The 2016 CHEP Guidelines: Evidence driven recommendations for hypertension

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  • Consulting Fees: None
  • Other:
2016 CHEP Guidelines Task Force
Evidence-based Annual Guidelines

• Canada has the world’s highest reported national blood pressure control rates

• CHEP is known as the most credible source for evidence-based hypertension guidelines, with annual updates, a well-validated review process and effective dissemination techniques across Canada
Guidelines Task Force

Topic Sub-Group

Central Review Committee

Topic Sub-Group

Topic Sub-Group

Dissemination and Implementation Committee

Outcomes Research Task Force
New Recommendations for 2016
What’s new?

- New thresholds and targets for high risk patients (SPRINT)
- **Assessing** clinic blood pressures using **automatic electronic** (oscillometric) monitors
- **Adopting** healthy behaviours is integral to the management of hypertension (focus on potassium supplementation)
- **Updating** the recommendation for lipid screening in patients with hypertension (now can be completed non-fasting)
- **Updating** the treatment of patients with hypertension with concurrent coronary artery disease
- **New** recommendations on the diagnosis and management of hypertension in pediatric patients (*NOT the focus of this presentation*)
Recommended Treatment Targets

Treatment consists of health behaviour ± pharmacological management

<table>
<thead>
<tr>
<th>Population</th>
<th>SBP</th>
<th>DBP</th>
</tr>
</thead>
<tbody>
<tr>
<td>High Risk</td>
<td>&lt;120</td>
<td>NA</td>
</tr>
<tr>
<td>Diabetes</td>
<td>&lt;130</td>
<td>&lt;80</td>
</tr>
<tr>
<td>All others (including CKD)*</td>
<td>&lt;140</td>
<td>&lt;90</td>
</tr>
</tbody>
</table>
New thresholds/targets for the high risk patient post-SPRINT: who does this apply to??

- Clinical or sub-clinical cardiovascular disease
  OR
- Chronic kidney disease (non-diabetic nephropathy, proteinuria <1 g/d, *estimated glomerular filtration rate 20-59 mL/min/1.73m^2)
  OR
- †Estimated 10-year global cardiovascular risk >15%
  OR
- Age ≥ 75 years

Patients with one or more clinical indications should consent to intensive management.

* Four variable MDRD equation
† Framingham Risk Score, D'Agastino, Circulation 2008
New thresholds/targets for the high risk patient post-SPRINT: who does this NOT apply to??

**Limited or No Evidence:**
- Heart failure (EF <35%) or recent MI (within last 3 months)
- Indication for, but not currently receiving a beta-blocker
- Frail or institutionalized elderly

**Inconclusive Evidence:**
- Diabetes mellitus
- Prior stroke
- eGFR < 20 ml/min/1.73m2

**Contraindications:**
- Patient unwilling or unable to adhere to multiple medications
- Standing SBP <110 mmHg
- Inability to measure SBP accurately
- Known secondary cause(s) of hypertension
SPRINT: SBPs achieved

Average no. of medications
Intensive care: 3
Standard care: 1.8
Primary Outcome

NNT=61

Hazard ratio with intensive treatment, 0.75 (95% CI, 0.64–0.89)

No. at Risk
Standard treatment 4683 4437 4228 2829 721
Intensive treatment 4678 4436 4256 2900 779

The SPRINT Research Group, NEJM, Nov 9th, 2015
Chep 2016 guidelines

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Office BP Measurement Methods

Office attended: OBPM

- Auscultatory (mercury, aneroid)
- Oscillometric (electronic)

Automated office (unattended): AOBP

- Oscillometric (electronic)
Automated office blood pressure measurement (AOBP) is the preferred method of performing in-office BP measurement (Grade D). When using AOBP, a displayed mean SBP ≥135 mmHg or DBP ≥85 mmHg is high (Grade D).
AOBP is Not Affected by the Setting in Which BP is Recorded

- Readings recorded in an ABPM unit or in an office waiting room are similar to AOBP recorded in a physician’s examination room


- AOBP results obtained in the pharmacy were comparable with AOBP results from the physician’s office

Comparisons of blood pressure readings obtained in clinical settings using different methods of blood pressure measurement

