Intracerebral Hemorrhage

J. Claude Hemphill III, MD, MAS

Kenneth Rainin Chair in Neurocritical Care
Professor of Clinical Neurology and Neurological Surgery
University of California, San Francisco
Director, Neurocritical Care
San Francisco General Hospital

Disclosures
Research Support: NIH/NINDS
Stock (options): Ornim
• Refer to Syllabus
• Please read the 2010 AHA/ASA ICH Guidelines

Guidelines for the Management of Spontaneous Intracerebral Hemorrhage

A Guideline for Healthcare Professionals From the American Heart Association/American Stroke Association

The American Academy of Neurology affirms the value of this guideline as an educational tool for neurologists.

The American Association of Neurological Surgeons and the Congress of Neurological Surgeons have reviewed this document and affirm its educational content.

Lewis B. Morgenstern, MD, FAHA, FAAN, Chair;
J. Claude Hemphill III, MD, MAS, FAAN, Vice-Chair; Craig Anderson, MBBS, PhD, FRACP;
Kyra Becker, MD; Joseph P. Broderick, MD, FAHA; E. Sander Connolly, Jr, MD, FAHA;
Steven M. Greenberg, MD, PhD, FAHA, FAAN; James N. Huang, MD; R. Loch Macdonald, MD, PhD;
Steven R. Messé, MD, FAHA; Pamela H. Mitchell, RN, PhD, FAHA, FAAN;
Magdy Selim, MD, PhD, FAHA; Rafael J. Tamargo, MD; on behalf of the American Heart Association
Stroke Council and Council on Cardiovascular Nursing
Non-Traumatic Intracerebral Hemorrhage (ICH)

- Bleeding into the brain parenchyma
- Not due to trauma
- Distinct from subarachnoid hemorrhage (SAH) and isolated intraventricular hemorrhage (IVH)
Non-Traumatic Intracerebral Hemorrhage (ICH)

- No approved treatment of proven benefit in decreasing morbidity or mortality

- Guidelines exist for
  - Evaluation
  - ICU care
  - Surgery
  - Blood Pressure control
Intracerebral Hemorrhage

• Epidemiology
  – 10-15% of all strokes
  – Incidence 12-15 cases per 100,000 persons
  – About 50-70,000 cases per year in US

• Outcome
  – ~40% 30-day mortality
  – ~20% of ICH patients independent at 6 mo

• Other stats
  – 34% of years of potential life lost to stroke
  – Lifetime cost per case ~ $124,000
  – Total lifetime cost for annual US cases >$4B

Taylor et al. Stroke 26:1459-1466, 1996
ICH – 1° and 2°

**Primary**
- Hypertension (60-70%)
- Cerebral Amyloid Angiopathy (CAA)
- CADASIL
- Coagulopathy
- Sympathomimetic Drug Abuse (phenylproylalimine, cocaine, amphetamines)

**Secondary**
- Vascular Malformations
  - Arteriovenous Malformations
  - Cavernous Malformation
  - Intracranial Aneurysms
  - Dural fistulas
  - Mycotic Aneurysm
  - Moya Moya Disease
- Ischemic Stroke
  - Hemorrhagic Conversion of an Ischemic Stroke
  - Thrombolysis-associated
  - Dural/Cortical Sinus Thrombosis
- Trauma
- Tumor associated ICH (1° or metastatic)
- Vasculitis
Hypertensive ICH

- **Hypertension**
  - Rupture of small arterioles (< 100 micron)
  - Typical locations – penetrating arteries

Thalamus | Cerebellum | Pons | Basal Ganglia
Lobar ICH

- Cerebral Amyloid Angiopathy
  - Dementia
  - Recurrent hemorrhage
    - Assoc w/ APOE ε2 and ε4 alleles
- Vascular Malformation
- Cerebral Venous Sinus Thrombosis
- Hemorrhagic Ischemic Infarct
- Underlying Tumor
- Moya-Moya
- Sympathomimetic Drugs
- Coagulopathy (esp. w/ “mild” trauma)
ICH Outcome Predictors

- Strongest Predictors from Observational Clinical Research Studies
  - Glasgow Coma Scale score (esp. ≤ 8)
  - Hematoma Volume (esp. > 60 ml)
  - Presence of intraventricular hemorrhage
  - Age (esp. ≥ 80 years old)
  - Infratentorial origin (esp. brainstem)
Other ICH Outcome Predictors

