University of Basrah
Al-Zahraa Medical College

Ministry of higher Education
and Scientific Researches

BLOCK:

Mental health care and Neurology

Stroke

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Objectives:

- Define the clinical and pathological entity of stroke and describe its classification, pathophysiology and risk factors.
- Describe how the clinical presentation following a stroke is determined by the neuroanatomical structures affected.
- Discuss the importance of the early evaluation and management in cases of stroke.
- When should neuroimaging take place? What are clinical indications for URGENT neuroimaging vs ROUTINE neuroimaging in stroke?.
- Discuss, in broad outline, current stroke treatment strategies.
- List some common complications of stroke.
- Where should stroke patients be treated and why?

The clinical & pathological entity of stroke, its classification, pathophysiology & risk factors.

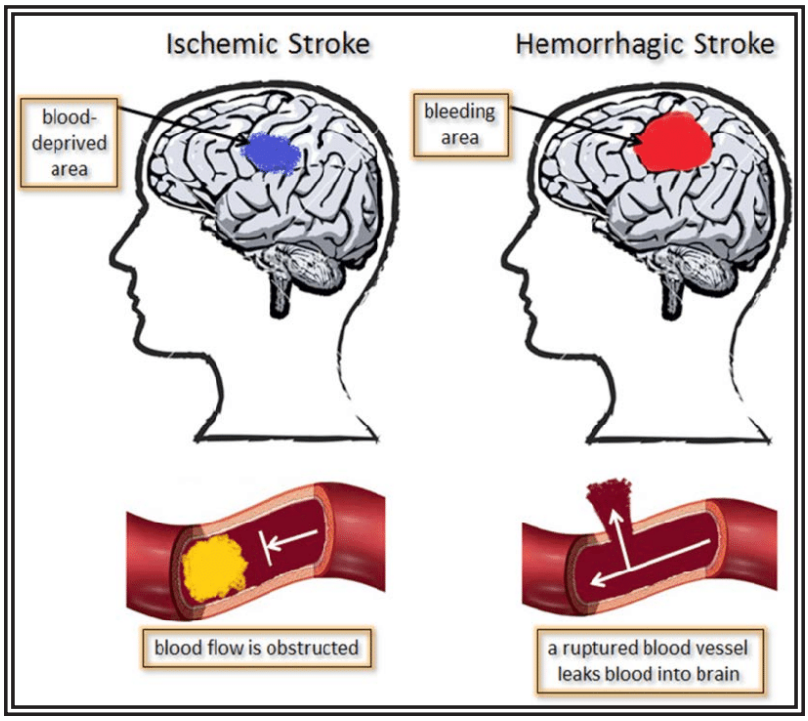
A stroke, sometimes called a brain attack, is a neurological disorder occurs when something blocks blood supply to part of the brain or when a blood vessel in the brain bursts. In either case, parts of the brain become damaged or die. A stroke can cause brain damage, long-term disability, or even death.

Classification

There are 2 types of stroke:

(1) Ischemic stroke: Occurs when a blood vessel supplying blood to the brain is obstructed. It accounts for 87 % of all strokes. A ***Transient Ischemic Attack (TIA)*** is sometimes called a "mini-stroke or warning stroke." is different from the major types of stroke, because blood flow to the brain is blocked for only a short time (usually no more than 5 minutes).

(2) Hemorrhagic stroke: Occurs when a weakened blood vessel ruptures. The two types of weakened blood vessels that usually cause hemorrhagic stroke are aneurysms and arteriovenous malformations (AVMs). The most common cause of hemorrhagic stroke is uncontrolled high blood pressure.

