Chapter 2

ACUTE AND CRITICAL STROKE CARE

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LEARNING OUTCOMES

After completing this chapter, you will be able to:

1. Identify the global impact of stroke
2. Describe the role of various stroke assessment tools and identify those used in your institution.
3. Describe the pathophysiology of both ischaemic and haemorrhagic stroke
4. Discuss the role of brain imaging in stroke assessment and the types of scans that may be performed
5. Discuss the immediate and ongoing care of a patient with stroke

ABBREVIATIONS

ABCD2	Arm, Blood pressure, Clinical features, duration and diabetes TIA score
ACT-FAST	Arm, chat, tap large vessel occlusion screening tool
AF	Atrial Fibrillation
AMP	Adenosine monophosphate
aSAH	Aneurysmal subarachnoid haemorrhage
ASPECTS	Alberta Stroke Programme Early CT Score
ATP	Adenosine triphosphate
BGL	Blood glucose level
BP	Blood pressure
CAA	Cerebral amyloid angiopathy
CBF	Cerebral blood flow
CPSS	Cincinnati prehospital stroke scale
CSF	Cerebrospinal fluid

CT	Computerised tomography
DOAC	Direct oral anticoagulant
ECG	Electrocardiogram
ECHO	Echocardiogram
EVT	Endovascular thrombectomy
GCS	Glasgow coma scale
HT	Hypertension
IPH	Intraparenchymal haemorrhage
LAMS	Los Angeles motor score
LAPSS	Los Angeles prehospital stroke screen
LVO	Large vessel occlusion
MASS	Melbourne ambulance stroke screen
MRI	Magnetic resonance imaging
NIHSS	National Institutes of Health Stroke Score
NMDA	N-methyl-D-aspartate
PES	Pharyngeal electrical stimulation
PFO	Patent foramen ovale
RACE	Rapid arterial occlusion evaluation scale
SAH	Subarachnoid haemorrhage
sICH	Symptomatic intracerebral haemorrhage
TIA	Transient Ischaemic Attack
VAN	Vision, aphasia, neglect score
VTE	Venous thromboembolism

INTRODUCTION

Stroke is a leading cause of death and disability globally. More than 15 million people have a stroke each year, over half of whom are aged 70 years or younger.(1) Internationally, the long-term cost of stroke care is immense, more than USD $890 billion per year; unchecked this number will exceed $1 trillion by the year 2050.(1) In the USA, only approximately one-quarter of stroke related health care costs come from the initial hospitalization, demonstrating the long-term cost of stroke disability.(2) These costs unfortunately particularly place undue financial stress on low- and middle-income countries who have the highest reported rates of stroke in the world. (1)

A stroke is a vascular condition that is caused by the sudden interruption of blood to the brain, spinal cord or retina, either through a blocked artery (ischaemic stroke) or a burst artery (haemorrhagic stroke).(3) This disruption of blood flow immediately reduces the supply of oxygen and nutrients to the brain, causing tissue ischaemia and death.(3) Ischaemic strokes account for approximately 80% of all strokes, while 20% are haemorrhagic. This chapter focuses on strokes occurring in the brain.

A stroke is a time-critical medical emergency, approximately 1.9 million neurons die each minute in a large vessel ischaemic stroke.(4) While treatment options exist for both ischaemic and haemorrhagic strokes, they are limited in both accessibility and timeframe eligibility, meaning not every hospital can treat patients with stroke and not every patient meets treatment criteria. It is essential that patients who develop stroke symptoms seek urgent medical care at stroke centres, and that nurses are knowledgeable and skilled in stroke assessment, management, and treatment to maximise patient outcomes.

TRANSIENT ISCHAEMIC ATTACK (TIA)

While traditionally defined as focal neurological symptoms that resolve completely within 24-hours from onset, approximately 30-40% of patients with a 'TIA', are found to have had a stroke on subsequent brain imaging. This means that time alone is not sufficient to differentiate a TIA from a stroke. All patients presenting with a TIA should therefore undergo routine imaging, preferably magnetic resonance imaging (MRI) which is more sensitive than a computed tomography (CT) scan, making MRI more likely to detect smaller strokes, especially early after symptom onset.(5, 6)
A TIA is a major risk factor for future stroke. While reported rates vary, about 40% of patients who experience a TIA will have a stroke in the subsequent three months, with the greatest risk being in the first 48-hours post TIA.(5, 7, 8)
Identifying the cause of the TIA and commencing targeted secondary prevention to reduce the risk of stroke is an important part of the assessment and management process.

ISCHAEMIC STROKE

Pathophysiology

The brain makes up only about 2% of the body's total weight, yet it has a high metabolic demand, requiring approximately 20% of total cardiac output. Unlike other organs, the brain is not able to store large quantities of glucose, making it dependent on a continuous blood supply for homeostasis.(9, 10) An ischaemic stroke is caused by an occlusion of an artery in, or leading to, the brain. These occlusions prevent the flow of blood, and therefore oxygen and glucose, to tissues which triggers the ischaemic cascade and results in cellular death.(10-12)

Tissue hypoxia causes cells to switch to anaerobic metabolism, which reduces the amount of ATP available for energy and creates lactic acid. Normal ion-transport pumps that rely on ATP fail. As a result, cells depolarize and allow an influx of calcium. This triggers the release of intracellular calcium stores, worsening the hypercalcaemic state. Glutamate is released, and receptors such as AMP and NMDA are stimulated. This perpetuates the calcium influx and generates excitotoxicity and the production of free radicals and other toxic enzymes and chemicals within the cell. Mitochondria begin to break down due to the toxic intracellular environment. Finally, processes that cause cellular death, including apoptosis, are triggered.(11-14)

As the cell dies, intracellular toxins are released into the extracellular space, damaging nearby neurons and causing a breakdown of the blood-brain barrier. Large molecules, such as albumin, that normally cannot cross the blood-brain barrier, now cross over, pulling water with them, causing vasogenic cerebral oedema within the infarcted area. Triggered by the necrotic process, phagocytic cells such as microglia and macrophages enter the area and over time scavenge the

dead tissue. This leaves a cavity in the brain which eventually fills with cerebrospinal fluid (CSF), resulting in a hypodense (dark) area on CT referred to as encephalomalacia.(11-14)

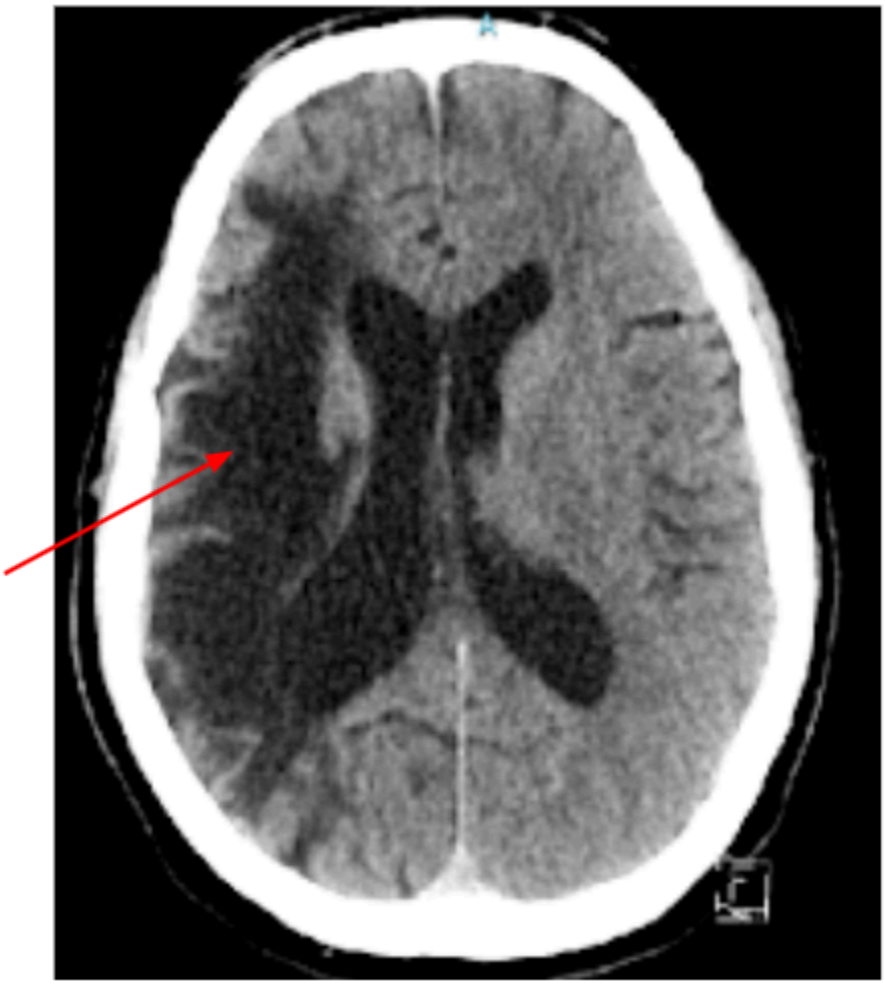

Figure 1. A CT scan showing an ischaemic stroke in the right middle cerebral artery distribution. The arrow shows infarcted tissue which has been scavenged and is now fluid filled, resulting in the hypodense (darker) appearance of the area as compared to the denser surrounding tissue. This is referred to as encephalomalacia. Image reprinted with permission from NET SMART (www.learnstroke.com), Health Outcomes Institute, LLC.

