

HYPERTENSIVE EMERGENCIES



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Hypertensive Emergencies : *Objectives*

- **Distinguish which hypertensive presentations require immediate therapy**
- **Describe appropriate therapies for each presentation**
- **Describe the risks of treatment**
- **Discuss the advantages and disadvantages of currently available antihypertensive drugs**

Cases

- **Asymptomatic 65 year-old, BP 200/115 (143)**
- **Embolic CVA, BP 215/105 (142)**
- **Hemorrhagic CVA, BP 200/100 (133)**
- **SAH, BP 180/100 (127)**
- **Aortic dissection, BP 175/105 (128)**
- **Pregnant female, BP 150/100**
- **Encephalopathy, BP 260/160 (194)**
- **Acute pulmonary edema, BP 220/120 (153)**

History of Hypertension

- “It is a mistake, and one made not infrequently, to begin treating the high blood pressure as if it were a disease.”
 - MacKenzie, 1908
- HTN as an associated symptom rather than as a cause of disease
- “Essential hypertension” (i.e., FDR)

Chronic Hypertension

- **Increases risk of**
 - atherosclerosis (CAD, MI)
 - cerebrovascular disease (CVA)
 - renal disease
- **These are long-term risks**
- **Elevation of BP is often a physiologic response to an acute condition**
- **Aggressive treatment of acute HTN may increase morbidity and mortality**

Hypertensive Emergencies : *Definition*

- **A rapid decompensation of vital organ function secondary to an inappropriately elevated BP**
- **Require lowering of BP within 1 hour to decrease morbidity**
- **Not determined by a BP level, but rather the imminent compromise of vital organ function**

Hypertensive *Emergencies*

- **CNS - Hypertensive encephalopathy**
- **CVS**
 - **Acute myocardial ischemia**
 - **Acute cardiogenic pulmonary edema**
 - **Acute aortic dissection**
 - **Post-op vascular surgery**
- **Renal - Acute renal failure**
- **Eclampsia**
- **Catechol excess- Pheochrom, Drugs**

