HYPERTENSIVE EMERGENCIES

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Hypertension (HT) is the most common primary diagnosis in America.

Globally, the overall prevalence of HT in adults >25 yrs is ~40% in 2008.

600 million in 1980 to ~1 billion in 2008.

Causes 7.5 million deaths (12.8% of total deaths) annually.
The New HT (JNC 8) Guidelines

• The last version (JNC 7) was published back in 2003

• The new (JNC 8) guidelines were published online Dec 18, 2013. Called “2014 guidelines”
Special Communication

2014 Evidence-Based Guideline for the Management of High Blood Pressure in Adults
Report From the Panel Members Appointed to the Eighth Joint National Committee (JNC 8)

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Definition

- A systolic blood pressure (SBP) >139 mmHg and/or
- A diastolic (DBP) >89 mmHg
- Based on the average of two or more properly measured, seated BP readings on each of two or more office visits
# Classification

## Table 1. Classification and management of blood pressure for adults*

<table>
<thead>
<tr>
<th>BP Classification</th>
<th>SBP* (mmHg)</th>
<th>DBP* (mmHg)</th>
<th>LIFESTYLE MODIFICATION</th>
<th>INITIAL DRUG THERAPY</th>
<th>WITH COMPPELLING INDICATIONS (SEE TABLE 8)</th>
</tr>
</thead>
<tbody>
<tr>
<td>NORMAL</td>
<td>&lt;120</td>
<td>and &lt;80</td>
<td>Encourage</td>
<td>No antihypertensive drug indicated.</td>
<td>Drug(s) for compelling indications.‡</td>
</tr>
<tr>
<td>PREHYPERTENSION</td>
<td>120–139</td>
<td>or 80–89</td>
<td>Yes</td>
<td>Thiazide-type diuretics for most. May consider ACEI, ARB, BB, CCB, or combination.</td>
<td>Drug(s) for the compelling indications.‡ Other antihypertensive drugs (diuretics, ACEI, ARB, BB, CCB) as needed.</td>
</tr>
<tr>
<td>STAGE 1 HYPERTENSION</td>
<td>140–159</td>
<td>or 90–99</td>
<td>Yes</td>
<td>Two-drug combination for most‡ (usually thiazide-type diuretic and ACEI or ARB or BB or CCB).</td>
<td></td>
</tr>
<tr>
<td>STAGE 2 HYPERTENSION</td>
<td>≥160</td>
<td>or ≥100</td>
<td>Yes</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*The initial drug therapy and management are indications for treatment and prevention based on existing clinical guidelines. The aim is to improve cardiovascular health and prevent complications. Additional measures include lifestyle modifications, medication adjustments, and periodic medical evaluations.

†In some cases, treatment may be adjusted or intensified depending on the individual’s response and health status.

‡Compelling indications may require more aggressive management or additional interventions based on the individual’s specific health profile and comorbidities.
# Hypertensive Crises

<table>
<thead>
<tr>
<th>Type</th>
<th>Urgencies:</th>
<th>Emergencies:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypertensive Urgencies</td>
<td>No progressive dysfunction</td>
<td>Progressive dysfunction</td>
</tr>
<tr>
<td>Hypertensive Emergencies</td>
<td>(Accelerated Hypertension)</td>
<td>(Malignant Hypertension)</td>
</tr>
</tbody>
</table>

**Hypertensive Crises**

- **Hypertensive Urgencies**: No progressive dysfunction (Accelerated Hypertension)
- **Hypertensive Emergencies**: Progressive dysfunction (Malignant Hypertension)
Hypertensive Emergencies

- Severely elevated BP (>180/120mmHg)
- With progressive target organ dysfunction
- Require emergent lowering of BP

- **Examples:** Severely elevated BP with
  - Hypertensive encephalopathy
  - Acute left ventricular failure with pulmonary edema
  - Acute MI or unstable angina pectoris
  - Dissecting aortic aneurysm
Pathophysiology of a Hypertensive Emergency

Pathophysiology of Hypertensive Emergency

- Not well understood
- Failure of normal autoregulation + abrupt rise in SVR
- Increase in SVR due to release of humoral vasoconstrictors from the stressed vessel wall
- Endothelium plays a central role in BP homeostasis via substances as Nitric Oxide and prostacyclin
- Increased pressure starts a cycle of
  - endothelial damage
  - local activation of clotting cascade
  - fibrinoid necrosis of small vessels
  - release of more vasoconstrictors
- Process leads to progressive increase in resistance and further endothelial dysfunction
Target Organs

- Nervous system
- CVS (Heart and Blood Vessels)
- The Eyes (Retinopathy)
- The kidneys

- Single organ involvement in approximately 83%
- Two organ involvement found in 14%
- Multiorgan involvement found in 3%
What Constitutes a Hypertensive Emergency?

