

Guidelines for Management of Stroke

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Abbreviations

AF	atrial fibrillation
BP	blood pressure
CAS	carotid artery stenting
CEA	carotid endarterectomy
CE-MRA	contrast-enhanced MR angiography
CSF	cerebral spinal fluid
CT	computed tomography
CTA	computed tomography angiography
CV	cardiovascular
DSA	digital subtraction angiography
DWI	diffusion-weighted imaging
ECG	electrocardiography
ED	emergency department
EEG	electroencephalography
EMS	emergency medical service
FLAIR	fluid attenuated inversion recovery
ICA	internal carotid artery
ICP	intracranial pressure
INR	international normalized ratio
ICH	Intracerebral hemorrhage
iv	intravenous
IS	Ischemic stroke
LDL	low density lipoprotein
MCA	middle cerebral artery
MI	myocardial infarction
MRA	magnetic resonance angiography
MRI	magnetic resonance imaging
mRS	modified Rankin score
NASCET	North American Symptomatic Carotid Endarterectomy Trial
NIHSS	National Institutes of Health Stroke Scale
NINDS	National Institute of Neurological Disorders and Stroke
OSA	obstructive sleep apnoea
PE	pulmonary embolism
PFO	patent foramen ovale
pUK	pro-urokinase
QTc	heart rate corrected QT interval
RCT	randomized clinical trial
rtPA	recombinant tissue plasminogen activator
SAH	Subarachnoid hemorrhage
TCD	transcranial Doppler
TOE	transoesophageal echocardiography
TIA	transient ischemic attack
TTE	transthoracic echocardiography
UFH	unfractionated heparin

Introduction

Stroke is one of the leading causes of morbidity and mortality worldwide. WHO statistics indicate that all types of stroke ranked cause of death (13-15%) as the third and surpassed only by heart disease and cancer. Each year 15.000.000 persons suffer from stroke worldwide out of which 5.000.000 and up with mortality and the remaining 10.000.000 have been deeply disabled. Each year, Mongolia registered 270-290 cases of stroke in 100.000 populations, thereby belonging to countries with higher incidence of stroke.

Many advances have been made in stroke prevention, treatment, and rehabilitation. For example, thrombolytic therapy can limit the extent of neurologic damage from stroke and improve outcome, but the time available for treatment is limited. Healthcare providers, hospitals, and communities must develop systems to increase the efficiency and effectiveness of stroke care. The “7 D’s of Stroke Care”— detection, dispatch, delivery, door (arrival and urgent triage in the emergency department [ED]), data, decision, and drug administration — highlight the major steps in diagnosis and treatment and the key points at which delays can occur.

The goal of stroke care is to minimize brain injury and maximize patient recovery. The community-oriented “Stroke Chain of Survival” that links actions to be taken by patients, family members, and healthcare providers to maximize stroke recovery are the following:

- Rapid recognition and reaction to stroke warning signs;
- Rapid emergency medical services (EMS) dispatch;
- Rapid EMS system transport and hospital pre-notification;
- Rapid diagnosis and treatment in the hospital;
- Rehabilitation;
- Primary prevention;
- Secondary prevention;

The guidelines summarize the management of 3 types of acute strokes: (1) Ischemic Stroke and Transient Ischemic Attack; (2) Intracerebral Hemorrhages; and (3) Aneurysmal Subarachnoid Hemorrhage in the adult patients.

The guidelines for management of strokes developed by leading experts of Mongolia were approved by the Council of Neurology at Ministry of Health Mongolia and recommended to introduce into out-of-hospital and ED assessment and In-hospital stroke management.

A. General

A1. Definition of Stroke

Stroke is an acute focal neurological deficit caused by a vascular lesion; The onset is sudden and the symptoms last longer than 24 hours, if the patient survives.

Ischemic stroke is an acute focal neurological deficit caused by a vascular occlusive lesion with sudden onset and symptoms lasting longer than 24 hours.

Transient ischemic attack is a neurological deficit lasting less than 24 hours, with complete clinical recovery, caused by focal hypoperfusion within the brain.

Intracerebral hemorrhage is an acute focal neurological deficit caused by rupture of microaneurysms secondary to chronic hypertension.

Subarachnoid hemorrhage is a spontaneous arterial bleeding into the subarachnoid space, caused by rupture of arterial aneurysm or AVM.

A.2. International Classification of Disease codes (ICD-10)

I63-I67:	Cerebral Infarction
G45-G46:	Transient Ischemic attack
I61-I67.9:	Intracerebral Hemorrhage
I60-I60.9:	Subarachnoid Hemorrhage

A.3. Users of this Guideline

The guidelines would be used by personnel of Emergency Aid, Stroke Units, Neurological Clinics and Neurosurgery and Rehabilitation specialists.

A.4. Objective

These guidelines are directed to emergency room personnel and stroke specialists for management of acute ischemic stroke, TIAs, Intracerebral and subarachnoid hemorrhages and their prevention in the modern era.

A.5. Processed Date: From June to September 2011

A.6. Update Date: 2016

A.7. Participants for preparing this guideline:

G.Tsagaankhuu	Consultant Neurologist, Department of Neurology, Health Sciences University
A.Kuruvilla	WHO consultant
J.Ariunaa	Chairman, Neurological Center, State Third Central Hospital
J.Sarangerel	Director, Reflex Hospital
R.Amarbayasgalan	Head of Neurological clinic, State First Central Hospital
G.Baigalmaa	Neurologist, State Third Central Hospital
U.Saraa	Neurologist, State Third Central Hospital
Ts.Delgermaa	Neurologist, State First Central Hospital
Ts.Ouyngerel	Neurologist, State First Central Hospital
L.Enkhsaikhan	Neurologist, State First Central Hospital

O.Tsetsgee	Anesthesiologist, State Third Central Hospital
D.Enkhbold	Neurosurgeon, State Third Central Hospital
R.Munkhbayar	Neurologist, Reflex Hospital
D.Baasanjav	Medical Research Institute, President of Mongolian Neurology Association
D.Ulziibayar	Department of Neurology, HSUM
L.Otgonbayar	Head, Department of Neurology, HSUM
Ts.Burmaa	Standardization and Measurement Agency
N.Ouynbileg	Millennium Challenge Account Mongolia, Project of Health personnel
S.R.Govind	PMO/Team Leader HSD, NCD, MCH, ENH WHO Mongolia
D.Narantuya	WHO, Project of "Stroke and Heart Infarction"

Review and confirmation of guidelines:

Organizations

Responsible for completion

Meeting for guidelines developing working groups under MOH; from Aug, 2011 to Nov,2012 (6 times)	Chief, Working group J.Ariunaa
Meeting of Mongolian Neurology Association's governors 27 th Aug,2011	President of Mongolian Neurology Association D.Baasanjav
MOH, Board of Neurology; 16 th February, 2012	MOH, Board of Neurology; G.Tsagaankhuu
HSUM, Council of terminology; 17 th February, 2012	HSUM, Council of terminology; G.Tsagaankhuu
MOH, Health medical aid and standardization technical committee;	MOH, Government Implementation Agency - Department of Health; Ts.Khun
Meeting of MOH governor 15 th Nov,2012	MOH, State secretary for Health J.Khatanbaatar

A.8. Used terminology:

- Aneurysm
- Ischemic stroke or cerebral Infarction
- Transient ischemic attack
- Reversible ischemic neurologic deficit (RIND)
- Progressing cerebral infarction
- Thrombus infarct
- Cardiogenic infarct
- Lacunar Infarct
- Hemodynamic infarct
- Intracerebralhemorrhage
- Subarachnoid hemorrhage
- Antithrombotic therapy
- Thrombolysis
- Stroke unit
- Carotid endarterectomy
- Angioplasty
- Stent

A.9. Epidemiology

In the Western industrialized countries age-adjusted stroke incidence rates range between 100-300 per 100,000 people per year. Every year, in the United States about 700,000 people of all ages suffer a new or repeat stroke. Approximately 158,000 of these people will die, making stroke the third leading cause of death in the United States. Each year, Mongolia registered 270-290 cases of stroke in 100,000 populations, thereby indicating that Mongolia has high rate of stroke incidence as compared with average stroke incidence rates in the world. In the developed countries, cerebral infarction accounted for 87% of all stroke types. In Mongolia, the ratio of brain infarction versus intracerebral hemorrhage is 1:1 thereby showing that adequate control of hypertension and preventive measures against stroke are not optimal in Mongolia.

Epidemiological studies suggest that ischemic strokes account for 80-85%, hemorrhagic strokes for 15-20% and subarachnoid hemorrhages for 10%. Of ischemic strokes, cardioembolism account for 15-30%, atherosclerotic infarction accounts for 15-40%.

B. Management of Ischemic Stroke (Cerebral Infarction)

Cerebral ischemia is caused by blockage vascular supply in the local region of the brain, except during the general circulatory failure due to cardiac arrest and systemic hypotension. Occlusion of the cerebral artery typically results in an area (the "core") so severely ischemic that will be damaged irreversibly within minutes or hours, surrounded by a less ischemic penumbra of neuronal tissue that may be temporarily inactivated. In fact, the ischemic penumbra can be defined as a severely ischemic area, functionally impaired; consisting of potentially surviving brain tissue that is at risk of infarction, but that can recover if it is reperfused in time. If the occluded artery reopens early and sufficiently with subsequent improvement or normalization of the blood supply, the ischemic lesion will be small or absent and its clinical expression might well be a TIA.

The three leading causes of cerebral infarcts are:

- Extra- and intracranial large artery disease
- Cardioembolism
- Small artery disease (microangiopathy)

About one-third of the sources of stroke remain undiagnosed even if carefully investigated.

Atherosclerotic large artery disease is the presumed cause of cerebral infarcts in 15-40% of patients. The principal sites of atherosclerotic plaque are in the internal carotid artery at the extracranial bifurcation, carotid siphon, and large intracranial arteries (more often seen in Asians and Black African Americans).

In the posterior circulation similar lesions occur in the proximal and distal vertebral arteries, and in the basilar artery.

