In this document, we explore the incidence, prevalence, and classification of cerebrovascular disorders, focusing on stroke in Canada. **Incidence & Prevalence**

- **Incidence**: 50,000/year
- **Prevalence**: 300,000 living with effects of stroke
- **Gender**: Male = Female
- **Age**: Mean age at onset 73 and 75 respectively
- **Disability**: Leading cause of adult disability
- **Mortality**: 3rd leading cause of death

**Classification of Stroke**

**Ischemic (80%)**
- Atherosclerotic (20-30%)
- Cardioembolic (20-30%)
- Small Vessel Disease (20-30%)
- Other (e.g., dissection, hypercoagulable state, vasculitis)

**Hemorrhagic (20%)**
- Subarachnoid Hemorrhage (60%)
  - Intracranial aneurysm
- Intracerebral and/or Intraventricular hemorrhage (40%)
  - Hypertension
  - Congophilic/amyloid angiopathy
  - Arteriovenous Malformation
### Cerebrovascular Anatomy & Physiology: Clinical Syndromes

**Large Vessel**

**MCA territory**
- Dominant – Aphasia and contralateral hemiparesis (+/- hemianopsia)
- Non-Dominant – Neglect and contralateral hemiparesis (+/- hemianopsia)

**ACA territory**
- Unilateral – Contralateral leg weakness
- Bilateral – Behaviour changes +/- leg weakness

**PCA territory**
- Dominant – contralateral hemianopsia
- Bilateral – cortical blindness (+/- short term memory loss, aphasia)

### Small Vessel

**Lacunar Syndromes**
- Involve anterior and posterior territories
- Commonly in deep structures (Basal Ganglia, Internal Capsule, Brainstem, Cerebellum)
- Clinical Presentation:
  - Ataxic hemiparesis
  - Clumsy-Hand dysarthria
  - Pure Motor
  - Pure Sensory
  - Mixed Sensorimotor
### Ischemic stroke and Transient ischemic attack (TIA)

#### Definitions
- **TIA**: temporary interruption in blood supply to retina (amaurosis fugax) or brain
- Sudden onset, sudden offset, duration usually less than 60 minutes
- If symptoms last more than 24 hours then it is a stroke

#### Signs and Symptoms
- Sudden
- Weakness
- Trouble speaking
- Vision problems
- Headache
- Dizziness

#### Pathophysiology
- Atherosclerosis
  - Atheroembolic
  - Thromboembolic
  - Complete occlusion from atherosclerosis
- Cardioembolic
- Small vessel disease
Non-Modifiable Risk Factors

- Age
- Sex
- Family history
- Race
- Ethnicity

Common Causes of Stroke

HEMORRHAGIC STROKE 20%  ISCHEMIC STROKE 80%

- Intracerebral Hemorrhage
- Subarachnoid Hemorrhage
- Intracranial Aneurysm

- Migraine
- Cardiovascular Disease
- Heart disease
- Diabetic retinopathy
- Hypertension
- Hypothyroidism
- Anemia
- HIV

- Cerebral Atheroendaritis
- Aneurysms
- Malignant Strokes
- Thrombolytic therapy
- Hypertension

- Thrombosis
- Hypertension
- Hypercholesterolemia
- Hypothyroidism
- Narrowing of the arteries
- Strokes
- Aneurysms
- Heart disease
- Diabetes
- Stroke prevention
- Anticoagulant therapy
- Aspirin
- Betablockers
- Calcium channel blockers
- Digitalis

Risk Factors for Stroke
Primary Stroke Prevention
- Lifestyle Management
- See Table 2

Risk Factors for Stroke
Secondary Stroke Prevention (Modifiable Risk Factors)
- See Table 3
- Pharmacological Intervention:
  - Antiplatelet for all patients (unless cardioembolic source – then anticoagulant used instead)
  - Treatments for risk factors (eg. diabetes, hypertension)