- Hematoma expansion
- Warfarin use, coagulopathy
- Very high blood pressure at onset
- Volume of IVH
- Contrast extravasation on CT scan
- Early care limitations
  - DNR, support withdrawal leading to self-fulfilling prophecies of poor outcome
- Fever
- Hyperglycemia
- Seizures (nonconvulsive)
- Genotype ApoE ε4 allele
  - Association with lobar hemorrhage
  - Increased recurrence risk
Mechanisms of Brain Injury in ICH

- **Primary**
  - tissue dissection
  - displacement and brain herniation

- **Secondary**
  - perihematoma injury (ischemia?)
  - hematoma expansion
  - cellular toxicity of blood products
Perihematoma Ischemia in ICH

- Initial animals models suggested a zone of perihematoma “ischemia” based on findings of low CBF
  - Bullock (1988) - blood injection into primate caudate; CBF below ischemic threshold of 18 ml/100 gm/min
  - Mendelow (1993) - neuroprotection with nimodipine in rodent ICH model
Perihematoma Ischemia in ICH

- More recent animal models and human neuroimaging suggest low blood flow, but not true ischemia
  - Dog model - low CBF and CMRO$_2$; found no ischemic penumbra (Qureshi 1999)
  - Human neuroimaging
    - PET, SPECT, MRI
    - low CBF, CMRO$_2$, but OEF ok -> no true ischemia; rare DWI + areas
    - Modest BP lowering (15%) does not create ischemia on PET (Powers)

- Conclusion
  - Ongoing ischemia in the perihematoma region is neither common nor the major mechanism of perihematoma injury
  - Metabolic depression in this region is present
ICH Secondary Injury Mechanisms

- **Concept** – “neurohemoinflammation”

- **Perihematoma edema**
  - BBB breakdown
  - Thrombin mediated brain injury (Xi)
  - Erythrocytes, hgb, iron
    - Deferoxamine decreases delayed edema

- **Inflammation**
  - Complement activation
    - Complement depletion decreases TNF-α
  - Matrix metalloproteinases
  - Regulated by NF-κβ (feedback loop)

- **Hemoxygenase -1 induction in porcine model (Wagner)**

- **Apoptotic cell death (Qureshi)**
Hematoma Expansion in ICH

Initial CT

2’ 45” later

Images Courtesy of Jonathan Rosand, MD
Hematoma Expansion in ICH

- Previously suggested as rare, suggestive of underlying AVM, coagulopathy

- Studies of early serial CT show as common
  - 72% of patients have some hematoma expansion over initial 24 hrs
  - 38% have significant (>33%) expansion over 24 hrs, usually clinically significant
    » w/in 1 hr in 26% of cases

- Hematoma expansion worsens outcome

Davis et al. Neurology 2006
Brott et al. Stroke 1997
Hematoma Expansion in ICH

• **Characteristics**
  – occurs mostly w/in 24 hours (esp. 6 hours) from onset
  – usually associated with clinical deterioration

• **Mechanism?**
  – Associated with elevated BP?
    » interaction of elevated glucose (or Hgb A$_1$C) and systolic blood pressure on admission $\geq$ 200 mm Hg (Kazui 1998)
    » Lack of association b/t hemodynamic factors and ICH hematoma growth (Jauch, *Stroke* 2006)
  – Perihematoma coagulopathy/DIC?
Warfarin-related hemorrhage

- Warfarin doubles ICH mortality
- Warfarin increases risk of hematoma expansion (OR 6.22)
- Hematoma expansion occurs over more prolonged time course

Flibotte et al. *Neurology*, 2004
ICH - Diagnosis

- **CT scanning – gold standard**
  - CTA
  - CT “spot sign” (on CTP or post-contrast CT)

- **MRI**
  - performs equivalently, including w/in 6 hours of onset
  - Gradient echo sequences
  - Less feasible due to patient acuity

- **Angiography (conventional)**
  - Indications
    - Isolated IVH (bleeding source in > 50%; Flint NCC 2008)
    - Obvious vascular malformation
    - Blood in unusual location such as sylvian fissure
ICH Volume

\[ A \times B \times C \times 2 \]

