Management of Spontaneous Intracerebral Hemorrhage (ICH)

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Doctors finally figured out what's wrong with my brain; on the left side, there's nothing right; and on the right side, there's nothing left.
Objectives

1. Define ICH
2. Discuss the incidence/prevalence of acute, spontaneous ICH
3. Identify the most common causes of ICH
4. Describe the pathophysiology of the two most common causes: HTN and Cerebral Amyloid Angiopathy (CAA).
5. Analyze current recommendations of both medical and surgical management of acute, spontaneous ICH.

Definition

Spontaneous, Nontraumatic Intracerebral Hemorrhage...

...occurs when a diseased blood vessel within the brain bursts, allowing blood to leak inside the brain.
Incidence / Prevalence

“An estimated **6.6 million Americans >= 20 years of age have had a stroke** (extrapolated to 2012 by use of NHANES 2009-2012). Overall stroke prevalence during this period is an estimated 2.6% (NHANES, NHLBI).”

NHANES = National Health and Nutrition Examination Survey
NHLBI = National Heart, Lung, and Blood Institute


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Incidence / Prevalence

• “By 2030, projections show an additional **3.4 million people** aged > 18 years will have had a stroke. A 20.5% increase in prevalence from 2012.”

• “Highest increase (29%) is projected to be in Hispanic men.”

Incidence / Prevalence

- Average age affected lower than that of ischemic stroke victims.
- ICH makes up approx **12%** of all strokes
- Incidence 10-20/100,000
- ICH mortality: 40% in first 30 days


Highest Risk Factors

- Advanced age (>70)
- HTN
- Kidney Disease
- Heavy ETOH use
- Substance abuse (cocaine, amphetamines)
- Very low cholesterol

Causes of Spontaneous ICH

Causes

Primary:
- Hypertension (35%)
- Amyloid angiopathy (20%)
- Antiplatelet use and bleeding diathesis (5%)
- Drug/substance abuse

Secondary:
- Vascular Anomalies (5% overall but ~38% in ICH < 45 yrs of age):
  - Arterio-Venous malformation
  - Aneurysm
  - Cavernoma
  - AV fistula, etc.
- Anticoagulation (14%)
- Trauma
- Tumors
- Infection
- Hemorrhagic transformation of cerebral infarct
- Moyamoya disease
- Cerebral venous thrombosis

Cause remains undetermined in approximately 21% of spontaneous ICH.

Causes of Spontaneous ICH

HTN & Amyloid Angiopathy cause >50% of spontaneous ICH

Pathophysiology Overview
Hypertension induced ICH

- Lipohyalinosis: Small vessel disease. Lipid accumulation in vessel wall causing narrowed lumen.

- Fibrinoid Necrosis: Accumulation of amorphous, basic, proteinaceous material in the tissue matrix with a staining pattern reminiscent of fibrin. NOT FIBRIN!
  - Antigen-Ab complexes stick to vessel wall → attract inflammatory cells → activate compliment leading to cell wall damage and necrosis.

- Hyperplasia of the media in artery walls due to proliferation of reactive smooth muscle cells die → cells replaced by collagen fibers → brittle vessel wall

Chakrabarty, A., Shivane, A. 8(1), 2008

Hypertension induced ICH:

- Charcot-Bouchard aneurysms: lenticulostriates, thalamoperforators, paramedian branches of the basilar artery, superior cerebellar arteries, and anterior inferior cerebellar arteries.

Chakrabarty, A., Shivane, A. 8(1), 2008
Cerebral Amyloid Angiopathy (CAA):

- Deposition of insoluble amyloid-beta peptides in the walls of leptomeningeal and cortical arteries, arterioles, and capillaries.
- Hemorrhages are superficial, lobar and commonly breach the cortical surface with associated subarachnoid hemorrhage.
- Can be multiple and recurrent
- Most commonly in age > 70
- Associated with Alzheimer’s

Case Report:

This is a 57 y/o female with PMHx significant for HTN and Afib who presents approx 1 hour after acute onset of HA, right-sided weakness, dysarthria, and associated N/V.

