MODULE 3: PRE-HOSPITAL AND EMERGENCY MANAGEMENT

Learning Objectives

Upon completion of this module, nurses will be able to:

- Understand the importance of symptom recognition and reaction
- Know the FAST signs of stroke
- Understand the role of EMS in hyperacute stroke
- Understand the role of thrombolytic therapy and endovascular treatment
- Identify stroke mimics
- Understand the role of ASA therapy for acute ischemic strokes
- Understand management of ischemic and hemorrhagic stroke

For the most up to date information please refer to the following Canadian Stroke Best Practice Guideline when completing this module:

NEW Acute Stroke Management | Canadian Stroke Best Practices

Management of Spontaneous Intracerebral Hemorrhage | Canadian Stroke Best Practices

SWO Stroke Network, 2023
Adapted from NEO Stroke Network (2010).

3.1 Stroke Warning Signs and Pre-hospital Care

The FAST Signs of Stroke

- FACE: Is it drooping?
- ARMS: Can you raise both?
- SPEECH: Is it slurred or jumbled?
- TIME: To call 9-1-1 right away.

Sometimes other symptoms appear, separately or in combination with F.A.S.T. signs:

- Sudden confusion, trouble speaking or understanding speech.
- Sudden numbness or weakness of face, arm or leg. Especially on one side of the body
- Sudden trouble walking, dizziness, loss of balance or coordination
- Sudden severe headache with no known cause.

Hyperacute stroke care is defined as the health care activities that take place from the time of first contact between a potential stroke patient and medical care. This period ceases once the patient is either admitted to hospital or discharged back into the community.

In broad terms, "hyperacute" refers to care offered in the first 24 hours after stroke (ischemic and hemorrhagic) and the first 48 hours after TIA. Canadian Best Practice Recommendations, 2013.

Pre-hospital Care

Patients who show signs and symptoms of hyperacute stroke in the community must be treated as **time-sensitive emergency cases** and should be transported without delay to the closest institution that provides emergency stroke care. Immediate contact with emergency medical services is strongly recommended because it improves the time to treatment for acute stroke. A system of rapid transport should be available to

Emergency Medical Service (EMS) dispatchers must triage patients showing signs of hyperacute stroke as a priority dispatch. EMS providers should use a standardized diagnostic screening tool. In Ontario, a **Paramedic Prompt Card** has been implemented for this purpose. The prompt card assists EMS providers in initially recognizing the signs and symptoms of stroke and subsequently in determining the most appropriate hospital to transport the patient.

As part of the Paramedic Prompt Card, EMS will complete the **Los Angeles Motor Scale (LAMS).** The LAMS is a three-item, 0-5-point (per side) motor scale that is validated in prehospital settings and is utilized as a secondary screen for Large Vessel Occlusion strokes. This screening tool helps to identify who may be eligible for EVT. A score of >4 is considered "positive".

Paramedic Prompt Card For Acute Stroke Protocol

Emergency Health Regulatory and Accountability Branch

Paramedic Prompt Card for Acute Stroke Bypass Protocol

This prompt card provides a quick reference of the *Acute Stroke Protocol* contained in the *Basic Life Support Patient Care Standards* (BLS PCS). Please refer to the BLS PCS for the full protocol.

Indications under the Acute Stroke Protocol

Redirect or transport to the closest or most appropriate Designated Stroke Centre* will be considered for patients who meet **ALL** of the following:

1. Present with a new onset of at least one of the following symptoms suggestive of the onset of an acute stroke:
    a. Unilateral arm/leg weakness or drift.
    b. Slurred speech or inappropriate words or mute.
    c. Unilateral facial droop.
2. Can be transported to arrive at a Designated Stroke Centre within 6 hours of a clearly determined time of symptom onset or the time the patient was last seen in a usual state of health.
3. Perform a secondary screen for a Large Vessel Occlusion (LVO) stroke using the Los Angeles Motor Scale (LAMS) and inform the CACC/ACS to aid in the determination of the most appropriate destination.