Select CT slice with largest ICH
\( A = \) longest axis (cm)
\( B = \) longest axis perpendicular to \( A \) (cm)
\( C = \) # of slices \( \times \) slice thickness (cm)

Estimated volume of spheroid
Correlates well w/ planimetric CT analysis

CT “Spot Sign” – CTA +/- CTP and post-con

Contrast extravasation on CT angiography predicts hematoma expansion in intracerebral hemorrhage

J.N. Goldstein, MD, PhD; L.E. Fazen, BA; R. Snider, BA; K. Schwab, BA; S.M. Greenberg, MD, PhD; E.E. Smith, MD; M.H. Lev, MD; and J. Rosand, MD, MS

NEUROLOGY 2007;68:889–894
Spot sign – hematoma expansion & time

Multivariable Predictors of Hematoma Expansion

<table>
<thead>
<tr>
<th>Variable</th>
<th>OR (95% CI)</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Contrast extravasation</td>
<td>18 (2.1-162)</td>
<td>0.009</td>
</tr>
<tr>
<td>ICH volume (per 10cc)</td>
<td>0.8 (0.6-1.0)</td>
<td>0.1</td>
</tr>
<tr>
<td>Time to CTA &lt; 3 hours</td>
<td>3.4 (0.5-22)</td>
<td>0.2</td>
</tr>
<tr>
<td>Age (per 10 years)</td>
<td>1.2 (0.6-2.3)</td>
<td>0.6</td>
</tr>
<tr>
<td>SBP (per 10mmHg)</td>
<td>0.94 (0.8-1.1)</td>
<td>0.5</td>
</tr>
</tbody>
</table>

Of those who suffered expansion, < 30% presented within 3 hours.

Figure 2.

Of those who suffered expansion, < 30% presented within 3 hours.
ICH – Treatment

- Blood Pressure
- Coagulopathy
- Hemostatic Agents
- Antiplatelet Agents
- ICP
- Fever
- Glucose
- DVT prophylaxis
- Steroids
- Anticonvulsants
- Surgery
ICH – Randomized Medical Trials

• 4 small trials prior to 1999 AHA ICH Guidelines
  – Corticosteroids v. placebo
  – Hemodilution v. best medical management (BMM)
  – Glycerol v. placebo
  – No benefit; steroids increase infectious complications

• Mannitol for ICH – 2005; no benefit

• CHANT (phase II) – NXY-059 neuroprotective; no benefit

• Factor VIIa
  – Phase II positive; phase III neutral (only p III ICH medical study, yet)

• BP
  – INTERACT – vanguard phase completed; ? Small benefit
  – ATACH – BP lowering safe
BP in ICH - Expert Consensus

Guidelines for the Management of Spontaneous ICH – AHA/ASA, 2010 (no change from 2007)

Table 6. Suggested Recommended Guidelines for Treating Elevated BP in Spontaneous ICH

1. If SBP is $>200$ mm Hg or MAP is $>150$ mm Hg, then consider aggressive reduction of BP with continuous intravenous infusion, with frequent BP monitoring every 5 min.

2. If SBP is $>180$ mm Hg or MAP is $>130$ mm Hg and there is the possibility of elevated ICP, then consider monitoring ICP and reducing BP using intermittent or continuous intravenous medications while maintaining a cerebral perfusion pressure $\geq 60$ mm Hg.

3. If SBP is $>180$ mm Hg or MAP is $>130$ mm Hg and there is not evidence of elevated ICP, then consider a modest reduction of BP (eg, MAP of 110 mm Hg or target BP of 160/90 mm Hg) using intermittent or continuous intravenous medications to control BP and clinically reexamine the patient every 15 min.

Note that these recommendations are Class C. SBP indicates systolic blood pressure; MAP, mean arterial pressure.
BP Lowering Trials in ICH

- **INTERACT** – Australia/NZ/China, possibly US
  - Randomized open-label study
  - Entry criteria
    - ≥ 2 SBP measurements (≥150 to ≤ 220 mm Hg)
    - BP-lowering regimen < 6 h of onset
  - BP Rx goals – SBP < 180 v. SBP < 140
  - “Vanguard” phase completed – 404 patients, 95% in China
    (Lancet Neurol 2008)
    - Possible modest effect on hematoma expansion in adjusted analysis
    - No clinical efficacy signal
    - ~2500 patient pivotal trial ongoing

