Impact of hypertension in the heart

Tomáš Seeman

Department of Pediatrics and Transplantation Center, University Hospital Motol, 2nd Faculty of Medicine, Charles University Prague, Czech Republic
• Cardiac consequences of hypertension

• Assessment of cardiac target organ damage (TOD)

• Prognostic value of the cardiac TOD in hypertensive children

• Is cardiac TOD reversible by antihypert. therapy?
2016 European Society of Hypertension guidelines for the management of high blood pressure in children and adolescents

Guidelines

J Hypertens 2016, 34: 1887-1920
Clinical cardiovascular consequences of hypertension

Adults:

Hypertension is a strong risk factor for

1. **cardiovascular** morbidity (MI, stroke) and mortality in general population
2. progression of chronic kidney disease (CKD)
Children:

Hypertension is a proven risk factor for


3. stroke in adulthood (Leiba et al., Ped Neph 2016, 31: 485-492)
Causes of death in children with ESRD

*Dutch Cohort Study (n=249)*

- Cardiovascular: 41%
- Infection: 21%
- Malignancies: 10%
- Complication of therapy: 8%
- Refusal further therapy: 11%
- Other: 9%

Groothoff et al., Kidney Int 2002, 61:621-629
Children **without** renal disease

**mortality** and clinical events of hypertension-associated cardiovascular morbidity (stroke, myocard infarction) are **very rare** (no population based data similar to adults)

**surrogate markers** of cardiovascular morbidity

= **subclinical changes** of the (mainly) heart induced by increased BP

= **target organ damage (TOD)**
Complications of Hypertension (adults): Target Organ Damage

Hypertension

- Hemorrhage, Stroke
- Retinopathy
- Peripheral Vascular Disease
- LVH, CHD, CHF
- Albuminuria, Proteinuria, Renal Failure

LVH = left ventricular hypertrophy
CHD = coronary heart disease
CHF = congestive heart failure


www.hypertensiononline.org
Target organs in children

1) Heart

2) Kidney

3) Small vessels

4) Large vessels

5) Central nervous system
Cardiac target organ damage

1) Heart: left ventricular hypertrophy (LVH) = the most prominent and the most thoroughly documented form of TOD caused by hypertension in children
Adults

(Schilacci, Verdecchia et al., Hypertension 2000)  (De Simone, Verdecchia et al., Hypertension 2002)
Children

No mortality studies.

Only association studies between BP and LV geometry
Relationship between BP and LVM

Children:

Belsha et al., Am J Hypert 1998:

- n=29 untreated adolescents with mild essential HT
- Clinic BP + ABPM + ECHO

LVH: 34% of children

Better correlation for Ambulatory SBP than for Clinic SBP (r=0.32)

The best correlation with LVMI for night-time SBP (r=0.41)

No correlation between LVMI and ambulatory DBP
Sorof et al., Hypertension 2002:

n=37 untreated children with essential HT
Clinic BP + ABPM + ECHO

LVH: 27 % of children
The best correlation for 24-hour SBP (r=0.43)
No correlation for Clinic SBP
No correlation between LVMI and ambulatory DBP
Left ventricular mass in children with various forms of hypertension

LVMI (g/m$^2.7$)

- Normotensives
- White-coat HT
- Masked HT
- Hypertensives

p<0.05

NS

(Stabouli et al., Pediatr Nephrol 2005, 20:1151-5)
Children

When to investigate a child for the presence of LVH?
TABLE 15. Laboratory investigation and imaging studies

**Laboratory tests**

- *Routine laboratory tests to be performed in all children with hypertension*
- Plasma creatinine, urea, electrolytes and uric acid
- Fasting plasma glucose
- Plasma cholesterol (total, HDL and LDL) and triglycerides
- Urinalysis and culture
- Quantification of albuminuria (albumin:creatinine ratio) proteinuria (protein:creatinine ratio)
- **Echocardiography**
- Renal ultrasonography
How to assess left ventricular mass and dg. LVH?