<table>
<thead>
<tr>
<th></th>
<th>Centre for Studies in Primary Care&lt;sub&gt;1&lt;/sub&gt;</th>
<th>ABPM referral unit&lt;sub&gt;2&lt;/sub&gt;</th>
<th>CAMBO trial&lt;sub&gt;3&lt;/sub&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td>Routine manual office BP</td>
<td>151/83</td>
<td>152/87</td>
<td>150/81</td>
</tr>
<tr>
<td>Automated office BP</td>
<td>140/80</td>
<td>132/75</td>
<td>135/77</td>
</tr>
<tr>
<td>Awake ambulatory BP</td>
<td>142/80</td>
<td>134/77</td>
<td>133/74</td>
</tr>
</tbody>
</table>

*The automated office blood pressure (BP) and awake ambulatory BP were similar, and both were lower than the routine manual BP obtained in community practice.*

Predictive value of AOBP

AOBP predicts end-organ damage

• Systolic AOBP correlates with LVMI similarly to awake ABPM
• AOBP and 24-h ABPM have similar predictive ability for microalbuminuria
• AOBP is more strongly associated with cIMT (compared to OBPM)

**cIMT:** Carotid Intima Media Thickness
**LVMI:** Left Ventricular Mass Index

What’s new?

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Potassium intake:

• In patients *not* at risk of hyperkalemia, increase dietary potassium intake to reduce blood pressure.
## Systematic Reviews showing a Significant Effect of Potassium on BP

<table>
<thead>
<tr>
<th>Author</th>
<th>Year</th>
<th>RCTs</th>
<th>Total N</th>
<th>Pooled effect SBP</th>
<th>Pooled effect DBP</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cappuccio</td>
<td>1991</td>
<td>19</td>
<td>586</td>
<td>-5.9 (-6.6 to -5.2)</td>
<td>-3.4 (-4.0 to -2.8)</td>
<td>Mixed status, 5-112 days, 10-150 participants; ?all RCTs</td>
</tr>
<tr>
<td>Whelton</td>
<td>1997</td>
<td>33</td>
<td>2609</td>
<td>-3.11 (-4.3 to -1.9)</td>
<td>-1.97 (-3.4 to -0.5)</td>
<td>Mixed status; 4d-3yrs; 10-484 N</td>
</tr>
<tr>
<td>Geleijnse</td>
<td>2003</td>
<td>27</td>
<td>NR</td>
<td>-2.4 (-3.8 to -1.1)</td>
<td>-1.57 (-2.6 to -0.5)</td>
<td>Mixed status; &gt;2 wks duration</td>
</tr>
<tr>
<td>Dickinson</td>
<td>2006</td>
<td>5</td>
<td>425</td>
<td>-3.9 (-8.6 to 0.8)</td>
<td>-1.5 (-6.2 to 3.1)</td>
<td>Cochrane; hypertensive only; &gt;8wks; 12-212 N; still significant heterogeneity; one trial not pooled – no ss dec in BP</td>
</tr>
<tr>
<td>van Bommel</td>
<td>2012</td>
<td>10</td>
<td>563</td>
<td>-7.12 (-8.5 to -5.7)</td>
<td>-4.9 (-5.8 to -4.0)</td>
<td>Hypertensive pts with high Na diet; heterogeneity dec. after exc. of outlier</td>
</tr>
<tr>
<td><strong>Aburto</strong></td>
<td>2013</td>
<td>22</td>
<td>1606</td>
<td>-3.49 (-5.2 to -1.8)</td>
<td>-1.96 (-3.1 to -0.9)</td>
<td>Mixed status; &gt;4 wks; measured urinary K</td>
</tr>
<tr>
<td>Binia</td>
<td>2015</td>
<td>15</td>
<td>917</td>
<td>-4.7 (2.4 to -7)</td>
<td>-3.5 (1.3 to 5.7)</td>
<td>Pts not on anti-htn Rx; mixed status; &gt;=4wks;</td>
</tr>
</tbody>
</table>
Increased Potassium intake decreases BP:

A K rich diet has additive effects to Na restriction

Systolic Blood Pressure (mm Hg)

- Control diet
  - High: -5.9 (-8.0 to -3.7)†
  - Intermediate: -2.1 (-3.4 to -0.8)‡
  - Low: -4.6 (-5.9 to -3.2)‡

