output increases during exercise, systolic blood pressure (BP) normally rises in responses to the increased demand of oxygen working load via from increased sympathetic tone. Some individuals present with abnormally exaggerated rise in systolic BP during exercise. This phenomenon is known as hypertensive response to exercise (HRE). However, there is no consensus about exact value of systolic BP to define a HRE. Studies conducted previously, defined HRE as a difference between peak and baseline systolic BP at least 60 mmHg in men and at least 50 mmHg in women during exercise testing or systolic BP exceeding the 90th percentile (approximately a systolic BP >210 mmHg in men and >190 mmHg in women) [2-5].

Mechanisms of hypertensive response to exercise

A hypertensive response to exercise (HRE) is frequently observed in individuals without hypertension or other cardiovascular disease. However, mechanisms and clinical implication of HRE is not fully elucidated. Increased stiffness of large artery and endothelial dysfunction contribute to development of HRE. From neurohormonal regulation factors, excess stimulation of sympathetic nervous system and augmented stimulation of angiotensin II production seems to be important contribution mechanism in HRE. Increasing evidences indicates that a HRE is associated with functional and structural abnormalities of left ventricle. A HRE harbors prognostic significance in future development of hypertension and increased cardiovascular events, particularly if a HRE is documented in moderate intensity

of exercise. Previous studies showed that HRE is not a benign phenomenon, however, currently, whether to treat a HRE is controversial with uncertain treatment strategy. Considering underlying mechanisms, angiotensin receptor blockers and beta blockers can be suggested in individuals with HRE, however, due to scarce evidence of proposed treatment warrants further studies. Also important role of this agents in regression of cardiac remodeling in this specific cohort [16].

While the impairment of endothelial function mainly contributes to HRE in younger individuals, arterial stiffness should be considered as the mechanism of HRE in older population. Increased arterial stiffness in elderly results in a reduction in arterial compliance, followed by a reduction of buffering capacity of BP, and finally leads to an abnormal increase in BP during exercise. Although a few studies report conflicting data, previous studies demonstrated positive association of HRE with large artery stiffness, assessed by pulse wave velocity, central pulse pressure, and mean arterial pressure [5].

Materials and Methods

Of the 84 eligible participants, men (n 46) women (n=38) with I and II degree hypertension (n=32) and pre hypertension (n=22). The study analysis included the subjects with an age range of 18-64 years (mean age 42.1±12.7 years) With or without left ventricular hypertrophy (LVH) described as normal, Concentric hypertrophy, Eccentric hypertrophy, concentric remodeling (see Table 1).

Table 1. *Description of LV hypertrophy and LV geometry*

LV GEPMETRY	LV MASS	RELATIVE WALL THICKNESS (RWT)
normal	≤115 g/m2 (men) or ≤95 g/m2 (women)	<0,42
Concentric hypertrophy	≥115 g/m2 (men) or >95 g/m2 (women)	>0,42
Eccentric hypertrophy	>115 g/m2 (men) or >95 g/m2 (women)	<0,42
Concnetric remodeling	≤115 g/m2 (men) or ≤95 g/m2 (women)	>0,42

Abbreviature [LV] – left ventricle.

To investigate the relation of exercise BP responses to submaximal and moderate exertion defined by us as (4 METSs) exercise with the incidence of hypertension and LVH.

LV mass detected by echocardiography calculated by formulae recommended in 2011y ESC/AHA guideline mass = 0.8[1.04 (LVEDD + IVST + PWT)3 2 (LVIDD)3] + 0.6, [2]/ where LVEDD is left ventricular end diastolic diameter measured from endocardial border) IVST and PWT are ventricular septum and posterior wall end diastolic thickness.

Submaximal Exercise Test

The submaximal exercise test was conducted using the modified Bruce protocol (Bruce Z) submaximal exertion considered ad 4.0 METs Stage 3 V (velocity 2.7 km/h) 12.0 % slope.

In all participants, BP was obtained on the treadmill in a standing position before the start of the exercise test. Participants then completed 2 stages of the submaximal exercise test (6 minutes of modified Bruce protocol), each stage lasting 2-3 minutes. Exercise BP was recorded at the midpoint of the first (2.7 mphat 0% grade) and second (2.7 mph at 5.0% grade) stages of the sub maximal exercise test. For the present investigation, the following exercise test variables were included: (1) exercise SBP, defined as SBP measured during the third stage of the submaximal exercise test; (2) exercise DBP, defined as DBP measured during the third stage of the submaximal exercise test; (3) SBP recovery, defined as exercise SBP minus SBP measured after 3 minutes of the submaximal exercise test (not taken in account for the present article).