- Initial Vitals:
  - BP 185/112, HR 115 bpm, RR 22, SpO2 99% on 2Lpm per NC
- Home medications: Lisinopril 20 mg QD, ASA 81 mg QD, and coumadin 5 mg Tu/Thurs and 2.5 mg on M/W/F/Sat
- Negative UDS
Case Report:

- On exam, the patient is very drowsy but arouses to noxious stimuli.
- Opens eyes to touch
- Severely dysarthric but follows commands with right sided facial droop and right sided motor deficits
- Slides RUE/RLE across a plane at best but not antigravity.
- Right sided sensory deficits and neglect
- Antigravity with good strength on left side

Case Report:

- EOMi, Pupils 3/=/+ 
- Unable to visualize gait due to level of consciousness
- Unable to manage oral secretions well with frequent coughing.
- Weak cough, gag intact
CT reveals a left thalamic, intracranial hemorrhage with intraventricular extension.

Radiographical Imaging for ICH:

- Initial – CT scan
  - Repeat imaging for assessment of expansion should be CT for accurate comparison...
    “Apples to apples...”

- Diagnostic evaluation
  - Vascular lesions: Angiography
  - Tumors / Infections: MRI w/wo contrast
  - Trauma: CT good for blood but MRI best for prognostication
Hemorrhage Assessment:

Location

- **Supratentorial:** Cerebrum, Lateral and third ventricle
- **Infratentorial:** Brain stem, Cerebellum, Fourth ventricle

Hemorrhage Assessment:

Size of hemorrhage

\[ A \times B \times C \times \frac{\text{slice thickness}}{2} \]

\[ \frac{ABC}{2} \]

\[ 2.4 \text{ cm} \times 2.8 \text{ cm} \times 5 \times 0.5 \times \frac{1}{2} \]

\[ = 8.4 \text{ ml ICH} \]
The ICH Score

Purpose: A clinical grading scale for ICH for accurate and rapid assessment of 30-day mortality.

ICH Score (Hemphill et al.)

<table>
<thead>
<tr>
<th>Feature</th>
<th>Finding</th>
<th>Points</th>
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<tbody>
<tr>
<td>GCS</td>
<td>3-4</td>
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<td>5-12</td>
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<td>13-15</td>
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<td>&lt;80</td>
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<td>Location</td>
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<td>ICH volume</td>
<td>&gt;=30cc</td>
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<tr>
<td></td>
<td>&lt;30cc</td>
<td>0</td>
</tr>
<tr>
<td>Intraventricular Blood</td>
<td>Yes</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>No</td>
<td>0</td>
</tr>
<tr>
<td>ICH SCORE</td>
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<td>0-6 points</td>
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ICH Score 30 Day Mortality

<table>
<thead>
<tr>
<th>ICH Score</th>
<th>30 Day Mortality</th>
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<tr>
<td>0</td>
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<tr>
<td>1</td>
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<td>2</td>
<td>26%</td>
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<td>3</td>
<td>72%</td>
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<td>4</td>
<td>97%</td>
</tr>
<tr>
<td>5</td>
<td>100%</td>
</tr>
<tr>
<td>6</td>
<td>100%</td>
</tr>
</tbody>
</table>

Hemphill, et al. 2001 Stroke

mY bRAIn iS eXPERIENCING tECHNICAL dIFFICULTIES

pLEASE StAnd By...
Quick Overview of
ACC AHA Guidelines
Classifications Scheme

LEVEL A
Multiple populations evaluated
data derived from multiple randomized clinical trials or meta-analyses
• Recommendation that procedure/therapy be performed or not performed

LEVEL B
Limited populations evaluated
data derived from a single randomized trial or nonrandomized studies
• Recommendation in favor of procedure/therapy
• Recommendation against procedure/therapy

LEVEL C
Very limited populations evaluated
Only consensus opinion of experts, case studies, or standard of care
• Suggested process for forming recommendations

CLASS I
Benefit >> Risk
Arbitrary criteria to which benefit must be attributed
IT IS REASONABLE to perform procedure/therapy

