The Stroke Clinician's Handbook

A Practical Guide to the Care of Stroke Patients

editors
Robert N Gan
N Venketasubramanian Ramani

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Dedicated to the undeniable reasons why we do not give up…

our families, friends, mentors, colleagues, students, and patients.

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POST-ACUTE CARE AND PREVENTION

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PREFACE

When we set out to put together this stroke handbook, we wanted to provide busy clinicians with a quick guide to help them address real-time issues often encountered in stroke care. In reality, many stroke patients worldwide are not seen by a neurologist at first contact and the question most often asked is "what should I do next?"

By arranging the topics in chronological order, from the pre-hospital setting to hospitalization and secondary prevention in the clinic, we are hoping that the use of this handbook will be instinctive. We tried to be as specific as possible in recommending actions to be taken, yet flexible enough for use under conditions that may be less than ideal. We avoided redundancies, overlaps, and inconsistencies by asking each author to address specific important clinical issues. This meant, however, that we had to rely on repeated references to specific sections elsewhere in the handbook when the need arises from another particular topic to close the loop, almost simulating the encounter with a stroke patient and the possible ups and downs one may go through during the process of stroke care.

This handbook is neither meant to replace stroke textbooks and published guidelines nor an attempt to oversimplify the issues behind the appropriate care of stroke patients. We all have the natural obsession and compulsion to know more about the science behind the disease and the treatments. The list of evidence and suggested readings at the end of each section will, therefore, help users in their search for more detailed and comprehensive information.

We are indebted to the authors for their excellent contributions. While this handbook may not be as extensive as textbooks and references, their work was by no means any less gargantuan. Often, it is more difficult to be concise and to pick out the most important things that really matter in the face of the mammoth information available instead of simply writing down everything that is known about a specific topic. The completion of this handbook is, therefore, a testament to the remarkable expertise of each contributor.

With the increasing popularity of "strokology" as an interest not only to neurologists and geriatricians, we hope that the availability of a practical handbook like this one will contribute to the collective efforts in reducing the burden of stroke worldwide.

Robert N. Gan
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LIST OF ABBREVIATIONS

ABC	airway, breathing, circulation
ACA	anterior cerebral artery
ACEI	angiotensin converting enzyme inhibitor
AF	atrial fibrillation
ANA	antinuclear antibody test
aPTT	activated partial thromboplastin time
ARB	angiotensin II receptor blocker
ARR	absolute risk reduction
AVM	arteriovenous malformation
BB	beta-blocker
BI	Barthel index
BID	twice a day
BMI	body mass index
BP	blood pressure
BSL	blood sugar level
BUN	blood urea nitrogen
CAD	coronary artery disease
CAS	carotid artery stenting
CAST	Chinese Acute Stroke Trial
CBC	complete blood count
CBF	cerebral blood flow
CCA	common carotid artery
CCB	calcium channel blocker
CEA	carotid endarterectomy
CHO	carbohydrates
CI	confidence interval
$CMRO_2$	cerebral metabolic rate of oxygen
CO_2	carbon dioxide
CPP	cerebral perfusion pressure
CRP	c-reactive protein
CS	carotid siphon
CT	computed tomography
CTA	computed tomographic angiography
CVP	central venous pressure
CVST	cerebral venous (sinus) thrombosis
°C	degree centigrade
DBP	diastolic blood pressure
DVT	deep venous thrombosis
DWI	diffusion weighted imaging
D10NS	dextrose 10% normal saline
D5NS	dextrose 5% normal saline

D50	dextrose 50%
ECA	external carotid artery
ECG/EKG	electrocardiogram
EC-IC	extracranial – intracranial
EDV	end diastolic velocity
ESR	erythrocyte sedimentation rate
FAST	face arm speech test
FBC	full blood count
GCS	Glasgow coma scale
GI	gastrointestinal
GRE	gradient echo
HbA1c	glycosylated hemoglobin
HDL	high density lipoprotein
HR	heart rate
H_2O	water
ICA	internal carotid artery
ICH	intracerebral/(intracranial) hemorrhage
ICP	intracranial pressure
ICU	intensive care unit
IMT	intima-media thickness
INR	international normalized ratio
IPC	intermittent pneumatic compression
IST	International Sroke Tial
IV	intravenous/intravenously
IVC	inferior vena cava
IVH	intraventricular hemorrhage
K	potassium
LACS	lacunar syndrome
LDL	low density lipoprotein
LES	lower extremity study
LMWH	low molecular weight heparin
LV	left ventricle/left ventricular
MAP	mean arterial pressure
MCA	middle cerebral artery
MES	microembolic signal
MI	myocardial infarction
MRA	magnetic resonance angiography
MRI	magnetic resonance imaging
mRS	modified Rankin scale
Na	sodium
NaCl	sodium chloride
NGT	nasogastric tube
NIHSS	National Institute of Health stroke scale
NNT	number needed to treat
NS	normal saline

OA	ophthalmic artery
OCSP	Oxfordshire Community Stroke Project
OD	once a day
OT	occupational therapy
PACS	partial anterior circulation syndrome
PCA	posterior cerebral artery
PCI	percutaneous coronary intervention
PCWP	pulmonary capillary wedge pressure
PE	pulmonary embolism
PEG	percutaneous endoscopic gastrostomy
PFO	patent foramen ovale
POCS	posterior circulation syndrome
PSV	peak systolic velocity
PT	physical therapy/physiotherapy
PT	prothrombin time/protime
RBC	red blood cells
RPR	rapid plasma reagin
RR	respiratory rate
rTPA	recombinant tissue plasminogen activator
RWMA	regional wall motion abnormality
SAH	subarachnoid hemorrhage
SBP	systolic blood pressure
SC	subcutaneous/subcutaneously
SI	soluble insulin
SPECT	single positron emission computed tomography
ST	speech therapy
TACS	total anterior circulation syndrome
TCD	transcranial Doppler
TEE	transesophageal echocardiography
TIA	transient ischemic attack
TOAST	Trial of Org 10172 in Acute Stroke Treatment
TTE	transthoracic (2D) echocardiography
US	ultrasound
UTI	urinary tract infection
VA	vertebral artery
VDRL	venereal disease research laboratory test
VQ	ventilation-perfusion
WC	waist circumference
WST	water swallowing test

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PREHOSPITAL STROKE EVALUATION AND TRIAGE

Dr. Chua Hoe Chin

Goals

To provide initial medical stabilization and to shorten delays in evaluation and treatment of patients with stroke.

Patient Data Needed

1. Early signs and symptoms of stroke:

   Sudden…
   - ☐ weakness on one side of the body
   - ☐ numbness or tingling of one side of the face, arm or leg
   - ☐ loss of vision or blindness, particularly in one eye, or double vision
   - ☐ difficulty in speaking, or slurring of speech
   - ☐ inability to understand what other people are saying
   - ☐ dizziness and loss of balance or sudden trouble in walking
   - ☐ severe and unusual headache

2. Oxygen saturation: _____ %
3. Capillary blood sugar: _____ mmol/L (or mg/dL)
4. Time of onset of stroke symptom(s)

Actions

1. Ensure ABC — Airway, Breathing and Circulation.
2. Give supplemental oxygen if oxygen saturation is ≤ 92%.
3. Perform rapid identification and assessment of stroke using the Cincinnati Prehospital Stroke Scale or the Face Arm Speech Test (FAST). (*See Insert 1 in this section*)
4. Check capillary blood sugar to exclude hypoglycemia. Give D50 intravenously if the blood sugar level < 4 mmol/L (< 70 mg/dL).
5. Send the patient promptly to a hospital with acute stroke facilities in view of the narrow time window for thrombolysis.
6. Notify (pre-arrival) the receiving hospital to mobilize the Emergency Department and Acute Stroke Team.
7. Exclude stroke mimics in the Emergency Department. (*See section on Stroke/TIA versus Mimics*)
8. Conduct fast-track evaluation of patients who present within 3 h of stroke for possible thrombolysis with rtPA. (*See section on Thrombolysis* (*rTPA*) *for Acute Ischemic Stroke*)

Evidence

1. Prehospital stroke care plays an important role in the "stroke chain of recovery".
2. Both the Cincinnati Prehospital Stroke Scale and FAST are highly reliable and reproducible for rapid identification of stroke patients. Interrater agreement is high amongst paramedics, the emergency department and neurologists.
3. Appropriately selected patients with acute ischemic stroke benefit from early treatment and thrombolysis.

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PREHOSPITAL STROKE EVALUATION AND TRIAGE

Insert 1. Prehospital Rapid Stroke Assessment Tools

Cincinnati Prehospital Stroke Scale

Suspect stroke if any of the following is abnormal:

a) Facial droop. Ask patient to smile or show teeth.
   Normal: Both sides of face move equally
   Abnormal: One side of face does not move as well as the other

b) Arm drift. Patient closes eyes and holds both arms straight out for 10 sec.
   Normal: Both arms move equally or not at all
   Abnormal: One arm drifts compared to the other

c) Speech. Ask patient to repeat "It is hot and sunny in Singapore".
   Normal: Patient says correct words without slurring
   Abnormal: Slurred or inappropriate words or mute

Face Arm Speech Test (*FAST*)

Suspect stroke if any of the following is answered "yes":

a) *Facial movements*
   Ask patient to smile or show teeth. Look for new asymmetry.
   Tick YES if there is an unequal smile or grimace or obvious facial asymmetry.

   YES ☐ NO ☐

b) *Arm movements*
   Lift the patient's arms together to 90° if sitting, or 45° if supine. Ask him to hold that position for 5 sec and then let go.
   Does one arm drift down or fall more rapidly?

   YES ☐ NO ☐

c) *Speech impairment*
   Look for new disturbances in speech. Look for slurred speech and word-finding difficulties. Ask the patient to name common nearby objects such as a cup, chair, key and pen.
   If there is a severe visual disturbance, place an object in the patient's hand and ask him to name it.

   YES ☐ NO ☐

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STROKE/TIA VERSUS MIMICS

Dr. Robert N. Gan

Goal

To differentiate true stroke or TIA from "stroke mimics".

Patient Data Needed

1. Duration of symptom(s) and mode of onset — e.g. sudden versus gradual
2. Pattern of weakness, if present, i.e. involvement of face, arm, and/or leg
3. History of risk factors for stroke
4. BP
5. Cardiac rhythm on auscultation or EKG

Actions

1. Check if the patient fulfils criteria for diagnosis of TIA or stroke:
   - ☐ acute or abrupt onset
   - ☐ neurological syndrome
   - ☐ referable to vascular abnormality
   - ☐ symptoms last from a few minutes (TIA) to > 24 h (stroke)
2. If weakness involves only the face, consider Bell's palsy.
3. Check for factors that make stroke or TIA more likely or less likely.

Factors that Make Stroke or TIA More Likely	Factors that Make Stroke or TIA Less Likely
Stroke risk factors (e.g. hypertension, diabetes mellitus, hypercholesterolemia, smoking)	No vascular risk factor
Definite focal signs/symptoms	Cognitive impairment
Clear and exact time of onset	Decreased level of consciousness/ confusion
Irregular cardiac rhythm on auscultation	Isolated dizziness or vertigo
Atrial fibrillation/atrial flutter on EKG	Seizures at onset
Diastolic BP > 90 mmHg	Abnormal findings in other systems, i.e. respiratory, abdominal, etc.
Abnormal visual fields	Signs and symptoms not consistent with neuroanatomical and vascular distribution
Abnormal eye movements	
History of angina	

4. If it is likely to be TIA or stroke, activate stroke team/protocol.

Evidence

1. As much as 20 to 30% of "strokes" are mimics.
2. Stroke mimics include: seizures, systemic infection, brain tumor, toxic-metabolic, positional vertigo, cardiac, syncope, trauma, subdural hematoma, encephalitis, transient global amnesia, dementia, demyelinating disease, spinal cord lesion, myasthenia gravis, Parkinsonism, hypertensive encephalopathy, conversion disorder, Bell's palsy, and migraine.
3. Stroke or TIA is unlikely in patients presenting with isolated dizziness or vertigo.

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ADMIT TO HOSPITAL VERSUS CLINIC

Dr. Nijasri C. Suwanwela

Goal

To identify patients with TIA or acute stroke who require hospital admission.

Patient Data Needed

1. Time from onset of stroke symptom(s)
2. Duration of TIA symptom(s): _____ minutes
3. Age: _____ years
4. BP: _____/_____ mmHg
5. Clinical features; presence of:
   - ☐ Unilateral motor weakness
   - ☐ Speech disturbance
6. History of diabetes mellitus
7. ECG rhythm

Actions

1. Patients who present within 3 h of stroke symptom(s) onset should be considered for thrombolytic therapy. (*See section on Thrombolysis* (*rtPA*) *for Acute Ischemic Stroke*)
2. Admit all patients with acute stroke or recent TIA within 7 days.
3. If admission is not possible for all patients, identify high-risk TIA patients using **ABCD²** score:

Age
- ☐ ≥ 60 years = 1
- ☐ < 60 years = 0

Blood pressure on first assessment
- ☐ Systolic ≥ 140 and/or diastolic ≥ 90 mmHg = 1
- ☐ Systolic < 140 and diastolic < 90 mmHg = 0

Clinical features
- ☐ Unilateral weakness = 2
- ☐ Speech disturbance without weakness = 1
- ☐ Other = 0

Duration of symptoms
- ☐ ≥ 60 min = 2
- ☐ 10–59 min = 1
- ☐ < 10 min = 0

Diabetes
- ☐ Yes = 1
- ☐ No = 0.

If score ≥ 4 (*medium to high risk*), *admit patient*
If score < 4 (*low risk*), *prompt evaluation in outpatient setting or TIA clinic*

4. Patients with neurological worsening or concomitant atrial fibrillation, carotid bruit, uncontrolled hypertension or hypotension, fever, dehydration, signs of congestive heart failure or acute coronary event should be admitted.
5. Perform CT scan or MRI of the brain as soon as possible. (*See section on Brain Imaging*)
6. Start oral aspirin if there is no contraindication.
7. Perform diagnostic tests according to stroke subtype as soon as possible. (*See sections on TIA and Acute Ischemic Stroke, Acute Intracerebral Hemorrhage, and Acute Subarachnoid Hemorrhage*)

Evidence

1. Patients with TIA have high risk of stroke (approximately 8%) especially during the first 7 days.
2. Clinical features of TIA provide substantial prognostic information. $ABCD^2$ score is one model which shows that the early risk of stroke after TIA is predictable. Two-day stroke risk is 0% for score of 0 or 1; 1–2% for 2; 0–3% for 3; 2–5% for 4; 3–7% for 5; 4–14% for 6; and 0–50% for 7. Patients with moderate (score of 4–5) or high (score > 5) risks account for 66% of patients presenting with TIA. Low risk patients (score < 4) have an overall 7–day stroke risk of 1.2%.
3. Prompt evaluation and treatment of patients with TIA or stroke improves outcome.
4. Antiplatelets are effective in preventing stroke after TIA or minor stroke. Patients with cardiac source of emboli, especially atrial fibrillation, are better treated with anticoagulation.

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ANTERIOR VERSUS POSTERIOR CIRCULATION SYNDROME

Dr. Maricar P. Yumul and Dr. Jose Navarro

Goal

To determine whether stroke syndrome is due to pathology in the anterior or posterior cerebral circulation to guide management in decision-making.

Patient Data Needed

1. Presenting symptoms:
   - speech ☐ confused or word-finding difficulty
     ☐ hoarse
   - visual ☐ could not see from one eye when good eye covered
     ☐ could not see half of objects with one eye or both eyes open
     ☐ diplopia/double vision
     ☐ completely blind
   - vertigo ☐ Yes ☐ No
   - imbalance ☐ Yes ☐ No
   - numbness around mouth ☐ Yes ☐ No
   - new-onset seizures ☐ Yes ☐ No
2. Neurological signs
   - limb weakness and/or numbness ☐ unilateral involvement
     ☐ bilateral involvement
   - facial droop or numbness ☐ same side as limb weakness/numbness
     ☐ side opposite limb weakness or numbness
   - eye movement ☐ looking away from side of limb weakness
     ☐ looking to side of limb weakness
     ☐ dysconjugate eye movements
   - aphasia ☐ Yes ☐ No
   - neglect ☐ Yes ☐ No
   - ptosis or partial Horner's ☐ Yes ☐ No
   - nystagmus ☐ Yes ☐ No
   - dysphonia ☐ Yes ☐ No
   - hearing loss ☐ Yes ☐ No
   - dysphagia, loss of gag reflex ☐ Yes ☐ No
   - homonymous hemianopsia ☐ Yes ☐ No
   - ataxia, dysmetria ☐ Yes ☐ No

Actions

1. Suspect anterior circulation stroke syndrome if:
   — speech is confused, word-finding difficulty, or aphasia
   — visual loss in one eye
   — new-onset seizures

   — facial droop or numbness on same side as limb weakness or numbness
   — eyes deviated (looking) away from side of limb weakness
   — neglect.
2. Suspect posterior circulation stroke syndrome if:
   — vertigo and imbalance
   — diplopia or dysconjugate eye movement
   — speech hoarse or dysphonic
   — numbness around mouth
   — eyes deviated towards (looking at) side of limb weakness
   — facial droop or numbness on side opposite limb weakness or numbness
   — bilateral weakness or numbness
   — isolated homonymous hemianopsia or cortical blindness
   — ptosis or partial Horner's syndrome
   — nystagmus
   — hearing loss
   — dysphagia or loss of gag reflex
   — ataxia or dysmetria without weakness on same side.
3. For posterior circulation stroke syndrome, consider performing MRI of the brain, if available, instead of CT scan (*See section on Brain Imaging*). Perform CT scan if:
   — The patient is candidate for thrombolytic therapy and MRI cannot be done within available time window. (*See section on Thrombolysis* (*rTPA*) *for Acute Ischemic Stroke*)
   — hemorrhagic stroke is suspected to be due to the presence of decreased level of consciousness, headache, vomiting, severe hypertension, and neck rigidity.
4. Proceed with diagnostic and treatment options as described in succeeding sections.

Evidence

1. Certain clinical features are helpful in distinguishing anterior from posterior circulation strokes.
2. Prognosis and choice of diagnostic examination and treatment may differ between anterior and posterior circulation strokes.

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4. Piechowski-Jówiak B, Boggouslavsky J. (2004) Vascular disorders of the posterior circulation — An anatomico-clinical overview. *ACNR* **4**: 6–9.
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6. Warlow CP, Dennis MS, van Gijn J, *et al.* (2001) *Stroke: A Practical Guide to Management*. Blackwell Science Ltd., Oxford, pp. 106–150.

NOTES

NOTES

BRAIN IMAGING

Dr. C C Tchoyoson Lim

Goals

To distinguish hemorrhagic from ischemic strokes, to direct specific treatments, and to detect and monitor complications, e.g. hydrocephalus, infarct extension or hemorrhagic transformation.

Patient Data Needed

1. Clinical diagnosis of acute stroke or TIA
2. Time of onset of symptoms
3. Relative and absolute contraindications to CT scan (e.g. pregnancy) or MRI (pacemaker, intraocular lens, aneurysm clips, metallic implants)

Actions

1. For all patients with suspicion of TIA or acute stroke, do non-contrast enhanced CT scan or MRI of the brain as soon as possible, preferably within 24 h of admission.
2. Do emergent brain CT or MRI for patients presenting within 3 h of symptom onset who may be candidates for thrombolysis with rTPA. (*See section on Thrombolysis* (*rTPA*) *for Acute Ischemic Stroke*)
3. Review brain scan as soon as possible.
4. Differentiate hemorrhagic stroke from ischemic stroke. (*See Inserts 1 and 2 in this section*)
5. Differentiate stroke from mimics. (*See section on Stroke/TIA versus mimics*)
6. If the clinical course of the patient is complicated by worsening, consider repeat brain imaging. (*See section on Worsening after Stroke*)

Evidence

1. CT shows a nonvascular lesion accounting for neurological symptoms (tumors, subdural hematoma) in about 1% of patients with TIA. CT and MRI may also identify other vascular lesions such as aneurysms or arteriovenous malformations that can be present in patients with TIAs.
2. Between 5–10% of clinically suspected ischemic stroke patients are found instead to have cerebral hemorrhage on CT scan.
3. Diagnostic errors based solely on clinical features may occur and this level of accuracy is insufficient to guide treatment decisions.
4. A brain imaging study is mandatory to distinguish ischemic stroke from hemorrhage or other structural brain lesions that may imitate stroke.
5. Due to bony artifacts, CT scan is inferior to MRI in the evaluation of posterior fossa lesions.

Selected Readings

1. Feinberg WM, Albers GW, Barnett HJM, *et al*. (1994) Guidelines for the management of transient ischemic attacks: From the Ad Hoc Committee on Guidelines for the Management of Transient Ischemic Attacks of the Stroke Council of the American Heart Association. AHA medical/scientific statement: Special report. *Circulation* **89**: 2950–2965.
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3. Muir KW, Weir CJ, Murray GD, *et al*. Comparison of neurological scales and scoring systems for acute stroke prognosis. *Stroke* **27**: 1817–1820.
4. Britton M, Hindmarsh T, Murray V, Tyden SA. (1984) Diagnostic errors discovered by CT in patients with suspected stroke. *Neurology* **34**: 1504–1507.
5. Interpretation of IST and CAST stroke trials (Correspondence). (1997) *Lancet* **350**: 440–450.

BRAIN IMAGING

Insert 1. Brain CT Scan

- Most widely used neuroimaging technique because it is quick, less expensive, and is widely available.
- Accurately demontrates intracranial hemorrhage, i.e. intracerebral hemorrhage (ICH, Fig 1) and subarachnoid hemorrhage (SAH, Fig 2).
- Demonstrates the extent of cerebral infarction (Fig 3) but has limitations if lesion is in posterior circulation.
- CT may be normal if performed very early after ischemic stroke onset, hence negative study does not rule out stroke.
- Contrast enhancement can provide CT angiographic and perfusion information.

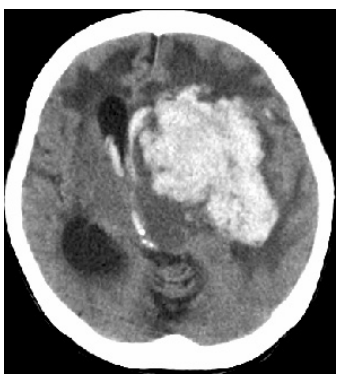

Figure 1. CT showing hyperdensity in the left basal ganglion due to hypertensive hemorrhage.

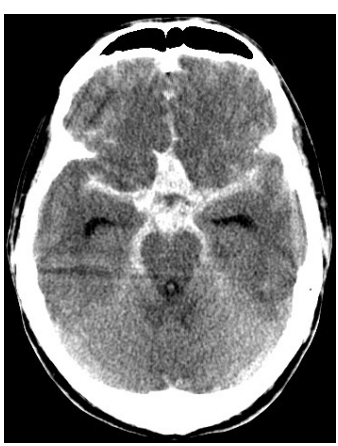

Figure 2. CT showing widespread hyperdensity in the subarachnoid space from SAH.