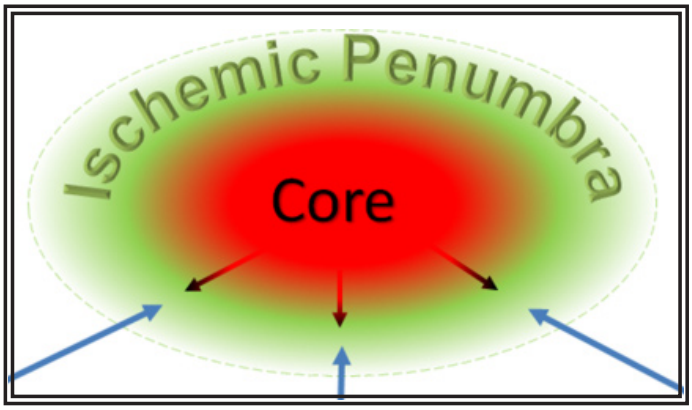


Pathophysiology

The ischaemic stroke is results from lack of sufficient blood flow to perfuse cerebral tissue, due to narrowed or blocked arteries leading to or within the brain. Ischaemic strokes can be broadly subdivided into ***thrombotic*** (atherosclerosis) and ***embolic*** strokes (blood clots or debris from elsewhere in the body, typically the heart valves, travel through the circulatory system and block narrower blood vessels). In the core area of a stroke, blood flow is so drastically reduced that cells usually cannot recover and subsequently undergo cellular death. The tissue in the region bordering the infarct core, known as the ***ischaemic penumbra***, is less severely affected. This region is rendered functionally silent by reduced blood flow but remains metabolically active. Cells in this area are endangered but not yet irreversibly damaged, and if blood flow and oxygen delivery is restored shortly after the onset of stroke, they are potentially recoverable.

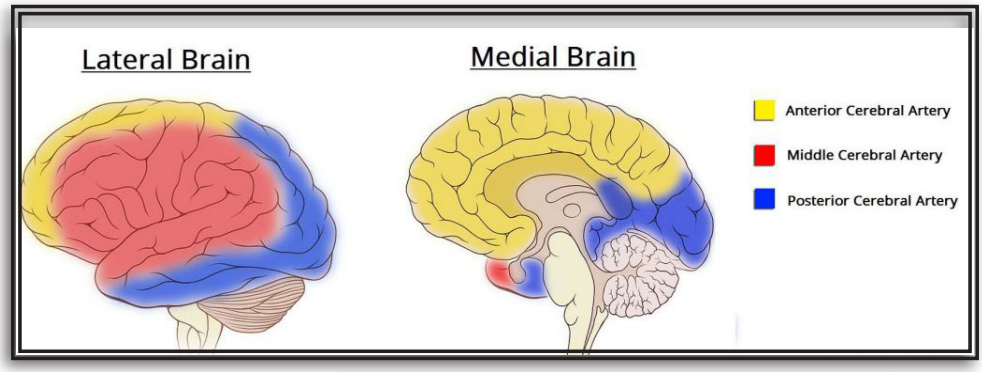
The Haemorrhagic stroke is due to the rupture of a blood vessels leading to compression of brain tissue from an expanding haematoma. In addition, the pressure may lead to a loss of blood supply to affected tissue with resulting infarction. ***Intracerebral haemorrhage*** – caused

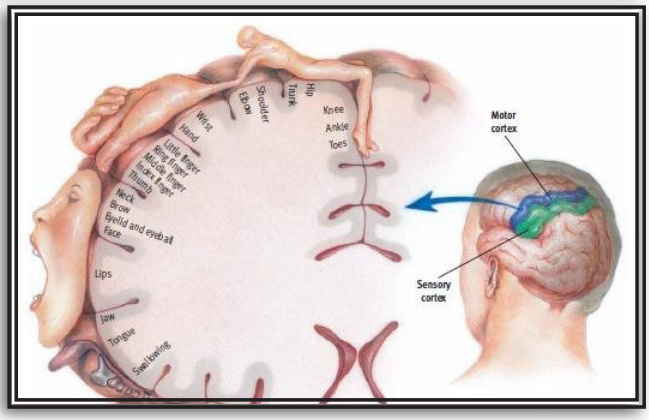
by rupture of a blood vessel and accumulation of blood within the brain is commonly the result of blood vessel damage from chronic hypertension, vascular malformations, or the use medications associated with increased bleeding rates, such as anticoagulants, thrombolytics, and antiplatelet agents. ***Subarachnoid haemorrhage*** is the gradual collection of blood in the subarachnoid space of the brain, typically caused by trauma to the head or rupture of a cerebral aneurysm.



How the clinical presentation following a stroke is determined by the neuroanatomical structures affected.