- Brain
  - Hypertensive Encephalopathy
  - Ischemic Stroke
  - Hemorrhagic Stroke
  - Subarachnoid Hemorrhage
- Retina
  - Hemorrhages
  - Exudates
  - Papilledema
- Cardiovascular System
  - Unstable Angina
  - Acute Heart Failure
  - Acute Myocardial Infarction
  - Aortic Dissection
- Kidney
  - Hematuria
  - Proteinuria
  - Decreasing Renal Function

Rynn et al. J Pharm Prac 2005;18:363-76
What End-Organs Are Typically Involved?

<table>
<thead>
<tr>
<th>End-organ damage type</th>
<th>Cases (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cerebral infarction</td>
<td>24.5</td>
</tr>
<tr>
<td>Intracerebral or subarachnoid bleed</td>
<td>4.5</td>
</tr>
<tr>
<td>Hypertensive encephalopathy</td>
<td>16.3</td>
</tr>
<tr>
<td>Acute pulmonary edema</td>
<td>22.5</td>
</tr>
<tr>
<td>Acute congestive heart failure</td>
<td>14.3</td>
</tr>
<tr>
<td>Acute myocardial infarction or unstable angina</td>
<td>12.0</td>
</tr>
<tr>
<td>Aortic dissection</td>
<td>2.0</td>
</tr>
<tr>
<td>Eclampsia</td>
<td>2.0</td>
</tr>
</tbody>
</table>
Effects on CVS

- Ventricular hypertrophy, dysfunction and failure, pulmonary edema
- Arrhythmias
- Myocardial ischemia, Acute MI
- Arterial aneurysm, dissection, and rupture
Effects on the Kidneys

- The renal system is impaired when high BP leads to arteriosclerosis, fibrinoid necrosis, and an overall impairment of renal protective autoregulation mechanisms.
- This may manifest as worsening renal function, hematuria, RBC cast formation, and/or proteinuria.
Nervous System

- Elevated BP overpowers the normal cerebral autoregulation. This results in transudate leak across capillaries and continued arteriolar damage. The end result of loss of autoregulation is hypertensive encephalopathy
- Hypertensive encephalopathy:
  - Clinical manifestation of cerebral edema and microhemorrhages, altered mental status, headache, vomiting and seizures with dysfunction of cerebral autoregulation
  - Defined as an acute organic brain syndrome or delirium in the setting of severe hypertension
- Stroke, intracerebral and subarachnoid hemorrhage
- Cerebral atrophy and dementia
HT Encephalopathy

• **Symptoms**
  - Severe headache
  - Nausea and vomiting
  - Visual disturbances
  - Confusion
  - Focal or generalized weakness

• **Signs**
  - Disorientation
  - Focal neurologic defects
  - Focal or generalized seizures
  - Nystagmus

Not adequately treated - cerebral haemorrhage, coma and death

BUT with proper treatment - completely reversible
The Eyes

- Retinopathy, retinal hemorrhages and impaired vision
- Vitreous hemorrhage, retinal detachment
- Neuropathy of the nerves leading to extraocular muscle paralysis and dysfunction
HT Retinopathy - Fundoscopy

- Keith-Wagener classification
  - Stage I- arteriolar sclerosis with thickening, irregularity and tortuosity
  - Stage II- AV dipping or compression
  - Stage III- Flame shaped haemorrhages and cotton wool spots
  - Stage IV- Papilledema

- “Stage III and IV lesions - imply failure of the CNS vascular autoregulation and makes the Dx of Malignant HT definitive”
Retina Normal and Hypertensive Retinopathy

A: Hemorrhages
B: Exudates (Fatty Deposits)
C: Cotton Wool Spots (Micro Strokes)
Stage-I Arteriolar Narrowing
Stage-II AV Nicking
Stage-III Hemorrhages (H), Cotton Wool Spots and Exudats (E)
Stage-IV Stage III+Papilledema
Diagnosis

