Hypertension in Emergency Medicine

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Questions to be addressed

In the Emergency Department
- When should HBP be treated?
- When should HBP not be treated?
- When should outpatient therapy be started?
- What agents should we use?
  - For what conditions?

Agenda for Our Discussion

- General Considerations
- Blood Pressure Readings in the ED
- Pathophysiology
- Pharmacologic Treatment Modalities
- Specific Emergencies Requiring BP Reduction in the ED
- Post ED Therapy
- Summary - Hypertension in the ED

Doctor,
The Patient’s Blood Pressure is Elevated!

Michael Jay Bresler, M.D
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• I will use primarily generic names
• But I will also include on the slides the brand names since these are most commonly used in the real world - where we practice
• When there are several brand names I will try to include them all
• I have no idea which companies make which drugs

I have no financial relationship with any drug company

What is Normal Blood Pressure??

Prehypertension
130-139/80-90
• Compared with normal BP
  – Double the risk for developing hypertension.
• Lifestyle & diet intervention warranted
  Joint National Committee on Hypertension, 2003

Incidence of Hypertension in U.S.A.
• > 140/90 (HTN)
  – 27% of adults
• > 130/90 (pre HTN + HTN)
  – 60% of adults!
  – 88% > 60 years old
  – 40% ages 18-39!!
  Wang Arch Intern Med, 2004

Scope of the Problem
• Normotensive people at age 55 have a 90% lifetime risk of developing HTN
  (Ref: Vasan)
• Between age 40-70, the risk of cardiovascular disease doubles for every (independent variables)
  – 20 mm Hg systolic above 115
  – 10 mm Hg diastolic above 70
  » Lewington Lancet, 2002

General Considerations

Prehypertension

**Should BP Rise with Age?**

**NO!!**

In societies with natural diet, less salt, and less obesity, more exercise
–BP does not rise with age
• Diet is a particular problem -
  –We love our unhealthy diet!

**BP and Gender**

• Estrogens protect
• After menopause, women catch up with men and eventually surpass the men
  (in blood pressure that is….)

**BP and Ethnicity**

• Incidence of HTN
  –1.5 - 2 x more common in Blacks
    • 1 in 3 African Americans
    • 1 in 4-5 Caucasian and Hispanic Americans
    • ? Asians
• African Americans
  –HTN begins earlier
  –More end organ damage
  –ACEI’s & ARB’s less effective

**High Blood Pressure Readings in the Emergency Department**

**Is that reading real?**

• Asymptomatic E.D. patients with BP >140/90
  –BP at home bid
  –> 1/2 continued >140/90
  –Most of rest continued at pre-hypertensive level
  –Independent of pain or anxiety in E.D.

• E.D. patients with BP >140/90 followed in clinic
  –54% continued >140/90
    »Cline Acad Emerg Med 2000

**Question**

Are ED BP readings accurate & reliable for screening asymptomatic patients for HTN?
Doctor,
The Patient’s Blood Pressure is Elevated!

ACEP Clinical Policy

- Level B Recommendation
  – If SBP persistently > 140 or
  – If DPB persistently > 90
  Refer for follow up of possible HTN and BP management


Question

Do asymptomatic patients with elevated BP benefit from rapid lowering of their BP?

ACEP Clinical Policy

- Level B Recommendation
  – Initiating Tx in the ED is not necessary if F/U is available
  – Rapid lowering of BP is not necessary and may be harmful
  – When Tx is initiated, BP should be lowered gradually and should not be expected to be normalized during the ED visit


HBP in the ED

- Most useful terminology
  – Hypertensive Emergency
  – Hypertensive Urgency
  – Elevated Blood Pressure

Why is this the most useful classification?

HBP in the ED

- Hypertensive Emergency
  – Treated in ED with IV meds
- Hypertensive Urgency
  – May be treated in ED - oral meds OK
  – Usually give prescription
- Elevated Blood Pressure
  – Not treated in ED
  – May or may not give prescription
  – We should refer for further evaluation

Hypertensive Emergency

- By definition
  – Evidence of acute end organ damage
  – Usually brain, heart, or kidney
- Definition implies that organ dysfunction is caused by acute HPB, rather than vice versa
- Systolic usually > 220
- Diastolic usually > 130
**Hypertensive “Urgency”**