Secondary Stroke Prevention
Carotid Revascularization
1. Carotid endarterectomy
2. Carotid angioplasty/stent
- Carotid Revascularization recommended within 14 days of TIA or non-disabling stroke (for appropriate patients)
- Carotid Revascularization may be contraindicated in the acute phase of disabling stroke secondary to the risk of reperfusion hemorrhage
Secondary Stroke Prevention

Carotid Revascularization
• Continued controversy in asymptomatic disease
• NASCET study
  – Proved benefit of carotid endarterectomy (symptomatic, moderate to severe disease)
• CREST study (compare endarterectomy with stent)
  – Older patients: surgical
  – Younger patients: angioplasty/stent

Carotid Revascularization

Carotid Endarterectomy
• Surgical exposure of artery in neck, plaque removed
• Usually GA
• Older patient, surgically accessible narrowing, ok if calcified disease
• Post operative care
  – See Table 4

Carotid Revascularization

Carotid Angioplasty/Stent
• Younger patient, recurrent disease, stenosis post radiation
• Local anaesthesia and sedation
• Post procedure care
  – See Figure 1 (Cerebral Angiography)
  – See Table 4 (post Carotid Intervention)
• Pharmacology
  – dual antiplatelet agents (clopidigrel and ASA)
Hyperacute Stroke

“Time is Brain”
- Promote public awareness to call 911 (initiates stroke protocols)
- Rapid assessment, diagnosis, investigation and treatment
- Patients eligible for IV tPA should have door-to-needle time of less than 60 minutes

Hyperacute Stroke – IV tPA

Patient Selection
- Within 4.5 hours of symptom onset
- Symptoms are not mild or rapidly resolving
- Contraindications to thrombolytics (eg. INR > 1.6, recent stroke, or recent MI)
Hyperacute Stroke – IV tPA

Administration and Monitoring

- Total dose of 0.9mg/kg (max 90 mg)
  - 10% bolus given by physician and remaining 90% given as infusion over 1 hour
- See Table 5 for nursing assessment and monitoring

Hyperacute Stroke: Intra-arterial treatments

Intra-arterial thrombolysis and/or intra-arterial thrombus extraction

- Role for this treatment is unclear
- Requires stroke centre with immediate access to cerebral angiography
- The intra-arterial approach should not delay IV tPA for those patients who are candidates for IV tPA
- See Table 5 (IV tPA) and/or Figure 1 (cerebral angiography) for the care of patients after these interventions

Acute Stroke

Goal: Minimize the effects of the Stroke

- Stroke Unit (evidence of better outcomes)
- Standardized Stroke Scale for monitoring (NIHSS or CNS)
- See Table 6 and Table 7
Acute Stroke

Surgical Treatment

Decompressive Craniectomy

1. Cerebellar Stroke
   – Suboccipital craniectomy to relieve brainstem compression

2. Malignant MCA Syndrome
   – Decompressive craniectomy for large strokes at risk of swelling and herniation
   – Evidence that early surgical intervention improves mortality and functional outcome

CNA Neuroscience Nursing Course: Cerebrovascular Disorders

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Hemorrhagic Stroke (20%)

Classification of Hemorrhagic Stroke

Subarachnoid Hemorrhage (SAH) 60%
- Aneurysm 85%
- Other 15-20%
  - Perimesencephalic 10%
  - AVMs, coagulopathies, tumors, cocaine

Intracerebral Hemorrhage (ICH) and/or Intraventricular Hemorrhage (IVH) (40%)
- Hypertension
- Congophilic/amyloid angiopathy
- Arteriovenous Malformation
  - Note bleeding may extend from one space into another, ie deep ICH \(\rightarrow\) IVH

Common Causes of Stroke

Hemorrhagic Stroke 20% Ischemic Stroke 80%

Intracranial Hemorrhage (Multiple etiologies)
Subarachnoid Hemorrhage
Subdural Hemorrhage
Epidural Hemorrhage
SAH
AVM
Hypertension
Congophilic/amyloid
angiopathy
Arteriovenous Malformation