- **Echocardiography** far better and more sensitive than ECG by an experienced pediatric cardiologist!

**左心室质量 (LVM)**
- Interventricular septum (IVS)
- Left ventricular posterior wall (LVPW)
- Left ventricular end-diastolic diameter (LVED)

\[
LVM (g) = 0.8 \times [1.04 \times (IVS + LVPW + LVED)^3 - (LVED)^3] + 0.6
\]

**左心室质量指数 (LVMI)** (LVMI = LVM corrected for height):

\[
LVMI \left( \frac{g}{m^{2.7}} \right) = 0.8 \times [1.04 \times (IVS + LVPW + LVED)^3 - (LVED)^3] + 0.6 \times m^{2.7}
\]

= Devereux equation (Devereux et al., Am J Cardiol 1986, 57:450-8)
METHODS

- The ASE-recommended formula for estimation of LV mass from 2D linear LV measurements

\[
LV \text{ mass} = 0.8 \times \{1.04[(LVIDd + PWTd + SWTd)^3 - (LVIDd)^3])\} + 0.6 \text{ g}
\]

- LV mass was indexed to body surface area

\[
RWT = 2 \times \frac{PWTd}{LVIDd}
\]


LVH = LVMI $\geq 95^{th}$ percentile in children < 9 years

$> 40 \text{ g/m}^{2.7}$ in girls > 9 years

$> 45 \text{ g/m}^{2.7}$ in boys > 9 years

ECHOCARDIOGRAPHIC REFERENCE VALUES IN CHILDREN AND ADULTS

Age-Specific Reference Intervals for Indexed Left Ventricular Mass in Children
Philip R. Khoury, MS, Mark Mitsnefes, MD, MS, Stephen R. Daniels, MD, PhD, and Thomas R. Kimball, MD, FASE, Cincinnati, Ohio; and Denver, Colorado
Assessment of left ventricular mass geometry = assessment of the type of LVH

← 1) LVMI

← 2) relative wall thickness (RWT)

RWT = (IVS+LVPW) / LVED (normal: adults <0.45, children <0.42)

→ 4 patterns of LV geometry:

1) normal LV geometry
2) concentric remodelling
3) excentric LVH
4) concentric LVH
Assessment of left ventricular mass geometry = assessment of the type of LVH

1. Normal Pattern
   normal LV mass + RWT < 0.42

2. Concentric remodelling
   normal LV mass + RWT > 0.42

3. Concentric LVH
   Increased LV mass + RWT > 0.42

4. Eccentric LVH
   Increased LV mass + RWT < 0.42
Macroscopic preparation
The type of LVH influences CV prognosis of the patients.

(Ganau, Devereux et al., J Am Coll Cardiol 1992)
**Systolic function**

**Shortening fraction (SF)**

**Ejection fraction (EF)**

\[ SV = EDV - ESV \]

\[ EF = \frac{SV}{EDV} \times 100\% \]
Systolic function

Ejection fraction (EF):
- decreased = $< 53\%$ = systolic dysfunction

Shortening fraction (SF)
- decreased = $< 25\%$ = systolic dysfunction

Overbeek, Kapusta et al., Eur J Echocardiog 2006
Prevalence of LVH in children

14 - 42% of children with primary HT

33 - 75% of children with chronic kidney diseases, chronic renal failure and after renal transplantation

(Daniels et al., Circulation 1998
Matteucci et al., JASN 2006,
Litwin Ped Neph 2018)
Can cardiac TOD be reversed by antihypertensive therapy?
Regression of LVH with antihypertensive therapy

- Adults:
  - many studies proved regression of LVH with therapy
  - one of the main aims of the antihypertensive therapy

(ESH Guidelines 2013)
Children in severe CKD - predialysis:

n=49, before and after 30 months of antiHT therapy

(Borzych+Zurowska – Gdańsk, Wiad Lek 2005, in Polish)
• **Children with end-stage renal disease-hemodialysis:**

  n=17, at start and after 16 months of dialysis and antiHT ther.

