Hypertensive Urgency and Emergency

First described by Volhard and Fahr in 1914; 1st study in 1939 by Keith
Estimated 60 million American have HTN

Common clinical occurrences → 27% of all medical emergencies presenting to ED
3% of all ED visits
Affect 1% of pts with HTN

Emergency → arbitrarily DBP>120, but more important is evidence of end-organ damage
* Encephalopathy, CVA, aortic dissection, unstable angina, ARF, AMI
* Rising BP thought to cause endothelin damage. This injury leads to platelets and fibrin deposition which leads to ischemia; releasing vasoconstrictor substances which further increase peripheral resistance.

Urgency → elevated BP but without evidence of end-organ damage; many article still use DBP>120.

Epidemiology → higher incidence among African-Americans, elderly, 2:1 ♂ to ♀
Majority of pts have h/o HTN
Common causes:
* Antihypertensive med withdrawal
* Renovascular hypertension
* Head trauma
* Autonomic hyperactivity
* Postoperative (4-35% of post-op pts)

Approach to the patient → 1) History, assess for end-organ damage
* Encephalopathy—HA, altered LOC, seizures, retinopathy, etc.
* Cardiac—angina, MI, decompensation (dyspnea, orthopnea, edema)
* Renal—ARF, oliguria, hematuria
* Pregnancy—HA, visual changes, seizures, RUQ pain, others as above
* H/o meds use, withdrawal, OTC drugs—esp. sympathomimetic, decongestants, antidepressants, amphetamines, cocaine, herbs

2) PE, start with ABCs, mental status
* Measure BP in both arms (R arm ~5-10mmHG higher) with appropriate cuff, compare manually with automatic
* Fundoscopic exam—papilledema, hemorrhages, exudates
* Careful cardiac, pulmonary and neurological exam

3) Labs
* CBC
* U/A
* Chemistries
* CXR
* EKG
* Echo
* Consider head imaging if neurological symptoms

Management → Emergencies require control of BP to stop ongoing organ damage (not to normal)
* Goal is to decrease MAP by 25% over 2-4hrs; and to 160/100 by 6hrs
* Unless dissection, then to SBP 120 in 30min
* Avoid rapid drop; may lead to renal, cerebral or coronary ischemia
* Frequent monitoring of BP response, q15-30mins

Urgencies require gradual decline in BP over days, usually with PO medications
CVA should not be lowered in most cases

Medications → clonidine (Catapres) PO or transdermal
* stimulates α-2 receptors (centrally acting)
* 0.1mg q20 mins used in HTN urgencies
* onset 30-90 mins, duration 6-8 hrs
* T ½ 12 hrs
* excellent choice when rapid control not required
enalaprilat (Vasotec) IV (1.25mg = 5mg PO)
* ACEI
* 1.25mg initially over 5 min (max 5mg q6 hrs)
* onset 15 mins, duration 12-24 hrs
* T ½ 11 hrs
* contraindicated in pregnancy

esmolol (Brevibloc) IV
* cardio selective β₁ receptor blocker
* 0.5mg/kg followed by infusion at 25-300mcg/kg/min
* onset 60 sec, duration 10-20 mins
* T ½ 9 mins
* not dependant on renal or hepatic function; safe in SV dysrythmias

fenolopam (Corlopam) IV
* dopamine agonist; acts in proximal/distal tubules
* 0.1mcg/kg/min, titrate 0.05mcg/kg/min (max 1.6mcg/kg/min)
* onset 5mins, max response 15min; duration 30-60 mins
* T ½ 5 mins
* advantage ↑ renal flow, diuresis, Na excretion (↑CrCl, UOP)
* may be drug of choice in pts with renal impairment

labetalol (Normodyne, Trandate) PO or IV
* α₁, β₁, β₂ receptor blocker
* 20mg IV over 2 mins, then 20-80 mg q10min or 1-2mg/min (max 300mg/d)
* onset 2-5 mins, peak at 5-15 mins, lasts 2-4 hrs
* 100-200mg PO, repeat q6 hrs prn
* T ½ 5-8 hrs
* maintains CO, cerebral, renal, coronary blood flow

nitroglycerine PO, SL, IV, transdermal
* stimulates cGMP production, results in venodilatation; reduces preload, CO, undesirable in pts with compromised flow; hypotension, reflex tachycardia
* 5-10mcg/min, increase by 5mcg/min q3-5 mins for response
* onset 2-5 mins
* T ½ 1-3 mins, metabolites up to 40 mins
* may be adjunct in pts with ACS, or acute pulmonary edema

nitroprusside (Nitropress) IV
* dilates peripheral vessels; decrease afterload and preload
* 0.05mcg/kg/min, titrate to max 2mcg/kg/min
* onset in seconds, lasts 3-5 mins
* T ½ 2 mins (circulating nitroprusside); T ½ 3 days (thiocyanate)
* metabolized into cyanogens, which is converted into thiocyanate in liver; pts with acute or chronic renal or hepatic disease, cyanide poisoning may occur after prolonged administration (>48 hrs) or if given too rapidly.
* only use when other agents not available

Other Agents† nifedipine (Adalat/Procardia) PO, SL; directly dilates vessels, sudden decrease in BP may precipitate cerebral, renal, or myocardial ischemia, therefore should not be used.

hydralazine IM, IV; directly dilates peripheral vessels, may have initial latent period 5-15min, then often leads to precipitous fall lasting up to 12 hrs. Been shown to bind to walls of muscular arteries, which may explain long effect of drug. Because of prolonged, unpredictable hypotensive effects and inability to titrate it should be avoided.
### Recommended Treatments for Hypertensive Crises

<table>
<thead>
<tr>
<th>Condition</th>
<th>Preferred Treatment</th>
<th>Mechanism</th>
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<tbody>
<tr>
<td>Acute Aortic Dissection</td>
<td>labetalol or esmolol + mitroprusside</td>
<td>Goal to ↓ pulsatile load by ↓ BP, and ↓ force of LV contraction</td>
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<tr>
<td>AMI</td>
<td>labetalol or esmolol + NTG</td>
<td>Maintains blood flow, ↓ preload/CO</td>
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<tr>
<td>ARF</td>
<td>fenoldopam</td>
<td>↑ renal blood flow</td>
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<tr>
<td>CVA</td>
<td>fenoldopam or labetalol</td>
<td>Reserve tx until DBP&gt;120mmHg; if treat ↓ by 20% in 24 hrs with short acting agent that will preserve flow; &gt;2/3 will spontaneously fall over 10d</td>
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<tr>
<td>Encephalopathy</td>
<td>labetalol</td>
<td>Preserves cerebral blood flow</td>
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<tr>
<td>Pulmonary Edema</td>
<td>fenoldopam, NTG, diuretics</td>
<td>Diuresis, decrease preload</td>
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<tr>
<td>Sympathetic Crisis</td>
<td>fenoldopam or CCB; NOTE: Ø β blockers</td>
<td>CCB→Decrease hemodynamic response, ?alleviate vasoconstriction due to cocaine (isradipine/Dynacirc shown to ↓ pressor effects of cocaine and methamphetamine) βB→inhibits β receptor induced vasodilation results in unopposed &amp; vasoconstriction</td>
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**REFERENCES:**

Cherney, D and Straus S.  Management of Patients with Hypertensive Urgencies and Emergencies: A Systematic Review of the Literature. JGIM 2002; 17: 937-945


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