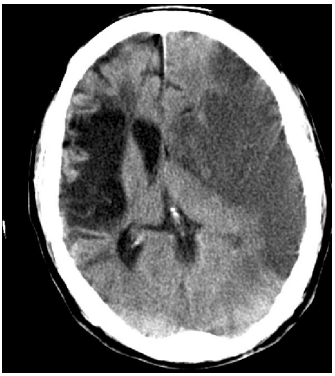

Figure 3. CT shows hypodensity of the left MCA territory due to acute infarction. Note the volume loss and hypodensity of the right temporal lobe due to previous MCA territory infarction.

BRAIN IMAGING

Insert 2. MRI Brain Scan

- DWI identifies ischaemic lesions (Fig 4) within minutes of the stroke onset, and can distinguish recent (within 2 weeks) infracts from chronic infarcts.
- MRA detects vascular abnormalities that led to the stroke, e.g. stenosis/occlusion (Fig 5), aneurysm, vascular malformation.
- GRE detects hemorrhage, both recent and old (Fig 6).
- Whilst DWI and MRA are well-established techniques, perfusion MRI is still largely investigational but promising.

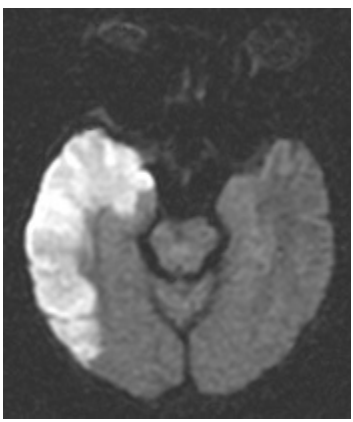

Figure 4. DWI showing hyperintensity of the right MCA territory due to acute infarction.

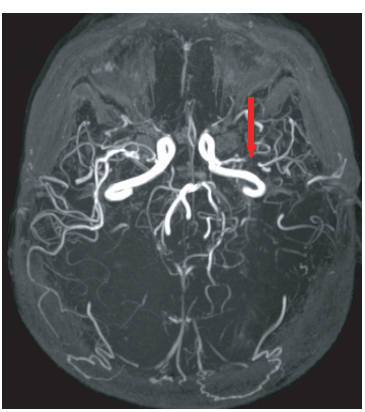

Figure 5. MRA showing occlusion of the left MCA (arrow).

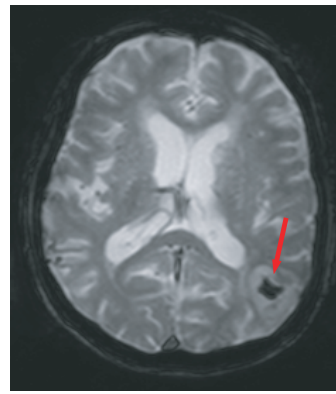

Figure 6. GRE showing hemorrhagic conversion (arrow) in an area of infarction.

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ADMIT TO STROKE UNIT OR WARD

Dr. N. Venketasubramanian Ramani

Goal

To manage a stroke patient in an appropriate hospital environment that further enhances good patient outcome.

Patient Data Needed

1. Clinical diagnosis of stroke
2. Conditions requiring more intensive care than can be provided by the current set-up of the stroke unit (e.g. respiratory failure, status epilepticus, etc.)
3. Active major co-morbid conditions best managed by another discipline (e.g. acute coronary syndrome, congestive heart failure, etc.)

Actions

1. Admit all suspected stroke patients to an organized geographical stroke unit, unless there are co-existing conditions that require them to be admitted to the intensive care unit or another discipline.
2. If a stroke unit or a bed in the stroke unit is not available, admit patient to an appropriate unit/ward and refer patient to a mobile stroke team or a neurologist.
3. Start stroke clinical pathway. (*See section on Basic Admitting Orders and Stroke Clinical Pathway*)
4. Perform baseline clinical assessments and regularly monitor patient's neurological status for any deterioration. (*See section on Stroke Scales and Classifications*)
5. Start treatments and perform diagnostic tests specific to the patient's stroke subtype. (*See sections on TIA and Acute Ischemic Stroke, Acute Intracerebral Hemorrhage, and Acute Subarachnoid Hemorrhage*)
6. Start coordinated multidisciplinary team care.
7. Closely monitor patients for complications, such as neurological (e.g. recurrent stroke, seizures), cardiovascular (e.g. myocardial infarction, arrythmia), infectious (e.g. UTI, pneumonia), thromboembolic (e.g. DVT, PE), gastrointestinal (e.g. GI bleeding, constipation), urinary (e.g. retention, hematuria), psychological (e.g. depression), and musculocutaneoskeletal (e.g. pressure sores, joint pain, falls).
8. Start a program of education and training for patient and family/caregivers.
9. Start early rehabilitation and discharge planning. (*See section on Rehabilitation, Physical Therapy, Occupational Therapy, and Speech Therapy*)
10. Institute early management policies e.g. early mobilization; avoid urinary catheterization; treatment of hypoxia, hyperglycemia, suspected infection.
11. Provide programs of education, training, research, and specialization of staff.
12. Regularly assess and audit services provided by the stroke unit/team.

Evidence

1. Stroke services are often provided in/by stroke ward (acute stroke unit, stroke rehabilitation unit), mobile stroke team, neurology ward, geriatrics ward, medical ward, mixed rehabilitation ward.
2. Compared with alternative services, organized inpatient stroke unit care reduces, at a median of 1 year, the odds of death by 14% (95% CI 6–29, p = 0.005), odds of death or institutionalized care by 20% (95% CI 10–29, p = 0.0002), and odds of death or dependency by 22% (95% CI 11–32, p = 0.0003).
3. Improved outcomes from stroke services are independent of the patient's age, sex, and stroke severity. Outcomes appear to be better in stroke units based in a discrete ward. Organized in-patient stroke unit care does not prolong hospital stay.
4. Reductions in case fatality occur as early as 1 to 4 weeks after the index stroke. Reduction in the odds of death is evident across all causes of death, but most marked for those deaths considered to be secondary to immobility.
5. Stroke unit care improves survival and functional state and increases the proportion of patients being able to live at home 10 years after their stroke.
6. The majority of stroke unit trials have similar approaches to assessment procedures, early management policies, and ongoing rehabilitation policies.
7. Improvements in outcomes after stroke care in stroke units are reproducible in the routine clinical setting.

Selected Readings

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5. Indredavik B, Bakke F, Slordahl SA, *et al*. (1999) Stroke unit treatment. 10-year follow-up. *Stroke* **30**: 1524–1527.
6. Stegmayr B, Asplund K, Hulter-Asberg K, *et al*. (1999) Stroke units in their natural habitat: Can results of randomised trials be reproduced in routine clinical practice? Riks-Stroke Collaboration. *Stroke* **30**: 709–714.
7. Langhorne P, Dey P, Woodman M, *et al*. (2005) Is stroke unit care portable? A systematic review of the clinical trials. *Age Ageing* **34**: 324–330.

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STROKE SCALES AND CLASSIFICATIONS

Dr. Robert N. Gan

Goal

To assess the clinical severity of stroke using standardized scales for prognostication and serial assessment of clinical status.

Patient Data Needed

1. Age
2. Neurological examination
3. Brain imaging, if available (*See section on Brain Imaging*)

Actions

1. Perform NIHSS, GCS, and OCSP classification at the initial presentation. (*Use Inserts 1, 2 and 3*)
2. If brain imaging reveals intracerebral hemorrhage, perform ICH scoring. (*Use Insert 4. See also section on Acute Intracerebral Hemorrhage*)
3. If brain imaging reveals subarachnoid hemorrhage, perform Hunt and Hess or World Federation of Neurosurgeons (WFNS) grading. (*Use Insert 5. See also section on Acute Subarachnoid Hemorrhage*)
4. Repeatedly perform NIHSS and GCS as often as necessary to monitor for changes in the patient's neurological status. NIHSS is usually performed at least daily in the acute stage. GCS may be performed hourly if necessary.

Evidence

1. The NIHSS score strongly predicts the likelihood of a patient's recovery after stroke. A score of ≥ 16 forecasts a high probability of death or severe disability whereas a score of ≤ 6 forecasts a good recovery. The score also correlates well with lesion size.
2. NIHSS and GCS show good inter-rater reliability. They are useful and valid in serial assessment of stroke.
3. The OCSP classification predicts size and location of the stroke on brain imaging in three quarters of patients. It also has prognostic value by predicting mortality, functional recovery and patterns of recurrent stroke. For example, patients with partial anterior circulation infarct (PACI) are at particularly high risk of early stroke recurrence.
4. The ICH score correlates well with mortality. A score of 5 has been shown to have a 100% 30–day mortality. The scoring is likewise predictive of functional outcome and has been validated in different populations, including Asians.
5. The Hunt and Hess and WFNS grading of subarachnoid hemorrhage help neurosurgeons in treatment decision-making.

Selected Readings

1. Adams HP Jr, Davis PH, Leira EC, *et al.* (1999) Baseline NIH Stroke Scale score strongly predicts outcome after stroke: A report of the Trial of Org 10172 in Acute Stroke Treatment (TOAST). *Neurology* **53**: 126–131.
2. Wityk RJ, Pessin MS, Kaplan RF, *et al.* (1994) Serial assessment of acute stroke using the NIH Stroke Scale. *Stroke* **25**: 362–365.
3. Lyden P, Lu M, Jackson C, *et al.* (1999) Underlying structure of the National Institutes of Health Stroke Scale — Results of a factor analysis. *Stroke* **30**: 2347–2354.
4. Mead GE, Lewis SC, Wardlaw JM, *et al.* (2000) How well does the Oxfordshire Community Stroke Project classification predict the site and size of the infarct on brain imaging? *J Neurol Neurosurg Psychiatry* **68**: 558–562.
5. Bamford J, Sandercock P, Dennis M, *et al.* (1991) Classification and natural history of clinically identifiable subtypes of cerebral infarction. *Lancet* **337**: 1521–1526.
6. Jamora RDG, Kishi-Generao EM Jr, Apaga NEP, *et al.* (2003) The ICH Score: Predicting mortality and functional outcome in an Asian population [Letter to the Editor]. *Stroke* **34**: 6–7.
7. Hunt WE, Hess RM. (1968) Surgical risk as related to time of intervention in the repair of intracranial aneurysms. *J Neurosurg* **28**:14–20.
8. Drake CG. (1988) Report of World Federation on Neurological Surgeons committee on a universal subarachnoid hemorrhage grading scale. *J Neurosurg* **68**: 985–986.

STROKE SCALES AND CLASSIFICATIONS

Insert 1. National Institute of Health Stroke Scale (*NIHSS*)

Instructions	Scale Definition	Score
1a. Level of Consciousness: The investigator must choose a response if a full evaluation is prevented by obstacles such as an endotracheal tube, language barrier, orotracheal trauma/bandages. A 3 is scored only if the patient makes no movement (other than reflexive posturing) in response to noxious stimulation.	0 = **Alert;** keenly responsive. 1 = **Not alert;** but arousable by minor stimulation to obey, answer, or respond. 2 = **Not alert;** requires repeated stimulation to attend, or is obtunded and requires strong or painful stimulation to make movements (not stereotyped). 3 = **Responds only with reflex** motor or autonomic effects or **totally unresponsive**, flaccid, and flexic.	_____
1b. LOC Questions: The patient is asked the month and his/her age. The answer must be correct — there is no partial credit for being close. Aphasic and stuporous patients who do not comprehend the questions will score 2. Patients unable to speak because of endotracheal intubation, orotracheal trauma, severe dysarthria from any cause, language barrier, or any other problem not secondary to aphasia are given a 1. It is important that only the initial answer be graded and that the examiner does not "help" the patient with verbal or non-verbal cues.	0 = **Answers both** questions correctly. 1 = **Answers one** question correctly. 2 = **Answers neither** question correctly.	_____
1c. LOC Commands: The patient is asked to open and close the eyes and then to grip and release the non-paretic hand. Substitute another one step command if the hands cannot be used. Credit is given if an unequivocal attempt is made but not completed due to weakness. If the patient does not respond to command, the task should be demonstrated to him or her (pantomime), and the result scored (i.e., follows none, one or two commands). Patients with trauma, amputation, or other physical impediments should be given suitable one-step commands. Only the first attempt is scored.	0 = **Performs both** tasks correctly. 1 = **Performs one** task correctly. 2 = **Performs neither** task correctly.	_____
2. Best Gaze: Only horizontal eye movements will be tested. Voluntary or reflexive (oculocephalic) eye movements will be scored, but caloric testing is not done. If the patient has a conjugate deviation of the eyes that can be overcome by voluntary or reflexive activity, the score will be 1. If a patient has an isolated peripheral nerve paresis (CN III, IV or VI), score a 1. Gaze is testable in all aphasic patients. Patients with ocular trauma, bandages, pre-existing blindness, or other disorder of visual acuity or fields should be tested with reflexive movements, and a choice made by the investigator. Establishing eye contact and then moving about the patient from side to side will occasionally clarify the presence of a partial gaze palsy.	0 = **Normal.** 1 = **Partial gaze palsy;** gaze is abnormal in one or both eyes, but forced deviation or total gaze paresis is not present. 2 = **Forced deviation,** or total gaze paresis is not overcome by the oculocephalic maneuver.	_____

Instructions	Scale Definition	Score
3. Visual: Visual fields (upper and lower quadrants) are tested by confrontation, using finger counting or visual threat, as appropriate. Patients may be encouraged, but if they look at the side of the moving fingers appropriately, this can be scored as normal. If there is unilateral blindness or enucleation, visual fields in the remaining eye are scored. Score 1 only if a clear-cut asymmetry, including quadrantanopia, is found. If patient is blind from any cause, score 3. Double simultaneous stimulation is performed at this point. If there is extinction, patient receives a 1, and results are used to respond to item 11.	0 = **No visual loss.** 1 = **Partial hemianopia.** 2 = **Complete hemianopia.** 3 = **Bilateral hemianopia** (blind including cortical blindness).	_____
4. Facial Palsy: Ask — or use pantomime to encourage — the patient to show teeth or raise eyebrows and close eyes. Score symmetry of grimace in response to noxious stimuli in the poorly responsive or non-comprehending patient. If facial trauma/bandages, orotracheal tube, tape or other physical barriers obscure the face, these should be removed to the extent possible.	0 = **Normal** symmetrical movements. 1 = **Minor paralysis** (flattened nasolabial fold, asymmetry on smiling). 2 = **Partial paralysis** (total or near-total paralysis of lower face). 3 = **Complete paralysis** of one or both sides (absence of facial movement in the upper and lower face).	_____
5. Motor Arm: The limb is placed in the appropriate position: extend the arms (palms down) 90° (if sitting) or 45° (if supine). Drift is scored if the arm falls before 10 sec. The aphasic patient is encouraged using urgency in the voice and pantomime, but not noxious stimulation. Each limb is tested in turn, beginning with the non-paretic arm. Only in the case of amputation or joint fusion at the shoulder, should the examiner record the score as untestable (UN), and clearly state the explanation for this choice.	0 = **No drift;** limb holds 90° (or 45°) for full 10 sec. 1 = **Drift;** limb holds 90° (or 45°), but drifts down before full 10 sec; does not hit the bed or other support. 2 = **Some effort against gravity;** limb cannot get to or maintain (if cued) 90° (or 45°), drifts down to the bed, but has some effort against gravity. 3 = **No effort against gravity;** limb falls. 4 = **No movement.** UN = **Amputation** or joint fusion, explain: _____	
	5a. Left Arm	_____
	5b. Right Arm	_____
6. Motor Leg: The limb is placed in the appropriate position: hold the leg at 30° (always tested supine). Drift is scored if the leg falls before 5 sec. The aphasic patient is encouraged using urgency in the voice and pantomime, but not noxious stimulation. Each limb is tested in turn, beginning with the non-paretic leg. Only in the case of amputation or joint fusion at the hip should the examiner record the score as untestable (UN), and clearly state the explanation for this choice.	0 = **No drift;** leg holds a 30° position for full 5 sec. 1 = **Drift;** leg falls by the end of the 5-sec period but does not hit the bed. 2 = **Some effort against gravity;** leg falls to the bed by 5 sec, but has some effort against gravity. 3 = **No effort against gravity;** leg falls to the bed immediately. 4 = **No movement.** UN = **Amputation** or joint fusion, explain: _____	
	6a. Left Leg	_____
	6b. Right Leg	_____

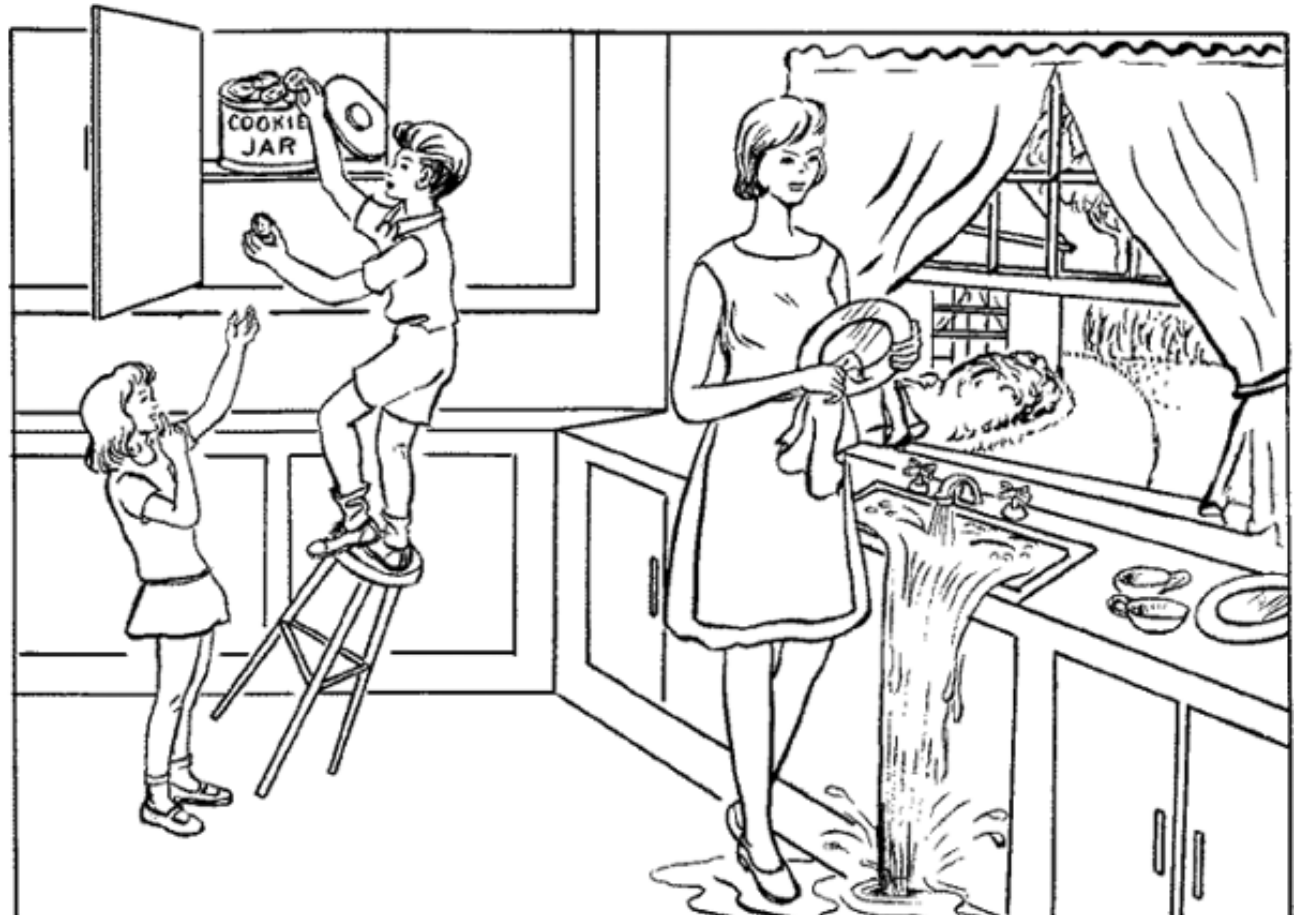
Instructions	Scale Definition	Score
7. Limb Ataxia: This item is aimed at finding evidence of an unilateral cerebellar lesion. Test with eyes open. In case of visual defect, ensure testing is done in an intact visual field. The finger-nose-finger and heel-shin tests are performed on both sides, and ataxia is scored only if present out of proportion to weakness. Ataxia is absent in the patient who cannot understand or is paralyzed. Only in the case of amputation or joint fusion, should the examiner record the score as untestable (UN), and clearly state the explanation for this choice. In case of blindness, test by having the patient touch nose from extended arm position.	0 = **Absent.** 1 = **Present in one limb.** 2 = **Present in two limbs.** UN = **Amputation** or joint fusion, explain: _____	_____
8. Sensory: Sensation or grimace to pinprick when tested, or withdrawal from noxious stimulus in the obtunded or aphasic patient. Only sensory loss attributed to stroke is scored as abnormal and the examiner should test as many body areas (arms [not hands], legs, trunk, face) as needed to accurately check for hemisensory loss. A score of 2, "severe or total sensory loss" should only be given when a severe or total loss of sensation can be clearly demonstrated. Stuporous and aphasic patients will, therefore, probably score 1 or 0. The patient with brainstem stroke who has bilateral loss of sensation is scored 2. If the patient does not respond and is quadriplegic, score 2. Patients in a coma (item 1a = 3) are automatically given a 2 on this item.	0 = **Normal;** no sensory loss. 1 = **Mild-to-moderate sensory loss;** patient feels pinprick is less sharp or is dull on the affected side; or there is a loss of superficial pain with pinprick, but patient is aware of being touched. 2 = **Severe to total sensory loss;** patient is not aware of being touched in the face, arm, and leg.	_____
9. Best Language: A great deal of information about comprehension will be obtained during the preceding sections of the examination. For this scale item, the patient is asked to describe what is happening in the attached picture, to name the items on the attached naming sheet and to read from the attached list of sentences. Comprehension is judged from responses here, as well as to all of the commands in the preceding general neurological exam. If visual loss interferes with the tests, ask the patient to identify objects placed in the hand, repeat, and produce speech. The intubated patient should be asked to write. The patient in a coma (item 1a = 3) will automatically score 3 on this item. The examiner must choose a score for the patient with stupor or limited cooperation, but a score of 3 should be used only if the patient is mute and follows no one-step commands.	0 = **No aphasia;** normal. 1 = **Mild-to-moderate aphasia;** some obvious loss of fluency or facility of comprehension, without significant limitation on ideas expressed or form of expression. Reduction of speech and/or comprehension, however, makes conversation about provided materials difficult or impossible. For example, in conversation about provided materials, examiner can identify picture or name card content from patient's response. 2 = **Severe aphasia;** all communication is through fragmentary expression; great need for inference, questioning, and guessing by the listener. Range of information that can be exchanged is limited; listener carries burden of communication. Examiner cannot identify materials provided from patient response. 3 = **Mute, global aphasia;** no usable speech or auditory comprehension.	_____

Instructions	Scale Definition	Score
10. Dysarthria: If patient is thought to be normal, an adequate sample of speech must be obtained by asking patient to read or repeat words from the attached list. If the patient has severe aphasia, the clarity of articulation of spontaneous speech can be rated. Only if the patient is intubated or has other physical barriers to producing speech should the examiner record the score as untestable (UN), and clearly state the explanation for this choice. Do not tell the patient why he or she is being tested.	0 = **Normal.** 1 = **Mild-to-moderate dysarthria;** patient slurs at least some words and, at worst, can be understood with some difficulty. 2 = **Severe dysarthria;** patient's speech is so slurred as to be unintelligible in the absence of or out of proportion to any dysphasia, or is mute/anarthric. UN = **Intubated** or other physical barrier, explain:_____	_____
11. Extinction and Inattention (formerly Neglect): Sufficient information to identify neglect may be obtained during the prior testing. If the patient has a severe visual loss preventing visual double simultaneous stimulation, and the cutaneous stimuli are normal, the score is normal. If the patient has aphasia but does appear to attend to both sides, the score is normal. The presence of visual spatial neglect or anosagnosia may also be taken as evidence of abnormality. Since the abnormality is scored only if present, the item is never untestable.	0 = **No abnormality.** 1 = **Visual, tactile, auditory, spatial, or personal inattention** or extinction to bilateral simultaneous stimulation in one of the sensory modalities. 2 = **Profound hemi-inattention or extinction to more than one modality;** does not recognize own hand or orients to only one side of space.	_____
	Total	_____

Administer stroke scale items in the order listed. Record performance in each category after each subscale exam. Do not go back and change scores. Follow directions provided for each exam technique. Scores should reflect what the patient does, not what the clinician thinks the patient can do. The clinician should record answers while administering the exam and work quickly. Except where indicated, the patient should not be coached (i.e., repeated requests to patient to make a special effort).