Children with end-stage renal disease: hemodialysis:

Fig. 1  a Left ventricular mass in 17 hemodialyzed children at onset and at the end of hemodialysis. Paired data are given for each patient and mean± standard error. Patients were observed over a mean duration of 16.3±3.0 months. The level of the 99th percentile (dashed line) of LVMI, defined as severe LVH, is given. b–e Time course of left ventricular mass (b); systolic (sbp), diastolic (dbp) and mean (mbp) arterial blood pressure index (c); hemoglobin level (d); plasma total protein level at onset (ptp1) and end (ptp2) of hemodialysis session (e). Patients were observed within 1 month following the onset of hemodialysis (zero point; n=17), then after at 6.3±0.5 (n=16), 10.6±0.5 (n=12) and 17.3±0.7 (n=7) months. Duration and parameters are expressed as mean and standard error. *P<0.05, **P<0.01, ***P<0.001

• Children **without ESRD:**

n=21, renal and primary HT, before and 6 months after ther.

**Regression of Left-Ventricular Hypertrophy in Children and Adolescents With Hypertension During Ramipril Monotherapy**

Tomáš Seeman, Jiří Gilík, Karel Vondrák, Eva Šimková, Hana Flögelová,
Ramipril monotherapy-induced decrease of ambulatory BP

Daytime SBP

Daytime DBP

Nighttime SBP

Nighttime DBP

p<0.001

p<0.01

p<0.001

(Seeman et al., Amer J Hypert 2007, 20:990-6)
Regression of LVH during therapy in $\frac{3}{4}$ of patients

Regression of LVH with antihypertensive therapy is possible also in children without ESRF = one of the treatment targets

(Seeman et al., Amer J Hypert 2007, 20:990-6)
• Children **with primary HT**: n=86, before and 12 months after pharma.+non-pharma.therapy
Children with primary HT:

n=86, before and 12 months after pharmacol.+non-pharma. therapy

\[ p < 0.0001 \]
Children with primary HT:

n=86, before and 12 months after pharmacol.+non-pharma.therapy

The main predictor of decrease of LVMi was a decrease of waist-to-height ratio

Fig. 2 Linear correlation between decrease in waist-to-height ratio (\(WHtR\)) and decrease in left ventricular mass (\(LVMi\)): \(r=0.263, p=0.03\)
• Children with CKD stages 2-4:

n=84, ESCAPE-trial substudy, 2 years

Change in Cardiac Geometry and Function in CKD Children During Strict BP Control: A Randomized Study

Maria Chiara Matteucci,* Marcello Chinali,† Gabriele Rinelli,† Elke Wahl,‡ Aleksandra Zurowska,§ Marina Chaebit,‖ Giacomo Pongiglione,† Franz Schaefer,† and the ESCAPE Trial Group.

(% of children with LVH)

but decrease of LVH / LVMI was

- restricted only to patients with LVH at baseline!

- independent of BP reduction!?
Children with CKD stages 2-4: n=84, ESCAPE-trial substudy, 2 years

**but** the decrease of LVH / LVMI was restricted only to patients with LVH at baseline:

- independent of BP reduction

←hypothesis of a possible direct cardiac benefit of ACEI

**Figure 2.** Reduction in LVMI during follow-up. Gray bars refer to mean change in LVMI between baseline and 12-month follow-up, and black bars refer to mean change in LVMI between baseline and 24-month follow-up (no differences could be observed in LVMI change according to follow-up time; all P>0.05). Bars are grouped
• Children with CKD stages 2-4:
  n=84, ESCAPE-trial substudy, 2 years