- DASH diet
  - High: -1.3 (-2.6 to 0.0)*
  - Intermediate: -2.2 (-4.4 to -0.1)*
  - Low: -1.7 (-3.0 to -0.4)†

Potassium supplementation leads to a decrease in BP

Effect most consistently seen in patients with hypertension

Effect of K is modified by Na intake, with greater effect at higher baseline Na
Identify those at Risk of Hyperkalemia with Potassium supplementation

Prior to advising increase in potassium intake, the following kinds of patients – who are at high risk of hyperkalemia, should be assessed for suitability, and monitored closely:

- Patients taking renin-angiotensin-aldosterone inhibitors
- Patients on other drugs that can cause hyperkalemia (trimethoprim and sulfamethoxazole, amiloride, triamterene)
- Patients with CKD (GFR < 45mL/min)
- Patients with baseline serum potassium > 4.5 mmol/L
What’s new?

• New thresholds and targets for high risk patients (SPRINT)
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Fasting Lipids & All-Cause Mortality


N=4299; NHANES
Figure 2. Association of Triglyceride Levels With Individual Cardiovascular End Points, According to Fasting Status

Women’s Health Study: N=26,509
Fasting vs. Non-fasting Cholesterol: (Cross-sectional, community-based study Calgary)

Table 1. Cholesterol Levels in Males by Fasting Time After Adjustment for the Effect of Age

<table>
<thead>
<tr>
<th>Fasting Time, h</th>
<th>Sample Size</th>
<th>Total Cholesterol</th>
<th>HDL Cholesterol</th>
<th>Calculated LDL Cholesterol</th>
<th>Triglycerides</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>724</td>
<td>166.9 (162.6-171.1)</td>
<td>49.0 (47.6-50.4)</td>
<td>88.9 (85.2-92.5)</td>
<td>150.6 (138.3-162.8)</td>
</tr>
<tr>
<td>2</td>
<td>370</td>
<td>171.6 (165.8-177.5)</td>
<td>47.9 (45.9-49.8)</td>
<td>93.5 (88.4-98.6)</td>
<td>160.5 (143.7-177.3)</td>
</tr>
<tr>
<td>3</td>
<td>313</td>
<td>172.4 (164.4-180.3)</td>
<td>48.8 (46.1-51.4)</td>
<td>92.1 (85.3-98.9)</td>
<td>159.0 (136.1-181.9)</td>
</tr>
<tr>
<td>4</td>
<td>307</td>
<td>172.9 (165.6-180.2)</td>
<td>47.9 (45.4-50.3)</td>
<td>95.2 (89.0-101.4)</td>
<td>153.4 (132.3-174.5)</td>
</tr>
<tr>
<td>5</td>
<td>210</td>
<td>173.1 (163.4-182.8)</td>
<td>50.5 (47.3-53.7)</td>
<td>94.2 (85.9-102.5)</td>
<td>153.6 (125.6-181.6)</td>
</tr>
<tr>
<td>6</td>
<td>160</td>
<td>173.0 (162.2-181.8)</td>
<td>48.6 (45.6-51.5)</td>
<td>93.4 (85.9-100.9)</td>
<td>165.6 (140.3-190.8)</td>
</tr>
<tr>
<td>7</td>
<td>141</td>
<td>173.0 (162.7-183.4)</td>
<td>44.3 (40.8-47.7)</td>
<td>96.8 (88.0-105.6)</td>
<td>161.7 (131.9-191.5)</td>
</tr>
<tr>
<td>8</td>
<td>305</td>
<td>169.5 (162.0-177.0)</td>
<td>50.6 (48.1-53.1)</td>
<td>94.6 (88.2-101.0)</td>
<td>127.0 (105.3-148.7)</td>
</tr>
<tr>
<td>9</td>
<td>846</td>
<td>167.0 (160.0-173.7)</td>
<td>48.3 (46.0-50.5)</td>
<td>92.7 (87.0-98.4)</td>
<td>134.9 (115.5-154.4)</td>
</tr>
<tr>
<td>10</td>
<td>10 050</td>
<td>170.4 (167.4-173.4)</td>
<td>50.3 (49.3-51.3)</td>
<td>96.1 (93.5-98.7)</td>
<td>122.9 (114.2-131.7)</td>
</tr>
<tr>
<td>11</td>
<td>13 668</td>
<td>166.6 (161.6-171.5)</td>
<td>51.2 (49.5-52.8)</td>
<td>92.6 (88.4-96.8)</td>
<td>117.7 (103.4-132.0)</td>
</tr>
<tr>
<td>12</td>
<td>32 199</td>
<td>171.0 (169.5-172.5)</td>
<td>49.6 (49.1-50.0)</td>
<td>97.3 (96.1-98.6)</td>
<td>124.3 (120.0-128.8)</td>
</tr>
<tr>
<td>13</td>
<td>16 842</td>
<td>171.0 (169.0-173.1)</td>
<td>50.0 (49.3-50.6)</td>
<td>97.2 (95.5-99.0)</td>
<td>122.9 (117.0-128.4)</td>
</tr>
<tr>
<td>14</td>
<td>16 622</td>
<td>175.6 (172.0-179.1)</td>
<td>50.0 (48.8-51.2)</td>
<td>101.7 (98.8-104.7)</td>
<td>123.0 (112.8-133.1)</td>
</tr>
<tr>
<td>15</td>
<td>5696</td>
<td>175.5 (172.9-178.1)</td>
<td>50.1 (49.2-50.9)</td>
<td>100.9 (98.7-103.2)</td>
<td>126.5 (119.0-134.1)</td>
</tr>
<tr>
<td>16</td>
<td>4677</td>
<td>175.1 (169.6-180.6)</td>
<td>51.4 (50.0-53.2)</td>
<td>100.2 (95.5-104.8)</td>
<td>120.4 (104.5-136.2)</td>
</tr>
</tbody>
</table>