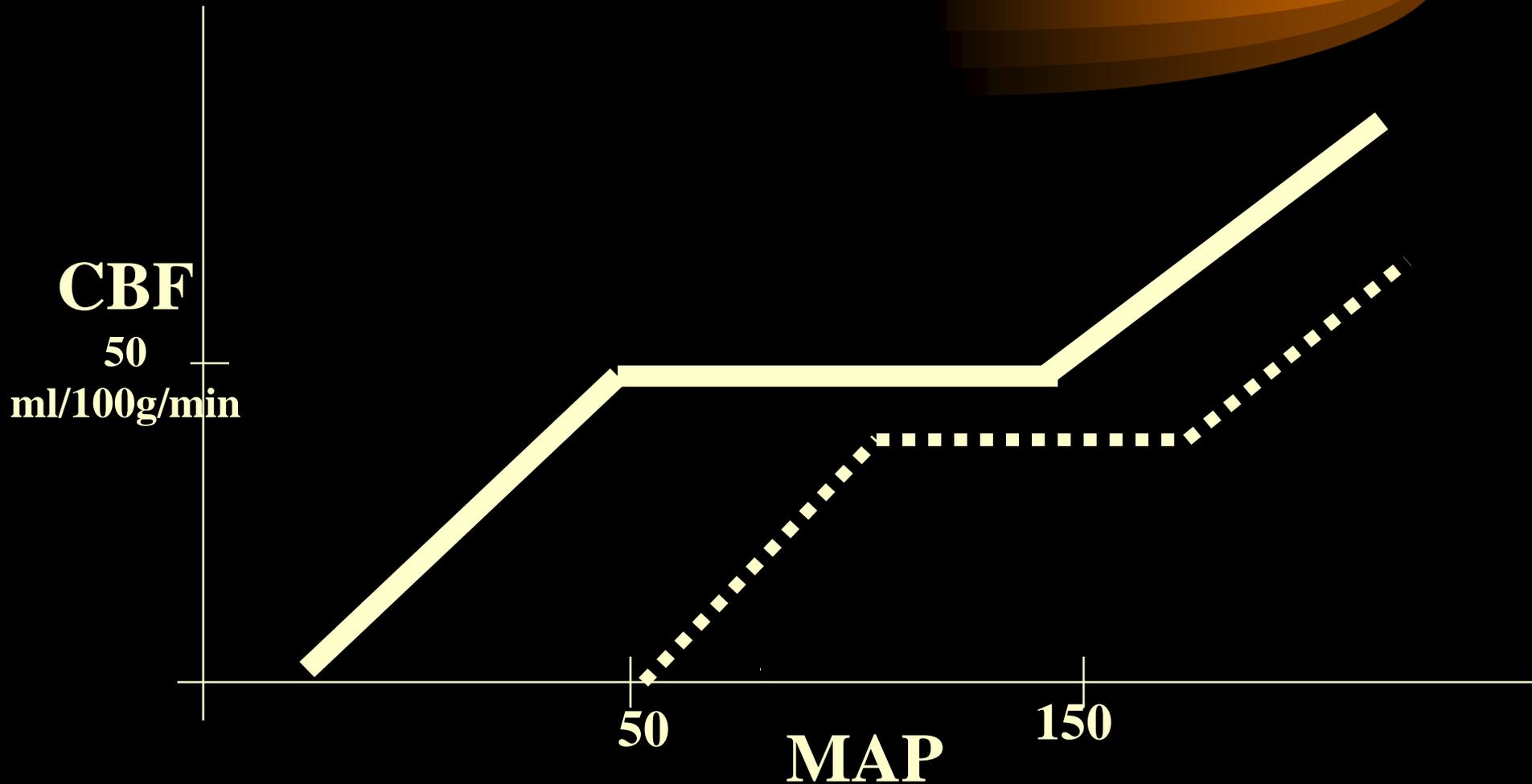
Hypertensive *Emergencies*

- High BP WITHOUT acute end-organ dysfunction IS NOT a hypertensive emergency
- “Hypertensive Pseudoemergency”

Cerebral Blood Flow

- **$CBF = CPP / CVR$**
- **$CPP = MAP - ICP$**
- **$MAP = DBP + 1/3 PP$**
- **Cerebral autoregulation**
 - normal between 50 - 150
 - 70/40 to 190/130
- **Vascular stenosis**

Cerebral Autoregulation



Cerebral Autoregulation

- **Shift to right**
 - **Chronic hypertensives**
 - **ICH, SAH, Ischemic infarct**
 - **Trauma**
 - **Cerebral edema**
 - **Age, atherosclerosis**
- **Some hypertensives suffer decrease CBF at MAP higher than 120**

Cerebral Autoregulation

- **CPP below lower limit**
 - **hypoperfusion with ischemia**
- **CPP above upper limit**
 - **“breakthrough” vasodilation**
 - **Segmental pseudospasm (“sausage-string”)**
 - **fluid extravasation**

Pathophysiology of Hypertensive Emergencies

- **Rate of change of BP determines likelihood**
- **Chronic HTN lowers probability**
 - adaptive vascular changes protect end-organs from acute changes in BP
- **Previous normotensives (eclampsia, acute GN) develop signs and symptoms at lower BP's**

Pathophysiology of Hypertensive Emergencies

- **Endothelial Role in BP Homeostasis**
 - Secretion of vasodilators (NO, Prostacyclin)
- **Sudden increased vasoreactivity**
 - norepinephrine, angiotensin II
 - activation of renin-angiotensin-aldosterone

Pathophysiology of Hypertensive Emergencies

- ? ATII direct cytotoxicity to vessel wall
- ? mechanical stretching
- **Inflammatory vasculopathy**
 - cytokines, endothelial adhesion molecules
- **Loss of endothelial function**
 - permeability
 - inhibition of local fibrinolysis
 - activation of coagulation cascade

Therapeutic considerations in hypertensive emergencies

- **Need for rapid reduction of BP**
- **Potential complications of therapy**
 - **Prevalence of cerebrovascular disease and coronary artery disease (Stenotic lesions)**
 - **Altered cerebral autoregulation**
 - **Impaired baroreflexes**
 - **Blood viscosity**
 - **Ability to increase oxygen extraction**

How far can BP be safely lowered?

- **Lower limit usually 25% below MAP**
- **50% of chronic hypertensives reached lower autoregulation limit with 11 to 20% reduction in MAP**
- **50% had lower limit above usual mean**
 - Kanaeko et al; J Cereb Blood Flow Metab 3:S51,1983
- **Most ischemic complications develop with reductions greater than 20 - 30 % (over 24 to 48 hours)**
- **Blindness, paralysis, coma, death, MI**

Initial Lowering of BP : **Therapeutic Guidelines**

- **Do not lower BP more than 20% over the first 1 to 2 hours unless necessary to protect other organs**
- **Decreasing to DBP of 110 or patients “normal” levels may not be safe**
- **Further reductions should be very gradual (days)**
- **Follow neuro status closely**

Concept of Hypertensive Urgencies

- **Potentially dangerous BP elevation without acute, life-threatening end-organ damage**
- **Examples (controversial!)**
 - **Retinal changes without encephalopathy or acute visual symptoms**
 - **High BP with nonspecific Sx (headache, dizziness, weakness)**
 - **Very high BP without symptoms**

Hypertensive Urgencies

- **Severe elevation of BP (DBP > 115)**
- **No progressive end-organ disease**
- **Joint National Committee on Detection, Evaluation, and Treatment of HBP**
 - **1984 - lower BP within 24 hours**
 - **1988 - urgent therapy rarely required**
 - **1993 - Gradual lowering of BP**
 - **Risks of rapid reduction (cerebral and myocardial ischemia)**