<table>
<thead>
<tr>
<th>Change in Cardiac Geometry and Function in CKD Children During Strict BP Control: A Randomized Study</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maria Chiara Matteucci,1 Marcello Chinalli,1 Gabriele Rinelli,1 Elke Wahl,1 Aleksandra Zurowska,2 Marina Chabiki,3 Giacomo Pongiglione,1 Franz Schaeter,1 and the ESCAPE Trial Group</td>
</tr>
</tbody>
</table>

Table 3. Changes in LV geometric pattern at follow-up according to tradition

<table>
<thead>
<tr>
<th></th>
<th>Conventional Baseline</th>
<th>Intensified Baseline</th>
<th>Overall Baseline</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal LV geometry</td>
<td>62</td>
<td>64</td>
<td>63</td>
</tr>
<tr>
<td>Concentric remodeling</td>
<td>7</td>
<td>6</td>
<td>6</td>
</tr>
<tr>
<td>Eccentric LVH</td>
<td>21</td>
<td>22</td>
<td>22</td>
</tr>
<tr>
<td>Concentric LVH</td>
<td>10</td>
<td>8</td>
<td>9</td>
</tr>
</tbody>
</table>

Data are given in percentages. No differences were found between treatment; LVH, left ventricular hypertrophy.

*P<0.05 comparing baseline versus follow-up.
• Children with CKD stages 2-4:
  
n=84, ESCAPE-trial substudy, 2 years
  
**AntiHT therapy improves systolic function**

Increase of systolic function was - higher in INTENS. BP target group than in STANDARD BP target group.
Children with CKD stages 2-4: n=84, ESCAPE-trial substudy, 2 years

AntiHT therapy improves systolic function

(% of systolic dysfunction)

before therapy

after therapy

p<0.05
• Children with CKD stages 2-4:

n=435, CKiD Study, after 4 years of observ.(no interv.)

(%) of children with LVH

Predictors of LVMI:
- systolic BP
- anemia
- use of non-ACEI antiHT drugs
• Children with CKD stages 2-4:
  n=435, CKiD Study, after 4 years of observ. (no interv.)

Predictors of LVMI/LVH:
- systolic BP
- anemia
- female sex
- use of non-ACEI/ARB antiHT drugs
Systolic function in HT children

• Children with primary HT:

n=46, cross-sectional study, Agu, Portman et al. JASH 2014

<table>
<thead>
<tr>
<th>Variables</th>
<th>Normotensive</th>
<th>Hypertensive</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>n</td>
<td>34</td>
<td>46</td>
<td></td>
</tr>
<tr>
<td>Relative wall thickness</td>
<td>0.36 (0.07)</td>
<td>0.40 (0.07)</td>
<td>.012</td>
</tr>
<tr>
<td>LVMI, g/m²</td>
<td>37.7 (9.1)</td>
<td>42.62 (10.3)</td>
<td>.046</td>
</tr>
<tr>
<td>Fractional shortening, %</td>
<td>38.6 (6.5)</td>
<td>40.8 (5.6)</td>
<td>.06</td>
</tr>
<tr>
<td>Ejection fraction, %</td>
<td>68.4 (7.5)</td>
<td>71.2 (6.6)</td>
<td>.073</td>
</tr>
<tr>
<td>Mid wall shortening fraction, %</td>
<td>18.5 (3.3)</td>
<td>17.9 (2.5)</td>
<td>.302</td>
</tr>
<tr>
<td>Mitral valve E, cm/s</td>
<td>87.4 (15.9)</td>
<td>94.7 (21.6)</td>
<td>.072</td>
</tr>
<tr>
<td>Mitral valve A, cm/s</td>
<td>44.8 (8.7)</td>
<td>45.5 (14.6)</td>
<td>.938</td>
</tr>
<tr>
<td>Mitral valve E/A</td>
<td>2.0 (0.6)</td>
<td>2.2 (0.7)</td>
<td>.131</td>
</tr>
<tr>
<td>Mitral valve deceleration time, ms</td>
<td>167.1 (40)</td>
<td>168.2 (39)</td>
<td>.408</td>
</tr>
</tbody>
</table>
How often should echocardiography be repeated in hypertensive children?
How quick can regression of LVH be seen in hypertensive children?
How often should echocardiography be repeated in hypertensive children?

