MODULE-3

RECOGNITION AND MANAGEMENT OF POST STROKE COMPLICATIONS
The core purpose
To upgrade practising physicians in the management of Stroke with the help of eminent Neurologists and to build a sustainable business relationship with the practising physicians of API

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RECOGNITION AND MANAGEMENT OF POST STROKE COMPLICATIONS (INCLUDING MOTOR WEAKNESS AND COGNITIVE IMPAIRMENT)
Introduction

Stroke is one of the most common life-threatening neurologic diseases and the third common cause of death globally. In India, stroke deaths are about 73/1,00,000 per year though there is no formal registry available. This burden is liable to increase with higher occurrence of risk factors like aging, smoking and adverse dietary patterns. It is one of the most common causes of disability and dependence with 70% of stroke survivors remaining vocationally impaired and the remaining 30% requiring assistance with daily activities.

It is estimated that 1 in 6 people will suffer from stroke in their lifetime. The speaker also elaborated that the burden of stroke is nearly 26/1,00,000/yr with incidence of ischaemic strokes, haemorrhagic strokes, subarachnoid haemorrhage (SAH) and undetermined strokes at 69%, 23%, 3% and 5% respectively.

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### Table 1: Stroke prevalence by subtype

<table>
<thead>
<tr>
<th>Stroke mechanism/Subtype</th>
<th>Prevalence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Borderzone</td>
<td>5%</td>
</tr>
<tr>
<td>Lacunar</td>
<td>20%</td>
</tr>
<tr>
<td>Cryptogenic and rare causes</td>
<td>20%</td>
</tr>
<tr>
<td>Artery-to-artery embolism</td>
<td>20%</td>
</tr>
<tr>
<td>Aortic arch atheroma</td>
<td>15%</td>
</tr>
<tr>
<td>Cardiac embolism</td>
<td>20%</td>
</tr>
</tbody>
</table>

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### Stroke subtype by underlying pathology

The speaker also stated that 72.5% of all strokes are caused by cerebral ischaemia with its incidence rising with increasing age.

Depending on the location and cause of the infarct, the various subtypes of stroke and their prevalence is outlined in table 1.
Based on the area affected (topographical subtype), there are 3 main stroke mechanisms involved (Fig 1). These can also be cryptogenic if the cause is not determined.

1. Artery-to-artery or cardiac/aortic embolism: The affected areas could be in the territory branch of the middle cerebral artery (MCA), posterior cerebral artery (PCA), anterior cerebral artery (ACA) or present as deep MCA infarcts.

2. In situ thrombosis due to small-vessel disease: present as lacunar infarcts.

3. Haemodynamic changes due to large artery disease: present as borderzone infarction.

Ischaemic and haemorrhagic strokes

Strokes can be mainly classified into two types: ischaemic and haemorrhagic strokes (Fig 2). Ischaemic strokes constitute >80% of all strokes. They are mainly caused by the blockage of a cerebral artery (or more rarely vein) impairing blood supply to that part of the brain. They are further subdivided into thrombotic strokes and embolic strokes.

Vascular dementia and stroke

Cerebrovascular injury caused by cerebral small vessel disease is one of the key causes for vascular dementia. Post-stroke syndromes can also result in vascular dementia. Depending on the vascular distribution of the lesions, they can be pathologically traced to presence of multiple lacunar infarcts, a single strategically placed infarct or hypoperfusion leading to white matter demyelination.

Thrombotic strokes are caused by atherothrombosis of large/small blood vessels of the brain. An embolic stroke is caused by an embolus arising either from the heart (most commonly due to atrial fibrillation or valvular disease) or another blood vessel.

Haemorrhagic strokes take place when a blood vessel supplying the brain ruptures and bleeds. Haemorrhagic are also subdivided into two types—intracerebral haemorrhage (ICH) and Subarachnoid haemorrhage (SAH). In ICH, bleeding occurs from the blood vessels within the brain and is most commonly caused by high blood pressure. In case of SAH, bleeding occurs between the brain and the meninges in the subarachnoid space. The most common causes of SAH are an aneurysm or an arteriovenous malformation (AVM) or by trauma.
**Lacunar infarction**

Strokes occurring due to infarcts in the very small blood vessels of the brain are called lacunar strokes (Fig 3). A lacunar infarct is typically ≤10mm in diameter. They are characterised by the lack of cortical signs and the classic lacunar syndromes, depending on the part of the brain affected, are purely sensory, purely motor (involving the face and arm or arm and leg), mixed sensorimotor, clumsy hand dysarthria (clumsiness of any one hand, disproportionate with any limb weakness) along with slurred speech & ataxic hemiparesis (presents as weakness of one side with ataxia (or unequal clumsiness) of the same side). Other lacunar syndromes include isolated dysarthria, unilateral asterixis, hemiballism and hemichorea.17

Lacunar ischaemic stroke is strongly associated with the presence of hypertension and diabetes.31 A study by Fisher CM revealed that the heart weighed >400gms in such patients.60 Prognosis in patients with lacunar infarcts is generally good. When there is an occlusion in the MCA, it might lead to infarction in the lenticulostriate arteries. These have a poorer prognosis than lacunar infarcts but better than that for cortical/subcortical infarcts.61 In case of anterior choroidal artery (AC/RA) infarcts, the underlying pathology is normally embolism for the larger infarcts, and they have a worse prognosis than the smaller infarcts.12

**Oxfordshire Community Stroke Project (OCSP) classification**

OCSP has divided strokes into four clinically identifiable subtypes as shown in Table 2.