Reference

Brott T, Adams HP Jr, Olinger CP, *et al*. (1989) Measurements of acute cerebral infarction: A clinical examination scale. *Stroke* **20**: 864–870.



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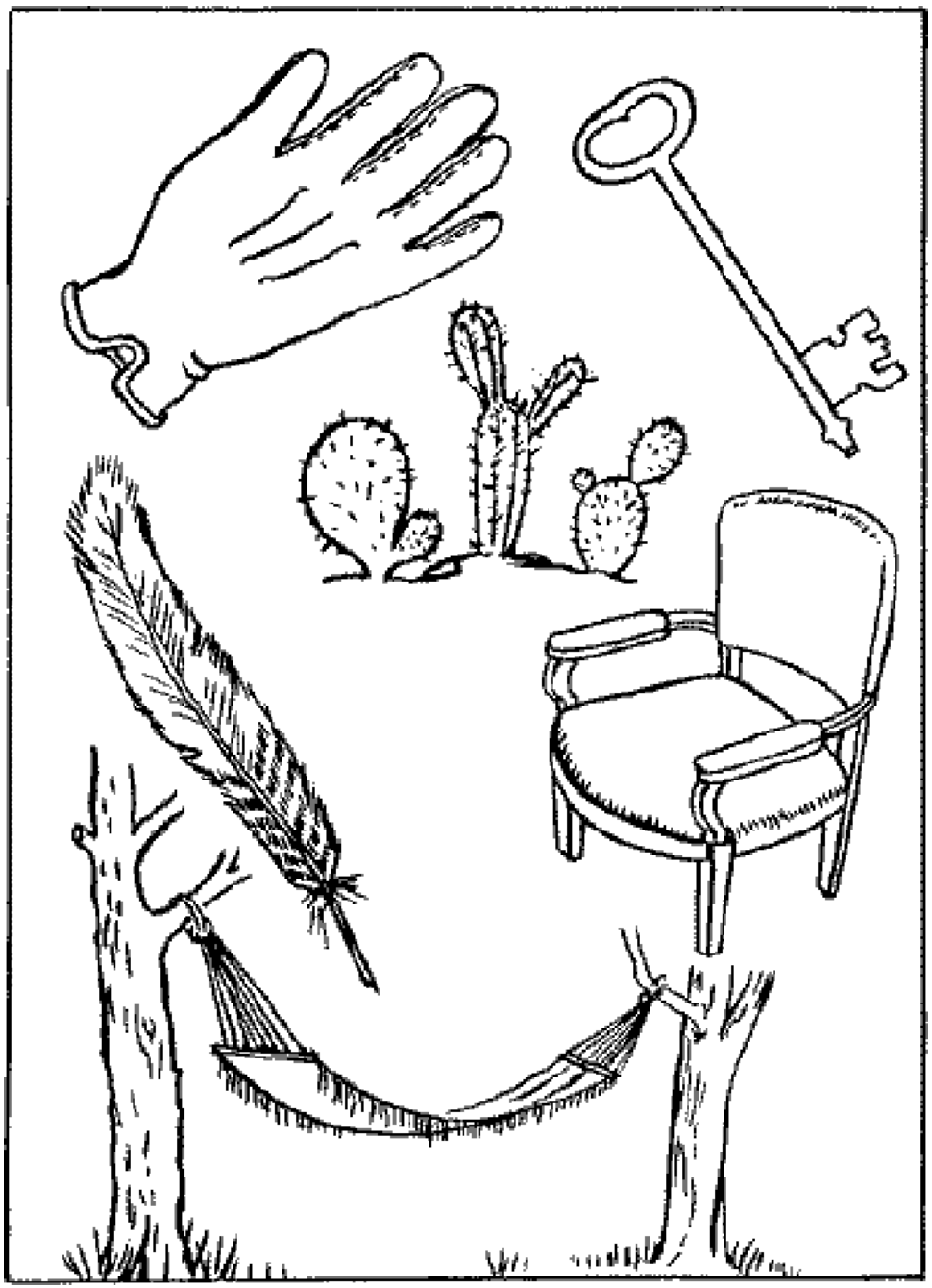

You know how.

Down to earth.

I got home from work.

Near the table in the dining room.

They heard him speak on the radio last night.

MAMA

TIP – TOP

FIFTY – FIFTY

THANKS

HUCKLEBERRY

BASEBALL PLAYER

Insert 2. Glasgow Coma Scale (*GCS*)

Eye Opening (E)

Spontaneous eye opening	4
Eye opening in response to verbal command, speech, or shout	3
Eye opening in response to pain	2
No eye opening	1

Best Verbal Response (V)

Oriented	5
— patient knows self, place, year, season and month	
Confused conversation	4
— able to answer questions but with disorientation or confusion	
Inappropriate speech	3
— random or exclamatory speech, no conversational exchange	
Incomprehensible speech	2
— no words uttered, only moaning	
No verbal response	1

Best Motor Response (M)

Obeys command	6
— does simple things you ask	
Purposeful movement and localizes to pain	5
Withdraws to pain	4
— pulls limb away from painful stimulus	
Flexor response to pain	3
— pain causes abnormal flexion of limbs or decorticate posture	
Extensor response to pain	2
— stimulus causes limb extension — decerebrate posture	
No motor response to pain	1

Total (E + V + M) = _____

Note: Lowest score is 3. Maximum score is 15.

Reference

Teasdale G, Jennett B. (1974) Assessment of coma and impaired consciousness: A practical scale. *Lancet* **2**: 81–83.

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STROKE SCALES AND CLASSIFICATIONS

Insert 3. OCSP Classification

Lacunar Syndrome (LACS)

- No visual field deficit
- No new disturbance of higher cortical functions
- No signs of brainstem disturbance
- PMS, PSS, SMS } must involve at least 2 out of 3 of Face, Arm (whole limb not just hand), or Leg
- AH/Clumpsy Hand-Dysarthria/Homolateral ataxia and crural paresis

Posterior Circulation Syndrome (POCS)

***Any** of:*

- Ipsilateral CN palsy (single or multiple with contralateral motor and/or sensory deficit
- Bilateral motor and/or sensory deficit
- Disorder of conjugate eye movement (horizontal or vertical)
- Cerebellar dysfunction w/o ipsilateral long tract deficit (as in Ataxic-Hemiparesis)
- Isolated hemianopsia or cortical blindness

May have disturbance of higher cortical function alongside any of the above

Total Anterior Circulation Syndrome (TACS)

***All** of:*

- Hemiplegia contralateral to the cerebral lesion
- Hemianopsia contralateral to the cerebral lesion
- New disturbance of higher cerebral function (e.g. dysphasia, visuospatial disturbance)

Partial Anterior Circulation Syndrome (PACS)

***Any** of:*

- Motor/sensory deficit and hemianopsia
- Motor/sensory deficit and new higher cerebral dysfunction
- New higher cerebral dysfunction and hemianopsia
- PMS/PSS deficit less extensive than for LACS (e.g. monoplegia)
- New higher cerebral dysfunction alone

When more than one type of deficit is present, they must all reflect damage in the same cerebral hemisphere.

Reference

Bamford J, Sandercock P, Dennis M, *et al*. (1991) Classification and natural history of clinically identifiable subtypes of cerebral infarction. *Lancet* **337**: 1521–1526.

Warlow CP, Dennis MS, van Gijn J, *et al*. (2001) *Stroke: A Practical Guide to Management*. Blackwell Science Ltd., Oxford, pp. 106–150.

May also use the following coding system:

Code	Findings on Neuro Exam
1	Unilateral weakness (and/or sensory deficit) affecting face
2	Unilateral weakness (and/or sensory deficit) affecting arm
3	Unilateral weakness (and/or sensory deficit) affecting hand
4	Unilateral weakness (and/or sensory deficit) affecting leg
5	Unilateral weakness (and/or sensory deficit) affecting foot
6	Dysphasia, dyslexia, dysgraphia (dominant hemisphere cortical)
7	Visuospatial disorder/inattention/neglect (non-dominant hemisphere)
8	Homonymous hemianopsia or quadrantanopsia
9	Brainstem/cerebellar signs other than ataxic-hemiparesis
10	Other deficits

TACS 1 + 2 + 3 + 4 + 5 + 6 + 7

LACS 1 + 2 + 3 + 4 + 5 or
1 + 2 + 3 or
2 + 3 + 4 + 5

POCS 8 or
9 or
8 + 9

PACS other combinations excluding 9 and 10

STROKE SCALES AND CLASSIFICATIONS

Insert 4. Intracerebral Hemorrhage (ICH) Score

Component	ICH Score Points
GCS score	
3–4	2
5–12	1
13–15	0
ICH volume, cm^3	
≥ 30	1
< 30	0
IVH	
Yes	1
No	0
Infratentorial origin of ICH	
Yes	1
No	0
Age (years)	
≥ 80	1
< 80	0
Total ICH Score (0–6)	_____

GCS score refers to Glasgow Coma Scale score on initial presentation (or after resuscitation).

IVH refers to presence of any intraventricular hemorrhage on initial CT scan.

ICH volume refers to hematoma volume on initial CT scan calculated by ABC/2 method:

$$\text{Hematoma volume (in cc)} = \frac{A \times B \times C}{2}$$

where: A = Largest diameter of hematoma (in cm)
B = Diameter perpendicular to A (in cm)
C = Number of slices on CT scan X slice thickness (in cm)

Count slice as 1 if size of hematoma is > 75% of the area seen on the slice with the largest hematoma
Count slice as 0.5 if size of hematoma is 25–75%
Disregard slice if size of hematoma is < 25%

Reference

Hemphill JC III, Bonovich DC, Besmertis L, *et al*. (2001) The ICH Score — A simple, reliable grading scale for intracerebral hemorrhage. *Stroke* **32**: 891–897.

Kothari RU, Brott T, Broderick JP, *et al.* (1996) The ABCs of measuring intracerebral hemorrhage volumes. *Stroke* **27**: 1304–1305.

STROKE SCALES AND CLASSIFICATIONS

Insert 5a. Hunt and Hess Classification for Subarachnoid Hemorrhage

Grade

I. Asymptomatic; or minimal headache and slight nuchal rigidity
II. Moderate to severe headache; nuchal rigidity; no neurologic deficit except cranial nerve palsy
III. Drowsy, minimal neurologic deficit
IV. Stuporous; moderate to severe hemiparesis; possibly early decerebrare rigidity and vegetative disturbances
V. Deep coma; decerebrate rigidity; moribund appearance

Reference

Hunt WE, Meagher JN, Hess RM. (1966) Intracranial aneurysm. A nine-year study. *Ohio State Med J* **62**: 1168–1171.

Insert 5b. World Federation of Neurosurgeons (WFNS) Grading Scale for Subarachnoid Hemorrhage

Grade

1 GCS 15
2 GCS 13–14, no focal neurological deficit, i.e. aphasia, hemiparesis/hemiplegia
3 GCS 13–14 with focal neurological deficit, i.e. aphasia, hemiparesis/hemiplegia
4 GCS 7–12
5 GCS 3–6

Reference

Drake CG. (1988) Report of World Federation on Neurological Surgeons committee on a universal subarachnoid hemorrhage grading scale. *J Neurosurg* **68**: 985–986.

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NOTES

NOTES

BASIC ADMITTING ORDERS AND STROKE CLINICAL PATHWAY

Nurse Clinician Noor Asiah Abu Aman and Nurse Clinician Winnie Soo

Goal

To coordinate comprehensive and up-to-date management of patients with TIA or acute stroke to improve outcome by preventing or limiting stroke progression and secondary complications.

Patient Data Needed

1. Clinical diagnosis of stroke
2. Stroke subtype ☐ Cerebral Infarct ☐ Intracerebral Hemorrhage ☐ Subarachnoid Hemorrhage

Actions

1. Start the stroke clinical pathway to streamline and coordinate care. (*See Insert 1 in this section*)
2. Do baseline evaluation and serial assessment, as often as necessary:
   — Stroke severity using appropriate stroke scales (*See section on Stroke Scales and Classifications*)
   — Blood pressure, heart rate, respiratory rate, and body temperature
   — Oxygen saturation
   — Blood sugar level
   — Pressure sores.
3. Apply elastic stockings in patients with impaired mobility or severely reduced power in the lower limbs. (*See also section on Deep Vein Thrombosis and Pulmonary Embolism*)
4. Do dysphasia screening (by trained staff) using the Water Swallow Test (WST). If patient fails WST, insert nasogastric tube and refer to Speech Therapist within 24 h of admission. (*See Insert 2 in this section*)
5. Refer all patients to a Physical Therapist and an Occupational Therapist for initial assessment. Start therapy within 24 h if necessary and if patient is able to. Refer to rehabilitation service for long-term rehabilitation plans, if needed.
6. If patient has diabetes mellitus, dyslipidemia, or hypertension, refer to a Dietician.
7. Identify psycho-social issues, if any, during admission and refer to a Medical Social Worker/Service as needed.
8. Institute bowel care. (*See Insert 3 in this section*)
9. Monitor for acute urinary retention and manage as necessary. (*See Insert 4 in this section*)
10. Start discharge planning as early as day 1 of admission.
11. Regularly assess the patient for complications and other medical needs that may indicate appropriateness of transfer to ICU, operating suite, rehabilitation ward, general ward or discharge home.

Evidence

1. Team agreement that optimal timing of treatments, reduced variations in coordinated care, pre-planned program of management, multidisciplinary approach, reduced amount of paper work through collaborative documentation, and identification of roles and responsibilities all positively influence quality and outcomes of stroke care.
2. Coordinated assessment procedures (medical, nursing and therapy assessments), early management policies (e.g. early mobilization; avoidance of urinary catheterization; treatment of hypoxia, hyperglycaemia and suspected infection), and ongoing rehabilitation policies (e.g. co-ordinated multidisciplinary team care, early assessment for discharge) are common characteristics of successful stroke units.
3. Organized inpatient stroke care improves outcome by prevention and early treatment of secondary complications from stroke, in particular infections, and by reducing need for institutional care through a reduction in disability by reducing stroke progression/recurrence.
4. While care pathways aim to promote organized and efficient patient care based on the best evidence and guidelines, the use of stroke care pathway may be associated with positive and negative effects. Patients treated with a care pathway may be less likely to suffer complications and more likely to have important tests performed. However its use may be associated with functional dependence upon discharge, poorer quality of life, and poorer satisfaction with hospital care.

Selected Readings

1. Underwood F, Parker J. (1998) Developing and evaluating an acute stroke care pathway through action research. *Nurse Res* **6**: 27–38.
2. Langhorne P, Pollock A. (2002) What are the components of effective strokes unit care? *Age Aging* **31**(5): 365–371.
3. Schwamm LH, Pancioli A, Acker JE III, *et al*. (2005) Recommendations for the establishment of stroke systems of care: Recommendations from the American Stroke Association's Task Force on the Development of Stroke Systems. *Stroke* **36**: 690–703.
4. Indredavik B, Bakke F, Slørdahl SA, *et al*. (1999) Treatment in a combined acute and rehabilitation stroke unit. Which aspects are most important? *Stroke* **30**: 917–923.
5. Leys D, Ringelstein EB, Kaste M, Hacke W for the European Stroke Initiative executive committee. (2007) The main components of stroke unit care: Results of a European expert survey. *Cerebrovasc Dis* **23**: 344–352.
6. Govan L, Langhorne P, Christopher J, Weir CJ for the Stroke Unit Trialists Collaboration. (2007) Does the prevention of complications explain the survival benefit of organized inpatient (stroke unit) care? Further Analysis of a Systematic Review. *Stroke* **38**: 2536–2540.
7. Kwan J, Sandercock P. (2004) In-hospital care pathways for stroke. Cochrane Database of Systematic Reviews, Issue 4. Art. No.: CD002924. DOI: 10.1002/14651858.CD002924.pub2.

BASIC ADMITTING ORDERS AND STROKE CLINICAL PATHWAY

Insert 1. Example of Stroke Clinical Pathway (from Acute Stroke Unit of Changi General Hospital)

LOS: 5 Days **ACUTE STROKE CLINICAL PATHWAY**

ACUTE STROKE CLINICAL PATHWAY

Ward	Bed	Unit	AFFIX PATIENT'S STICKY LABEL
		Med	NAME: .....
			NRIC: .....

Drug Allergy: Yes / No	Specialist in-charge: .....
If Yes, specify:	Case Manager: .....

Notes and Guidelines

- This clinical pathway commences upon clinical suspicion of stroke.

Inclusion criteria for admission to the Acute Stroke Unit (ASU)

1 Age below 80 yrs

2 Clinical diagnosis of acute TIA or stroke, ischemic or haemorrhagic particularly those occuring within 48 hrs

Exclusion criteria

1 Imminent intubation

2 High risk of cardiac or pulmonary collapse or other medical illness of greater severity that are better managed in ICU or by other specialties

3 Patients for withdrawal of treatment or not for active intervention

4 Persistent vegetative state

5 Premorbid status - Modified Rankin Score of 5

Investigations	Appointment Date & Time	Tick if done	Signature
CT scan brain			
MRI/MRA brain			
2D echocardiogram			
Carotid ultrasound			

Seen by:	Date	Signature
Physiotherapist		
Occupational Therapist		
Speech Therapist		
Dietitian		
Medical Social Worker		
Nurse Clinician		

Note : This clinical pathway is only a guideline. Patients' problems & interventions may be changed and individualised according to patient's condition and assessment data. It is not to be used as a substitute or replacement for independent clinical assessment and management.

ACUTE STROKE CLINICAL PATHWAY

AFFIX PATIENT'S STICKY LABEL

NAME: .....

NRIC: .....

A&E

Goals: 1. Rapid Triage of stroke patient **DATE:**

ASSESSMENT

Date & time of suspected stroke / TIA: ...../...../..... (DD MM YY) at .....:.....hours

Blood pressure (...../.....mmHg)

GCS E:__,V:__,M:__= total __ / 15

Pain score : _____

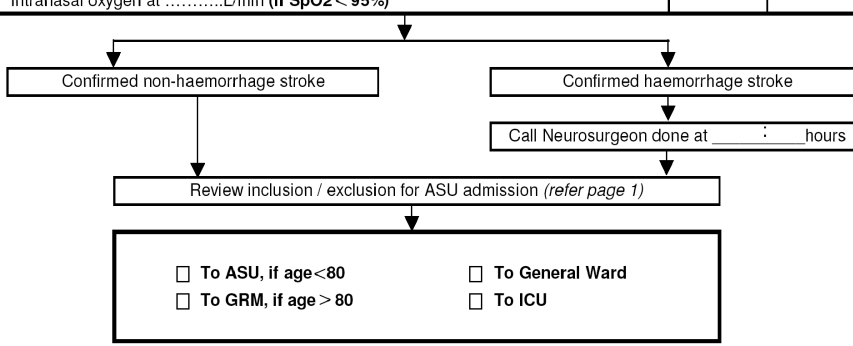
Temp: _____°C HR: _____/min RR: _____/min

Hypocount stat: .....mmol/L

SpO2 .....%

(Don't treat post-stroke hypertension unless SBP>220 or DBP>120mmHg; if needed, use labetolol 10 mg IV to give over 2 min)

Orders:	**DR**	**N**
☐ Urgent brain scan head done at _____hrs		
☐ CXR		
☐ ECG		
☐ NBM		
☐ IV therapy **(NS 1 pint x 12 hr unless dehydrated)**		
☐ Intranasal oxygen at .....L/min **(if SpO2 < 95%)**		



ADDITIONAL ORDERS	**DR**	**N**

NURSES' ACTIVITY	**N**
Maintain clear airway (insert airway if necessary)	
Elevate head at 30 degrees (if GCS < 15 or haemorrhagic stroke)	

DOCTOR I/C	**NURSE I/C**

ACUTE STROKE CLINICAL PATHWAY

Ward	Bed	Unit	AFFIX PATIENT'S STICKY LABEL
			NAME: .....
			NRIC: .....