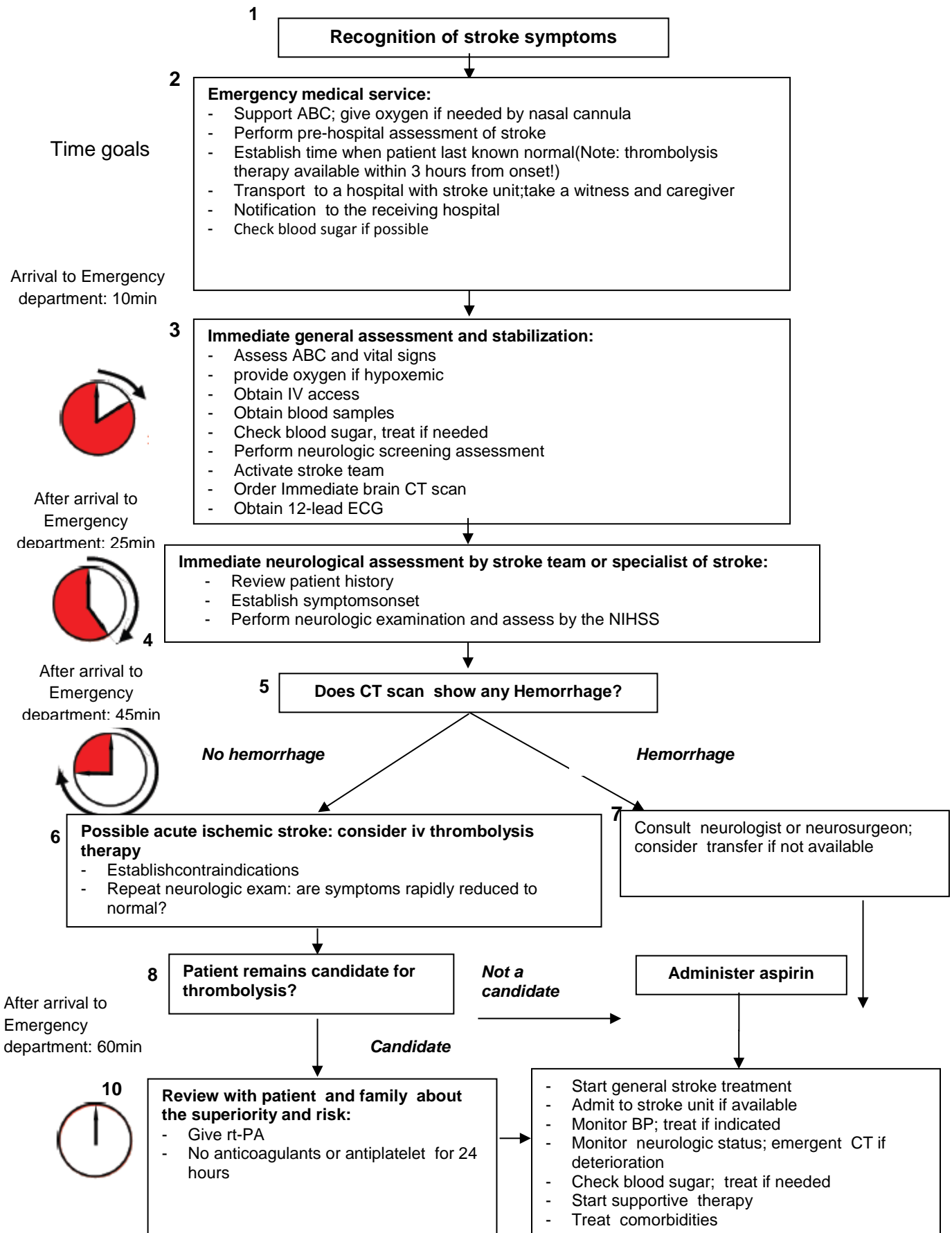
Ischemic stroke is also classified into subgroups based on the postulated mechanisms of infarction and duration of progressing ischemia:

- **Embolic stroke** occurs when thrombus from heart (cardioembolic stroke) or another blood vessel (artery-to-artery embolism) breaks and occludes more distal cerebral artery.
- **Lacunar infarct** or small vessel disease develops when focal atherosclerotic lesion leads to occlusion of penetrating artery deep in the brain parenchyma.
- **Hemodynamic infarct** is considered to be hemodynamic when there is evidence of flow failure. Mostly, this may involve the boarder-zone of cerebral arteries and caused by severe stenosis or occlusion of a large artery (ICA or VA).

- **Transient ischemic attack (TIA)** is defined as temporary episode of focal ischemic neurologic dysfunction that completely resolves within 24 hours.
- **Reversible ischemic neurologic deficit (RIND)** or a minor stroke is considered when focal ischemic deficit persists for longer than 24 hours but resolves within 3 weeks.
- **Cerebral infarctions(Ischemic strokes)** is characterized by persisting of focal neurologic deficits longer than 3 weeks.
- **Progressing cerebral infarction** is considered when rapid evolution of ischemic cerebral vascular events occurs in patients who have increasing neurologic deficit for as long as 72 hours after the onset symptoms. This stroke syndrome is more common in the territory of vertebrobasilar system.

Defined diagnosis of stroke and the differentiation between ischemic and hemorrhagic stroke cannot be made on clinical grounds alone. Specific treatments can be initiated only after the diagnosis of an ischemic or hemorrhagic stroke has been established with an adequate radiological examination.

Goals for management of patients with suspected stroke algorithm



B.1. Evaluation and management of acute stroke

Any patient suspected of having a stroke should be transported as quickly and safely as possible to the nearest hospital with staff experienced in acute stroke management and emergency brain imaging. The emergency aid to the stroke patients can be taken in a short time in following orders:

- Pre-hospital recognition stroke symptoms;
- Transportation to the hospital with stroke unit;
- Evaluation of general condition and neurological status of patients in stroke unit;
- Immediate clinical and neuroimaging diagnosis;
- Immediate starting of general and specific appropriate treatment;

B.1.1. Orders and steps of emergency medical services

Step 1: Identify signs of a possible stroke

Step 2: Call 103 immediately (activate EMS system)

This is an important step because EMS responders can transport the patient to a hospital that provides acute stroke care and notify the hospital that the patient is coming. The hospital staff can then prepare for efficient evaluation and management of the patient. Currently, half of all stroke victims are driven to the ED by family members or friends.

B.1.2. Referral and patient transfer

Step 3. Emergency medical service assessments and actions

Table 1. EMS personnel assessments and actions

Assessments	Actions
Define and recognize the signs of stroke	Support the ABCs (airway, breathing, and circulation)
Assess the patient using the “ Face-Arm-Speech Test ”	Give oxygen as needed
Establish time zero	Time Zero: set the time when the patient was last known to be neurologically normal. If the patient was sleeping and wakes up with symptoms, time zero is the last time the patient was seen to be normal
Consider triage at a stroke center, if possible	Transport the patient quickly.
Assess neurologic status while the patient is being transported	Bring a family member or witness to confirm time zero
	Alert the receiving hospital
	Check glucose levels

Table 2. Prehospital stroke Scale (Cincinnati criteria)

Facial Droop (have patient show teeth or smile):
• Normal—both sides of face move equally
• Abnormal—one side of face does not move as well as the other side
Arm Drift (patient closes eyes and holds both arms straight out for 10 seconds):
• Normal—both arms move the same or both arms do not move at all (other findings, such as pronator drift, may be helpful)
• Abnormal—one arm does not move or one arm drifts down compared with the other
Abnormal Speech
• Normal—patient uses correct words with no slurring
• Abnormal—patient slurs words, uses the wrong words, or is unable to speak
Interpretation: If any 1 of these 3 signs is abnormal, the probability of a stroke is 72%.

Table 3. Prehospital stroke Scale (Los Angeles criteria)

Criteria	Yes	Unknown	No
1. Age >45 years	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
2. History of seizures or epilepsy	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
3. Symptom duration <24 hours	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
4. At baseline, patient is not wheelchair bound or bedridden	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
5. Blood glucose between 60 and 400	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
6. Obvious asymmetry (right vs left) in any of the following 3 exam categories (must be unilateral):	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
	Equal	R Weak	L Weak
Facial smile/grimace	<input type="checkbox"/>	<input type="checkbox"/> Droop	<input type="checkbox"/> Droop
Grip	<input type="checkbox"/> Weak grip <input type="checkbox"/> No grip	<input type="checkbox"/> Weak grip <input type="checkbox"/> No grip	<input type="checkbox"/> Weak grip <input type="checkbox"/> No grip
Arm strength	<input type="checkbox"/>	<input type="checkbox"/> Drifts down <input type="checkbox"/> Falls rapidly	<input type="checkbox"/> Drifts down <input type="checkbox"/> Falls rapidly
Los Angeles criteria sensitivity 93%, specificity 97%			

B.1.3. Emergency management in the Emergency Department (ED)

Time goal: 10 min

Step 4: Within 10 minutes of the patient's arrival in the ED, take the following actions

Table 4. Stroke unit services

Actions
Assess ABCs and evaluate vital signs.
Give oxygen if patient is hypoxemic (less than 92% saturation). Consider oxygen if patient is not hypoxemic.
Make sure that an IV has been established.
Take blood samples for blood count, coagulation studies, and blood glucose. Check the patient's blood glucose and treat if indicated. Give dextrose if the patient is hypoglycemic. Give insulin if the patient's serum glucose is more than 300. Give thiamine if the patient is an alcoholic or malnourished.
Assess the patient using a neurologic screening assessment, such as the NIH Stroke Scale (NIHSS).
Order a CT brain scan and have it read quickly by a qualified specialist.
Obtain a 12-lead ECG and assess for arrhythmias.
Do not delay the CT scan to obtain the ECG. The ECG is taken to identify a recent or ongoing acute MI or arrhythmia (such as atrial fibrillation) as a cause of embolic stroke. Life-threatening arrhythmias can happen with or follow a stroke.

B.1.4. Diagnosis of stroke

Time goal: 25 min

Step 5. Within 25 minutes of the patient's arrival, take the following actions:

Table 5. Stroke team actions

Actions
Review the patient's history, including past medical history.
Perform a physical exam.
Establish time zero, if not already done.
Perform a neurological exam to assess patient's status using the NIHSS.
The CT scan should be completed within 25 minutes from the patient's arrival in the ED and should be read within 45 minutes; Establish neurological and CT diagnosis

Table 6. **Emergency diagnostic tests in acute stroke patients**

In all patients	
1	Brain Imaging: CT or MRI
2	ECG
3	<i>Laboratory Tests:</i> Complete blood count and platelet count, prothrombin time or INR, Partial Thrombin Time (PTT) Serum electrolytes, blood glucose, C-reactive protein (CRP) or sedimentation rate, Hepatic and renal chemical analysis
When Indicated	
4	Extracranial and transcranial Duplex / Doppler ultrasound
5	MRA or CTA
6	Diffusion and perfusion MR or perfusion CT
7	Echocardiography (transthoracic and/or transoesophageal)
8	Chest X-ray
9	Pulse oxymetry and arterial blood gas analysis
10	Lumbar puncture
11	EEG

Table 7. **Recommended requirements for centers managing acute stroke patients**

Primary stroke center	Comprehensive stroke center
Availability of 24-hour CT scanning	MRI / MRA / CTA
Established stroke treatment guidelines and operational procedures, including intravenous rtPA protocols	Transoesophageal echocardiography
Close co-operation of neurologists, internists and rehabilitation experts	Cerebral angiography
Specially trained nursing personnel	Transcranial Doppler sonography
Early multidisciplinary stroke unit rehabilitation including speech therapy, occupational therapy and physical therapy	Extracranial and intracranial colour-coded duplex sonography
Neurosonological investigations within 24 hours (extracranial Doppler sonography)	Specialized neuroradiological, neurosurgical and vascular surgical consultation (including telemedicine networks)
Transthoracic echocardiography	Carotid surgery
Laboratory examinations (including coagulation parameters)	Angioplasty and stenting

Monitoring of blood pressure, ECG, oxygen saturation, blood glucose, body temperature	Automated monitoring of pulse oxymetry, blood pressure
Automated ECG monitoring at bedside	Established network of rehabilitation facilities to provide a continuous process of care, including collaboration with outside rehabilitation center

Table 8. National Institutes of Health Stroke Scale (NIHSS)

Stroke scale			
Assess level of consciousness Alert Drowsy Stuporous Coma	0 1 2 3	Movement of legs: (elevate both legs to 30° for 5 seconds) a) left b) right No drift Drift Some effort against gravity No effort against gravity No movement	0 1 2 3 4
Assess orientation (month, age) Both correct One correct Two incorrect	0 1 2	Coordination of limb Normal One arm or one leg no coordination Two limb no coordination	0 1 2
Follow commands (1) Open & close eyes; (2) make fist & release Obeys both correctly Obeys one correctly Two incorrect	0 1 2	Sensory Normal Partial loss Dense loss	0 1 2
Follow finger (gaze) Normal Partial gaze palsy Forced deviation	0 1 2	Language (aphasia) Normal Mild to moderate aphasia Severe aphasia Mute/ Complete -Global aphasia	0 1 2 3
Visual field Normal Partial hemianopia Complete hemianopia Bilateral loss	0 1 2 3	Speech clarity Normal articulation Mild to moderate slurring Nearly unintelligible, mute	0 1 2
Facial palsy Normal Minor asymmetry Partial Complete	0 1 2 3	Inattention No neglect Partial neglect Profound neglect	0 1 2
Movement of arms: (elevate both arms to 90° for full 10 seconds) a) left b) right No drift Drift Some effort against gravity No effort against gravity No movement	0 1 2 3 4		
		Total	

B.1.5. Treatment decisions by stroke team

Time goal: 45 min

Step 6. Within 45 minutes of the patient's arrival, the specialist must decide, based on the CT scan or MRI, if a hemorrhage is present.