Summary of Evidence: Non-fasting Lipids

1. *Fasting has little effect* on overall lipid profile.
   - TG vary up to 20-30%; TC, HDL, LDL vary up to 10%.

2. *Prognostic value* of non-fasting lipids similar to fasting in predicting: all-cause mortality, CVD mortality, and vascular events (CHD & stroke) across multiple populations and subgroups.
   - In largest combined analysis, *Fasting TG not predictive of CVD outcomes* whereas non-fasting TG ↑ predictive CVD.

2015 Proposed Recommendation:

Lipids may be evaluated fasting or non-fasting (Grade C).
CHEP 2016 Guidelines

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Prior recommendation:
For patients with stable angina, β-blockers are preferred as initial therapy. *CCBs may also be used.*

New recommendation:
For patients with hypertension and stable angina pectoris but without prior HF, MI or coronary artery bypass surgery, *either a beta blocker or a calcium channel blocker can be used as initial therapy.*
Beta blocker vs CCB in Treatment of CAD

The TIBET Trial

Events =MI, CV death, HF, ACS
Hard events + ischemic ST changes on 24 h ECG

Total Ischaemic Burden European Trial (TIBET): Effects of atenolol (N=226), nifedipine SR (N=232) or combination (N=224) on outcome in chronic stable angina. Dargie et al. EHJ 1996;17:104-112
The curves were not extended beyond 5 years as few patients were followed thereafter.

APSIS: metoprolol vs verapamil in stable angina pectoris
No difference: CV events (30.8% v 29.3%) CV mortality 4.7% vs 4.7%), Non-fatal CV events 26.1 v 24.3%

Hjemdahl P. et al. Favourable long term prognosis in stable angina pectoris: an extended follow up of the angina prognosis study in Stockholm (APSIS); Heart 2006;92:177-182
CCB vs. Non-CCB in treatment of CAD

The INVEST trial

As required to achieve blood pressure control:

**CCB strategy:**
Verapamil sustained release + Trandolapril + HCTZ

**Non-CCB strategy:**
Atenolol + HCTZ, + Trandolapril

- 22,000 HT patients with CAD
- Primary Outcome:
  - Alive, Free of MI or Stroke
- Total FU: 61,807 pt-y, mean FU 2.7y,
- Annual event rate = 3.6%

Pepine JC et al. JAMA 2003 290(21):2805-16
hypertension.ca

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