Stroke Network
Southwestern Ontario
Serving Erie St. Clair and South West LHINs

ACUTE STROKE UNIT ORIENTATION

2014

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 5 warning signs of stroke
- Understand the role of EMS in hyperacute stroke
- Understand the role of thrombolytic therapy and administration of tPA
- Identify complications post administration of tPA
- Identify stroke mimics
- Understand the role of ASA therapy for acute ischemic strokes
- Understand management of ischemic and hemorrhagic stroke

SWO Stroke Network, 2014.
Adapted from NEO Stroke Network (2010).

3.1 Stroke Warning Signs and Pre-hospital Care

The 5 Warning Signs of Stroke

Sudden onset of …

- Weakness or numbness
- Speech disturbances
- Unexplained dizziness
- Visual changes
- Sudden severe headache of unknown cause

Hyperacute stroke are strokes where there is a very recent onset of symptoms (i.e., within minutes or hours).

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.

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.

Paramedic Prompt Card For Acute Stroke Protocol

Indications for Patient Redirect or Transport to a Designated Stroke Centre Patients who present with a new onset of at least one of the following symptoms suggestive of the onset of an acute ischemic stroke:

- unilateral arm/leg weakness or drift
- slurred speech or inappropriate words or mute
- unilateral facial droop

AND

Can be transported to arrive at a Designated Stroke Centre within 3.5 hours of a clearly determined time of symptom onset or the time the patient was “last seen in a usual state of health”.

Exclusions for Patient Redirect to a Designated Stroke Centre

Any of the following conditions exclude a patient from being redirected to a Designated Stroke Centre:

- CTAS Level 1 and/or uncorrected airway, breathing or circulation problem
- Patients whose symptoms have resolved prior to paramedic assessment
- Blood sugar < 3.0mmol/l
- Seizure at onset of symptoms or observed by paramedic
- Glasgow Coma Scale <10
- Terminally ill or palliative care patient

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

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.*

Stroke Bypass Protocols/Direct Transport Protocols exist in each region whereby the EMS providers will bypass the local community hospital and transport the patient, up to 2 hours in the ambulance, to a centre providing specialized acute stroke treatment, and arrive within 3.5 hours from the time of stroke onset.

The 2013 *Canadian Best Practice Recommendations for Stroke Care* also emphasizes the need for rapid transport of acute stroke patients to appropriate facilities. However, there is an unfortunate lack of public awareness of stroke signs and symptoms, and still a lack of knowledge that stroke is an emergency. 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 acute stroke patients should be given at least **160mg of ASA immediately** as a one-time loading dose after brain imaging has excluded intracranial hemorrhage [Evidence Level A] (ESO, NZ, RCP, SIGN 13) (2013 Canadian Best Practice Recommendations for Stroke Care).

- For patients treated with tPA (tissue plasminogen activator) (tPA or Alteplase) (see section 3.3 below), ASA should be delayed until after the 24-hour post-thrombolysis scan has excluded intracranial hemorrhage.
- ASA (80-325 mg daily) should then be continued indefinitely or until an alternative antithrombotic regime is started.
- For dysphagic patients, ASA may be given by enteral tube or by rectal suppository.
- For patients already on ASA prior to ischemic stroke or TIA, clopidogrel may be considered as an alternative; if rapid action is required then a loading dose of 300mg of clopidogrel followed by a maintenance dose of 75mg/day (2013 Canadian Best Practice Recommendations for Stroke Care).

*Research has concluded that the administration of ASA within 48 hours of onset of presumed ischemic stroke **reduces the risk of early recurrent ischemic stroke** without a major risk of early hemorrhagic complications and improves long-term outcomes. This is why ASA is administered in the hyperacute phase of stroke (Cochrane Database Systematic Review, 2007). * Refer to hospital protocols and standing order sets to guide initial management*

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 tissue plasminogen activator (tPA or Alteplase).

What is tissue plasminogen activator (tPA or Alteplase)?

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 (Faaast FAQs for Nurses, page 7, Heart and Stroke Foundation of Ontario, 2007).

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

Eligible patients are those who can receive tPA within 4.5 hours of the onset of stroke symptoms, in accordance with criteria adapted from the *National Institute of Neurological Disorders and Stroke tPA Stroke Study* and *Third European Cooperative Acute Stroke Study (ECASS III)*. Beyond the 4.5 hour window, the risks of giving tPA outweigh the benefits.

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.

NOTE: The introduction of thrombolytic therapy has provided a proven treatment for acute ischemic stroke patients if given as soon as possible and within the time window. However, it is a high risk treatment that should only be given by personnel trained in its use, in a centre equipped to investigate and monitor patients appropriately.

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 scan imaging or MRI.

tPA is most often administered intravenously or sometimes intra-arterially directly to the site of the clot via catheter, allowing for a greater dose of the drug with fewer potential side effects.

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

Each centre will have standard order sets and protocols.

- Determine last seen normal time (less than 4.5 hours)
- 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 tPA

The following is from the 2013 *Canadian Best Practice Recommendations for Stroke Care*, and are designed to guide clinical decision making:

Treatment Inclusion Criteria

- Diagnosis of ischemic stroke causing measurable neurologic deficit in a patient who is 18 years of age or older.
  - For adolescents, decision to administer tPA should be based on clinical judgment, presenting symptoms, and patient age; and, if possible, consultation with a pediatric stroke specialist.
- Time from last known well (onset of stroke symptoms) less than 4.5 hours before tPA administration.

Exclusion Criteria

Historical

- History of intracranial hemorrhage in previous six months.
- Stroke or serious head or spinal trauma in the preceding three months.
- Recent major surgery, such as cardiac, thoracic, abdominal, or orthopedic.
- Arterial puncture at a non-compressible site in the previous seven days.
- Any other condition that could increase the risk of hemorrhage after tPA administration.