Every 6 – 12 months

Repeat echocardiography may be performed to monitor improvement or progression of target organ damage at 6- to 12-month intervals. Indications to repeat echocardiography include persistent HTN despite treatment, concentric LV hypertrophy, or reduced LV ejection fraction; and
Conclusions

Presence of cardiac target organ damage must be evaluated in all children with hypertension.

Echocardiography (assessment of the presence of LVH) should be performed instead of ECG in all hypertensive children regardless of age and severity of hypertension at the time of diagnosis and periodically during follow-up (every 6-12 months).

Treatment-induced regression of LVH is possible in children with ESRD as well as in children with CKD without ESRD and with primary hypertension.
Definition of LVH in children acc. USA Guidelines, Flynn et al. 2017

For this document, the following definitions for LV target organ injury have been chosen regarding hypertrophy, relative wall thickness, and ejection fraction. These definitions are based on published guidelines from the American Society of Echocardiography and associations of thresholds for indexed LV mass with adverse outcomes in adults:

- LVH is defined as LV mass $>51 \text{ g/m}^2.7$ or LV mass $>115 \text{ g per body surface area (BSA) for boys and LV mass} >95 \text{ g/BSA for girls. (Note that the values for LVH are well above the 95th percentile for distributions of LV mass in children and adolescents.}$

$369$ The clinical significance of values between the
Cumulative Incidence of Cardiovascular Events in Women and Men Without Hypertension, According to BP Category at Baseline

Meta Analysis: Lower Mean BP Results in Slower Rates of Decline in GFR in Diabetics and Non-Diabetics

MAP (mmHg)

GFR (mL/min/year)

Untreated HTN


Hypertension = risk factor for progression of chronic renal insufficiency

(Wingen et al., Lancet, 1997, 349: 1117-1123)
## Risk factors for overall mortality in end-stage renal disease (ESRD) children

<table>
<thead>
<tr>
<th>Risk Factor</th>
<th>RR</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Long lasting dialysis</td>
<td>7.2</td>
<td>4.4 - 11.8</td>
</tr>
<tr>
<td>2. Long lasting hypertension</td>
<td>3.1</td>
<td>2.1 - 4.6</td>
</tr>
</tbody>
</table>

Groothoff et al., Kidney Int 2002, 61:621-629
Hypertension in childhood increases 3-times the risk of cerebrovascular mortality in adulthood.


Fig. 2: Cumulative cerebrovascular disease (CVA) mortality among young normotensives (gray line) and young hypertensives (black line) followed up for 45,729,521 person-years and adjusted for year of birth, age at examination, sex, body mass index, height, country of origin, socio-economic status and education by Cox proportional hazards modeling (see also Table 3: Model 3).

