Η σημασία της αρτηριακής σκληρίας στην εκτίμηση της διαστολικής δυσλειτουργίας στην υπέρταση. Θεραπευτικές παρεμβάσεις

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Αττικό Νοσοκομείο
Prevalence of raised blood pressure*, ages 25+, age standardized
Both sexes, 2008

Prevalence of raised blood pressure (%)
- <35
- 35–39.9
- 40–44.9
- 45–49.9
- ≥50
- Data not available
- Not applicable

* SBP ≥140 and/or DBP ≥90 or using medication to lower blood pressure

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Data Source: World Health Organization
Map Production: Public Health Information and Geographic Information Systems (GIS)
World Health Organization
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Hypertension, Hyperlipidemia, Diabetes Mellitus, Smoking
Worldwide blood pressure control in treated hypertensive patients

- Canada: 66.0
- USA: 63.1
- Mexico: 21.8
- England: 29.2
- Greece: 49.5
- Turkey: 19.8
- Germany: 33.6
- Japan: 55.7
- China: 28.8
- Taiwan: 18.0
- Egypt: 33.5
- South Africa: 47.6
- Italy: 37.5

Arterial hypertension is a highly widespread cardiovascular risk factor in the general population and particularly in advanced age.

Though randomized pharmacological trials have shown a marked reduction in cardiovascular morbidity and mortality in treated elderly essential hypertensive patients, adequate blood pressure control and effective cardiovascular prevention is achieved only in a minor portion of this population in clinical practice.

This contrast may be explained by the fact that in elderly individuals, the presence of multiple cardiovascular risk factors, target organ damage, associated cardiovascular conditions and a large blood pressure variability all make achievement of optimal blood pressure control very difficult.
Subclinical organ damage represents an intermediate stage in the continuum of vascular disease and a determinant of overall cardiovascular risk.

Signs of subclinical organ involvement, otherwise called target organ damage (TOD), should be investigated thoroughly in hypertensive patients with valid noninvasive methods at baseline leading to proper treatment choices and during follow-up of hypertensive patients in order to determine reduction of cardiovascular risk and treatment success.

TODs include arterial stiffness, left ventricular hypertrophy, left ventricular diastolic dysfunction, left atrium (LA) enlargement, impaired coronary flow reserve (CFR), increased intima-media thickness (IMT), elevated levels of microalbuminuria, and cognitive impairment.
2013 ESH/ESC Guidelines for the management of arterial hypertension

The Task Force for the management of arterial hypertension of the European Society of Hypertension (ESH) and of the European Society of Cardiology (ESC)

## Epidemiology and Total Cardiovascular Risk

<table>
<thead>
<tr>
<th>Other risk factors, asymptomatic organ damage or disease</th>
<th>Blood Pressure (mmHg)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>High normal SBP 130–139 or DBP 85–89</td>
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<tr>
<td>No other RF</td>
<td>Low risk</td>
</tr>
<tr>
<td>1–2 RF</td>
<td>Low risk</td>
</tr>
<tr>
<td>≥3 RF</td>
<td>Low to Moderate risk</td>
</tr>
<tr>
<td>OD, CKD stage 3 or diabetes</td>
<td>Moderate to high risk</td>
</tr>
<tr>
<td>Symptomatic CVD, CKD stage ≥4 or diabetes with OD/RFs</td>
<td>Very high risk</td>
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</tbody>
</table>

**Figure 1** Stratification of total CV risk in categories of low, moderate, high and very high risk according to SBP and DBP and prevalence of RFs, asymptomatic OD, diabetes, CKD stage or symptomatic CVD. Subjects with a high normal office but a raised out-of-office BP (masked hypertension) have a CV risk in the hypertension range. Subjects with a high office BP but normal out-of-office BP (white-coat hypertension), particularly if there is no diabetes, OD, CVD or CKD, have lower risk than sustained hypertension for the same office BP.
Arterial stiffness represents an independent predictor for cardiovascular mortality in patients with essential hypertension, diabetes, or end-stage renal disease.

Arterial stiffness can noninvasively be estimated by measuring pulse wave velocity (PWV), the velocity of the pulse wave to travel a given distance between two sites of the arterial system.

PWV measurement is widely used as an index of large artery compliance.
Diastolic heart failure (HFpEF) is characterized by the symptoms and signs of heart failure, a preserved ejection fraction and abnormal left ventricular (LV) diastolic function caused by a decreased LV compliance and relaxation.