The rate of tissue death during a stroke is variable. There are three main zones within the stroke area – the necrotic core, the ischaemic penumbra and benign oligemia. The necrotic core is immediately proximal to the occlusion and dies quickly due to the sudden and severe reduction in cerebral blood flow (CBF) to <10-12ml/100g brain tissue/min; (normal CBF in healthy brain tissue is 50-80ml/100g/min).(15)

Spreading out from the necrotic core is an area of hypoxic tissue called the ischaemic penumbra. The CBF in the penumbra is low enough (<20ml/100g/min) to cause significant cellular dysfunction producing stroke symptoms, but is not

low enough to cause immediate cell death. This means the penumbral tissue is potentially salvageable, but only if blood flow is restored quickly, before the area begins to infarct. Lastly benign oligemia has reduced CBF (<35ml/100g/min), but the cells still largely function normally and don't contribute to the clinical presentation of the patient.(16, 17)

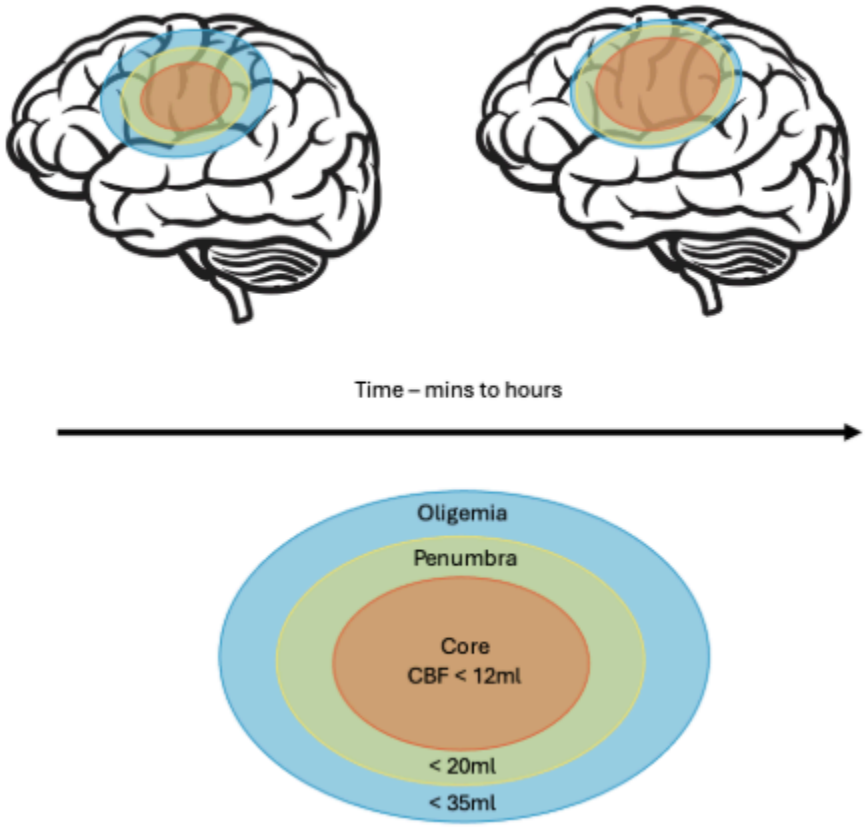

Figure 2. The penumbra concept – cerebral blood flow (CBF) is variable across the stroke area. Tissue within the necrotic core zone will die quickly due to a severe and abrupt reduction in blood flow. Tissue within the penumbra may temporarily remain viable due to collateral circulation, but unless the occlusion is quickly removed and CBF restored, the penumbra will begin to infarct. The border areas of oligemic tissue begin to fail, turning into smaller zones of penumbra as time passes. Without urgent revascularization, the final infarction size increases. *Image produced by S.Coote 2025.*

The aim of ischemic stroke treatments is to save the penumbra while it remains viable, and prevent recruitment of benign oligemic tissue into the penumbral and infarct zones.(16) Collateral circulation can provide a limited supply of blood to penumbral tissue to maintain viability for a period, but this is temporary at best. Collateral vessels are small, fragile vessels that are designed as a rescue or backup pathway only. The extensiveness and longevity of the collateral circulation varies significantly between individuals; some patients have an extensive collateral network, while others have a collateral circulation that is insufficient to hold off infarction. Some collaterals will fail quickly, causing a rapid progression of the stroke, while others have more functional collaterals, resulting in a slower progression of infarction.(13, 16, 17) All of

this is unknown when a patient first presents with stroke symptoms, so reperfusion therapies must be given without delay. Left untreated, penumbral tissue will infarct once collaterals fail, turning the penumbra into necrotic core, and increasing the final size of the stroke.(16, 17)

Mechanism

Ischaemic stroke occlusions are usually either thromboembolic in nature, which are commonly associated with atherosclerosis (particularly in the internal carotid artery), or result from cardioembolism, most notably atrial fibrillation (AF).(13) Other causes can include small vessel disease, or unusual causes of stroke such as arterial dissection and hematological disorders.(13)

In the case of atherosclerosis, long-term hypertension (HT), diabetes, smoking and hypercholesterolaemia lead to endothelial damage and plaque development, narrowing the arterial lumen. Depending on the degree of stenosis, haemodynamic changes to blood pressure (BP) can reduce flow to critical levels distal to the stenosis, resulting in a stroke.(18, 19) Changes to flow dynamics past the obstruction can further damage the vessel wall and contribute to plaque ulceration and rupture. Once the plaque cap is ruptured, platelet and fibrin aggregation occur with the resulting thrombus occluding the artery and causing a stroke.(9) Because atherosclerosis is a progressive disease that develops over time, patients with atherosclerosis are more likely to have developed a collateral circulation network.

A cardioembolic stroke results from emboli that have developed in, or moved through, the heart. Cardioembolic strokes tend to involve the large arteries in the brain and are more likely to be a spontaneous event, meaning the brain has not developed an extensive collateral circulation that may be present with atherosclerotic disease or other chronic risk factors such as HT and smoking. As a result, cardioembolic strokes tend to produce severe neurological deficits and result in poorer patient outcomes.(20) AF is the most common form of cardioembolic stroke and accounts for approximately 20-25% of all ischaemic strokes.(21) Other cardioembolic causes can include valvular heart disease, left ventricular dyskinesis, acute myocardial infarction and even venous thromboembolism (VTE) with a venous to arterial embolic transfer through an intracardiac right-to-left shunt, from an atrial septal defect and/or patent foramen ovale (PFO).(13, 21)

Lacunar strokes develop from small vessel disease, and account for approximately 25% of ischaemic strokes.(22) Lacunar strokes are caused by an occlusion of the tiny perforator vessels off larger intracranial arteries. Given the size of these vessels (≤ 0.5mm), even a small occlusion can block flow. Despite the comparative size of a lacunar stroke to other strokes, small vessel strokes can cause similar rates of disability.(22, 23) Lacunar strokes were previously thought to be exclusively caused by lipohyalinosis-related inflammatory changes from poorly controlled stroke risk factors (e.g., HT, diabetes, hypercholesterolaemia, smoking), but new evidence suggests that microembolic events and even microatheromas may occlude these perforating vessels at their proximal root. Small vessel disease/lacunar stroke is closely associated with the development of cognitive impairment and vascular dementia, due to the often-cumulative occurrence of strokes in association with poorly controlled risk factors.(23)

Less common causes of ischaemic stroke (stroke of unusual cause) include:

- Arterial dissection – a tear within the intimal layer of an artery with a resultant haematoma within the wall of the vessel (intramural thrombus). Often caused by trauma, stroke from an arterial dissection is usually associated with younger adults. However, the degree of trauma does not need to be severe, and has been reported in cases following heavy lifting, sneezing and falls.(13, 24)
- Haematological disorders – such as sickle cell disease, antiphospholipid syndrome or other prothrombotic or hypercoagulable conditions including pregnancy and malignancies that predispose to thrombus formation.(13)
- Cerebral vasculitis – inflammation of the arterial wall can reduce lumen diameter and accelerate rates of atherosclerosis, leading to thrombus formation.(13, 25)

