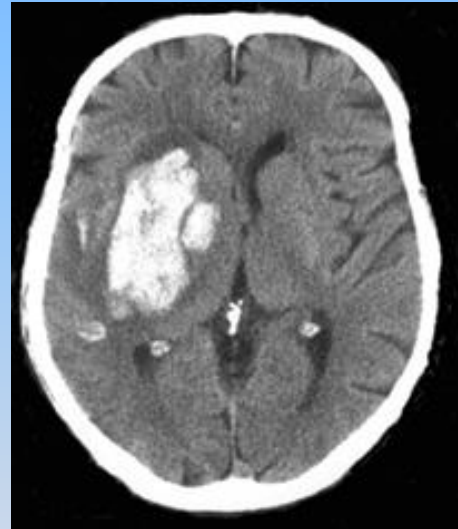




# Acute Intracerebral Hemorrhage



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*Quinte Physician Stroke Champion*

# Disclosure

- **Relationships with commercial interests:** I have no financial relationship with any commercial interests including any manufacturers of any product or class of products discussed in this presentation.
- **Potential for conflict of interest:** None
- I have received financial support from the Canadian Institutes of Health Research and the PSI Foundation for research in an area unrelated to stroke, and have received support from the Heart and Stroke Foundation for activities unrelated to this talk.

