### Overview

- **10%** of strokes is caused by ICH.
- **Main Causes:**
  - Less than 40 years old: vascular malformations and illicit drug use.
  - 40 to 69 years old: hypertension predominates.
  - Elderly: cause is often unknown, possible amyloid angiopathy.
  - Other causes:
    - Use of anticoagulants, thrombolytics, and probably aspirin.
    - Carotid endarterectomy (especially in the setting of recent ipsilateral infarct or severe postoperative hypertension)
    - Head trauma.

### Prognosis

- **Mortality:** varies between 20% and 56%.
- Depends on several variables:
  - **Age**
  - **Level of consciousness**
  - **Hematoma volume**
  - **Ventricular extension of the hemorrhage**
- 30 days after onset: 12% normal or with minor handicap.
Management

Hypertension:

- It is recommended that mean arterial pressure (MAP) > 125-135 mm Hg be treated emergently.
- The ideal antihypertensive agent would be short acting and easily titrated and would not cause cerebral vasodilation with its associated increased ICP.
- An alpha- and beta-adrenergic blocker Labetalol:
  - 10 mg iv bolus
  - followed by 10-20 mg after 10 minutes as needed, to a maximum of 160 mg.
- If the BP continues to be severely elevated, iv sodium nitroprusside:
  - 2 ug/kg/min and then titrated to maintain a MAP of 100-125 mm Hg or systolic BP of 140-160 mm Hg.
- Sublingual nifedipine (10 mg) is potentially useful for moderate hypertension, but has the disadvantage of causing, at times, an excessive drop in BP.
- Other agents that can be used:
  - subcutaneous clonidine (0.075 mg)
  - trimethaphan
  - angiotensin-converting enzyme inhibitors.

Early endotracheal intubation: If Glasgow Coma Scale (GCS) score < 8,

- Premedicate with short-acting agent to block the increases in ICP that can result from tracheal stimulation:
  - Thiopental (1-5 mg/kg) or
  - Lidocaine (1 mg/kg)

Treatment of Increased Intracranial Pressure:

- Maintain the cerebral perfusion pressure (CPP) of 70-100 mm Hg
- This is dependent on ICP and MAP (CPP = MAP - ICP).
- Hyperventilation (to PaCO2 of 28-35 mm Hg)
- Advantage: rapid effect
- Disadvantage: brief effect lasting only a few hours.
- Osmotic diuretics
- Mannitol creates an osmotic gradient that moves water out of the brain and a
reduction of CSF production. In addition, mannitol decreases blood viscosity, resulting in improved cerebral blood flow.
- 0.75-1 g/kg, followed by 0.25-0.5 g/kg every 3-5 hours
- attempting to maintain serum around 320 mosm/L and euvolemia.
- Effect can be enhanced by the concomitant use of loop diuretics such as (furosemide).

Intravenous barbiturate has limited role, because of systemic hypotension, and inability to follow the neurologic examination.

Pentobarbital at 1-5 mg/kg, it consistently reduces brain metabolism, cerebral blood volume, and ICP.
- This tends to be a short-lasting effect, approximately 6 hours.
- Most useful as an emergency measure while the patient is in the process of receiving a more definitive therapy.

Ventriculostomy for CSF drainage
- Effective in reducing ICP in the presence of hydrocephalus.
- Disadvantage: infection, invasive procedure

Corticosteroids are controversial in the management of increased ICP owing to ICH.
- Has established effectiveness in reducing the vasogenic edema that surrounds cerebral metastases
- Ability to reduce ICP and improve outcome in ICH patients has not been documented.
- In a randomized trial in which dexamethasone was used over 9 days, cases and controls had similar mortality.

Urine toxic screen: especially in the young and the normotensive. Substances implicated in the causation of the ICH, mostly sympathomimetic agents used as recreational drugs, decongestants, or appetite suppressants, including cocaine hydrochloride, crack cocaine, amphetamines, Talwin-pyribenzamine (T's and blues), methylphenidate, phencyclidine & phenylpropanolamine.

Patients who develop ICH after cocaine use have been found to have a high frequency of aneurysms or arteriovenous malformations as the mechanism of the hemorrhage. Further imaging studies should be considered.
Correction of Coagulopathy

Patients on heparin therapy:

- Check aPTT
- Use
  - Protamine sulfate should be given slow iv over 10-minute in doses not to exceed 50 mg, because of the potential risk of severe systemic hypotension.
  - Patients who received warfarin tend to develop large hematomas, often evolve over periods of many hours or even days.

- Check
- Use intravenous vitamin K1 5-25 mg.
  - For immediate normalization of INR: fresh-frozen plasma (10-20 mL/kg) or prothrombin complex concentrate.
  - Patients who received thrombolytic agents generally develop large hematomas that are associated with high mortality, in the 40-50% range.

  - Risk factors for ICH in this setting are old age, low body weight, hypertension, recent ischemic stroke or head injury, previous ICH, recent major surgery, and probably, concomitant use of anticoagulants.
  - There are no useful data regarding the preferred treatment of ICH caused by thrombolytic therapy.
  - Cryoprecipitate or fresh-frozen plasma (4-6 U), and 1 U of single-donor platelets are recommended.
  - The use of epsilon-aminocaproic acid (5 g over 60 minutes loading dose, followed by 1 g/h until bleeding is controlled) is controversial, increased risk of deep vein thrombosis and pulmonary embolism.

Precautions in the CT diagnosis of ICH

- Severe anemia (hematocrit < 20): a fresh hematoma may appear isodense to the brain.
- Coagulopathy: presence of a blood-fluid level in the hematoma.
- Primary brain tumor: may cause bleeding into the corpus callosum.
- Bleeding into an underlying mass lesion: cause a disproportionate amount of low-density edema surrounding an acute hemorrhage.
Surgical vs Nonsurgical Treatment

No data from randomized clinical trials
Hematomas of putaminal, thalamic, and lobar location.

Data from many nonrandomized clinical studies with multiple types of biases seems to indicate:

- Small supratentorial hematomas (volume < 20 cm²) do well without surgical treatment.
- Large hematomas (volume > 60 cm²) do poorly regardless of whether they are treated surgically or nonsurgically.

Cerebellar Hemorrhage:

- Small hemorrhage < 3 cm in diameter in a cerebellar hemisphere and that is without hydrocephalus by CT scan, tend to do well without requiring surgical treatment.
- Hemorrhages > 3 cm in diameter and are associated with hydrocephalus and effacement of the quadrigeminal cistern on CT scan have a notorious tendency to progress to brainstem compression and poor outcome unless they are treated surgically.

In addition, the presence of clinical signs of lateral pontine tegmental involvement (ipsilateral horizontal gaze palsy, facial palsy, facial hypesthesia) are an indication for surgical drainage, since further progression of symptoms is likely to result in respiratory arrest and death.

If hydrocephalus is present, should include immediate ventriculostomy while preparations are being made for posterior fossa craniotomy for drainage of the hematoma.

Lobar Hemorrhage

Intermediate size: volume of 20-60 cm² are often considered candidates for surgical treatment, especially in instances when progressive deterioration in the level of consciousness occurs in association with CT evidence of enlargement of the hematoma.

This approach is favored by the superficial location of the hematoma, which makes surgical trauma less likely.

An additional reason to consider surgical treatment is the documentation of a vascular lesion with potential for rebleeding, such as an arteriovenous malformation or cavernous angioma.

Thalamic and Caudate Hemorrhage

Deeply located paraventricular hemorrhages often cause ventricular hemorrhage and hydrocephalus.

Emergent ventriculostomy can result in a dramatic reversal of obtundation and neuroneurologic deficits.