Table 9. Stroke unit specialist actions

Take these actions if a hemorrhage is present	Take these actions if a hemorrhage is NOT present
Note that the patient is not a candidate for thrombolytics.	Decide if the patient is a candidate for thrombolytic therapy.
Arrange for a consultation with a neurologist or neurosurgeon.	Review criteria for IV thrombolytic therapy by using the thrombolytic checklist
Consider transfer, if available.	Repeat the neurologic exam (NIHSS)

If the patient is rapidly improving and moving to normal, thrombolytics may not be necessary.

B.1.6. Treatment of Ischemic Stroke

B.1.6.1. General stroke treatment

The term “general treatment” refers to treatment strategies aimed at stabilizing the critically ill 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, the prevention and treatment of conditions such as seizures, venous thromboembolism, dysphagia, aspiration pneumonia, other infections, or pressure ulceration, and occasionally management of elevated intracranial pressure. It is common practice to actively manage neurological status and vital physiological functions such as blood pressure, pulse, oxygen saturation, blood glucose and temperature. Neurological status can be monitored using validated neurological scales such as the NIH Stroke Scale or the Scandinavian Stroke Scale. There is little direct evidence from randomized clinical trials to indicate how intensively monitoring should be carried out, but in stroke unit trials it was a common practice to have a minimum of 4-hourly observations for the first 72 hours after stroke. Close monitoring is also required for the first 24 hours after thrombolysis. More invasive monitoring procedures, such as central venous catheters or intracranial pressure monitoring, are used only in highly selected patient groups.

General principles of caring for patients with Ischemic stroke

1. Provide ongoing monitoring of neurologic status (level of consciousness, focal deficit, GCS);
2. Establish cardiac monitoring;
3. Airway and oxygenation; Oxygen should be administered if the oxygen saturation falls below 95%;

4. Elevate head of bed 30 degrees;
5. Routine blood pressure lowering is not recommended following acute stroke; Cautious blood pressure lowering is recommended in patients with extremely high blood pressures (>220/120 mmHg) on repeated measurements, or with severe cardiac failure, aortic dissection, or hypertensive encephalopathy;
6. It is recommended that low blood pressure secondary to hypovolemia or associated with neurological deterioration in acute stroke should be treated with volume expanders;
7. Provide enteral nutrition with nasogastric tube for patients with decreased level of consciousness or impaired gag reflex;
8. Maintain normovolemia and normal sodium level by starting with administration of 2-3 liters/day solutions of 0.9 normal saline;
9. Treat increased ICP as needed;
10. Monitoring serum glucose levels is recommended; Treat if serum glucose levels >180 mg/dl (>10mmol/l) with insulin titration; Severe hypoglycaemia (<50 mg/dl [<2.8mmol/l]) should be treated with intravenous dextrose or infusion of 10–20% glucose;
11. Give therapy directed at specific aspects of stroke pathogenesis (recanalization of vessel occlusion or prevention of mechanisms leading to expansion the infarct size).
12. No maintenance dextrose / lactate drip should be given in acute stroke. Only normal saline infusions should be given as maintenance infusion.

B.1.6.2. Specific treatment

Time goal: 60 min (from onset of stroke symptoms)

If the **patient is a candidate** for thrombolytic therapy (recombinant tissue plasminogen activator), review the risks and benefits of therapy with the patient and family (the main complication of IV tPA is intracranial hemorrhage) and give the tissue plasminogen activator.

Do not give anticoagulants or antiplatelet treatment for 24 hours after tPA until a follow-up CT scan at 24 hrs does not show intracranial hemorrhage.

If the **patient is NOT a candidate** for thrombolytic therapy, give the patient aspirin.

For both groups (those treated with tPA and those given aspirin), give the following basic stroke care:

Begin stroke pathway
Support patient's airway, breathing, and circulation
Check blood glucose
Watch for complications of stroke and fibrinolytic therapy
Transfer patient to intensive care if indicated

Patients with acute ischemic stroke who are hypoglycemic tend to have worse clinical outcomes, but there is no direct evidence that active glucose control improves outcomes. Consider giving IV or subcutaneous insulin to patients whose serum glucose levels are greater than 10 mmol/L (about 200 mg/dL).

B.1.6.2.1. Thrombolytic therapy

Use the thrombolytic checklist to screen candidates for thrombolytic therapy.

Table 10. Thrombolytic Checklist

Inclusion criteria	Exclusion criteria	Exclusion criteria
Age: 18 yrs or older	Evidence of intracranial hemorrhage from CT scan	Active internal bleeding or acute trauma, such as a fracture
Diagnosis of an ischemic stroke with neurologic deficit	Clinical presentation suggestive of a subarachnoid hemorrhage, even with normal CT	Acute bleeding diathesis, including the following but may include other manifestations: <ul style="list-style-type: none"> • Platelet count < 100,000/mm³ • Heparin received within 48 hours prior to onset of stroke, with elevated activated partial thromboplastin time (aPTT) • Current use of anticoagulant (e.g., warfarin) with an elevated international normalized ratio (INR) > 1.7
Time from onset of symptoms is within 3 hours	Evidence of multilobar infarction in more than one-third of the cerebral hemisphere on CT	Intraspinal surgery, serious head trauma, or previous stroke within the past 3 months
	History of intracranial hemorrhage	Arterial puncture at a non-compressible site within the past 7 days
	Uncontrolled hypertension based on repeated measurements of >185 mm Hg systolic pressure or > 110 mm Hg diastolic pressure	
	Known AV malformation, neoplasm, or aneurysm	
	Witnessed seizure at stroke onset	
Relative Contraindications/Precautions		
Minor or rapidly improving stroke symptoms		
Major surgery or serious trauma within the past 14 days		
Recent gastrointestinal or urinary tract hemorrhage within the past 3 weeks		
Post-myocardial infarction pericarditis		
Recent acute myocardial infarction within the past 3 months		
Abnormal blood sugar level < 50 mg/dl or > 400 mg/dl		

Complications. The major complication of IV tPA is intracranial hemorrhage. Other bleeding complications, ranging from minor to severe, may also happen. Angioderma and transient hypotension also can occur.

Research. Several studies have shown that good to excellent outcomes are more likely when tPA is given to adults with acute ischemic stroke within 3 hrs of onset of symptoms. However, these results happened when tPA was given in hospitals with a stroke protocol that adheres closely to the therapeutic regimen and eligibility requirements of the NINDS protocol. Evidence from prospective randomized studies in adults documented a greater likelihood of benefit when early treatment is started.

Table 11. **Intravenous infusion recombinant tissue plasminogen activator (rt-PA) in patients with an acute Ischemic stroke**

Intravenous infusion of rt-PA
<ul style="list-style-type: none"> • Alteplase (actilyse) 0.9 mg/kg, with 10% of the dose given as a bolus followed by a 60 min infusion, maximum dose is 90 mg; • Patient should be admitted to stroke unit or emergency department with a skilled care facility and close observation unit; • Neurological examination should be repeated during rt-PA infusion in every 15 min, following 6hrs in each 30 min, and next 24hrs in every 1 h • If patient's condition worsens during infusion of rt-PA, the infusion should be discontinued and send for rapid brain CT scan to detect possible bleeding; • Frequent checking of BP: after starting infusion for 2hrs in each 15 min, following 6hrs in each 30 min, next 24hrs in each 1 h • Other precautions see table 10 • CT scan to detect possible bleeding for any patient with neurological worsening after administration of rt-PA; • Management life-threatening hemorrhagic complications by (1) discontinuing ongoing infusion of rt-PA, (2) obtaining blood samples for coagulation testing, (3) obtaining surgical consultation as needed and (4) implementation of other interventions that may be useful (plasma transfusion, fibrinogen cryoprecipitate (6-8 units), platelets concentrate (6-8 units);

B.1.6.3. Management for Hypertension

B.1.6.3.1. Managing Hypertension in patients eligible or not eligible for thrombolytic therapy (tPA)

For patients who are candidates for thrombolytic therapy, control their blood pressure to lower their risk of intracerebral hemorrhage following administration of tPA.

Table 12. Candidates eligible for thrombolytic therapy

PRETREATMENT	
Systolic > 185 or diastolic > 110	Labetalol 10 to 20 mg IV for 1–2 min—may repeat 1 time or nitropaste 1–2 inches
During or after TREATMENT	Check blood pressure every 15 min for 2 hrs, then every 30 min for 6 hrs, and finally every hr for 16 hrs
Monitor blood pressure	Check blood pressure every 15 min for 2 hrs, then every 30 min for 6 hrs, and finally every hr for 16 hrs
Diastolic > 140	Sodium nitroprusside 0.5 µg/kg per minute IV infusion as initial dose and titrate to desired blood pressure
Systolic > 230 or diastolic 121 to 140	Labetalol 10 mg IV for 1–2 min—may repeat or double every 10 min to maximum dose of 300 mg or give initial labetalol dose and then start labetalol drip at 2 to 8 mg/min OR Nicardipine 5 mg/hr IV infusion as initial dose and titrate to desired effect by increasing 2.5 mg/hr every 5 min to maximum of 15 mg/hr; if blood pressure is not controlled by nicardipine, consider sodium nitroprusside
Systolic 180 to 230 or diastolic 105 to 120	Labetalol 10 mg IV for 1–2 min—may repeat or double every 10 to 20 min to a maximum dose of 300 mg or give initial labetalol dose, then start labetalol drip at 2 to 8 mg/min

Table 13. Candidates NOT eligible for thrombolytic therapy

Blood pressure level, mm Hg	Treatment
Systolic ≤220 or diastolic ≤120	Observe patient unless there is other end-organ involvement. Treat the patient's other symptoms of stroke (headache, pain, nausea, etc). Treat other acute complications of stroke, including hypoxia, increased intracranial pressure, seizures, or hypoglycemia.
Systolic > 220 or diastolic 121 to 140	Labetalol 10 to 20 mg IV for 1–2 min—may repeat or double every 10 min to a maximum dose of 300 mg OR Nicardipine 5 mg/hr IV infusion as initial dose; titrate to desired effect by increasing 2.5 mg/hr every 5 min to max of 15 mg/hr Aim for a 10% to 15% reduction in blood pressure
Diastolic > 140	Nitroprusside 0.5 µg/kg per min IV infusion as initial dose with continuous blood pressure monitoring
	Aim for a 10% to 15% reduction in blood pressure

B.1.6.4. Antiplatelet and anticoagulant therapy

1. Aspirin (81-325 mg/day, commonly 325 mg)
 - Initial treatment low grade stenosis in anterior or posterior circulation
 - Initial treatment in symptomatic high grade stenosis intracranial carotid artery, vertebrobasilar system, or extracranial carotid artery, if not candidate for warfarin or operation
 - Initial treatment in selected patients with transient ischemic attack (TIA) or minor stroke (nonsurgical candidate in anterior circulation ischemia)
2. Clopidogrel (75 mg/day)
 - Recurrent symptoms in spite of aspirin therapy /aspirin failure, contraindication to warfarin in setting of high grade arterial stenosis (nonsurgical candidate in anterior circulation ischemia) or cardiac source emboli
 - Initial treatment in selected patients with transient ischemic attack (TIA) or minor stroke (nonsurgical candidate in anterior circulation ischemia)
 - Allergic or sensitive to aspirin, requiring antiplatelet therapy
3. Warfarin anticoagulation (INR 2.0-3.0), short-term (3 months), followed by antiplatelet agent
 - Symptomatic low grade stenosis anterior and posterior circulation
 - Symptomatic occlusion of carotid, vertebral, or basilar artery with associated thromboembolic symptoms (use warfarin 4-6 wk or 3 months, INR 2.0-3.0)
 - Symptomatic high grade stenosis in intracranial carotid artery, vertebrobasilar system, or extracranial carotid artery, if not a surgical candidate
 - Cardiac source of emboli, level and duration of anticoagulation depending on cause
4. Heparin anticoagulation
 - Intravenous infusion in appropriate patients with cerebral infarction may be initiated with a bolus of 5000 U followed by constant infusion of 800-1000 U/h under the control of activated partial thromboplastin time (aPTT) with the normal control value 1.5-2.0 times - should be monitored every 6 hours until the therapeutic value has been documented and then daily during the time of infusion (hemorrhagic complications 1-7% in large ischemic stroke)
 - Treatment sometimes includes low-molecular-weight heparins (flaxiparine, calciparine), which have less side effects for causing complications than usual unfractionated heparin.
 - Low dose Enoxaparin (Lovenox) 30 mg sc daily for deep vein thrombosis prophylaxis among seriously ill patients.