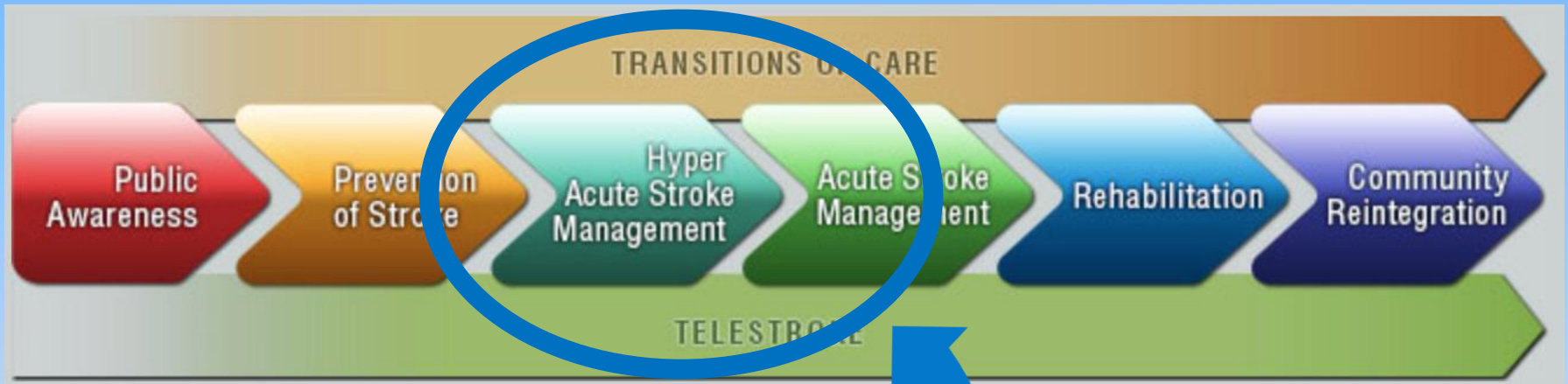
# Outline

- Definition
- Epidemiology & Pathophysiology
- Presentation
- Treatment



# Definitions

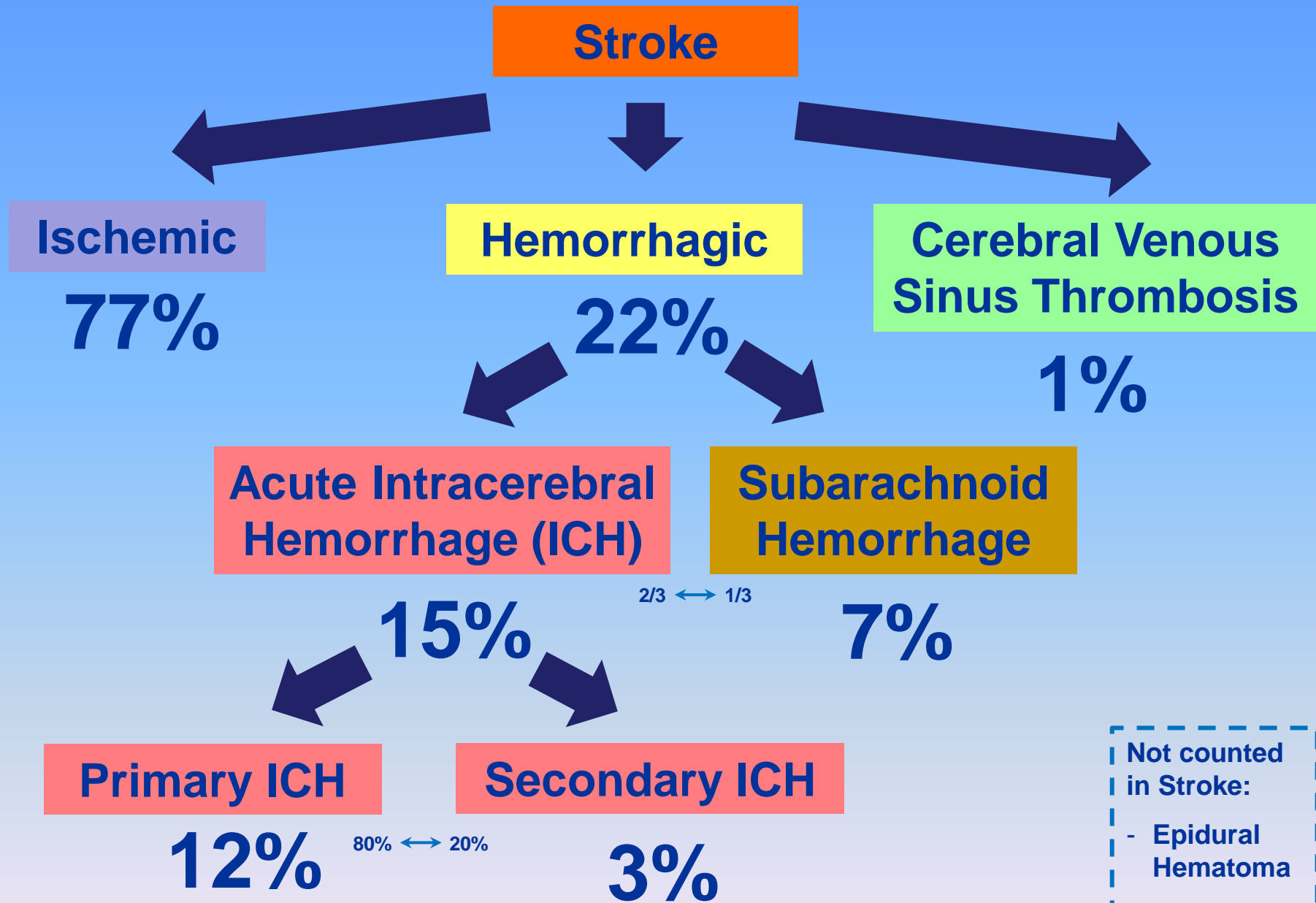
# Hyper Acute



# Hyper Acute

- assessment, stabilization, and treatment in the first hours
- all prehospital and initial emergency care
- assessment, diagnosis, early neurovascular imaging, emergency neurosurgical procedures





Not counted in Stroke:

- Epidural Hematoma
- Subdural Hematoma

# Definition

## **Acute Intracerebral Hemorrhage**

**Acute  
extravasation of  
blood into the brain  
parenchyma**





# Definition



## Primary Acute ICH

Unrelated to an underlying congenital or acquired brain lesions or abnormality – spontaneous

- Hypertension
- Cerebral Amyloid Angiopathy
- Anticoagulants & Thrombolytics
- Drug Use
- Bleeding Diathesis