<table>
<thead>
<tr>
<th>Type of infarct</th>
<th>Detailed description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total anterior circulation infarcts, TACI</td>
<td>large anterior circulation infarcts with both cortical and subcortical involvement</td>
</tr>
<tr>
<td>Partial anterior circulation infarcts, PACI</td>
<td>restricted and predominantly cortical infarcts</td>
</tr>
<tr>
<td>Posterior circulation infarcts, POCI</td>
<td>infarcts associated with the vertebrobasilar arterial territory</td>
</tr>
<tr>
<td>Lacunar infarcts, LACI</td>
<td>infarcts confined to the territory of the deep perforating arteries</td>
</tr>
</tbody>
</table>

**B.E.F.A.S.T.**

The B.E.F.A.S.T. acronym helps in swift identification of a person having a stroke based on signs of motor weakness

B – Balance: sudden loss of balance or coordination
E – Eyes: sudden change in vision or trouble seeing
F – Face: drooping of one side of the face when asked to smile
A – Arms: drifting of one arm downward when asked to raise both arms
S – Speech: speech slurred or strange while speaking
T – Time: call 9-1-1 immediately, if any of the above signs are seen
Vascular dementia and associated cognitive disturbances

Vascular dementia, specifically the subcortical ischaemic form (SVI), often causes dementia and cognitive dysfunction in elderly people. The ischaemic injury can be a complete infarction (including lacunar infarcts and microinfarcts) or an incomplete infarction of deep cerebral white matter (Fig 4).

The incomplete infarction of deep white matter presents as Binswanger’s disease. Symptoms include slowly progressive depression, bradykinesia, dysexecutive syndrome, gait apraxia and urinary incontinence.

Differential diagnosis of stroke

There are several conditions which mimic stroke. Some of these include seizure, migraine, transient global amnesia, tumours, metabolic disorders and multiple sclerosis.

The resulting interruption of the parallel circuits from the prefrontal cortex to the basal ganglia and corresponding thalamocortical connections gives rise to several symptoms.

Clinical manifestations of the lacunar state include apathy, depression, abulia, Parkinsonism and dysexecutive syndrome. Dementia due to a single strategically placed lacunar infarct presents as executive dysfunction and frontal lobe syndrome.
A TIA is also called a "mini-stroke." Unlike other major stroke types, blood flow to the brain in TIA is blocked for only a short time, often without causing any permanent damage (Fig 5). However, it should not be ignored since it is a warning sign of a future stroke. Just like a major stroke, TIA is considered a medical emergency.29

Guide 1 and 2: History and examination
Use of a standardised stroke clerking proforma to take a thorough history at the point of admission helps in recording essential information at the most appropriate time. This, in turn, enhances patient assessment and might help in efficient execution of the management plan.29

The general physical examination of the patient is essential to rule out other potential causes for the symptoms observed, presence of comorbidities, to establish the cause of stroke and to assess issues that may influence the management of stroke.29

A thorough neurological exam and use of a standardised assessment and stroke scale can help measure the extent of the neurological deficit and pinpoint the stroke subtype to optimise management.29

Guide 3: Cardiopulmonary resuscitation
Along with other symptoms, post circulation strokes can present with impaired consciousness.29 The speaker stated that in case of stroke associated with impaired consciousness, resuscitation is rarely successful.4

Guide 4: Chest X-ray
In case of acute stroke, a chest X-ray obtained 12 to 24hrs after admission guided clinical management in 4% cases. Routine chest X-ray is not advisable unless underlying pulmonary, cardiac or vascular disease is suspected.29

Guide 5: Electrocardiogram (ECG)
The cardiovascular status of patients with acute stroke can be assessed with an ECG and cardiac biomarkers. They may help in the identification of coexisting cardiac arrhythmias or myocardial ischemia. A repeat ECG along with serial cardiac enzymes can also indicate developing silent ischaemia or paroxysmal arrhythmias not observed initially.29

TIA usually lasts for only a few minutes and the majority of the signs and symptoms disappear within an hour’s time. The predominant ones are similar to those of a stroke such as weakness/numbness of the upper limb or both upper and lower limb, speech difficulties, monocular blindness, loss of balance or coordination and sudden, severe headache without any cause.29

Atherosclerosis is the main cause of TIA. Thrombus formation at the site of the plaque can cause further occlusion of the cranial artery; TIA can also be caused by embolism.29
Most common ECG abnormalities (up to 40%) in stroke patients included T-wave abnormalities, QT prolongation and arrhythmias. Some of the causes for these changes include involvement of the insular cortex, right sided lesions and presence of heart disease or hypertension. \(^5\) The speaker added that age and diabetes can also be causative factors for abnormal ECGs. \(^5\) These patients are also extremely vulnerable to sudden cardiac death. \(^5\) \(^5\)

**Guide 6: Echocardiogram**

An echocardiogram helps in identifying the mechanism of stroke. Presence of certain specific abnormalities such as left ventricle thrombus, patent foramen ovale (PFO), infective endocarditis, atrial fibrillation, aortic atherosclerotic plaques and cardiac masses can be detected with the help of echocardiography. The two methods are Transoesophageal Echocardiography (TEE) and Transthoracic Echocardiography (TTE), with TEE being more useful than TTE. \(^4\) The speaker stated that there is higher yield with TEE in case of ischaemic lesions and recommended its use in available settings. \(^8\)

**Guide 7: Computed Tomography (CT) scan of the brain**

It is the most widely used brain imaging technique. It helps to differentiate between ischaemia and haemorrhage. Prompt usage ensures that patients with ischaemic stroke receive appropriate thrombolytic therapy. Some early signs of ischaemic brain injury include the hyperdense MCA sign, Insular ribbon sign and sulcal effacement. Helical CT angiography is also useful in detecting vessel occlusions or stenosis in stroke. \(^8\) The speaker emphasized that CT scan of the brain is a must for all strokes. \(^6\)

**Guide 8: Magnetic resonance imaging (MRI)**

Diffusion-weighted imaging (DWI) is very sensitive and specific technique for imaging of an acute infarct. It is better than any other MRI sequence. Along with intracranial angiography (MRA), it gives very valuable information. \(^7\) The speaker affirmed that this technique is not routinely indicated. \(^7\)

**Guide 9: Doppler studies**

The different methods of Doppler ultrasound to visualize the cranial vasculature include the B-mode, Duplex, continuous wave and pulsed Doppler systems, colour Doppler flow imaging and transcranial Doppler (TCD). These tests show changes in arterial blood flow patterns near plaques and also give an idea about the vulnerability of the plaque. They are useful in assessing vasospasm, collateral circulation, hemodynamic effects, reserve capacity and during or post-carotid endarterectomy. \(^2\) The speaker thus confirmed that Doppler studies are useful in appropriate clinical settings. \(^9\)