*A Designated Stroke Center is a Regional Stroke Centre, District Stroke Centre or a Telestroke Centre regardless of EVT capability.

Contraindications under the Acute Stroke Protocol

ANY of the following exclude a patient from being transported under the Acute Stroke Protocol:

1. CTAS Level 1 and/or uncorrected airway, breathing or circulatory problem.
2. Symptoms of the stroke resolved prior to paramedic arrival or assessment**.
3. Blood sugar <3 mmol/L***.
4. Seizure at onset of symptoms or observed by paramedics.
5. Glasgow Coma Scale <10.
6. Terminally ill or palliative care patient.
7. Duration of out of hospital transport will exceed two hours.

**Patients whose symptoms improve significantly or resolve during transport will continue to be transported to a Designated Stroke Centre.

*** If symptoms persist after correction of blood glucose level, the patient is not contraindicated.

CACC/ACS will authorize the transport once notified of the patient's need for redirect or transport under the Acute Stroke Protocol.

Ontario

NOTES:

• A Designated Stroke Centre is a Regional Stroke Centre, District Stroke Centre or Telestroke Centre.

• Patients will be redirected or transported to the closest Designated Stroke Centre.

• Patients whose symptoms improve significantly or resolve during transport will continue to a Designated Stroke Centre.

• Out-of-hospital transport will not exceed two hours.

The Central Ambulance Communications Centre (CACC) will authorize the transport once notified of the patient's need for redirect or transport under the Acute Stroke Protocol.

The 2022 Acute Stroke Management Canadian Stroke Best Practice Recommendations also emphasize the need for rapid transport of acute stroke patients to appropriate facilities. A system of rapid transport should be available to facilitate the movement of patients from one emergency department to another when time-sensitive stroke-specific care cannot be provided in the emergency department where the patient is first assessed.

It is important to create public awareness about the necessity of calling 911, so patients may be taken to the nearest Designated Stroke Centre.

Emergency Evaluation and Management of Patients with Transient Ischemic Attack and Ischemic Stroke

Time is Brain! All patients presenting to the emergency department with suspected stroke or transient ischemic attack must have an immediate clinical evaluation and investigations to establish the diagnosis, rule out stroke mimics, determine eligibility for thrombolytic therapy, and develop a plan for further management (2013 Canadian Best Practice Recommendations for Stroke Care: Hyperacute Stroke Care, p 32).

Stroke Mimics

Not all cases that appear as a stroke are in fact a stroke. Be aware of the many other conditions that would be part of the differential diagnoses as they can present much like a stroke.

- Seizure
- Infection
- Hypoglycemia
- Syncope
- Brain abscess or tumour
- Drug overdose
- Head trauma
- Migraine
- Bell's palsy
- Hypertensive encephalopathy

3.2 Acute ASA Therapy

All patients with acute ischemic stroke or transient ischemic attack (TIA) who are not already on an antiplatelet agent should be treated with at least **160 mg of acetylsalicylic acid (ASA) immediately** as a one-time loading dose after brain imaging has excluded intracranial hemorrhage [Strong recommendation; High quality of evidence]. (2022 Acute Stroke Management, Canadian Stroke Best Practice Recommendations)

For patients with delayed swallow screen or potential dysphagia, ASA (81 mg daily) or clopidogrel (75 mg daily) may be administered by enteral tube or ASA (325 mg daily) by rectal suppository [Strong recommendation; Moderate quality of evidence]. Note: ASA and clopidogrel should only be administered orally once dysphagia screening has been performed and indicates an absence of potential dysphagia (p. 84). (2022 Acute Stroke Management, Canadian Stroke Best Practice Recommendations)

For patients treated with intravenous thrombolysis, antiplatelet therapy should be delayed until after the 24-hour post-thrombolysis scan has excluded intracranial hemorrhage.

Note: Use or non-use of anticoagulants, including the timing of the last dose taken, should be sought and recorded during the initial patient assessment.

