INTRODUCTION

Stroke is the third most common cause of mortality and morbidity after all cardiac and cancer disorders. The prognosis after acute ischemic stroke (AIS) varies greatly, depending upon the pre-morbid condition, stroke severity, age, and post-stroke complications. The overall mortality rate at 30 days after stroke is 28%. Stroke is the clinical expression of a vascular lesion be it atherothrombotic or haemorrhagic. Acute ischemic stroke (AIS) can be defined as acute loss of perfusion to vascular territory of the brain and a corresponding loss of neurologic function. It has a heterogeneous group of causes including thrombosis, embolism, and hypoperfusion. Stroke, as the name suggests, is an emergency. Management till recently consisted of masterly inactivity to some of empirical measures of doubtful value. With better anatomical and metabolic imaging available, efforts are being made to salvage ischemic cerebral tissues before permanent damage sets in.

The concept of the ischemic penumbra is an important one. All proved and experimental therapies for acute stroke must begin during a narrow and specific time window. This led to concept of “time is brain”. The current trend is to have an organised stroke care team and a ward that exclusively manages patients with stroke, intravenous thrombolysis with recombinant tissue plasminogen activator (rt-PA) within three hours of symptom onset. Although treatments with broad efficacy and safety should be considered for all patients, specific treatment should ideally target the underlying pathophysiology.

PATHOPHYSIOLOGY
The brain is the most metabolically active organ in the body. It represents 2% of the body’s mass, but requires 15-20% of resting cardiac output meet requirement of glucose and oxygen for its metabolism. Consequent to focal cerebral ischemia, there develops a central dense core of necrosed tissue with very little blood flow (approximately 0-10 ml/100 gm/min) surrounded by less defined area of ischemia (approximately 10-20 ml/100 gm/min). The ‘penumbra’ is a concept coined in animal experiments suggesting that functionally impaired tissue can survive and recover if sufficient reperfusion is re-established within a limited time period, which depends on the level of residual flow. However, there is no physical demonstration of this ischemic penumbra. Neurons in this area can carry out all functions for biological maintenance (structurally intact) but do not produce required neurotransmitters (functionally and electrically silent). This process is dependent on speed and duration of ischemia, region of brain involved, and state of collateral circulation. ‘Therapeutic window’ (can be called as ‘window of opportunity’) in relation to stroke is defined as a period during which reperfusion and treatment may lessen the neuronal injury and improves the clinical outcome. MRI and PET data suggest that therapeutic window may vary from 1 hour to 24 hours.

HISTORY AND PHYSICAL EXAMINATION
The neurologic examination must be thorough and focused examination. Clinical features alone may not distinguish ischemic stroke from intracerebral haemorrhage. Misdiagnosis is common and as much as 20% of emergency department diagnoses are incorrect. Common misdiagnoses are seizure, confusional states, syncope, brain tumours,
subdural haematoma, toxic or metabolic disorders, including hypoglycaemia and hyponatremia, Bell’s palsy, peripheral nerve pressure palsies, myasthenia gravis, sepsis, drug overdose.

INVESTIGATIONS
Investigations are done to identify stroke, its mimics, aetiology and systemic conditions that influence choices for acute treatment (Table I). Because time is essence in acute stroke care, hospitals should have diagnostic studies available on a 24X7 day basis.

Brain Imaging
Rapid imaging of the penumbra appears to be the most promising approach to tailor treatment based on specific pathophysiological findings. The diagnostic yield and clinical utility of various neuroimaging procedures must be assessed in terms of information provided, time required, availability, and cost.

Till recently, computed tomography scan (CT) was used for excluding haemorrhage and stroke mimics. With introduction of thrombolytic treatment, its main use is now in treatment decision and to predict/exclude therapeutic complications. It needs to be performed and interpreted within 30 minutes of arrival to the emergency department. The typical early signs are the dense artery sign, loss of gray-white matter interface, loss of the insular ribbon, sulcal effacement, and mass. These early CT signs have been found alone or in combination to be present in up to 92% of cases.