SAH

Anatomy and common sites of aneurysms
- Bleeding into subarachnoid space
- Skull
  - Epidural space
  - Dura mater
- Arachnoid
  - Subarachnoid space
  - (circle of willis)
- Pia mater
- Brain
SAH

Traumatic SAH vs aneurysmal SAH

SAH

Aneurysmal SAH vs perimesencephalic hemorrhage

SAH

Diagnosis of aneurysmal SAH

- History
- CT head
  - Positive in 95% within 24 hours of bleed
- If in doubt...lumbar puncture
  - Persisting elevated red cell count
  - Supernatant is xanthochromic
Cerebral aneurysm

- It’s a brain thing
- At arterial bifurcations
  - Congenital weakness, flow related
  - Develop over time, thin walled
- Family history??
- Presentation
  - Incidental
  - Rupture
  - Mass effect

SAH

Aneurysmal SAH: History, incidence, prognosis

- Sudden onset worst headache of life
- +/- LOC, vomiting, focal deficit, neck stiffness, photophobia
- Incidence 10/100,000/year
- About 10-15% die outright
- Overall 50% dead/disabled at 6 months
- It’s a bad thing.....

SAH grading systems

- Clinical grade
  - WFNS (Table 8)
  - Hunt and Hess (Table 9)
    - Influences decision to treat
    - Prognostic of outcome
- Radiologic Grade
  - Fisher Scale (Table 10)
    - Volume of hemorrhage
    - May help predict risk of cerebral vasospasm
SAH

CT information

- Volume of hemorrhage (Fisher Scale)
- Location of blood
- Presence of intracerebral hemorrhage (clot)
- Ventricular hemorrhage
- May see aneurysm itself
SAH

Source of SAH: CT angiography

SAH

Early Management

ABC’s
- ABC’s
- Airway: smooth intubation
- Breathing: normal pO2, pCO2
- Circulation: BP (less than 160 systolic), arrhythmias

Neurologic intervention
- Treat hydrocephalus
- Treat elevated ICP
- Find source of hemorrhage: plan treatment to prevent rebleed

SAH

Cerebral angiography
Ruptured intracranial aneurysm: m&m

- See Tables 7 and 11
- Effects of initial hemorrhage
  - Surgery, coils will not impact (exception ICH, IVH)
- Potential to rebleed
- Hydrocephalus
- Vasospasm
- Disorders of salt/water balance

m&m: rebleed

- **Sudden** deterioration
- Highest risk in first 24 hours
- Dx: CT
- Treatment/prevention
  - SAH routine, secure (clip/coil), BP target low
  - If bleeds...ABCs
- Drugs
  - Bowel, analgesics, sedatives, avoid blood thinners
  - Antihypertensives...labetalol, hydralazine

Prevention of rebleed: clip vs coil

- Aneurysm factors
- Brain factors: ICH, swelling
- Accessibility factors
- Clinician experience/preference
- Durability vs safety
Prevention of rebleed: clip vs coil

Coil
• endovascular, GA, long term followup required
Clip
• surgical, GA, “permanent” fix
(See Table 12)
• ISAT (Lancet October 2002)
– Surgical 30% dependent or dead
– Endovascular 23% dependent or dead

Endovascular coiling

Surgical clipping
SAH

m&m: hydrocephalus

- Population at risk
  - IVH, large SAH
- Decreased LOC, “failure to thrive”
- Time frame:
  - Acute (15-80%) & urgent, delayed (8-48%)
- Dx: CT
- Treatment/prevention
  - EVD, lumbar puncture, VP shunt
  - Rarely Intraventricular tpa for severe IVH

SAH

Normal ventricles vs hydrocephalus

SAH

m&m: hydrocephalus EVD nursing care

- See Table 13
- Complications
  - Brain hemorrhage r/t catheter
  - Infection
  - Aneurysmal rebleed
  - Intracranial hypotension
SAH

m&m: cerebral vasospasm (delayed cerebral ischemia)