Pharmacologic
Therapy

Nitroprusside

- **Arteriolar and venous dilation**
- **Predictably effective in lowering BP**
- **Usual dosage 0.5 - 8 $\mu\text{g}/\text{kg}/\text{min}$**
 - **50 mg/250 ml D5W; start @ approx.
10 $\mu\text{drops}/\text{min}$ (10 ml/hr) in 70 kg patient =
0.5 $\mu\text{g}/\text{kg}/\text{min}$**
- **Potential cyanide or thiocyanate toxicity
with prolonged infusion**

Nitroglycerin

- **Predominant venodilation at low infusion rates; significant arteriolar dilation at higher dosages**
- **Effective in management of hypertension complicated by CHF or cardiac ischemia**
- **Usual dosage 10-250 $\mu\text{g}/\text{min}$**
 - **50 mg/250 ml D5W; start at approx..**
3 $\mu\text{drops}/\text{min}$ [3 ml/hr] = 10 $\mu\text{g}/\text{mins}$

Nitroglycerin and *Nitroprusside*

- **Rapid onset and offset; ability to smoothly titrate BP**
- **Potential hypotension and end-organ hypoperfusion**
- **Require continuous IV infusion, constant patient monitoring**
- **Adversely effect cerebral autoregulation**
- **May increase ICP**

Nifedipine

- **Peripheral and coronary arteriolar v.d.**
- **Rapid onset of antihypertensive effect**
 - **5-20 minute onset**
 - **peak effect in 30-60 min**
 - **duration 4-5 hr**
- **Potential hypotension and/or reflex cardiac stimulation**
- **Several case reports of cerebral or myocardial ischemia after rapid decrease**

Sublingual Nifedipine

- “Should a Moratorium be Placed on Sublingual Nifedipine capsules given for hypertensive emergencies and pseudoemergencies?”
 - Grossman, Messerli, Grodzicki, Kowey
 - JAMA, 276 : 1328 - 1331,1996

Sublingual Nifedipine

- **“Inappropriate physician habits in prescribing nifedipine capsules in hospitalized patients”**
 - Rehman et al; Am J Hypertension 9 ; 1035, 1996
- **Ordered over phone for asymptomatic**
- **Arbitrary use**
- **No evidence of bedside evaluation in 98%**
- **No follow-up exam documented**

Labetalol

- **Combined α , β adrenergic blockade**
- **Usual contraindications to β -blockade**
- **Rapidly effective when given IV;**
- **Onset < 5 min, peak 5-10 min, duration 2-6 hr (sometimes longer)**
- **Usual dosage 20 mg IV, then 40-80 mg IV q 10-15 min until achieving desired effect, or total of 300 mg**

Diazoxide

- **Rapid effect when given as IV bolus**
- **Potential hypotension (long-lasting) → cerebral and myocardial ischemia**
- **Marked reflex increase in HR and CO (myocardial oxygen demand)**
- **Rarely a first-line**
- **Dose 1-3 mg/kg IV bolus q 10-15 min until desired BP achieved**

Hydralazine

- **Unpredictable hypotensive effect**
- **Delayed onset compared to other parenteral agents**
- **Reflex increase in HR and CO**
- **Largely outmoded for acute therapy except in pre-eclampsia/eclampsia, where it is “traditional” therapy**
- **Adverse effects on cerebral autoreg**

Clonidine

- **Central α -agonist; \downarrow sympathetic tone to heart and peripheral vessels**
- **Usual regimen: 0.1-0.2 mg po, then 0.1 mg po q hr until desired BP achieved**
- **Onset 30-60 min, peak 2-4 hr, duration 6-12 hr**
- **Sedation may interfere with neurologic assessment of pt**
- **Rarely a first-line agent**

ACE inhibitors

- **IV enalaprilat, oral captopril potentially useful for acute BP reduction**
- **Little clinical experience in patients with hypertensive emergencies**
- **Difficult to titrate (sometimes ineffective, sometimes excessive BP ↓)**
- **Positive effects on cerebral autoregulation**

New Agents

- *Fenoldopam*
 - *peripheral Dopamine-1 receptor agonist*
 - *direct vasodilation*
 - *renal artery vasodilation*
 - *natriuresis*
- *Nicardipine*
 - *dihydropyridine CCB*



Management of
Specific Hypertensive
Emergencies

Hypertensive Encephalopathy

- **Abrupt, sustained increased BP exceeds limits of cerebral autoregulation**
 - **MAP 150 -200**
- **Variable vasospasm, edema, hemorrhages**
- **Headaches, nausea, vomiting, confusion**
- **Patchy focal neuro deficits**
- **Papilledema, retinopathy**
- **Signs + symptoms resolve with reduction of BP**