ASU DAY 1

Goals : 1. Identify etiology of stroke 2. Reduce early mortality and complications
3. Stabilise concurrent medical condition

Day..... of Stroke Date:_____

Clinical

Assessment :

Blood pressure ...../.....mmHg

GCS E:__,V:__,M:__= total __ / 15

Pain score: _____

Temp: _____°C HR: _____/min RR: _____/min

Hypocount stat: .....mmol/L *(if not done in A&E)*

SpO2:_____*(if not done in A&E)*

NIHSS: _____

Investigations *(If not done in A&E)*	**DR**	**Nurse**
☐ FBC		
☐ PT/ INR		
☐ PTT		
☐ U/E/Cr		
☐ Fasting glucose		
☐ LFT		
☐ Fasting lipids		
☐ ECG		
☐ CXR		
☐ CT scan brain		
If required		
☐ Carotid ultrasound		
☐ MRI / MRA brain		
☐ 2D echocardiogram		
☐ Young stroke workup		
☐ DVT prophylaxis specify :		
☐ Oxygen therapy specify :		
Additional orders		

Doctors' Notes

Time

GENOGRAM

Name & Signature of Doctor

ACUTE STROKE CLINICAL PATHWAY

Nurse / Allied Health Service Notes	Nurses Notes
Nurse	
Physiotherapist	
Occupational Therapist	
Speech Therapist	
Dietitian	
Medical Social Worker	

Nurses' activity	AM	PM	ND
Proper positioning of patient's head and limbs			
Catheterise patient (if on IV mannitol)			
Water swallowing test done ☐ Passed ☐ Failed - refer to ST ☐ Not done			
Automatic referral to: ☐ PT ☐ OT ☐ Dietitian if on N/G tube			
Give Stroke education pamphlet to patient			
ASK - Is patient a chronic smoker? ☐ Yes ...../day ☐ No			
ADVISE - if patient is a smoker, is he /she agreeable to consider a smoking cessation program? ☐ Yes ☐ No ☐ Non smoker If Yes- refer smoking cessation counselor			

Inform doctor and document if: ☐ **Deterioration in conscious level** ☐ **SBP<100 or >220 or DBP>120**

(Tick in the box if present) ☐**SpO2<95% (Give I/N O2 if not on)** ☐ **Temp>38°C**

☐**Suspect dehydration** ☐ **Acute retention of urine ***

☐**Calf is tender/swollen (Right/Left)** ☐ **Constipation ***

☐**Choke or wet voice during feeding. Put on NBM and refer ST**

** Refer to annex 2 for acute retention of urine and bowel care*

	NAME OF NURSE IN-CHARGE	SIGNATURE
AM		
PM		
ND		

ACUTE STROKE CLINICAL PATHWAY

ASU DAY 1

Date:_____

NATIONAL INSTITUTE OF HEALTH STROKE SCALE (NIHSS)

Administer stroke scale items in the order listed. Record performance in each category after each subscale exam. Do not go back and change scores. Follow directions provided for each exam technique. Scores should reflect what the patient does, not what the clinician thinks the patient can do. The clinician should record answers while administering the exam and work quickly. Except where indicated, the patient should not be coached (i.e., repeated requests to patient to make a special effort).

Scale Definition	Score
1a. Level of Consciousness 0 Alert; keenly responsive. 1 Not alert, but arousable by minor stimulation to obey, answer, or respond. 2 Not alert, requires repeated stimulation to attend, or is obtunded and requires strong or painful stimulation to make movements(not stereotyped). 3 Responds only with reflex motor or autonomic effects or totally unresponsive, flaccid, areflexic.	
1b. LOC Questions 0 Answers both questions correctly. 1 Answers one question correctly. 2 Answers neither question correctly.	
1c. LOC Commands 0 Performs both tasks correctly 1 Performs one task correctly 2 Performs neither task correctly	
2. Best Gaze 0 Normal 1 Partial gaze palsy. This score is given when gaze is abnormal in one or both eyes but where forced deviation or total gaze paresis are not present. 2 Forced deviation, or total gaze paresis not overcome by the oculocephalic maneuver.	
3. Visual 0 No visual loss 1 Partial hemianopia 2 Complete hemianopia 3 Bilateral hemianopia (blind including cortical blindness)	
4. Facial Palsy 0 Normal symmetrical movement 1 Minor paralysis (flattened nasolabial fold, asymmetry on smiling) 2 Partial paralysis (total or near total paralysis of lower face) 3 Complete paralysis of one or both sides (absence of facial movement in the upper and lower face)	
5 & 6. Motor Arm and Leg 0 No drift, limb holds 90 (or 45) degrees for full 10 seconds 1 Drift, Limb holds 90 (or 45) degrees, but drifts down before full 10 seconds; does not hit bed or other support 2 Some effort against gravity, limb cannot get to or maintain (if cued) 90 (or 45) degrees, drifts down to bed, but has some effort against gravity 3 No effort against gravity, limb falls 4 No movement 9 Amputation, joint fusion explain: _____ 5a. Left Leg 5b. Right Leg 0 No drift, leg holds 30 degrees position for full 5 seconds 1 Drift, leg falls by the end of the 5 second period but does not hit bed 2 Some effort against gravity; leg falls to bed by 5 seconds, but has some effort against gravity 3 No effort against gravity, leg falls to bed immediately 4 No movement 9 Amputation, joint fusion explain: _____ 6a. Left Leg 6b. Right Leg	

Scale Definition	Score
7. Limb Ataxia 0 Absent 1 Present in one limb 2 Present in two limbs If present, is ataxia in? 9 amputation or joint fusion, explain: _____ Left arm 1 = Yes 2 = No Right arm 1 = Yes 2 = No 9 amputation or joint fusion, explain : _____ Left leg 1 = Yes 2 = No Right leg 1 = Yes 2 = No	
8. Sensory 0 Normal; no sensory loss. 1 Mild to moderate sensory loss; patient feels pinprick is less sharp or is dull on the affected side; or there is a loss of superficial pain with pinprick but patient is aware he/she is being touched. 2 Severe to total sensory loss; patient is not aware of being touched in the face, arm, and leg.	
9. Best Language 0 No aphasia, normal 1 Mild to moderate aphasia; some obvious loss of fluency or facility of comprehension, without significant limitation on ideas expressed or form of expression. Reduction of and/or comprehension, however, makes conversation about provided material difficult or impossible. For example, in conversation about provided materials examiner can identify picture or naming card from patient's response. 2 Severe aphasia; all communication is through fragmentary expression; great need for inference, questioning, and guessing by the listener. Range of information that can be exchanged is limited; listener carries burden of communication. Examiner cannotidentify materials provided from patient response. 3 Mute, global aphasia; no usable speech or auditory comprehension.	
10. Dysarthria 0 Normal 1 Mild to moderate; patient slurs at least some words and, at worst, can be understood with some difficulty. 2 Severe; patient's speech is so slurred as to be unintelligible in the absence of or out of proportion to any dysphasia, or is mute/anarthric. 9 Intubated or other physical barrier, explain: _____	
11. Extinction and Inattention (formerly Neglect) 0 No abnormality. 1 Visual, tactile, auditory, spatial, or personal inattention or extinction to bilateral simultaneous stimulation in one of the sensory modalities. 2 Profound hemi-inattention or hemi-inattention to more than one modality. Does not recognize own hand or orients to only one side of space.	
Total	

Date:_____ Time:_____

Done by (name):_____

Signature: _____

ACUTE STROKE CLINICAL PATHWAY

Acute Stroke Unit		
Ward	Bed	NAME: …… NRIC: ……

ABBREVIATED MENTAL TEST (AMT)

Ten-point AMT score. Score one point for each correct answer.	On admission	Upon transfer/ Disch
DATE		
Please remember the following phrase: "37, Bukit Timah Road". I will be asking you to repeat the phrase to me later.		
1 What is the present year? (Western calender, ie. 20__)		
2 What time is it now? (within 1 hour)		
3 What is your age? (for Chinese, +1yr is usually the norm and hence acceptable)		
4 What is your date of birth? (Western year +/- month and day)		
5 Where are we now? (For community survey, "my home" or "my son's home" etc. is probably acceptable)		
6 What is your home address? (complete address excluding postal code)		
7 Who is Singapore's present Prime Minister?		
8 Show picture of a nurse or doctor - what is his/ her job?		
9 Count backwards from 20 to 1		
10 Please recall the memory phrase.		
TOTAL SCORE out of 10		
REMARKS Add comment if communication or mood abnormal, eg. Deaf, dysphasic, depressed* may affect AMT. ** If depression suspected, consider referral to Psychiatrist*		
SIGNATURE		

ACUTE STROKE CLINICAL PATHWAY

Ward	Bed	Unit	AFFIX PATIENT'S STICKY LABEL NAME: .... NRIC: ....

ASU DAY 2

Goals : 1. Reduce early mortality and complications 2. Stabilise concurrent medical condition 3. Improve functional status

Day………….. of Stroke Date:____________

Clinical

Assessment :

Blood pressure ……./…….mmHg

GCS E:__,V:__,M:__= total __ / 15

Pain score: ______________

Temp: _____^{0}C HR: _____/min RR: _____/min

Hypocount: ………………mmol/L

SpO2:_______________________

NIHSS: _______________

Investigations (if required)	DR	Nurse
☐ FBC		
☐ PT/ INR		
☐ PTT		
☐ U/E/Cr		
☐ Fasting glucose		
☐ LFT		
☐ Fasting lipids		
☐ ECG		
☐ CXR		
☐ CT scan brain		
☐ Carotid ultrasound		
☐ MRI / MRA brain		
☐ 2D echocardiogram		
☐ Young stroke workup		
☐ DVT prophylaxis specify: ………………….....		
☐ Oxygen therapy specify: ………………….....		
Additional orders		

Doctors' Notes
Time

Name & Signature of Doctor

ACUTE STROKE CLINICAL PATHWAY

Nurse / Allied Health Service Notes
Nurse
Physiotherapist
Occupational Therapist
Speech Therapist
Dietitian
Medical Social Worker

Nurses Notes

Nurses' activity	AM	PM	ND
Assess if patient is able to feed			
Give 'Stroke education pamphlet' to patient if not done			
Assessed by smoking cessation counselor ☐ Yes ☐ No ☐ NA			

Inform doctor and document if: (Tick in the box if present)

- ☐ **Deterioration in conscious level**
- ☐ **SpO2 <95% (Give I/N O2 if not on)**
- ☐ **Suspect dehydration**
- ☐ **Calf is tender/swollen (Right/Left)**
- ☐ **SBP<100 or >220 or DBP>120**
- ☐ **Temp >38°C**
- ☐ **Acute retention of urine ***
- ☐ **Constipation ***
- ☐ **Choke or wet voice during feeding. Put on NBM and refer ST**

** Refer to annex 2 for acute retention of urine and bowel care*

	NAME OF NURSE IN-CHARGE	SIGNATURE
AM		
PM		
ND		

INSERT SHEET

INSERT DAY.................. **ACUTE STROKE CLINICAL PATHWAY**

Ward	Bed	Unit	AFFIX PATIENT'S STICKY LABEL NAME: .... NRIC: ....

ASU DAY ....

Goals : 1. To prevent deterioration 2. Maximise functional status 3. Stabilise current medical condition

Day.............. of Stroke Date:____________

Clinical

Assessment :

Blood pressure ..../.......mmHg

GCS E:__,V:__,M:__= total __ / 15

Pain score:_________________

Temp: _____ ^{0}C HR: _____/min RR: _____/min

Hypocount stat: ....mmol/L

SpO2:_______________________

NIHSS: _______________

Investigations (if required)	DR	Nurse
☐ FBC		
☐ PT/ INR		
☐ PTT		
☐ U/E/Cr		
☐ Fasting glucose		
☐ LFT		
☐ Fasting lipids		
☐ ECG		
☐ CXR		
☐ CT scan brain		
☐ Carotid ultrasound		
☐ MRI / MRA brain		
☐ 2D echocardiogram		
☐ Young stroke workup		
☐ DVT prophylaxis		
☐ specify:		
☐ Oxygen therapy		
specify:		
Additional orders		

Doctors' Notes
Time

Name & Signature of Doctor

F/WD/02-066.R0

ACUTE STROKE CLINICAL PATHWAY

Nurse / Allied Health Service Notes
Nurse
Physiotherapist
Occupational Therapist
Speech Therapist
Dietitian
Medical Social Worker

Nurses Notes

Nurses' activity	AM	PM	ND
Side rails checked & call bell within reach			
Assess patient if able to feed			

Inform doctor and document if:
(Tick in the box if present)

- ☐ Deterioration in conscious level
- ☐ SBP <100 or >220 or DBP >120
- ☐ SpO2 <95% (Give I/N O2 if not on)
- ☐ Temp >38°C
- ☐ Suspect dehydration or retention of urine
- ☐ Acute retention of urine *
- ☐ Calf is tender/swollen (Right/Left)
- ☐ Constipation *
- ☐ Choke or wet voice during feeding. Put on NBM and refer ST

** Refer to annex 2 for acute retention of urine and bowel care*

	NAME OF NURSE IN-CHARGE	SIGNATURE
AM		
PM		
ND		

ACUTE STROKE CLINICAL PATHWAY

Acute Stroke Unit		AFFIX PATIENT'S STICKY LABEL
Ward	Bed	NAME: …… NRIC: ……

DISCHARGE / TRANSFER SHEET

(Delete if not applicable)

Date: _____

Final Diagnosis : ☐ Stroke Specify : ……
☐ Not Stroke Specify : ……

Other medical conditions : …… ……
…… ……

DNR ☐ Yes ☐ No **AMD** ☐ Available ☐ Not available

NIHSS : …… AMT : …… GCS: ……

	Discharged	Ongoing	Not applicable
Physiotherapist			
Occupational Therapist			
Speech Therapist			
Dietitian			
Medical Social Services			
Rehab medicine :			

Planned final discharge destination
(to be filled by doctor):
☐ Home with no rehab
☐ Home with Caregiver or Day Centre
☐ Rehabilitation
☐ Nursing Home
☐ Community hospital
☐ Others……

ASU Discharge Destination :
☐ Home
☐ Nursing Home ☐ Community Hospital
☐ Others ……

Discharge criteria from ASU
- Neurologically / medically stable
- Requires no intravenous injection
- No fever
- Caregiven training completed
- Rehabilitation plans made (if needed)

ASU Transfer Destination: ☐ General Ward : Ward _____Bed _____
☐ Rehabilitation: Ward _____ Bed _____

Reason for transfer out of ASU (tick one):
☐ 1. Rehabilitation:
☐ 2. Neurologically/ medically stable & awaiting bed for rehab (acute or slow-stream)
☐ 3. Neurologically/ medically stable & awaiting community hospital or nursing home transfer
☐ 4. Stroke now beyond acute period and ASU bed needed by new admission
☐ 5. As requested by patient / family - options discussed
☐ 6. Others: _____

Discharge orders *(upon patient's discharge)*

☐ TCU Neurology Clinic x ……..weeks	Name of Doctor: ……
☐ TCU Rehab clinic x ……weeks	Name of Doctor: ……
☐ TCU General medicine clinic x ……weeks	Name of Doctor: ……
☐ TCU Cardiovascular clinic x ……weeks	Name of Doctor: ……
☐ TCU Psychiatrist x ……weeks	Name of Doctor: ……
☐ Follow up OPS / GP	☐ Others : Specify: ……

Nurses' activity	N
☐ Inform patient &/or relative of transfer or discharge	
☐ Arrange for transport if needed	
☐ Instruct patient &/or family on the appropriate diet before discharge	
☐ Complete discharge checklist	

DOCTOR I/C	SIGN	NURSE I/C	SIGN

ANNEX 1

ACUTE STROKE CLINICAL PATHWAY

BARTHEL INDEX OF ACTIVITIES OF DAILY LIVING (BI)

Aim to record what the patient actually DOES do in daily life, not what he/she can do. The score reflects the degree of INDEPENDENCE from help provided by another person:

* If supervision is required, the patient is NOT independent
* If aids and devices are used but no help is required, the patient IS independent

Middle categories imply that the patient supplies over 50% of the effort.

A : Estimated premorbid ADL **B :** Upon discharge from ASU **Date**	A	B
BOWELS 2 Continent 1 Occ. Accident < 1/week 0 Incontinent		
BLADDER 2 Continent 1 Occ. Accident < 1/day 0 Incontinent		
FEEDING 2 Independent 1 Needs help 0 Dependent		
GROOMING (Face/hair/teeth/shave) 1 Independent 0 Needs help		
DRESSING 2 Independent 1 Can do half 0 Dependent		
TRANSFER 3 Independent 2 Minor help 1 Major help (can sit) 0 Unable		
TOILET USE 2 Independent 1 Needs some help 0 Dependent		
MOBILITY 3 Independent 2 Walks with one person / assistance 1 Wheelchair independent 0 Unable		
STAIRS 2 Independent 1 Needs help 0 Unable		
BATHING 1 Independent 0 Dependent		
TOTAL SCORE		
OT Signature		
PT Signature		

Reference: Wade DT (1992)

BOWELS
2 Continent (for preceding week)
1 Occasional accident (once a week or less)
0 Any worse grade of incontinence (or needs enemas for continent

Bladder
2 Continent (for preceding week), or unable to manage any device (eg. Catheter & bag) without help
1 Occasional accident (once a day or less), or catheterised & needs help with device
0 Any worse grade of incontinence

Feeding, food place within reach by others
2 Able to cut up food, spread butter, etc. without help
1 Needs some help cutting or spreading
0 Needs to be fed

Grooming
1 Independent washing face, combing hair, shaving & cleaning teeth (when implements are provided)
0 Needs help

Dressing
2 Independent putting on all clothes, including fastening buttons, zips, etc. (clothes may be adapted)
1 Needs some help, but can do at least half
0 Needs more help than this

Transfer, bed to chair & back
3 Needs no help
2 Needs minor help, verbal or physical: can transfer with one person easily, or needs supervision
1 Needs major help: two people or one strong/trained person, but can sit unaided
0 Cannot sit; needs skilled lift by two people (or hoist)

Toilet use
2 Able to get on & off toilet or commode, undress & dress sufficiently, & wipe self without physical or verbal help
1 Needs some help, can wipe self & do some of the rest with minimal help only
0 Needs more help than this

Mobility around house or ward, indoors
3 May use aid (stick or frame etc. but not wheelchair)
2 Needs help of one person, verbal or physical, including help standing up
1 Independent in wheelchair, including able to negotiate doors & corners un aided
0 Needs more help than this

Stairs
2 Independent up & down, & can carry any necessary walking aid
1 Needs help, verbal or physical or help carrying aid
0 Unable

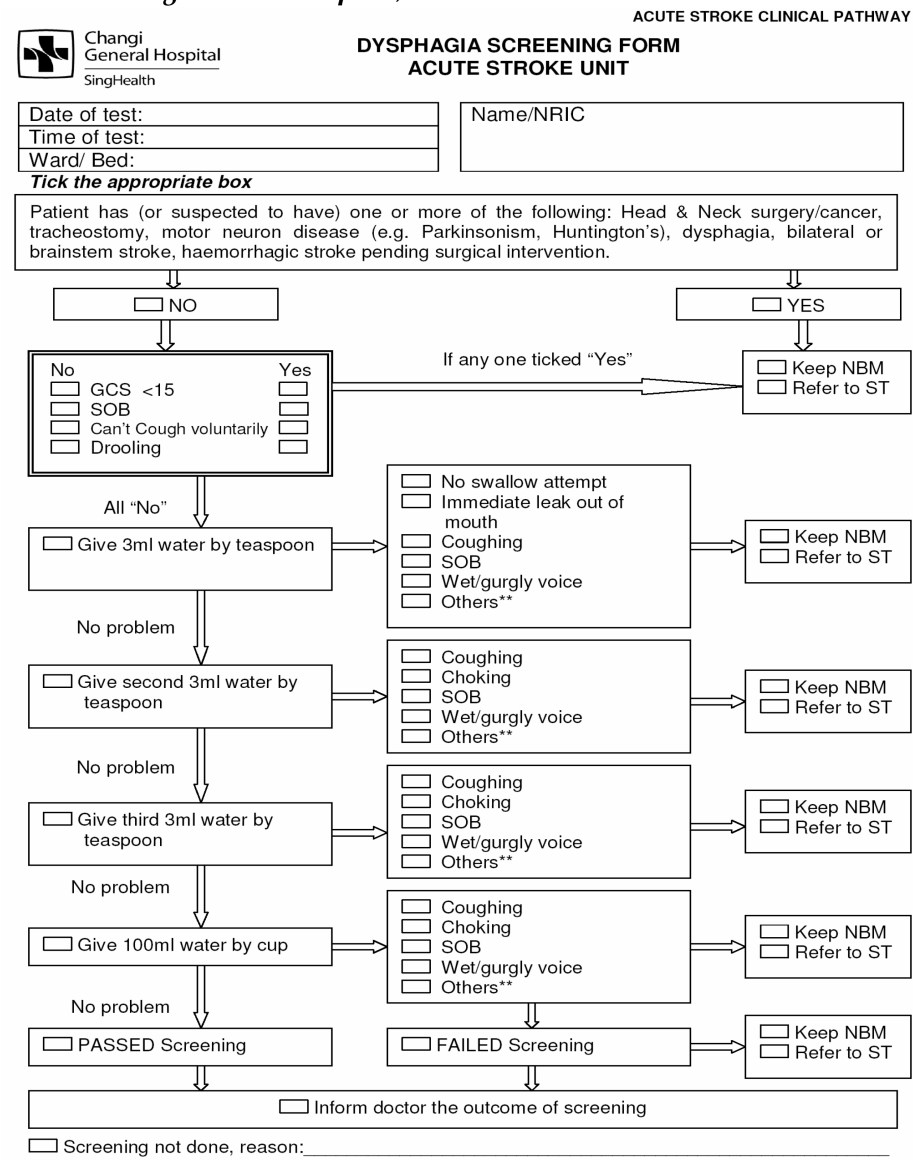
Bathing
1 Able to get in & out of bath or shower, wash self without help (may use any aids)
0 Unable

Circle the appropriate score

THE MODIFIED RANKIN SCORE	A	B
0 No symptoms, no significant disability 1 No significant disability, able to carry out all usual duties & activities 2 Slight disability, unable to carry out all previous activities 3 Moderate disability, requiring some help, but able to walk without assistance 4 Moderately severe disability, unable to walk without assistance & unable to attend to own bodily need without assistance. 5 Severe disabilty, bed ridden, incontinent & requiring constant nursing care & attention 6 Dead		

BASIC ADMITTING ORDERS AND STROKE CLINICAL PATHWAY

Insert 2. Example of Water Swallowing Test Protocol (from Acute Stroke Unit of Changi General Hospital)