- **History**
  1) Focus on presence of symptoms of end-organ dysfunction
  2) Any identifiable etiology
- **Hypertension Hx**
  - Last known normal BP
  - Prior diagnoses
  - Dietary and social factors
- **Medication**
  - Steroid use
  - Estrogens
  - Sympathomimetics
  - MAO inhibitors
- **Social history**
  - Smoking, alcohol
  - Illicit drugs (cocaine, stimulants)
- **Family history**
  - Early HT in family members
  - Cardiovascular and cerebrovascular disease
  - Diabetes
  - Pheochromocytoma
- **Pregnant?**
Diagnosis

• **History (cont)**
  • Symptom specific Hx - suggesting EOD
  • **CVS Hx**
    - Previous MI/angina/arrhythmias
    - Chest pain/claudication/flank or back pain
  • **Neurologic Hx**
    - Prior strokes, neuro dysfunction
    - Visual changes, blurriness, loss of visual fields, severe headaches, nausea and vomiting, change in mental status
  • **Renal Hx**
    - Underlying renal disease (RF)
    - Acute onset changes in renal frequency (anuria/oliguria)
  • **Endocrine Hx**
    - Diabetes, thyroid dysfunction, Cushing’s syndrome
Diagnosis

Examination
1) Confirm elevated BP
   » Proper position, appropriate cuff size
   » Supine and standing and both arms

2) Asses – EOD present
   • Fundoscopy
     • Chronic HT will have findings
     • Acute changes
       New retinal bleeds
       Superficial/flame shaped
       Deep/punctuate
     Exudates
     hard/cotton wool spots
     Papilledema
   • Neck
     Enlarged thyroid, carotid bruit, jugular venous distention
   • CVS
     Enlarged heart, S3, asymmetric pulses, arrhythmias
   • Pulmonary
     Signs of LV dysfunction (crackles, rhonchi)
   • Renal
     Renal bruit, abdominal masses
   • Neurologic
     Level of consciousness, evidence of stroke, any focal signs
Workup

**Lab studies**
- Electrolytes, urea and creatinine
- Urinalysis - Dipstix + microscopy
- Optional - Tox screen
  - B-HCG
  - Endocrine testing

**Imaging studies**
- CXR (pulmonary edema, aortic arch, cardiac enlargement)
- Head CT/MRI brain (abn neurology)
- Chest CT/TEE/Aortic angio (Aortic dissection)

**Other Tests**
- ECG (LVH, signs of ischemia, injury, infarct)
Aortic Dissection?
- Suspect with severe tearing chest pain, unequal pulses, widened mediastinum
- Contrast Chest CT Scan or MRI

Pulmonary Edema/CHF
- Transthoracic Echocardiogram
- Differentiate between systolic dysfunction, diastolic dysfunction, mitral regurgitation
How Quickly?

- **Cerebral Blood Flow Autoregulation**
  
  - For constant Cerebral Blood flow in normotensive individuals MAPs of 60 -120 mmHg
  
  - In chronically hypertensive patients autoregulatory range is higher: MAP Range 120-160 mmHg

- Autoregulation also impaired in the elderly and those with cerebrovascular disease
Management

• Where?
  - ICU with close monitoring
  - Severe cases require intra-arterial BP monitoring

• Which Parenteral meds?

• Depends on the situation

• In 2008: According to a Cochrane systematic review—there is no evidence that antihypertensive drugs reduced mortality or morbidity in hypertensive emergencies

• Therefore; treatment recommendations are consensus based
Treatment Typically Parenteral

- Adrenergic receptor blockers
  - Esmolol ($\beta_1$)
  - Labetalol ($\alpha_1$ and $\beta$)
  - Phentolamine ($\alpha_1$)
  - Urapidil ($\alpha_1$)
- Ca$^{2+}$ channel blockers
  - Nicardipine
  - Clevidipine
- ACE inhibitors
  - Enalaprilat

- NO donors
  - Nitroprusside
  - Nitroglycerin
  - Isosorbide dinitrate
- BNP analogue
  - Nesiritide
- Dopamine agonist
  - Fenoldopam
- Direct vasodilator
  - Hydralazine
What Is Used Most Commonly?