C. Management of Spontaneous Intracerebral Hemorrhage (ICH)

Intracerebral hemorrhage occurs when the hemorrhage penetrates into the parenchyma of the brain and it most commonly caused by arterial hypertension and subsequent small vessel disease and amyloid angiopathy (primary ICH) or by vascular malformation (secondary ICH).

The clinical signs of ICH range from asymptomatic bleeding to sudden death. Immediate stages are:

- Mild to severe head ache
- Focal neurologic deficits
- Global cerebral deficits, ranging from mild disturbance of consciousness to coma

The pattern of clinical signs and secondary complications reflect the neurotopology and size of the ICH (hematoma in the cortex, subcortex, brain stem and cerebellum, or intraventricular

hemorrhages) and indicate prognosis. Clinically, ICH cannot be distinguished from cerebral ischemia, although some clues suggest one possibility more than the other. For example, ICH shows rapid onset, whereas cerebral ischemia may present with TIA. Signs of increased ICP and edema (headache, vomiting, hiccups, seizures, and disturbance of consciousness) may also be misleading and should not be considered in the differential diagnosis, although rapid progression of symptoms and impaired consciousness occur more often after ICH.

C.1. Diagnosis of ICH

- ICH is a medical emergency of the highest degree with frequent early neurological deterioration or death. Vomiting, early change in level of consciousness and high elevation of blood pressure in a patient with acute stroke suggest ICH.
- CT of the head is the imaging procedure of choice in the initial evaluation of suspected ICH.
- Angiography should be considered for all patients without a clear cause of hemorrhage who are surgical candidates, particularly young, normotensive patients who are clinically stable.
- Angiography is not required for older hypertensive patients who have a hemorrhage in the basal ganglia, thalamus, cerebellum, or brain stem and in whom CT findings do not suggest a structural lesion. Older patients with deep hemorrhages die or have severe morbidity related to the hemorrhage and are not candidates for angiography.
- Timing of cerebral angiography depends on the patient's clinical state and the neurosurgeon's judgment concerning the urgency of surgery, if needed.
- MRI and MRA are helpful and may obviate the need for contrast cerebral angiography in selected patients. They should also be considered to look for cavernous malformations in normotensive patients with lobar hemorrhages and normal angiographic results who are surgical candidates.

Algorithm:

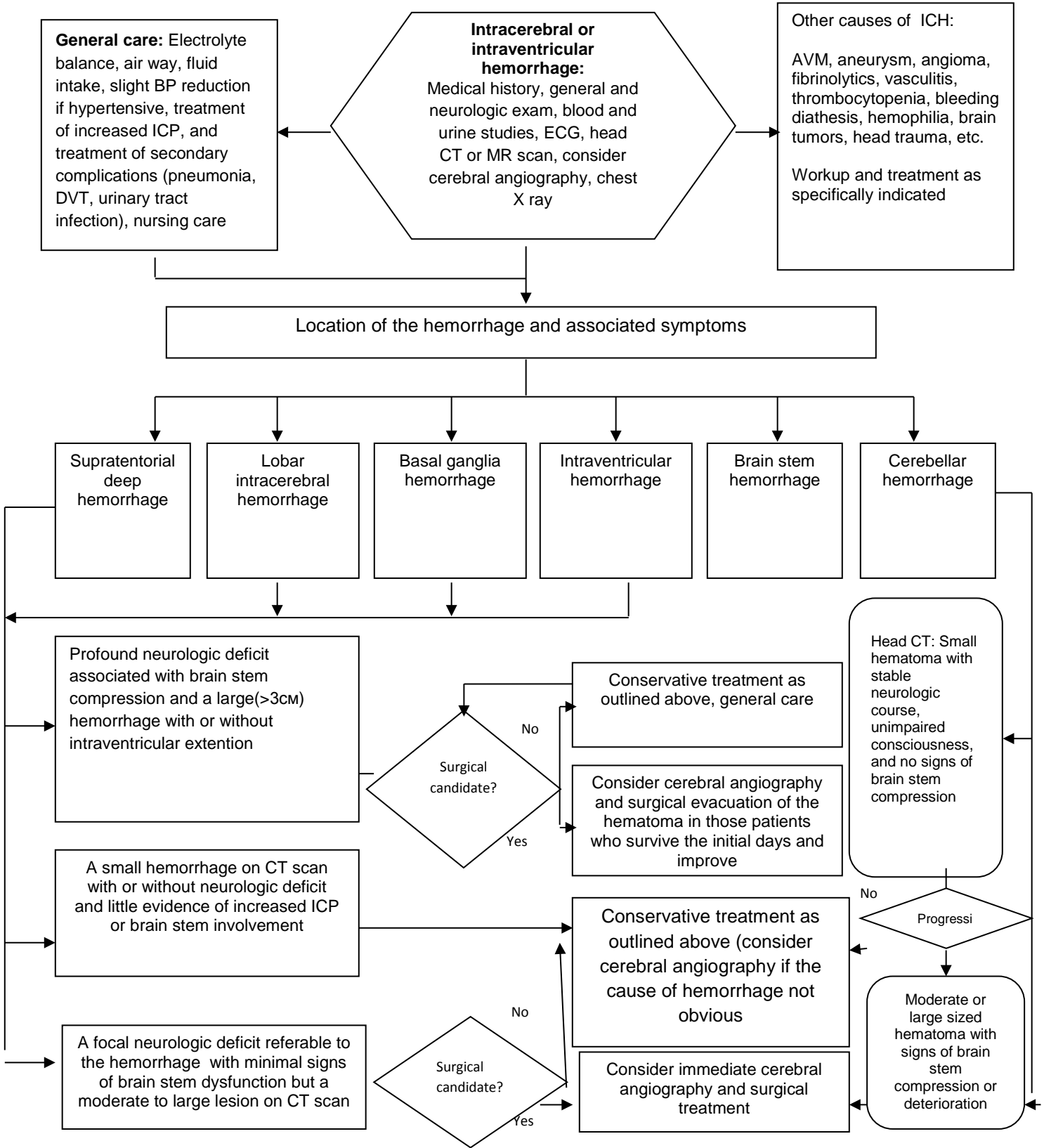


Table 14. Medical history, examination and diagnostic measures in the ED

	Explanation
History reveiw	
set the time when the patient was last known to be neurologically normal	
Initial symptoms, its progression	
Vascular risk factors	High blood pressure, diabetes mellitus, hyperlipidemia, smoking
Drug consumption	Anticoagulants (heparin, warfarin), antiplatelet drugs (aspirin, clopidogrel), decongestives, hypotensive drugs, weight lowering, sympathomimetic substances
Resent trauma, surgery	Carotid surgery, stent
Dementia	
Alcohol and narcotic substances	cocaine and other sympathomimetic substances
Seizures	
Liver disorders	Changes in coagulation factors
Cancer and hematological disorders	Changes in coagulation factors
Examination	
General condition and vital functions	Fever associated with early deterioration and complications The risk of stroke its complications and mortality is directly related to the degree of hypertension
Immediate and accurate neurologic examination	Needed assessment of severity of speech and motor impairments by NIH and NINDS scale, and unconsciousness by GCS
Blood and urine analysis	
Blood cell count, electrolytes, creatinine, urea, glucose, nitrogen	High creatinine and glucose levels may potentiate volume expanding hematoma and poor outcome
Coagulation factors: PT, INR, pATT	Warfarin associated hemorrhage may rapidly expand,so is high risk factor of morbidity and mortality
Toxicology test: check cocaine, sympathomimetic substances	Cocaine and other sympathomimetic substances mostly caused cerebral hemorrhages
Urine test	
Other analyses	
ECG	Assessment of heart failure and previous myocardial infarction, and evaluation of cardiac ejection fraction
Chest X-ray	
Brain CT or MRI	

C.2. Treatment of acute ICH

General principles of caring for patients with ICH

1. Place patient on bed rest in quiet room
2. Provide ongoing monitoring of neurologic status (level of consciousness, focal deficit, GCS)
3. Consider cardiac monitoring
4. Elevate head end of bed to 30 degrees
5. Prevent straining (stool softeners and antitussive agents as needed)
6. Provide oral nutrition for alert patients with intact gag reflex
7. Provide enteral nutrition with nasogastric tube for patients with decreased level of consciousness or impaired gag reflex
8. Maintain normovolemia and normal sodium level by starting with administration of 2-3 liters/day solutions of 0.9 normal saline
9. Mildly sedate patient if agitated (phenobarbital 30-60 mg 2 times a day)

10. Control mild pain with acetaminophen or propoxyphene and severe pain with codeine(60mg, IM or PO, every 3-4 hr); use morphine (1-2 mg IV) only as last resort
Reduce BP conservatively and careful monitoring if patient has extremely increased BP
12. Treat increased ICP as needed

C.2.1. Airway and Oxygenation

Although intubation is not required for all patients, airway protection and adequate ventilation are critical. Patients who exhibit a decreasing level of consciousness or signs of brain stem dysfunction are candidates for aggressive airway management. Intubation should be guided by imminent respiratory insufficiency rather than an arbitrary cutoff such as a specific Glasgow Coma Scale (GCS) score. Intubation is indicated for insufficient ventilation as indicated by hypoxia ($pO_2 < 60$ mm Hg or $PCO_2 > 50$ mm Hg) or obvious risk of aspiration with or without impairment of arterial oxygenation. Orotracheal intubation should be performed carefully, following institutional protocols such as maximal preoxygenation and administration of drugs to avoid reflex arrhythmias and/or blood pressure derangement, eg, atropine, thiopental, midazolam, propofol, and succinylcholine. Precautions should always be taken to prevent aspiration of gastric contents. All patients with endotracheal tubes receive nasogastric or orogastric tubes to prevent aspiration and are monitored for cuff pressure every 6 hours. Endotracheal tubes with soft cuffs can generally be maintained for ≤ 2 weeks. In the presence of prolonged coma or pulmonary complications, elective tracheostomy should be performed after ≈ 2 weeks. Oxygen should be administered to all patients presenting with a possible ICH.

C.2.2. Medical treatment

- In the ICH associated with coagulopathies and severe thrombocytopenia should be administrated appropriate coagulation factors and platelets concentrate (6-8 unit, IV infusion),
- In patients with ICH associated with warfarin therapy (increased INR) for cardioembolic cerebral infarction, warfarin should be discontinued and given vitamin "K" 10-20 mg, or fresh frozen plasma, or particularly recombinant activated factor VIIa within 3 hours of onset of symptoms,
- In heparin associated ICH, patient should be initiated on Protamine sulfate and its dosage is 1 mg to every 100 mg heparin and depended on discontinuation time of heparin infusion (protamine sulfate 5mg/min, IV, maximal dose less than 50mg),
- If ICH occurred in association with fibrinolytic therapy, necessary administration of blood transfusion, Recombinant activated Factor VII, cryoprecipitate, platelets (6-8 unit) etc may be initiated.