HAEMORRHAGIC STROKE

Intracerebral Haemorrhage (also called Intraparenchymal Haemorrhage)

An intracerebral haemorrhage (ICH) occurs when there is bleeding into the brain tissue (the parenchyma) from an arterial rupture. The most common cause of an ICH is uncontrolled HT. The small perforating arteries - which arise from the main feeder arteries off the circle of Willis - are the most vulnerable to rupture as they receive the highest in-flow pressures. Hypertensive ICH is therefore classically found in the subcortical regions of the brain and in areas where small perforating arteries arise from large arteries.(26-28)

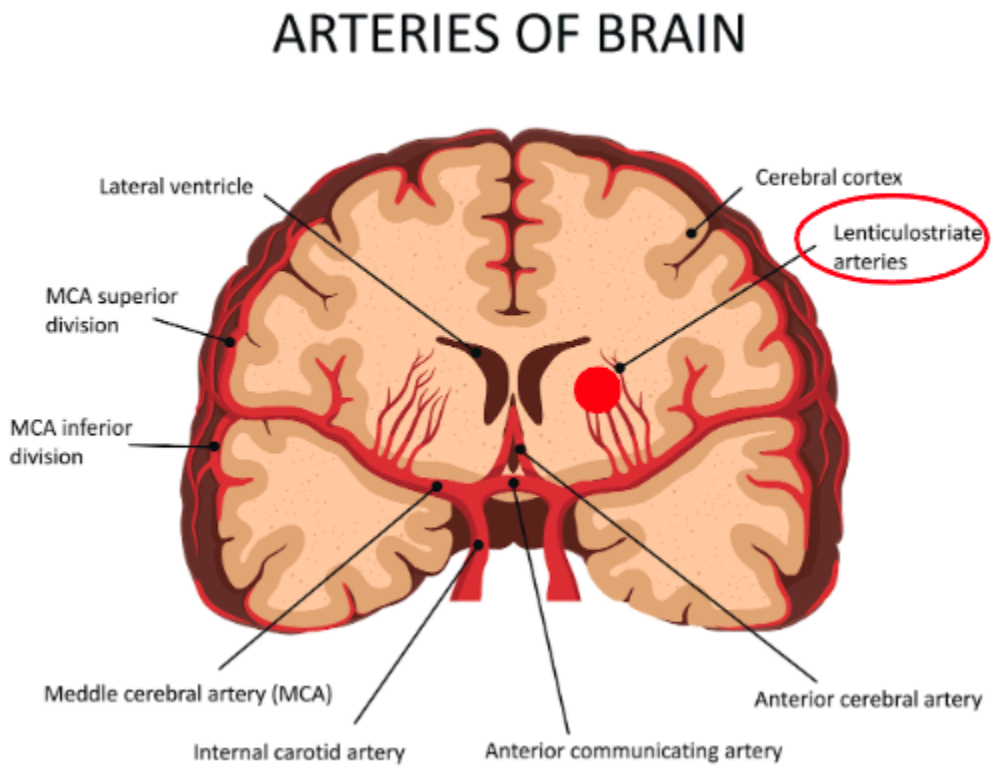

Figure 3. Tiny perforating lenticulostriate arteries arising from the middle cerebral arteries. These small vessels are a common site of a HT ICH. Their small caliber and reduced smooth muscle make them vulnerable to HT.

Source: logika600/Shutterstock.com

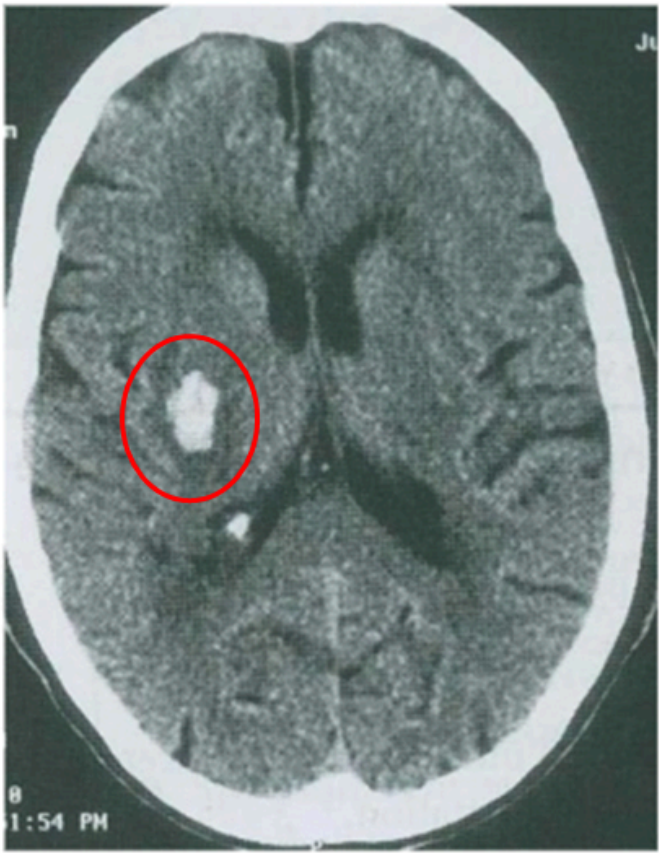

Figure 4. An axial CT scan of the brain showing a right basal ganglia (subcortical) intraparenchymal haemorrhage, likely due to HT. Image reprinted with permission from NET SMART (www.learnstroke.com), Health Outcomes Institute, LLC.

Cerebral amyloid angiopathy (CAA) is another common cause of an ICH. β-amyloid proteins can line cerebral vessels, weakening them and making them more prone to rupture.(26, 28) CAA is associated with older patients and Alzheimer's disease but also may be genetic.(28) CAA classically presents as a haemorrhage in the lobar and cortical areas of the brain and may have some subarachnoid involvement. CAA bleeds vary significantly, ranging from larger lobar bleeds through to small 'ribbons' on the brain's cortical surface. It's important that clinicians interrogate all brain imaging from the base of skull through to the vertex (top) of the brain so as not to miss subtle signs of blood.(26, 28, 29)

Less common causes of an ICH include ruptured vascular formations and aneurysms, bleeding disorders including therapeutic anticoagulation, trauma, tumors, haemorrhagic conversion of an ischaemic stroke, vasculitis, and alcohol and drug use (especially cocaine and methamphetamine).

Haematoma expansion is a common cause of early deterioration in patients with an ICH and is associated with increased mortality and morbidity rates. Up to 40% of patients will have a haematoma expansion between 33% to 50% of the initial volume (27), while up to 70% of patients will have some degree of expansion in the first 24-hours, often in the first hour, making prevention of haematoma expansion a time critical treatment target in ICH management.(26, 27)
Tissue injury in an ICH occurs through numerous mechanisms. The initial injury comes from the space occupying effect of the haematoma and the associated cerebral oedema, as it exerts a compression force on the surrounding brain tissue and raises intracranial pressure. If the haematoma and oedema volume is large enough, a midline shift may occur. If blood has expanded into the ventricular system, hydrocephalus can develop.(27, 29) Secondary injury develops over days to weeks from the toxicity of the iron and blood components within the tissues, inflammatory processes and oxidative stress.(29) Lastly, depending on the size of the bleed, ischaemia to the tissues distal to the rupture may occur, creating a secondary ischaemic injury.(27)

The impact of an ICH varies depending on the size and location of the bleed. Even small haematomas can produce life-threatening injuries if located in critical areas, such as in or around the brainstem.(26, 27, 29) While there is no definitive set of symptoms that can clinically distinguish an ICH from an ischaemic stroke, haemorrhages are more commonly associated with headaches, vomiting, altered levels of consciousness, pupillary changes, haemodynamic instability and electrocardiograph (ECG) changes, in addition to the classic stroke hemi-motor and hemi-sensory changes.(27)

Subarachnoid Haemorrhage

A subarachnoid haemorrhage (SAH) is bleeding within the subarachnoid space - between the arachnoid mater and the pia mater. While SAH makes up only about 5% of strokes each year, it has a very high mortality rate, with over half the patients dying within the first six months, and one-quarter within the first 24-hours. Many patients with a SAH die before reaching the hospital.(30)