The diagnosis of diastolic heart failure is often one of exclusion. The majority of patients with HFpEF have a history of hypertension. Hypertension induces a compensatory thickening of the ventricular wall in an attempt to normalize wall stress, which results in LV concentric hypertrophy, which in turn decreases LV compliance and LV diastolic filling.

There is an abnormal accumulation of fibrillar collagen accompanying the hypertension-induced LV hypertrophy, which is also associated with decreased compliance and LV diastolic dysfunction.
The filling phase of the cardiac cycle moves along the end-diastolic pressure-volume relationship or passive filling curve of the ventricle. A shift of the curve from A to B indicates that a higher LV pressure is required to distend the left ventricle to a similar volume, in this case from 100 to 120 ml.

Fig. Abnormal left ventricular (LV) diastolic function caused by a decreased LV compliance and relaxation.
## Diastolic Dysfunction Grades

LV relaxation, filling pressures and 2D and Doppler findings according to LV diastolic function

<table>
<thead>
<tr>
<th></th>
<th>Normal</th>
<th>Grade I</th>
<th>Grade II</th>
<th>Grade III</th>
</tr>
</thead>
<tbody>
<tr>
<td>LV Relaxation</td>
<td>N</td>
<td>Impaired</td>
<td>Impaired</td>
<td>Impaired</td>
</tr>
<tr>
<td>LAP</td>
<td>N</td>
<td>Low or N</td>
<td>Elevated</td>
<td>Elevated</td>
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<tr>
<td>Mitral E/A Ratio</td>
<td>≥0.8</td>
<td>≤ 0.8</td>
<td>&gt;0.8 to &lt;2</td>
<td>&gt;2</td>
</tr>
<tr>
<td>Average E/e' ratio</td>
<td>&lt;10</td>
<td>&lt;10</td>
<td>10-14</td>
<td>&gt;14</td>
</tr>
<tr>
<td>Peak TR velocity</td>
<td>&lt;2.8</td>
<td>&lt;2.8</td>
<td>&gt;2.8</td>
<td>&gt;2.8</td>
</tr>
<tr>
<td>LA volume index</td>
<td>N</td>
<td>N or Increased</td>
<td>Increased</td>
<td>Increased</td>
</tr>
</tbody>
</table>

### Diastolic Heart Failure

- Normal
- Impaired Relaxation
- Pseudonormal
- Restrictive

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*ASE/EACVI Guidelines And Standards, J Am Soc Echocardiogr 2016*
Left atrium (LA) enlargement is caused by hypertension and is detected earlier than left ventricular hypertrophy or dilatation in the course of hypertensive heart disease.

Office pulse pressure is the only non-invasive index of arterial stiffness, which can strongly and independently determine left atrial size in non-dipper hypertensive patients with newly diagnosed essential hypertension.

By ROC analysis, the cutoff value of PP > 57.5 mm Hg predicted the presence of LA enlargement in non-dippers.
An independent relationship of PWV with left ventricular diastolic dysfunction, and LA enlargement was revealed.

When dipping status was taken into account, PWV associated with left ventricular diastolic dysfunction, and LA enlargement in dippers.

It seems that in patients with increased 24-h hypertensive burden and subsequent augmented cardiovascular risk, nondippers, a different TOD pattern compared to dippers is observed.
Although not proven, an improvement in aortic stiffness during hypertension treatment may reduce CV morbidity and mortality.
PWV improvement due to successful anti-hypertensive treatment is evident when at least moderately impaired arterial stiffness is present at baseline, while the magnitude of PWV decrease is independent of the observed BP decrease.

(a) PWV0 increases with age in treated uncontrolled hypertensives,
(b) PWV0 remains almost unchanged in controlled hypertensives,
(c) PWV0 decreases in those controlled hypertensives with a baseline PWV >12.4 m/s who are older with more severe baseline hypertension
(d) Using ROC curve, the cutoff level of baseline PWV >10.4 m/s predicts aortic stiffness improvement after 3 years of successful treatment.
Lifestyle changes like physical activity, smoking cessation, salt and weight reduction, and balanced diet might decrease arterial stiffness; however, findings are conflicting.

It seems that the administration of statins might lead to a neutral (pravastatin), intermediate (simvastatin), or positive (fluvastatin) effect regarding PWV improvement in patients with hypertension and hyperlipidemia.