**Guide 10: Fever**

Patients of acute stroke presenting with fever generally have a poor outcome. \(^2\) The metabolic rate increases by 7% for every 1F increase in body temperature. \(^2\) Treatment of the fever to achieve normothermia should be carried out and has achieved good results. Though inducing hypothermia could be an option, its benefits have not been fully elucidated. \(^9\)

**Guide 11: Oxygenation**

Post-stroke hypoxia is common and hypoxic-ischemic (HI) disorders can result from the presence of other medical conditions such as seizures, cardiac arrhythmias, pneumonia. \(^9\) Oxygenation has been found to be beneficial. Hyperbaric oxygen maybe safe. \(^9\) The speaker confirmed that oxygen administration is useful and recommended. \(^9\)

**Guide 11B: Hyperglycaemia**

Diabetes mellitus and hyperglycaemia were associated with larger infarcts and fasting with smaller infarcts. \(^1\) Moreover, hyperglycaemia exacerbates brain lactic acidosis. \(^1\) Blood glucose levels should be maintained between 140 to 180mg/dl in all hospitalised patients. Insulin can be used to treat hyperglycaemia. \(^1\)
Guide 12A: Anti-oedema measures

Steroids are ineffective in ischaemic brain swelling. Mannitol, glycerol and hypertonic saline are useful in some cases. Loop diuretics are also useful, though administration of high-dose albumin has not been proved in major trials. Hyperventilation is useful for short periods of time.

Guide 12B: Blood pressure (BP)

In patients with hypertension, controlled reduction in BP is preferred after stroke, except in case of hypertensive emergencies like hypertensive encephalopathy or aortic dissection. Angiotensin converting enzyme inhibitors (ACEIs) are very useful in managing hypertension and diuretics can also be used in combination. The speaker reiterated that there was no specific BP level to be achieved in these patients.

Guide 13: Atrial fibrillation (AF)

Oral administration of aspirin (initially 325 mg) within 24 to 48 hours after stroke onset is recommended for most patients. The speaker emphasised that for those with AF/ left ventricular (LV) clot, warfarin is to be given after 48 hrs, along with heparin. The European Atrial Fibrillation Trial (1995) showed that there was no treatment effect for anticoagulation if international normalised ratio (INR) was <2. On the other hand, The Stroke Prevention in Atrial Fibrillation study (SPAF III 1996) showed that INR>3 increases bleeding.

Guide 14: Other considerations

Haemodilution with plasma volume expanders such as plasma, dextran or hydroxyethyl starch can be carried out, if needed. The combination of induced hypertension, hypervolaemia, and haemodilution (triple-H therapy) is useful in SAH. The speaker reiterated that ideal levels of mean arterial pressure would be 120-130mmHg and that of central venous pressure (CVP) and pulmonary capillary wedge pressure (PCWP) would be 10-12 and 14-18mmHg, respectively. Arterial blood gas is to be checked only if hypoxia is suspected.

Barbiturate coma and propofol to reduce the elevated intracranial pressure (ICP) have been useful, though they produce hypotension in some cases and must be used judiciously. Indomethacin 50mg IV has been used in stroke to lower ICP and may reduce cerebral blood flow (CBF) too.

The speaker also stated that sedation, pain control and neuromuscular blockade may be necessary in patients with altered sensorium as pain and irritation impede cerebral venous return. He added that sedation reduces sympathetic overactivity, increases co-operation for procedures and nursing care and is also helpful in reducing cerebral metabolism.
MANAGEMENT OF STROKE

The several aspects of general stroke management include maintenance and assessment of the patient’s airway, breathing, and circulation (ABGs). Other aspects to be screened for and assessed are fluids & electrolytes, dysphagia, aspiration, urinary dysfunction, venous thromboembolism, seizures, skin care and depression.  

Management of acute ischaemic stroke

Systemic thrombolysis

IV recombinant tissue plasminogen activator (rtPA; 0.9 mg/kg, maximum dose 90mg) should be administered within 3hrs of onset of stroke. Intra arterial thrombolysis can be used in certain categories of patients within 6hrs of stroke onset. However, this procedure is technically demanding. Both aspirin and heparin are not recommended for 24hrs after thrombolysis.  

Anticoagulants like heparin/low-molecular weight heparin are (LMWH) NOT recommended in acute ischemic stroke routinely. The speaker stated that they may however be recommended in setting of AF, acute myocardial infarction (MI) risk, prosthetic valves, coagulopathies and for prevention of deep vein thrombosis (DVT).  

Intra-arterial thrombolitics are an option for treatment of selected patients with major stroke of <6hrs duration, due to the presence of large vessel occlusion.  

Hypertension

Moderate hypertension might be helpful since low BP could precipitate perfusion failure. Reduction of BP in acute stroke remains controversial. In case of severe end organ damage like pulmonary oedema, encephalopathy and uraemia, BP reduction can be carried out. Markedly elevated BP (>220/110mmHg) can be managed with nitroglycerin, labetalol and sodium nitroprusside. The speaker recommended a more aggressive approach upon institution of thrombolytic therapy.  

Glucose levels and pyrexia

In case of hypoglycaemia, blood glucose should be kept within physiological levels using oral or IV glucose. In case of hyperglycaemia insulin can be used to maintain glucose levels <200mg/dL. The speaker advised against routine glucose infusions. In case of hyperthermia, use of antipyretics and cooling devices can improve the prognosis.  

Ischaemic stroke treatment highlights

IV rtPA (0.9mg/kg, maximum dose 90mg) is recommended for selected patients who may be treated within 3 hours of onset of ischaemic stroke. The first 10% of the calculated dose to be given as bolus, remaining 90% given in infusion over 1 hour. The risk of haemorrhage with rtPA is approximately 6%.  

