Background

• 30% of patients have history of hypertension prior to stroke
• 80% will present with elevated B/P
• In normotensive patients, cerebral blood flow is maintained over a wide range of MAP
  – 50-150 mm Hg
• Chronically hypertensive patients require higher MAP to maintain normal cerebral blood flow
Impaired Neurogenic CV Control

Autonomic Dysregulation

Reflex Response to Cerebral Ischemia

Baroreflex Failure

Long standing chronic hypertension

Increased Sympathetic Drive

Increased ICP

Pain

Mental Stress

Elevated B/P

Long standing chronic hypertension

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Reflex Response to Cerebral Ischemia

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Elevated B/P
“A major component of stroke care should focus upon strategies to limit neurological damage within the ischemic penumbra by controlling abnormal physiology”
What is Cerebral Autoregulation?

• Mechanism by which cerebral blood flow (CBF) remains constant across a wide range of cerebral perfusion pressures (CPP)
• Reflex vasoconstriction or vasodilation of the cerebral arterioles in response to changes in perfusion pressure
• If ICP is constant and not elevated, MAP and CPP are proportional
Eames and Colleagues

• Evidence shows that autoregulation is impaired in stroke patients
• Global impairment in dynamic autoregulation found in all stroke patients but not in controls
• Dysfunction occurred in both hemispheres
Cerebral Blood Flow

- Cerebral blood flow is dependent on systemic blood pressure
- \(\downarrow\) systemic blood pressure = \(\downarrow\) cerebral blood flow = \(\downarrow\) perfusion = neuronal death
- When cerebral blood flow is less than 12ml/100 g/min, neuronal death is likely to occur
Cerebral Blood Flow

• Not homogenous in the area of infarction
• Critical threshold below which neurons cease to function
  – May continue to survive for a short time
  – May return to normal functional state if blood flow is restored
• Time period for reversal may be first several hours
  – May depend on the location of the occlusion, rapidity of occlusion, adequacy of collateral blood flow
Cerebral Blood Flow

- Infarct: <8 mL/100 g/min
- Normal: 50 mL/100 g/min
- Penumbra: 8-20 mL/100 g/min
What does research show?

• Contradictory results
• Some show association of poor outcomes in patients who are hypertensive on admission
• Others note decreased risk of deterioration from stroke with higher B/P and worse outcomes in those patients whose B/P is decreased after admission
• Does higher B/P reflect greater stroke severity rather than causing worse outcomes?
What does the research show?

• Patients with lacunar or large artery atheroembolic /cardioembolic strokes had better outcomes when B/P was higher

• “U-shaped” relationship between B/P and mortality
  – Very low or high admission B/P linked to poor outcomes
Minnesota Project

• Lit Review
• 3 sets of guidelines related to B/P management in the acute phase
  • AHA/ASA
  • ISH (International Society of Hypertension)
  • EUSI (European Stroke Initiative)
• AHA/ASA and ISH guidelines are identical
Summery of Guidelines

ASA guideline: Treat cautiously and only when systolic BP (SBP) >220 mm Hg or diastolic BP (DBP) >120 mm Hg or mean BP >130 mm Hg.

EUSI guideline: Treatment is to be withheld unless SBP >180 mm Hg or DBP >105 mm Hg at the least, although higher thresholds are acceptable.
Figure 3.
Distribution of systolic and diastolic blood pressures that triggered as-needed medication use in the acute phase (n=126). The bold lines show the thresholds recommended by American Stroke Association (ASA) and the European Stroke Initiative (EUSI) guidelines. The lower left quadrant (bounded by the systolic and diastolic thresholds of each guideline) represents the overtreated blood pressures.
Barriers

• No clear time window for leaving B/P elevated
• Potential Exceptions
  – Hypertensive encephalopathy
  – Aortic dissection
  – Acute MI
  – Acute renal failure
  – Acute pulmonary edema
• B/P control in post-acute period is not addressed in guidelines
• Timing for resumption of prestroke regimen ignored
A delicate balance...

Low CPP
- Ischemia Occurs

High CPP
- Increased ICP
- Increased Intracranial Blood Volume
- Vasogenic Edema
Factors to Consider

• Type of Neurovascular Injury
• Level of hypertension
• Blood Pressure History
• Perceived condition of native autoregulatory system
• Kathy start

Applying this evidence and knowledge
Why Do We Treat HTN?