Magnetic resonance imaging (MRI) with angiography (MRA) is a major advance in the neuroimaging of stroke. Its major limitations are cost, availability and the skills required to interpret the images. Diffusion-weighted imaging (DWI) can detect areas of ischemic brain within minutes of onset of symptoms and perfusion-weighted imaging (PWI) demonstrates areas of decreased perfusion. Combination of DWI and PWI can identify potentially salvageable tissues and may be helpful in enlarging therapeutic window as well as in identifying patients to be benefited with therapy.

Advanced imaging aims at vascular and metabolic status of tissue at risk and they include CT perfusion, CT angiography, automated or semi-automated calculations of perfusion and penumbral maps on CT, MR spectroscopy, Doppler carotid imaging, transcranial Doppler ultrasonography, catheter angiography, single photon-emission computed tomography (SPECT) xenon-enhanced CT oxygen-15 positron-emission tomography (PET). In near future vascular imaging will be a key component of the evaluation.

MANAGEMENT: GENERAL
Management of AIS patients at a specialised stroke unit, general ICU, acute ward or at home is a debatable issue and we do not think that a single answer will be appropriate. One must look into medical, financial, social, cultural and geographical aspects before recommending any one of them. Principles and various steps in the management of a stroke have been summarised in Tables II.

Pre-hospital management
Public awareness in recognition of stroke, availability of ambulance, immediate evacuation of patient to a stroke centre and training of health professionals are of great importance. Pre-hospital and in-hospital delays and lack of stroke centre coverage are major issues that negatively impact stroke care. The main steps at this stage are;

a) Secure airways and administering 2-4 L O2/min.
b) Monitor glucose and body temperature and correct if indicated.
c) Pre-hospital triage and notification to a stroke centre.

<table>
<thead>
<tr>
<th>All patients</th>
<th>Selected patients</th>
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<tr>
<td>* Brain CT / MRI</td>
<td>* Hepatic function tests</td>
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<tr>
<td>* Complete blood count, including platelet count</td>
<td>* Toxicology screen</td>
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<tr>
<td>* Blood glucose</td>
<td>* Blood alcohol determination</td>
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<td>* Serum electrolytes</td>
<td>* Pregnancy test</td>
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<tr>
<td>* Renal function tests</td>
<td>* Oxygen saturation or arterial blood gas tests</td>
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<td>* Electrocardiogram</td>
<td>* Chest radiography</td>
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<tr>
<td>* BT, CT, Prothrombin time/ INR</td>
<td>* Lumbar puncture</td>
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<tr>
<td>* Activated partial thromboplastin time</td>
<td>* Electroencephalogram</td>
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Emergency department

**The First 15 Minutes:** After assuring stable airway, breathing, and circulation, relevant blood samples are withdrawn (Table I). Glasgow coma scale (GCS) score and NIH stroke scale score (NIHSS) are recorded with a focused history and physical examination. Two large-bore intravenous lines (IVs) should be placed and a CT scan is performed.

**No blood on the CT scan, <3 hours from symptom onset:** If the diagnosis is clear, inclusion/exclusion criteria for IV tPA should be reviewed. After tPA is given in the emergency, the patient should be transferred to an intensive care unit (ICU) setting for 24 hours.

**No blood on CT scan, >3 hours, but ≤8 hours:** Intra-arterial (IA) thrombolysis is a possibility.

**No blood on the CT scan, >8 hours:** All patients who do not qualify for thrombolysis in the 8-hour time window should receive aspirin. Further inpatient care is tailored to the severity of the acute stroke and comorbid illnesses as described subsequently in this review.

**Stroke care unit (SCU)**

The most important therapeutic advance in the treatment of patients with acute stroke is the development of stroke services and stroke units. There is no strict definition of what constitutes a stroke unit. In general, it is a geographically defined facility staffed by a group of skilled professionals. The units should have monitoring capabilities, which permit close observation for neurological worsening or other complications (Table III).

Optimally all acute stroke patients should have cardiac monitoring and oximetry. More invasive monitoring procedures, such as ICP monitoring, microdialysis, invasive tissue oxygen and brain temperature monitoring are used in selected patient groups. Important interventions at SCU are appropriate hydration, early mobilisation, thorough investigations, management for blood sugar and blood pressure fluctuations, attention to swallowing and oral diet, prevention of deep venous thrombosis and early rehabilitation.