3.3 Acute Thrombolytic Therapy

All patients with acute ischemic stroke who can be treated within 4.5 hours after symptom onset should be evaluated without delay to determine their eligibility for treatment with thrombolytics.

Thrombolysis

Tissue plasminogen activator (tPA or Alteplase) is a thrombolytic agent (clot-busting drug) that can destroy an existing blood clot that is approved for use in select patients having an ischemic stroke.

Tenecteplase (TNK) may be considered as an alternative to alteplase within 4.5 hours of acute stroke symptom onset [Strong recommendation; Moderate quality of evidence] (2022, Acute Stroke Management Canadian Stroke Best Practice Recommendations). TNK is a genetically modified version of Alteplase. If administering tenecteplase, the dose of 0.25 mg/kg up to a maximum of 25 mg should be administered, given as a single bolus over 5 seconds [Strong recommendation; Moderate quality of evidence] (p. 61). Caution: The dosing of alteplase and tenecteplase for stroke is NOT the same as the dose protocols for administration of these medications for myocardial infarction or massive pulmonary embolism

Time is brain *- the closer to the time of stroke onset that reperfusion occurs, the better the patient prognosis.*

Ultimately, the goal of thrombolytic therapy is to limit irreversible ischemic damage caused by an arterial occlusion. Thrombolysis will promote reperfusion of the viable tissue of the penumbra, improving stroke prognosis and outcome.

Prior to administration of the drug, the patient must undergo specific diagnostic procedures to determine if there is any hemorrhage. This requires **immediate** access to CT and CTA imaging. Additional imaging such as MRI and MRA may be considered, however this should not delay decision and/or treatment with thrombolysis or EVT (2015 Canadian Best Practice Recommendations for Stroke Care: Hyperacute).

*N*OTE: Intravenous thrombolytics should not routinely be administered to patients on Direct Oral Anticoagulants (DOACs) who present with acute ischemic stroke. Treatment may be considered in consultation with a stroke expert

What is the usual process prior to a patient receiving thrombolysis?

Each center will have standard order sets and protocols.

- Determine last seen normal time (less than 4.5 hours) However, remember, patients whose last seen normal time is 6.0hrs may still be candidates for EVT.
- Ensure history and physical symptoms are consistent with acute ischemic stroke
- CT to rule out hemorrhagic stroke (or any etiology other than ischemic stroke)
- Bloodwork: CBC, platelets, electrolytes, glucose, INR, PTT, renal function, troponin, fasting lipid profile, fasting glucose level and HbA1c, and TSH
- Assessment by a *Physician* with stroke expertise; considering inclusion/exclusion criteria for thrombolysis.

Sites providing thrombolysis should have local protocols and assessments to support IV thrombolysis inclusion/exclusion criteria, and pre and post thrombolysis administration monitoring.

In addition, sites should consider complications such as symptomatic intracranial hemorrhage following administration of intravenous thrombolysis.

Intracranial hemorrhage should be considered if there is a change in neurological status, a spike in blood pressure with persisting elevation or a new/worsened headache. In this case, an immediate non-contrast CT head should be done, with a stroke team member accompanying the patient. If intracranial hemorrhage is identified the thrombolysis infusion should be stopped immediately.

For additional clinical considerations on thrombolysis administration, refer to the 2022 Canadian Stroke Best Practice Guidelines: Acute Stroke Management.

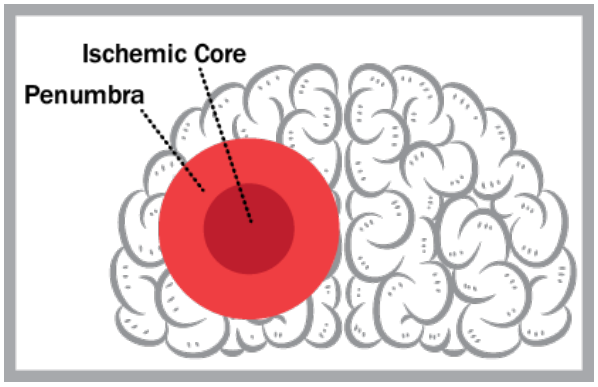
Why work quickly to determine if thrombolysis is the appropriate treatment?