C.2.3. Blood Pressure Management

Table 15. Blood Pressure Management in ICH

High blood pressure	
Labetalol	5–100 mg/h by intermittent bolus doses of 10–40 mg or continuous drip (2–8 mg/min)
Esmolol	500 µg/kg as a load; maintenance use, 50–200 µg/kg/min
Nitroprusside	0.5–10 µg/kg/min
Hydralazine	10–20 mg Q 4–6 h

Enalapril

0.625–1.2 mg Q 6 h as needed

Low blood pressure

Volume replenishment is the first line of approach. Isotonic saline or colloids can be used and monitored with central venous pressure or pulmonary artery wedge pressure. If hypotension persists after correction of volume deficit, continuous infusions of pressors should be considered, particularly for low systolic blood pressure such as <90 mm Hg.

Phenylephrine	2–10 µg/kg/min
Dopamine	2–20 µg/kg/min
Norepinephrine	Titrate from 0.05–0.2 µg/kg/min

Nitroprusside, the most commonly used agent for severe elevations of blood pressure, is a vasodilatory agent that theoretically can increase cerebral blood flow and thereby intracranial pressure. This possible disadvantage has yet to be demonstrated in a clinical study.

C.2.4. Surgical Removal of ICH

The decision about whether and when to operate remains controversial. Patients with small hemorrhages (<10 mL) or minimal neurological deficits should be treated medically because they generally do well with medical treatment alone. Patients with a GCS score ≤4 should also be treated medically because they uniformly die or have extremely poor functional outcome that cannot be improved by surgery. Patients with cerebellar hemorrhage >3 cm in diameter who are neurologically deteriorating or who have brain stem compression and hydrocephalus from ventricular obstruction should have surgical removal of the hemorrhage as soon as possible. Stereotactic aspiration may be associated with better outcomes than standard craniotomy for moderate-sized cerebellar hemorrhages, but this hypothesis has yet to be tested in a randomized study. Young patients with large lobar hemorrhages (≥50 mL) who deteriorate during observation often undergo surgical removal of the hemorrhage. However, the efficacy of this approach is supported only by the small endoscopic study of Auer and colleagues. An ICH associated with a structural lesion such as an aneurysm or a vascular malformation may be removed if the patient has a chance for a good outcome and the structural vascular lesion is surgically accessible. Ultra-early removal of ICH by localized, minimally invasive surgical procedures is promising but untested.

Table 16. Recommendations for Surgical Treatment of ICH
Nonsurgical candidates

1. Patients with small hemorrhages (<10 mL) or minimal neurological deficits).
2. Patients with a GCS score ≤4. However, patients with a GCS score ≤4 who have a cerebellar hemorrhage with brain stem compression may still be candidates for lifesaving surgery in certain clinical situations.

Surgical candidates

-
1. Patients with cerebellar hemorrhage >3 cm who are neurologically deteriorating or who have brain stem compression and hydrocephalus from ventricular obstruction should have surgical removal of the hemorrhage as soon as possible.
 2. ICH associated with a structural lesion such as an aneurysm, arteriovenous malformation, or cavernous angioma may be removed if the patient has a chance for a good outcome and the structural vascular lesion is surgically accessible).
 3. Young patients with a moderate or large lobar hemorrhage who are clinically deteriorating. Best therapy is still unclear.
-

D. Management of Aneurysmal Subarachnoid Hemorrhage

Subarachnoid hemorrhage occurs when the site of hemorrhage is in the subarachnoid spaces surrounding the brain. Aneurysmal SAH is caused by rupture of arterial aneurysm and AVM, and other etiological factors. Therefore SAH is divided into clinical forms: Aneurysmal SAH and non-aneurysmal SAH.

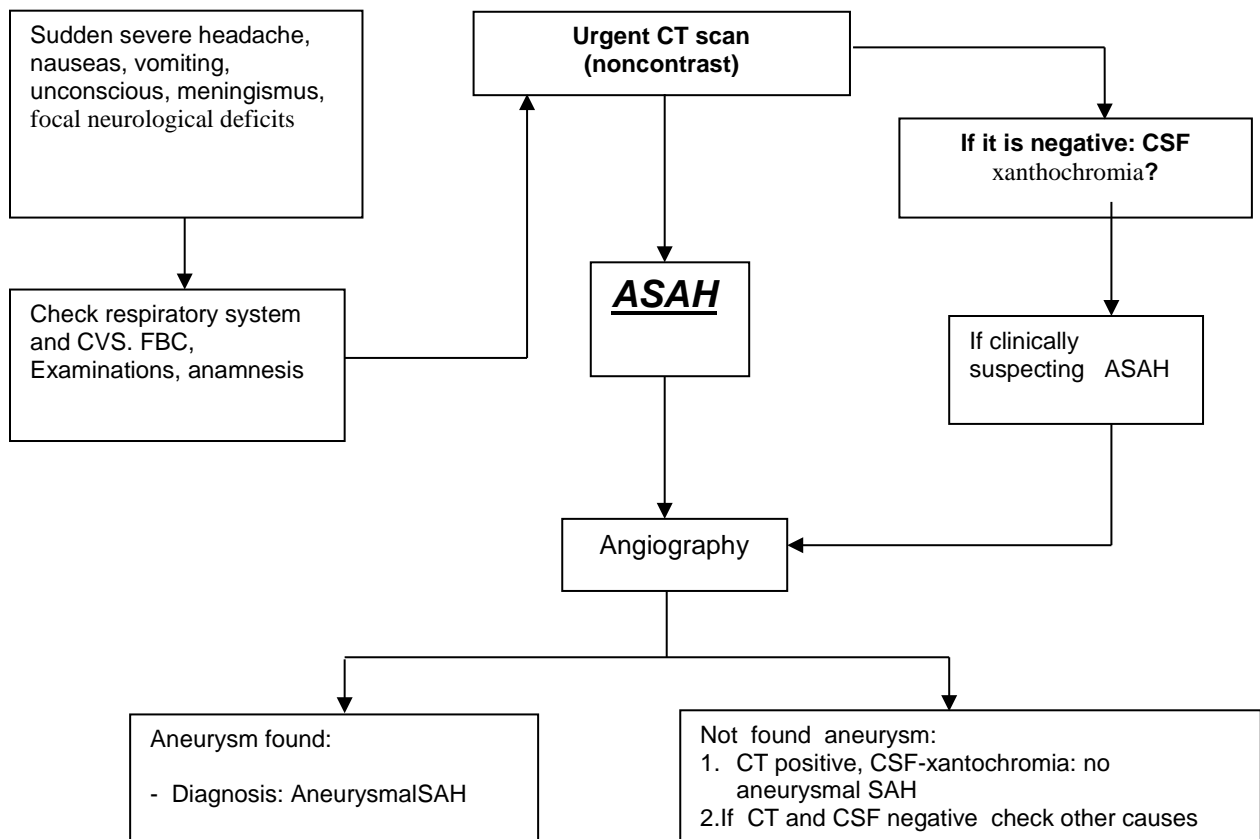
- Arterial aneurysm -80%,
- AVM -5% (angioma, dural arteriovenous fistula),
- Other rare causes-5% (hypertension, head trauma, arteritis, dissection, coagulation disorders, hematologic diseases, sickle cell anemia),
- Unknown causes- 10%
- Non-aneurysmal SAH, mostly in the perimesencephalic cisterns - 10%

D.1. Manifestations and Diagnosis of SAH

- SAH is a medical emergency that is frequently misdiagnosed. A high level of suspicion for SAH should exist in patients with acute onset of severe headache.
- CT scanning for suspected SAH should be performed, and lumbar puncture for analysis of CSF is strongly recommended when the CT scan is negative.
- Selective cerebral angiography should be performed in patients with SAH to document the presence and anatomic features of aneurysms.
- MRA and CTA may be considered when conventional angiography cannot be performed in a timely fashion.

Table 17. Clinical grades of SAH

World Federation of Neurosurgery, SAH grading scale			Hunt-Hess scale	
I	GCS score	Motor deficit	Grade	Criteria
I	15	Absent	I	Asymptomatic, minimal headache, stiff neck
II	14-13	Absent	II	Moderate to severe headache, stiff neck, cranial nerve palsy, no focal neurologic symptoms
III	14-13	Present	III	Confusion, slight neurologic symptoms
IV	12-7	Present or absent	IV	Stupor, moderate-severe hemiparesis, possibly early decerebrate signs
V	6-3	Present or absent	V	Deep coma