CLASS IIa
Benefit >> Risk
Arbitrary criteria to which benefit must be attributed
IT IS REASONABLE to perform procedure/therapy

CLASS IIb
Benefit >> Risk
Arbitrary criteria to which benefit must be attributed
IT IS REASONABLE to perform procedure/therapy

CLASS III
Benefit << Risk
Arbitrary criteria to which benefit must be attributed
IT IS NOT REASONABLE to perform procedure/therapy

Best Level
Multiple RCTs or meta-analysis

Limited populations evaluated
Single RCT or nonrandomized studies

Lowest level
Limited evidence
Expert opinion
Case Report: Assessment & Plan

This is a 57 y/o female with PMHx significant for HTN and Afib (on coumadin), who presents approx 1 hour after acute onset of HA, right-sided weakness, dysarthria, and associated N/V. Found per CT to have a hypertensive, left thalamic hemorrhage with intraventricular extension.

Supratentorial hematoma volume of 8 ml
ICH score of 2, predicting a 26% 30-day mortality

Medical Management
Medical Management:

1st and utmost important...

Correction of Coagulopathy!!!
Coagulation Cascade

Correction of Coagulopathy

**Heparin / LMWH**

Mechanism: Binds to the enzyme inhibitor antithrombin III (AT) → inactivates thrombin, factor Xa and other proteases

Treatment:
- Ascertain last dose
- Reversal: Protamine sulfate
- May consider rFVIIa in LMWH if protamine contraindicated or continued hemorrhage despite protamine.

Frontera, et al. Neurocritical Care, 24, 2016
Warfarin (Coumadin)

Mechanism: “Vitamin K Antagonist” but rather antagonizes vitamin K1 recycling, depleting active vitamin K1.

Depletes factors II, VII, IX, and X (“27, 910”)

Reversal: Vitamin K → (INR > 2) FFP or Prothrombin Complex Concentrate (PCC)

INR correction with FFP + Vit K ~ 4-5 times slower than with PCC.


Frontera, et al. Neurocritical Care, 24, 2016
 Correction of Coagulopathy

**Direct Thrombin Inhibitors (Dabigatran)**

- **Bivalent**: Hirudin, Bivalirudin, Desirudin, Lepirudin
- **Univalent**: Argatroban

**Mechanism:** Directly inhibits the enzyme thrombin (factor II)

**Treatment:**
- Ascertain time of last dose
- If < 2 hrs since ingestion, administer activated charcoal
- Reversal: idarucizumab (Praxbind) – Adheres to thrombin binding sites
- Administer PCC
- Approx 50% dialyzable

Frontera, et al. Neurocritical Care, 24, 2016
Coagulation Cascade

Direct Xa Inhibitors (Rivaroxaban, Apixaban, Edoxaban)

Mechanism: Inhibits factor Xa thereby interrupting the intrinsic and extrinsic pathway → inhibits thrombin formation

Treatment:
- Ascertain time of last dose
- If < 2 hrs, administer 50 g activated charcoal
- Prothrombin Complex Concentrate (PCC)
- Reversal: Andexanet – alfa may be a future option.