**Telemedicine:**

Though perceived obstacles exist, telestroke has been effective in the management of acute ischemic stroke. It can provide treatment options not previously available at the remote hospital.

<table>
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<th>Table II. Steps in Management of Stroke</th>
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<td>3. Pre-hospital management</td>
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<td>4. Emergency management</td>
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<td>5. Treatment</td>
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<td>i. Airway protection, respiratory and cardiac care</td>
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<td>ii. General - Control of BP, glucose, fever, fluid &amp; electrolytes, physiotherapy</td>
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<td>iii. Antiplatelet agents</td>
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<td>iv. Anticoagulants</td>
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<td>v. Thrombolysis: intravenous, intra-arterial</td>
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<td>vi. Endovascular treatment</td>
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<td>viii. Management of medical complications</td>
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<th>Table III. Essential components for acute stroke care</th>
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<td>- Rapid triage of patients and public education to recognise stroke symptoms</td>
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<tr>
<td>- Comprehensive team and dedicated unit: experienced physician or neurologist, stroke nurse coordinator, neuroradiologist, neurosurgeon, rehabilitation specialist, physiotherapist, occupational therapist, speech and language therapist, and dietician</td>
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<td>- Rapid access to CT (and MRI)</td>
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<td>- Ability to deliver IV rt-PA 24 hours a day, 7 days a week</td>
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<td>- Access to a monitored bed with one-to-one nursing</td>
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<td>- Access to IA/interventional approaches</td>
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MANAGEMENT: SPECIFIC
Treatment for AIS can be broadly divided into two i.e. revascularisation measures and ancillary measures.

Revascularisation measures
The optimal treatment for AIS is reperfusion. To be successful, it must be achieved before the ischemic area in brain is completely infarcted. To date, only intravenous administration of rtPA has been proven to be effective.

Thrombolysis:
The occluding thrombus is bound together within fibrin. Fibrinolysis acts by activation of plasminogen to plasmin; plasmin splits fibrinogen and fibrin and lyases the clot. Recombinant tissue plasminogen activators (rt-PAs) cleave plasminogen to plasmin and its action is potentiated by fresh fibrin. Thus, it has a selective target at the site of thrombus rather than at freely circulating plasminogen as with streptokinase and urokinase.

A new era in therapy for AIS began in 1995, when the National Institute of Neurologic Disorders and Stroke (NINDS) approved the use of tPA in carefully selected patient group. NINDS trial and subsequent studies have demonstrated that relative risk reduction provided by it is 44%, absolute risk reduction is 13% and the number needed to treat to save one person from death or disability is seven. It is beneficial irrespective of the patient's age, gender, ethnicity, presumed cause of stroke or extent of deficit. However, there is 10-fold increase in the risk of symptomatic intracranial hemorrhage (6.4% vs 0.6%). The risk of hemorrhagic complications is greater in certain subgroups of patients (severe strokes, significant early CT changes, and older age). Balancing the risk of hemorrhagic transformation against the therapeutic aim to salvage the ischemic penumbra remains a formidable challenge.

In view of narrow therapeutic window and a large number of exclusion (or debatable) criteria, only a small number of patients (<5%) qualify for thrombolytic therapy when present to a hospital emergency. The recently published European Cooperative Acute Stroke Study-3 (ECASS-3) trial demonstrated that IV tPA has efficacy with adequate safety up to 4.5 hours after the onset of symptoms. Thrombolysis in developing nations is hampered by constraints of resources, delay in arrival of the patients, ignorance or disbelief amongst neurologists as to the efficacy of this form of treatment and a fear of serious rtPA-related complications.

rt-PA is administered in a dose of 0.9 mg/kg with a maximum of 90 mg (given 10% as bolus and remaining as infusion for 60 minutes). No anticoagulation or antiplatelet agents are given for next 24 hours. Bleeding, if occurs, should be treated with cryoprecipitate. CT scan is repeated at 24 hours unless indicated earlier. Important causes of deterioration following thrombolysis are hemorrhagic transformation, reperfusion injury, evolving infarction, re-embolisation and systemic factors e.g. electrolyte imbalances.