Weakness affecting the face and arm greater than the leg suggests a stroke in the ***middle cerebral artery territory***, whereas a deficit mainly involving the leg is characteristic of an ***anterior cerebral artery stroke***. Aphasia, neglect, apraxia, and seizures are other signs of involvement of the ***Internal carotid artery territory***. Gaze deviation to the side opposite the hemiparesis is highly suggestive as well. Amaurosis fugax or transient monocular visual loss implies ischemia in the ***territory of the ophthalmic artery***, the first branch of the internal carotid artery. Unilateral infarctions of the occipital lobe due to ***Posterior cerebral artery strokes*** may cause contralateral homonymous hemianopia with macular sparing..





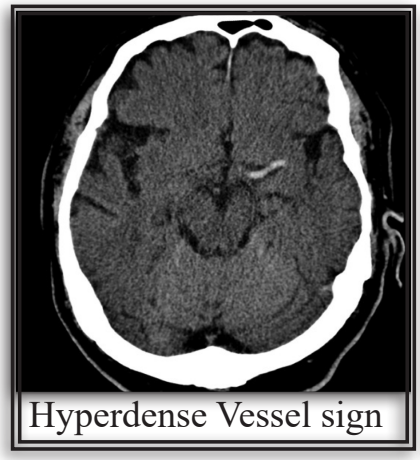
The importance of the early evaluation and management in cases of stroke

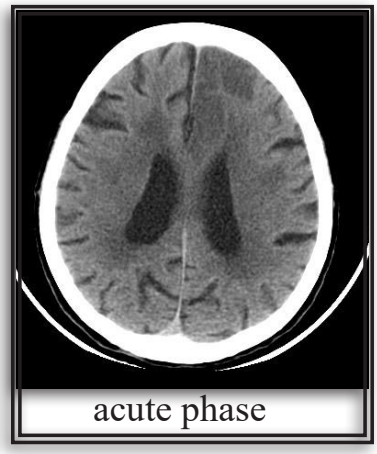
Nerve cells are the core components of the brain, spinal cord and central nervous system and the more that are lost, the greater the chance of permanent disability. As time from initial stroke increases, the penumbra is quickly replaced by infarcted tissue. Earlier treatment results in a greater chance of recovery, a reduced likelihood of permanent disability and lesser need for extensive rehabilitation. This is why is stroke a medical emergency.

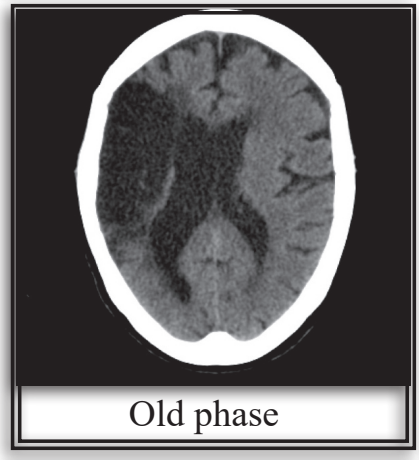
When should neuroimaging take place? What are clinical indications for URGENT neuroimaging vs ROUTINE neuroimaging in stroke?

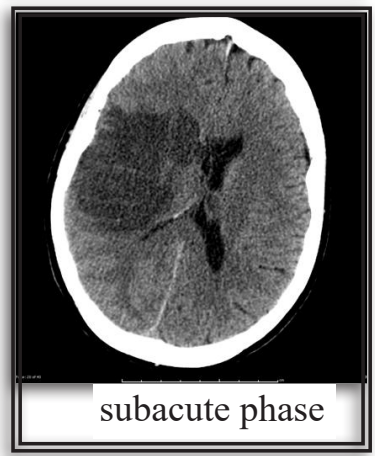
Imaging studies are used to exclude hemorrhage in the acute stroke patient, to assess the degree of brain injury, and to identify the vascular lesion responsible for the ischemic deficit. Some advanced computed tomography (CT) and magnetic resonance imaging (MRI) technologies are able to distinguish between brain tissue that is irreversibly infarcted. ***The plain film radiograph has no role in stroke imaging***.