Management algorithm 1

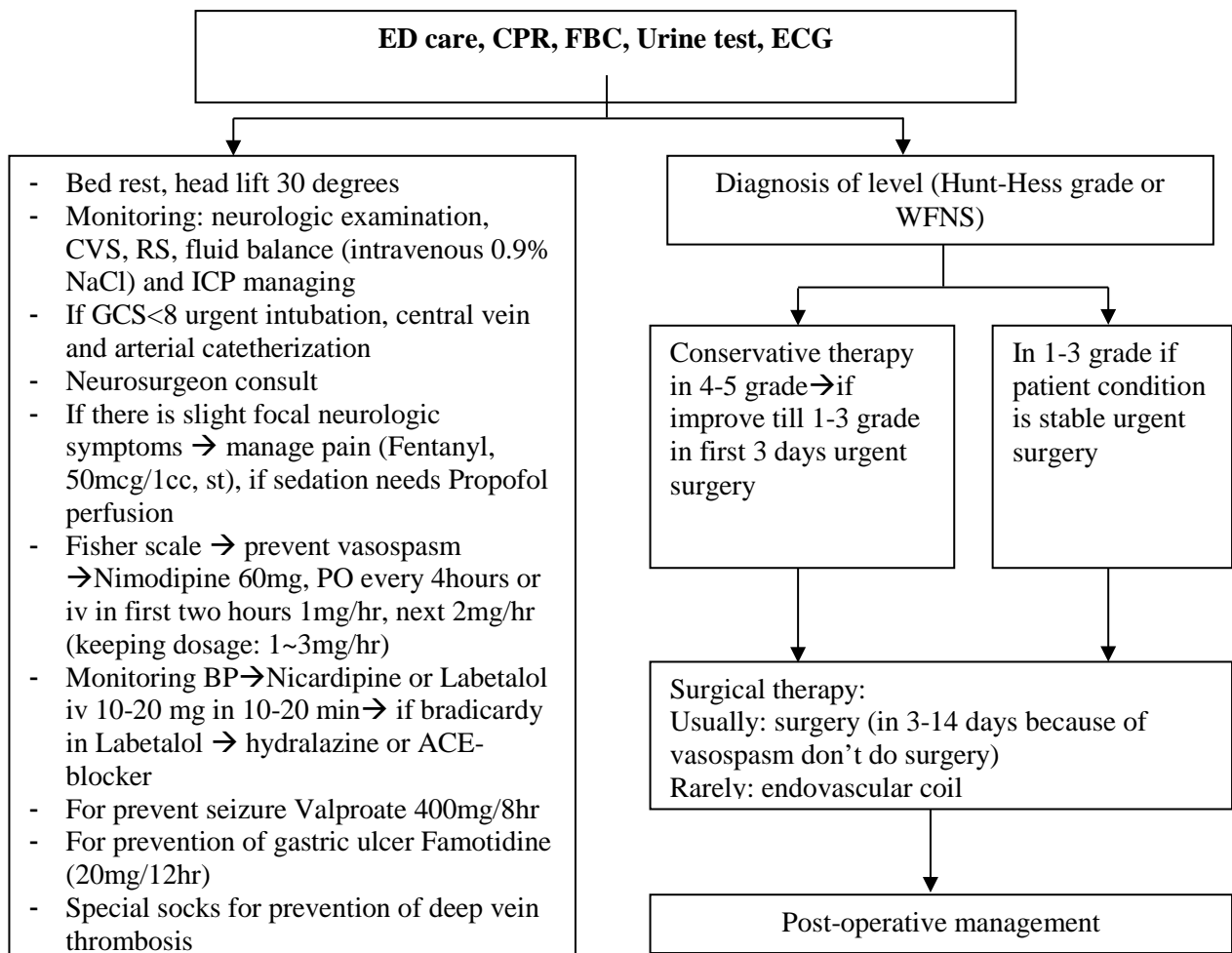
D.2. Medical management of SAH

General principles of caring for patients with SAH

1. Place patient on bed rest in quiet room
2. Provide ongoing monitoring of neurologic status (level of consciousness, focal deficit, GCS)
3. Consider cardiac monitoring
4. Elevate head end of bed 30 degrees
5. Prevent straining (stool softeners and antitussive agents as needed)
6. Provide oral nutrition for alert patients with intact gag reflex
7. Provide enteral nutrition with nasogastric tube for patients with decreased level of consciousness or impaired gag reflex
8. Maintain normovolemia and normal sodium level by starting with administration of 2-3liters/day solutions of 0.9 normal saline
9. Mildly sedate patient if agitated (phenobarbital 30-60 mg 2 times a day)
10. Control mild pain with acetaminophen or propoxyphene and severe pain with codeine(60mg, IM or PO, every 3-4 hr); use morphine (1-2 mg IV) only as last resort
11. Reduce BP conservatively and careful monitoring if patient has extremely increased BP
12. Treat increased ICP as needed

The following algorithm shows a general guidance of treatment in SAH

Treatment algorithm



D.3. Surgical or Endovascular Treatment of Ruptured Aneurysms

1. Surgical clipping or endovascular coiling should be performed to reduce the rate of re-bleeding after aneurysmal SAH.
2. Wrapped or coated aneurysms and incompletely clipped or coiled aneurysms have an increased risk of re-hemorrhage compared with those that are completely occluded and therefore require long-term follow-up angiography. Complete obliteration of the aneurysm is recommended whenever possible.
3. For patients with ruptured aneurysms judged by an experienced team of cerebrovascular surgeons and endovascular practitioners to be technically amenable to both endovascular coiling and neurosurgical clipping, endovascular coiling can be beneficial. Nevertheless, it is reasonable to consider individual characteristics of the patient and the aneurysm in deciding the best means of repair, and management of patients in centers offering both techniques is probably indicated.
4. Although previous studies showed that overall outcome was not different for early versus delayed surgery after SAH, early treatment reduces the risk of re-bleeding after SAH, and newer methods may increase the effectiveness of early aneurysm treatment. Early aneurysm treatment is reasonable and is probably indicated in the majority of cases.

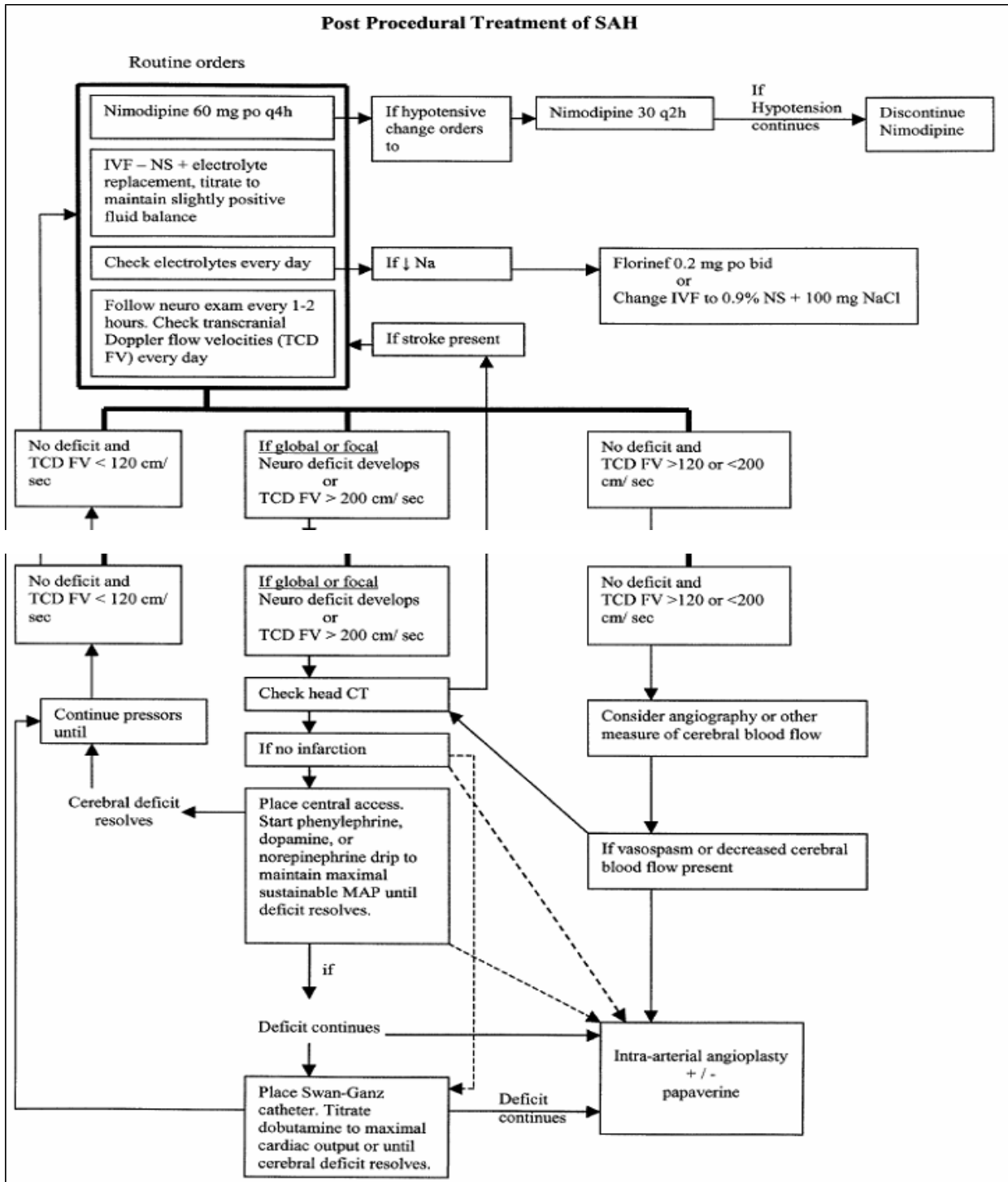
D.4. Medical Measures to Prevent Re-bleeding After SAH

- Blood pressure should be monitored and controlled to balance the risk of stroke, hypertension-related re-bleeding, and maintenance of cerebral perfusion pressure.
- Bed rest alone is not enough to prevent re-bleeding after SAH. It may be considered a component of a broader treatment strategy, along with more definitive measures.
- Although older studies demonstrated an overall negative effect of antifibrinolytics, recent evidence suggests that early treatment with a short course of antifibrinolytic agents (EACA 24-36 g/day 1000ml of 5% dextrose solution, or tranexamic acid 1 g IV or 1.5 g orally 4-6 times daily) combined with a program of early aneurysm treatment followed by discontinuation of the antifibrinolytic and prophylaxis against hypovolemia and vasospasm may be reasonable, but further research is needed. Furthermore, antifibrinolytic therapy to prevent rebleeding may be considered in certain clinical situations, in patients with a low risk of vasospasm and/or a beneficial effect of delaying surgery.

D.5. Management of Cerebral Vasospasm

- Oral nimodipine is indicated to reduce poor outcome related to aneurysmal SAH. The value of other calcium antagonists, whether administered orally or intravenously, remains uncertain.
- Treatment of cerebral vasospasm begins with early management of the ruptured aneurysm, and in most cases, maintaining normal circulating blood volume and avoiding hypovolemia are probably indicated.
- One reasonable approach to symptomatic cerebral vasospasm is volume expansion, induction of hypertension, and hemodilution (triple-H therapy).

- Alternatively, cerebral angioplasty and/or selective intra-arterial vasodilator therapy may be reasonable after, together with, or in the place of triple-H therapy, depending on the clinical scenario.



E. Management of complications in Strokes

E.1. Therapy of elevated Intracranial Pressure and Hydrocephalus

ICP is considered a major contributor to mortality after Stroke; thus, its control is essential. ICP may be managed through osmotherapy, controlled hyperventilation, and barbiturate coma.

Elevated ICP is defined as intracranial pressure ≥ 20 mm Hg for >5 minutes. A therapeutic goal for all treatment of elevated ICP is ICP < 20 mm Hg and cerebral perfusion pressure (CPP) > 70 mm Hg. Optimal head position (raised 15-30) can be adjusted according to ICP values. Patients with suspected elevated ICP and deteriorating level of consciousness are candidates for invasive ICP monitoring. The GCS level that requires ICP monitoring should be based on rate of decline and other clinical factors such as CT evidence of mass effect and hydrocephalus. In general, ICP monitors should be placed in patients with a GCS score of < 9 and all patients whose condition is thought to be deteriorating due to elevated ICP. The type of device depends on availability, experience, and situation. Intraventricular ICP monitors and intraparenchymal fiberoptic ICP devices are 2 commonly used methods of monitoring ICP.

In addition to the mass effect of the hematoma, secondary hydrocephalus may contribute to elevated ICP. Ventricular drains should be used in patients with or at risk for hydrocephalus. Drainage can be initiated and terminated according to clinical performance and ICP values. Because of infectious complications, external drainage devices must be checked regularly, and duration of placement ideally should not exceed 7 days. Use of anti-infectious prophylaxis is recommended.

Although universally accepted standardized therapy protocols for elevated ICP have not been established, The beneficial effect of *sustained* hyperventilation on ICP is unresolved. In theory, reduction of ICP by hyperventilation ceases when the pH of cerebrospinal fluid (CSF) reaches equilibrium. In practice, this may not occur for many hours. Some authors believe that prolonged hyperventilation has a beneficial effect on brain water volume. As with osmotherapy, adverse rebound effects can occur if normal ventilation is resumed too quickly. When hyperventilation is deemed no longer necessary, gradual normalization of serum PCO_2 should occur over a 24- to 48-hour period. In general, if hyperventilation is instituted for elevated ICP, PCO_2 should be maintained between 30 and 35 mm Hg until ICP is controlled. In addition, most patients will require sedation with agents such as *propofol*, *benzodiazepines*, or *morphine* and treatment with intermittent muscular paralysis.

E.1.2. Management of ICP

- **Osmotherapy:** The first medical line of defense is osmotherapy. However, it should not be used prophylactically. Mannitol 20% (0.25–0.5 g/kg every 4 h) or glycerol (10%, 250ml every 6 h) are reserved for patients with progressively increasing ICP values, or clinical deterioration associated with mass effect. Due to its rebound phenomenon, mannitol is recommended for only ≤ 5 days. To maintain an osmotic gradient, furosemide (10 mg Q 2–8 h) may be administered simultaneously with osmotherapy. Intravenous hypertonic saline solutions (NaCl 23.4%, 30ml) are probably similarly effective. Serum osmolality should be measured twice daily in patients receiving osmotherapy and targeted to ≤ 310 mOsm/L.
- **Corticosteroids** in stroke are generally avoided because multiple potential side effects must be considered and clinical studies have not shown benefit.