Secondary prevention of stroke

Average annual risk of stroke recurrence ranges from 4 to 6%. Five year recurrence rates range from 20 to 41%. Since case fatality was higher the first 30 days after recurrent stroke, high priority should be given to secondary prevention. Moreover, patients with TIA or stroke have an increased risk of MI or vascular event.  

For secondary prevention of stroke, BP goal is <140/90mmHg. Statins can be used to lower low density lipoprotein cholesterol (LDL-C) to <100mg/dL. Warfarin is especially recommended in patients with cardioembolic stroke. Other measures include appropriate lifestyle modifications such as smoking, alcohol consumption, diet and exercise.  

Antiplatelet agents which can be utilised include aspirin (50-325 mg), clopidogrel (75 mg), ticlopidine, aspirin + extended release dipyridamole, sulfipyrazone59 and salutocidil. Therapy with combination of two agents can also be carried out.  

Complications of stroke

Cerebral oedema

One third of patients worsen after stroke due to cerebral oedema with cytotoxic oedema peaking after 3 to 4 days. The speaker added that the excitatory amino acids (EAA) produce neurotoxic oedema and accelerate apoptosis.  

Haemorrhagic transformation

Haemorrhagic transformation can occur in up to 40% of stroke patients. It can occur within the first 2 weeks with worsening observed in 10% of patients. Increased risk is observed with antithrombotic, anticoagulant and thrombolytic therapy. Greater size of the infarct and advanced age are also some of the factors that increase the chances for haemorrhagic transformation.  


Management of acute haemorrhagic stroke

The speaker reiterated that analgesics (especially those with sedative properties) and antianxiety agents might prove useful in relieving headache associated with the stroke.\textsuperscript{26} Hyperosmotic agents like mannitol can also be used to reduce cerebral oedema and raised intracranial pressure. The patient should be adequately hydrated. The use of surgical intervention may prove essential in certain cases.\textsuperscript{30}

Immediate diagnostic measures\textsuperscript{36}

Physical examination of the patient is done to assess vital signs as well as to establish neurological activity, if any, eg examination of face and neck may reveal signs of trauma or seizure activity. Neurological examination of the patient is also equally important.

Brain imaging via cranial CT or MRI is carried out to differentiate between ischaemic and haemorrhagic stroke.

Stroke Doppler ultrasonography/Angiography help in detection of large vessel atherosclerosis whereas ECG/Echocardiography help in detection of cardiac embolism.

Differential diagnosis of stroke is essential to exclude conditions which mimic stroke such as hypoglycaemia, migraine and seizure.

Multimodal monitoring

Since each technique covers different aspects of cerebral ischaemia, multiple modalities are used to derive an accurate picture. CBF monitoring can be done via several methods such as Xenon enhanced CT scanning, laser doppler flowmetry (qualitative measurement) and thermal diffusion (quantitative measurement).\textsuperscript{37,38}

Cerebral oxygenation can be monitored by assessing the tissue partial pressure of oxygen (P\textsubscript{T}O\textsubscript{2}) directly with the help of electrodes.\textsuperscript{39}
The speaker also confirmed that emergency medical care personnel who respond to neurologic emergencies first provide reassurance, ensure proper airway and breathing in the patient, place the patient in a position of comfort and finally assess and care for any injuries (if trauma of any type is suspected).


RECOGNITION AND MANAGEMENT OF POST STROKE COMPLICATION

Speech, Sensory impairment and Depression
RECOGNISING AND MANAGING POST STROKE COMPLICATIONS – Why is it important?

Introduction
Medical complications after acute stroke are related to poor outcomes. They are fairly common with their frequency ranging from 40% to 96%.

Impact of stroke
As observed in the accompanying figure, the impact of stroke is substantial. It was the second largest cause of death in 2012. Stroke caused 6.7 million deaths, amounting to 11.9% of all deaths in that year.

Table 1: Common complications after stroke

<table>
<thead>
<tr>
<th>Condition</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Falls</td>
<td>25%</td>
</tr>
<tr>
<td>UTI</td>
<td>24%</td>
</tr>
<tr>
<td>Chest Infection</td>
<td>22%</td>
</tr>
<tr>
<td>Pressure Sore</td>
<td>21%</td>
</tr>
<tr>
<td>Depression</td>
<td>33%</td>
</tr>
<tr>
<td>Shoulder Pain</td>
<td>9%</td>
</tr>
<tr>
<td>DVT</td>
<td>2%</td>
</tr>
<tr>
<td>Pulmonary Embolism</td>
<td>1%</td>
</tr>
<tr>
<td>Somatosensory impairment</td>
<td>45-80%</td>
</tr>
</tbody>
</table>

Common complications after stroke
The common medical complications that arise after stroke are given in Table 1. It was observed that the complications with the highest prevalence were depression (33%) and somatosensory impairment (45-80%).

Mortality from post stroke complications
Mortality in week 1 after the stroke is attributed maximally (90%) to the infarct. The complications range from oedema, extension, rebleed to herniation. During week 2-4, the causes are infections and pulmonary embolism with the risk remaining high at 3 months. The speaker confirmed that during week 8-12, bronchopneumonia and heart disease are the key reasons for mortality in stroke patients.
BASIC PRINCIPLES
OF THERAPY

Prevention
An integrated stroke and rehabilitation unit utilizing comprehensive order sets, care pathways along with dedicated nursing help in prevention of common complications such as fever, hyperglycaemia, hypoperfusion, fever and hypoglycaemia.19

Diagnosis
Accurate diagnosis requires the clinician to have a high index of suspicion and enhanced awareness about post-stroke complications.19

Early treatment
Early treatment of complications should be complication specific and aggressive.19
Both care and outcome are improved with the availability of specialized multidisciplinary stroke units.7

POST STROKE ACUTE NEUROLOGICAL COMPLICATIONS

Cerebral oedema19
All infarcts, especially ones with large volume, present with cerebral oedema. The clinical findings range from being silent to swift fatal deterioration. The location of the stroke, degree of pre-existing atrophy, the infarct volume and the patient’s age all play a role in this parameter. Cytotoxic oedema is usually at its highest 3 to 4 days after injury. However, early reperfusion of a bigger volume of necrotic tissue can speed up and aggravate the oedema towards a course of malignancy with reduced cerebral perfusion pressure within the first 24 hours.