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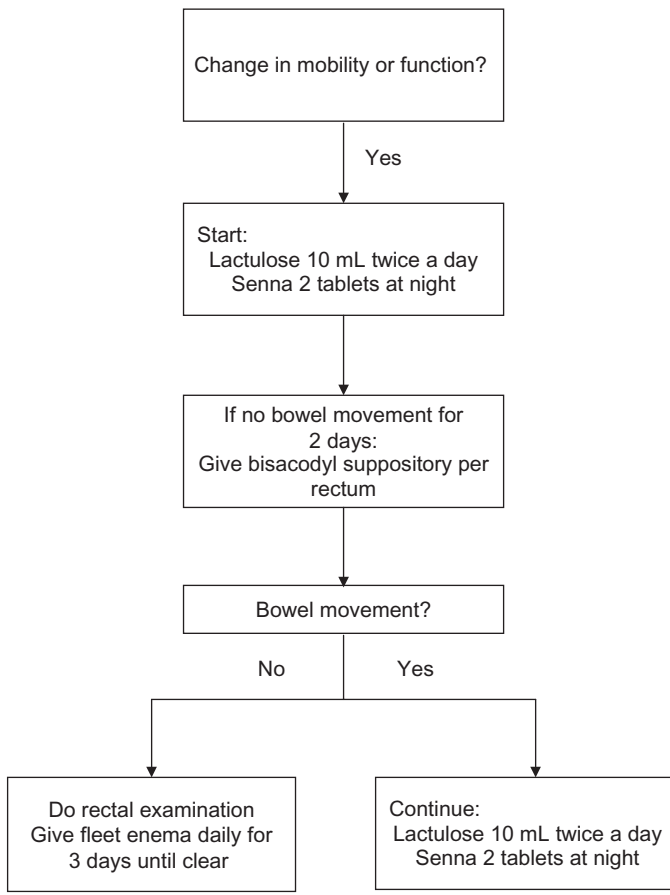
BASIC ADMITTING ORDERS AND STROKE CLINICAL PATHWAY

***Insert 3. Algorithm for Bowel Care* (*from Acute Stroke Unit of Changi General Hospital*)**



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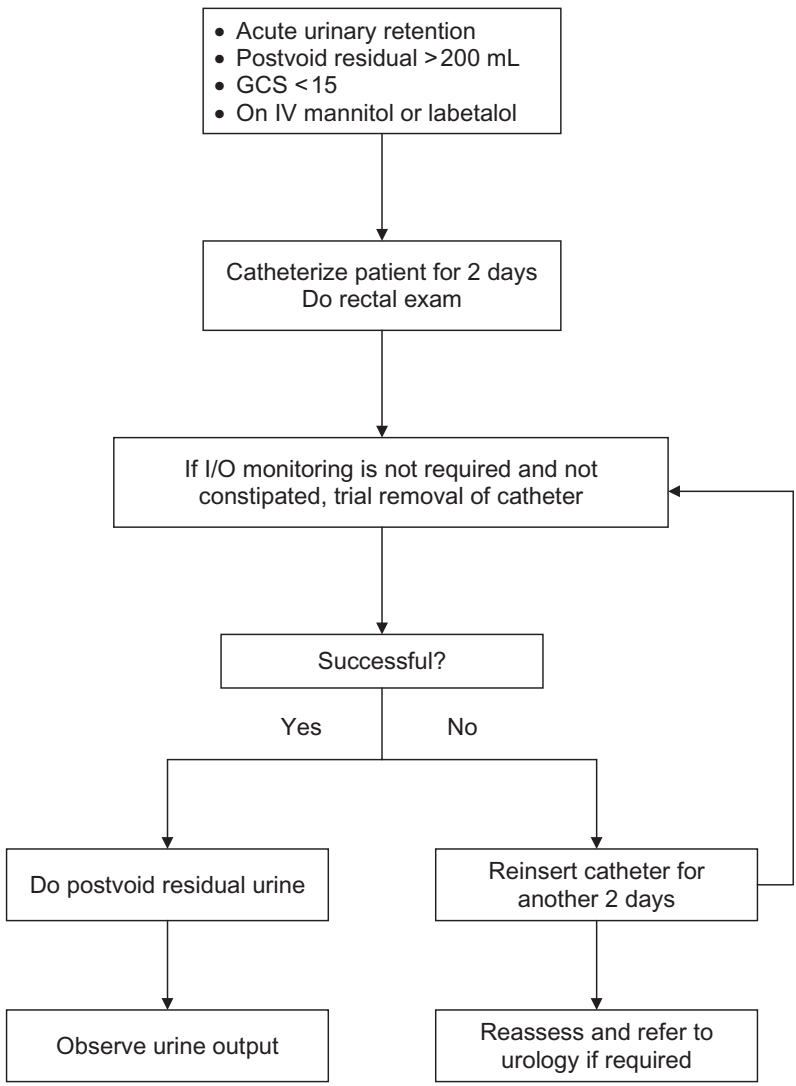
BASIC ADMITTING ORDERS AND STROKE CLINICAL PATHWAY

Insert 4. Algorithm for Management of Acute Urinary Retention (from Acute Stroke Unit of Changi General Hospital)



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TIA AND ACUTE ISCHEMIC STROKE: BASIC INVESTIGATIONS

Dr. Jorge L. Padilla

Goals

To confirm the stroke and elucidate its pathology, topography, mechanism, and prognosis.

To detect other medical conditions related to the stroke that may influence the strategy for acute intervention and secondary prevention.

Patient Data Needed

1. Clinical diagnosis of stroke
2. Cardiac examination, e.g. irregular heart rhythm, signs of valvular disease

Actions

1. Check capillary blood sugar level and oxygen saturation, if not yet done by paramedics or in the emergency room. (*See section on Prehospital Stroke Evaluation and Triage*)
2. Perform brain imaging as soon as possible, if not yet done. (*See section on Brain Imaging*)
3. Order the following first line hematological and biochemical tests
   - Full (complete) blood count
   - PT/INR
   - aPTT
   - ESR
   - Fasting blood glucose
   - Serum creatinine, urea and electrolytes
   - Fasting lipid profile
   - Liver function test.
4. Perform an ECG, especially in patients with irregularly irregular rhythm or concomitant cardiac signs and symptoms.
5. Perform chest X-ray, especially if cardiac or pulmonary disease is suspected.
6. Perform the following, if necessary and concomitant conditions are suspected:
   - Cervical spine X-ray — if patient unresponsive and trauma is suspected
   - Arterial blood gas — if hypoxia or hypercarbia is suspected
   - Blood culture and septic work-up — if patient febrile or infective endocarditis is suspected
   - Lumbar puncture — if subarachnoid hemorrhage is suspected but no blood is seen on brain imaging or if infectious cause for stroke is suspected
   - Toxicology screen — if drug abuse is suspected
   - Pregnancy test — if suspected or uncertain.

7. Order second-line (non-emergent) laboratory tests and procedures to determine underlying ischemic stroke mechanism by TOAST classification. (*See Insert 1 in this section and section on TIA and Acute Ischemic Stroke: Diagnostic Tests for TOAST Classification*)

Evidence

1. Hyperglycemia (or hypoglycemia) causes stroke-like syndrome and is associated with unfavorable outcome.
2. Clinical stroke scoring systems have a poor accuracy in distinguishing ischemic and hemorrhagic strokes. Acute stroke management requires neuroimaging prior to treatment.
3. Anemia and blood viscosity worsen the prognosis in ischemic stroke, more so in the presence of atherosclerosis and small vessel disease. Hematocrit levels above 60% increase the risk of stroke from 44% to 59%.
4. Many patients hospitalized with ischemic stroke/TIA, including those with known dyslipidemia and those taking lipid lowering agents, have measured LDL that is higher than recommended by national guidelines.
5. Cerebral cardioembolism, particularly atrial fibrillation, is the cause of acute ischemic strokes in 6% to 36% of patients.

Selected Readings

1. Working Group MOH Clinical Practice Guidelines. (2003) *Stroke and Transient Ischemic Attacks (TIA's): Assessment, Investigation, Immediate Management and Secondary Prevention*. Ministry of Health Singapore.
2. Adams HP, Adams RJ, Brott T, *et al.* (2003) Guidelines for the early management of patients with ischemic stroke: A scientific statement from the stroke council of the American Stroke Association. *Stroke* **34**: 1056–1083.
3. Editorial Team and Expert Panel. (2006) National Stroke Association Guidelines for the management of transient ischemic attacks. American Neurological Association. *Ann Neurol* **60**: 301–313.
4. Smith EE, Abdullah AR, Amirfarzan H, Schwamm LH. (2007) Serum lipid profile on admission for ischemic stroke. *Neurology* **68**: 660–665.
5. Warlow CP, Dennis MS, van Gijn J, *et al.* (2001) *Stroke: A Practical Guide to Management*. Blackwell Science Ltd., Oxford, pp. 265–300.
6. Bamford J. (2001) Assessment and Investigation of stroke and transient ischemic attack. *J Neurol Neurosurg Psych* **70** (Suppl I): i3–i6.

TIA AND ACUTE ISCHEMIC STROKE: BASIC INVESTIGATIONS

Insert 1. TOAST Classification

Large-artery Atherosclerosis

These patients will have clinical and brain imaging findings of either significant (> 50%) stenosis or occlusion of a major brain artery or branch cortical artery, presumably due to atherosclerosis. Clinical findings include those of cerebral cortical impairment (aphasia, neglect, restricted motor involvement, etc.) or brain stem or cerebellar dysfunction. A history of intermittent claudication, transient ischemic attacks (TIA's) in the same vascular territory, a carotid bruit, or diminished pulses helps support the clinical diagnosis. Cortical or cerebellar lesions and brain stem or subcortical hemispheric infarcts greater than 1.5 cm in diameter on CT or MRI are considered to be of potential large-artery atherosclerotic origin. Supportive evidence by duplex imaging or arteriography of a stenosis of greater than 50% of an appropriate intracranial or extracranial artery is needed. Diagnostic studies should exclude potential sources of cardiogenic embolism. The diagnosis of stroke secondary to large-artery atherosclerosis cannot be made if duplex or arteriographic studies are normal or show only minimal changes.

Cardioembolism

This category includes patients with arterial occlusions presumably due to an embolus arising in the heart. Cardiac sources are divided into high-risk and medium-risk groups based on the evidence of their relative propensities for embolism. At least one cardiac source for an embolus must be identified for a possible or probable diagnosis of cardioembolism stroke. Clinical and brain imaging finding are similar to those described for large-artery atherosclerosis. Evidence of a previous TIA or stroke in more than one vascular territory or systemic embolism supports a clinical diagnosis of cardiogenic stroke. Potential large-artery atherosclerotic sources of thrombosis or embolism should be eliminated. A stroke in a patient with a medium-risk cardiac source of embolism and no other cause of stroke is classified as a possible cardioembolic stroke.

Small-artery Occlusion (*lacune*)

This category includes patients whose strokes are often labeled as lacunar infarcts in other classifications. The patient should have one of the traditional clinical lacunar syndromes and should not have evidence of cerebral cortical dysfunction. A history of diabetes mellitus or hypertension supports the clinical diagnosis. The patient should also have a normal CT/MRI or examination or a relevant brain stem or subcortical hemispheric lesion with a diameter of less than 1.5 cm demonstrated. Potential cardiac sources for embolism should be absent, and evaluation of the large extracranial arteries should not demonstrate a stenosis of greater than 50% in an ipsilateral artery.

Stroke of Undetermined Etiology

In several instances, the cause of a stroke cannot be determined with any degree of confidence. Some patients will have no likely etiology determined despite an extensive evaluation. In others, no cause is found but the evaluation was cursory. This category also includes patients with two or more potential causes of stroke so that the physician is unable to make a final diagnosis. For example, a patient with a medium-risk cardiac source of embolism who also has another possible cause of stroke identified would be classified as having a stroke of undetermined etiology. Other examples would be a patient who has atrial fibrillation and an ipsilateral stenosis of 50%, or a patient with a traditional lacunar syndrome and an ipsilateral carotid stenosis of 50%.

Stroke of Other Determined Etiology

This category includes patients with rare causes of stroke, such as nonatherosclerotic vasculopathies, hypercoagulable states, or hematologic disorders. Patients in this group should have clinical and CT or MRI findings of an acute ischemic stroke, regardless of the size or location. Diagnostic studies such as blood tests or arteriography should reveal one of these unusual causes of stroke. Cardiac sources of embolism and large-artery atherosclerosis should be excluded by other studies.

Features of TOAST Classification of Subtypes of Ischemic Stroke

	Subtype			
Features	**Large Artery Athero-sclerosis**	**Cardio-embolism**	**Small Artery Occlusion (Lacune)**	**Other Cause**
Clinical				
Cortical or cerebellar dysfunction	+	+	−	+/−
Lacunar syndrome	−	−	+	+/−
Imaging				
Cortical, cerebellar, brainstem, or subcortical infarct > 1.5 cm	+	+	−	+/−
Subcortical or brainstem infarct < 1.5 cm	−	−	+/−	+/−
Tests				
Stenosis of extracranial internal carotid artery	+	−	−	−
Cardiac source of emboli	−	+	−	−
Other abnormality on tests	−	−	−	+

Reference

Adams HP, Bendixen BH, Kappelle LJ, *et al.* (1993) Classification of subtype of acute ischemic stroke: Definitions for use in a multicenter clinical trial. *Stroke* **24**: 35–41.

NOTES

NOTES

THROMBOLYSIS (rTPA) FOR ACUTE ISCHEMIC STROKE

Dr. Robert N. Gan

Goal

To improve long-term functional outcome by the safe administration of rTPA in eligible patients admitted for acute ischemic stroke.

Patient Data Needed

1. Date and time of onset of stroke: Date: _____ Time: _____
2. Brain imaging (*See section on Brain Imaging*)
3. Capillary blood sugar: _____ mg/dL or _____ mmol/L
4. Weight: _____ kg
5. Platelet count: _____ /mm^3
6. Prothrombin time (PT): _____ sec
7. Activated partial thromboplastin time (aPTT): _____ sec
8. Eligibility (*See Insert 1 in this section*): ☐ Yes ☐ No
9. Pretreatment NIHSS: _____ BP: _____ / _____ mm Hg

Actions

1. Confirm the date and time of onset of stroke symptom(s).
   - — Patients who noticed their stroke upon awakening should be considered to have the onset at the time when they went to sleep or was last known well.
   - — Do not include patients with uncertain time of onset of stroke symptoms).
2. Ensure there is ample time available for intravenous rTPA to be given within 3 h of stroke onset.
   - — For patients who are ineligible solely due to time frame (> 3 h), consider treatment with intra-arterial thrombolysis or mechanical clot extraction if facility and personnel are available and can be performed within 6 to 12 h.
3. Call Radiology to get the CT (or MRI) scanner ready.
4. Check the capillary blood sugar. Send off basic laboratory tests stat, if not yet done. (*See section on TIA and Acute Ischemic Stroke: Basic Diagnostic Tests*)
5. Check the blood pressure and perform NIH Stroke Scale. (*See section on Stroke Scales and Classifications*)
6. Perform a non-contrast CT scan (or MRI) of the brain and check for the presence of hemorrhage or distinct hypodensity that suggests that the stroke may have occurred more than 3 h prior. (*See section on Brain Imaging*)
7. Weigh the patient.
8. Insert the intravenous access, if not yet done earlier.
9. Trace the results of platelet count, PT, and aPTT.
10. Check the eligibility for rTPA administration. (*See Insert 1 in this section*)

11. If the patient is eligible, obtain informed consent if required by and according to local law.
12. Re-check and monitor the blood pressure. (*See Insert 2 in this section or section on BP in Acute Ischemic Stroke*)
13. Compute the rTPA total dose at 0.9 mg/kg (do not exceed maximum of 90 mg).
14. Give 10% of the total dose intravenously as bolus over 1 min. Infuse the remaining rTPA dose (90% of total) intravenously over 60 min.
15. No nasogastric tube (NGT), bladder catheter or intra-arterial insertion for 24 h.
16. Do not give aspirin, heparin or warfarin for 24 h.
17. Monitor the neurological status using NIHSS and GCS. (*See section on Stroke Scales and Classifications*)
18. If there is any acute neurological deterioration, new headache, acute hypertension, or nausea and vomiting following the start of rTPA infusion, suspect intracranial hemorrhage. (*See Insert 3 in this section*)
19. Obtain a follow-up CT scan of the brain at 24 h before starting antithrombotic agent.

Evidence

1. Intravenous administration of rTPA in appropriately selected patients within 3 h of ischemic stroke onset improves the likelihood of good clinical outcome at 3 months post-stroke. Risk of symptomatic intracerebral hemorrhage is 6%.
2. The benefit seen from rTPA is sustained up to 12 months after stroke.

Selected Readings

1. The National Institute of Neurological Disorders and Stroke rt-PA Stroke Study Group. (1995) Tissue plasminogen activator for acute ischemic stroke. *N Engl J Med* **333**:1581–1587.
2. Kwiatkowski TG, Libman RB, Frankel M, *et al*. (1999) Effects of tissue plasminogen activator for acute ischemic stroke at one year. *N Engl J Med* **340**: 1781–1787.
3. Adams HP, Jr, del Zoppo G, Alberts MJ, *et al*. (2007) Guidelines for the early management of adults with ischemic stroke: A guideline from the American Heart Association/American Stroke Association Stroke Council, Clinical Cardiology Council, Cardiovascular Radiology and Intervention Council, and the Atherosclerotic Peripheral Vascular Disease and Quality of Care Outcomes in Research Interdisciplinary Working Groups. *Stroke* **38**: 1655–1711.

THROMBOLYSIS (RTPA) FOR ACUTE ISCHEMIC STROKE

Insert 1. NINDS TPA Stroke Study Group Eligibility Guidelines

Eligibility for IV treatment with rTPA

- ☐ Age 18 years or older
- ☐ Clinical diagnosis of ischemic stroke causing a measurable neurological deficit
- ☐ Time of symptom onset well established to be less than 180 min before treatment would begin.

Contraindications

- ☐ Evidence of intracranial hemorrhage on pretreatment CT
- ☐ Clinical presentation suggestive of subarachnoid hemorrhage, even with normal CT
- ☐ Active internal bleeding
- ☐ Known bleeding diathesis, including but not limited to:
  - ☐ Platelet count < 100 000/mm^3
  - ☐ Current use of oral anticoagulants (e.g., warfarin sodium) or recent use with an elevated PT > 15 sec or INR > 1.5*
  - ☐ Patient has received heparin within 48 h and has an elevated aPTT (greater than upper limit of normal for laboratory)*
- ☐ Within 3 months of any intracranial surgery, serious head trauma, or previous stroke
- ☐ Myocardial infarction in the previous 3 months
- ☐ History of intracranial hemorrhage
- ☐ Known arteriovenous malformation, or aneurysm
- ☐ On repeated measurements, systolic blood pressure greater than 185 mm Hg or diastolic blood pressure greater than 110 mm Hg at the time treatment is to begin, and patient requires aggressive treatment to reduce blood pressure to within these limits (*See also Insert 2 in this section*)

Warnings and Cautions

- ☐ Only minor or rapidly improving stroke symptoms
- ☐ NIH Stroke Scale Score greater than 22
- ☐ CT scan showing multilobar infarction (hypodensity > 1/3 of cerebral hemisphere)
- ☐ Patient has had major surgery or serious trauma excluding head trauma in the previous 14 days
- ☐ History of gastrointestinal or urinary tract hemorrhage within 21 days
- ☐ Recent arterial puncture at a noncompressible site in the previous 7 days
- ☐ Recent lumbar puncture
- ☐ Blood glucose of < 50 mg/dL (< 2.7 mmol/L) or > 400 mg/dL (22.2 mmol/L)
- ☐ Post myocardial infarction pericarditis
- ☐ Patient was observed to have seizure at the same time the onset of stroke symptoms were observed.

**In patients without recent use of oral anticoagulants or heparin, treatment with rTPA can be initiated prior to the availability of coagulation study results but should be discontinued if either the PT is greater than 15 sec or the aPTT is elevated by local laboratory standards.*

THROMBOLYSIS (rTPA) FOR ACUTE ISCHEMIC STROKE

Insert 2. Guidelines for Blood Pressure Control in Patients Selected for rTPA

Pretreatment

- Monitor BP every 15 min. BP should be below 185/110 mm Hg to be eligible for treatment with rTPA.
- If systolic BP (SBP) > 185 mm Hg or diastolic BP (DBP) > 110 mm Hg, give:
  - — 1 or 2 doses of Labetalol 10–20 mg given IV over 1 to 2 min, or
  - — Nitroglycerin paste 1 to 2 inches, or
  - — Nicardipine infusion at 5 mg/h, titrate up by 2.5 mg/h every 5 to 10 min interval, maximum of 15 mg/h; when desired BP is achieved, reduce to 3 mg/h
- If these measures do not reduce BP below 185/110 and keep it down, the patient should not be treated with rTPA.

During and After Treatment

- Monitor BP for the first 24 h after starting treatment:
  - — Every 15 min for 2 h after starting the infusion, then
  - — Every 30 min for 6 h, then
  - — Every hour for 16 h.
- If DBP > 140 mm Hg on two readings 5 to 10 min apart:
  - — Start intravenous (IV) infusion of sodium nitroprusside (0.5 to 10 μg/kg/min).
- If SBP > 230 mm Hg or DBP is 121 to 140 mm Hg on two readings 5 to 10 min apart:
  - — Give labetalol 10 mg IV over 1 to 2 min. Dose may be repeated or doubled every 10 min, maximum dose 300 mg.
  - — Alternatively, following the first bolus of labetalol, IV infusion of 2 to 8 mg/min labetalol may be initiated and continued until the desired BP is reached.
  - — Another option is nicardipine infusion at 5 mg/h, titrate up to desired effect by increasing 2.5 mg/h every 5 min to maximum of 15 mg/h.
  - — If satisfactory response is not obtained, use IV sodium nitroprusside (0.5 to 10 μg/kg/min).
- If SBP is 180 to 230 mm Hg or DBP is 105 to 120 mm Hg on two readings 5 to 10 min apart:
  - — Give labetalol 10 mg IV over 1 to 2 min. The dose may be repeated or doubled every 10 to 20 min, maximum dose 300 mg.
  - — Alternatively, following the first bolus of labetalol, IV infusion of 2 to 8 mg/min labetalol may be initiated and continued until the desired BP is reached.

- Monitor BP every 15 min during the antihypertensive therapy. Observe for hypotension.
- If, in the clinical judgment of the treating physician, an intracranial hemorrhage is suspected, the administration of rTPA should be discontinued and an emergency CT scan or MRI should be obtained.

THROMBOLYSIS (rTPA) FOR ACUTE ISCHEMIC STROKE

Insert 3. Guidelines for Management of Suspected and Proven Intracerebral Hemorrhage in Patients Treated with rTPA

Suspected Intracranial Hemorrhage

- Suspect the occurrence of intracranial hemorrhage following the start of rTPA infusion if there is any acute neurological deterioration, new headache, acute hypertension, or nausea and vomiting.
- Discontinue rTPA infusion unless other causes of neurological deterioration are apparent.
- Immediately perform CT scan or MRI.
- Draw blood for PT, aPTT, platelet count, fibrinogen, and type and cross (may wait to do actual type and cross).
- Prepare 6 to 8 units of cryoprecipitate containing factor VIII.
- Prepare 6 to 8 units of platelets.