The most common trigger of a SAH is a ruptured aneurysm.(30, 31) Causes of aneurysmal SAH (aSAH) include HT causing arterial mechanical stress particularly at arterial bifurcations, smoking, connective tissue disorders, mycotic aneurysms and genetic familial mechanisms.(32) Vascular formations, most commonly arteriovenous malformations (AVM) are another cause of a SAH. Typically congenital, AVMs occur when an abnormal tangle of blood vessels causes high pressured arterial blood to feed directly into thin-walled veins. This weakens the veins and makes them prone to rupture.(33)

In most patients, there is a biphasic mechanism of injury with aSAH – early brain injury and delayed cerebral ischaemia. The exact pathophysiology of both mechanisms is not entirely understood, but early brain injury occurs from the time of the rupture and lasts for the first two to three days, while delayed cerebral ischaemia due to vasospasm peaks around a 5 to 7 days after rupture.(31, 34)

Blood within the subarachnoid space irritates the meninges, resulting in a classic presentation that includes intense/unusual headache, photophobia and neck stiffness. It also causes intracranial pressure to rise, reducing CBF and cerebral perfusion pressure and creating 'haemorrhagic ischaemia'. This initial ischaemia can trigger the ischaemic cascade and thus cellular death. Acute vasospasm can further reduce CBF, worsening the ischaemia.(31, 34) Blood in the subarachnoid space can block the arachnoid villi, reducing the reabsorption of CSF. The resultant build-up of CSF causes hydrocephalus, further increasing intracranial pressure and reducing CBF. Other mechanisms of injury include altered autoregulation, increased permeability of the blood-brain barrier, diffuse cerebral oedema - both vasogenic (extracellular) and cytotoxic (intracellular), neuronal toxicity from the iron and blood components, neuroinflammation, micro-thrombi formation and oxidative cascades.(30, 31, 34) Longer term, ongoing vasospasm, cortical spreading depolarization and cerebral oedema contribute to delayed cerebral ischaemia.(30)

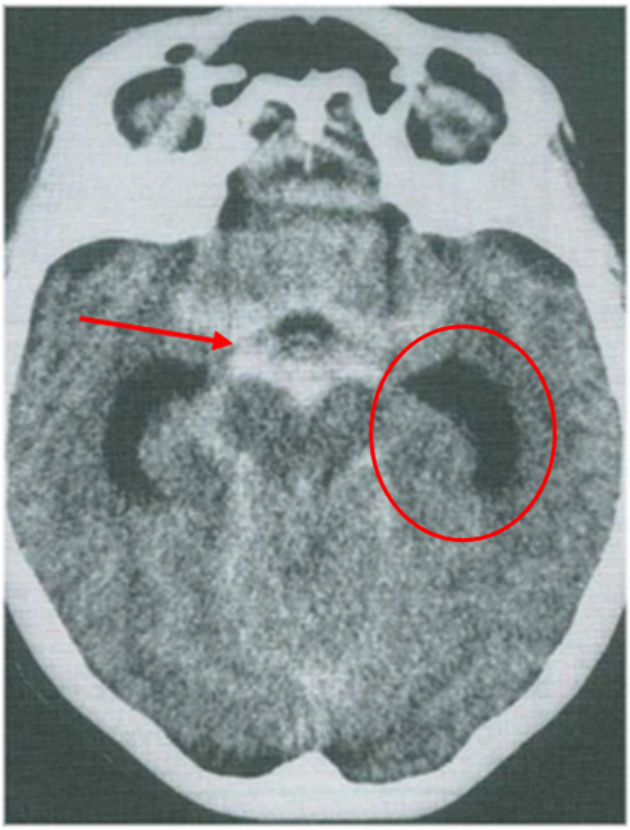

Figure 5. An axial CT scan showing a diffuse subarachnoid haemorrhage (SAH) (arrow) with enlarged temporal horns of the lateral ventricles (circle) indicating the presence of hydrocephalus. Reprinted with permission from NET SMART (www.learnstroke.com), Health Outcomes Institute, LLC.

CASE STUDY – Part 1

Arif is a 65-year man who lives at home with his wife. He has a past medical history of hypertension, hypercholesterolaemia, type II diabetes and atrial fibrillation. He is prescribed perindopril, atorvastatin, metformin and apixaban, however he does not always remember to take his tablets. He has issues with his feet due to his diabetes, and walks with a single point stick, but is otherwise independent in his personal and community-based activities of daily living.

At 7.30am he was in the kitchen making breakfast with his wife when he was witnessed to slump to the ground (no head strike occurred) with right sided weakness. His wife immediately attended to him and noted that he was not speaking properly. She tried to lift him up to the couch but couldn't, so she went to her neighbours for help. When they arrived, they called for an ambulance.

- What are your initial differential diagnoses for Arif?
- Based on his medical and medication history, what would be the possible causes of stroke for Arif?
- What are the immediate priorities for Arif's care?

PATIENT ASSESSMENT

The history of the stroke event, the patient's presenting symptoms, and brain imaging findings are all used to make a stroke diagnosis. Stroke symptoms can be some of the most complex and nuanced in medicine. While typically unilateral in nature, stroke symptoms will vary depending on the artery, and therefore brain territory, impacted by the stroke.(35) There are several different stroke tools in use around the world, the application of these can vary based on the stage of the stroke and stroke type. Nurses should therefore be skilled in assessing patients using a variety of assessment tools and understand their relevance and limitations.(35)

Prehospital Stroke Assessment Tools

In the prehospital setting, two main types of tools are used:

1. General stroke tools which assess whether the patient has symptoms consistent with a stroke (based on the most common stroke symptoms)
2. Stroke severity tools used to identify patients with severe stroke symptoms, which could indicate a large vessel occlusion (LVO).(36, 37)

Given the nature of emergency ambulance work, and the small proportion of stroke in daily paramedic workloads, it is impractical to expect paramedics to maintain proficiency with lengthy and complex stroke assessment tools. Therefore, general stroke tools typically assess only the most common stroke symptoms – usually a combination of facial droop, arm/hand weakness and speech alterations (dysphasia and/or dysarthria).(36, 38) These symptoms originate from middle cerebral artery strokes, the artery most pathologically impacted by ischaemic stroke.(39)

Examples of general stroke assessment tools include the Cincinnati Prehospital Stroke Scale (CPSS) and the Melbourne Ambulance Stroke Screen (MASS).(36, 40) These scales have the benefit of being fast and easy to administer with little training required, with high sensitivities and reasonable specificities for stroke diagnosis.(36, 40) However, due to the limited nature of their assessment items, they can miss strokes originating from other arteries, particularly from the posterior circulation, making their overall use somewhat limited.

Strokes caused by a LVO tend to present with more severe symptoms. Stroke severity tools therefore usually measure the magnitude of the symptoms, rather than yes/no to symptom presence like general stroke tools. These tools assess for 'cortical signs', namely deficits arising from areas on the cerebral cortex, including aphasia, vision loss, abnormal gaze and neglect. Recognising this cohort of patients in the prehospital setting allows paramedics to directly transport patients to comprehensive or endovascular thrombectomy (EVT) capable hospitals for treatment, bypassing smaller or primary hospitals that lack EVT capabilities. This can potentially expedite treatment by more than two hours, improving patient outcomes.(41, 42)

Examples of commonly used stroke severity-based scales are:

- The ACT-FAST algorithm – assesses arm drift, language and neglect (43)
- VAN screening tool – assesses vision, language and neglect (44)
- RACE (Rapid Arterial Occlusion Evaluation) – assesses facial droop, arm and leg drift, gaze deviation, aphasia and agnosia (45)

Not all patients arrive by ambulance, and these prehospital tools may be used by triage nurses to quickly identify patients with stroke symptoms and activate appropriate stroke protocols. By understanding the use and the implication of these assessment tools, nurses contribute to the streamlining of hospital pathways to reduce door to CT, door to needle and door to arterial puncture times.

Glasgow Coma Scale - GCS

The Glasgow Coma Scale (GCS) was designed to assess and prognosticate altered consciousness and coma.(46) The GCS assesses three items: eye opening, motor response and verbal response. As it assesses overall brain function, it asks the clinician to only score the patient's 'best' response, ignoring unilateral deficits.(47) This means a patient with a dense hemiparesis will not lose points on their motor assessment if they can obey commands on their unaffected side. Conversely, an aphasic patient who is conscious and alert will lose four points, scoring one for 'no verbal response', giving them a best possible score of 11/15 points. This makes the GCS an ineffective tool for general stroke assessment.