Medical management of cerebral oedema19
The strategies used for medical management of cerebral oedema include reducing utilisation of free water to avoid hypo-osmolar fluid, carrying out osmotherapy involving IV mannitol (0.25 to 0.5 g/kg) and/or glycerol or 3% normal saline, giving an acute IV bolus of furosemide 40mg, preventing surplus glucose administration, decreasing hypercarbia and hypoxemia, treating hyperthermia, discontinuing antihypertensive use (especially those that result in cerebral vasodilatation) and raising the head end of the bed to 20°~30° to assist in venous drainage.

Decompressive Surgery19
Surgical decompression with decompressive hemicraniectomy performed ≤48 hours of stroke onset in the age group of 18 to 60 years (with malignant infarctions) significantly reduced mortality from 78 to 29% with significantly increased favourable outcomes. Use of this procedure must however be individualized due to the risk of survival with moderate to severe disability.

Haemorrhagic transformation19
Haemorrhagic transformation may be accompanied by petechial or symptomatic haemorrhage. The former is observed in patients not treated with recanalization strategies, whereas the latter in patients after IV rtPA, anticoagulant use and intra-arterial (IA) recanalization strategies.
NEUROPSYCHIATRIC COMPLICATIONS OF STROKE

A large number of them occur within the first 24 hours after IV rPA. Moreover, those that occur within the first 12 hours are fatal. The symptoms include falling mental status, deteriorating neurological symptoms, higher BP/pulse, headache and vomiting.

Upon display of signs of symptomatic haemorrhage, remaining IV rPA, if any, should be withheld immediately. Along with an emergent non-contrast CT scan and a complete blood count, testing of coagulation parameters (prothrombin time [PT], activated partial thromboplastin time [aPTT], INR), type and screen and fibrinogen levels is absolutely essential. Antithrombotics can be used safely after the haemorrhagic infarction.

Seizures

Seizures affect nearly 8.9% of all stroke patients. They are often partial with potential for secondary generalization. Inhibitory seizures, mimicking TIAs, occur in 7.1% cases. Most early-onset seizures occur within 24h after the stroke. Early-onset seizures are more common in severe disabling stroke, with cortical involvement, and in haemorrhagic stroke. General causes of seizures to be considered include anti-epileptic drug withdrawal, electrolyte imbalance (especially in renal disease), hypocalcaemia and hypomagnesaemia. There is very little information available regarding the effects of prophylactic anticonvulsants after ischemic stroke or their long-term use after seizure.2

Treatment specifics

The speaker reiterated that early-onset seizures have a recurrence rate of 16% with high in-hospital mortality.4 In these cases, discontinuation of treatment can be considered after a few months. Late-onset seizures, on the other hand, have a recurrence rate of 50% and result in vascular cognitive impairment increasing disability.5

Lorazepam and phenytoin can be used as the initial treatment options.6 Several anticonvulsants are available for use. The classic drugs include Carbamazepine, Ethosuximide, Phenobarbital, Primidone and Valproic acid whereas the newer ones include Lamotrigine, Levetiracetam, Lacosamide, Topiramate, Gabapentin and Pregabalin.7 Choice of the drug can be made on the basis of drug interactions, patient profile and clinician’s discretion since specific guidelines are not available.

Cognitive Syndrome/Post Stroke Dementia

Nearly 30% of all stroke patients present with post stroke dementia. It has a slow progression and is more common in the elderly and those with a history of diabetes. The location of the infarct determines the symptoms displayed by the patient. Subcortical vascular ischaemic dementia can present with features of frontal lobe dementia along with pronounced executive dysfunction.6

The American Heart Association/American Stroke Association (AHA/ASA) Guidelines for Adult Stroke Rehabilitation and Recovery state that*

Screening for cognitive deficits is recommended for all stroke patients before discharge home. (Class IIB)

When screening reveals cognitive deficits, a more detailed neuropsychological evaluation to identify areas of cognitive strength and weakness may be beneficial. (Class IIA C)

Psychiatric Syndrome

Several symptoms are associated with the psychiatric syndrome in post-stroke patients. Overt sadness occurs in 72% of patients,6,8 60% present with depression (associated with the left insular cortex)6 and 25% with anxiety.6 Psychosis/delusional disorder is a rarer complication after stroke.6 Mania is often associated with right hemisphere stroke.6 Another psychiatric complication is the presence of post-traumatic stress disorder (PTSD)-like syndrome.6

Psychological complications

In patients with stroke, depression and anxiety were screened for at discharge and after 6 weeks. At the end of one year, psychological complications included depression (20-50%), anxiety (20-30%) and emotionalism (10%).9

Predictors of post stroke depression

Depression before stroke, physical disability, stroke severity, cognitive impairment, lack of family and social support or anxiety after stroke were positively associated with development of post-stroke depression. On
the other hand, female sex, older age, stroke subtype, previous stroke diabetes mellitus, education level and living alone were not consistently associated with the subsequent development of depression.10

**Outcomes of post stroke depression**

As shown in Fig 2, post stroke depression results in worsened social outcomes with reduced quality of life, decreased rehabilitation treatment efficacy, reduced cognitive impairment, greater mortality and poor functional recovery (recovery may be delayed by 2 years).11

**Screening for post stroke depression**

There are 3 scales available for the screening of post stroke depression. These include The Center of Epidemiological Studies Depression Scale (CES-DO; sensitivity: 0.75; 95% CI 0.60 to 0.85; specificity: 0.88; 95% CI 0.71 to 0.95), Patient Health Questionnaire (PHQ)-9; sensitivity: 0.86; 95% CI 0.70 to 0.94; specificity: 0.79; 95% CI 0.60 to 0.90) and the Hamilton Depression Rating Scale (HDRS; sensitivity: 0.84; 95% CI 0.75 to 0.90; specificity:0.83); 95% CI 0.72 to 0.90).12 Out of the 3, the PHQ-9 was found to be easy-to-use, less time-consuming and validated clinical tool.13