Intracranial Hemorrhage Confirmed

- Obtain fibrinogen results.
- Consider administering cryoprecipitate or platelets, if needed.
- Consider alerting and consulting a hematologist or neurosurgeon.
- Consider decision regarding further medical and/or surgical therapy.
- Consider second CT to assess progression of intracranial hemorrhage.
- Emergent neurosurgical consultation is highly recommended.

References

This protocol (Inserts 1 to 3) is based on research supported by the National Institute of Neurological Disorders and Stroke (NINDS) (N01–NS-02382, N01–NS-02374, N01–NS-02377, N01–NS-02381, N01–NS-02379, N01–NS-02373, N01–NS-02378, N01–NS-02376, N01–NS-02380).

Reference is also made to the manufacturer's prescribing information for alteplase (Genentech Inc., South San Francisco, California).

Adams HP, Jr, del Zoppo G, Alberts MJ, *et al.* (2007) Guidelines for the early management of adults with ischemic stroke: A guideline from the American Heart Association/American Stroke Association Stroke Council, Clinical Cardiology Council, Cardiovascular Radiology and Intervention Council, and the Atherosclerotic Peripheral Vascular Disease and Quality of Care Outcomes in Research Interdisciplinary Working Groups. *Stroke* **38**: 1655–1711.

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TIA AND ACUTE ISCHEMIC STROKE: ANTIPLATELETS

Dr. Jose Leonard Pascual and Dr. Chang Hui Meng

Goal

To improve long-term functional outcome by early administration of antiplatelet therapy in patients admitted for acute ischemic stroke or TIA.

Patient Data Needed

1. Clinical diagnosis of ischemic stroke or TIA
2. Brain imaging (CT scan or MRI)
3. Platelet count: _____ /mm^3
4. Prothrombin time (PT): _____ sec
5. Activated partial thromboplastin time (aPTT): _____ sec
6. Eligibility for intended thrombolysis with rTPA: ☐ Yes ☐ No
7. Hypersensitivity to aspirin: ☐ Yes ☐ No
8. Contraindication to antiplatelets (e.g. active gastrointestinal bleeding, hemophilia): ☐ Yes ☐ No

Actions

1. Confirm that the presenting TIA or stroke is not hemorrhagic using brain imaging. (*See section on Brain Imaging*)
2. Trace results of platelet count, PT and aPTT.
3. If patient is eligible to receive or was given thrombolytic (rTPA) therapy, do not give antiplatelet within 24 h of rTPA administration. (*See section on Thrombolysis (rTPA) in Acute Ischemic Stroke*)
4. If patient is not for thrombolysis with no contraindication to aspirin, immediately start aspirin 160–300 mg/day within 48 h of stroke onset. Alternatively, clopidogrel 75 mg/day (with or without initial loading dose of 300 mg) may be considered in patients with hypersensitivity to aspirin but no contraindication to antiplatelet therapy.
5. In patients who are unable to have brain imaging done within 48 h of onset, aspirin 160–300 mg may be considered if:
   - — signs and symptoms have completely resolved, suggestive of TIA, or
   - — stroke has no feature to suggest hemorrhagic type of stroke (e.g. decreased level of consciousness, headache, vomiting, severe hypertension, neck rigidity, etc.)

   Stop antiplatelet drug if brain imaging subsequently reveals intracranial hemorrhage.

Evidence

1. For every 1000 acute ischemic stroke patients treated early with aspirin, seven recurrent ischemic strokes can be prevented and 13 less patients will be dead

or severely disabled at 6 months compared to the risk of causing 2 intracranial hemorrhages.
2. The primary effect of aspirin in acute ischemic stroke is likely on early secondary prevention of recurrent events, as it is still unclear if it limits the neurological sequelae of the acute ischemic stroke itself.
3. More than 8900 patients included in IST and CAST did not have brain CT scan done prior to randomization. Among about 800 patients who were inadvertently entered in CAST and IST with a cerebral hemorrhage rather than an ischemic stroke, there was no evidence of an increased adverse effect of aspirin.
4. Loading doses of clopidogrel 300 mg followed by 75 mg per day have been efficacious in acute coronary syndromes among patients with aspirin hypersensitivity. However, there is currently no available data on the utility of this drug, alone or in combination with aspirin, in the setting of acute stroke.

Selected Readings

1. Albers GW, *et al*. (2004) Antithrombotic and thrombolytic therapy for ischemic stroke: The Seventh ACCP Conference on Antithrombotic and Thrombolytic Therapy. *Chest* **126** (Suppl 3): 483S–512S.
2. Gubitz G, Sandercock P, Counsell C. (2000) Antiplatelet therapy for acute ischaemic stroke. *Cochrane Database Syst Rev* CD000029.
3. Adams HP Jr, del Zoppo G, Alberts MJ, *et al*. (2007) Guidelines for the early management of adults with ischemic stroke: A guideline from the American Heart Association/American Stroke Association Stroke Council, Clinical Cardiology Council, Cardiovascular Radiology and Intervention Council, and the Atherosclerotic Peripheral Vascular Disease and Quality of Care Outcomes in Research Interdisciplinary Working Groups. *Stroke* **38**: 1655–1711.
4. International Stroke Trial Collaborative Group. (1997) The International Stroke Trial (IST): A randomised trial of aspirin, subcutaneous heparin, both, or neither among 19 435 patients with acute ischaemic stroke. *Lancet* **349**: 1569–1581.
5. CAST (Chinese Acute Stroke Trial) Collaborative Group. (1997) CAST: Randomised, placebo-controlled trial of early aspirin use in 20 000 patients with acute ischaemic stroke. *Lancet* **349**: 1641–1649.
6. Barer D, Cohen A, Bradford APJ, *et al.* (1997) Interpretation of IST and CAST stroke trials. *Lancet* **350**: 440–444.
7. Early use of Existing Preventive Strategies for Stroke (EXPRESS) study. (2007) Effect of urgent treatment of transient ischaemic attack and minor stroke on early recurrent stroke (EXPRESS study): A prospective population-based sequential comparison. *Lancet* **370**: 1432–1442.

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BLOOD PRESSURE IN ACUTE ISCHEMIC STROKE

Dr. Artemio Roxas

Goal

To maintain BP for optimal and safe cerebral perfusion.

Patient Data Needed

1. BP: _____ / _____ mm Hg
2. MAP = $\frac{\text{SBP} + (2 \times \text{DBP})}{3}$ = _____ mm Hg
3. ICP, if available: ____ mm Hg (1 mm H_2O = 0.07 mm Hg)
4. Eligible to receive or received thrombolytic therapy? ☐ Yes ☐ No

Actions

1. If patient is eligible to receive or was given thrombolytic therapy, control BP according to rTPA protocol. (*See Insert 1 in this section or section on Thrombolysis (rTPA) for Acute Ischemic Stroke*)
2. If patient has an ischemic stroke and is not eligible for thrombolytic therapy, treat only if BP > 220/120 mm Hg unless there is other end-organ involvement (e.g., aortic dissection, acute myocardial infarction, pulmonary edema, hypertensive encephalopathy). (*See Insert 2 in this section*)
3. Monitor the BP every 15 min during the antihypertensive therapy. Observe for hypotension.
4. Aim for a BP reduction of only ≈15% of pretreatment values during the first day.
5. Avoid drugs that may precipitously drop the BP and have prolonged effects, such as sublingual calcium channel antagonists.
6. Consider other medical conditions when selecting an antihypertensive agent, e.g. avoid beta-blocker in patients with history of asthma.
7. If the BP is uptrend and the heart rate is downtrend, consider increased ICP. (*See section on Increased Intracranial Pressure*)
8. If a direct ICP measurement is available, the BP and ICP control must aim to keep cerebral perfusion pressure (CPP) > 70 mmHg, where:

   $$\text{CPP} = \text{MAP} - \text{ICP}.$$

9. Treat and search for cause if the BP is substantially lower than expected (e.g. SBP < 90 mm Hg) for a given patient, considering past history of hypertension (treated or untreated). Therapeutic options include IV isotonic saline, treatment of congestive heart failure and arrhythmia, correction of blood loss, and infusion of pressor agents such as:

   — Phenylephrine 2 to 10 µg/kg/min
   — Dopamine 2 to 20 µg/kg/min
   — Norepinephrine 0.05 to 0.2 µg/kg/min.

Evidence

1. Extremely high and low BP correlate with poor prognosis. Evidence from randomized clinical trials with sufficient size and power to clarify the issue of optimal BP management in the acute stroke setting is scarce.
2. Most experts agree that in acute phase of ischemic stroke, elevated BP should not be lowered unless patient will receive or has received thrombolytic treatment or has evidence of end organ damage (aortic dissection, hypertensive encephalopathy, acute renal failure, acute pulmonary edema or acute MI).
3. The presence of an ischemic "penumbra" (brain tissue that is ischemic but still viable) surrounding the area of infarction and impaired autoregulatory response are major arguments against lowering systemic BP in patients with acute ischemic stroke. In the zone of ischemia, reduction in MAP may potentially further reduce cerebral blood flow (CBF) below the threshold of viability.
4. Patients with chronic hypertension and those with high-grade carotid stenosis may be particularly vulnerable to the effects of BP lowering in the acute setting. The limits of autoregulation are shifted to higher values in patients with chronic hypertension as compared with those without hypertension. As a result, CBF may be reduced even at a relatively higher MAP than in normotensive patients.
5. Treatment of acute extreme hypertension may reduce cerebral edema, risk of hemorrhagic transformation, further vascular injury and likelihood of early recurrence. However, firm experimental evidence supporting these potential benefits is lacking or contradictory.

Selected Readings

1. Adams HP, Jr, del Zoppo G, Alberts MJ, *et al*. (2007) Guidelines for the early management of adults with ischemic stroke: A guideline from the American Heart Association/American Stroke Association Stroke Council, Clinical Cardiology Council, Cardiovascular Radiology and Intervention Council, and the Atherosclerotic Peripheral Vascular Disease and Quality of Care Outcomes in Research Interdisciplinary Working Groups. *Stroke* **38**: 1655–1711.
2. Klijn CJ, Hankey GJ for the American Stroke Association and European Stroke Initiative. (2003) Management of acute ischaemic stroke: New guidelines from the American Stroke Association and European Stroke Initiative. *Lancet Neurol* **2**: 698–701.
3. EUSI writing committee. (2003) European Stroke Initiative recommendations for stroke management: Update 2003. *Cerebrovasc Dis* **16**: 311–337.
4. Massachusetts General Hospital Emergency Department Acute Stroke Protocol for Thrombolysis (lV/IA), http://www.massgeneral.orq/stopstroke/protocollVlAThrom.asp
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BLOOD PRESSURE IN ACUTE ISCHEMIC STROKE

Insert 1. Guidelines for BP Control in Patients Selected for rTPA

Pretreatment

- Monitor the BP every 15 min. The BP should be below 185/110 mm Hg to be eligible for treatment with rTPA.
- If the SBP > 185 mm Hg or the diastolic BP (DBP) > 110 mm Hg, give:
  - — 1 or 2 doses of Labetalol 10–20 mg via IV over 1 to 2 min, or
  - — Nitroglycerin paste 1 to 2 inches, or
  - — Nicardipine infusion at 5 mg/h, titrate up by 2.5 mg/h every 5 to 10 min interval, maximum of 15 mg/h; when the desired BP is achieved, reduce to 3 mg/h.
- If these measures do not reduce the BP below 185/110 and keep it down, the patient should not be treated with rTPA.

During and After Treatment

- Monitor the BP for the first 24 h after starting treatment:
  - — Every 15 min for 2 h after starting the infusion, then
  - — Every 30 min for 6 h, then
  - — Every hour for 16 h.
- If the DBP > 140 mm Hg on two readings 5 to 10 min apart:
  - — Start intravenous (IV) infusion of sodium nitroprusside (0.5 to 10 μg/kg/min).
- If the SBP > 230 mm Hg or the DBP is 121 to 140 mm Hg on 2 readings 5 to 10 min apart:
  - — Give labetalol 10 mg IV over 1 to 2 min. The dose may be repeated or doubled every 10 min, up to a maximum dose of 300 mg.
  - — Alternatively, following the first bolus of labetalol, an IV infusion of 2 to 8 mg/min labetalol may be initiated and continued until the desired BP is reached.
  - — Another option is a nicardipine infusion at 5 mg/h, titrate up to the desired effect by increasing 2.5 mg/h every 5 min to a maximum of 15 mg/h.
  - — If a satisfactory response is not obtained, use an IV sodium nitroprusside (0.5 to 10 μg/kg/min).
- If the SBP is 180 to 230 mm Hg or the DBP is 105 to 120 mm Hg on 2 readings 5 to 10 min apart:
  - — Give labetalol 10 mg IV over 1 to 2 min. The dose may be repeated or doubled every 10 to 20 min, ip to a maximum dose of 300 mg.
  - — Alternatively, following the first bolus of labetalol, an IV infusion of 2 to 8 mg/min labetalol may be initiated and continued until the desired BP is reached.

- Monitor the BP every 15 min during the antihypertensive therapy. Observe for hypotension.
- If, in the clinical judgment of the treating physician, an intracranial hemorrhage is suspected, the administration of rTPA should be discontinued and an emergency CT scan or MRI should be obtained.

BLOOD PRESSURE IN ACUTE ISCHEMIC STROKE

Insert 2. Guidelines for BP Control in Patients Not Eligible for rTPA

- If the SBP ≤ 220 mm Hg or the diastolic BP (DBP) ≤ 120 mm Hg:
  - — Observe only unless there is other end-organ involvement (e.g., aortic dissection, acute myocardial infarction, pulmonary edema, hypertensive encephalopathy).
  - — Treat other symptoms of stroke (e.g., headache, pain, agitation, nausea, vomiting).
  - — Treat acute complications of stroke (e.g., hypoxia, increased ICP, seizures, hypoglycemia).
- If SBP > 220 mm Hg or DBP of 121–140 mm Hg on 2 readings 5–10 min apart:
  - — Give labetalol 10–20 mg intravenously (IV) over 1–2 min. May repeat or double every 10 min (up to a maximum dose of 300 mg).
  - — Alternatively, the first bolus of labetalol may be followed by an IV infusion of 2 to 8 mg/min labetalol and continued until the desired BP is reached.
  - — May also consider a nicardipine IV infusion at 5 mg/h as initial dose; titrate to desired effect by increasing 2.5 mg/h every 5 min to a maximum of 15 mg/h.
- If DBP > 140 mm Hg on 2 readings 5–10 min apart:
  - — Start sodium nitroprusside 0.5 μg/kg/min IV as initial dose with continuous blood pressure monitoring.
- Monitor BP every 15 min during the antihypertensive therapy. Observe for hypotension.

Reference

Adams HP, Jr, del Zoppo G, Alberts MJ, *et al*. (2007) Guidelines for the early management of adults with ischemic stroke: A guideline from the American Heart Association/American Stroke Association Stroke Council, Clinical Cardiology Council, Cardiovascular Radiology and Intervention Council, and the Atherosclerotic Peripheral Vascular Disease and Quality of Care Outcomes in Research Interdisciplinary Working Groups. *Stroke* **38**: 1655–1711.

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TIA AND ACUTE ISCHEMIC STROKE: DIAGNOSTIC TESTS FOR TOAST CLASSIFICATION

Dr. Disya Ratanakorn and Dr. Jesada Keandoungchun

Goal

To determine underlying mechanism of TIA or ischemic stroke so as to guide the treatment choices in acute management and secondary prevention.

Patient Data Needed

1. Clinical diagnosis of ischemic stroke
2. Stroke syndrome by OCSP classification (*See section on Stroke Scales and Classifications*)
   - ☐ TACS
   - ☐ PACS
   - ☐ POCS
   - ☐ LACS
3. Cardiac examination, e.g. irregular heart rhythm, signs of valvular disease
4. ECG result
5. modified Ranking Scale (mRS) (*See section on Rehabilitation, PT, OT, and ST*)

Actions

1. Perform basic investigations, if not yet done. (*See section on TIA and Acute Ischemic Stroke: Basic Investigations*)
2. Perform extracranial carotid and vertebral duplex ultrasonography. (*See Insert 1 in this section*)
   a. The alternatives are extracranial magnetic resonance angiography (MRA) or computed tomogram angiography (CTA).
   b. If an abnormality is detected, determine if the abnormal vessel is "symptomatic" for current TIA or stroke syndrome. Consider the abnormal vessel as "symptomatic" if:
      — stenosis in the carotid artery is on the same side as ischemic stroke or TIA responsible for syndrome (TACS, PACS or LACS), or
      — abnormality in the vertebral artery is detected in setting of POCS.
   c. If the patient is elderly and the stroke is severely disabling (mRS ≥ 4), an assessment of extracranial vessels may be postponed until the patient's functional status improves.
3. Assess the intracranial vessel for evidence of stenosis, altered hemodynamics, and collateral flows using TCD (*See Insert 2 in this section*), MRA or CTA.
4. Perform tranthoracic 2-dimensional echocardiography (TTE).
   — TTE is optional if the stroke syndrome is LACS, the CT scan or MRI shows subcortical infarct ≤ 1.5 cm, and ECG is normal.
   — Consider TEE if TTE is unrevealing but cardiac or aortic arch source is highly suspected.
5. Consider cardiac monitoring during the first 24 h or holter monitoring to screen for atrial fibrillation and other potentially serious cardiac arrhythmias.

6. Perform vascular (i.e. carotid and vertebral duplex, TCD or MRA or CTA) and cardiac (i.e. echocardiogram) studies in all stroke patients ≤ 45 years old. In addition, do tests to determine unusual causes of ischemic stroke. (*See section on Ischemic Stroke in Young Adults and Cerebral Venous Sinus Thrombosis*)
7. Use the results of diagnostic procedures and laboratory tests to determine the underlying ischemic stroke mechanism by TOAST classification. (*See Insert 3 in this section*).
8. Consider a catheter angiography if:
   — surgical or endovascular intervention is planned
   — further studies are needed for treatment decision-making, or
   — unusual cause for stroke is suspected and less invasive tests are equivocal (e.g. dissection, arteritis, moyamoya, and other non-atherosclerotic vasculopathies).
9. Decide on choice of antithrombotic treatment for early secondary prevention. (*See section on Antithrombotics: Antiplatelets and Anticoagulant*)

Evidence

1. There is excellent correlation between duplex ultrasonography and other neuroimaging modalities in the diagnosis of carotid stenosis.
2. Established clinical indications for TCD in acute stroke are to identify intracranial stenoocclusive disease, to measure vasomotor reactivity test in severe ICA stenosis/occlusion, to detect microembolic signal, to assess intracranial hemodynamics and collateral in extracranial ICA stenosis, to detect vasospasm in subarachnoid hemorrhage, and to enhance thrombolysis in combination with intravenous rTPA.
3. Cardiogenic sources of embolism in stroke include atrial fibrillation or flutter, acute myocardial infarction with left ventricular thrombus, cardiomyopathy, valvular heart disease (rheumatic mitral valve disease, prosthetic heart valve, aortic valve disease, mitral valve prolapse, mitral annular calcification), and endocarditis.
4. Less common causes of ischemic stroke include non-atherosclerotic vascular abnormalities (e.g. dissection, vasculitis/arteritis, moyamoya, fibromuscular dysplasia), hypercoagulable state, paradoxical embolism through a patent foramen ovale, congenital metabolic abnormality (e.g. homocysteinemia, Fabry disease), and CADASIL.

Selected Readings

1. Nederkoorn PJ, Mali WP, Eikelboom BC, *et al*. (2002) Preoperative diagnosis of carotid artery stenosis: Accuracy of noninvasive testing. *Stroke* **33**: 2003–2008.
2. Tegeler CH, Ratanakorn D. (2004) Carotid and vertebral duplex scanning in secondary stroke prevention and stenting. In: *Andrei V. Alexandrov, ed. Cerebrovascular Ultrasound in Stroke Prevention and Treatment*. NY: Blackwell Publishing, Inc./Futura Division, pp. 161–169.
3. Sloan MA, Alexandrov AV, Tegeler CH, *et al*. (2004) Assessment: Transcranial Doppler ultrasonography. *Neurology* **62**: 1468–1481.
4. WASID Trial Investigators. (2006) Predictors of ischemic stroke in the territory of a symptomatic intracranial arterial stenosis. *Circulation* **113**: 555–563.
5. Sacco RL, Adams R, Albers G, *et al*. (2006) Guidelines for prevention of stroke in patients with ischemic stroke or transient ischemic attack. *Stroke* **37**: 577–617.

TIA AND ACUTE ISCHEMIC STROKE: DIAGNOSTIC TESTS FOR TOAST CLASSIFICATION

Insert 1. Extracranial Carotid and Vertebral Duplex Ultrasonography

- Insonate both extracranial carotid and vertebral arteries to identify CCA, bifurcation, ICA, ECA, and VA. Screen using cross-sectional and longitudinal views.
- Use Doppler flow velocities to measure PSV and EDV of all vessels at proximal, distal, and stenotic points.
- Use brightness mode (B-mode) imaging, color-flow imaging, and power Doppler imaging to describe and measure the IMT, plaque location, size and characteristics, degree of stenosis, vertebral flow velocity and direction as well as abnormalities observed in nearby structures, such as thyroid mass, internal jugular vein abnormality, carotid body tumor, etc.
- Assess for the presence of dissection (thrombus, flap, etc.) and other arterial abnormalities.
- Estimate degree of the ICA stenosis by using each laboratory's validated criteria or by using the the Society of Radiologists in Ultrasound Consensus criteria below:

Consensus Panel Gray-Scale and Doppler US Criteria for Diagnosis of ICA Stenosis

	Primary Parameters		**Additional Parameters**	
Degree of Stenosis (%)	**ICA PSV (cm/sec)**	**Plaque Estimate (%)***	**ICA/CCA PSV Rato**	**ICA EDV (cm/sec)**
Normal	< 125	None	< 2.0	< 40
< 50	< 125	< 50	< 2.0	< 40
50–69	125–230	≥ 50	2.0–4.0	40–100
≥ 70 but < near occlusion	> 230	≥ 50	> 4.0	> 100
Near occlusion	High, low, or undetectable	Visible	Variable	Variable
Total occlusion	Undetectable	Visible, no detectable lumen	Not applicable	Not applicable

* Plaque estimate (diameter reduction) by gray-scale and color Doppler US

- Use the ischemic cuff test to identify subclavian-vertebral steal if early systolic deceleration, alternating flow, or flow reversal is detected in the vertebral artery.
- Promptly notify attending physicians of any crucial abnormalities observed, such as severe carotid stenosis, moving thrombus, or dissection.

Reference

Grant EG, Benson CB, Moneta GL, *et al*. (2003) Carotid artery stenosis: Gray-scale and Doppler US diagnosis — Society of Radiologists in Ultrasound Consensus Conference. *Radiology* **229**: 340–346.