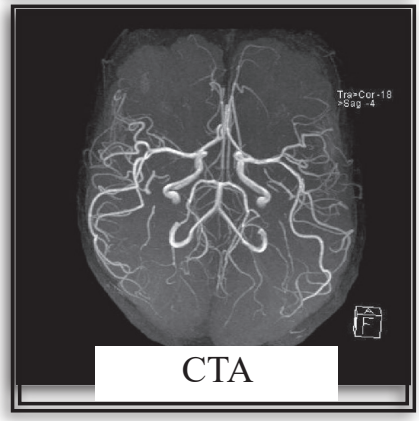
CT scan: Non-contrasted computed tomography (NCCT) is the first imaging technique done in all patients suspected of stroke mainly to exclude hemorrhagic stroke. Besides, NCCT is sensitive to identifying calcification, which is vital in the detection of any lesion. NCCT ***should be done immediately as the patient has stabilized in the emergency room***. NCCT findings in ischemic stroke depend on the age of infarction: *hyperacute* (less than 12 hours), acute (12 to 24 hours), *subacute* (24 hours to 5 days), and *old* (within weeks after stroke). In the "***hyperacute phase***," the main role of non-contrast brain CT is to exclude intracranial hematoma. Sometimes an intra-arterial thrombus has high attenuation in NCCT and can be detected. This phenomenon is called "*hyperdense vessel sign*." In "***acute***" infarction NCCT can show subtle loss of gray-white matter interface (differentiation) due to the rise of water content result of cytotoxic edema. In "***subacute***" infarction, NCCT shows vasogenic edema with mass effect and well-defined margins. In "***old***" findings, NCCT shows volume loss of brain parenchyma (encephalomalacia).

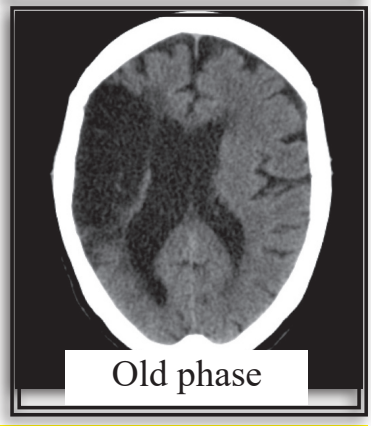

Hyperdense Vessel sign


acute phase


Old phase


subacute phase

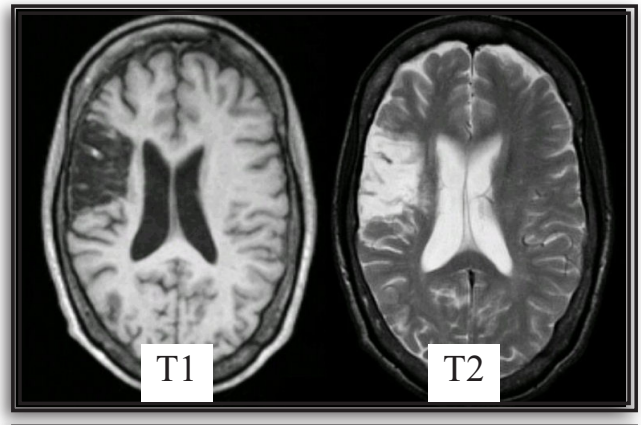

CTA


Old phase

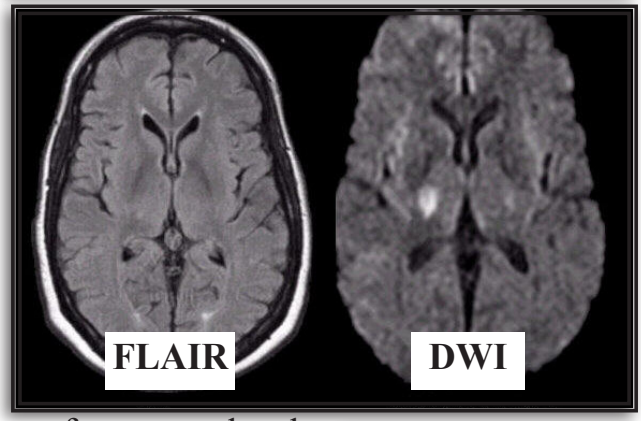
Computed tomography angiography (CTA) is performed by administration of intravenous CT contrast through a line in antecubital fossa. In the acute stroke setting, CTA is done to identify

any vessel thrombosis or occlusion, vascular malformations, dissection, vasculitis, and aneurysm. 3D reconstructions are suitable for the rapid detection of distal vascular stenosis and cloth length. CTA is a reliable method to detect any stenosis or occlusion in the large intracranial vessels such as internal carotid artery and middle cerebral artery trunk.

MRI: Given higher soft-tissue contrast of MRI, MRI is superior to CT in hyperacute and acute phases.