Hypertensive Encephalopathy: *Differential Dx*

- **Stroke (Ischemic)**
- **Intracranial (intracerebral or subarachnoid) hemorrhage**
- **Intracranial mass**
- **Encephalopathy due to drug ingestion, CNS infection, uremia**

Hypertension with Stroke *Syndromes*

- **Need for BP therapy controversial**
 - rebleed, hemorrhagic transformation
 - increased edema and ICP
- **Hypertension often transient, physiologic response which resolves spontaneously**
- **BP reduction may cause ischemic neurologic deterioration**
 - Ischemic penumbra
 - Cerebral autoregulation (right shift)
- **Cautious reduction of very high BP**

Subarachnoid Hemorrhages

- **20% rebleed within 2 weeks (24 hrs)**
- **Increased risk if SBP >160 or MAP > 110**
- **No study has shown that treatment of BP reduces risk of rebleeding**
- **Acute right shift of curve (ICH,hydrocephalus)**
- **Nimodipine for cerebro-protection (vasospasm)**
- **Cautious decrease in BP by 20% initially, then below SBP of 160 (if not yet clipped)**

Intracerebral *Hemorrhage*

- **HTN associated with increased mortality**
- **HTN may be a marker for more advanced chronic arterial compromise**
- **Physiologic response to increased ICP from clot**
 - **Decrease in BP may raise ICP**
- **Ischemic penumbra may exist in ICH**
- **No evidence that acute lowering of BP reduces risk of hematoma expansion, rebleed rare after 12 hrs**
- **Rate of 24-hour BP decline and mortality after spontaneous ICH**
 - **Qureshi et al; CCM 1999, 27: 480 - 485**

Intracerebral *Hemorrhage*

- **NSA recommendations**
 - **SBP > 220 or DBP > 120**
- **NINDS recommendations**
 - **SBP > 180 , MAP > 130**
 - **Lower BP to MAP 100 - 130**
- **Control of BP not been demonstrated to decrease ongoing or recurrent bleeding**

Thromboembolic (Ischemic)

CVA's

- **NSA recommendations**
 - SBP > 220, DBP > 120
- **NINDS recommendations**
 - DBP > 140 - NTP
 - SBP > 220, DBP > 120, MAP > 130
 - Labetalol, Enalapril, esmolol, Nitropaste
- **Ischemic penumbra**
- **Thrombolytic therapy**

Aortic Dissection

- **Tear in intima → separation or “dissection” of wall longitudinally**
- **50% mortality in first 48 hours; begin treatment based on suspicion of Dx**
- **Decrease pulse wave contour (dP/dT)**
- **Therapeutic regimens: (SBP 100 -120, HR < 80)**
 - propranolol plus nitroprusside
 - labetalol
 - trimethaphan
- **Definitive diagnosis (CT, TEE, aortography, MRI) after control of BP, contractility, pain**

Acute LV failure / Acute cardiac ischemia

- **HTN→ increased afterload; may precipitate LV failure or ischemia**
- **Dyspnea, pain, anxiety may cause HTN**
- **Specific BP therapy indicated if patient remains hypertensive after conventional measures for CHF or ischemia**
- **NTG, NTP, ACEI**
- **BB, CCB**

Pre-eclampsia/ Eclampsia

- **Preeclampsia**
 - mild = 140/90 with proteinuria
 - severe = 160/110, 5 gm protein, Sx
- **"Standard" therapy is hydralazine**
- **Other agents: Nifedipine, labetalol, diazoxide (small doses), methyldopa**
- **nitroprusside (risk of fetal CN toxicity)**
- **Additional measures: MgSO₄; Delivery**

Drug Associated Hypertension

- "Hypercatecholamine state"

Cases

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CLINICAL CHARACTERISTICS OF HYPERTENSIVE CRISIS

- **BP: Usually >140 mm Hg diastolic**
- **Funduscopy findings:**
 - Hemorrhage, exudate, papilledema
- **Neurological status:**
 - Headache, confusion, somnolence, stupor, visual loss, focal deficits, seizures, coma
- **Cardiac findings:**
 - Prominent apical impulse, cardiac enlargement, congestive failure
- **Renal: Oliguria, azotemia**
- **Gastrointestinal: Nausea, vomiting**

DIFFERENTIATED FROM A HYPERTENSIVE CRISIS

Acute left ventricular failure

Uremia from any cause, particularly with volume overload

Cerebrovascular accident, Subarachnoid hemorrhage

Brain tumor, Head injury

Epilepsy (postictal)

Collagen diseases (i.e., lupus), with cerebral vasculitis

Encephalitis

Overdose and withdrawal from narcotics, amphetamines

Hypercalcemia

Acute anxiety with hyperventilation syndrome

circumstances.