- **ATACH**
  - PI – Adnan Qureshi
  - “Dose-escalation” study of feasibility of achieving 3 successive BP goals for 24 hours after acute ICH using nicardipine
    - 170-200, 140-170, 110-140 mmHg
  - Safety evaluation by decline in GCS of 2 points or NIHSS of 4 points
  - Total – 60 patients, conclusions: safe (CCM 2010)
  - ATACH 2 ongoing
Reversal of Anticoagulation

- **Principle** – any ICH in patient on warfarin (with INR > 1.4) should be considered “life-threatening”
- **Goal** – normal INR ASAP

- **Guidelines from US, UK, Australasia recommend**
  - Prothrombin complex concentrate (PCC) (30-50 U/kg)
  - Vitamin K (1 mg IV or 10 mg SQ)
  - Fresh frozen plasma (10-15 ml/kg)
  - rFVIIa as option (80 μg/kg ?)

- **Less hematoma growth with PCC, with no difference with FFP if INR corrected w/in 2 hours (Huttner, Stroke 2006)**

- **FFP slower due to thawing, cross-matching, volume**
- **No randomized trials**

Effect of Antiplatelets

- Conflicting evidence on risk of poor outcome (presumably mediated through hematoma expansion)
- Likely increase risk a little
- No info on utility of reversal strategies (platelet transfusion, DDAVP)

The usefulness of platelet transfusions in ICH patients with a history of antiplatelet use is unclear and is considered investigational (Class IIb; Level of Evidence: B). (New recommendation)
Hemostatic Agents

- FAST - Phase III Trial of rFVIIa in acute ICH
- ICH patients
  - Without coagulopathy
  - CT scan w/in 3 hours
  - Rx w/in 1 hour of CT scan
  - 841 patients randomized; 821 patients dosed

- F/U of phase IIb trial that showed
  - Decreased hematoma expansion
  - Lower mortality and better functional outcome
  - Modest increase in thrombotic events

- Largest ICH medical trial ever conducted
- Protocol similar to phase IIb trial
- rFVIIa 80 µg/kg vs 20 µg/kg vs placebo

Mayer et al., NEJM 358: 2117-2137
# FAST: Primary Results

<table>
<thead>
<tr>
<th>Hematoma Growth at 24 hrs</th>
<th>Placebo</th>
<th>20 μg/kg</th>
<th>80 μg/kg</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean % change</td>
<td>26%</td>
<td>18%</td>
<td>11%</td>
<td>&lt;0.001 (80 μg/kg vs placebo)</td>
</tr>
<tr>
<td>Absolute difference</td>
<td>7.8 ± 18.7</td>
<td>4.7 ± 14.8</td>
<td>3.8 ± 15.3</td>
<td>0.009 (80 μg/kg vs placebo)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>Placebo</th>
<th>20 μg/kg</th>
<th>80 μg/kg</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Modified Rankin Score ≥ 5 at 90 days</td>
<td>24%</td>
<td>26%</td>
<td>29%</td>
<td>NS</td>
</tr>
<tr>
<td>Mortality</td>
<td>19%</td>
<td>18%</td>
<td>21%</td>
<td>NS</td>
</tr>
<tr>
<td>Arterial Thrombotic Events</td>
<td>4%</td>
<td>5%</td>
<td>8%</td>
<td>0.04</td>
</tr>
<tr>
<td>Cerebral Infarction</td>
<td>1%</td>
<td>1%</td>
<td>3%</td>
<td>0.14</td>
</tr>
</tbody>
</table>

- Reduces hematoma expansion
- No effect on clinical outcome
- Increase in arterial thrombotic events

Mayer et al., *NEJM* 358: 2117-2137
Other Aspects

- ICP, fever, glucose all mentioned in guidelines
- No randomized trials
- CPP $\geq 60$ mmHg per BP guidelines
  - Range of 50-70 mmHg depending on autoregulation status
- Fever is probably bad
  - Normothermia is goal with little data to guide us
- Glucose
  - Little ICH specific data
  - NICE-SUGAR backlash
- Glucocorticosteroids
  - No specific recommendation in the guidelines
Patients with ICH should have intermittent pneumatic compression for prevention of venous thromboembolism in addition to elastic stockings (Class I; Level of Evidence: B). (Unchanged from the previous guideline)