The faster the thrombolysis takes place; the less brain tissue is affected by the stroke. Surrounding the ischemic core (infarcted tissue) is the ischemic penumbra (moderately ischemic tissue that is still viable but lacking perfusion and, therefore, at risk).

The human brain requires an uninterrupted blood supply of glucose and oxygen because the brain does not store them. An interruption in either can lead to cellular dysfunction.

For example, a complete interruption of blood supply to part of the brain for only 30 seconds can alter brain metabolism and neuronal function may cease after 1 minute. After 5 minutes, anoxia initiates a chain of events that may lead to death of brain tissue.

Penumbra tissue remains viable for **several hours** after stroke. Penumbra cells are supplied by collateral arteries which contribute to reperfusion. Thrombolytic therapy also works to perfuse the penumbra.



A stroke patient should receive thrombolytic therapy as soon as possible. A rapid and coordinated emergency department response facilitates early diagnosis and treatment. National benchmarks for ED assessment and triage include:

- Door to CT/CTA: ≤ 15 minutes
- Stroke onset to tPA administration: ≤ 4.5 hours
- Door to Needle/tPA: ≤ 30 minutes
- Door to Groin Puncture: ≤ 60 minutes
- Door to Revascularization: ≤ 90 minutes

3.4 Adverse effects of tPA

Nurses should be aware of the adverse effects of tPA.

Hemorrhage

Superficial Bleeding

- Observe potential bleeding sites: venous and arterial puncture, lacerations, etc.
- Avoid invasive procedures during tPA and for 24 hours after (including nasogastric (NG) and foley catheter)
- Monitor all secretions for bleeding
- Notify *Physician* if bleeding is present or suspected

Intracranial hemorrhage

- Observe for deterioration of neurological status, including: a reduction in level of consciousness, a spike in blood pressure or a worsened headache.
- If suspected, stop tPA and notify *Physician*
- Obtain immediate non-contrast CT scan and coagulation work-up such as complete blood count, INR and type and cross.

Angioedema

Risk assessment

- Inquire if patient has had angioedema in past
- Take Angiotensin Converting Enzyme Inhibitors (ACE) history
- Although angiotensin II (ATII) receptor antagonists have not been implicated in the angioedema reaction, caution is advised in patients reporting a history of ATII antagonist use

Monitoring

- Observe for facial, tongue, and/or pharyngeal angioedema 30 minutes, 45 minutes, 60 minutes and 75 minutes after initiation of IV tPA infusion, and periodically for 24 hours afterwards
- Acute Ischemic Stroke Non tPA: Vital signs (including temperature) should be assessed as follows or as indicated by hospital protocol:
  - q 1 hour for 24 hours
  - q 4 hours for 24 hours

3.5 Endovascular Thrombectomy (EVT)

Endovascular Thrombectomy (also known as endovascular treatment) is a newer avenue for hyperacute stroke care.

It is an image guided procedure for clot removal using a catheter commonly inserted through the groin. EVT is typically performed in an EVT centre by a specialist with neurointerventional expertise. In Southwestern Ontario the 2 EVT centres are located at University Hospital; and Windsor Regional Hospital. The window for this therapy is slightly longer: 6 hours -24 hours.

Patients can receive EVT with or without I.V thrombolysis. Using both thrombolysis and EVT therapies in combination provide effective results.