Correction of Coagulopathy

Frontera, et al. Neurocritical Care, 24, 2016
Correction of Coagulopathy

**Anticoagulant Reversal strategy Mechanism Approval status**

<table>
<thead>
<tr>
<th>Anticoagulant</th>
<th>Reversal strategy</th>
<th>Mechanism</th>
<th>Approval status</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heparin, LMWH</td>
<td>protamine</td>
<td>Binding and inactivation of heparins</td>
<td>FDA-approved</td>
</tr>
<tr>
<td>Warfarin</td>
<td>Vitamin K</td>
<td>Overwhelm warfarin inhibition of VKOR-C1</td>
<td>FDA-approved</td>
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<tr>
<td></td>
<td>KCentra® (4-factor PCC)</td>
<td>Factor replacement</td>
<td>FDA-approved</td>
</tr>
<tr>
<td>Dabigatran (Pradaxa®)</td>
<td>Idarucizumab (Praxbind®)</td>
<td>Monoclonal antibody that binds dabigatran</td>
<td>FDA- approved</td>
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<tr>
<td>Rivaroxaban (Xarelto®)</td>
<td>KCentra® (4-factor PCC)</td>
<td>Factor replacement</td>
<td>Not-FDA approved</td>
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<tr>
<td>Apixaban (Eliquis®)</td>
<td>Andexanet-alfa</td>
<td>Decoy Xa molecule</td>
<td>Under FDA review</td>
</tr>
<tr>
<td>Edoxaban (Savaysa®)</td>
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</tbody>
</table>

ASA/Plavix

- If no NSG intervention, **NO** routine platelet transfusion recommended
- If undergoing NSG intervention, **SHOULD** transfuse platelets
- Platelet function testing if able
- Single dose desmopressin (DDAVP)

NSAIDs

- No platelet transfusion

GP IIb/IIIa inhibitors

- No platelet transfusion
- No good evidence for rFVIIa or Fibrinogen, but may be considered

Frontera, et al. Neurocritical Care, 24, 2016

**Lancet 2016; 378: 2605-13**
Medical Management:

Next most important...
Blood Pressure Control

Blood Pressure Goals

What are we trying to achieve?

...Maximal cerebral perfusion of both global parenchyma AND local, perihematomal tissue being compressed...
What are we MOST afraid of? MORE BLOOD!!!!

So what SBP is best to obtain maximal perfusion with the least risk of worsening hemorrhage?

120???, 140???, 180???, 160???
What do the studies show?

- Vemmos et al evaluated the relationship between SBP and DBP on admission and early or late mortality in acute stroke patients
- Prospective study of hospitalized, first-ever stroke patients over 8 yrs
- N = 1,121
- Admitted within 24 hrs of stroke onset
- Followed for 12 months
- Main outcome measures were mortality at 1 month and 12 months after stroke.


Results

At both 1 month and 12 months, less mortality in SBP 121-140 mmHg.

Table 6. Relationship between admission systolic blood pressure (SBP) values and causes of death at 1 and 12 months after acute stroke

<table>
<thead>
<tr>
<th>Causes of death</th>
<th>SBP ≤130 mmHg (n = 247)</th>
<th>SBP &gt;130 mmHg (n = 883)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 month</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Neurological damage</td>
<td>13 (27.7)</td>
<td>94 (50.3)</td>
<td>0.005</td>
</tr>
<tr>
<td>Cardiovascular disease</td>
<td>14 (29.7)</td>
<td>21 (11.8)</td>
<td>0.004</td>
</tr>
<tr>
<td>Infections</td>
<td>15 (27.7)</td>
<td>52 (28.0)</td>
<td>0.968</td>
</tr>
<tr>
<td>Other or unknown</td>
<td>7 (14.9)</td>
<td>17 (9.1)</td>
<td>0.246</td>
</tr>
<tr>
<td>12 months</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Neurological damage</td>
<td>18 (18.4)</td>
<td>102 (12.7)</td>
<td>0.001</td>
</tr>
<tr>
<td>Cardiovascular disease</td>
<td>22 (11.4)</td>
<td>91 (20.3)</td>
<td>0.041</td>
</tr>
<tr>
<td>Infections</td>
<td>22 (23.6)</td>
<td>75 (28.5)</td>
<td>0.565</td>
</tr>
<tr>
<td>Other or unknown</td>
<td>23 (26.7)</td>
<td>42 (34.9)</td>
<td>0.011</td>
</tr>
</tbody>
</table>

Values are given as n (%).

Both high and low admission BP-values are associated with poor outcome.

Is early, intensive BP-lowering safe?

**SAFE!!!**

N = 2,794
Randomized to SBP < 140 mmHg or < 180 mmHg

Intensive lowering of blood pressure **did not** result in a significant reduction in the rate of death or severe disability.

Significantly better functional outcomes, as well as, better physical and psychological well-being among patients who received intensive treatment.