- Major elevation of BP (roughly in range of >220/>120) but
  – Without evidence of acute organ failure
  – No acute symptoms directly attributable to elevated BP

**Hypertensive Urgency**

- Treatment may be administered in the ED if BP remains very elevated
  – Controversial
  – Trend toward *not* treating in the ED
- Outpatient treatment should generally be initiated, however
- Basic studies may be indicated

**Diagnostic Studies in the ED**

- Incidental finding of moderate HBP
  – ED workup not necessarily indicated -> refer
- If initiating outpatient treatment
  – Basic studies in ED may be considered
    – CBC, lytes, renal, glucose, UA, EKG
- If ED treatment required
  – Basic studies usually indicated
- If hypertensive emergency - basic plus
  – Studies specific to disorder (CT, etc.)

**Pathophysiology of Hypertension**

**Regulation of Blood Pressure**

**A Balance Between**

- Inherent stiffness of the arterial wall
- Vasodilation
- Vasoconstriction

**Inherent stiffness of arterial wall**

Cardiovascular risk factors lead to:

- Replacement of elastin in arterial walls by collagen and fibrous tissue
  – Decreased compliance
  – Increased resistance
- Endothelial dysfunction
Doctor,  
The Patient’s Blood Pressure is Elevated!

Pathophysiology

\[ \uparrow \text{BP} \rightarrow \text{Endothelial wall stretch/stress} \]

- Endothelial Dysfunction
  - Capillary permeability
  - Depletion of NO
  - Inflammation

Acute Regulation of BP

- Vasodilation
  - Beta-2 adrenergic innervation
  - Nitric oxide \( \rightarrow \) c-AMP

- Vasoconstriction
  - Alpha-1 adrenergic innervation
  - Circulating catecholamines
  - Angiotensin II

Renin-Angiotensin-Aldosterone

- Angiotensinogen
- Renin
- Angiotensin I
- ACE
- Angiotensin II

Renin-Angiotensin-Aldosterone

- Angiotensin II
  - Powerful vasoconstrictor
  - Release of aldosterone
  - Inflammatory response
  - Hypertrophy of smooth muscle cells
  - Decreased nitric oxide \( \rightarrow \) further vasoconstriction

Auto-Regulation and Hypertensive Crisis

- Organ-specific autoregulation
  - Normally maintains capillary pressure & flow within an acceptable range
    - Increased systemic BP \( \rightarrow \) vasoconstriction
    - Decreased systemic BP \( \rightarrow \) vasodilation
**Autoregulation of Cerebral Blood Flow**

- Cerebral arterial resistance varies directly with BP to maintain cerebral perfusion within acceptable limits
- “Set point” rises with chronic HBP
- Rapid ED reduction of BP may drop CPF below adequate level
- Lower BP gently;
- And *usually* never < 110 diastolic
  - Except
    - *with aortic dissection*

**Mean Arterial Pressure**

<table>
<thead>
<tr>
<th>Hypertensive Person</th>
<th>Normotensive Person</th>
</tr>
</thead>
</table>

Adapted from Elliott Crit Care Clin 2001;17:435

**Autoregulation and Hypertensive Crisis**

Hypertensive crisis

Autoregulation fails ->

- Endothelial dysfunction
- Capillary permeability & edema
- Inflammatory response
- Prothrombotic response
- Decreased nitric oxide
- Release of vasoconstrictors
- Cell necrosis

**Pharmacologic Treatment Modalities**

- Parenteral Vasodilators
- Beta Blockers
- Calcium Channel Blockers
- Angiotensin Converting Enzyme Inhibitors
- Angiotensin II Receptor Blockers
- Direct Renin Inhibitors
- Diuretics
- Others

**Parenteral Vasodilators**
Doctor,
The Patient’s Blood Pressure is Elevated!

**Parenteral Vasodilators**

Nitroprusside (Nipride™, Nitropress™)
- Arterial > venodilator
- Advantages
  - Most commonly used agent in EM
  - Extremely effective
  - Very short half-life
- Are there better agents??