## Secondary Acute ICH

Related to a pre-existing intracranial abnormality

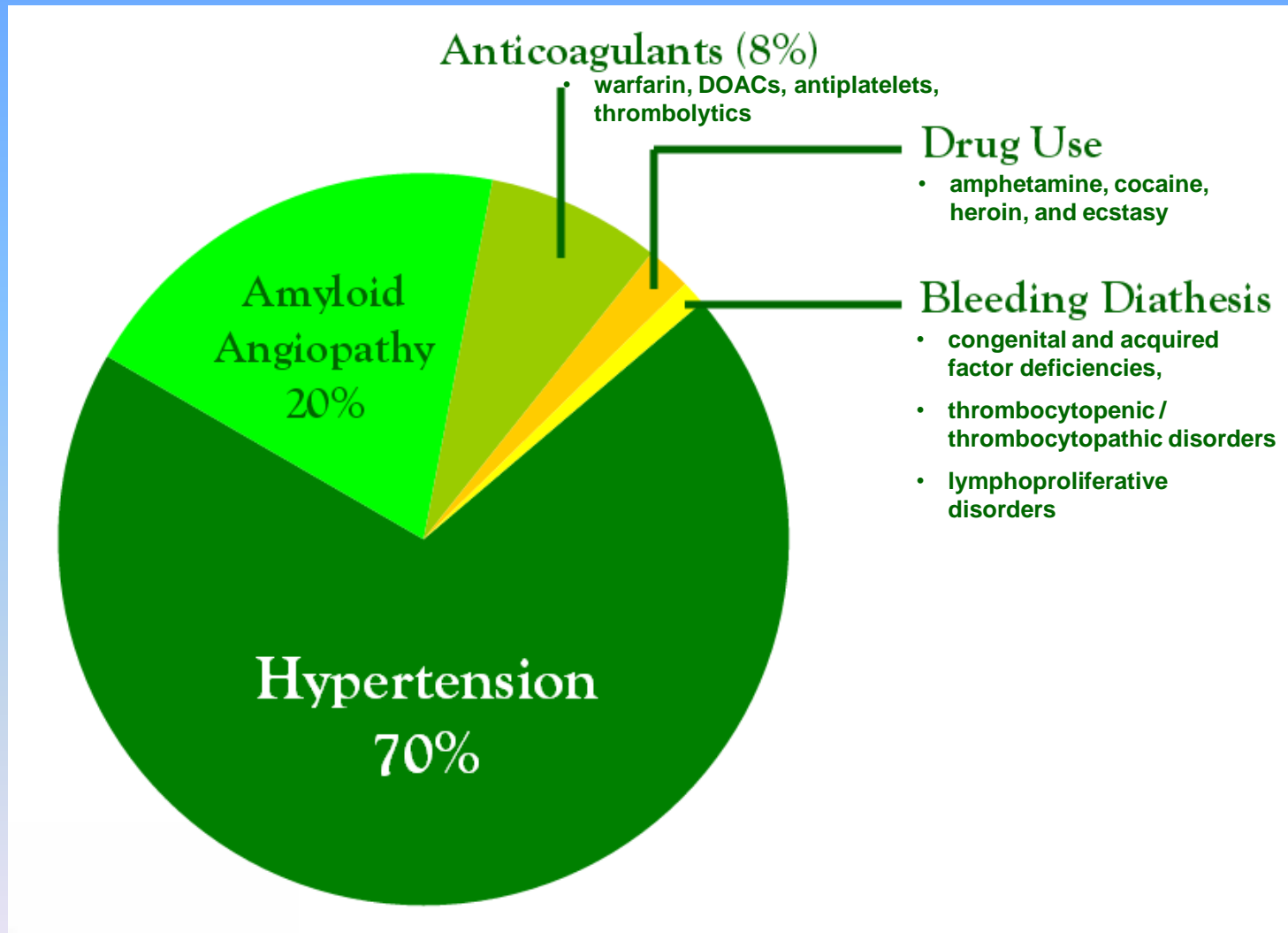
- Vascular Malformations
- Aneurysms
- Intracranial Neoplasm
- Cerebral Infarctions
- Venous Infarction
- Moyamoya Disease
- Cerebral Vasculitis

1. Qureshi AI, Tuhim S, Broderick JP, Batjer HH, Hondo H, Hanley DF. Spontaneous intracerebral hemorrhage. *N Engl J Med.* 2001; 344: 1450–1460.