While poor eGFR predicts poor outcome, intensive BP reduction does not affect this.

Anderson, et al. NEJM, 368(25), 2013

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Is early, intensive BP-lowering efficacious?

**Potentially Efficacious...**

**INTERACT2 Trial**

- Did not significantly reduce the rate of death or disability.
- Trend toward improved functional outcomes
- **No significant difference** in size of hematoma between two groups.
Primary Outcome: Death or disability at 3 months after randomization.

N = 1000 (500 to each group)
Standard group (SBP 140 – 179 mmHg)
Intensive group (SBP 110 – 139 mmHg)

Eligibility:
ICH volume < 60 ml
GCS > 5

Conclusions:
Target SBP 110 – 139 mmHg did not result in lower rate of death or disability compared to the standard treatment group 140 – 179 mmHg.
Stopped early due to futility and adverse events (ie. renal injury)

So now we know that rapid, intensive, BP-lowering to 140 vs 180 is safe and potentially efficacious...

However, closer to 120 has worsened adverse events...

...but does a SBP of < 140 mmHg provide enough cerebral perfusion?
Hypothesis: CT perfusion will not demonstrate evidence of perihematomal ischemia following acute BP reduction.

Randomized, blinded, end-point trial

Acutely hypertensive ICH patients are randomized to SBP < 150 or < 180 mmHg.

CT perfusion obtained 2 hr post randomization and cerebral blood flow (CBF) measured

Secondary outcomes:
- Difference in BP at 1 and 2 hr post randomization and hematoma expansion at 24 hrs.
The Intracerebral Haemorrhage Acutely Decreasing Arterial Pressure Trial: ICH ADAPT

“A randomized clinical trial using CT perfusion in primarily small and medium ICH found no clinically significant reduction in cerebral blood flow within the perihematomal region related to early intensive BP lowering to an SBP target of <140 mm Hg within several hours of ICH.”

2013, AHA, Inc. Stroke.

AHA / ASA Recommendation:

For ICH patients presenting with SBP between 150 and 220 mm Hg and without contraindication to acute BP treatment, acute lowering of SBP to 140 mm Hg is safe (Class I; Level of Evidence A) and can be effective for improving functional outcome (Class IIa; Level of Evidence B) (Revised from the previous guideline)

For ICH patients presenting with SBP >220 mm Hg, it may be reasonable to consider aggressive reduction of BP with a continuous intravenous infusion and frequent BP monitoring. (Class IIb; Level of Evidence C). (New recommendation)
So how do we achieve this blood pressure goal?

Antihypertensive Pharmacotherapy:

**Nicardipine hydrochloride (Cardene)**
- Dihydropyridine class of CCB
- More selective for cerebral and coronary blood vessels
- Does not intrinsically decrease myocardial contractility

Avoid nitroprusside (Nipride) and nitroglycerin infusions due to cerebral vasodilatory effects
Hydralazine (Apresoline)

- Direct-acting smooth muscle relaxant
- Decreases PVR thereby lowering afterload
- May increase HR and CO due to reflex sympathetic stimulation (baroreceptor reflex)
- May increase plasma renin concentration → fluid retention
- Can be given in combo with B-Blocker and a diuretic

Antihypertensive Pharmacotherapy:

Labetalol hydrochloride (Normodyne)

- Mixed alpha/beta adrenergic antagonist
- Short-term → decreases SVR without effect on SV, HR, or CO
- Long-term → decreases HR while maintaining CO with an increase in SV
- SE = postural hypotension

Better BP control than HR control
Glucose Management:

- High blood glucose on admission predicts increased risk of mortality and poor outcome
- Aggressive glucose management leads to increased incidence of both systemic and cerebral hypoglycemic events = increased risk of mortality

AHA / ASA Recommendation:
Closely monitor glucose but avoid both hyperglycemia and hypoglycemia. Goal 140-180 mgdL.

(Class I; Level of Evidence C).