National Institutes of Health Stroke Score - NIHSS

The National Institutes of Health Stroke Scale (NIHSS) is an internationally recognised and validated stroke assessment tool.(48) It is an 11-point scale that features many of the aspects of the previously mentioned tools, including elements of

the GCS, general stroke tools and stroke severity tools.(48) Designed initially for use in clinical research trials, it objectively measures stroke symptoms, removing subjective interpretations found in standard neurological charts such as 'mild' or 'moderate' weakness.(49) Unlike many of the other stroke scores, the NIHSS requires users to undergo certification prior to use to maximise accuracy and inter-rater reliability.(48)

While quite comprehensive, the NIHSS is not without its limitations. There is a bias in the assessment towards patients with strokes in the anterior circulation, due to a very limited number of assessment items associated exclusively with posterior circulation stroke symptoms.(48, 50) There is also a scoring bias for patients presenting with left hemispherical strokes due to the number of items assessing language. Language centres are most commonly found in the left brain, even in left-handed individuals, if impacted by stroke, the NIHSS scores a maximum of seven language-associated points. Conversely, patients with a right brain stroke will not have a language deficit, but instead will more likely display neglect, which scores only a maximum of two points. Therefore, NIHSS scores for left hemisphere strokes will have a higher points value than right-sided strokes, even when an equivalent amount of brain infarction has occurred.(48-50)

Assessment Item	Score
1a. Level of consciousness	0 – Alert, keenly responsive 1 – Not alert, but rousable by minor stimulation 2 – Not alert, requires repeated stimulation 3 – Unresponsive or responds only with reflex
1b. Level of consciousness questions *What is your age?* *What is the month?*	0 – Answers both questions correctly 1 – Answers one question correctly 2 – Answers neither questions correctly
1c. Level of consciousness commands *Open and close your eyes* *Grip and release your hand*	0 – Performs both tasks correctly 1 – Performs one task correctly 2 – Performs neither task correctly
2. Best gaze	0 – Normal 1 – Partial gaze palsy 2 – Forced deviation
3. Visual	0 – No vision lost 1 – Partial hemianopia 2 – Complete hemianopia 3 – Bilateral hemianopia
4. Facial palsy	0 – Normal symmetrical movements 1 – Minor paralysis 2 – Partial paralysis 3 – Complete paralysis of one or both sides
5. Motor arm *a. Left arm* *b. Right arm*	0 – No drift 1 – Drift 2 – Some effort against gravity 3 – No effort against gravity 4 – No movement
6. Motor leg *a. Left arm* *b. Right arm*	0 – No drift 1 – Drift 2 – Some effort against gravity 3 – No effort against gravity 4 – No movement
7. Limb ataxia	0 – Absent 1 – Present in one limb 2 – Present in two limbs
8. Sensory	0 – Normal, no sensory loss 1 – Mild to moderate sensory loss 2 – Severe to total sensory loss
9. Best language	0 – Normal, no aphasia 1 – Mild to moderate aphasia 2 – Severe aphasia 3 – Mute, global aphasia

10. Dysarthria	0 – Normal 1 – Mild to moderate dysarthria 2 – Severe dysarthria
11. Extinction and neglect	0 – No abnormality 1 – Visual, tactile, auditory, spatial or personal inattention 2 – Profound hemi-inattention or extinction

Figure 6. The National Institutes of Health Stroke Scale (NIHSS). An internationally validated and recognised tool used in the assessment of patients with stroke. Stroke severity increases with the total score. Mild strokes = 0-4, moderate strokes = 5-15, moderate-severe strokes = 16-20, severe strokes = 21 and above.

Patients arriving at hospital should have an NIHSS performed to establish a baseline and to monitor progress, however, the baseline score alone should not be used to determine eligibility for reperfusion treatments. Instead, clinicians should assess whether the patient is disabled by their symptoms, compared to their usual pre-stroke status. Nurses are best prepared to make this assessment and determine the impact a disability could have on the patient's ability to manage their usual activities of daily living, and should advocate for reperfusion treatments as appropriate.

It should also be noted that because the NIHSS does not capture the complete constellation of stroke symptoms, some patients may score a zero on the NIHSS despite having new stroke-related disability.(51) As an example, the NIHSS assesses ataxia (uncoordinated movements) in the arms and legs, but it does not assess truncal ataxia or the patient's gait. This means a patient may be severely disabled, unable to sit or walk without assistance, yet presents with an NIHSS score of zero. These symptoms may also not be apparent while the patient is lying on the ambulance stretcher or hospital bed. Similarly, sudden onset dysphagia or apraxia are not scored on the NIHSS and may result in severe disabilities impacting quality of life long-term.

Assessment tools can therefore be insufficient when used in isolation and should be used and interpreted in the context of the complete clinical picture.(37) Nurses performing the NIHSS should be aware of these assessment gaps and perform additional neurological assessments as required so that disabilities are not overlooked.(50)

ABCD2 Score

The ABCD2 score was developed to determine the imminent risk of stroke in patients who present with a TIA. Patients at high risk (scores ≥ 4 points) may be admitted to hospital for workup and investigation, with prioritisation for outpatient clinic follow-up, while patients at low risk (scores 0-3) may be safe to discharge with urgent tests and follow up performed by their local general practitioners in the following few days.(8, 13)

The ABCD2 comprises several key stroke risk factors, including HT and diabetes, but it does not consider other important factors such as a history of AF, atherosclerosis or previous stroke.(52) Because of this, not all clinicians advocate the use of the ABCD2 score and instead rely on clinical acumen and the overall history of the patient to guide admission and investigations.(52)

Clinical criteria		Score
Age	≥ 60 years	1
	< 60 years	0
Blood Pressure	≥ 140/90	1
	< 140/90	0
Clinical features	Unilateral weakness	2
	Speech disturbance without weakness	1
	Other	0
Duration	≥ 60 minutes	2
	10-59 minutes	1
	< 10 minutes	0
Diabetes	Yes	1
	No	0

Figure 7. The ABCD2 score used to assess risk factors for imminent stroke after a transient ischaemic attack (TIA).

CASE STUDY – Part 2

The paramedics arrive and assess Arif. His BP is 175/108 mmHg, heart rate 87 in a controlled AF, respiratory rate 18 breaths per minute, oxygen saturation 98% on room air. He is afebrile and has a blood glucose level of 13.4 mmol/L (241 mg/dL).

He is presenting with right sided weakness and altered speech. He is not talking much, and what he is saying is gibberish and slurred. The paramedics determine Arif is likely having a stroke.

- What would be a good assessment tool for the paramedics to initially use and why?
- What is the value in using a stroke severity assessment tool in the prehospital setting? How would it benefit Arif's overall stroke management?
- What assessment tools are used in your workplace? Have you used them in your practice?

BRAIN IMAGING

Stroke is primarily a clinical diagnosis that is made by localizing the symptom pattern to a specific arterial territory in the brain. However, the type of stroke – ischaemic vs. haemorrhagic – is diagnosed by imaging the brain and must be performed before any definitive treatment can begin. Either a CT scan or a MRI can be used, but due to the limited availability of MRI globally, cost, and the need for thorough MRI safety checks, which can be difficult in patients with aphasia, CT is more commonly used.(53)

Non-contrast CT scans are exquisitely sensitive to the presence of blood, allowing them to be confidently used to differentiate ICH from ischaemic stroke for thrombolysis decision making.(53, 54) While haemorrhage is visible acutely on CT, ischaemic changes take longer to be seen. The first signs of ischaemic stroke ('early ischaemic changes') are usually

loss of the differentiation between the grey and white matter and swelling around nearby structures like the folds of the cerebral cortex. These changes occur because of cerebral oedema and take on average six to eight hours to become visible.(55) This means in the first few hours of an ischaemic stroke, the CT scan should be normal, making the diagnosis of ischaemic stroke solely a diagnosis based on the clinical examination.(53, 54)

If more established signs of ischaemia are present, it can mean that the onset time may be inaccurate, and the stroke started before the patient realized. Established changes can also be present in patients with poor collateral circulation, causing the tissue to infarct faster than average. A non-contrast CT can also show a 'hyperdense artery' sign – an area within a cerebral artery that is radiologically denser than the surrounding tissue. This sign is highly suggestive of an intraluminal thrombus and can indicate a large vessel occlusion needing thrombectomy.(56)