After documentation of cessation of bleeding, low-dose subcutaneous low-molecular-weight heparin or unfractionated heparin may be considered for prevention of venous thromboembolism in patients with lack of mobility after 1 to 4 days from onset (Class IIb; Level of Evidence: B). (Revised from the previous guideline)
Antiepileptic Drugs

- Overt clinical seizures in 4-8% of patients
  - Lobar location as risk

- Non-convulsive seizures found by cEEG in 28% of patients (Vespa, 2003)
  - Assoc w/ increased midline shift and poorer outcome
  - Chicken v. egg unclear

- Prophylaxis commonly done, esp for lobar ICH
  - 2 studies suggest worsened outcome (Messe, 2009; Naidech 2009)

Clinical seizures should be treated with antiepileptic drugs (Class I; Level of Evidence: A). (Revised from the previous guideline) Continuous EEG monitoring is probably indicated in ICH patients with depressed mental status out of proportion to the degree of brain injury (Class IIa; Level of Evidence: B). Patients with a change in mental status who are found to have electrographic seizures on EEG should be treated with antiepileptic drugs (Class I; Level of Evidence: C). Prophylactic anticonvulsant medication should not be used (Class III; Level of Evidence: B). (New recommendation)
# Surgery for ICH – Small Trials

## Representative Prior Randomized ICH Treatment Studies

<table>
<thead>
<tr>
<th>Author (year)</th>
<th>Treatment</th>
<th>Control</th>
<th>Enrollment Criteria</th>
<th>Time Window</th>
<th># of Subjects</th>
<th>Outcome Measure</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>Juvela (1989)</td>
<td>craniotomy &amp; evacuation</td>
<td>BMM</td>
<td>supratentorial ICH</td>
<td>arbitrary; 6-48 hrs</td>
<td>52</td>
<td>6-month dead or dependent</td>
<td>no difference</td>
</tr>
<tr>
<td>Auer (1989)</td>
<td>endoscopic aspiration</td>
<td>medical</td>
<td>supratentorial ICH vol &gt; 10 cc</td>
<td>48 hours</td>
<td>100</td>
<td>function at 6-months</td>
<td>overall negative; subgroups with aspiration benefit</td>
</tr>
<tr>
<td>Batjer (1990)</td>
<td>craniotomy &amp; evacuation</td>
<td>medical ICP</td>
<td>hypertensive putaminal ICH &gt; 3 cm diameter</td>
<td>none; considered as emergencies</td>
<td>21</td>
<td>6-month dead or dependent</td>
<td>no difference</td>
</tr>
<tr>
<td>Morgenstern (1998)</td>
<td>craniotomy &amp; evacuation</td>
<td>BMM</td>
<td>Supratentorial ICH &gt; 9 cc</td>
<td>12 hours</td>
<td>34</td>
<td>6-month mortality</td>
<td>no difference</td>
</tr>
<tr>
<td>Zuccarello (1999)</td>
<td>craniotomy or stereotactic evacuation</td>
<td>BMM (+/- ICP monitor)</td>
<td>GCS &gt; 4; supratentorial ICH vol &gt; 10 cc</td>
<td>24 hours</td>
<td>20</td>
<td>3-month GOS</td>
<td>non-significant trend for surgery</td>
</tr>
</tbody>
</table>
Surgical Trial for ICH (STICH)

- Completed in 2003
- Largest study of surgery in ICH (>1000 pts)
- Does a policy of “Early Surgery” improve outcome in patients with spontaneous supratentorial ICH compared with a policy of “Initial Conservative Treatment”?  
  - Randomization within 72 hours of ictus  
  - Surgery within 24 hours of randomization  
  - Selection based on “uncertainty principle”

Mendelow Lancet, 2005
### STICH - Results

<table>
<thead>
<tr>
<th>Mortality</th>
<th>Early Surgery</th>
<th>Initial Conservative tx</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alive</td>
<td>304 (64%)</td>
<td>316 (63%)</td>
</tr>
<tr>
<td>Dead</td>
<td>173 (36%)</td>
<td>189 (37%)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Primary Outcome</th>
<th>Early Surgery</th>
<th>Initial Conservative tx</th>
</tr>
</thead>
<tbody>
<tr>
<td>(“Prognosis based” functional outcome)</td>
<td>112 (26%)</td>
<td>118 (24%)</td>
</tr>
<tr>
<td>Favourable</td>
<td>346 (74%)</td>
<td>351 (76%)</td>
</tr>
</tbody>
</table>

\[ P = 0.71 \]