$p < 0.001$

HR 3.12

95% CI 1.76-5.54
### ESH-ESC Guidelines: Factors Influencing Prognosis

<table>
<thead>
<tr>
<th>Risk factors for CV disease used for stratification</th>
<th>Target Organ Damage (TOD)</th>
<th>Diabetes Mellitus</th>
<th>Associated Clinical Conditions (ACC)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Levels of SBP and DBP</td>
<td>Left ventricular hypertrophy (electrocardiogram: Sokolow-Lyons &gt; 38 mm; Cornell &gt; 2440 mm*ms; echocardiogram: LVMI ≥ 125, W ≥ 110 g/m²^2)</td>
<td>Fasting plasma glucose ≥ 7.0 mmol/l (126 mg/dl)</td>
<td>Cerebrovascular disease: ischaemic stroke; cerebral haemorrhage; transient ischaemic attack</td>
</tr>
<tr>
<td>Men &gt; 55 years</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Women &gt; 65 years</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Smoking</td>
<td>Ultrasound evidence of arterial wall thickening (carotid IMT &gt; 0.9 mm) or atherosclerotic plaque</td>
<td>Postprandial plasma glucose &gt; 11 mmol/l (198 mg/dl)</td>
<td>Heart disease: myocardial infarction; angina; coronary revascularization; congestive heart failure</td>
</tr>
<tr>
<td>Dyslipidaemia (total chol. &gt; 250 mg/dl, or LDL-chol. &gt; 155 mg/dl, or HDL-chol. M &lt; 40, W &lt; 48 mg/dl)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Family history of premature CV disease (at age &lt; 55 years M, &lt; 65 years W)</td>
<td>Slight increase in serum creatinine (M 115-133, W 107-124 μmol/l; M 1.3-1.5, W 1.2-1.4 mg/dl)</td>
<td>Microalbuminuria (30-300 mg/24h; albumin-creatinine ratio M ≥ 22, W ≥ 31 mg/g; M ≥ 2.5, W ≥ 3.5 mg/mmol)</td>
<td>Renal disease: diabetic nephropathy; renal impairment (serum creatinine M &gt; 1.5, W &gt; 1.4 mg/dl) proteinuria (&gt; 300 mg/24h)</td>
</tr>
<tr>
<td>Abdominal obesity (abdominal circumference &gt; 102 cm, W &gt; 88 cm)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>C-reactive protein ≥ 1 mg/dl</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Adults:
Adults:

Children:
Regeneration of LVH during antihypertensive therapy is possible also in children = one of the treatment targets.

(Seeman et al., Am J Hypert 2007, 20:990-6)
Children:

Litwin et al., Pediatr Nephrol 2004, 19:767-774

n=49 hypertensive children vs. controls
carotid IMT (cIMT), femoral IMT + ABPM

**higher cIMT in hypertensive children**

no correlation with ABPM results!
correlation with **office SBP** and pulse pressure !!
strongest correlation with BMI (HT children heavier!)

**BMI (obesity) predicts cIMT more than BP (hypertension)**
Diastolic function:

**E/A ratio** = the ratio of peak velocity blood flow in *Early* diastole (the E wave) to peak velocity flow in *late* diastole caused by *Atrial* contraction (the A wave)

- decreased: **<1.0 = diastolic dysfunction**
Diastolic dysfunction:

- E/A ratio = the ratio of peak velocity blood flow in early diastole (the E wave) to peak velocity flow in late diastole caused by atrial contraction (the A wave)
  - normal: ...

Systolic vs. Diastolic

- Diastolic dysfunction
  - EF normal or increased
  - Hypertension
  - Due to LVH and chronic replacement by fibrous tissue - decrease in distensibility
- Systolic dysfunction
  - EF < 40%
  - Usually from coronary disease
  - Due to ischemia-induced decrease in contractility
- Most common is a combination of both
Diastolic function in HT children

- Children with primary HT:
  
n=46, cross-sectional study, Agu, Portman et al. JASH 2014

<table>
<thead>
<tr>
<th>Variables</th>
<th>Normotensive</th>
<th>Hypertensive</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>n</td>
<td>34</td>
<td>46</td>
<td></td>
</tr>
<tr>
<td>Relative wall thickness</td>
<td>0.36 (0.07)</td>
<td>0.40 (0.07)</td>
<td>.012</td>
</tr>
<tr>
<td>LVMI, g/m²</td>
<td>37.7 (9.1)</td>
<td>42.62 (10.3)</td>
<td>.046</td>
</tr>
<tr>
<td>Fractional shortening, %</td>
<td>38.6 (6.5)</td>
<td>40.8 (5.6)</td>
<td>.06</td>
</tr>
<tr>
<td>Ejection fraction, %</td>
<td>68.4 (7.5)</td>
<td>71.2 (6.6)</td>
<td>.073</td>
</tr>
<tr>
<td>Mid wall shortening fraction, %</td>
<td>18.5 (3.3)</td>
<td>17.9 (2.5)</td>
<td>.302</td>
</tr>
<tr>
<td>Mitral valve E, cm/s</td>
<td>87.4 (15.9)</td>
<td>94.7 (21.6)</td>
<td>.072</td>
</tr>
<tr>
<td>Mitral valve A, cm/s</td>
<td>44.8 (8.7)</td>
<td>45.5 (14.6)</td>
<td>.938</td>
</tr>
<tr>
<td>Mitral valve E/A</td>
<td>2.0 (0.6)</td>
<td>2.2 (0.7)</td>
<td>.131</td>
</tr>
<tr>
<td>Mitral valve deceleration time, ms</td>
<td>167.1 (40)</td>
<td>168.2 (39)</td>
<td>.408</td>
</tr>
</tbody>
</table>
Diastolic function in HT children