Temperature Management:

- Fevers worsen outcome
- Increases cerebral metabolic demand
- Common in ICH, especially those with IVH, due to huge inflammatory response
- Hypothermia thought to reduce perihematomal edema but no evidence.
- Goal should be normothermia

AHA / ASA Recommendation:
Treatment of fever after ICH is reasonable.

(Class IIb; Level of Evidence C). (New recommendation)
Temperature Management:

**Pharmacological Management:**
- Acetaminophen and/or NSAIDs
- Alternate, Dual antipyretics best!
- Watch for renal and/or hepatic toxicity
- With NSAIDs, watch for gastric irritation/ulcers and/or bleeding.


Temperature Management:

**Non-Pharmacological Interventions:**
- Cold fluid IV infusions
- Air circulating blankets
- Water-circulating blankets and pads
- Intravascular cooling catheters

**Shiver Control:**
- Meperidine, buspirone, IV MgSO4
- Surface warming

Lopez, G.A. Curr Treat Options Neurol, 18:12, 2016
Seizures and Seizure Prophylaxis:

- Frequency of clinical sz within 1 week after ICH = up to 16%
- Despite prophylaxis, 28-31% still have electrographic seizures on continuous EEG
- Cortical involvement single highest predictor

Passero, et al (2002) performed study followed 761 patients with spontaneous, nontraumatic ICH in order to characterize seizures after ICH, evaluate risk for relapse, predisposing factors, prognostic significance, and to assess the utility of AED therapy.

“...short-term mortality was not affected, and the risk of epilepsy was lower than previously thought”

Would a short duration of AEDs soon after ICH reduce the risk of early seizures?

Seizures and Seizure Prophylaxis:

Are post intracerebral hemorrhage seizures prevented by anti-epileptic treatment?

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§ Statistical Unit, Edith Wolfson Medical Center, Holon, Sackler Faculty of Medicine, Tel Aviv University, Tel Aviv, Israel

Purpose: Assess the occurrence of seizures and neurologic outcome in SICH patients randomized to either valproic acid (VPA) or placebo x 1 month after SICH.

N = 7

Small study...

No benefit on mortality but could provide improved neurological outcomes.

VPA treated pts exhibited improved neuroprotective effects

Figure 1. The NIHSS subgroups before and after treatment comparing the VPA group to the placebo group.


Seizures and Seizure Prophylaxis:

“"No statistical difference found for seizure prevention in those prescribed prophylactic anticonvulsants"”

Seizures and Seizure Prophylaxis:

**AHA / ASA Recommendation:**

Clinical or subclinical seizures should be treated with anti-seizure drugs.  
(Class I; Level of Evidence A)

Prophylactic anti-seizure medication is not recommended  
(Class III; Level of Evidence B)

Consider continuous EEG when mental status disproportionate to degree of brain injury.  
(Class IIa; Level of Evidence C)

Great! Wait... so you want to start prophylaxis anyway?!?!
So which drug is best?

Seizures and Seizure Prophylaxis:

Levetiracetam is more effective than phenytoin for seizure prophylaxis without suppression of cognitive abilities in patients with ICH.
ICP Monitoring and Treatment

Ventricular drainage for hydrocephalus is reasonable, especially in the setting of decreased LOC.

(Class IIa; Level of Evidence B). (Revised from the previous guideline)

Those with extensive intraventricular hemorrhage with concern for developing hydrocephalus.

(Class IIb; Level of Evidence C)

GCS < 8 and/or evidence of transtentorial herniation.

(Class IIb; Level of Evidence C)

Corticosteroids should not be administered for treatment of elevated ICP in ICH.

(Class III; Level of Evidence B). (New recommendation)
**Intraventricular Hemorrhage (IVH)**

- IVH occurs in ≈45% of patients with spontaneous ICH
- Can lead to a clot in the CSF conduits blocking its flow and leading to **obstructive hydrocephalus** which may quickly result in increased ICP and death.
- Inflammatory response damages the arachnoid granulations, inhibiting the regular reabsorption of CSF and resulting in permanent **non-obstructive hydrocephalus**.