- No Difference
- 26% of patients randomised to Initial Conservative Treatment later had surgery
- Early surgery is not harmful
- There is no evidence favoring early surgery in supratentorial ICH
- Possible benefit in lobar ICH < 1 cm from cortical surface (STICH II ongoing)

Mendelow *Lancet*, 2005
Ongoing Surgical Studies

- Minimally invasive surgery +/- t-PA
- Intraventricular t-PA

- US Experience
  - Zuccarello, Carhuapoma and others
  - UCLA – Vespa

- MISTIE – Hanley (Johns Hopkins)
  - Multi-center NIH-sponsored clinical trial
  - Catheter-directed t-PA; endoscopic surgery

- CLEAR III
  - Intraventricular t-PA
  - Phase II showed benefit compared with expected outcome
ICH Surgery – the 2010 Guidelines

1. For most patients with ICH, the usefulness of surgery is uncertain \((Class\ IIb;\ Level\ of\ Evidence: \ C)\). (New recommendation) Specific exceptions to this recommendation follow.

2. Patients with cerebellar hemorrhage who are deteriorating neurologically or who have brainstem compression and/or hydrocephalus from ventricular obstruction should undergo surgical removal of the hemorrhage as soon as possible \((Class\ I;\ Level\ of\ Evidence: \ B)\). (Revised from the previous guideline) Initial treatment of these patients with ventricular drainage alone rather than surgical evacuation is not recommended \((Class\ III;\ Level\ of\ Evidence: \ C)\). (New recommendation)

3. For patients presenting with lobar clots >30 mL and within 1 cm of the surface, evacuation of supratentorial ICH by standard craniotomy might be considered \((Class\ IIb;\ Level\ of\ Evidence: \ B)\). (Revised from the previous guideline)

4. The effectiveness of minimally invasive clot evacuation utilizing either stereotactic or endoscopic aspiration with or without thrombolytic usage is uncertain and is considered investigational \((Class\ IIb;\ Level\ of\ Evidence: \ B)\). (New recommendation)

5. Although theoretically attractive, no clear evidence at present indicates that ultra-early removal of supratentorial ICH improves functional outcome or mortality rate. Very early craniotomy may be harmful due to increased risk of recurrent bleeding \((Class\ III;\ Level\ of\ Evidence: \ B)\). (Revised from the previous guideline)
Withdrawal of Support

Aggressive full care early after ICH onset and postponement of new DNR orders until at least the second full day of hospitalization is probably recommended (Class IIa; Level of Evidence: B). Patients with preexisting DNR orders are not included in this recommendation. Current methods of prognostication in individual patients early after ICH are likely biased by failure to account for the influence of withdrawal of support and early DNR orders. Patients who are given DNR status at any point should receive all other appropriate medical and surgical interventions unless otherwise explicitly indicated. (Revised from the previous guideline)
ICH Prevention

1. In situations where stratifying a patient’s risk of recurrent ICH may affect other management decisions, it is reasonable to consider the following risk factors for recurrence: lobar location of the initial ICH, older age, ongoing anticoagulation, presence of the apolipoprotein E ε2 or ε4 alleles, and greater number of microbleeds on MRI (Class IIa; Level of Evidence: B). (New recommendation)

2. After the acute ICH period, absent medical contraindications, BP should be well controlled, particularly for patients with ICH location typical of hypertensive vasculopathy (Class I; Level of Evidence: A). (New recommendation)

3. After the acute ICH period, a goal target of a normal BP of <140/90 (<130/80 if diabetes or chronic kidney disease) is reasonable (Class IIa; Level of Evidence: B). (New recommendation)
ICH Prevention – for example

• Warfarin-related ICH
  – Lobar or cerebral amyloid angiopathy
    » Do not restart warfarin
    » Antiplatelet agents may mildly increase ICH risk, but probably ok
Intracerebral Hemorrhage

• For the test,
  – consult the 2010 AHA/ASA Guidelines
  – Read the syllabus
Beef. It's what's for dinner!

Stroke. It's what's for later.