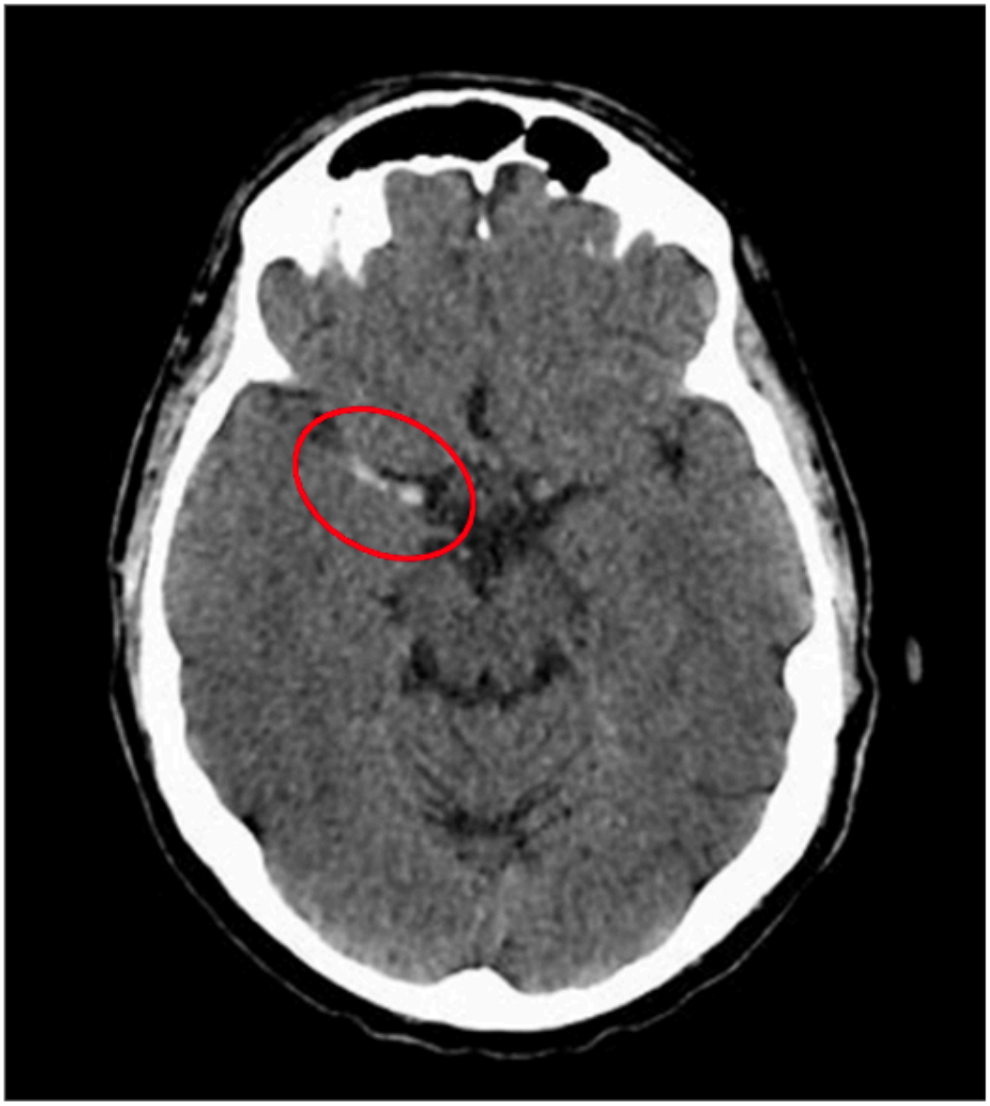

Figure 8. A hyperdense artery sign (circled) in the proximal right middle cerebral artery. This sign indicates a likely thrombus present in the artery. As the thrombus is denser than the surrounding blood and tissue, it becomes hyperdense (brighter white) on CT. A hyperdense artery can indicate the presence of a large vessel occlusion (LVO). Image reprinted with permission from NET SMART (www.learnstroke.com), Health Outcomes Institute, LLC.

While a non-contrast CT scan is the only scan that is required to make a thrombolysis treatment decision, additional imaging such as a CT angiogram and CT perfusion scans can provide important diagnostic information.(54) However, to ensure that the patient receives thrombolysis without delay, these additional scans should ideally be performed after treatment with a tissue plasminogen activator drug (alteplase or tenecteplase) is administered.(54)

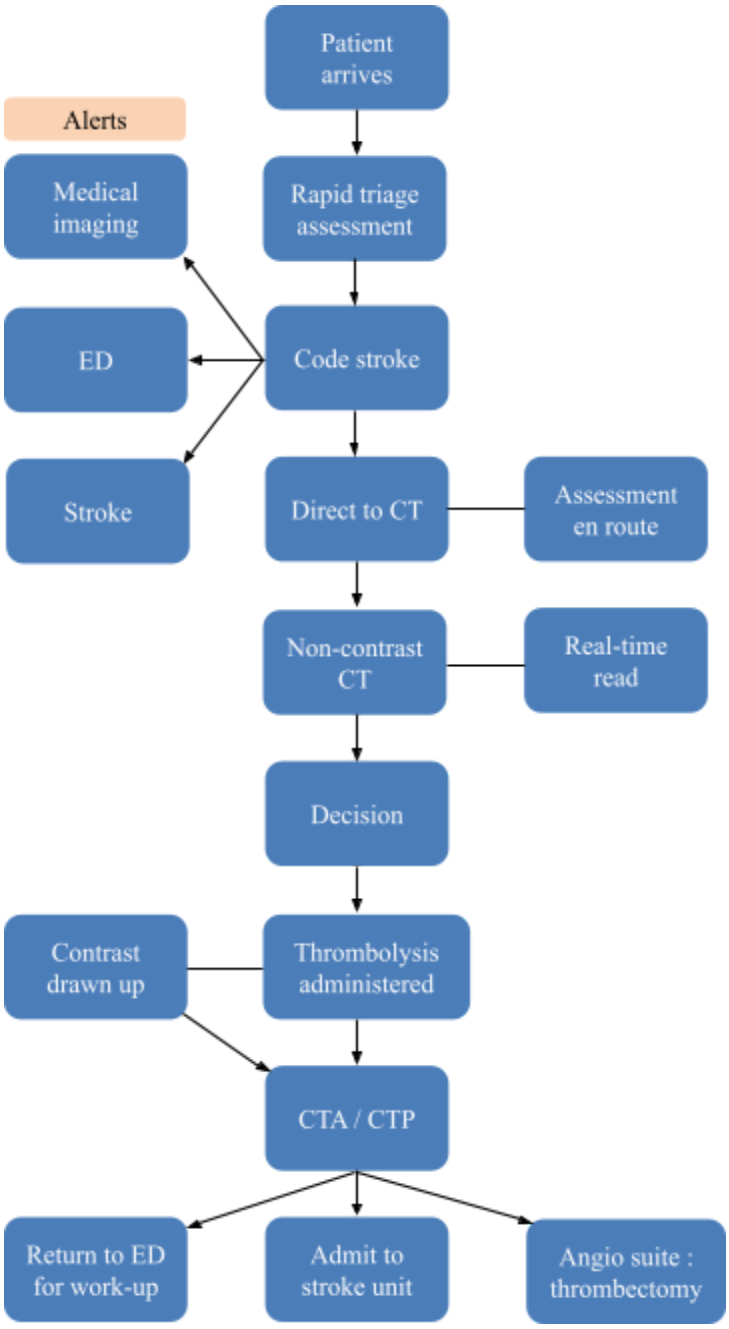

Figure 9. A suggested workflow for patients arriving with suspected stroke to an emergency department (ED). Designed to maximise parallel tasks to reduce door-CT and door-needle times. Image produced by S Coote.

A CT angiogram is a contrast scan that visualises vessel patency. It can confirm the presence of a large vessel occlusion and can give an indication of collateral circulation.(53, 55) In the case of a haemorrhagic stroke, a CT angiogram can diagnose aneurysms or AVMs and can demonstrate ongoing bleeding within a haematoma.(53) CT angiograms should be performed from the aortic arch, through the large arteries of the neck, to the brain's intracranial vessels so the entire patency of the cardio-cerebrovascular system can be assessed. If the neck vessels are not initially imaged, patients should undergo carotid ultrasound before discharge to check for atherosclerosis as a possible mechanism. If found, patients should be considered for carotid revascularization depending on the degree of flow-limiting stenosis and plaque ulceration.(57)

A CT perfusion scan is a contrast scan that assesses the severity of blood flow reduction in the brain. While called 'perfusion' scans, these scans do not measure actual tissue perfusion and instead make inferences about likely tissue infarction based on blood flow compromise within the arterial territory. The software produces colour maps indicating areas assumed to be infarcted or likely salvageable. CTP imaging is only valid in anterior circulation strokes. While these scans can be considered unnecessary in patients arriving within standard treatment windows, they do provide useful information that can be used for prognostication and can also be used to determine presence of a small, distal occlusion that may not be visible on a non-contrast CT or CT angiogram.(53, 54, 58) Given how variable brain tissue viability is during a stroke, some stroke centres use CT perfusion to increase patient eligibility for thrombolysis and thrombectomy, rather than automatically excluding late presenting patients based on symptom onset time alone.(58) However, others prefer using methods such as the Alberta Stroke Programme Early CT Score (ASPECTS) to expertly interrogate the non-contrast CT scan to identify subtle changes and enable patient selection for treatment.(59)

Brain imaging should commence within 30 minutes of patients arriving at hospital.(60) However, large volume stroke centres aim for completion of brain imaging in 10-15 minutes of arrival, utilising a 'Direct to CT' approach, where the patient is taken from triage, on the ambulance stretcher, directly to the CT scanner without off-loading into the emergency department first. This reduces the time to decision making and the time to treatment, therefore increasing the chances of an improved outcome for the patient.(60)

Most patients will undergo additional brain imaging during their hospitalisation. This is done to monitor the evolution of the stroke, look for any haemorrhagic transformation of ischaemic strokes (bleeding into the infarcted tissue), particularly if treated with intravenous thrombolysis or thrombectomy, assess reperfusion and revascularization, and to determine the likely mechanism of the stroke (e.g. atherosclerosis, cardioembolism, etc.).