**Intra-thecal tPA Administration**

**A multicenter, randomized, double-blinded, placebo-controlled phase III study of Clot Lysis Evaluation of Accelerated Resolution of Intraventricular Hemorrhage (CLEAR III)**

- In IVH pts, rt-PA has been shown to reduce morbidity and mortality by accelerating blood clearance and clot lysis.
- **CLEAR-IVH Trial**: Patients treated with rtPA had significantly lower intracranial pressures, fewer VC obstructions that required replacement, and non-significantly shorter duration of VC requirement.
  1. Slightly increased risk of symptomatic bleeding
  2. Less incidence of permanent CSF diversion
  3. **No statistical difference in mRS or mortality**
Conclusion

AHA / ASA Recommendations:
Although intraventricular administration of rtPA in IVH appears to have a fairly low complication rate, the efficacy and safety of this treatment are uncertain.

The efficacy of endoscopic treatment of IVH is uncertain.

(Class IIb; Level of Evidence B). (New recommendation)

Supratentorial Intracerebral Hemorrhage

- Above the tentorium
- Majority of intracranial hemorrhages
- High morbidity and mortality
Surgery vs Conservative Treatment

Early surgery versus initial conservative treatment in patients with spontaneous supratentorial intracerebral haematomas in the International Surgical Trial in Intracerebral Haemorrhage (STICH): a randomised trial

STICH Trial
Question: Does early surgery reduce mortality and improve neurological outcome compared with conservative management?

Answer: 26% vs 24% = No overall statistically significant difference in mortality or functional outcome.

The Lancet, Volume 365, Issue 9457, 387 - 397

Surgery vs Conservative Treatment

Early surgery versus initial conservative treatment in patients with spontaneous supratentorial lobar intracerebral haematomas (STICH II): a randomised trial

STICH II Trial
Question: Is early surgery would be beneficial for conscious patients with superficial lobar hemorrhage within 1 cm of the cortical surface?

Answer: 41% (early surgery group) vs 38% (medical arm) had favorable outcomes; this difference was not statistically significant.

Non-significant survival advantage
Conclusion

Study Findings:
Patients with spontaneous supratentorial intracerebral hemorrhage in neurosurgical units show no overall benefit from early surgery when compared with initial conservative treatment.

However, the STICH II results confirm:
1. That early surgery does not increase the rate of death or disability at 6 months
2. Early surgery might have a small but clinically relevant survival advantage for patients with spontaneous superficial intracerebral haemorrhage without intraventricular hemorrhage.

Supratentorial hematoma evacuation in deteriorating patients might be considered as a life-saving measure.

(Class IIIb; Level of Evidence C). (New recommendation)

Minimally Invasive Clot Evacuation
Conclusion

Preliminary Findings:
Thus far study has demonstrated a **significant reduction in perihematomal edema** in the hematoma evacuation group with a **trend toward improved outcomes**.

AHA / ASA Recommendations:
The effectiveness of minimally invasive clot evacuation with stereotactic or endoscopic aspiration with or without thrombolytic usage is **uncertain**.

*(Class IIb; Level of Evidence B)*
Infratentorial Intracerebral Hemorrhage

- Below the tentorium
- Narrow confines of the posterior fossa can quickly lead to deterioration in cerebellar hemorrhage caused by obstructive hydrocephalus or local mass effect on the brainstem.

Conclusion

AHA / ASA Recommendations:
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)

Initial treatment of these patients with ventricular drainage rather than surgical evacuation is **not recommended.**

(Class III; Level of Evidence C)
Summary
Top 5 things to remember

1. Spontaneous, atraumatic ICH has significant impact on not only mortality, but also functional impairment.
2. Correction of coagulopathy and BP management (SBP < 140) are of utmost importance in the acute management phase.
3. Direct catheter injected tPA has shown promise in both IVH and IPH resolution.
4. Spontaneous, supratentorial, intracerebral hemorrhage show no overall benefit from early surgery when compared with initial conservative treatment.
5. Spontaneous, infratentorial hemorrhages should undergo emergent evacuation as soon as possible.