The impact of screening was observed in the AIM (Activate-Initiate-Monitor) randomised, controlled trial (N=188) where a care management strategy (n=89 at 12 weeks) was used. Here, psychoeducational sessions were used to inform survivors and their families about understanding depression and get them to accept treatment (Activate), antidepressant treatment was started (Initiate), and treatment was overseen with scripted telephone calls twice a month (Monitor). The controls were the usual care group (n=93 at 12 weeks) who received the same number of telephone sessions that focused only on recognition and monitoring of stroke symptoms and risks. Remission (HDRS<8) was achieved in 39% of the intervention group vs 23% of the control group (P=0.01). Decrease in depression symptoms (HDRS<8 or a 50% reduction in scores from baseline) was significantly greater in the former group (51%) vs the latter group (30%; P=0.005).14
The AHA/ASA Guidelines for Adult Stroke Rehabilitation and Recovery state that
Administration of a structured depression inventory such as the Patient Health Questionnaire-2 is recommended to routinely screen for post stroke depression. (Class I B)

Patient education about stroke is recommended. Patients should be provided with information, advice, and the opportunity to talk about the impact of the illness on their lives. (Class I B)

Patients diagnosed with post stroke depression should be treated with antidepressants in the absence of contraindications and closely monitored to verify effectiveness. (Class I B)

Periodic reassessment of depression, anxiety and other psychiatric symptoms may be useful in the care of stroke survivors. (Class IIa B)

Consultation by a qualified psychiatrist or psychologist for stroke survivors with mood disorders causing persistent distress or worsening disability can be useful. (Class IIa C)

The usefulness of routine use of prophylactic antidepressant medications is unclear. (Class IIb A)

Patient education, counselling and social support may be considered as components of treatment for post stroke depression. (Class IIb B)

An exercise program of at least 4 weeks duration may be considered as a complementary treatment for post stroke depression. (Class IIb B)

Early effective treatment of depression may have a positive effect on the rehabilitation outcome. (Class IIb B)

No recommendation for the use of any particular class of antidepressants is made. SSRIs are commonly used and generally well tolerated in this patient population. (Class III A)

Post stroke sensory impairment can be divided into somatosensory impairment (occurring in 45-80% patients), visual impairment (30%) and hearing impairment.¹

**Somatosensory deficits**

These range from loss of simple sensory modalities to complex sensory disorders such as dysesthesia (numbness, tingling, or abnormal sensations) and hyperesthesia (excessive reactions to sensory stimuli). ¹,⁶ 'Tactile deficits may be the most common form of sensory deficit after stroke.' In the months after a stroke, patients show substantial but variable somatosensory recovery, especially for proprioception. The bedside exam involves testing of sensory modalities such as pain, proprioception, kinesthesia (sense of movement), temperature and pallesthesia (sense of vibration).¹ ⁶

Assessment of sensory deficits can be done with a pin in the proximal portions of all 4 limbs. The clinician can ask the patient how the stimulus feels (sharp or dull). The eyes need not to be necessarily closed. Response to the response to stimulus on the right and left are compared. If the patient does not respond to noxious stimulus on one side, it is scored as 1. Persons with severe depression of consciousness are assessed and scored as 0 (normal) if no sensory loss to pin is detected; 1 (partial loss) if mild to moderate diminution in perception to pain stimulation is recognized (may involve more than one limb) and 2 (dense loss) if there is severe sensory loss (the patient is not aware of being touched; does not respond to noxious stimuli applied to that side of body).¹ ⁶

**Visual disorders**

Homonymous hemianopia is one of the most common visual deficits observed post stroke. It is essential to assess visual field defect vs visual neglect since visual neglect may improve spontaneously while visual field deficits do not. Other deficits include disruption of colour vision, paralysis of conjugate gaze (poor prognostic sign) and disturbances due to brain stem stroke such as vertigo, diplopia, visual distortions and oscillopsia.⁷

The greatest recovery takes place early after the injury, with maximum spontaneous recovery of visual fields being within the first 2 to 10 days, the first 30 days or first 90 days.¹
Hearing impairment
Posterior circulation ischemia can often result in hearing impairments in approximately 21% of patients. This is usually the result of ischemia in the distribution of the anterior inferior cerebellar artery (majorly due to an inner ear infarct). Hence, stroke-related hearing loss is observed along with vertigo and other deficits related to brainstem/cerebellar infarction. Audiometry is more sensitive than bedside assessment of hearing loss. Majority of the patients show partial or complete recovery at the end of 1 year post stroke.¹

The AHA/ASA Guidelines for Adult Stroke Rehabilitation and Recovery state that³ Evaluation of stroke patients for sensory impairments, including touch, vision, and hearing, is probably indicated. (Class Ila B)

Introduction
Nearly 30-50% of patients suffer from dysphagia at the acute stage of stroke with the incidence falling to 10% after 6 months. Dysphagic patients are prone to dehydration and malnutrition. They also are an increased risk of acquiring aspiration pneumonia. Aspiration pneumonia risk increases by up to 12-fold in dysphagic stroke patients and sometimes occurs in nearly 30% of patients. Due to the likelihood that it would become life-threatening, morbidity and mortality are significantly increased in dysphagic stroke patients compared to those without the disorder. It is thus important to detect it earlier and implement a suitable nutritional management to manage it.⁴⁵

Screening and assessment for dysphagia³⁵
Which methods should be used for dysphagia screening? How should the risk of aspiration be evaluated?

- Water-Swallowing-Test (WST): A 50 ml-WST is recommended by the Scottish Intercollegiate Guidelines Network (SIGN) guidelines
- Multiple-Consistency-Test: The ‘Gugging Swallowing Screen (GSS)’ grades dysphagia into severe, moderate, mild or no dysphagia with a stepwise evaluation for non-fluid and fluid textures
- Swallowing-Provocation-Test (SPT): The swallowing-provocation-test (SPT) specifically examines the involuntary swallowing reflex by bolus injection of 0.4 ml of distilled water through a small nasal catheter into the oropharynx

The AHA/ASA Guidelines for Adult Stroke Rehabilitation and Recovery state that³
Assessment of swallowing before the patient begins eating, drinking, or receiving oral medications is recommended. (Class I B)
Oral hygiene protocols should be implemented to reduce the risk of aspiration pneumonia after stroke. (Class I B)
In which patients is assessment of dysphagia indicated?