TIA AND ACUTE ISCHEMIC STROKE: DIAGNOSTIC TESTS FOR TOAST CLASSIFICATION

Insert 2. TCD Ultrasonography

- Insonate the basal intracranial arteries around the circle of Willis via temporal windows for middle cerebral arteries (MCA-M1), anterior cerebral arteries (ACA-A1), and posterior cerebral arteries (PCA-P1 and P2), orbital windows for ophthalmic arteries (OA) and carotid siphons (CS), and occipital windows for vertebral arteries (VA) and basilar artery (BA).
- Identify the specific arteries by probe position, depth of insonation, flow direction, and continuity of insonation along the length of vessel.
- Record the spectral Doppler flow velocities, waveforms, and pulsatility indices at proximal, distal, and stenotic sites.
- Evaluate the degree of intracranial artery stenosis using validated PSV or MFV criteria, e.g. MFV ≥ 80 cm/sec for MCA-M1, MFV ≥ 60 for ACA-A1, MFV ≥ 50 for PCA, MFV ≥ 70 for CS, MFV ≥ 60 for BA, and MFV ≥ 50 for VA suggesting intracranial artery stenosis of more than 50%.
- Interpret the overall flow velocities in the context of side-to-side differences and spectral waveforms.
- Assess the collateral blood flow pattern, including the flow reversal in OA and ACA-A1.
- Consider assessment of vasomotor reactivity in patients with severe stenosis or occlusion in the extracranial ICA or CS.
- Consider TCD monitoring to detect MES in patients with significant extra-intracranial stenosis or cardioembolic source.
- Consider TCD bubble study to detect right-to-left shunt (PFO, pulmonary shunt), especially in young patients.

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TIA AND ACUTE ISCHEMIC STROKE: DIAGNOSTIC TESTS FOR TOAST CLASSIFICATION

Insert 3. TOAST Classification

Large-artery Atherosclerosis

These patients will have clinical and brain imaging findings of either significant (> 50%) stenosis or occlusion of a major brain artery or branch cortical artery, presumably due to atherosclerosis. Clinical findings include those of cerebral cortical impairment (aphasia, neglect, restricted motor involvement, etc.) or brain stem or cerebellar dysfunction. A history of intermittent claudication, TIAs in the same vascular territory, a carotid bruit, or diminished pulses helps support the clinical diagnosis. Cortical or cerebellar lesions and brain stem or subcortical hemispheric infarcts greater than 1.5 cm in diameter on CT or MRI are considered to be of potential large-artery atherosclerotic origin. Supportive evidence by duplex imaging or arteriography of a stenosis of greater than 50% of an appropriate intracranial or extracranial artery is needed. Diagnostic studies should exclude potential sources of cardiogenic embolism. The diagnosis of stroke secondary to large-artery atherosclerosis cannot be made if duplex or arteriographic studies are normal or show only minimal changes.

Cardioembolism

This category includes patients with arterial occlusions presumably due to an embolus arising in the heart. Cardiac sources are divided into high-risk and medium-risk groups based on the evidence of their relative propensities for embolism. At least one cardiac source for an embolus must be identified for a possible or probable diagnosis of cardioembolism stroke. Clinical and brain imaging finding are similar to those described for large-artery atherosclerosis. Evidence of a previous TIA or stroke in more than one vascular territory or systemic embolism supports a clinical diagnosis of cardiogenic stroke. Potential large-artery atherosclerotic sources of thrombosis or embolism should be eliminated. A stroke in a patient with a medium-risk cardiac source of embolism and no other cause of stroke is classified as a possible cardioembolic stroke.

Small-artery Occlusion (*lacune*)

This category includes patients whose strokes are often labeled as lacunar infarcts in other classifications. The patient should have one of the traditional clinical lacunar syndromes and should not have evidence of cerebral cortical dysfunction. A history of diabetes mellitus or hypertension supports the clinical diagnosis. The patient should also have a normal CT/MRI or examination or a relevant brain stem or subcortical hemispheric lesion with a diameter of less than 1.5 cm demonstrated. Potential cardiac sources for embolism should be absent, and evaluation of the large extracranial arteries should not demonstrate a stenosis of greater than 50% in an ipsilateral artery.

Stroke of Undetermined Etiology

In several instances, the cause of a stroke cannot be determined with any degree of confidence. Some patients will have no likely etiology determined despite an extensive evaluation. In others, no cause is found but the evaluation was cursory. This category also includes patients with two or more potential causes of stroke so that the physician is unable to make a final diagnosis. For example, a patient with a medium-risk cardiac source of embolism who also has another possible cause of stroke identified would be classified as having a stroke of undetermined etiology. Other examples would be a patient who has atrial fibrillation and an ipsilateral stenosis of 50%, or the patient with a traditional lacunar syndrome and an ipsilateral carotid stenosis of 50%.

Stroke of Other Determined Etiology

This category includes patients with rare causes of stroke, such as nonatherosclerotic vasculopathies, hypercoagulable states, or hematologic disorders. Patients in this group should have clinical and CT or MRI findings of an acute ischemic stroke, regardless of the size or location. Diagnostic studies such as blood tests or arteriography should reveal one of these unusual causes of stroke. Cardiac sources of embolism and large-artery atherosclerosis should be excluded by other studies.

Features of TOAST Classification of Subtypes of Ischemic Stroke

	Subtype			
Features	**Large Artery Athero-sclerosis**	**Cardio-embolism**	**Small Artery Occlusion (Lacune)**	**Other Cause**
Clinical				
Cortical or cerebellar dysfunction	+	+	–	+/–
Lacunar syndrome	–	–	+	+/–
Imaging				
Cortical, cerebellar, brainstem, or subcortical infarct > 1.5 cm	+	+	–	+/–
Subcortical or brainstem infarct < 1.5 cm	–	–	+/–	+/–
Tests				
Stenosis of extracranial internal carotid artery	+	–	–	–
Cardiac source of emboli	–	+	–	–
Other abnormality on tests	–	–	–	+

Reference

Adams HP, Bendixen BH, Kappelle LJ, *et al.* (1993) Classification of subtype of acute ischemic stroke: Definitions for use in a multicenter clinical trial. *Stroke* **24**: 35–41.

NOTES

NOTES

TIA AND ACUTE ISCHEMIC STROKE: ANTICOAGULATION

Dr. Hamidon Basri

Goal

To identify and safely treat patients with acute stroke or TIA who may benefit from early anticoagulation.

Patient Data Needed

1. Brain imaging (*See section on Brain Imaging*)
2. 12–lead ECG
3. Holter monitoring or telemetry for suspected paroxysmal atrial fibrillation (AF)
4. Reembolization risk analysis
   - ☐ Findings on 2D echocardiogram (trans-esophageal, if possible)
   - ☐ Presence of artificial valves
   - ☐ Recent myocardial infarction (within 4 weeks)
   - ☐ Valve vegetations/infective endocarditis
5. Evidence on US, MRI/MRA, CT angiography, or angiography of:
   - ☐ Extracranial carotid or vertebral dissection
   - ☐ Cerebral venous/sinus thrombosis
   - ☐ Severe basilar artery stenosis
   - ☐ Severe carotid stenosis
6. Evidence of DVT or PE (*See section on Deep Venous Thrombosis/Pulmonary Embolism*)

Actions

1. Do not urgently anticoagulate cerebral infarcts of moderate to large size or those with evidence of significant hemorrhage/hemorrhagic conversion.
2. Do not anticoagulate patients with evidence of infective endocarditis.
3. Consider early anticoagulation with IV heparin (*See Inserts 1 and 2 in this section*) or low molecular weight heparin (LMWH), if not contraindicated, for patients with evidence of:
   - — Intracardiac clot
   - — Artificial cardiac valve
   - — MI within 4 weeks
   - — Extracranial carotid or vertebral dissection
   - — Cerebral venous/sinus thrombosis
   - — DVT/PE (*See section on Deep Venous Thrombosis/Pulmonary Embolism*)
   - — Severe basilar artery stenosis
   - — Severe carotid stenosis with progressing or recurrent TIA/stroke.
4. In patients with AF or paroxysmal AF or evidence of hypercoagulable state (*See section on Stroke in Young Adults*) without concomitant conditions listed in #3

above, anticoagulation with IV heparin or LMWH may be started when the patient is neurologically and medically stable.
5. Do not combine antiplatelets with anticoagulation, unless there is strong medical indication (e.g. emergent intervention for ongoing MI, etc.).
6. For patients requiring and amenable to long-term anticoagulation, adjusted-dose warfarin to maintain target INR between 2.0 to 3.0 may be started in the hospital. (*See section on* Antithrombotics: Antiplatelets and Anticoagulant)
7. Closely monitor the patient for systemic and intracranial bleeding. Perform plain CT scan of the brain to rule out hemorrhage if there is new headache, nausea or vomiting, neurological deterioration, or acute hypertension.

Evidence

1. Early anticoagulation after an acute cardioembolic stroke does not reduce recurrent stroke in the first 14 days nor death or disability on follow-up and is associated with an increase in risk of symptomatic intracranial bleeding.
2. In patients with AF and TIA or minor stroke, risk of death, myocardial infarction, stroke, and systemic embolism is 19% per year. Anticoagulation reduces the annual risk to 8% (stroke risk reduced from 12% to 4% per year) while aspirin reduces risk to 15%. Risk of major bleeding for anticoagulation is 2.8% per year and for aspirin is 0.9% per year.
3. Based on small trials, indirect evidence, and consensus, most clinicians give heparin in large artery dissection and cerebral vein thrombosis. There are gaps in evidence for early anticoagulation in high-grade large artery stenosis with repeated events and in high-risk cardiac lesions (e.g. atrial or ventricular thrombus). Decisions to anticoagulate early should be individualized.

Selected Readings

1. Paciaroni M, Agnelli G, Micheli S, Caso V. (2007) Efficacy and safety of anticoagulant treatment in acute cardioembolic stroke. *Stroke* **38**: 423–430.
2. Caplan L. (2003) Resolved: Heparin may be useful in selected patients with brain ischemia. *Stroke* **34**: 230–231.
3. Sacco RL, Adams R, Albers G, *et al*. (2006) Guidelines for prevention of stroke in patients with ischemic stroke or TIA: A statement for healthcare professionals from the American Heart Association/American Stroke Association Council on Stroke. *Circulation* **113**: 409–449.
4. Atrial Fibrillation Investigators. (1994) Risk factors for stroke and efficacy of anti-thrombotic therapy in atrial fibrillation. Pooled data from five RCTs. *Arch Intern Med* **154**: 1449–1457.
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6. Bath PM, Iddenden R, Bath FJ. (2000) Low-molecular-weight heparin and heparinoids in acute ischaemic stroke: A meta-analysis of RCTs. *Stroke* **3**1: 1770–1778.
7. Hart RG, Palacio S. (2005) Cardioembolic stroke. EMedicine in http://www.emedicine.com/NEURO/topic45.htm

TIA AND ACUTE ISCHEMIC STROKE: ANTICOAGULATION

Insert 1. Example of Guideline for Intravenous Administration of Unfractionated Heparin in Stroke Patients (from NNI-TTSH)

1. Do baseline FBC (CBC), PT, and aPTT before starting IV heparin therapy.
2. Dilute 10 000 units of heparin in a micro-infusion chamber with normal saline to a total volume of 100 ml.
   - The diluted concentration is 100 units/ml.
   - Use 0.45% NaCl (1/2 normal saline) if patient has hypernatraemia > 155 mmol/l.
3. Infuse the diluted heparin through an IV line at the following recommended starting dose:

Body Weight (kg)	Starting Dose (units/h)
< 50	700
50–59	800
60–69	900
> 70	1000

4. Do not give any bolus dose.
5. This IV infusion must not be stopped or interrupted for other medications or activities.
6. aPTT must be repeated 4–6 h after starting IV heparin infusion and with every dose adjustment.
   - Blood for aPTT must be taken only after 4 h of uninterrupted continuous heparin infusion.
   - To avoid falsely prolonged APTT results due to heparin contamination, specimens should not be collected from the same extremity used for heparin infusion.
7. Adjust the IV heparin infusion dose by using the following titration nomogram as a guide:

APTT (sec)	Dose Change (units/h)	Additional Action
< 36	+200	Repeat aPTT in 4–6 h
36–59	+100	Repeat aPTT in 4–6 h
60–82	0	Repeat aPTT in 4–6 h (see #9)
83–120	–100	Stop heparin for 1 h; repeat aPTT
> 120	–200	in 4–6 h after restarting heparin

8. Change the infusion rate according to the dose-infusion rate table below with each dose adjustment. Do not change the heparin concentration.

Dose (units/h)	Infusion Rate (ml/h)
300	3
400	4
500	5
600	6
700	7
800	8
900	9
1000	10
1100	11
1200	12
1300	13
1400	14
1500	15
1600	16

9. aPTT may be repeated only once (or twice) daily if 2 consecutive measurements are within the therapeutic range.
10. Do FBC (CBC) twice a week to detect heparin-induced thrombocytopenia.

TIA AND ACUTE ISCHEMIC STROKE: ANTICOAGULATION

Insert 2. Example of Weight-based Normogram used for Heparin Therapy (from Royal University Hospital, Saskatoon, Saskatchewan, Canada)

PHYSICIAN'S ORDERS

Heparin-Adjusted Normogram for Stroke/Transient Ischemic Attack

Date:	Time:	Processed at/by:

1. Medication is provided intravenously. No intramuscular injections.
2. APTT, INR, and CBC must be drawn prior to start of therapy.
3. CBC must be drawn at least every three days during therapy.
4. If patient has a transient ischemic attack, a bolus of intravenous heparin may be given at the discretion of the admitting physician. Suggested IV heparin bolus is 50 U/kg to a maximun of 5000 U. If a bolus is desired, this must be indicated:

   ☐ Give _____ U IV heparin as bolus.

5. Initial dosing for continuous infusion of intravenous heparin:

WEIGHT (kg)	INITIAL INFUSION
☐ < 50	500 U/h = 10 mL/h
☐ 50–59	600 U/h = 12 mL/h
☐ 60–69	700 U/h = 14 mL/h
☐ 70–79	800 U/h = 16 mL/h
☐ 80–89	900 U/h = 18 mL/h
☐ 90–99	1000 U/h = 20 mL/h
☐ 100–109	1100 U/h = 22 mL/h
☐ 110–119	1200 U/h = 24 mL/h
☐ > 119	1400 U/h = 28 mL/h

6. aPTT is to be drawn 6 h after heparin therapy initiation.

7. Adjusted heparin therapy as according to APTT based on sliding scale:

aPTT (sec)	STOP INFUSION	RATE CHANGE	REPEAT APTT
< 40	–	Increase by 250 U/h	6 h
40–49	–	Increase by 150 U/h	6 h
50–59	–	Increase by 100 U/h	6 h
60–90	–	–	Next a.m.
91–100	–	Decrease by 100 U/h	6 h
101–120	–	Decrease by 150 U/h	6 h
> 120	60 min	Decrease by 250 U/h	6 h

8. If significant bleeding occurs, stop heparin and call physician to reassess.

Physician's signature: _____ Date: _____

Reference

Toth C, Voll C. (2002) Validation of a weight-based nomogram for the use of intravenous heparin in transient ischemic attack or stroke. *Stroke* **33**: 670–674.

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ISCHEMIC STROKE IN YOUNG ADULTS AND CEREBRAL VENOUS THROMBOSIS

Dr. Jose Leonard Pascual and Dr. Christopher Chen Li-Hsian

Goal

To facilitate the recognition, diagnosis, and treatment of ischemic stroke in young adults (≤ 45 years old) and patients with cerebral venous sinus thrombosis (CVST) through a rational, methodical, and cost-effective approach.

Patient Data Needed

1. History of any known risk factors for accelerated atherosclerosis
   - ☐ hypertension
   - ☐ diabetes mellitus
   - ☐ dyslipidemia
   - ☐ cigarette smoking
2. History of any known non-atherosclerotic disease or risks
   - ☐ heavy consumption of alcoholic beverages (≥ 4 servings per day)
   - ☐ cardiac condition (e.g. valvular disease, atrial fibrillation, PFO, etc.)
   - ☐ oral contraceptive pill use
   - ☐ substance abuse
   - ☐ head or neck trauma
   - ☐ any sudden/worsening severe headaches or migraine
3. Conditions concomitant with stroke:
   - ☐ pregnancy or post-partum
   - ☐ seizures
   - ☐ headache
   - ☐ fever or infection

Actions

1. Check eligibility and consider thrombolysis if the patient presents within 3 h of stroke onset. (*See section on Thrombolysis* (*rTPA*) *in Acute Ischemic Stroke*)
2. Perform a brain imaging if not yet done. (*See section on Brain Imaging*)
   - — Rule out stroke "mimics" (e.g. multiple sclerosis, neoplasms, etc.)
   - — Suspect CVST if lesion(s) is/are in unusual locations (e.g. high convexity, bilateral, not in specific arterial distribution) with hemorrhagic component especially in the setting of associated new-onset seizures and/or headache.
3. Perform the basic investigations if not yet done. Do pregnancy test in women of child-bearing age. (*See sections on TIA and Acute Ischemic Stroke: Basic Investigations*)
4. Start antiplatelet. (*See section on TIA and Acute Ischemic Stroke: Antiplatelets*)
5. Perform the second-line diagnostic tests if not yet done. (*See sections on TIA and Acute Ischemic Stroke: Diagnostic Tests for TOAST Classification*)
6. In addition, order the following if no obvious mechanism of stroke is detected:
   - — serum fasting homocysteine
   - — protein C and protein S
   - — antithrombin III, activated protein C resistance (or factor V Leiden mutation)

   — ESR, ANA
   — anticardiolipin antibodies, lupus anticoagulant
   — VDRL or RPR
   — toxicology tests (do early to avoid negative results due to "washout").
7. Screen for DVT if PFO is detected.
8. Perform further arterial or venous imaging if needed, i.e. MR angiogram or venogram, CT angiogram or venogram, or catheter angiogram.
9. Consider early anticoagulation if patient has cervical arterial dissection, CVST, or other indications. (*See section on TIA and Acute Ischemic Stroke: Anticoagulation*)
10. Discontinue oral contraceptive intake in women and offer alternative methods.
11. Determine choice of long-term antithrombotic. (*See section on Antithrombotics: Antiplatelets and Anticoagulant*)

Evidence

1. Cardioembolism (20–25%) and non-atherosclerotic causes (7–22%) are more common than atherothrombosis (7–8%) in young Asian patients with ischemic stroke. Among non-atherosclerotic strokes, cervical artery dissection is the most common cause (24%). Other causes are thrombophilic states, substance abuse, pregnancy, arteriopathies (e.g. moyamoya, arteritis/vasculitis, Takayasu's), etc.
2. The rarity of known prothrombotic states (hyperhomocysteinemia and protein C, protein S, or antithrombin III deficiencies) precludes the conduct of controlled trials to assess their interaction with oral contraceptive use. Case reports and studies in large families indicate that young women with these prothrombotic states are at high risk for extracerebral venous thrombosis.
3. Data from a few small controlled trials favor the use of heparin in patients with CVST, with the reduction of risk of death and severe disability without undue increase in intracranial hemorrhage. No controlled data is available regarding the benefit and optimal duration of oral anticoagulation therapy in patients with CVST.
4. Ischemic strokes in young adults have diverse etiologies and treatment should be individualized accordingly, depending on the underlying disorder.

Selected Readings

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2. Einhaupl K, Bousser M-G, de Bruijn SFTM, *et al*. (2006) EFNS guideline on the treatment of cerebral venous and sinus thrombosis. *Eur J Neurol* **13**: 553–559.
3. Stam J, de Bruijn SFTM, deVeber G. (2001) Anticoagulation for cerebral venous thrombosis. The Cochrane Database of Systematic Reviews; Issue 4. Art. No.: CD002005.
4. Lee TH, Hsu WC, Chen CJ, Chen ST. (2002) Etiologic study of young ischemic stroke in Taiwan. *Stroke* **33**: 1950–1955.
5. Lipska K, Sylaja PN, Sarma PS, *et al*. (2007) Risk factors for acute ischaemic stroke in young adults in South India. *J Neurol Neurosurg Psychiatry* **78**: 959–963.
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7. Marcoux M. (2000) Stroke in young adults. *CNI Rev.* **11**(2).

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ACUTE INTRACEREBRAL HEMORRHAGE: DIAGNOSTIC TESTS

Dr. Sherwin Joy Agustin and Dr. Robert N. Gan

Goal

To document the presence of spontaneous ICH and determine its underlying etiology

Patient Data Needed

1. Clinical diagnosis and features of ICH
   - ☐ sudden focal neurological deficits
   - ☐ headache
   - ☐ vomiting
   - ☐ impaired consciousness
   - ☐ decreased sensorium
2. No definite history of trauma before onset of symptom(s)
3. History of:
   - ☐ hypertension
   - ☐ intake of anticoagulant or antiplatelet medications
   - ☐ smoking
   - ☐ alcohol abuse
   - ☐ blood dyscrasias
   - ☐ recreational drug abuse
   - ☐ connective tissue disease
4. Allergy to intravenous contrast agent ☐ Yes ☐ No

Actions

1. Check the capillary blood sugar level and oxygen saturation, if not yet done by paramedics or in the emergency room. (*See section on Prehospital Stroke Evaluation and Triage*)
2. Order the following first line hematological and biochemical tests
   - Full (complete) blood count
   - Prothrombin time and international normalized ratio (PT/INR)
   - Activated partial thromboplastin time (aPTT)
   - Serum creatinine, urea and electrolytes
   - Glucose
   - Liver function test.
3. Perform non-contrast CT scan or MRI (including gradient echo and/or FLAIR sequences) of the brain as soon as possible, if not yet done. (*See section on Brain Imaging*)
4. Perform ECG.
5. Perform chest X-ray.
6. Perform the following, if necessary and concomitant conditions suspected:
   - Toxicology screen — if drug abuse is suspected

- Pregnancy test in women — if suspected or uncertain
- Cervical spine X-ray — if patient unresponsive and trauma is suspected
- Arterial blood gas — if hypoxia or hypercarbia is suspected
- Blood culture — if patient febrile or infective endocarditis is suspected.
- Erythrocyte sedimentation rate and C reactive protein — if connective tissue disease, vasculitis, or infective endocarditis is suspected.

7. Consider catheter angiography if there is/are:
   - associated subarachnoid hemorrhage
   - abnormal calcifications
   - obvious vascular anomalies
   - blood in unusual locations (e.g. sylvian fissure, isolated IVH)
   - no obvious cause of hemorrhage, such as young, normotensive subjects.

   If the patient cannot tolerate procedure, CTA or MRA may be performed instead. If the patient has allergy to contrast agent or frank renal failure, perform a non-contrast MRA.
8. If angiogram is normal, hemorrhage is lobar in location, the patient has no history of hypertension, and is a surgical candidate, consider a follow-up MRI to look for cavernous malformations.