Table IV. Prognostication with thrombolytic therapy

| 1. | Size and site of infarct: Improved outcome with smaller & deeper infarct |
| 2. | Angiography findings: Improved outcome with good collateral & perfusion |
| 3. | Neuroimaging findings: Improved outcome with CT negative patients and patients between diffusion and perfusion weighted MRI Mismatch |
| 5. | Dose of thrombolytic agents: Complications are more with increase in dose |
| 7. | Type of tissue: Hippocampus, striatum, neocortex are more vulnerable |
| 8. | Associated diseases: Absence of diabetes, hypertension & cardiac diseases - better prognosis |
from snake venom that degrades fibrinogen, was tested in a series of clinical studies. None of these agents have been tested extensively. Use of multiple thrombolytic agents used serially or in combination may prove to be more effective. Till now thrombolytic therapy has been used without any regard for underlying vascular pathology. Newer approaches will address specific pathophysiology.

**Intra-arterial (IA) Thrombolysis:**
IA thrombolysis has been tried with streptokinase, rtPA, urokinase, and recombinant prourokinase (r-proUK). It should be performed by physicians who are experienced in neurointervention and in centres with neurological expertise. There is a higher rate of recanalisation with IA thrombolysis (67%) against intravenous (40%) one. IA thrombolysis is an option for treatment of selected patients with major stroke of ≤6 hours’ duration due to large vessel occlusions. The feasibility of combining IV and IA rtPA in treatment of ischemic stroke has been examined recently.

**Endovascular Treatment:**
U.S. Food and Drug Administration (FDA) have approved two devices for mechanical thrombectomy in cerebral vasculature for use up to 8 hours from symptom onset. These are MERCI (mechanical embolus removal in cerebral ischemia) and Penumbra System. Other techniques undergoing evaluation are direct mechanical balloon angioplasty of thrombus, mechanical removal of clot from middle cerebral artery, intravascular stenting for restoring arterial patency, suction thrombectomy, laser-assisted thrombolysis of emboli, and power-assisted Doppler thrombolysis. Because of the lack of evidence about the safety and efficacy of these procedures, they are not recommended outside of a research setting.

**Surgical treatment:**
Occasionally drainage of cerebrospinal fluid is necessary to relieve acute hydrocephalus caused by a posterior fossa stroke. Surgical decompression and evacuation of large cerebellar or cerebral infarctions can be considered in small number of patients.

**Malignant MCA infarction** is defined as large infarction (1/2 or 2/3) of the MCA territory and with severe deficits, that goes on to severe cerebral oedema, mass effect, and often herniation with death. Maximal oedema occurs 2 to 5 days from stroke onset. It is often resistant to medical management with a mortality rate of 70% to 80%. Early decompressive hemicraniectomy (≤48 h) and durotomy appears to significantly reduce the mortality to approximately 20%.

**Ancillary measures**

**Airway:**
There is no convincing evidence that routine low flow rate oxygen (2-4 L/min) is of benefit in AIS patients. Ventilation support and endotracheal intubation are recommended for patients with severe airway dysfunction.

**Fluid and electrolyte management:**
The goal of fluid management is euvoemia. Due to the breach of blood brain barrier, the infarcted tissue tends to retain fluid resulting in cerebral oedema. On the contrary, excess fluid loss by diuretics may lead to cerebral hyponatremia and dehydration. IV fluid is commonly considered in patients with reduced consciousness or impaired swallowing. In general, normal saline is recommended. If hypovolemia is suspected, CVP should be maintained between 5 and 12 mm Hg or pulmonary wedge pressure at 10 to 14 mm Hg. Electrolytes (sodium, potassium, calcium, and magnesium) should be checked and substituted according to normal values. Acidosis and alkalosis should be corrected according to blood gas analysis.

Hemodilution and hypertensive treatment is based on the principle that blood flow is inversely proportional to the viscosity. At present, changing rheological characteristics of the blood or by increasing perfusion pressure are not established as useful.

**Blood pressure:**
Elevated blood pressure (BP) is frequent in patients with AIS. It results from stress of the stroke, a full bladder, pain, pre-existing hypertension, a physiological response to hypoxia, or increased intracranial pressure. Theoretical reasons to lower the blood pressure include reducing brain oedema, reducing risk of hemorrhagic transformation of infarction and early recurrent stroke. However, aggressive treatment could be detrimental because of reduction of perfusion in the area of ischemia.