2. Manno EM et al. Emerging Medical and Surgical Management Strategies in the Evaluation and Treatment of Intracerebral Hemorrhage. *Mayo Clin Proc.* 2005;80(3):420-433

Epidemiology  
and  
Pathophysiology

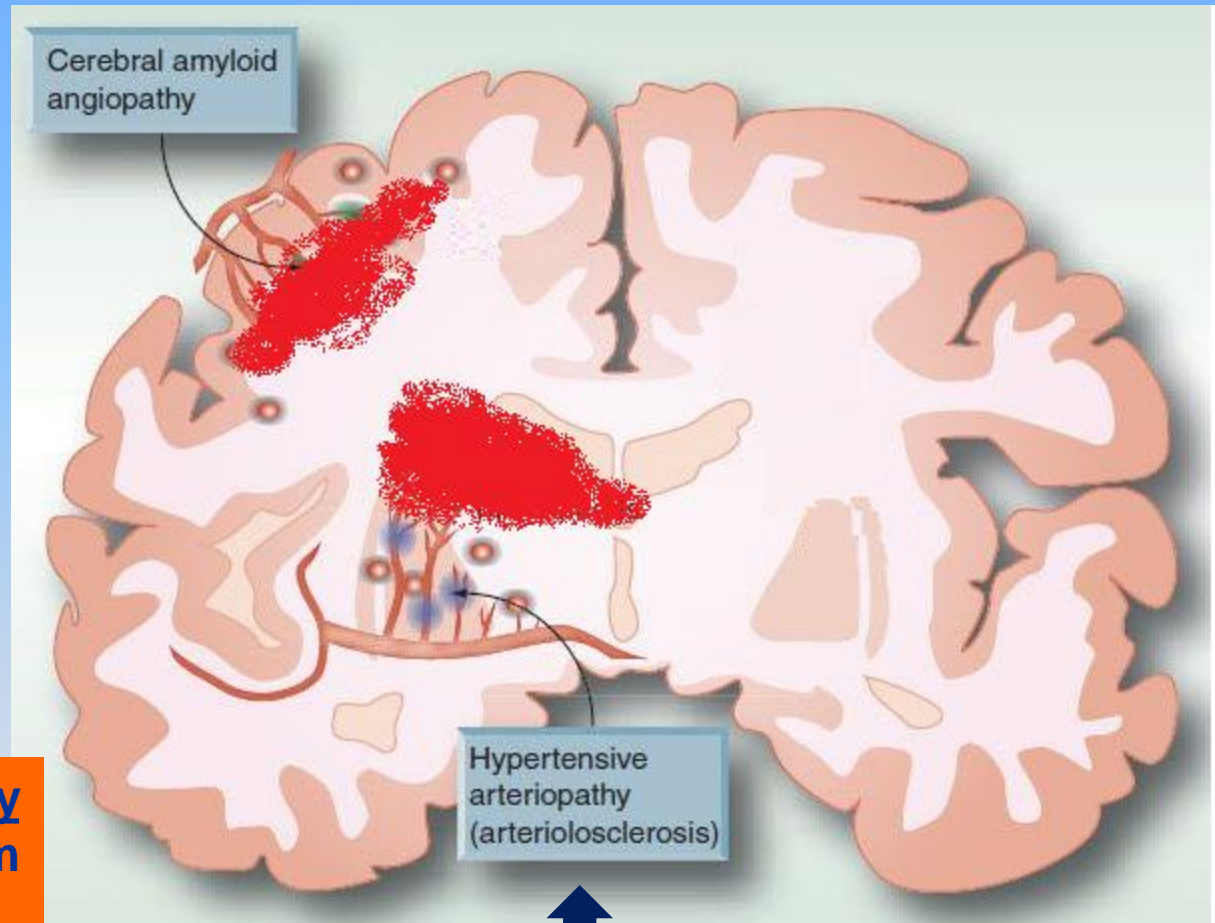
# Primary Acute ICH



1. Qureshi AI, Tuhrim S, Broderick JP, Batjer HH, Hondo H, Hanley DF. Spontaneous intracerebral hemorrhage. *N Engl J Med.* 2001; 344: 1450–1460.
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# Primary Acute ICH

**Cerebral Amyloid Angiopathy** affects small arteries and arterioles of the cerebral cortex and gray–white matter junction by the deposition of amyloid in the vessel walls.