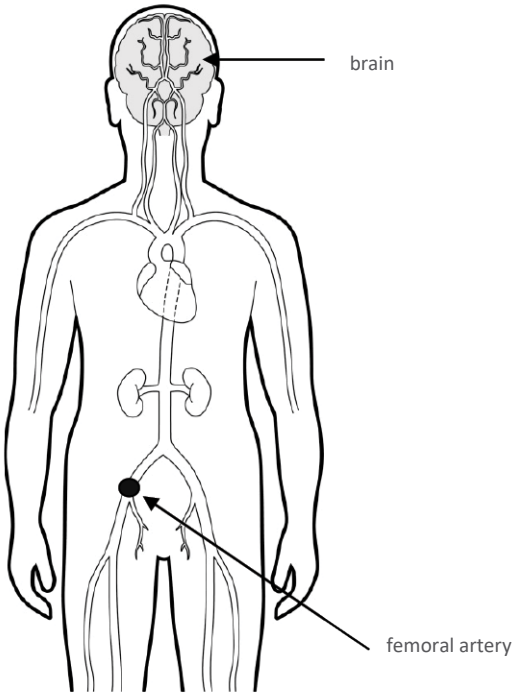
As per the 2022 Acute Stroke Management: Canadian Stroke Best Practice Guidelines the use of intravenous thrombolysis and/or EVT in patients who are not functionally independent may be considered, based on careful review.

When a patient who is eligible for both intravenous thrombolysis and EVT presents to an EVT site, a decision not to administer intravenous thrombolysis and proceed straight to EVT may occur. With this, the main focus remains "time is brain" and maintaining improved patient outcomes is key.

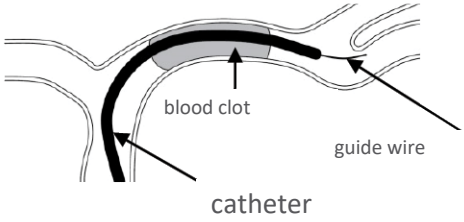
NOTE: Anticoagulation is not a contraindication for EVT, and the decision to treat should be based on individual patient factors and assessment of benefit and risk.

Mechanical Thrombectomy (Clot Retrieval)

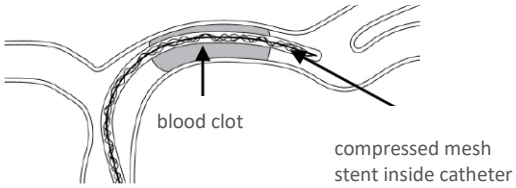
How the procedure is done:



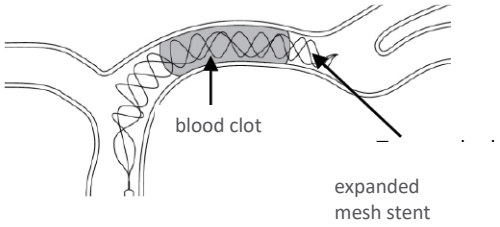
A small thin tube, called a sheath, is inserted in the femoral artery in the groin area.



A guide wire and catheter are inserted through the sheath into the femoral artery and passed to the artery with the clot in the brain.



The guide wire is removed and a compressed mesh stent is inserted through the catheter to the clot.



The catheter is pulled back causing the mesh stent to expand through the clot. Once the clot is “trapped” in the stent, the clot can be safely removed with the stent.

Obtained from LHSC: Clot Retrieval for Stroke, Patient and Family Education pamphlet, 2017.

Potential candidates for EVT

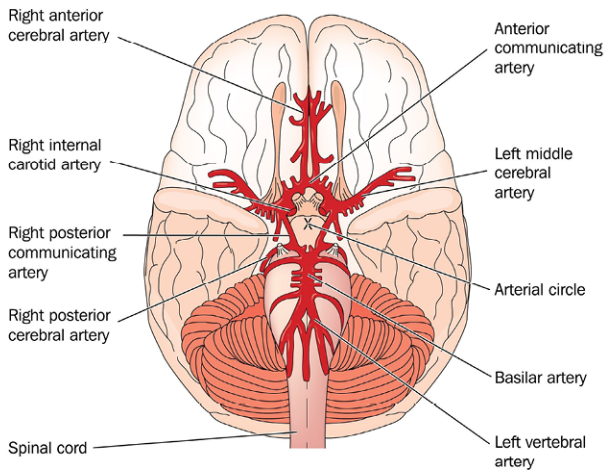
- Patients with an occluded proximal intracranial artery, which is a target lesion of:
  - ICA terminus, M1, M2 –M1 equivalent, basilar artery.
  - The presence of good collaterals on multiphase CTA (CT Angiography)
  - 6-24 hours from stroke symptoms onset.