- Reduce formation of brain edema
- Reduce risk of hemorrhagic transformation
- Prevent further vascular damage

Aggressive lowering of BP can cause a reduction of perfusion in the ischemic zone, which may expand the region of infarction.
Basic Points

• Don’t treat isolated BP
  – Always recheck value prior to treating BP

• Too High
  – What is the cause?
  – Antihypertensives

• Too Low
  – What is the cause?
  – Fluid Boluses, vasopressors
Impaired Neurogenic CV Control

Autonomic Dysregulation

Reflex Response to Cerebral Ischemia

Mental Stress

Agitation

fear

confusion

Over-stimulation

Baroreflex Failure

Long standing chronic hypertension

Increased Sympathetic Drive

Increased ICP

Pain

Foley

IV

ET tube

EVD

HA

SCDs

crani

Impaired Hypertension

Elevated B/P

Mental Stress:
- Anger
- Agitation
- fear
- confision
- Over-stimulation

Autonomic Dysregulation:
- Reflex Response to Cerebral Ischemia

Long standing chronic hypertension:
- Agitation
- fear
- Over-stimulation

Elevated B/P:
- Mental Stress
- Autonomic Dysregulation
- Reflex Response to Cerebral Ischemia

Increased Sympathetic Drive:
- Mental Stress
- Autonomic Dysregulation
- Reflex Response to Cerebral Ischemia

Increased ICP:
- Pain
- Foley
- ET tube
- EVD
- HA
- SCDs
- crani
# Titratable Anti-Hypertensives

<table>
<thead>
<tr>
<th>DRUG</th>
<th>MECHANISM</th>
<th>DOSE</th>
<th>ONSET</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nicardipine</td>
<td>L-type CCB (dihydropyridine)</td>
<td>5-15 mg/h infusion</td>
<td>5-10 min</td>
</tr>
<tr>
<td>Labetalol</td>
<td>$\alpha_1 \beta_1 \beta_2$ antagonist</td>
<td>10-80 mg bolus q 10min; max 300 mg; 0.5-2 mg/min infusion</td>
<td>5-10 min</td>
</tr>
<tr>
<td>Esmolol</td>
<td>$\beta_1$ antagonist</td>
<td>500 $\mu$g/kg bolus; 50-300 $\mu$g/kg/min infusion</td>
<td>1-2 min</td>
</tr>
<tr>
<td>Enalaprilat</td>
<td>Ace Inhibitor</td>
<td>0.625 mg bolus, then 1.25-5 mg q 6h</td>
<td>15-30 min</td>
</tr>
</tbody>
</table>
Provider Practice

• Fear of letting B/P run high
  – End organ damage
  – ICH
  – More comfortable with low BP

• Patient “looks fine”
Challenges

• Case load – how to manage increased work load of frequent monitoring of BP with aggressive treatment regimen
  – Other causes of high or low BP
    • Time required to ferret out the specific cause and take action
• Level of care change for aggressive BP management
  – Patients being moved from one area to another
• Clarity of orders... what do they mean?
  – MAP vs SBP
• Provider doesn’t take action when notified of BP outside of range
Challenges

• Standards for ischemic different from hemorrhagic management
  – HOB elevation
  – Acceptable high/low ranges

• Evidence suggests age-related differences in outcomes at 3 months
  – SBP reductions >27 mmHG within first 8 hours
    • Poor outcomes likelihood: 6x greater age 70-76; 10x greater age 76-80; 15x greater >age 80
  – SBP reductions 10-27 mmHG better outcomes until age >76 years
Challenges

• HOB recommendations related to recanalization
  – Incomplete recanalization pts should have HOB 0°
    • Increases mean flow velocity
  – Complete recanalization pts should have HOB 30°
• Variability of BP management
  – Higher variability at 72 hours shown to increase risk of death or dependent outcomes
  – Co-medication of antihypertensive agents associated with unfavorable outcomes (ECASS-II)
Challenges

• Tough to link BP mismanagement with outcomes

• Tough to get acute care nurses to appreciate that the results of their actions may only be evident months after discharge
Case Review: Q3 2012

• TW is a 33-year-old female transferred in with right hemiplegia
  – Patient was evaluated via LionNet telestroke
• NIHSS was 17 on arrival to ED
• Patient restless
  – Intubated and sedated to complete CTA
Case Review

• CTA showed large perfusion defect in left MCA territory without evidence of penumbra

• Plan for hypothermia
  – Goal temp 35 C
  – MAP goals 90-120
Case Review
Case Review

• 1.8% NaCl started for cerebral edema
• MRI showed hemorrhagic conversion of left MCA infarct
• Patient went to OR for decompressive hemicraniectomy on Day #2
• Post-op, hypothermia continued through Day #3
Case Review
Case Review

- Discharged to Acute Rehab on Day #13
- NIHSS was 15 on discharge
- NIHSS at 90 day visit was 14
- Living at home with mother’s help
Post-op hemicraniectomy

Albumin 250mg x 2

Dobutamine

Norepinephrine

MRI
Management of BP – A Delicate Balance

Hypertension may promote hemorrhage

Treating hypertension may exacerbate ischemia
So what do we do now?

• Throw our hands up and stomp our feet
• Recognize that BP control is within our scope of practice
• Be/become familiar with medications
• Know your patient’s specific orders for BP management
• Commit to monitoring and responding to your patient’s BP
The ultimate endpoint

Limiting neurological damage within the ischemic penumbra by controlling abnormal physiology

... save the brain.
References