As a secondary analysis, we created 4 new exercise BP variables: (1) change in exercise SBP, defined as exercise SBP minus preexercise standing SBP; (2) change in exercise DBP, defined as exercise DBP minus preexercise standing DBP;

Results

Association between BP responses to sub maximal exercise and LVH

The observation revealed sub maximal exertion in different age groups shown in Table 1 and 2. Overall, several statistically significant associations were noted. In multivariable-adjusted models, there was a positive association of exercise SBP with and cardiac remodeling revealed as left ventricular hypertrophy (LVH). All observed associations between exercise BP variables and indicators of subclinical CVD remained statistically significant.

Table 2. *Characteristics of study sample*

	MEN 46	WOMEN 38
Characteristics of patienes		
Median Age, y	52±8	50±6
Body Mass Index BMI, kg/m^2	26.4±3.2	24.3±3.1
Standing pre-exercise SBP, mm Hg	132±10	128±12
Standing pre-exercise DBP, mm Hg	79±12	76±13
Resting HR, beats per minute BPM	67±17	66±13
Sub maximal HR, BPM	120±18	122±20
SPB sub maximal exercise	158±18	155±16
LV hypertrophy	12±3	11±2
Exercise test data		
Exercise SBP, mm Hg moderate exertion (4 METs)	142±12	148±16
Exercise DBP, mm Hg moderate exertion (4METs)	76±14	73±15
Exercise SBP, mm Hg, Peak	168±16	164±14
Exercise DBP, mm Hg, Peak	88±8	91±7

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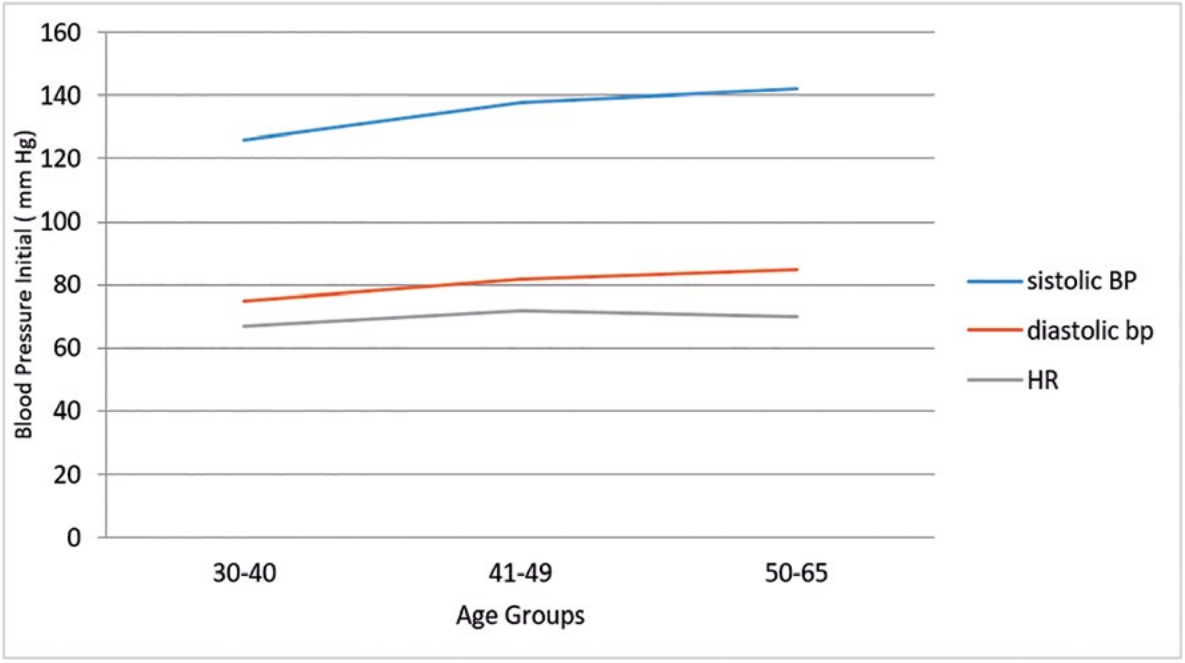