- Large volume SAH, younger
- Focal deficit, can be LOC, behavior change
- Day 4-14, peak 7-11 days
- Dx: TCD, CTA, Cerebral Angiography
- Treatment/prevention
  - Nimodipine, statins
  - Metabolic (O2, glucose, Mg+)
  - Maintain blood flow to brain (HHH, hold antihypertensives)
  - Endovascular: IA milrinone, angioplasty

SAH

Normal vessels vs cerebral vasospasm

m&m: Salt/water imbalance

- Global decline
- Time frame: anytime
- Dx: simultaneous urine and se osmo and lytes
- Etiology:
  - Cerebral salt wasting (linked to spasm)
  - SIADH
SAH

m&m: salt/water imbalance: etiologies

• Cerebral salt wasting
  – Brain tells kidneys to secrete Na, fluid follows
  – Prevention/treatment
    • Hypertonic solutions, salt, fludrocortisone

• SIADH
  – Brain tells kidneys to hold onto water, brain waterlogged (water is problem), Na is diluted
  – Prevention/treatment
    • Fluid restriction

Unruptured intracranial aneurysms

Clinical management

• ISUOA (2003)
  • Anterior circulation aneurysms
    – <7 mm…no bleeding,
    – 7-12 mm 2.6%,
    – 13-24mm 14.5%
    – >25mm 40%
  • Posterior circulation aneurysms
    – <7mm 2.7%
    – 7-12mm 14.5%
    – 13-24mm 18.5%
    – >25mm 50%

• Multiple aneurysms with previously ruptured aneurysms
  • Higher risk of second aneurysm rupturing

• Surgical m&m…15%

• Guidelines for practice

SAH

Drugs in SAH, cerebral aneurysms

• *Nimodipine
• Sedatives
• Analgesics
• Albumin, pentaspan
• Inotropes
• Antihypertensives
• antiplatelets
SAH

Themes in management
- Crisis
- Uncertainty
- Grief/Loss

Intracerebral Hemorrhage/Intraventricular Hemorrhage

Acute Management
- ABC’s
- Blood Pressure
- Reversal of coagulopathy (see Table 14)
- ICP (EVD, evacuation of hematoma)
- See Tables 7 and 15 for additional nursing management

Etiology

1. Hypertension
   - Most common, varied degree of severity
   - Basal ganglia, pons, cerebellum
   - Vessels stiffen and rupture
   - Blood extends along white matter tracts
   - Edema, extension of bleeding, gradual resolution
### Etiology

#### Intracerebral Hemorrhage/Intraventricular Hemorrhage

2. Congophilic (amyloid) angiopathy
   - Amyloid (protein) deposit in media and adventitia and weakens vessel
   - Over age 60
   - Lobar hemorrhage often with subarachnoid extension
   - Prone to recurrent hemorrhage in different locations
   - Avoid anticoagulants

3. Arteriovenous Malformations
   - Classification
     - Arteriovenous malformation (AVM)
     - May have associated aneurysms
     - Cavernous angioma/cavernoma
     - von Hippel Lindau
     - Developmental venous anomaly (DVA)
     - Often in association with cavernomas
     - Arteriovenous fistulas
     - Acquired post venous thrombosis, injury

   - Congenital (0.5% of population)
   - Abnormal connection between arteries and veins
   - No capillary bed
   - high flow
   - Feeding arteries, draining veins, “nidus”
   - May be associated aneurysms
Arteriovenous Malformations

- Presentation
  - Hemorrhage (50%)
  - Seizure (25-50%)
  - Headache (12-35%)
  - Progressive neurologic deficit (10-25%)

Arteriovenous Malformations

- Natural History:
  - Risk of rupture 1.5-3% per year
  - Risk of recurrent rupture, highest in first months after bleed
  - Mortality 20-30%
  - Mortality 10-15%

- Higher risk features
  - Associated aneurysm, venous outflow narrowing
Intracerebral Hemorrhage/Intraventricular Hemorrhage

Arteriovenous Malformations: Treatment Options

- Conservative
- Embolization (often multiple)
- Surgical excision
- Radiosurgery (less than 3 cm)
- *Combination therapies common (see Table 16)