**Potential problems**
- Unstable to UV light—must be wrapped
- Orthostatic hypotension—keep supine
- Metabolized to cyanide/thiocyanate
- Toxic at higher dose
  - Potentially toxic to fetus
- Tissue necrosis if extravasation
- Increases intracranial pressure

**Parenteral Vasodilators**

Nitroglycerin
- Venodilation > arterial dilation
- Good for CHF & angina
- Not a good drug for hypertensive crisis

**Beta Blockers**

- β₁ blockade
  - Lusitropic
  - (decreased cardiac contractility)
- Decrease renin
- Decrease norepinephrine
**Beta blockers**

- Advantages
  - Especially good with CAD
  - Decreased myocardial oxygen demand
  - Good with anxiety
  - Long acting preparations best for PO

**Beta blockers**

- Most useful for Emergency Medicine
  - Labetalol (IV, also alpha blocker)
  - Metoprolol (PO & IV)
  - Esmolol
    - (short acting cardioselective IV agent)
  - Among many other preparations available
    - Propranolol
    - Atenolol
    - Nadolol
    - Carvedilol (also alpha blocker)

**Calcium Channel Blockers**

- Decrease heart rate & contractility
- Dilate peripheral vasculature
- 2 classes
  - Dihydropyridines
  - Nondihydropyridines

**Calcium Channel Blockers**

- Nondihydropyridines
  - Cardiac effect > vascular
    - verapamil, diltiazem
- Dihydropyridines
  - Vascular effect > cardiac
    - nifedipine, amlodipine,
    - felodipine, nicardipine
- Dihydropyridines thus best for HBP

**Calcium Channel Blockers**

- Most useful for Emergency Medicine
- In the ED (for blood pressure control)
  - Nicardipine (Cardene™) IV
  - Clevidipine (Cleviprex™) IV
- Outpatient Rx
  - Long acting formulations of
    - nicardipine (DynaCyrc™, Cardene™)
    - nifedipine (Procardia™, Adalat™)
  - Do not use short acting dihydropyridines
Calcium Blockers vs. Nitroprusside

Advantages of IV calcium blockers (nicardipine, clevidipine)

- As effective as nitroprusside
- No cyanide/thiocyanate toxicity
- Not light sensitive; no need for foil wrap
- Less need for rate adjustment (1/3 as often)
- No need for arterial line
- No intracerebral vasodilation causing edema

Angiotensin Converting Enzyme (ACE) Inhibitors

Regulation of BP

Renin-Angiotensin-Aldosterone

Angiotensin II

- Powerful vasoconstrictor
- Release of aldosterone
- Inflammatory response
- Hypertrophy of smooth muscle cells
- Decreased nitric oxide -> further vasoconstriction

ACEI’s block these effects

ACE Inhibitors

- Also block metabolism of bradykinin
- Bradykinin is a strong vasodilator
- However, bradykinin may cause the principal potential side effects of ACEI’s
  - Cough
  - Angioedema

ACE Inhibitors

- Especially beneficial with
  - Diabetes
  - Renal failure
  - Heart failure
- Potential side effects - bradykinin mediated
  - Cough (1/10)
  - Angioedema (1/2,000)
Doctor,
The Patient’s Blood Pressure is Elevated!

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ACE Inhibitors
Most useful for Emergency Medicine
• In the ED
  – Enalaprilat IV (Vasotec™)
• Outpatient Rx examples
  – Captopril (Capoten™)
  – Benazepril (Lotensin™)
  – Enalapril/enalaprilat (Vasotec™)
  – Lisinopril (Prinivil™, Zestril™)
  – Quinapril (Accupril™)

Angiotensin II Receptor Blockers

Angiotensin II receptors blockers
• Similar therapeutic effect as ACEI’s
• Fewer side effects because unlike ACEI’s, they do not block bradykinin breakdown. Therefore:
  – No bradykinin mediated cough
  – Extremely rare angioedema
• Rx examples: losartan (Cozaar™), valsartan (Diovan™), irbesartan (Avapro™)

Direct Renin Inhibitors

ACE Inhibitors
Regulation of BP
Renin-Angiotensin-Aldosterone

Angiotensinogen

Angiotensin I

ACE

Angiotensin II

X

Angiotensinogen

Angiotensin I

ACE
**Direct Renin Inhibitor**

- Similar therapeutic effect as ACEI’s
- Fewer side effects because unlike ACEI’s, they do not block bradykinin breakdown. Therefore:
  - No bradykinin mediated cough
  - Extremely rare angioedema
- Rx examples: aliskiren (Tekturna™)