All antihypertensive drugs reduce arterial stiffness, since the reduction of BP unloads the stiff components of the arterial wall, leading to a passive decrease of PWV. A recent meta-analysis and meta-regression analysis of RCTs documented that ACE inhibitors and ARBs reduce PWV beyond BP reduction. However, it is not clear whether they are superior to other antihypertensive agents in their effect on arterial stiffness.

Is Arterial Hypertension Control Enough to Improve Aortic Stiffness in Untreated Patients With Hypertension? A 3-Year Follow-Up Study

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Changes in PWV during long-term studies are not dependent on changes in mean BP leading to the conclusion of a **decoupling of PWV and BP when control of BP is effective for a long period of time.**

It is suggested that the reduction in PWV occurs in **2 phases** in treated patients with hypertension; in the first one, **PWV falls in parallel with the short term fall in BP**, and in the second phase, **PWV continues to fall significantly, despite a limited BP reduction.**

The second phase is more likely related to the arterial remodeling occurring in response to long-term administration of antihypertensive drugs and/or normalization of other CV risk factors.
We revealed the significance of an already activated RAAS with subsequent impaired baseline aortic stiffness in order for RAAS inhibitors to exhibit their destiffening effect.

We believe that antihypertensive treatment reduces future CV events when it takes into consideration both increased BP and impaired aortic stiffness at baseline and targets both BP decrease and aortic stiffness improvement as well.

Hypertensive patients need a follow-up in arterial stiffness.
Sauna exposure leads to improved arterial compliance: Findings from a non-randomised experimental study.

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Author information

Abstract

Background Heat therapy has been suggested to improve cardiovascular function. However, the effects of hot sauna exposure on arterial compliance and the dynamics of blood flow and pressure have not been well documented. Thus, we investigated the short-term effects of sauna bathing on arterial stiffness and haemodynamics. Design The design was an experimental non-randomised study. Methods There were 102 asymptomatic participants (mean age, 51.9 years) who had at least one cardiovascular risk factor. Participants were exposed to a single sauna session (duration: 30 min; temperature: 73°C; humidity: 10-20%). Pulse wave velocity, augmentation index, heart rate, blood pressure, mean arterial pressure, pulse pressure, augmented pressure and left ventricular ejection time were assessed before, immediately after, and 30 min after a single sauna session. Results Sauna bathing led to reductions in pulse wave velocity, blood pressure, mean arterial pressure and left ventricular ejection time. Mean pulse wave velocity value before sauna was 9.8 m/s and decreased to 8.6 m/s immediately after sauna bathing (p < 0.001 for difference), and was 9.0 m/s after the 30-minute recovery period (p < 0.001 for analysis of variance). Systolic blood pressure was 137 mm Hg before sauna bathing, decreasing to 130 mm Hg after sauna (p < 0.001), which remained sustained during the 30-minute recovery phase (p < 0.001 for analysis of variance). After a single sauna session, diastolic blood pressure decreased from 82 to 75 mm Hg, mean arterial pressure from 99.4 to 93.6 mm Hg and left ventricular ejection time from 307 to 278 m/s (p < 0.001 for all differences). Pulse pressure was 42.7 mm Hg before the sauna, 44.9 mm Hg immediately after the sauna, and reduced to 39.3 mm Hg after 30-minutes recovery (p < 0.001 for analysis of variance). Heart rate increased from 65 to 81 beats/min post-sauna (p < 0.001); there were no significant changes for augmented pressure and pulse pressure amplification. Conclusion This study shows that pulse wave velocity, systolic blood pressure, diastolic blood pressure, mean arterial pressure, left ventricular ejection time and diastolic time decreased immediately after a 30-minute sauna session. Decreases in systolic blood pressure and left ventricular ejection time were sustained during the 30-minute recovery phase.
Diastolic dysfunction and arterial stiffness: the chicken or the egg

J. Daemen

Arterial stiffness by itself is associated with left ventricular diastolic dysfunction.

Both left ventricular hypertrophy and diastolic dysfunction have been linked to cardiovascular morbidity and mortality, irrespective of blood pressure.

Whether arterial stiffness truly precedes and predicts the development of left ventricular dysfunction or whether both parameters are simply the result of the ageing of the cardiovascular system remains the question.