**Hypertensive Arteriopathy** affects small vessels from deep arterial perforators to white matter and deep gray nuclei



# Primary Acute ICH

**Hypertensive  
Arteriopathy**

*Deep Pattern*



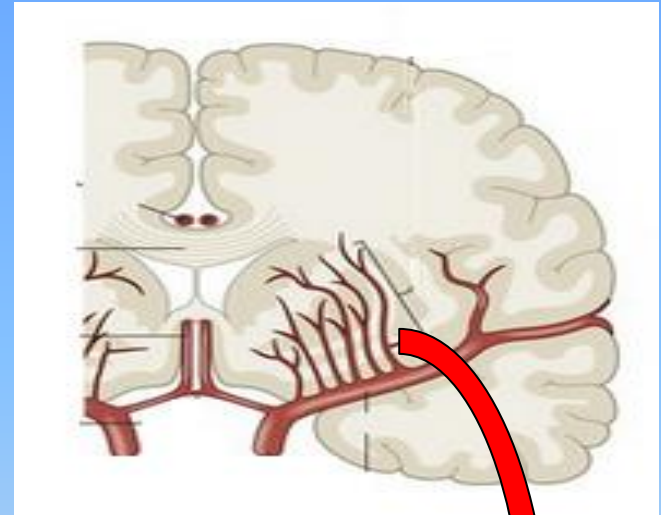
**Cerebral Amyloid  
Angiopathy**

*Lobar Pattern*



# Hypertensive Acute ICH

- territory of penetrator arteries that branch off major intracerebral arteries, often at 90° angles
- particularly susceptible to the effects of hypertension as directly exposed to the pressure of the much larger parent vessel without the protection gradual decrease in vessel calibre

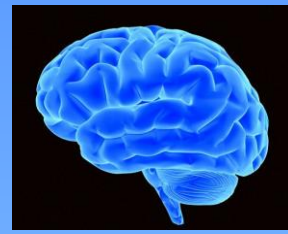


# Clinical Presentation





# ICH



- most fatal form of stroke
- poorest prognosis for survival and functional recovery.
- patients often left with moderate to severe persistent functional deficits
- about half of patients with primary ICH die in first month

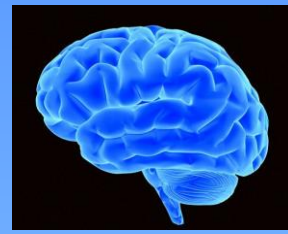
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2. Vermeer SE, Algra A, Franke CL, Koudstaal PJ, Rinkel GJ. Long-term prognosis after recovery from primary intracerebral hemorrhage. Neurology. 2002; 59: 205–209.





# ICH



- Baseline hematoma volume strong predictor of outcome (>30mL = poor outcome), but not a modifiable risk factor
- 38–46% of patients will continue to bleed and experience hematoma expansion = predictor of poor outcome.
- Risk Factors for hematoma expansion<sup>1</sup>:
  - a ‘spot sign’ (i.e. contrast extravasation) on CT
  - early presentation to medical attention
  - anticoagulation use
  - large initial hematoma volume

## ***Clinical assessment cannot reliably distinguish intracerebral hemorrhage from ischemic stroke - brain imaging is required***

<b>Presentation</b>	<b>Ischemic Stroke</b>	<b>Acute Primary ICH</b>
Altered LOC	Not always present	50%
Sudden onset	Often onsets over minutes	Sudden onset of symptoms often in seconds
Risk Factors	previous stroke, DM, a fib, MI, and intermittent claudication more common risk factor	Smoking, high alcohol intake, anticoagulation more common
Nausea & Vomitting	Less common	40-50%
Sudden Severe Headache	14%	40%
Seizures	Less common	6-7%
Elevated Blood Pressure	Not always present	90%
Progression	Can wax and wane	Early progression over minutes to hours
Severity	Less severe, less mortality when compared to ICH	More severe stroke, higher mortality



# 5 Components to the Treatment of Acute ICH



1. Initial Assessment
2. Blood Pressure Management
3. Management of Anticoagulation
4. Consultation with Neurosurgery
5. Initial Interventions

# 1. Initial Assessment

- Patients with ICH are a medical emergency
- Conduct an assessment of severity (Level B evidence)

**Awake or Drowsy**



**NIHSS**

**Obtunded, semi-comatose, or comatose**



**GCS**

- CT or MRI immediately to confirm diagnosis, location, and extent of hemorrhage (Level A evidence)