**Diuretics**

- Reduce blood volume
- Dilate vessels
- 3 types
  - Loop (furosemide) - best for diuresis
  - Thiazide (hydrochlorothiazide) - best for lowering blood pressure
  - K⁺ sparing (spironolactone)

**Diuretics**

- Advantages of thiazide diuretics
  - Inexpensive
  - Chronic Tx: at least as effective as newer drugs (ACEI & Ca blockers) in:
    - Lowering BP
    - Preventing CV complications of HBP
      (Ref: ALLHAT, 2002)
  - Most patients will require additional meds
    - (Ref: Joint National Committee on Hypertension, 2003)

**Diuretics**

Value for treating HBP in Emergency Medicine

- In the ED
  - None
- Outpatient Rx
  - Hydrochlorothiazide
  - Chlorthalidone

**Other Antihypertensive Agents**
**Alpha Adrenergic Agents**

**Blocked & Agonists**

- **Alpha-1 receptors**
  - Vasoconstriction
  - Alpha-1 blockers --> lower BP
- **Alpha-2 receptors**
  - Inhibition of sympathetic (adrenergic) NS
  - Alpha-2 agonists --> lower BP

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**Alpha Adrenergic Agents**

**Blocked & Agonists**

Alpha-1 receptors

- Vasoconstriction
  - Alpha-1 blockers --> lower BP
  - Phentolamine IV and
  - Phenoxybenzamine PO
    - Pheochromocytoma (with β-blocker)
    - MAOI toxicity

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**Alpha Adrenergic Agents**

**Blocked & Agonists**

Alpha-2 receptors

- Inhibition of sympathetic (adrenergic) NS
- Alpha-2 agonists --> lower BP

Most useful in Emergency Medicine

- Clonidine (Catapres™)
  - PO for hypertensive urgency

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**Rarely used older agents**

- Ganglionic blockers
  - Trimethaphan (Arfonad™)
- Central sympatholytics
  - Reserpine
  - Alpha methyldopa (Aldomet™)
- Direct vasodilators
  - Hydralazine (Apresoline™)
  - Minoxidil (Lonitin™)

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**Specific Emergencies**

**Requiring Blood Pressure Reduction in the ED**

- 72 year old male
- Gradual onset headache past 2 days
- Nausea & vomiting
- Blurred vision
- No motor weakness
- BP = 260/140

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**Your Patient**

- 72 year old male
- Gradual onset headache past 2 days
- Nausea & vomiting
- Blurred vision
- No motor weakness
- BP = 260/140
**Hypertensive Encephalopathy**

- Acute HTN overwhelms cerebral autoregulation ->
  - arteriolar spasm
  - cerebral ischemia
  - vascular permeability
  - edema
  - hemorrhage

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**Your Patient**

- 72 year old male
- Awakens not moving right side
- Mild headache and nausea
- BP = 180/110
- CT = early infarct signs
- What drug to lower his BP?

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**Ischemic Stroke**

Acutely elevated BP on ED presentation
- Common response to the stroke
- Probably beneficial
  - May increase CBF to ischemic region
  - Usually transient
- Don’t treat!
  - Unless stays very high
  - Danger of cerebral hypoperfusion

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**Ischemic Stroke**

- If BP remains very high, *gentle* reduction *may* be reasonable
  - 10-15% reduction of MAP
  - To diastolic no lower than 110
- May lower to 180/110 in ischemic stroke to meet t-PA criteria

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**Ischemic Stroke**

- “The level of blood pressure that would mandate such treatment is not known, but consensus exists that medications should be withheld unless the systolic blood pressure is >220 mm Hg or the diastolic blood pressure is >120 mm Hg”
  - Class I, Level of Evidence C
  
  *Adams: American College of Neurology Circulation 2007*

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**Your Patient**

- 67 year old female
- Sudden onset of severe headache and vomiting
- Not moving left side
- BP = 230/130
- CT = intracranial hemorrhage
Hemorrhagic Stroke

- Recent evidence that size of hemorrhage may be lessened – with no deleterious effect on perihematomal edema - if systolic BP is lowered to the 140’s
- Preliminary studies

Arima, Hypertension 2010
Anderson, Stroke 2010

Acute Brain Syndromes

- Hypertensive Encephalopathy
- Ischemic Stroke
- Hemorrhagic Stroke
  - What Agents Should We Use??

