

Arterial stiffness increases in response to an acute arterial load challenge induced by an isometric handgrip in healthy individuals

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INTRODUCTION

Arterial stiffness plays an essential role in the development of cardiovascular disease and its complications, including mortality [1]. The Arterial Stiffness Index (SI_{DVP}) can be measured non-invasively by analyzing the arterial digital volume pulse (DVP) [2]. SI_{DVP} correlates well with resting blood pressure (BP) although it is unknown whether it changes with a BP elevation frequently occurring during the day in all people.

Various physiological provocations can cause temporary BP increases, including an isometric handgrip (IHG) stimulating the sympathetic nervous system and increasing arterial load, which represents an opposition that needs to be overcome during ejection by the left ventricle (LV). However, an IHG is also accompanied by acceleration of the heart rate (HR). Furthermore, it is unknown whether the potential effect of an increase in BP upon SI_{DVP} is independent of a changing HR.

We attempted to find out if a rapid change in BP, caused by an IHG, can influence SI_{DVP} and, if such an association exists, whether it depends on the effects of an accelerating HR in healthy people.

METHODS

A total of 22 healthy adult volunteers were recruited. The participants were informed about the study, and written consent was obtained. The local Ethics Committee approved the study protocol.

As for the inclusion criteria, none of the study subjects could be on any medication or suffer from chronic conditions. Out of 23 screened subjects, one was rejected

due to high resting blood pressure (value >140/90 mm Hg).

Isometric handgrip exercise

Resting brachial BP was obtained using an oscillometric method (705 IT, Omron Healthcare Co. Ltd., Kyoto, Japan). Maximal IHG strength was measured in a sitting position using the Jamar hydraulic hand dynamometer (Sammons Preston Rolyan, Bolingbrook, IL, US). Subsequently, the participants were instructed to compress the dynamometer for 3 minutes and maintain 30% of their previously determined maximal compression pressure. Systolic (SBP) and diastolic (DBP) blood pressure, pulse pressure (PP), and the heart rate (HR) were assessed at rest and between 2.45 and 3.15 minutes after the beginning of the IHG. The IHG was performed with the dominant hand (right hand for all participants), BP and SI_{DVP} were estimated on the contralateral limb.

Stiffness Index by digital volume pulse analysis

The digital volume pulse waveforms were recorded at rest and between the 2.45-minute and 3.15-minute marks of the 30% maximal IHG using a finger photoplethysmograph (Pulse Trace 2000, MicroMedical, Rhymney, UK). SI_{DVP} which is an estimate of pulse wave velocity and arterial stiffness of the large arteries, was obtained from the subject's body height (h) divided by the time between the systolic and diastolic peaks of the DVP. Measurement represents the mean SI_{DVP} of 6 consecutive beats during 10 seconds.