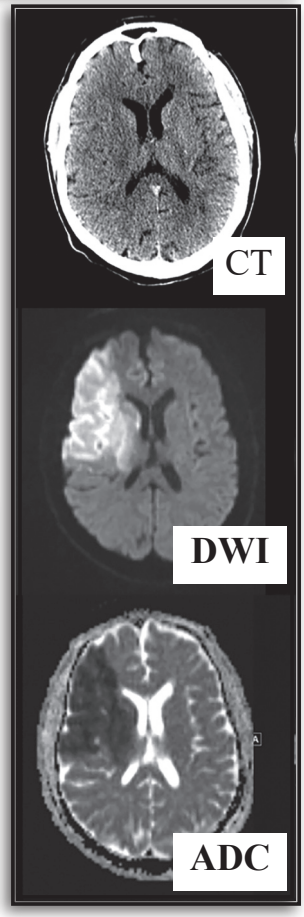


Conventional MRI sequences: FLAIR images may show abnormal signal within *3 hours* after stroke onset. High signal intensity on T2 usually appears about *8 hours* after the stroke onset. T1 may take even longer than *8 hours* to show low signal intensity. Besides, FLAIR is highly sensitive to detect *subarachnoid hemorrhage* and make a high signal in sulci in patients with subarachnoid bleeding.

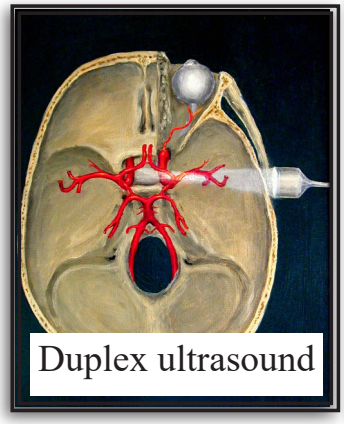


Diffusion-weighted imaging (DWI) is now the best sequence to detect brain infarction earlier than CT or other MR sequences. The diffusion restriction of water molecules in the infarcted tissue, which can be detected by the DW sequence. The diffusion restriction is seen on DWI as hyper signal intensity. To differentiate the "true" diffusion restriction (infarction) versus "T2 shine effect" (like cerebral edema), another sequence called **Apparent Diffusion Coefficient (ADC)** is done by repeat the DW sequences with different parameters and directions. The true diffusion restriction has a lower signal on the ADC than the T2 shine effect.

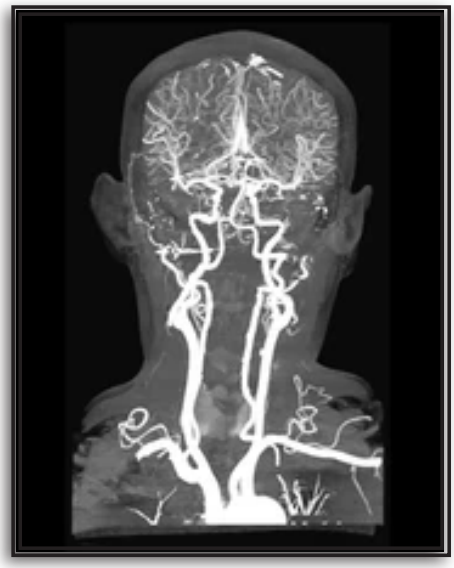
Magnetic resonance angiography (MRA): Like CTA, MRA can be performed in stroke patients to evaluate large vessel occlusions and atherosclerotic lesions. It is useful in patients with allergy to CT contrast material. In comparison to CTA, the MRA is more time consuming and is not available in all hospitals. It is most common in the subacute phase of infarction.



Ultrasonography: Duplex ultrasound is the usual choice for screening of carotid artery stenosis in patients suspected of stroke. Transcranial Doppler ultrasound is commonly used for screening of cerebral artery vasospasm after SAH (SubArchnoid Hemorrhage).



Clinical Indications: (1) All patients with suspected acute stroke should undergo brain imaging with non-contrast CT or MRI.
(2) All patients with suspected acute ischemic stroke who arrive within 3 hours and are potentially eligible for intravenous thrombolysis should undergo immediate brain imaging with non-contrast CT (NCCT) without delay to determine eligibility for thrombolysis.
(3) All patients with suspected acute ischemic stroke who arrive within 6 hours and are potentially eligible for endovascular thrombectomy should undergo immediate brain imaging with non-contrast CT and CT angiography (CTA) without delay, from arch-to-vertex including the extra- and intra-cranial circulation, to identify large vessel occlusions eligible for endovascular thrombectomy.