- Labetolol: 32%
- Metoprolol: 17%
- Nitroglycerin: 15%
- Hydralazine: 15%
- Nicardapine: 8%
- Sodium nitroprusside: 5%
- Other: 8%
### Specific Indications

<table>
<thead>
<tr>
<th>Comorbidity</th>
<th>Preferred Agent(s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute aortic dissection</td>
<td>Esmolol&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>Acute congestive heart failure</td>
<td>Nesiritide,&lt;sup&gt;c&lt;/sup&gt; nitroglycerin, nitroprusside</td>
</tr>
<tr>
<td>Acute intracerebral hemorrhage</td>
<td>Labetalol, nicardipine</td>
</tr>
<tr>
<td>Acute ischemic stroke</td>
<td>Labetalol, nicardipine</td>
</tr>
<tr>
<td>Acute myocardial infarction</td>
<td>Clevidipine,&lt;sup&gt;d&lt;/sup&gt; esmolol, labetalol, nicardipine, nitroprusside</td>
</tr>
<tr>
<td>Acute pulmonary edema</td>
<td>Nesiritide,&lt;sup&gt;c&lt;/sup&gt; nitroglycerin, nitroprusside</td>
</tr>
<tr>
<td>Acute renal failure</td>
<td>Clevidipine, fenoldopam, nicardipine</td>
</tr>
<tr>
<td>Eclampsia or preeclampsia</td>
<td>Hydralazine, labetalol, nicardipine</td>
</tr>
<tr>
<td>Perioperative hypertension</td>
<td>Clevidipine, esmolol, nicardipine, nitroprusside</td>
</tr>
<tr>
<td>Sympathetic crisis or catecholamine toxicity</td>
<td>Clevidipine, fenoldopam, nicardipine, phentolamine</td>
</tr>
</tbody>
</table>

<sup>a</sup>Agents listed in alphabetical order, not in order of preference.

<sup>b</sup>May be used in combination with a vasodilatorlike dihydropyridine calcium-channel blocker or nitroprusside; however, β-blockade must precede administration of these agents.

<sup>c</sup>Use is controversial.

<sup>d</sup>May be used in patients with heart rate of <70 beats/min.
How Low Should You Go?

• Simple answer
  - 25% reduction in MAP within 1\textsuperscript{st} hour
  - Target ~ 160/100 mmHg by 2-6 hours
How Low Should You Go?

• Better answer
  - It really depends on clinical condition
    • Less aggressive with ischemic stroke
    • More aggressive with hemorrhagic stroke, acute HF and aortic dissection
AHA/ASA Recommendations for BP Management in AIS

1. Patients eligible for treatment with intravenous thrombolytics or other acute reperfusion intervention and SBP > 185 mm Hg or DBP > 110 mm Hg should have BP lowered before the intervention. A persistent SBP of > 185 mm Hg or a DBP > 110 mm Hg is a contraindication to intravenous thrombolytic therapy. After reperfusion therapy, keep SBP < 180 mm Hg and DBP < 105 mm Hg for at least 24 hours.

2. Patients who have other medical indications for aggressive treatment of BP should be treated.

3. For those not receiving thrombolytic therapy, BP may be lowered if it is markedly elevated (SBP > 220 mm Hg or DBP > 120 mm Hg). A reasonable goal would be to lower BP by approximately 15% during the first 24 hours after onset of stroke.

4. In hypotensive patients, the cause of hypotension should be sought. Hypovolemia and cardiac arrhythmias should be treated and in exceptional circumstances, vasopressors may be prescribed in an attempt to improve cerebral blood flow.
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AHA/ASA Recommendations for BP Management in ICH

1. If SBP is >200 mm Hg or MAP is >150 mm Hg, consider aggressive reduction of BP.

2. If SBP is >180 mm Hg or MAP is >130 mm Hg and ICP may be elevated, consider monitoring ICP and reducing BP to keep cerebral perfusion pressure between 60 and 80 mm Hg.

3. If SBP is >180 mm Hg or MAP is >130 mm Hg and there is no evidence of or suspicion of elevated ICP, consider modest BP reduction (eg, MAP of 110 mm Hg or target blood pressure of 160/90 mm Hg).
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AHA/ACC Recommendations for BP Management in Acute HF

The role of intravenous vasodilators for the patient hospitalized with HF can not be generalized. The goals of HF therapy with vasodilators, in the absence of more definitive data, include a more rapid resolution of congestive symptoms; relief of anginal symptoms while awaiting coronary intervention; control of hypertension complicating HF; and, in conjunction with ongoing hemodynamic monitoring while the intravenous drug is administered, improvement of hemodynamic abnormalities prior to instituting oral HF medications.
Wrap Up

• Critical first step is to differentiate true emergencies from poorly controlled chronic hypertension

• Intervention for emergencies should be driven by condition-specific goals
  - Involve more than just a number!
  - Equate with problems caused by acute HT
  - Best achieved by using co-morbidity congruous agents
THANK YOU

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