Clinical

- Symptoms suggestive of subarachnoid hemorrhage.
- Stroke symptoms due to another non-ischemic acute neurological condition such as a seizure with post-ictal Todd's paralysis or focal neurological signs due to severe hypo- or hyperglycemia.
- Hypertension refractory to antihypertensives such that target blood pressure less than 185/110 cannot be achieved.

Laboratory

- Blood glucose concentration below 2.7 mmol/L or above 22.2 mmol/L.
- Elevated activated partial-thromboplastin time.
- International Normalized Ratio greater than 1.7.
- Platelet count below 100,000 per cubic millimetre.

CT or MRI Findings

- Any hemorrhage on brain CT or MRI.
- CT showing early signs of extensive infarction, represented by a score of less than five on the Alberta Stroke Program Early CT Score [ASPECTS], or MRI showing an infarct volume greater than 150 cc on diffusion-weighted imaging.

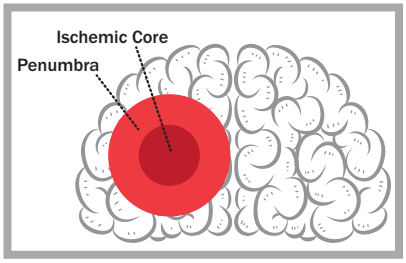
Why work quickly to determine if tPA is the appropriate treatment?

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, within a maximum of 60 minutes of arrival to the designated stroke centre. A rapid and coordinated emergency department response facilitates early diagnosis and treatment. The following **maximum target times** for emergency management of ischemic stroke have been established:

Door-to-triage	1 minute
Door-to stroke team notification	5 minutes
Door-to-CT scan 25 minutes	25 minutes
Door-to-needle	60 minutes

NOTE: Absolute and relative exclusion criteria for administration of tPA and the tPA Order Set are included in the Supplemental Modules: Hospital-Specific Chart Forms section (packages that are available at tPA administering sites)

Acute Ischemic Stroke receiving tPA

Vital signs (including temperature) should be assessed as follows after beginning tPA infusion:

- q 15 minutes for 2 hours
- q 30 minutes for 2 hours
- q 1 hour for 6 hours
- q 4 hours for 14 hours

(Black et al., 2012, p.9)

Intravenous Tissue Plasminogen Activator for Stroke

Nursing Monitoring and Interventions and Potential Complications	Clinical Implications
Monitoring: • Baseline Vital signs: blood pressure (BP) and heart rate (HR) • Baseline ECG and Oxygen saturation continuous monitoring • Baseline neurological assessment: using standardized stroke scale e.g., National Institutes of Health Stroke Scale (NIHSS) • Establish 2 intravenous sites • Initiating tPA is the priority, do not delay for other line insertion unless clearly indicated (e.g., Urinary catheter, NG feeding tube)	• If SBP greater than 185 mmHg or DBP greater than 110 mmHg for 2 consecutive readings greater than 5 minutes apart, notify *Physician* and be prepared to treat with anti-hypertensive (IV Labetolol, nitroglycerin paste, or IV Hydralazine). If these measures do not decrease the BP, tPA will not be given • Glasgow Coma Scale was not designed to capture stroke deficits and deterioration • Cannot administer any other medication through tPA infusion line • Inserting these devices should not be considered standard treatment and should not delay the start of tPA infusion. They may be clinically necessary in some situations (e.g., urinary catheter in elderly male with diabetes and nocturia) • Increase the frequency of BP measurements if SBP greater than 180 mmHg or DBP greater than 105 mmHg • Headache, decreased level of consciousness, or worsening neurological deficit may be symptoms of hemorrhage into stroke. Notify *Physician*.
During tPA Infusion (1 hour): • Vital signs (BP and HR) q 15 minutes • Neurological assessment q 15 minutes	
Post tPA Infusion (23 hours): • Vital signs (BP and HR) q 15 minutes X 1 hour then q 30 minutes X 6 hours then q 1 hour X 16 hours • Neurological assessment q 1 hour X 23 hours	
Systemic bleeding: • No antiplatelets, intramuscular injections, or non-compressible invasive lines for 24 hours	• If bleeding occurs, notify *Physician*; may need to stop tPA infusion (if still infusing) • If bleeding occurs in compressible area, may require extended pressure to area • If significant bleeding occurs, patient may require blood products (e.g., fresh frozen plasma, platelets, and/or packed red blood cells)
Allergic reaction: • Monitor for cough, wheezing, or angioedema	• Angioedema of the tongue can potentially cause airway obstruction and may require intubation. Treatment may also include corticosteroids and antihistamines
Bedrest X 24 hours	• To prevent complications secondary to falls • Turn patient q 1 – 2 hours
Nutrition	• NPO

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 (ex. NIHSS/CNS)
- If suspected, stop tPA and notify *Physician*
- Obtain CT scan and coagulation work-up

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

2013 Canadian Best Practice Recommendations for Stroke Care (3.4.1) with respect to angioedema management state: For tPA-induced angioedema, discontinue the tPA infusion if it is still running, obtain assistance for airway management if required, and give I.V.:

- *Hydrocortisone 100 mg*
- *Diphenhydramine 50 mg*
- *Ranitidine 50 mg*

**The use of epinephrine should be weighed against the risk of sudden HTN and the risk of intracranial hemorrhage [Evidence Level C].*

3.5 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.

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)

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 and oxygen applied if oxygen saturation is less than 94% (American Heart Association/ American Stroke Association, 2013). 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*.

3.6 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 Glascow Coma Scale is highly predictive of death (Martin, 2013).

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; Treat BP greater than 180 mmHg and high intracranial pressure (ICP) with medications such as labetolol (Martin, C., 2013)

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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