Update – 2014
Hypertensive Emergencies

Michael Jay Bresler, MD, FACEP

Clinical Professor
Division of Emergency Medicine
Stanford University School of Medicine

I have no conflicts of interest to disclose
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

Agenda for Our Discussion

- New ACEP Guidelines
- Medications
- Treatment of Specific Hypertensive Emergencies
Update - 2014

8th Joint National Committee - JNC8
Guidelines for Outpatient Treatment – Controversial

New ACEP Clinical Policy
Evaluation and Treatment in the ED

Update - 2014

8th Joint National Committee - JNC8
Guidelines for Outpatient Treatment
Controversial

• Guidelines loosened
  • Threshold for initiating treatment
  • Target blood pressure in older folks
Update - 2014

New ACEP Clinical Policy

October 2013

ACEP Clinical Policies
Guidelines – NOT Commandments
Older Classification

- Hypertensive Emergency
- Hypertensive Urgency
- Elevated Blood Pressure

Hypertensive Emergency – Term still used

• 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

• Treated with IV medication
New Terminology

“Asymptomatic Markedly Elevated Blood Pressure”

• No symptoms due to blood pressure
• Pressure “markedly elevated”
  – Equal to or greater than
    • 160 systolic, or
    • 100 diastolic
• Definition depends on absence of acute end organ injury, not on the BP per se
ACEP Clinical Policy - 2013

Critical Issues
in the Evaluation and Management
of Adult Patients
in the Emergency Department
With Asymptomatic Elevated Blood Pressure

ACEP Clinical Policy - 2013

First Question

In ED patients with asymptomatic elevated blood pressure, does screening for target organ injury reduce rates of adverse outcomes?
(1) In ED patients with asymptomatic markedly elevated blood pressure, routine screening for acute target organ injury (e.g., serum creatinine, urinalysis, ECG) is not required.

(2) In select patient populations (e.g., poor follow-up), screening for an elevated serum creatinine may identify kidney injury that affects disposition (e.g., hospital admission).
Second Question

*In patients with asymptomatic markedly elevated blood pressure, does ED medical intervention reduce rates of adverse outcomes?*

ACEP Clinical Policy – 2013

Second Question - Intervention

Level C recommendations.

(1) In patients with asymptomatic markedly elevated blood pressure, routine ED medical intervention is not required. [Consensus recommendation]
ACEP Clinical Policy – 2013
Second Question- Intervention

Level C recommendations.

(2)
In select patient populations (eg, poor follow-up), emergency physicians may treat markedly elevated blood pressure in the ED and/or initiate therapy for long-term control.

[Consensus recommendation]
Pharmacologic Treatment Modalities for Hypertensive Emergencies

- Parenteral Vasodilators
- Beta Blockers
- Calcium Channel Blockers

Parenteral Vasodilators
**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**

- **Fenoldopam (Corlopam™)**
  - Newer IV alternative to nitroprusside
  - Peripheral dopamine (DA-1) receptor agonist
  - Rapid onset & offset of action
  - Improves renal function?
  - Less chance of overshoot vs. nitroprusside
  - No thiocyanate toxicity or light sensitivity

**Parenteral Vasodilators**

**Nitroglycerin**

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

- $\beta_1$ 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

- **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

Hypertensive Emergencies
Requiring Blood Pressure Reduction in the ED

![Heart Image]
Auto-Regulation and Hypertensive Crisis

Organ-specific autoregulation

• Normally maintains capillary pressure & flow within an acceptable range
  – Increased systemic BP -> vasoconstriction
  – Decreased systemic BP -> 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**

Adapted from Elliott: Crit Care Clin 2001;17:435
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
Your Patient

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

**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
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

---

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
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**

- “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

Hypertensive Encephalopathy, Ischemic or Hemorrhagic Stroke

*If* treated
- **Controlled** reduction of BP over 1 hour
- Never < 110 diastolic
  - Labetalol
  - Nicardipine – increasingly used by stroke neurologists
  - Clevidipine and Fenoldopam may be alternatives
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
**Acute Aortic Dissection**

- **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
Cocaine & Amphetamine Toxicity

- Benzodiazepines
  - Usually effective & sufficient

- BETA BLOCKERS CONTRAINDICATED
  - Unopposed alpha adrenergic effect

Pheochromocytoma

- Mixed alpha + beta adrenergic toxicity
- Treatment somewhat like aortic dissection
  - Both alpha & beta blockade
- Phentolamine (alpha-blocker) or nitroprusside
  + beta blocker
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

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

Asymptomatic Markedly Elevated BP

- Screening for target organ injury?
  - Routine screening is not required
  - In selected populations (eg. poor follow-up, may affect disposition

- Treatment in the E.D.?
  - Routine medical intervention is not required
  - In selected populations (eg. poor follow-up) may treat in the E.D. or initiate therapy for long-term control


**Agenda for Our Discussion**

- New ACEP Guidelines
- Medications
- Treatment of Specific Hypertensive Emergencies

**Update – 2014 Hypertensive Emergencies**

*Michael Jay Bresler, MD, FACEP*

Clinical Professor
Division of Emergency Medicine
Stanford University School of Medicine