Main Vessels Treated with Thrombectomy

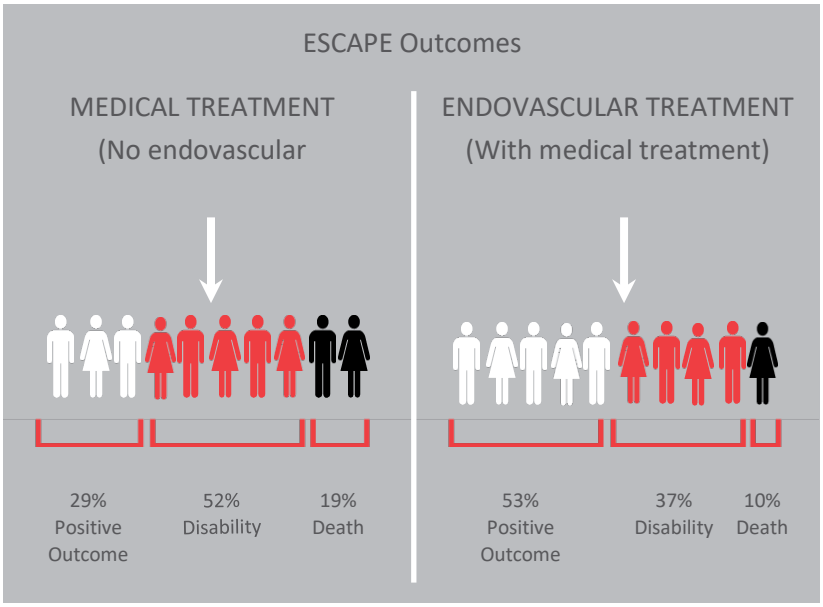
The larger cerebral arteries are accessible for thrombectomy, namely:

- Middle Cerebral Artery (MCA) –M1* and M2* segments
- Anterior Cerebral Artery (ACA) –A1* segment
- Internal Carotid Artery (ICA)
- Basilar Artery
- Posterior Cerebral Artery (PCA)

* Refers to the larger branch of that specific artery



What is the impact of EVT?



M.Hill Escape Trial 2015

Pre and Post EVT treatment

General management considerations, including: airway management, anesthesia consideration, contrast allergy management and pre and post EVT patient monitoring considerations are highlighted in Section 5, Box 5D of the 2022, Canadian Stroke Best Practice Recommendations: Acute Stroke Management. In addition, all EVT sites should continue to follow local protocols and assessments.

3.6 Acute Ischemic Stroke Management

The goals of Acute Ischemic Stroke Management are:

- Reduce or minimize ischemic damage
- Reduce cerebral edema
- Prevent secondary complications
- Determine etiology of stroke
- Prevent recurrent stroke
- Facilitate access to an acute stroke unit, rehabilitation and support community reintegration

Contributing Factors to Ischemic Damage

Restoring blood flow to the penumbra is the goal of acute stroke management; there are multiple factors to consider and address as part of the management plan. Factors that contribute to a potential size increase in of the infarct include:

- Blood pressure
- Blood glucose
- Body temperature
- Oxygen saturation

It is important to **assess and monitor vital signs** to keep this goal at the forefront. (Heart and Stroke: Best Practice Guidelines for Stroke Care, 2003)

Blood Pressure

Acute stroke patients often experience hypertension (HTN) in the immediate hours after stroke onset. Initially, elevated blood pressure (BP) may act as a compensatory mechanism to maintain cerebral perfusion.