# 1. Initial Assessment

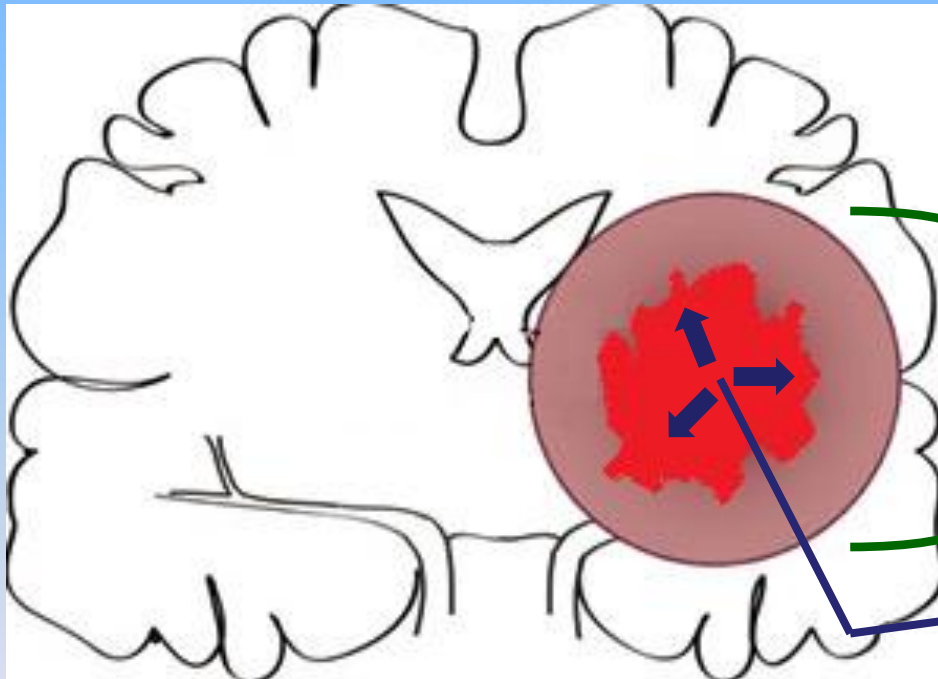
- In proven ICH, CT angiography, MR angiography, or catheter angiography is recommended to exclude aneurysm or AVM (Evidence Level B).
- Evaluation of patients with acute ICH should include:
  - questions about anticoagulant therapy (level A evidence)
  - platelet count, PTT, and INR (level A evidence)
  - medication history (level C evidence)

# 1. Initial Assessment

- Patients should be assessed for clinical signs of increased intracranial pressure (Evidence Level B).
  - Headache
  - Nausea and vomiting
  - Decreasing LOC
  - +/- Papilloedema (can take hours to days to develop)
- A validated neurological scale should be conducted by RN staff at baseline and at least hourly for the first 24 hrs, depending on stability of patient (Evidence Level C).

# 2. Blood Pressure Management

## *Two contradicting theories*



**Peri-Hematomal Penumbra**

Region of perihematomal “penumbra” at risk of ischemia if the blood pressure is reduced precipitously.

**Hematoma**

Hypertension increases hematoma growth

# 2. Blood Pressure Management

- Assess BP on arrival to ED and q15 min thereafter until (Evidence Level C).
- BP targets in may be challenging to achieve and require careful monitoring, and in some cases aggressive repeated dosing or IV infusion of antihypertensive medications (Evidence Level C)
  - My choice labetalol 10–80mg IV boluses q5-15min or starting an infusion of 2mg/min, if brady then hydralazine 5-10 mg every 30 minutes max 3 doses,
- Close BP monitoring (eg. q30-60 min, or more frequently if above target) should continue for at least the first 24 to 48 h (Evidence Level B).

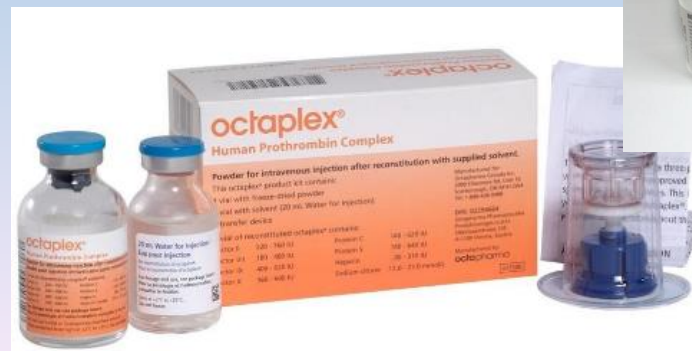


# 2. Blood Pressure Management

- Insufficient evidence that lower BP targets have better outcomes (research ongoing) but evidence to support safety for SBP < 140 mmHg (Evidence Level B). Older guidelines were SBP < 160, MAP <110
- Labetalol recommended as first-line treatment for BP management if no contraindications (Evidence Level B).
- After 24 h further BP lowering should be continued with parenteral or oral antihypertensive medications (depending on swallowing ability), to achieve individualized BP targets for usual secondary stroke prevention (Evidence Level B).