In stroke patients who fail the dysphagia screening, a thorough assessment of swallowing function should be conducted.

How often should the assessment of dysphagia be repeated?

Initially, daily assessment of dysphagia is indicated. Continued presence of dysphagia can result in assessments carried out at least twice per week and before discharge. If dysphagia is present even after discharge, monthly assessments for 6 months after the stroke can be carried out.

Enteral nutrition

Enteral nutrition within 7 days of admission reduces mortality by 5.8%. In patients who have a decreased level of consciousness and mechanical ventilation, enteral nutrition is needed for a longer period of time and tube feeding can be initiated early. In patients where prolonged severe dysphagia is expected (>7 days) tube feeding should be initiated. During the acute phase of stroke, if sufficient oral food intake is not possible, enteral nutrition can be preferably given via a nasogastric tube.

The AHA/ASA Guidelines for Adult Stroke Rehabilitation and Recovery state that

Enteral feedings (tube feedings) should be initiated within 7 days after stroke for patients who cannot safely swallow. (Class I A)

Nasogastric tube feeding should be used for short term (2–3 weeks) nutritional support for patients who cannot swallow safely. (Class I B)

Which route of enteral feeding should be preferred?

In the acute stroke, in case sufficient oral food intake is not possible, enteral nutrition can be initiated via a nasogastric tube. If enteral feeding is likely to last for more than a month (>28 days), a percutaneous endoscopic gastrostomy (PEG) should be placed in a stable clinical phase (after 14 – 28 days). Early placement of PEG is necessary in mechanically ventilated stroke patients. If a nasogastric tube is repeatedly removed accidentally by the patient and if artificial nutrition is needed for >14 days, early placement of a PEG should be considered.

The AHA/ASA Guidelines for Adult Stroke Rehabilitation and Recovery state that

Nutritional supplements are reasonable to consider for patients who are malnourished or at risk of malnourishment. (Class IIa B) Drug therapy, neuromuscular electrical stimulation (NMES), pharyngeal electrical stimulation, physical stimulation, transcranial direct current stimulation (tDCS) and transcranial magnetic stimulation are of uncertain benefit and not currently recommended. (Class III A)

In the case of conscious dysphagic stroke patients, along with tube feeding oral intake should also be initiated. This can be in accordance to the kind and severity of dysphagia. Oral nutritional supplements should be given to stroke patients who are malnourished or at an increased risk for malnutrition and those who are at risk for pressure sores. If hydration proves inadequate by oral or enteral nutrition, parenteral hydration should be begun immediately. Consultation with a dietician along with appropriate nutrition support should be initiated in cases of insufficient Intake over a prolonged period of time.
POST STROKE SPASTICITY

Spasticity is defined as a velocity-dependent resistance to stretch of a muscle. It is a component of the upper motor neuron syndrome (Fig 3). Post stroke spasticity may have involuntary muscle activity and limb positioning (dystonic features). Spasticity is correlated with activity limitations associated with hygiene, pain and dressing. These aspects increase caregiver burden and decrease quality of life. Severe proximal and distal limb weakness on acute hospital or rehabilitation admission is the strongest predictor of moderate to severe spasticity (Ashworth scale score ≥2).”

POST STROKE COMMUNICATION IMPAIRMENT

Sensory impairment (Aphasia)

The impairment or loss of language caused by brain damage is called aphasia. It manifests in 21-38% of acute stroke patients and is one of the most devastating cognitive impairments of stroke.1

Types of Aphasia

Out of the several types of aphasia, the 3 main types are Broca’s aphasia, Wernicke’s aphasia and Global aphasia (Fig 4). Broca’s aphasia is characterized by sparse, halting speech, frequently missing function words, misarticulated speech and bound morphemes. There is disturbance in speech planning and production mechanism. Wernicke’s aphasia results in poor reading and auditory comprehension, morphological and semantic paraphasias and fluent speech with phonemic. There is disturbance of permanent representations of the sound structures of words. Global aphasia leads to disruption of all language processing components and severe reduction or loss of all language.”

Fig 3: Upper and lower limb spasticity

The Adducted/Internally Rotated Shoulder
The Clinched Fist
Equinovarus
Flexed Knee
Adducted Thighs

The Flexed Wrist
The Flexed Elbow
Striatal Toe

The Pronated Forearm
The Thumb-in-Palm Deformity
Stiff Knee

Fig 4: Types of Aphasia and their lesions

Broca’s aphasia
Wernicke’s aphasia
Global aphasia
Differential diagnosis of aphasia

There are other syndromes or disorders which mimic aphasia. These include developmental disorders of speech, developmental suppression of the motor fluency, congenital deaf-mutism, pure dysarthria, anarthria, disorders of phonation, conversion reaction, Munchausen syndrome and psychosis (especially schizophrenia). 4,5,6

Clinical examination of aphasia

The accompanying figures mention the components of a brief and detailed clinical examination of aphasia (Fig 5 and 6).

Fig 5: Brief clinical examination of aphasia

(Adapted from Hauser A. C.)

<table>
<thead>
<tr>
<th>Auditory comprehension</th>
</tr>
</thead>
<tbody>
<tr>
<td>Understanding words, questions, directions and stories</td>
</tr>
<tr>
<td>Verbal expression</td>
</tr>
<tr>
<td>Producing sequences</td>
</tr>
<tr>
<td>Repetition of words/sentences</td>
</tr>
<tr>
<td>Naming objects</td>
</tr>
<tr>
<td>Describing pictures</td>
</tr>
<tr>
<td>Responding to questions and having conversations</td>
</tr>
<tr>
<td>Reading and writing</td>
</tr>
<tr>
<td>Reading: Recognition, matching and discrimination of graphic symbols of alphabets</td>
</tr>
<tr>
<td>Writing: Reproduction of letters, words, sentences and paragraphs</td>
</tr>
<tr>
<td>Functional communication</td>
</tr>
<tr>
<td>Using gestures, drawing, pointing when in trouble getting a point across verbally</td>
</tr>
</tbody>
</table>

Fig 6: Detailed clinical examination of aphasia

(Adapted from Hauser A. C.)