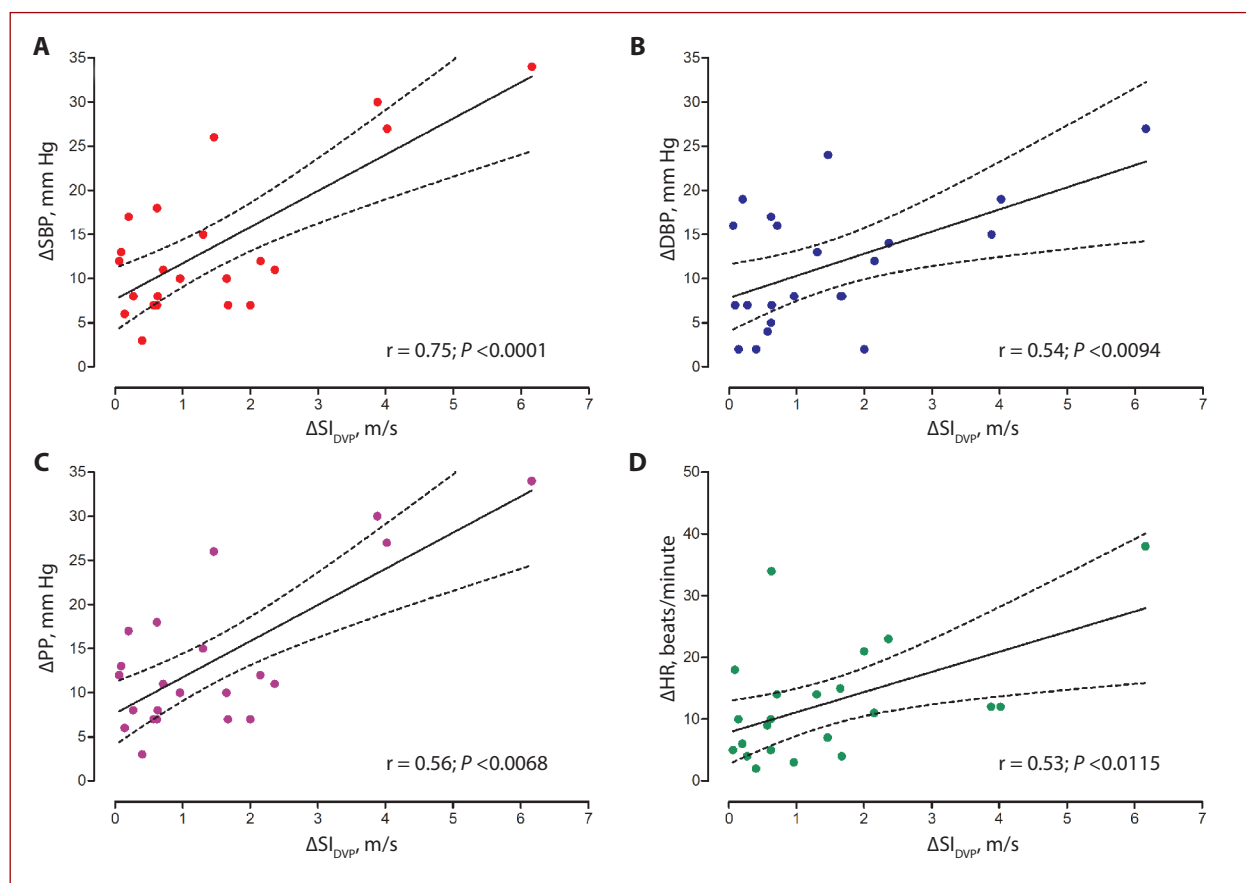


Figure 1. Correlation between the increase (Δ) in systolic BP, diastolic BP, PP pressure, heart rate, and the increase (Δ) in SI_{DVP} at the peak of handgrip exercise

Abbreviations: ΔSBP , increase in systolic blood pressure; ΔDBP – increase in diastolic blood pressure; ΔHR , increase in the heart rate; ΔPP , increase in pulse pressure; ΔSI_{DVP} increase in the Stiffness Index

Statistical analysis

The continuous data distribution was normal (the D'Agostino-Pearson normality test); the results are reported as the mean (standard deviation [SD]). The rest-to-peak figures of the IHG measurements in respect of the SI_{DVP} , SBP, DBP, and HR were compared using paired t-tests. Correlations between the rest-to-peak of the IHG differences (Δ) of these parameters, i.e., ΔSI_{DVP} and ΔSBP , ΔPP or ΔDBP , or ΔHR , were analyzed through the Pearson correlation. Linear regression adjusted to the ΔHR was applied to study the relation between ΔSI_{DVP} and ΔSBP or ΔDBP . All statistical analyses were considered to be significant if $P < 0.05$. Analyses were conducted using MedCalc® statistical software version 20.014 (MedCalc Software Ltd., Ostend, Belgium).

RESULTS AND DISCUSSION

Clinical characteristics and results of the IHG are shown in Supplementary material, *Table S1*. The mean age of the studied subjects was 35.4 (12.3) years and there were 12 women. On average, the participants were slightly overweight, with a body mass index (BMI) of 26.0 (4.2) kg/m^2 , and the maximal strength of the handgrip was 29.8 (7.9) kg.

The mean values in respect of the SBP, DBP, BPmean, PP, HR, and SI at rest and near the end of the IHG are presented and compared in Supplementary material, *Table S1*. All were within normal values. Compared to the rest, the IHG caused significant increases in all measured parameters.