Normally, cerebral autoregulation maintains cerebral blood flow. However, as cerebral perfusion pressure decreases in the presence of stroke, normal autoregulation is lost and blood flow depends on blood pressure.

Many factors cause HTN secondary to stroke: full bladder, nausea, pain, pre-existing HTN, anxiety, a physiological response to hypoxia, or increased intracranial pressure.

Both hypertension *and* hypotension have been associated with poor patient outcomes. There are good reasons to lower blood pressure: HTN can increase cerebral edema, increase risk of hemorrhagic transformation, cause further vascular damage, or cause stroke recurrence. However, reducing blood pressure too quickly, or too low, may cause neurological damage as a result of reduced perfusion pressure to the ischemic areas; it can result in serious consequences.

For some stroke patients, blood pressure may decline spontaneously within the first few hours, resulting from interventions like moving the patient to a quieter area, emptying the bladder, allowing the patient to rest, or controlling pain. The treatment of increased intracranial pressure may also result in a lowering of blood pressure.

Tips on Blood Pressure Reduction

- Blood pressure reduction should be addressed cautiously
- Measure blood pressure accurately, continuously monitor
- Clear data is lacking on how and when to reduce blood pressure but:

NOTE: Please refer to the anti-hypertensive guidelines set out on the preprinted order sets at your hospital.

2013 American Heart Association/American Stroke Association Guidelines recommend:

- Initiate treatment if SBP greater than 220mmHg or DBP greater than 120mmHg
- tPA candidates: Initiate treatment if SBP greater than 185mmHg or DBP greater than 110mmHg
- Lower blood pressure by 15-25% within 24 hours
- Medication selection on case by case basis but consider ability to lower blood pressure carefully and ability for rapid reversal

NOTE: Use of sublingual Nifedipine is contraindicated due to its prolonged effect and rapid decline in BP.

2015 Canadian Stroke Best Practice Recommendations:

- Avoid rapid or excessive lowering of blood pressure
  - This may exacerbate existing ischemia or may induce ischemia, especially in the setting of intracranial arterial occlusion or extracranial carotid or vertebral artery occlusion (Evidence Level C) p.9.
- Treat SBP>220mmHg or diastolic>120mmHg.
  - Should be reduced by about 15% and not more than 25% over the first 24hrs with further gradual reduction thereafter to targets for long-term secondary stroke prevention (Evidence level C) p.9.

Blood Glucose

(See your hospital's protocols for specific thresholds)

- All patients with suspected acute stroke should have their blood glucose concentration checked immediately.
- Blood glucose measurement should be repeated if the first value is abnormal or if the patient is known to have diabetes.
- Markedly elevated blood glucose concentrations (hyperglycemia) should be treated with glucose lowering agents immediately. (Lindsay, 2005)

Hyperglycemia is associated with worse stroke outcomes and is a risk factor for hemorrhagic transformation. It can also have a serious effect on aphasia, hemiparesis, and changes in mental status. It is unclear whether hyperglycemia increases cerebral damage, or to what extent post-stroke hyperglycemia is a normal physiological response.

Studies have also shown that hyperglycemia is linked to increased risk for in-hospital mortality in non-diabetic patients, and/or increased risk of poor functional recovery. Keep in mind, many stroke patients may be unaware they have diabetes (a modifiable risk factor) until admission to hospital.

Body Temperature

(See your hospital's protocols for specific parameters)

Temperature should be routinely monitored and treated if greater than 37.5 Celsius. Increased body temperature of greater than 37.6 Celsius (hyperthermia) in the setting of acute ischemic stroke is associated with poor neurological outcome (increased risk of morbidity and mortality), and is possibly secondary to increased metabolic demand, enhanced release of neurotransmitters, and increased free radical production. The source of any fever should be ascertained. The fever may be secondary to a cause of stroke, such as infective endocarditis, or it may represent a complication such as pneumonia, urinary tract infection or sepsis.