MOBILE STROKE UNITS

Until now, the need for brain imaging has been the rate-limiting step in moving stroke treatment to the prehospital setting. However, the introduction of mobile stroke units (MSUs) has allowed patients to be assessed, scanned and treated, at their location prior to going to hospital. MSUs are customized ambulances with a built-in CT scanner. While team members vary between MSU programs, most consist of paramedics, radiographer (or technologist), a stroke specialised

nurse or Nurse Practitioner, and a physician (either on-board or via telemedicine).(61) MSUs are dispatched to patients with suspected stroke in the community. At the scene the team can quickly but comprehensively assess the patient and obtain CT scans, usually both non-contrast and CT angiogram. This provides an immediate diagnosis and allows treatments including intravenous thrombolysis, to start without delay.

Ultra-early treatment of stroke on MSUs has been proven to significantly improve patient outcomes. Out of every 100 patients treated on an MSU, 27 will have less final stroke disability and 11 more will be disability free compared to patients treated at hospital.(62, 63)

Figure 10. A view of the rear cabin of the Melbourne Mobile Stroke Unit, showing the OmniTom scanner, track for stretcher loading, and collapsed attendant seats. Reprinted with permission of The Melbourne Brain Centre at The Royal Melbourne Hospital.

CASE STUDY – Part 3

The paramedics use a stroke severity tool and determine that Arif is likely having a large vessel occlusion stroke. They call through to the nearest hospital with EVT facilities. On arrival, the ED, medical imaging and stroke team are waiting.

- What are the priorities on arrival to hospital?
- What brain scans should be ordered for Arif and why?
- What other tests and investigations are needed?

MANAGEMENT OF STROKE

The gold standard treatment for ischaemic stroke is thrombolysis, and/or thrombectomy. Both treatments, while highly effective, are limited in terms of patient selection and time parameters.(64)

Thrombolysis

Initially licensed for use within three hours from stroke onset (or time last known well)(65), intravenous thrombolysis is now approved in most countries for use up to 4.5 hours.(66) Some countries, such as the United States of America are still restricted to three hours, although most clinicians will treat up to 4.5 hours regardless. Using advanced imaging, select patients may now be eligible for thrombolysis up to twenty four hours after onset, and in some cases even when the onset time is unknown, or patients have woken with symptoms.(67-69)

Alteplase has been the mainstay of stroke thrombolysis treatment, however trials have shown the safety and efficacy of tenecteplase in treating ischaemic stroke, especially in LVO strokes, where it has been shown to be superior to alteplase.(70) In non-LVO strokes, individual research trials have found that tenecteplase is non-inferior to alteplase, with recent meta-analyses demonstrating superiority.(71) Clinicians and clinical guidelines now recommend tenecteplase as a suitable alternative agent to alteplase.(72-75)

Both alteplase and tenecteplase are fibrinolytic agents, they work to dissolve the fibrin strands holding the components of the thrombus together. Tenecteplase has greater fibrin specificity, is more resistant to being broken down, and has a longer half-life than alteplase. This longer half-life means that tenecteplase can be given as a bolus injection only, rather than a bolus and infusion which is required with alteplase.(76, 77) This makes tenecteplase quicker and easier to give, it requires less nursing time to manage, and is particularly advantageous for use on MSUs and for patients requiring interhospital transfers, as management of an infusion is not necessary.

Thrombolysis is not without risks. As a fibrinolytic agent, systemic bleeding can occur, and patients need to be carefully screened to avoid unwanted complications. The main concern is a symptomatic haemorrhagic transformation (sICH), where capillaries in the infarct-ischaemic zone leak blood into the stroke area. Rates of sICH are generally 2-6%.(78, 79) Careful patient selection, treatment in high volume stroke centres and MSUs with well-established protocols, and excellent control of blood pressure reduce the risk of bleeding.(75)

Endovascular Thrombectomy

Endovascular thrombectomy (EVT) is a highly specialised procedure that mechanically removes a thrombus from within the artery. It is standard of care for patients with a proximal large vessel occlusion and some medium-sized vessel occlusions. EVT is highly effective and significantly improves patient outcomes, especially when performed as early as possible after stroke onset; improving disability in as much as one patient out of every 2.6 patients treated.(80) EVT is most effective when given in combination with intravenous thrombolysis prior to the procedure; therefore, when patients are candidates for both treatments, both should be given.(81) To further improve efficacy, current research is examining the effect of a small intra-arterial bolus dose of thrombolytic agent during EVT, with recent results showing improved patient outcomes without major safety concerns. Initial trials recommended EVT up to six hours after stroke onset (or time last known well), however research has shown benefit out to 24-hours (82-85) and possibly beyond in select patients with

favourable imaging.(86-88) Current research is also examining the safety and efficacy of EVT for smaller clots in medium-sized arteries in the brain.

Like percutaneous coronary intervention for myocardial infarctions, arterial access for EVT is usually via the femoral or radial artery, with the catheter passed into the cerebrovasculature.(89) Other access sites can include the brachial artery or even directly into the carotid artery. A catheter, usually a retrievable stent and/or an aspiration catheter is advanced to, or into, the clot. When a retrievable stent is used, the stent is deployed, gripping the thrombus in its mesh and then retrieved, whereas with aspiration catheters, suction is applied to retrieve the clot. In some cases both techniques are used to ensure complete clot removal.(89)

While EVT is generally safe, there are safety concerns including arterial perforation and dissection, groin and retroperitoneal haematomas from femoral punctures, vasospasm, intracranial haemorrhage and partial embolization of the clot to smaller distal branches which cannot be mechanically retrieved.(83, 89) To minimise complications and optimise outcomes, EVT should only be performed at specialised stroke centres with a great deal of experience in the procedure. This maintains clinician skills, ensures currency of practice, and appropriate clinical and case quality oversight. Because of this, patients requiring EVT often need to be transferred to centres that can perform EVT safely and effectively, demonstrating the importance of early identification of patients with likely LVOs.(89)

CASE STUDY – Part 4

On arrival at hospital, Arif's brain scans show no signs of haemorrhage or established ischaemic changes. He has a hyperdense artery sign in his left middle cerebral artery which is confirmed as a large vessel occlusion on CT angiogram. Arif is unable to answer questions, but his wife can confirm that he has not taken any of his prescribed medication for the last 2 days.

What is the most suitable treatment for Arif?

- Thrombolysis
- Thrombectomy
- Both
- Neither

Neurosurgery

Unlike ischaemic stroke, there is no definitively proven standard treatment for ICH stroke, but research is ongoing. Some patients may be eligible for neurosurgery, but in most instances, ICH treatment consists of medical management of blood pressure, reversal of anticoagulation or coagulopathic states if present, concomitant management of other medical problems (e.g. fever, hyperglycemia, etc.) and general supportive care.(27) The Code ICH approach ensures rapid diagnosis, use of appropriate imaging (CT/CT angiogram), standardized measurement of neurologic disability using the

NIHSS, and head elevation to 30-degrees height, alongside blood pressure control, coagulopathic correction, and consultation of neurosurgery to determine if operative treatment is appropriate.(90)

It is a common misnomer that all patients with ICH require neurosurgery. Considerations for surgery include the location (surface ICH vs. deep subcortical ICH), hematoma size, patient age, premorbid condition, and severity of the deficits, particularly the patient's conscious state. Until recently, surgical evacuation trials have not shown a clear benefit compared to best medical management, and surgery has therefore been considered lifesaving, but incapable of restoring the patient to their previous functional capabilities.(27) However, a recent trial showed the benefit of minimally invasive surgery in patients with a small-moderate sized (30-80ml) haemorrhages, with reduced mortality, and improved functional outcomes six months post stroke; however, the trial's efficacious findings were driven primarily by enrollment of lobar haemorrhages and the adaption of the protocol to exclude deep subcortical haemorrhages from enrollment.(91) Ongoing research with newer technologies will hopefully offer broader criteria for surgery in the future.