Hemodynamic and Stiffness Index response to IHG maneuver

The initial study determined that the 3-minute IHG resulted in maximal hemodynamic response. Prolonging the IHG results in muscle weakness and diminished IHG. The increased SI_{DVP} and BP return to pre-exercise level within 1–3 minutes after cessation of the handgrip (data not shown).

Correlation of ΔSI_{DVP} with changes in SBP, DBP, PP, and HR during IHG

Figure 1 shows the results of the Pearson correlations between ΔSI_{DVP} and ΔSBP , ΔDBP , ΔPP , and ΔHR during the IHG. All correlations were positive and significant. Similarly, ΔBP_{mean} was significantly correlated with ΔSI_{DVP} (data not shown).

Linear regression models adjusted for Δ HR show that Δ SI_{DVP} was significantly related to Δ SBP ($P = 0.0003$; adjusted model's R^2 0.61) and Δ DBP ($P = 0.0212$; adjusted model's R^2 0.40). In other words, Δ SI_{DVP} during the IHG was significantly correlated with Δ SBP or Δ DBP, regardless of the effects of Δ HR.

We demonstrate that an IHG-induced surge in BP increases arterial stiffness, regardless of the HR effects. An IHG is a physiological maneuver that stimulates the sympathetic nervous system, increases vascular resistance, and elevates BP. Consequently, raised SBP and DBP transfer the mechanical load from elastin to collagen fibers, distending and deforming the arterial walls and, thus, increasing arterial stiffness, which translates further into an increased LV arterial load.

Augmented arterial stiffness is a significant risk factor for cardiovascular events, particularly in elderly subjects, as well as in those suffering from diabetes, chronic renal disease, and hypertension [1]. The measurement of arterial stiffness is recommended in people with cardiovascular risk factors and diseases, particularly hypertension [3, 4]. Arterial stiffness is frequently described as segmental and local pulse wave velocity, or a general marker, such as the Stiffness Index. Prolonged exposure to hypertension results in adaptive structural changes in the arterial wall, which is difficult to reverse; therefore, acute decreases in BP do not reduce the markers of arterial stiffness. However, a long-term BP reduction decreases arterial stiffness.

In our study, the sustained (approximately) 3-minute increase in BP significantly augmented SI_{DVP}. Noteworthy is the fact that, on average, the increases in both SBP and DBP were within normal range values during the IHG. Nevertheless, these increases were sufficient to affect the distensibility of the arterial walls in healthy people.

Several investigators examined short-term vascular hemodynamics related to various forms of exercise in both young adults and the elderly [5–7]. However, BP and arterial stiffness were measured after a period of rest. In contrast, our study examines arterial stiffness at the peak of the IHG, during peak SBP and DBP. In patients with hypertension, an exaggerated response to exercise is frequently observed, resulting in higher arterial stiffness measures.

Blood pressure surges are commonly observed in healthy people and those with hypertension, e.g., during exercise, emotions, or after awakening [8]. Arterial stiffness measured during such events may likely differ from data acquired in different circumstances.

It was also noticed that an increase in sympathetic activity influences the mechanical properties of arterial vessels through various mechanisms, including the HR increase [9]. A positive relationship between the HR and high arterial stiffness was reported in normotensive and hypertensive individuals [10]. We have also noted similar findings, with the increase in the HR correlating positively with the increase in SI_{DVP}. However, the impacts of BP upon

arterial stiffness appear to be unaffected by the potential influence of the HR. Nevertheless, it is currently difficult to separate the effect of BP and the heart rate on SI_{DVP}.

Study limitation

We studied only healthy subjects to evaluate whether arterial stiffness is a dynamic feature. Consequently, conclusions should not be extrapolated to patients with existing cardiovascular disease. Similar or even exaggerated responses could likely be observed in individuals suffering from hypertension. Nevertheless, this will require a separate study. Moreover, additional investigation is warranted in healthy populations, including improved sex balance and older subjects.

In summary, we showed that in healthy people a rapid increase in BP triggered by the 3-minute IHG elevates arterial stiffness, regardless of the change in the HR. As arterial stiffness depends on the current BP, it appears that the results of arterial stiffness measurements should be reported together with BP readings.

Supplementary material

Supplementary material is available at https://journals.viamedica.pl/kardiologia_polska.

Article information

Conflict of interest: None declared.

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