(American Heart Association/American Stroke Association, 2013)

Oxygen Saturation

(See your hospital's protocols for specific thresholds)

Ensuring adequate oxygenation of tissues is important to acute stroke management to prevent worsening of ischemic damage. Oxygen saturation should be monitored with the use of pulse oximetry maintaining oxygen saturation above 92%. Oxygen should be provided if oxygen saturation is less than 95%, p.9. 2015 Canadian Stroke Best Practice Recommendations. However, supplemental oxygen given to patients who are not hypoxic may result in production of oxygen-free radicals and contribute to worse outcome.

Consideration and assessment of other causes of hypoxia should also be reviewed. These include pneumonia, partial airway obstruction, hypoventilation and atelectasis. Patients who have brain stem infarcts have the greatest risk of airway compromise due to impaired oropharyngeal mobility and loss of reflexes.

Sleep apnea, another influencing factor, is more common in stroke survivors than in the general population. Obstructive sleep apnea has been identified as both a risk factor for stroke and a secondary condition that develops post-stroke. It is associated with uncontrolled hypertension, and the onset of atrial fibrillation. Nurses should monitor patients for potential signs and risk factors for sleep apnea, including:

- Snoring
- Tiredness (although, research shows that stroke survivors may not present with excessive fatigue)
- Pauses in breathing when sleeping
- Hypertension
- Aged over 50
- Male
- Large neck circumference

If you observe any of these, document what you have seen and speak to the *Physician*.

3.7 Hemorrhagic Stroke Management

Hemorrhagic Stroke occurs when a blood vessel bursts and there is bleeding into the brain. Twenty percent of all strokes are hemorrhagic, and they can be classified as either subarachnoid or intracerebral (see *Module 1: Pathophysiology of Stroke, Neuroanatomy, and Stroke Syndromes).*

Subarachnoid hemorrhage: characterized by bleeding around the brain and is often caused by rupture of a weakened blood vessel (aneurysm) on the surface of the brain.

Intracerebral hemorrhage: characterized by bleeding into the brain and is most often caused by high blood pressure due to rupture of a deep penetrating artery (Mink and Miller, 2011), or from cerebral atherosclerosis.

Damage can occur quickly due to pressure caused by increasing amounts of blood, or because of the blood itself. Blood is irritating to brain tissue and causes it to swell.

Hemorrhagic Stroke has a 30 day mortality rate between 35% and 52%, with half of the deaths occurring within the first two days of intracranial hemorrhage (Miller and Mink, 2011). Size matters in hemorrhagic stroke, and decreasing Glasgow Coma Scale is highly predictive of death (Martin, 2013). For detailed information on the management of intracerebral hemorrhage refer to the CSBPR module Management of Spontaneous Intracerebral Hemorrhage (2020).

Nurses should:

- Understand how large the bleed is and where it is located in the brain
- Maintain head of bed 30 degrees; keep head positioned midline in bed
- Manage blood pressure; In patients with symptomatic ICH who are hypertensive (>185/110 mmHg), blood pressure should be lowered, however, the specific target and duration of therapy are unknown at this time

Predictors of poor outcome include:

- Temperature elevation greater than 37.5 Celsius (higher risk of death)
- Increased age greater than 85 years
- Increased ICP
- Increased time from onset of bleed until hospitalization

Treatment

The treatment of a hemorrhagic stroke depends upon the cause of the bleeding (e.g., high blood pressure, use of anticoagulant medications, head trauma, blood vessel malformation). Most patients are monitored closely in an intensive care unit during and after a hemorrhagic stroke. The initial care of a person with hemorrhagic stroke includes several components:

- Determine cause of bleeding.
- Control blood pressure.
- Stop any medication that could increase bleeding (e.g., warfarin, aspirin). If the patient has been taking warfarin, specific treatments such as factor VIIa or transfusions of blood clotting factors, may be given to stop ongoing bleeding.
- Measure and control the pressure within the brain (Caplan, 2013).

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