Most studies suggest a negative association between increased levels of BP and clinical outcome, whereas a few studies showed clinical improvement with higher BP levels, clinical deterioration with decreased BP, or no association at all. Because of these conflicting issues and the lack of unambiguous data, the appropriate treatment of the BP in the setting of AIS remains controversial. Situations that require urgent antihypertensive therapy include hypertensive encephalopathy, aortic dissection, acute renal failure, acute pulmonary oedema, or acute myocardial infarction.
There is general agreement to recommend a cautious approach toward treatment of hypertension in the acute setting. Antihypertensive agents should be used when systolic BP is >220 mm Hg or diastolic BP is >120 mm Hg. It should never be lowered rapidly. Agents that have a short duration of action and little effect on cerebral blood vessels are preferred.

Causes of hypotension include aortic dissection, volume depletion, and decreased cardiac output secondary to myocardial ischemia or cardiac arrhythmias. They should be treated with adequate fluids, whole blood and/or vasopressor agents.

**Cardiac care:**
ECG changes secondary to stroke include ST segment depression, QT interval prolongation, inverted T waves, and prominent U waves. Myocardial infarction, cardiac failure, cardiac arrhythmias and sudden death are potential complications of AIS. Significant arrhythmias occur in 5-10% cases of acute cerebral infarction.

**Glucose:**
Hyperglycaemia in AIS is a dynamic stress-related reaction having a significant positive correlation between serum cortisol and glucose levels. The detrimental effects of hyperglycaemia are not clearly understood but include tissue acidosis secondary to anaerobic glycolysis and increased blood-brain barrier permeability. It is unclear whether the impact of hyperglycaemia on stroke outcome is similar in lacunar and non-lacunar infarctions. In multivariate analysis, hyperglycaemia was associated with worse functional outcome in a concentration-dependent manner in non-lacunar stroke, whereas in lacunar stroke hyperglycaemia was associated with favourable outcome. It is postulated that lactate produced by astrocytes has neuroprotective effect.

Current guidelines recommend hyperglycaemia should be treated with insulin and oral agents have no place. Aggressive therapy should be avoided because it can result in fluid shifts, electrolyte abnormalities, and hypoglycaemia.

**Fever & Infections:**
Increased body temperature in AIS has been associated with poor neurological outcome, possibly due to increased metabolic demands, enhanced release of neurotransmitters, and increased free radical production. Acetaminophen and other cooling devices should be used to treat temperature >38.5° C.

In all febrile patients or those at risk for infection, source of infection should be identified. Pneumonia is an important cause of death following stroke. Urinary tract infections are common and secondary sepsis can develop in approximately 5% of patients. An indwelling bladder catheter should be avoided.

**Antiplatlet Agents:**
Data about the usefulness of aspirin or other antiplatelet agents in patients with AIS are less certain than those for treatment of acute myocardial ischemia. Aspirin (150-300 mg/day) should be given within 24 to 48 hours of stroke. The effect is modest with only one extra independent survivor per 100 patients treated. The primary benefit of aspirin seems to be in preventing recurrent events.

Recommendations for other antiplatelet agents such as clopidogrel, dipyridamole, and sulphinpyrazole are less strong. Clopidogrel can be considered for urgent use in patients with aspirin failure or in those where it is contraindicated. Combination of aspirin with clopidogrel in the setting of AIS though claimed to be beneficial but has not been tested with large number of patients.

**Anticoagulants:**
Several trials have evaluated low-molecular-weight (LMW) as well as conventional heparins in treatment of AIS patients with varying benefits. There administration is associated with an increased risk of bleeding complications. Despite a lack of evidence, many physicians use heparin for treatment of acute basilar occlusive disease. Current recommendations do not support anticoagulation as a routine measure in AIS. They can be considered in recurrent TIAs, evolving strokes, dissection of the carotid or vertebral arteries, cardiogenic embolisation to the brain, and lone atrial fibrillation.