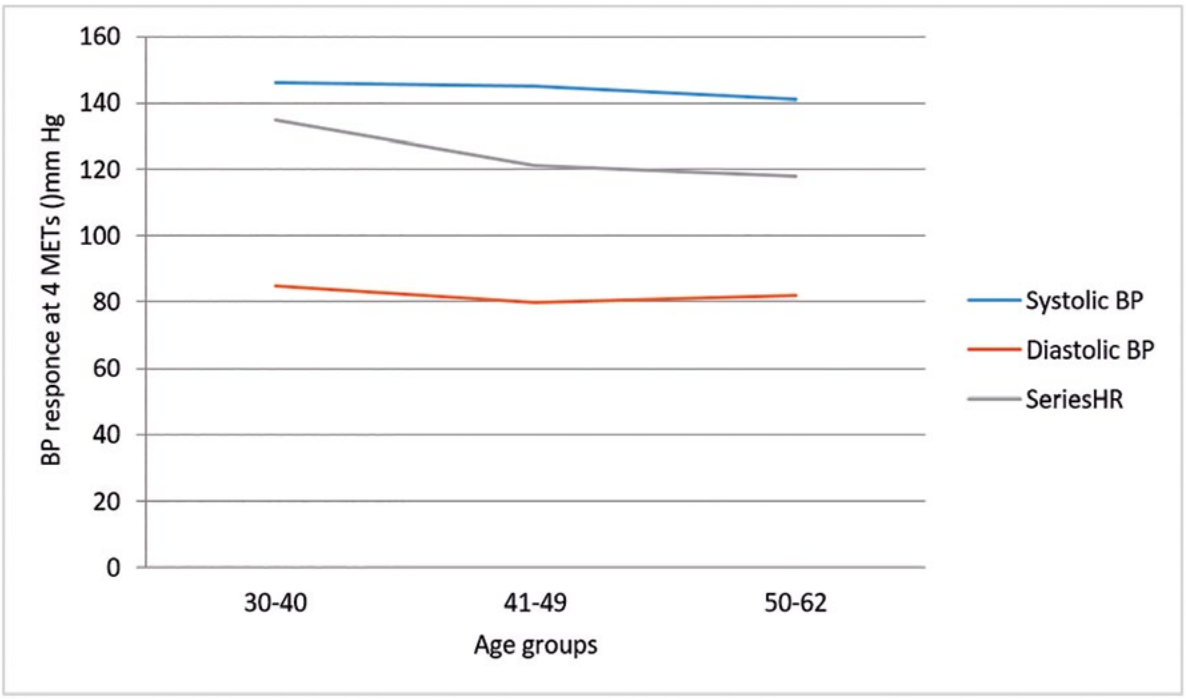

Fig. 1 (A) Initial BP values according to age groups. (B) 4 METS BP response.

Discussion

Hypertensive response

There is no definition of hypertensive response in normotensive asymptomatic standard patients, however there are proposed pressure figures. SBP max effort is commonly referred to at effort.

Although the relevance of the diagnosis and the prognosis of a HR is not fully clarified, some of these patients have an increased risk of future hypertension [1,11,12,] left ventricular hypertrophy or abnormal mobility [12,13] stroke [11], cardiovascular incidents [11-13], increased mortality, and endothelial dysfunction 48.

Results

Many studies regarding the diagnosis and prognosis of an abnormal response of the BP during physical exertion have been undertaken, studying response to sub maximal activity. However, until now, no widely excepted standard values have been

determined. The abnormal response of the BP during effort is related to an increased risk of future hypertension or cardiovascular events and mortality. In the event of an abnormal response in an asymptomatic patient, a study of primary hypertension should be considered. More frequent clinical control of resting BP, an echocardiogram, a 24 hour BP ambulatory monitoring as a more dynamic test, and at least annual repetition of the exercise test, to follow up the same abnormal response [8] As more studies are needed and a consensus regarding the abnormal response of the BP during exercise. Our study can be of clinical importance to prevent future adverse events linked to hypertension induced organ damage.

Mean while, based on the studies mentioned previously but considering sex and physical condition of the patient, and above all the clinical context, we can consider an abnormal.

Response of BP during effort in the following cases: SBP submaximal values greater than 158±18 mmHg in men and women respectively, may represent an exaggerated response in adults; Abnormal response of SBP in progressive tests was considered approximately more then 12 to 15 mmHg by MET, about 25 watts, although there are no standard values. The tension response at submaximal and maximal exercise, is dependent on age, sex and physical condition, which should be taken into account when assessing a response in normal exercise testing (Figure 1). Almost all patients with hypertensive response to sub maximal exertion revealed LVH as marker of the HIOD and cardiac remodeling leading to CVD.

Conclussion

Presented investigation is a continuation of the ideas and theory developed in works [8, 9]. In these ones, it is showed that the center of resistance of the tooth does not always exist and for those cases, a new concept of region of resistance is introduced. The results of this study are the developing of technique to determinate the location of the region of resistance analytically or by finding the set of translational action lines. The relation between the type of the region of resistance and the geometric parameters of the “tooth–periodontium” system is established. The process of transition from one type of region to another is considered. Also, it is experimentally shown, that the center of resistance may exist in bodies with the axis of symmetry of n-order ($n > 2$).

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