In the case of aSAH, occlusion of aneurysms or AVMs by surgical clipping or endovascular treatment reduces the initial size of the lesion, and in many cases permanently occludes the structure to prevent the risk of re-bleeding. Endovascular occlusion is generally favoured with better odds of recovery and reduced disability.(92) In the case of AVMs, often multiple approaches are used, especially in larger AVMs that may require treatment by endovascular occlusion to reduce the structure's functionality and size, followed by surgical removal when close to the surface of the brain, or stereotactic radiosurgery treatment when located in deep, difficult to reach brain structures. Treatment of ruptured aneurysms is usually commenced quickly after hospital admission to avoid rebleeding which carries high rates of death and severe disability; whereas treatment of AVMs can be planned at a slower pace since early rebleeding is rarely a concern. Patients with aneurysms or AVMs should be followed over the course of many years with repeated CT angiogram imaging to determine if the treatment was sufficient to occlude the lesion.

Blood Pressure

All patients presenting with a stroke should have their BP managed, however there is no single BP target that works best for all types of strokes. Instead, BP aims vary by the severity of the BP, the type of stroke and individual patient factors.(93) Unfortunately, BP targets are not based on robust clinical data and are instead more a gestalt of expert opinion derived from secondary analyses of multiple BP studies, which have become best practice.

BP management will be determined by local protocols and medication availability, however intravenous agents that allow good control, without causing hypotension, rebound HT or cardiac instability are recommended in the hyperacute phase before changing to oral agents. Uncontrolled HT is the most common factor associated with the development of a sICH following thrombolysis. For this reason, patients who are thrombolysed should have their BP closely monitored.(75)
For patients with an ischaemic stroke, guidelines recommend maintaining BP less than 180/105 mmHg before and after treatment with intravenous thrombolysis. However, it is not recommended to drop the BP too low as reduced arterial flow may worsen ischaemia and contribute to penumbral tissue infarction. Guidelines also suggest that for patients not treated

with intravenous thrombolysis, permissive hypertension up to approximately 220/120 mmHg may be considered, however this should be tailored to the individual patient and their medical history.(93)

Conflicting BP research findings challenge the knowledge of appropriate BP targets for patients undergoing EVT, with numerous studies ongoing. In the absence of alternative evidence, thrombolysis BP targets are generally recommended for the pre- and peri-procedural phase, with a gentle reduction to systolic BP between 140 to 160 mmHg in patients with successful clot extraction with good blood flow restoration.(93, 94) However, most BP targets post-EVT are individualized based on patient outcomes and individual practitioner preferences.

Rapid BP reduction is generally recommended for patients with an ICH, with many guidelines suggesting maintenance of systolic BP at 140 mmHg to reduce the risk for haematoma expansion.(95) However, the data supporting this recommendation come from patients whose starting BPs were generally no higher than 180 mmHg. BP targets for patients with severe hypertensive emergencies (e.g. >220 mm Hg) on admission have yet to be established, with most practitioners favoring higher BP values such as 160 mmHg as the reduction target.

In aSAH, BP should be reduced to approximately 140 mmHg systolic, until the aneurysm is occluded to reduce the risk for rebleeding. Once occluded, permissive hypertension or medication-induced hypertension may be used to enhance perfusion during vasospasm.(96) Once the initial hyperacute phase has passed, BP aims should be reviewed and adjusted to maintain appropriate blood flow for the patient, considering factors such as known stenoses or spastic segments in an aSAH. (93)

Patients with an impaired swallow may need enteral tubes to allow administration of oral medications. It is therefore important that nurses ensure tubes are placed without delay and medications are provided in an appropriate form for administration (e.g., tablets that can be crushed, syrup, etc.). Patients will often need multiple agents to maintain BP control. Each medication should be added slowly and adjusted to effect and polypharmacy considerations should be given to class and mechanism of action.(93)

Coagulation Status

Patients who present with an anticoagulation related haemorrhage should have their coagulation status urgently assessed, and any coagulopathies reversed. Warfarin related haemorrhages should be treated with vitamin K, prothrombin complex concentrates or cryoprecipitate. Specific anticoagulation agents for direct oral anticoagulants (DOACs) such as idarucizumab, should be used in preference for generic reversal agents when available. Non-specific agents such as Factor VII may reduce some haematoma expansion (97), but are yet to be proven in studies that have excluded DOAC patients, while tranexamic acid has failed to show a benefit.(97, 98)

For patients who have an ischaemic stroke while on dabigatran, the reversal agent, idarucizumab, can safely be administered, quickly and efficiently reversing the anticoagulation, allowing for thrombolysis to be given.(99)

Fever, sugar, swallow

Hyperglycaemia, hyperthermia and dysphagia have all been shown to worsen neurological outcomes, but research demonstrates that good nursing care and prompt management of these conditions can significantly improve patient outcomes post stroke. This applies to patients with both haemorrhagic and ischaemic stroke,(100), and should be a priority in all stroke care plans. Treatment of dysphagia using pharyngeal electrical stimulation (PES) is a recent advancement that should be considered in all patients with dysphagia that require a feeding tube. PES treatment has been shown to increase the rate of tube removal and reduce the risk of aspiration and related complications.(101)

Stroke Unit Care

All patients with a stroke or TIA should be admitted to an acute stroke unit for their ongoing management until discharge, this is in preference to a general ward and even an intensive care unit.(102, 103) A stroke unit is a geographically discreet area or ward with co-located beds, that is staffed by a multidisciplinary team of stroke experts who are best equipped to manage the patients' complex needs.(104) Patients who are admitted to a stroke unit are more likely to be alive, independent and living at home 12-months post stroke compared to patients admitted to non-stroke units. This benefit applies to patients who have had both a haemorrhagic or an ischaemic stroke and is irrespective of any reperfusion or hyperacute therapies the patient may have had.(104)

Patients should be monitored closely in the first hours post stroke for signs of deterioration, especially if they have had reperfusion therapies. Deterioration can be due to several reasons including haematoma expansion in a haemorrhagic stroke, haemorrhagic transformation of an ischaemic stroke, vessel reocclusion, vasospasm and expected evolution of the existing infarct.(64) Frequency of observations and the assessment tools used will be determined by local protocols and individual patient condition, but nurses must be vigilant to any signs of neurological change and escalate care appropriately.

Palliative Care

Despite best medical and surgical care, stroke has a high mortality rate. In the case of a massive infarct or haemorrhage with a poor prognosis, or patients who fail to improve following treatment, it may be appropriate to move care to a palliative approach rather than subjecting them to lengthy and ultimately futile medical investigations and treatments. Patients' wishes (if known) should always be considered, and sensitive discussions with patients' family, ideally in conjunction with a palliative care team, should be undertaken.(105)

Ongoing Management

While in hospital, patients with stroke should undergo all necessary tests and investigations to determine the cause of their stroke so that tailored secondary prevention measures can be commenced. These may include cardiac monitoring for AF, including implantable loop recorders or similar long-term devices, echocardiography, ECGs, carotid imaging,

pathology tests, sleep studies, and repeat brain imaging. Individual patient risk factors such as smoking, HT, diabetes, hypercholesterolaemia, sleep apnoea, drug use, etc., need to be identified and targeted treatment and education, including general lifestyle education, provided to reduce the risk of future strokes. It is important that this process start in hospital, but stroke prevention education must also be reinforced in rehabilitation sessions and by primary providers.(106)

CASE STUDY – Part 5

Arif is given thrombolysis and is taken for thrombectomy. Afterwards he is admitted to the stroke unit where he begins to show steady signs of improvement.

- What are the priorities of care during Arif's in-patient stay?
- What tests and investigations should be performed, if any?
- What education would you give Arif and his wife about his stroke and secondary prevention?

CONCLUSION

In summary, stroke produces a multifaceted, variable and highly nuanced global disease burden, and is the number one cause of preventable adult disability. Stroke requires prompt recognition and aggressive treatment at specialised centres to reduce mortality and morbidity. While thrombolysis and EVT therapies improve outcomes, these treatments are not universally available, making early recognition and appropriate hospital routing by paramedics an essential first step in the stroke chain of survival. Nurses, the emergency department interdisciplinary staff, neuroimaging personnel, and stroke teams all contribute to patients' recovery by initiating stroke protocols for rapid diagnosis and treatment. Once in a stroke unit, the interdisciplinary team is the first line of defense against neurological deterioration and complication avoidance. A team approach, conducted by highly skilled and specialised stroke clinicians, practicing evidence-based care ultimately are patients' best hope for recovery.

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