**Neuroprotection:**
Neuroprotection is a recent concept entailing the use of drugs to protect the brain from injurious insults thereby limiting cell injury and cell death. It aims at a) enhancing neuronal tolerance thereby decreasing cellular injury, and b) promoting functional recovery subsequent to an ischemic insult. They should be administered early to decrease the net loss of neurons. Other potential application of neuroprotection will as an adjunct in pre-hospital management and in extending therapeutic window. As per current scenario, no agent with putative neuroprotective effects is recommended. Hypothermia and barbiturate coma are promising form of neuroprotection. They decrease metabolic demand but are not easy to use in critically ill patients.
Venous Thrombosis:
Pulmonary embolism accounts for approximately 10% of deaths after stroke. Patient with advanced age, immobility, severe paralysis, and atrial fibrillation are associated with an increased risk of deep vein thrombosis. Anticoagulants can be given to prevent deep vein thrombosis and pulmonary embolism. Aspirin also may be effective for patients who have contraindications to the use of anticoagulants.

Alimentation:
Swallowing impairments are associated with an increased mortality. An abnormal gag reflex, impaired voluntary cough, dysphonia, and cranial nerve palsies are important pointer for the risk. An assessment of the ability to swallow is important before patient is allowed to eat or drink. When necessary, a nasogastric tube can be inserted to provide feedings and to expedite administration of medications.

Seizures:
The reported frequency of seizures during the first days after stroke ranges from 4% to 43% depending on study designs. There are no data about the utility of prophylactic administration of anticonvulsants after stroke.

Brain Oedema and Increased Intracranial Pressure
Brain oedema is initially cytotoxic and later on vasogenic with considerable overlap. Increased ICP depends on size of infarction, degree of oedema and brain compliance. It usually peaks at 3 to 5 days after stroke. Increased ICP also can also be secondary to obstruction of cerebrospinal fluid pathways by a large cerebellar lesion.

The goals of management are to maintain adequate cerebral perfusion and prevent brain herniation. Initial care includes elevation of head end of bed by 20 to 30 degrees and mild restriction of fluids. Factors that exacerbate ICP (e.g., hypoxia, seizures, hypercarbia, and hyperthermia) should be treated. Further managed is through osmotherapy, hyperventilation, barbiturate coma, hypothermia, or surgery. However, there is no standardisation or uniformity for their use. Osmotherapy includes intravenous mannitol (0.25 to 0.5 g/kg) and / or glycerol. An acute IV bolus of 40 mg of furosemide can be used as an adjunct. Hyperventilation is an emergency measure that acts almost immediately. It is a temporary measure and should be supplemented with another intervention to control brain oedema. Oral glycerol is neither tolerated nor is effective in these situations.

Hyperbaric oxygen:
Hyperbaric oxygen therapy aims at achieving normal tissue oxygenation despite a decrease in blood flow. Though it has a potential, evidences from randomized controlled trials are insufficient to provide clear guidelines.

SUMMARY
Till recently, stroke care was an area with therapeutic nihilism among treating physicians. This negative perception is shared by the general public, who often has a poor understanding of the early symptoms and significance of a stroke. We have now entered in an era of proactive approach. Management of patients with acute ischemic stroke is multifaceted, and indications for specific therapies vary among patients. The shortening of pre-hospital period requires education of patients and health professionals and optimization of transport strategies. Lack of stroke centres and pre-hospital and in-hospital delays are major issues that negatively impact stroke care. Telestroke expertise and SCU will help to treat larger number of acute stroke patients.

Key Points
1. There is a need for public education, strengthening of emergency transport system, telestroke and training of paramedical staff. A national as well as local program to expedite stroke care is recommended.
2. Need for urgent evaluation and treatment has to be understood.
3. Stroke care system should be developed with aim of reducing delay and urgent evaluation.
4. CT or MRI evaluation is must as it helps in diagnosis and guiding therapy.
5. Intravenous rtPA within 3 hours of onset of stroke is recommended for all eligible patients and emergency physician has to be conversant with it.
6. Aspirin in a dose of 300 mg/day is administered in all AIS patient not suitable for thrombolysis

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7. Routine administration of other antiplatelet agents and anticoagulants are not recommended.
8. Secondary damage to brain and its consequences needs to be addressed. They are very effective in reduction of mortality and morbidity.
9. Surgical consideration and neurosurgeon is integral part of care in select patients. Endovascular treatment is fast evolving.

REFERENCES