<table>
<thead>
<tr>
<th>Linguistic analysis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Phonology, morphology, syntax, semantics, discourse</td>
</tr>
<tr>
<td>Psychosocial status of the patient</td>
</tr>
<tr>
<td>Educational, linguistic, occupational, economic, social, psychiatric and familial background</td>
</tr>
<tr>
<td>Appropriate laboratory and imaging investigations</td>
</tr>
<tr>
<td>Laboratory investigations: General workup and evaluation for stroke risk factors</td>
</tr>
<tr>
<td>Imaging: MRI brain and MR angiogram to document the location, size and morphology of the lesions in brain and its vascular supply</td>
</tr>
<tr>
<td>Functional neuroimaging (fMRI): PET, SPECT techniques for defining discrete neural modules or networks concerned with cognitive functions such as speech</td>
</tr>
</tbody>
</table>

The AHA/ASA Guidelines for Adult Stroke Rehabilitation and Recovery state that 7

Speech and language therapy is recommended for individuals with aphasia. (Class I a)

Intensive treatment is probably indicated, but there is no definitive agreement on the optimum amount, timing, intensity, distribution, or duration of treatment. (Class II a)

Computerized treatment may be considered to supplement treatment provided by a speech language pathologist. (Class II b)

Group treatment may be useful across the continuum of care, including the use of community-based aphasia groups. (Class II b)

Pharmacotherapy for aphasia may be considered on a case-by-case basis in conjunction with speech and language therapy, but no specific regimen is recommended for routine use at this time. (Class II b)
Neurological improvement with piracetam, better than placebo

Several studies have shown that neurological improvement by piracetam is better than placebo as outlined in Table 2 below.

Table 2: Piracetam vs placebo in acute stroke

<table>
<thead>
<tr>
<th>Study (no. of pts)</th>
<th>Daily piracetam dose/Duration of therapy</th>
<th>Time to treatment</th>
<th>Principal outcome Parameter</th>
<th>Results (% of pts)</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Creyten (50)</td>
<td>6g/7 days 4.8g/30 days</td>
<td>&lt;1 day</td>
<td>Neurological improvement</td>
<td>44/8</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Independence</td>
<td>80/71</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Normal neurological function:</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Consciousness</td>
<td>86/66</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Motor function</td>
<td>13/13</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Speech</td>
<td>70/43</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Normal functional ability:</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Orientation</td>
<td>73/37</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Gait</td>
<td>33/11</td>
<td></td>
</tr>
<tr>
<td>Karutana (60)</td>
<td>12g/12 days 4.8g/18 days</td>
<td>&lt;2 days</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Herrschaft (44)</td>
<td>12g/14 days 4.8g/14 days</td>
<td>&lt;5 days</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Piracetam was effective and showed positive results as an add-on treatment in patients with aphasia receiving language therapy. The subtest scores for the Aachen Aphasia Test (AAT) are shown in Table 3.

Table 3: Aachen Aphasia Test (AAT) subtest scores

<table>
<thead>
<tr>
<th>AAT subtest</th>
<th>AAT scores after treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Piracetam (n=24)</td>
</tr>
<tr>
<td>Token test</td>
<td>51.0 (29.2)</td>
</tr>
<tr>
<td>Repetition</td>
<td>51.7 (24.5)</td>
</tr>
<tr>
<td>Written language</td>
<td>58.3 (29.6)</td>
</tr>
<tr>
<td>Naming</td>
<td>55.0 (21.9)</td>
</tr>
<tr>
<td>Comprehension</td>
<td>58.3 (21.9)</td>
</tr>
<tr>
<td>Profile level</td>
<td>51.2 (8.0)</td>
</tr>
</tbody>
</table>
The Communicative Ability Scale rating of spontaneous speech also showed impressive improvement in the piracetam group.²

Motor speech impairment (Dysarthria, Apraxia)³

Dysarthria is an umbrella term for a group of speech disorders and occurs in 20% of stroke patients. These are caused by paralysis, weakness or incoordination of the speech musculature after neurological damage. It can affect one or more of the subsystems underlying speech production: the respiratory, laryngeal, velopharyngeal and oral-articulatory subsystems.

Apraxia of speech is a disorder of motor planning or programming. This results in difficulty in voluntarily producing the correct sounds of speech. In addition to articulatory disturbances, prosodic deficits such as slow rate of speech and restricted variations in pitch and loudness may be present. Apraxia of speech often coexists with non-fluent aphasia. Behavioural treatments include strategies to increase the precision of articulation, to improve prosody and to modify the rate and loudness of speech.

The AHA/ASA Guidelines for Adult Stroke Rehabilitation and Recovery state that⁴

- Interventions for motor speech disorders should be individually tailored and can include behavioural techniques and strategies that target: Physiological support for speech, including respiration, phonation, articulation, and resonance and Global aspects of speech production such as loudness, rate, and prosody. (Class I B)
- Augmentative and alternative communication devices and modalities should be used to supplement speech. (Class I C)
- Telehabilitation may be useful when face-to-face treatment is impossible or impractical. (Class IIa C)
- Environmental modifications, including listener education, may be considered to improve communication effectiveness. (Class III b C)

The post-stroke period requires a sustained and coordinated effort from a large team, including the patient and his/her goals, family and friends, other caregivers (e.g., personal care attendants), physicians, nurses, physical and occupational therapists, speech-language pathologists, recreation therapists, psychologists, nutritionists, social workers and others.⁵

Communication and coordination among team members dealing with various aspects of stroke care is extremely important to maximize the effective and efficient measures use for the management of post-stroke complications. Only then can the stroke patients achieve their full potential.⁶