- Children with primary HT: n=29, cross-sectional study, Lee et al. Clinical Hypert 2015

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Normal BP index (BP index &lt;1.0) vs high BP index (BP index ≥1.0)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>BP index &lt;1.0</td>
</tr>
<tr>
<td>N</td>
<td>9</td>
</tr>
<tr>
<td>Age (year)</td>
<td>15.0 ± 2.6</td>
</tr>
<tr>
<td>BMI</td>
<td>20.2 ± 2.7</td>
</tr>
<tr>
<td>BP index</td>
<td>0.94 ± 0.06</td>
</tr>
<tr>
<td>BP load (day) (%)</td>
<td>19.6 ± 17.9</td>
</tr>
<tr>
<td>Night dip (%)</td>
<td>7.0 ± 5.4</td>
</tr>
<tr>
<td>Daytime average SBP</td>
<td>125.9 ± 7.4</td>
</tr>
<tr>
<td>Night time average SBP</td>
<td>117.0 ± 7.7</td>
</tr>
<tr>
<td>LVMI (g/m²)</td>
<td>32.2 ± 8.1</td>
</tr>
<tr>
<td>LVMI (g/m²)</td>
<td>73.2 ± 17.6</td>
</tr>
<tr>
<td>LVH (%)</td>
<td>0 (0 %)</td>
</tr>
<tr>
<td>E/E'</td>
<td>7.6 ± 2.5</td>
</tr>
<tr>
<td>E/A</td>
<td>1.9 ± 0.5</td>
</tr>
<tr>
<td>Tei index</td>
<td>0.3 ± 0.1</td>
</tr>
</tbody>
</table>

Test method = Mann-Whitney U test
N sampler number, BMI body mass index, BP index blood pressure index, BP load (day) blood pressure load during daytime, LVMI left ventricular mass index, LVH left ventricular hypertrophy, E/E' early transmial filling velocity/ early diastolic tissue velocity at septal mitral annulus, E/A early transmial filling velocity/late transmilar filling velocity
Target organ damage in children

1) Heart

2) **Kidney** = microalbuminuria

3) Small vessels

4) Large vessels

5) Central nervous system
Relationship between
renal TOD = microalbuminuria
and
cardiac TOD = LVH

[Assadi et al., Ped Cardiol 2008]
LVH influence CV prognosis of the patients

BACKGROUND

- Left ventricular hypertrophy (LVH) and alterations in LV geometry have been associated with increased mortality and other cardiovascular (CV) events.

- Patients with concentric LVH (increased relative wall thickness [RWT] and LV mass index [LVMi]), have been shown to have the highest incidence of adverse CV events, including death.
Classic pathway in the progression from Hypertension to Heart Tension

Hypertension

Eccentric LVH

Heart Failure

Pressure overload

Concentric LVH

HF with Preserved LVEF

Increase in LV Mass

LVH

HF with reduced LVEF

Adapted from Kamran Diaz, Yasmin Subhi, Hypertensive Heart Disease. Medscape Reference, Jan 2012