Hemorrhagic Stroke

- “In patients presenting with a systolic BP of 150 to 220 mm Hg, acute lowering of systolic BP to 140 mm Hg is probably safe”
- Class Ila; Level of Evidence: B
- New recommendation

Morgenstern, AHA/ASA Guidelines 2010

Acute Brain Syndromes

- Nitroprusside may not be best agent
  – Increases ICP
  – Impairs cerebrovascular reactivity to PCO₂ changes
  – Exacerbates drop in CPP in response to a given decrease in peripheral BP

(Ref: Adams)

Acute Brain Syndromes

Labetalol
Both alpha & beta adrenergic blocker
  – Theoretically
    - Alpha blockade shifts cerebral autoregulation “set point” to lower level
      (Ref: Adams)
    - Preserves CO₂ reactivity
    - Preserves CBF at lower BP level

Acute Brain Syndromes

Treatment
- Controlled reduction of BP over 1 hour
- Never < 110 diastolic
  – Labetalol
  – Nicardipine – increasingly used by stroke neurologists
  – Clevidipine and Fenoldopam may be alternatives
Doctor,
The Patient’s Blood Pressure is Elevated!

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Your Patient

• 65 year old male with hx of HBP
• Sudden onset of excruciating chest pain radiating to the back
• EKG = LVH
• CXR = ? Widened mediastinum
• BP = 180/110

Acute Aortic Dissection

• Goals
  – Rapid reduction of BP to nearly hypotensive level
    • Systolic 100 - 120
    • Within 20 minutes
      – The only time a rapid drop is indicated - or safe
      – Prevention of reflex tachycardia

• BP Reduction: Vasodilator
  – Nitroprusside (most rapid)
  – Alternatives: fenoldopam, nicardipine

• Tachycardia prevention: Beta blocker
  – Metoprolol or esmolol

• Alternatively
  – Labetalol alone ->
    • alpha + beta blockade

Your Patient

• 55 year old female
• Chest pain for 1 hour
• Dyspnea increasing x 2 days, severe x 2 hours
• Rales throughout chest
• CXR = acute pulmonary edema
• BP = 170/110

Acute Coronary Syndromes & Pulmonary Edema

• Nitroglycerin
• If BP stays high, cause is usually insufficient nitroglycerin or analgesia
  – Increase nitroglycerin infusion rate
• Nitroprusside is rarely needed
  – An indication that acute HTN may be the cause of the acute cardiac problem rather than vice versa

Your Patient

• 35 year old pregnant female
• Headache & blurred vision
• Nausea & vomiting
• Hyper-reflexic
• Pre-tibial edema
• Proteinuria
• BP = 150/90
**Eclampsia/Pre-eclampsia**

Treatment
- Classically
  - IV hydralazine
- Better alternatives
  - Labetalol, nicardipine
  - Nitroprusside falling out of favor
    - concern re fetal cyanide

**Your Patient**
- 22 year old male
- Partying with friends
- (Not your son….)
- Chest pain and dyspnea
- Freaked out
- Jittery
- BP = 220/140

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**Cocaine & Amphetamine Toxicity**

- Benzodiazepines
  - Usually effective & sufficient
- BETA BLOCKERS CONTRAINDICATED
  - Unopposed alpha adrenergic effect

**Your Patient**
- 33 year old female
- Diabetic
- Increasing creatinine over past month
- Creatinine 8.0
- Lungs with slight basilar crackles
- Cannot dialyze till morning
- BP = 220/120

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**Acute Renal Failure**

- Nitroprusside has been traditional Tx
  - Slowly metabolized by kidney
  - Danger of cyanide toxicity in ARF
- Probably safer
  - Fenoldopam
  - Nicardipine, clavidipine

**Your Patient**
- 55 year old male
- Sprained ankle
- No other symptoms
- No medical history
- Reading sports page
- Ready for discharge
- BP = 240/130
Hypertensive Urgency

- Sustained BP in range of >220/>120 without evidence of acute organ dysfunction
- Growing trend NOT to treat in the ED
- If treated, JNC-7 recommends
  - Oral clonidine
    - 0.1 - 0.2 mg PO to start
    - then 0.1 mg/hr
    - Goal: 20% reduction of MAP or to 110 diastolic

Post ED Therapy

Guidelines for Writing Prescriptions

Post ED Therapy

- If BP stays high, Rx from ED may be indicated, especially in patients with
  - Consistently > 100 diastolic
  - Chronic CHF
  - Coronary artery disease
  - Chronic renal failure
  - Diabetes