Arteriovenous Malformations: Embolization

- Neuroradiology, usually GA, 3-5 hours
- Post procedure monitoring:
  - Cerebral angiography (Figure 1)
  - Stroke (ICH, progressive thrombosis with ischemia)
- Staged, multiple
- Occasionally used to ameliorate symptoms (bruit, headache) without definitive cure

Arteriovenous Malformations: Surgical Excision

- Post-op Care
  - Craniotomy
    - Often lengthy OR time
  - Reperfusion syndrome:
    - "clot waiting to happen"
    - Tight blood pressure control
    - May elect to keep sedated x24-48 hours

Post-op Care
- Craniotomy
  - Often lengthy OR time
- Reperfusion syndrome:
  - "clot waiting to happen"
  - Tight blood pressure control
  - May elect to keep sedated x24-48 hours
Arteriovenous Malformations: Radiosurgery
- Specialized centre (with Gamma knife or Linear Accelerator)
- "nidus" under 3 cm.
- Delay in occlusion (2-3 years)
- Single treatment
- Need follow up imaging
- Acute and delayed complications
  - Bleed, edema, seizures

Cavernous Angioma/Cavernoma
- Collection of tightly packed venous channels
- Occult on angiography
- 0.1 - 0.5% of population
- Presentation:
  - Seizure, hemorrhage, headache, progressive deficit
- Treatment
  - Conservative
  - Surgery if multiple symptoms and accessible
Intracerebral Hemorrhage/Intraventricular Hemorrhage

Other Etiologies

4. Coagulopathies
   - acquired
     • Antiplatelets, anticoagulants
     • Thrombocytopenia, ITP
   - Congenital
     • Factor deficiencies

5. Vasculitis
   - Inflammation of blood vessels
     a. Intracranial eg. Cerebral vasculitis
        • Headache, seizures, behaviour change, or focal deficit
        • May result in ischemic or hemorrhagic stroke
        • Inflammatory markers, CSF analysis, cerebral angiography and/or brain and blood vessel biopsy may help diagnosis
        • Treatment includes immunosuppressant therapy (steroids, cyclophosphamide)
   b. Extracranial eg. Temporal arteritis
        • Vision loss, headache/temporal tenderness
        • May result in blindness
        • ESR, temporal artery biopsy
        • Treat with steroids

6. Illicit drug use
   - Cocaine

7. Cerebral venous thrombosis
Cerebral venous thrombosis

• Risk factors
  – Acquired
    • OCP, pregnancy, obesity, parameningeal infections (otitis media)
  – Genetic
    • Hypercoagulable syndromes
• Incidence 0.5-1% of strokes

Symptoms:
• Headache, visual obscurations, seizures, focal deficit
• May result in ischemic stroke (often with hemorrhagic transformation)
• Edema from venous engorgement → IICP, papilledema
• If left untreated papilledema → blindness (pseudotumor)

Anticoagulation
• If worsens despite anticoagulation → interventional angioplasty, stent or thrombectomy

Treatment to prevent vision loss:
– Lumboperitoneal or ventriculoperitoneal shunt
– Diamox to reduce CSF production (temporize)
– Optic nerve sheath fenestration
Primary and secondary prevention

- Blood pressure control
- Limit alcohol consumption
- Smoking cessation
- Etiology specific
  - Treat structural lesion
  - Reverse coagulopathy

Classification of Stroke

<table>
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<tr>
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| • Atherosclerotic (20-30%) | • Subarachnoid Hemorrhage (60%)
| • Cardiembolic (20-30%) |   - Intracranial aneurysm
| • Small Vessel Disease (20-30%) |   - Intracerebral and/or Intraventricular hemorrhage (40%)
| • Other (eg. dissection, hypercoagulable state, vasculitis) |   - Hypertension
|                            |   - Congophilic/amyloid angiopathy
|                            |   - Arteriovenous Malformation

- Hypertension
- Congophilic/amyloid angiopathy
- Arteriovenous Malformation