Current stroke treatment strategies

General stroke treatment: aimed at stabilizing the patient in order to control systemic problems that may impair stroke recovery; the management of such problems is a central part of stroke treatment. General treatment includes respiratory and cardiac care, fluid and metabolic management, blood pressure control and treatment of conditions such as seizures, venous thromboembolism, dysphagia, aspiration pneumonia, other infections, or pressure u lceration, and occasionally management of elevated intracranial pressure.

Specific stroke treatment:

Ischemic stroke **(A) treat the cause of stroke (B) is to preserve tissue in the ischemic penumbra**, where perfusion is decreased but sufficient to prevent infarction. Tissue in this area of oligemia can be preserved by restoring blood flow to the compromised area and optimizing collateral flow. ***(1) Recanalization strategies***, including *(a) Thrombolysis* the administration of intravenous (IV) recombinant tissue-type plasminogen activator (rt-PA) if patient reach the hospital within *3 hours* of the first symptoms of an ischemic stroke because of hemorrhagic transformation particularly for the patients who received a delayed administration of tPA, and *(b) Thrombectomy* intra-arterial approaches (inserting a catheter into an artery, often in the groin. A small device is passed through the catheter into the artery in the brain), attempt to establish revascularization so that cells in the penumbra can be rescued before irreversible injury occurs. Restoring blood flow can reduce the effects of ischemia only if performed quickly. ***(2) Clot Preventing Strategies*** *(a) Aspirin and other antiplatelets* which reduces the chances of another clot forming *(b) Anticoagulants* (heparin, Warfarin) to help reduce their risk of developing new blood clots in the future.

Haemorrhagic stroke **(A) treat the cause of stroke** if the patient was taking anticoagulants before the stroke, he may need treatment to reverse the effects of the medicine and reduce your risk of further bleeding (Sodium bicarbonate regardless of the serum pH, as it enhances elimination of aspirin in the urine, and Vitamin K is the reversal agent for warfarin, and Protamine is a medication used to reverse and neutralize the anticoagulant effects of heparin). **(B) Remove the mass effect of hematoma by surgery** if it is endanger of patient life and repair any burst blood vessels.

Common complications of stroke

The most common complications of stroke are:

- **Brain edema** — swelling of the brain after a stroke.
- **Pneumonia** — causes breathing problems, a complication of many major illnesses. Pneumonia occurs as a result of not being able to move as a result of the stroke. Swallowing problems after stroke can sometimes leading to aspiration pneumonia.
- **Urinary tract infection (UTI) and/or bladder control**. UTI can occur as a result of having a foley catheter placed to collect urine when the stroke survivor cannot control bladderd function. • Seizures — abnormal electrical activity in the brain causing convulsions. These are common in larger strokes.
- **Clinical depression** — a treatable illness that often occurs with stroke and causes unwanted emotional and physical reactions to changes and losses. This is very common after stroke or may be worsened in someone who had depression before the stroke.
- **Bedsores** — pressure ulcers that result from decreased ability to move and pressure on areas of the body because of immobility.
- **Limb contractures** — shortened muscles in an arm or leg from reduced ability to move the affected limb or lack of exercise.
- **Shoulder pain** — stems from lack of support of an arm due to weakness or paralysis. This usually is caused when the affected arm hangs resulting in pulling of the arm on the shoulder.
- **Deep venous thrombosis (DVT)** — blood clots form in veins of the legs because of immobility from stroke.

Sudden death is an important but widely under-recognised consequence of stroke. Acute stroke can disturb central autonomic control, resulting in myocardial injury, electrocardiographic abnormalities, cardiac arrhythmias, and ultimately sudden death.

Strategies to reduce mortality rates from stroke include providing timely transportation of patients, timely delivery of reperfusion therapy through pre-hospital triage via telephone or in the ambulance, evidence-based medical interventions and access to high-quality specialised facilities such as ***stroke units***.

Where should stroke patients be treated and why?

In **Stroke Unit** which is an organized in-hospital facility that entirely is devoted to care for patients with stroke. It is staffed by a team with special knowledge in stroke care.