Diuretics are the Bedrock of Outpatient Therapy

- If not on HBP medication
  - Start hydrochlorothiazide (HCTZ)
    - low dose
    - 12.5 - 25 mg per day
  - If taking other HBP medication(s),
    - Add HCTZ
      - 6.25 - 12.5 mg per day

Post ED Therapy – ALLHAT recommendations

- Diuretics are the bedrock of therapy
- Probably all patients should be on a diuretic (usually a thiazide), with additional meds added as needed
- Additional meds eventually will be needed in most patients
- But start with thiazides

ALLHAT JAMA 2002
Moser J Hypertens 2007
Post ED Therapy

- If already taking a diuretic, additional drug may be tailored to other conditions
  - CAD - Beta blocker
  - CHF - ACEI or ARB
  - Renal failure - ACEI or ARB
  - Diabetes - ACEI or ARB
  - Isolated systolic hypertension
    - Long acting CCB or ACEI/ARB

Post ED Therapy

- Regardless of the ALLHAT recommendations, many physicians begin with an ACI, ARB, or beta blocker, and then add a diuretic if needed
- This alternative is acceptable for beginning treatment from the ED

Post ED Therapy - Combined Preparations

- Many new products now with varying combinations of 2 or even 3 classes of anti-hypertensive agents
- Also combinations with lipid-lowering statins
- Disadvantage - cost
- Advantage - convenience and therefore compliance

Summary

Hypertension in the Emergency Department

- High BP readings in the ED
  - Usually decline before discharge
  - Rarely require treatment
    - in the ED
  - Often do reflect real HTN
  - Sometimes warrant writing a prescription

Summary - Hypertensive Emergencies

- Hypertensive emergencies with acute organ damage require IV treatment in the emergency department
**Summary - Hypertensive Emergencies**

- In general
  - Reduce MAP about 20% gradually over at least 1 hour
  - Aortic dissection -> over 20 minutes
  - Not lower than 110 diastolic
  - As low as 100 systolic with dissection OK

- Aortic dissection
  - Nitroprusside, fenoldopam, or nicardipine
  - Beta-blocker: metoprolol or esmolol
  - OR
  - Labetalol alone

- Acute coronary syndromes
  - Nitroglycerin, analgesic
  - Beta-blockers, ?ACEI

- Acute CHF
  - Nitroglycerin, diuretic (?)
  - ? ACEI

- Cocaine/amphetamine toxicity
  - Benzodiazepine

- Pheochromocytoma
  - Nitroprusside IV or phentolamine
  - PLUS beta-blocker

- Encephalopathy

- Stroke – if treated
  - Labetalol
  - Nicardipine
  - Alternatives
    - Clevidipine, Fenoldopam

- Pre-/Eclampsia/Eclampsia
  - Labetalol or nicardipine
  - ? Hydralazine

- Acute renal failure
  - Nicardipine
  - Alternatives: Fenoldopam, clevidipine
Doctor,
The Patient’s Blood Pressure is Elevated!

Summary - Hypertensive Emergencies

- Hypertensive URGENCY
  - Clonidine PO (if treated)
- Not as a prescription, however

Summary - Outpatient Rx

Start with diuretic or add diuretic
If already on diuretic:
- CAD - beta-blocker
- CHF - ACEI or ARB
- CRF - ACEI or ARB
- DM - ACEI or ARB
- Isolated systolic HTN - long acting CCB
  – Often eventually need ACE or ARB

64 year old female you’ve diagnosed with acute bronchitis
- Initial BP = 250/130
- On no meds
- No history of hypertension
- Feels fine except for cough
- Ready for discharge: BP = 210/110

“Hey Doc, whadya want to give her?”

64 year old female you’ve diagnosed with acute bronchitis
- Initial BP = 250/130
- On no meds
- No history of hypertension
- Feels fine except for cough
- Ready for discharge: BP = 250/140

“Hey Doc, whadya want to give her?”

64 year old male complaining of severe chest pain for 3 hours
- Initial BP = 230/120
- EKG normal
- Widened mediastinum on CXR
- Repeat BP = 170/90
- “Doc, they’re ready in CT.”

“Hey Doc, whadya want to give him?”

Hypertension in Emergency Medicine

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