- **Hyperventilation:** Hypocarbica causes cerebral vasoconstriction. Reduction of cerebral blood flow is almost immediate, although peak ICP reduction may take up to 30 minutes after pCO₂ is changed. Reduction of pCO₂ to 35–30 mm Hg, best achieved by raising ventilation rate at constant tidal volume (12–14 mL/kg), lowers ICP 25% to 30% in most patients. Failure of elevated ICP to respond to hyperventilation indicates a poor prognosis.
- **Muscle relaxants:** Neuromuscular paralysis in combination with adequate sedation can reduce elevated ICP by preventing increases in intrathoracic and venous pressure associated with coughing, straining, suctioning, or "bucking" the ventilator. Nondepolarizing agents, such as vecuronium or pancuronium, with only minor histamine liberation and ganglion-blocking effects, are preferred in this situation. Patients with critically elevated ICP should be pretreated with a bolus of a muscle relaxant before airway suctioning. Alternatively, lidocaine may be used for this purpose.

- If elevated ICP cannot be controlled with the above mentioned treatment alternatives, induced **barbiturate coma** may be instituted. However, high-dose barbiturate therapy should be viewed as an option and not part of a standardized algorithm in the treatment of elevated ICP in patients with stroke. Short-acting barbiturates such as *thiopental* are known to effectively reduce elevated ICP. The effect is presumably mediated through reduction of cerebral blood flow and volume. In addition to reducing the volume of the normal brain, barbiturates reduce brain swelling, perhaps as a result of mild systemic hypotension, and may act as free radical scavengers. The complications of high-dose barbiturate administration (safe limit: ≈10 mg/kg per day) include hypotension, which is most pronounced at the time of bolus administration, and possible predisposition to infection. Systemic hypotension mainly results from decreased venous tone, baroreflex tone, and sympathetic activity. Cardiovascular side effects may be aggravated by concomitant dehydration promoted by osmotherapy and diminished cardiac filling pressures. Maximal reduction in cerebral metabolism is accompanied by electrocerebral silence (continuous EEG recording). Since some tolerance develops with continued administration of barbiturate, use of multiple small boluses may be considered (0.3 to 0.6 mg/kg).

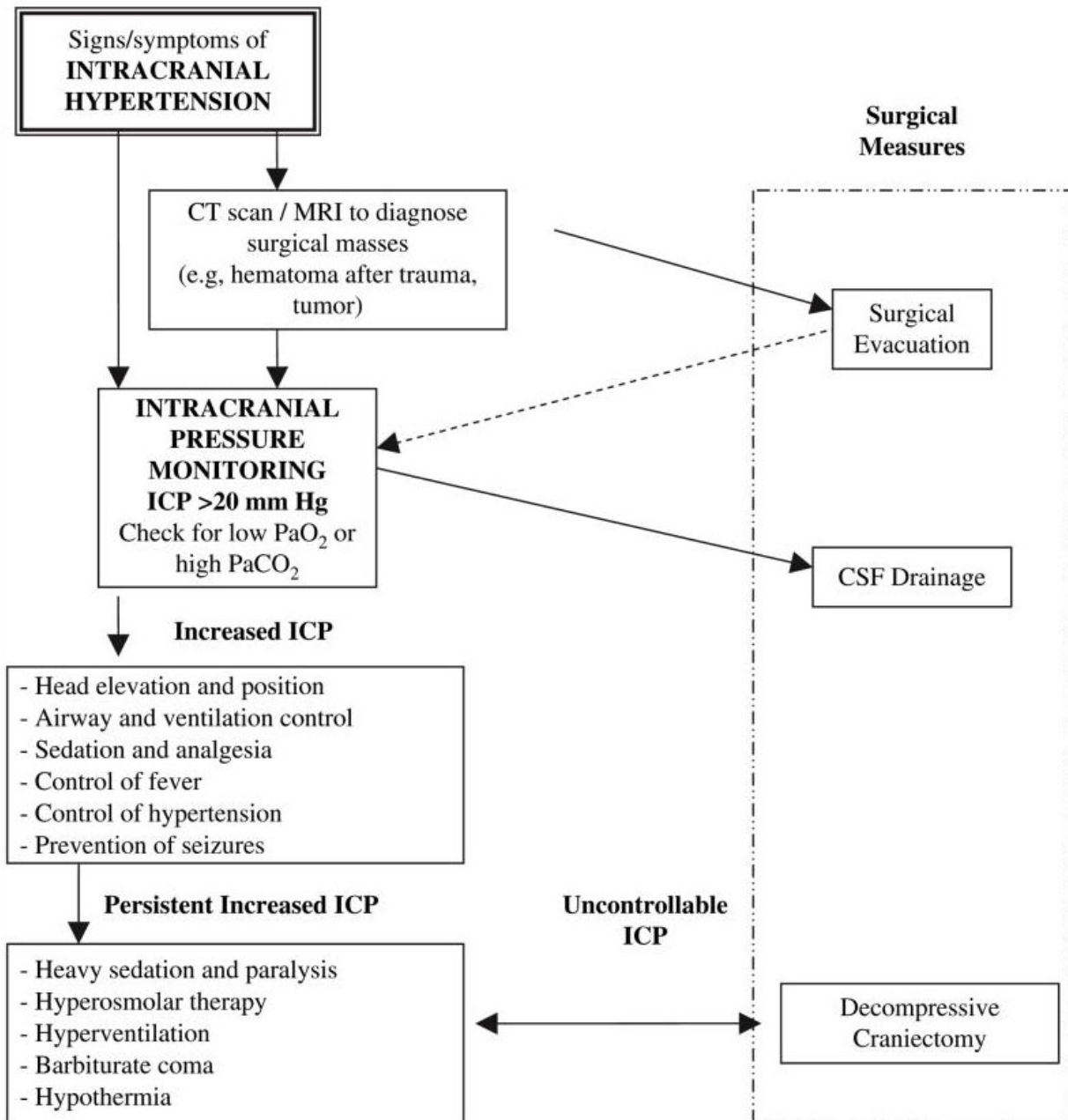
Management of Hydrocephalus:

- Temporary or permanent CSF diversion is recommended by external ventricular drainage in symptomatic patients with chronic hydrocephalus after SAH.
- Ventriculostomy can be beneficial in patients with hydrocephalus and diminished level of consciousness after acute SAH.

Management of Seizures

- The administration of prophylactic anticonvulsants may be considered in the immediate post-hemorrhagic period
- The routine long-term use of anticonvulsants is not recommended but may be considered for patients with risk factors such as prior seizure, parenchymal hematoma, infarct, or middle cerebral artery aneurysms

Algorithm for management ICP



Management of Hyponatremia

- Avoid increased use of hypotonic solutions
- Test for any changes in antidiuretic hormone level and sodium loss syndromes. (Hypotonic hyponatremia, extracellular dehydration)
- If sodium loss syndromes caused by hypovolemia: for correcting hyponatremia use isotonic sodium solution, Ringer lactate or colloid solutions (central vein pressure must be 8-12mm Hg)
- If there are changes in antidiuretic hormone levels, reduce intake of fluids (less than 1 litre per day) and use Lasix(Feruosimide) daily 40mg or Demeclocycline 300-600 mg twice a day PO

- Decide about including in the complex therapy Fludrocortisone Acetate by 1mg twice a day.
- Decide in rare case of hyponatremia (<120 mEq/l) use of 3% sodium chloride 25-50ml/hr.
- Avoid quick correction of sodium level in hyponatremia (during 24hours ≤ 20 mEq/l or 1,5-2mEq/l/hr) , to avoid potential danger of central pontine myelinolysis (CPM) with rapid correction.

E.2. Prevention and management of other complications in Strokes

- Infections after strokes should be treated with appropriate antibiotics. Prophylactic administration of antibiotics is not recommended;
- Early rehydration and graded compression stockings / pneumatic compression devices are recommended to reduce the incidence of venous thromboembolism;
- Early mobilization is recommended to prevent complications such as aspiration pneumonia, DVT and pressure ulcers;
- It is recommended that low-dose subcutaneous heparin or low molecular weight heparins should be considered for patients at high risk of DVT or pulmonary embolism;
- Administration of anticonvulsants is recommended to prevent recurrent post-stroke seizures. Prophylactic administration of anticonvulsants to patients with recent stroke who have not had seizures is not recommended;
- An assessment of falls risk is recommended for every stroke patient;
- Calcium/vitamin-D supplements are recommended in stroke patients at risk of falls;
- In stroke patients with urinary incontinence, specialist assessment and management is recommended;
- Swallowing assessment is recommended as a routine, but there are insufficient data to recommend a specific approach for treatment;
- Oral dietary supplements are only recommended for non-dysphagic stroke patients who are malnourished;
- Early commencement of nasogastric (NG) feeding (within 48 hours) is recommended in stroke patients with impaired swallowing;
- It is recommended that percutaneous enteral gastrostomy (PEG) feeding should not be considered in stroke patients in the first 2 weeks.

F. Rehabilitation (see special guideline worked out on it)

Even with optimal stroke unit care including thrombolysis, fewer than one third of patients recover fully from stroke. Rehabilitation aims to enable people with disabilities to reach and maintain optimal physical, intellectual, psychological and/or social function. Goals of rehabilitation can shift from initial input to minimize impairment to more complex interventions designed to encourage active participation.

Setting for rehabilitation

- Admission to a stroke unit with rehabilitation facility is recommended for acute stroke patients to receive coordinated multidisciplinary rehabilitation;
- Early initiation of rehabilitation is recommended;

- It is recommended that early discharge from stroke unit is possible in medically stable patients with mild or moderate impairment provided that rehabilitation is delivered in the community by a multidisciplinary team with stroke expertise;
- Tricyclic or anticonvulsant therapy are recommended to treat post-stroke neuropathic pain in selected patients;
- Drug therapy and non-drug interventions are recommended to improve cognitive function and mood in depressive patients;
- It is recommended to continue rehabilitation after discharge during the first year after stroke;
- It is recommended to increase the duration and intensity of rehabilitation;

The results from stroke unit trials favor coordinated multidisciplinary teams of staff with expertise in stroke care. The composition of these teams is not formally prescribed, but usually includes stroke physicians, neurosurgeons, physiatrists, neuroradiologists (including interventionists), vascular surgeons, nutritionists, cardiologists, nursing staff, physiotherapists, occupational therapists, and speech and language therapists.

H. Prevention

H.1. Primary prevention

The aim of primary prevention is to reduce the risk of stroke in asymptomatic people.

Management of vascular risk factors

- Provide educational programs to increase awareness of stroke at the population level;
- Provide educational programs to increase stroke awareness among professionals (paramedics/emergency physicians);
- Provide a community awareness for prevention of stroke and urgent referral to emergency medical service if stroke occurs;
- Blood pressure should be checked regularly. It is recommended that high blood pressure should be managed with lifestyle modification and individualized pharmacological therapy aiming at normal levels of 120/80 mmHg. For prehypertensive (120-139/80-90 mmHg) with congestive heart failure, MI, diabetes, or chronic renal failure antihypertensive medication is indicated;
- The relationship between hypertension and aneurysmal SAH is uncertain. However, treatment of high blood pressure with antihypertensive medication is recommended to prevent ischemic stroke, intracerebral hemorrhage, and cardiac, renal, and other end-organ injury.
- Careful control of the anticoagulation level in patient's warfarin therapy reduces the risk of subsequent ICH.
- Careful selection of patients for thrombolytic treatment for acute myocardial infarction and acute ischemic stroke should result in a decline in ICH rates.
- Blood glucose should be checked regularly. It is recommended that diabetes should be managed with lifestyle modification and individualized pharmacological therapy. In diabetic patients, high blood pressure should be managed intensively aiming for levels below 130/80 mmHg. Where possible, treatment should include an angiotensin converting enzyme inhibitor or angiotensin receptor antagonist;

- Blood cholesterol should be checked regularly. It is recommended that high blood cholesterol (e.g. LDL > 150 mg/dl [3.9 mmol/l]) should be managed with lifestyle modification and a statin;
- It is recommended to quit cigarette smoking;
- Regular physical activity is recommended;
- A diet low in salt and saturated fat, high in fruit and vegetables and rich in fiber is recommended; avoidance of heavy alcohol and use of sympathomimetic drugs may decrease risk of ICH
- Subjects with an elevated body mass index are recommended to take a weight-reducing diet;
- Antioxidant vitamin supplements are not recommended;
- Hormone replacement therapy is not recommended for the primary prevention of stroke;

A healthy lifestyle, consisting of abstinence from smoking, low-normal body mass index, regular exercise and healthy diet, is associated with a reduction in strokes.