# 3. Management of Anticoagulation

- Patients with acute ICH and coagulopathy or hx of anticoagulation medications should get prompt bloodwork (INR/PTT) and have a medical treatment plan to control bleeding (Evidence Level B).
- Warfarin and elevated INR should be treated with prothrombin complex concentrate (PCC) and vitamin K. PCC is preferred (fast) but FFP and Vit K be used as alternative if PCC is not available (Evidence Level B).
- Antiplatelet agents (ASA, clopidogrel, dipyridamole/ASA) should be stopped immediately (Evidence Level C).



# 3. Management of Anticoagulation

- DOAC use = urgent consultation with hematologist (Evidence Level C).



- If strong indication for anticoagulation (e.g. a fib, mechanical heart valve), decision to restart anticoagulant made on a case-by-case basis (Evidence Level C).
- Evidence is unclear re: timing to restart anticoagulation. Consultation with stroke expert may be considered to optimize individual patient care (Evidence Level C).

# 4. Consultation with Neurosurgery

- Cerebellar hemorrhage needs urgent neurosurgical consultation, especially with altered LOC or brainstem symptoms (Evidence Level C)
- New onset of acute hydrocephalus may require placement of external ventricular drain and needs urgent neurosurgical consultation (Evidence Level C).
- Surgical intervention has not been shown to be superior to conservative management to improve outcomes in most patients with supratentorial ICH (Evidence Level B).
- In select patients with a higher LOC (especially GCS 9–12), early surgical intervention may be considered (Evidence Level B).
- Early consultation with a neurosurgeon is recommended in cases where decompressive craniectomy is considered (Evidence Level C).

## Neurosurgery Division

Dr Fraser Saunders



Dr Ronald Pokrupa



Dr Christopher Wallace



Dr Ron Levy



Dr DJ Cook



# 5. Initial Interventions

- Medically stable patients with acute ICH should be admitted to a **stroke unit or ICU** (Evidence Level B), and undergo interprofessional stroke team assessment to determine their rehabilitation and other care needs.
- Administration of **recombinant Factor VIIa** (NiaStase) prevents hematoma growth, but increases risk of arterial thromboembolic phenomena and does not provide clinical benefit for survival or outcome. Not recommended for use outside of clinical trials at this time. Clinical trials are ongoing (Evidence Level A).
- ***Statin*** therapy is not indicated for prevention of ICH. For ICH patients who have indication for cholesterol lowering treatment, statin therapy should be individualized and should take into account the patient's overall thrombotic risk as well as the possibility of increased risk of ICH on statin therapy (Evidence Level B).



# 5. Initial Interventions

- Beyond the acutely symptomatic period, patients with ICH are managed the same as those with ischemic stroke, except for avoidance of antithrombotic medications (Evidence Level B).
- Currently there is no role for prophylactic anti-convulsant treatment (Evidence Level C). If a patient were to present with or proceed to have a seizure, anti-convulsants should be initiated.

# 5. Initial Interventions - DNR

- Goals of care should be established with patient and/or designated substitute decision-maker (Evidence Level B).
- For most patients, decisions related to DNR or palliative care should be deferred for 24 to 48 h after onset, to see if there is a significant response to medical therapy or if there is worsening (Evidence Level C). Exceptions are those with preexisting wishes for DNR.
- Patients given DNR status should receive all other medical and surgical interventions unless otherwise indicated. Preexisting DNR orders should be reassessed after 24-48 hrs (Evidence Level C).
- Early DNR orders are not always inappropriate but difficulty lies in deciding when they are appropriate. Prognostication with acute ICH is an uncertain science and current guidelines suggest careful consideration of aggressive, full care during the first 24 hrs and postponement of new DNR orders during that time. This recommendation does not apply to patients with preexisting DNR orders
- 30-day mortality from ICH ranges from 35-52%; one-half of deaths occur within the first two days. A systematic review estimated that 12-39% of patients achieve independent function