Antithrombotic therapy

- Low-dose aspirin is recommended in women aged 45 years or more who are not at increased risk for intracerebral hemorrhage and who have good gastro-intestinal tolerance;
- It is recommended that low-dose aspirin may be considered in men for the primary prevention of myocardial infarction; however, it does not reduce the risk of ischemic stroke;
- Antiplatelet agents other than aspirin are not recommended for primary stroke prevention;
- Aspirin may be recommended for patients with non-valvular AF who are younger than 65 years and free of vascular risk factors;
- Unless contraindicated, either aspirin or an oral anticoagulant (international normalized ratio [INR] 2.0-3.0) is recommended for patients with non-valvular AF who are aged 65-75 years and free of vascular risk factors;
- Unless contraindicated, an oral anticoagulant (INR 2.0–3.0) is recommended for patients with non-valvular AF who are aged >75, or who are younger but have risk factors such as high blood pressure, left ventricular dysfunction, or diabetes mellitus;
- It is recommended that patients with AF who are unable to receive oral anticoagulants should be offered aspirin;
- It is recommended that patients with AF who have mechanical prosthetic heart valves should receive long-term anticoagulation with a target INR based on the prosthesis type, but not less than INR 2.0–3.0;
- Low dose aspirin is recommended for patients with asymptomatic internal carotid artery (ICA) stenosis >50% to reduce their risk of vascular events;

Carotid surgery and angioplasty

- Carotid surgery is not recommended for asymptomatic individuals with significant carotid stenosis (60-99%), except in those at high risk of stroke;
- Carotid surgery is recommended for symptomatic individuals with significant carotid stenosis (>70%),

- Carotid angioplasty, with or without stenting, is not recommended for patients with asymptomatic carotid stenosis;
- It is recommended that patients should take aspirin before and after surgery;

H.2. Secondary prevention

Optimal management of vascular risk factors

- It is recommended that blood pressure be checked regularly. Blood pressure lowering is recommended after the acute phase of strokes;
- It is recommended that blood glucose should be checked regularly. It is recommended that diabetes should be managed with lifestyle modification and individualized pharmacological therapy;
- In patients with type 2 diabetes who do not need insulin, treatment with pioglitazone is recommended after stroke;
- Statin therapy is recommended in subjects with non-cardioembolic stroke;
- It is recommended to quit cigarette smoking;
- It is recommended that heavy use of alcohol be discouraged;
- Regular physical activity is recommended;
- A diet low in salt and saturated fat, high in fruit and vegetables, and rich in fiber is recommended;
- Subjects with an elevated body mass index are recommended to adopt a weight-reducing diet;
- Antioxidant vitamin supplements are not recommended;
- Hormone replacement therapy is not recommended for the secondary prevention;
- Sleep-disordered breathing such as obstructive sleep apnoea is recommended to be treated with continuous positive airway pressure breathing;
- It is recommended that endovascular closure of PFO be considered in patients with cryptogenic stroke and high risk PFO;

Antithrombotic therapy

- It is recommended that patients receive antithrombotic therapy;
- It is recommended that patients not requiring anticoagulation should receive antiplatelet therapy. Where possible, combined aspirin and dipyridamole, or clopidogrel alone, should be given;
- The combination of aspirin and clopidogrel is not recommended in patients with recent ischemic stroke, except in patients with specific indications (e.g. unstable angina or non-Q-wave MI, or recent stenting); treatment should be given for up to 9 months after the event;
- It is recommended that patients who have a stroke on antiplatelet therapy should be re-evaluated for pathophysiology and risk factors;
- Oral anticoagulation (Warfarin 5mg; INR 2.0–3.0) is recommended after ischemic stroke associated with AF. Oral anticoagulation is not recommended in patients with co-morbid conditions such as falls, poor compliance, uncontrolled epilepsy, or gastrointestinal bleeding. Increasing age alone is not a contraindication to oral anticoagulation;
- It is recommended that patients with cardioembolic stroke unrelated to AF should receive anticoagulants (INR 2.0-3.0) if the risk of recurrence is high;

- It is recommended that anticoagulation should not be used after non-cardio-embolic ischemic stroke, except in some specific situations, such as aortic atheromas, fusiform aneurysms of the basilar artery, cervical artery dissection, or patent foramen ovale in the presence of proven deep vein thrombosis (DVT) or atrial septal aneurysm;
- It is recommended that combined low dose aspirin and dipyridamole should be given if oral anticoagulation is contraindicated;

Surgery and angioplasty

- Carotid endarterectomy (CEA) is recommended for patients with 70–99% stenosis. CEA should only be performed in centers with a perioperative complication rate (all strokes and death) of less than 6%;
- It is recommended that CEA be performed as soon as possible after the last ischemic event, ideally within 2 weeks;
- It is recommended that CEA may be indicated for certain patients with stenosis of 50–69%; males with very recent hemispheric symptoms are most likely to benefit. CEA for stenosis of 50–69% should only be performed in centers with a perioperative complication rate (all stroke and death) of less than 3%;
- CEA is not recommended for patients with stenosis of less than 50%;
- It is recommended that patients remain on antiplatelet therapy both before and after surgery;
- Carotid percutaneous transluminal angioplasty and/or stenting (CAS) is only recommended in selected patients. It should be restricted to the following subgroups of patients with severe symptomatic carotid artery stenosis: those with contraindications to CEA, stenosis at a surgically inaccessible site, re-stenosis after earlier CEA, and post-radiation stenosis. Patients should receive a combination of clopidogrel and aspirin immediately before and for at least 1 month after stenting;
- It is recommended that endovascular treatment may be considered in patients with symptomatic intracranial stenosis;

I. Application of the guidelines for management of stroke In each level of medical organizations

The using of the guidelines for management of acute stroke into each level of medical organizations should be needed to provide the next requirements (table 17).

Table 17. Requirements for managing acute stroke patients in each hospital level

Requirements for managing acute stroke patients	State central hospitals (3rd level)	Zonal diagnostic centers, aimag and district hospitals (2nd level)	Soumon hospitals (1st level)
Emergency medical service (Recognize the signs of stroke using the "Face-Arm-Speech Test)	Transport the patient quickly to stroke unit or emergency department	Transport the patient quickly to neurological or emergency department	Transport the patient to primary care unit
Investigation and diagnostic actions	Perform neurologic and cardiologic assessments; Brain CT or MRI, ECG, TCD; Lab.tests: complete blood analysis and platelet count, PT or INR, PTT, Serum electrolytes, blood glucose, CRP or ESR, Hepatic and renal chemical analysis	Perform neurologic and cardiologic assessments; Brain CT, ECG, TCD; Lab.tests: complete blood analysis and platelet count; PT or INR, PTT, Serum electrolytes, blood glucose, CRP or ESR, Hepatic and renal chemical analysis	Recognize the signs of stroke using the "Face-Arm-Speech Test; Lab.tests: complete blood analysis and platelet count; Order general treatment for stroke; Obtain neurologist consultation and establish close observation
Acute therapy	Depending on stroke type and time of symptoms onset start general and specific appropriate treatment (thrombolysis or antiplatelet, or anticoagulant therapy, and neurosurgery intervention)	Depending on stroke type and time of symptoms onset start general and specific appropriate treatment (antiplatelet, or anticoagulant therapy, and neurosurgery intervention)	Depending on stroke type and time of symptoms onset start general and specific appropriate treatment (antiplatelet therapy)
Primary and second prevention	Perform by the guidelines for management of stroke		

References

1. Баасанжав Д.Улаанбаатар хотын хүн амын дунд тархины харвалтын эпидемиологи: судалгааны дүн, сүүлийн 30 жилийн хандлага, Baasanjav D. Epidemiology of stroke in population of Ulaanbaatar, Улаанбаатар, 2005, 81 p.
2. Цагаанхүү Г.Мэдрэл судлал (эмч нарын сурах бичиг), Tsagaankhuu G. Textbook of Neurology, Ulaanbaatar, 2011, p. 344-389
3. Мөнхбаяр Р., Сарангэрэл Ж., Цагаанхүү Г. Тархины цус хомсрох хамшинжийн үеийн гемодинамик өөрчлөлтийг доплерографийн аргаар судалсан нь. “Эрүүл мэндийн шинжлэх ухаан ухаан” сэтгүүл, 2005, 1, х. 59-61.
4. Цагаанхүү Г., Мөнхбаяр Р., Сарангэрэл Ж.: Эрүүл монгол хүний тархины судасны доплерографийн үзүүлэлтүүдийн насны хамаарал. Монголын анагаах ухаан, 2005, 2, х3-6.
5. Adams HP Jr, Davis PH, Leira EC, Chang KC, Bendixen BH, Clarke WR, et al. Baseline NIH Stroke Scale score strongly predicts outcome after stroke: A report of the Trial of Org 10172 in Acute Stroke Treatment (TOAST). *Neurology*. Jul 13 1999;53(1):126-31.
6. Adams and Victor's Principles of Neurology. 8-th edition. A.H.Ropper, R.H.Brown New York, McGaw-Hill, 2005, p. 660-746
7. Alan Guberman, An Introduction to Clinical Neurology. Boston, New York, Toronto, London, 1994, p. 373-418.
8. David A.Greenberg, Michael J. Aminoff, Roger P. Simon. Clinical Neurology. New York, Chicago, London, McGaw-Hill, 2002, p. 381-425
9. David O.Wiebers, Valery L.Feigin, Robert D.Brown. Handbook of Stroke (Russian translated version). Rochester, Minnesota, 1997, Бином, Moscow., 2005, 608 p.
10. Guidelines for Management of Ischemic Stroke and Transient Ischemic Attack 2008. The European Stroke Organization (ESO), Executive Committee and the ESO Writing Committee. Version 16.03.2008.
11. Guidelines for the Management of Spontaneous Intracerebral Hemorrhage. A Guideline for Healthcare Professionals From the American Heart Association/American Stroke Association (*Stroke*. 2010;41:00-00.)
12. Joshua B. Bederson, E. Sander Connolly, et cetera. Guidelines for the Management of Aneurysmal Subarachnoid Hemorrhage: A Statement for Healthcare Professionals From a Special Writing Group of the Stroke Council, American Heart Association. *Stroke*. 2009; 40:994-1025.
13. Lehmon-Horn A., Ludolph A. Neurologisch Therapie. ELSEVIER, 2005, p. 393-430
14. Luis R. Caplan. Stroke. AAN Press, New York, 2005, 255 p.
15. Michael G Hennereci, Julien Bogousslavsky, Ralph LSacco. Stroke, ELSEVIER, Churchill Livingstone, 2005, 200 p.
16. The world health report 2002 - Reducing Risks, Promoting Healthy Life 10172 in Acute Stroke Treatment (TOAST). *Neurology*. Jul 13 1999;53(1):126-31.