Evidence

1. ICH is a medical emergency with frequent, early, ongoing bleeding, progressive deterioration, severe clinical deficits, subsequent high mortality and morbidity rates, and should be promptly recognized and diagnosed.
2. The volume of ICH on admission and presence of hydrocephalus on brain scan are powerful predictors of death at 30 days.
3. CT and MRI show equal ability to identify the presence of acute ICH, its size and location, and hematoma enlargement. In patients with contraindications to MRI, CT scan should be obtained.
4. Causes of ICH include hypertension (40–60%), aneurysm and AVM (10–20%), amyloid angiopathy (5–10%), coagulopathy (5%), tumor (2–10%), drug-related (5%), hemorrhagic transformation of ischemic stroke (5–7%), trauma, and arteritis.
5. Common locations of hypertensive ICH are the basal ganglia, thalamus, cerebellum, and brainstem (pons).

Selected Readings

1. Broderick JP, Conolly S, Feldman E, *et al*. (2007) Guidelines for the management of spontaneous intracerebral hemorrhage in adults: Update: A guideline from the American Heart Association/American Stroke Association Stroke Council, High Blood Pressure Research Council, and the Quality of Care and Outcomes in Research Interdisciplinary Working Group. *Stroke* **38**: 2001–2023.
2. Qureshi AI, Tuhrim S, Broderick JP, *et al*. (2001) Spontaneous intracerebral hemorrhage. *N Engl J Med* **344**: 1450–1460.
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4. Caplan LR. (1992) Intracerebral hemorrhage. *Lancet* **339**: 656–658.

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ACUTE INTRACEREBRAL HEMORRHAGE: MEDICAL AND BP MANAGEMENT

Dr. Robert N. Gan and Dr. Ernest Wang

Goal

To prevent clinical worsening and maintain blood pressure for optimal and safe cerebral perfusion after acute ICH.

Patient Data Needed

1. BP: _____ / _____ mm Hg
2. MAP = $\frac{SBP + (2 \times DBP)}{3}$ = _____ mm Hg
3. ICP, if available: _____ mm Hg (1 mm H_2O = 0.07 mm Hg)
4. Given antiplatelet, anticoagulation, or thrombolytic treatment ☐ Yes ☐ No

Actions

1. Stop the antiplatelet, anticoagulation, or thrombolytic treatment if given. Reverse the antithrombotic effect and correct coagulopathy if necessary.
2. If the BP is uptrend and the heart rate is downtrend, suspect increased ICP and institute measures to control the ICP. (*See section on Increased Intracranial Pressure*)
3. Maintain SBP ≤ 180 mm Hg or MAP < 130 mm Hg.
   - — If the SBP is > 200 mm Hg or the MAP is > 150 mm Hg, treat the BP with frequent BP monitoring every 5 min.
   - — If the SBP is 181–200 mm Hg or the MAP is 130–150 mm Hg, and
     i. with evidence of or suspicion of an increased ICP, consider ICP monitoring and treat BP but keep cerebral perfusion pressure (CPP = MAP – ICP) > 60 mm Hg.
     ii. with no evidence or suspicion of an increased ICP, target modest reduction to a BP of about 160/90 mm Hg or a MAP of 110 mm Hg.
4. Use any of the following for elevated BP management:

Drug	Intravenous Bolus	Infusion
Labetalol	5–20 mg every 15 min	2–8 mg/min (max 300 mg/d)
Nicardipine	–	5–15 mg/h
Esmolol	250 µg/kg loading dose	25–300 µk/kg/min
Enalapril	0.625–5 mg every 6 h	–
Hydralazine	5–20 mg every 30 min	1.5–5 µg/kg/min
Sodium Nitroprusside	–	0.1–10 µg/kg/min
Nitroglycerin	–	20–400 µg/min

5. Consider other medical conditions when selecting antihypertensive agent, e.g. avoid beta-blocker in patients with history of asthma.

6. Avoid drugs that may precipitously drop the BP and have prolonged effects, such as sublingual calcium channel antagonists.
7. Monitor the BP and neurological status every 15 min during antihypertensive therapy. Observe for hypotension.
8. Consider referring to a neurosurgeon. (*See section on Acute Intracerebral Hemorrhage: Surgery*)
9. Avoid MAP > 110 mm Hg in the immediate postoperative period if the patient undergoes surgery.
10. Treat the BP that is substantially lower than expected (e.g. SBP < 90 mm Hg) for a given patient, considering past history of hypertension (treated or untreated). Therapeutic options include IV isotonic saline, treatment of congestive heart failure and bradycardia, and consideration of pressor agents infusion such as:
    - — Phenylephrine 2 to 10 μg/kg/min
    - — Dopamine 2 to 20 μg/kg/min
    - — Norepinephrine 0.05 to 0.2 μg/kg/min.

Evidence

1. Up to a third of patients with ICH, particularly those who present within 24 h of onset, have subsequent enlargement of the hematoma.
2. Although it is unclear whether severe hypertension is a cause or an effect of hematoma expansion, the main rationale for controlling BP is to avoid hemorrhagic expansion from potential sites of bleeding.
3. It is unclear if perihematomal penumbra is actually due to ischemia. Functional and biochemical studies show that the penumbra around ICH has reduced $CMRO_2$ and oxygen extraction fraction, mitochondrial dysfunction, and biochemical features more akin to traumatic brain injury than the ischemic penumbra around a cerebral infarct.
4. While severe hypotension may be detrimental, modest BP reduction in ICH may be safe. Autoregulation of CBF in small- to medium-sized acute ICH is preserved despite reduction of mean arterial blood pressure by 16.7 (±5.4)% from 143 (±10) mm Hg to 119 (±11) mm Hg.

Selected Readings

1. Broderick JP, Conolly S, Feldman E, *et al*. (2007) Guidelines for the management of spontaneous intracerebral hemorrhage in adults: 2007 Update: A guideline from the American Heart Association/American Stroke Association Stroke Council, High Blood Pressure Research Council, and the Quality of Care and Outcomes in Research Interdisciplinary Working Group. *Stroke* **38**: 2001–2023.
2. Fujii Y, Takeuchi S, Sasaki O, *et al*. (1998) Multivariate analysis of predictors of hematoma enlargement in spontaneous intracerebral hemorrhage. *Stroke* **29**: 1160–1166.
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4. Morgenstern LB, Yonas H. (2001) Lowering blood pressure in acute intracerebral hemorrhage. Safe but will it help? *Neurology* **57**: 5–6.
5. Powers WJ, Zazulia AR, Videen TO, *et al*. (2001) Autoregulation of cerebral blood flow surrounding acute (6 to 22 h) intracerebral hemorrhage. *Neurology* **57**: 18–24.

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ACUTE INTRACEREBRAL HEMORRHAGE: SURGERY

Dr. Annabell Chua

Goal

To reduce mortality and/or improve functional outcome among patients presenting with acute spontaneous ICH.

Patient Data Needed

1. Neurological status of the patient
   - Serial GCS (*See section on Stroke Scales and Classifications*)
   - Signs of neurological deterioration from baseline (*See section on Worsening after Stroke*)
2. Significant co-morbid medical conditions, e.g. cardiac disease, pulmonary disease, etc.
3. Pre-stroke functional status
4. Brain CT scan findings
   - Location of ICH ☐ Supratentorial (basal ganglia, thalamus, lobar)
     ☐ Infratentorial (cerebellum, brainstem)
   - Volume of hematoma (*See Insert 1 in this section*): _____ mL
   - Cerebral edema and/or mass effect ☐ Yes ☐ No
   - Intraventricular extension of hemorrhage ☐ Yes ☐ No
   - Hydrocephalus ☐ Yes ☐ No

Actions

1. Assess GCS upon admission and monitor as often as necessary.
2. Review the brain CT scan.
3. Refer to a neurosurgeon if *any* of the following:
   a. GCS < 13
   b. Clinical evidence of neurological deterioration
   c. Brain CT scan shows
      i. For supratentorial ICH
         - hematoma volume > 30 mL
         - intraventricular extension of hemorrhage
         - hydrocephalus
         - midline shift.
      ii. For infratentorial ICH
         - cerebellar hematoma > 3 cm in diameter
         - brainstem compression
         - obstructive hydrocephalus.

Evidence

1. There is insufficient evidence to favor surgery over initial medical management for patients with supratentorial ICH.
2. Surgery is the treatment of choice for spontaneous cerebellar hemorrhages > 3 cm in diameter with brainstem compression and/or obstructive hydrocephalus.
3. Patients with superficial spontaneous intracerebral hemorrhages (1 cm or less from cortical surface) may benefit from surgery.
4. Surgical options in the management of spontaneous intracerebral hemorrhage include craniotomy, stereotactic clot lysis and/or aspiration, endoscopic clot lysis and/or aspiration, ventriculostomy, decompressive craniectomy or a combination of these procedures.

Suggested Readings

1. Mendelow A, Gregson B, Fernandes H, *et al*. (2005) Early surgery versus initial conservative treatment in patients with spontaneous supratentorial intracerebral haematomas in the International Surgical Trial in Intracerebral Haemorrhage (STICH): A randomized trial. *Lancet* **365**: 387–397.
2. Fewel M, Thompson G, Hoff J. (2003) Spontaneous Intracerebral Hemorrhage: A Review. *Neurosurg Focus* **15**(4).
3. Psuratian N, Kaselle N, Dumont A. (2003) Update on Management of Intracerebral Hemorrhage. *Neurosurg Focus* **15**(4).
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6. Hseeh PC, Awad IA. Getch CC, *et al*. (2006) Current updates on perioperative management of intracerebral hemorrhage. *Neurol Clin* **24**: 745–764.

ACUTE INTRACEREBRAL HEMORRHAGE: SURGERY

Insert 1. ABC Method of Estimating ICH Volume on CT Scan

$$\text{Hematoma volume (in mL)} = \frac{A \times B \times C}{2}$$

where:

A = Largest diameter of hematoma (in cm)
B = Diameter perpendicular to A (in cm)
C = Number of slices on CT scan × slice thickness (in cm)

For C:

Count slice as 1 if size of hematoma is > 75% of the area seen on the slice with the largest hematoma
Count slice as 0.5 if size of hematoma is 25–75% of the area seen on the slice with the largest hematoma
Disregard slice if size of hematoma is < 25% of the area seen on the slice with the largest hematoma

Reference

Kothari RU, Brott T, Broderick JP, *et al.* (1996) The ABCs of measuring intracerebral hemorrhage volumes. *Stroke* **27**: 1304–1305.

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ACUTE SUBARACHNOID HEMORRHAGE: DIAGNOSTIC TESTS

Dr. Kathleen Joy Khu and Dr. Annabell Chua

Goal

To document the presence of spontaneous subarachnoid hemorrhage and determine its underlying cause.

Patient Data Needed

1. Clinical diagnosis and features of subarachnoid hemorrhage
   - Presenting symptom of sudden severe headache, usually characterized as the worst headache ever experienced
   - May or may not be associated with:
     - ☐ loss of consciousness
     - ☐ vomiting
     - ☐ decreased sensorium
     - ☐ focal neurological deficits
     - ☐ nuchal rigidity or "catch"
2. No definite history of trauma before onset of symptom(s)
3. History of:
   - ☐ intake of anticoagulant or antiplatelet medications
   - ☐ blood dyscrasias
   - ☐ connective tissue disease
   - ☐ smoking
   - ☐ hypertension
   - ☐ arteriosclerosis
4. Allergy to intravenous contrast agent ☐ Yes ☐ No

Actions

1. Check the capillary blood sugar level and oxygen saturation, if not yet done by paramedics or in the emergency room. (*See section on Prehospital Stroke Evaluation and Triage*)
2. Order the following first line hematological and biochemical tests
   - Full (complete) blood count
   - PT/INR
   - aPTT
   - Serum creatinine, urea and electrolytes
   - Blood type and screen.
3. Perform a non-contrast CT scan of the brain as soon as possible, if not yet done (*See section on Brain Imaging*). If the CT scan shows no evidence of SAH, but
   a. the clinical history and feature are strongly suggestive of SAH, perform lumbar puncture (LP) for CSF analysis to determine the presence of significant amount of red blood cells and xanthochromia.
   b. the clinical history and neurologic examination are equivocal, less invasive procedure like CTA or MRA may be performed to detect intracranial aneurysm.
4. Perform an ECG, especially in older patients or if concomitant cardiac signs and symptoms are present.

5. Perform a chest X-ray, especially if cardiac or pulmonary disease is suspected.
6. Arrange for a 4–vessel angiography once a SAH is confirmed. If the patient cannot tolerate procedure, a CTA or MRA may be performed instead. If the patient has allergy to contrast agent or frank renal failure, perform a MRA.
7. Perform the following, if necessary and concomitant conditions suspected:
   - Cervical spine X-ray — if the patient is unresponsive and trauma is suspected
   - Arterial blood gas — if hypoxia or hypercarbia is suspected
   - Blood culture — if patient febrile or infective endocarditis is suspected.
   - Erythrocyte sedimentation rate and C reactive protein — if connective tissue disease, vasculitis, or infective endocarditis is suspected
   - Lumbar puncture — if infectious cause or vasculitis is suspected
   - Toxicology screen — if drug abuse is suspected
   - Pregnancy test in women — if suspected or uncertain
   - Liver function test — if liver disease or blood dyscrasia is suspected
   - Fasting lipid profile, glucose — if the patient has dyslipidemia or diabetes mellitus.
8. Perform serial transcranial Doppler studies, if available, to evaluate for presence of vasospasm and to monitor course.

Evidence

1. For detecting SAH, sensitivity of CT scan is 98% if done within 12 h of ictus, and 93% if done within 24 h. Overall, CT scan is positive in 92% of patients with SAH. LP is most sensitive within 12 h from onset of symptoms. LP is negative in 10–15% of patients with SAH. Negative CT and LP findings indicate a favorable prognosis.
2. 4–vessel angiography remains the gold standard for detecting vascular pathologies or etiologies of spontaneous SAH, e.g. aneurysms and vascular malformations. Alternatives include CTA and MRA.
3. CTA demonstrates aneurysms as small as 2–3 mm, whereas MRA detects those with a diameter of at least 3–5mm.
4. CTA is 90.5% sensitive and 93.3% specific in detecting the presence of an aneurysm, while MRA is 86.3% sensitive and 96.9% specific.

Selected Readings

1. Vermeulen M, van Gijn N. (1990) The diagnosis of subarachnoid hemorrhage. *J Neurol Neurosurg Psychiatry* **53**: 365–372.
2. Bederson JB, Awad IA, Wiebers DO, *et al.* (2000) Recommendations for the management of patients with unruptured intracranial aneurysms: A statement for healthcare professionals from the Stroke Council of the American Heart Association. *Circulation* **102**: 2300–2308.
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4. Edlow JA, Caplan LR. (2000) Avoiding pitfalls in the diagnosis of subarachnoid hemorrhage. *N Engl J Med* **342**: 29–36.
5. Aaslid R, Huber P, Nornes H. (1984) Evaluation of cerebrovascular spasm with transcranial Doppler ultrasound. *J Neurosurg* **60**: 37–41.

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ACUTE SUBARACHNOID HEMORRHAGE: MEDICAL MANAGEMENT

Dr. Kathleen Joy Khu and Dr. Annabell Chua

Goal

To reduce morbidity and mortality among patients presenting with spontaneous SAH by prevention, recognition, and treatment of potential complications while awaiting definitive intervention.

Patient Data Needed

1. Neurological status of the patient (*See section on Stroke Scales and Classifications*)
   - Hunt and Hess or World Federation of Neurological Surgeons (WFNS) grading
   - Serial GCS
2. Significant co-morbid medical conditions, e.g. cardiac or pulmonary disease, etc.
3. BP: _____ / _____ mm Hg
4. MAP = $\frac{\text{SBP} + (2 \times \text{DBP})}{3}$ = _____ mm Hg
5. ICP, if available: _____ mm Hg (1 mm H_2O = 0.07 mm Hg)
6. History of antiplatelet or anticoagulation intake ☐ Yes ☐ No

Actions

1. Stop antiplatelet or anticoagulation treatment if given. Reverse the antithrombotic effect and correct coagulopathy if necessary.
2. Assess clinical severity of SAH (GCS, Hunt and Hess or WFNS grading) and refer to a neurosurgeon. (*See section on Subarachnoid Hemorrhage: Surgery and Intervention*)
3. Reduce risk of rebleeding by the following measures:
   a. Complete bed rest with no bathroom privileges.
   b. Adequate analgesia, mild sedation, stool softeners.
   c. Maintain MAP < 110 mm Hg. (*See Insert 1 in this section*)
   d. Start prophylactic anticonvulsant.
4. If the BP is uptrend and the heart rate is downtrend, suspect an increased ICP and institute measures to control the ICP. (*See section on Increased Intracranial Pressure*)
5. Institute measures to prevent vasospasm and/or infarction.
   a. Do serial TCD and/or CT scans as needed.
   b. Maintain normovolemia, normothermia, and normal oxygenation.
   c. Start nimodipine 60 mg every 4 h, orally or by nasogastric tube, to be given for 21 days. Adjust the dose if hypotension occurs.
6. Suspect symptomatic vasospasm if neurological deterioration is noted with rising intracranial arterial flow velocity (MCA PSV > 200 cm/sec) on TCD. Treat with the following only when the aneurysm has been secured or obliterated.
   a. Transfer the patient to the ICU with adequate team support.
   b. Insert Swan Ganz catheter and arterial line.

c. Start the Triple H (hypertension, hypervolemia, hemodilution) therapy.
   — Maintain the CVP at 10–12 mm Hg, the PCWP at 12–16 mm Hg, and the hematocrit at 30–35%. Intravenous albumin and fluid may be used to achieve targets.
   — Maintain the SBP at 160 to < 200 mm Hg with target CPP > 75 mm Hg. May use vasopressors to maintain desired blood pressure:

Phenylephrine	2 to 10 μg/kg/min
Dopamine	2 to 20 μg/kg/min
Norepinephrine	0.05 to 0.2 μg/kg/min.

d. Regularly monitor the neurological and cardiopulmonary status, electrolytes, osmolality, and the status of vasospasm (by daily TCD).
e. If the Triple H therapy fails to reverse the neurologic deficit from vasospasm, consider endovascular techniques such as angioplasty or intra-arterial papaverine.

Evidence

1. While non-randomized studies have shown only a non-significant trend towards benefit of prophylactic anticonvulsants in SAH, seizures occur in 10–25% of patients after SAH with potential risk of rebleeding if it occurs. Administration of anticonvulsant is, therefore, recommended in the immediate posthemorrhage period.
2. There is a higher rate of rebleeding in patients not receiving antihypertensive therapy, despite lower BP, as compared to those who receive treatment.
3. The use of nimodipine, a calcium channel blocker, reduces poor outcome when given for 21 days from the ictus in all grades of patients with spontaneous SAH.
4. Triple H (hypervolemia, hypertension and hemodilution) or hyperdynamic therapy may reverse neurological deficits from vasospasm before actual infarction occurs and improve outcome compared to historical controls.
5. The role of statins to prevent delayed cerebral ischemia is still unclear. Data from early phase studies suggest that they ameliorate vasospasm and improve outcome.
6. Routine use of antifibrinolytics in aneurysmal SAH is not recommended. While they reduce risk of rebleeding, they increase ischemic complications, with no net benefit.

Suggested Readings

1. Suarez JL, Tarr RW, Selman WR. (2006) Aneurysmal subarachnoid hemorrhage. *N Engl J Med* **354**: 387–396.
2. Dorhout Mees SM, Rinkel GJE, Feigin VL, *et al*. (2007) Calcium antagonists for aneurysmal subarachnoid haemorrhage. *Cochrane Database of Systematic Reviews*, Issue 3. Art. No.: CD000277. DOI: 10.1002/14651858.CD000277.pub3.
3. Roos YBWEM, Rinkel GJE, Vermeulen M, *et al*. (2003) Antifibrinolytic therapy for aneurysmal subarachnoid haemorrhage. *Cochrane Database of Systematic Reviews*, Issue 2. Art. No.: CD001245. DOI: 10.1002/14651858.CD001245.
4. Wijdicks EF, Vermeulen M, Murray GD, *et al*. (1990) The effects of treating hypertension following aneurysmal subarachnoid hemorrhage. *Clin Neurol Neurosurg* **92**: 111–117.
5. Awad IA, Carter LP, Spetzler RF, *et al.* (1987): Clinical vasospasm after subarachnoid hemorrhage: Response to hypervolemic hemodilution and arterial hypertension. *Stroke* **18**: 365–372.

SUBARACHNOID HEMORRHAGE: MEDICAL MANAGEMENT

Insert 1. Blood Pressure Management in Acute Spontaneous SAH prior to Surgical or Endovascular Intervention of Aneurysm

- Maintain a MAP of < 110 mm Hg or a BP of < 160/90 mm Hg.
- Consider inserting an ICP monitor if the GCS is ≤ 8 or in the presence of clinical signs and symptoms of increased intracranial pressure. Keep the cerebral perfusion pressure (CPP = MAP – ICP) > 60 mm Hg.
- Use any of the following for elevated BP management:

Drug	Intravenous Bolus	Infusion
Labetalol	5–20 mg every 15 min	2–8 mg/min (max 300 mg/d)
Nicardipine	–	5–15 mg/h
Esmolol	250 µg/kg loading dose	25–300 µk/kg/min
Enalapril	0.625–5 mg every 6 h	–
Hydralazine	5–20 mg every 30 min	1.5–5 µg/kg/min
Sodium Nitroprusside	–	0.1–10 µg/kg/min
Nitroglycerin	–	20–400 µg/min

- Consider other medical conditions when selecting an antihypertensive agent, e.g. avoid beta-blocker in patients with history of asthma.
- Avoid drugs that may precipitously drop the BP and have prolonged effects, such as sublingual calcium channel antagonists.
- Monitor the BP and neurological status every 15 min during antihypertensive therapy. Observe for hypotension.

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ACUTE SUBARACHNOID HEMORRHAGE: SURGERY AND INTERVENTION

Dr. Shree Kumar Dinesh and Dr Ivan Ng

Goal

To reduce mortality and morbidity among patients presenting with acute SAH by prevention and treatment of complications and by obliteration of underlying vascular anomaly.

Patient Data Needed

1. Neurological status of patient (*See section on Stroke Scales and Classifications*)
   - Hunt and Hess or World Federation of Neurological Surgeons (WFNS) grading
   - Serial GCS
2. Significant co-morbid medical conditions, e.g. cardiac disease, pulmonary disease, etc.
3. Pre-stroke functional status
4. Brain CT scan findings
   - Location of hematoma, if present (may indicate location of aneurysm or vascular malformation)
   - Location of SAH ☐ Supratentorial ☐ Infratentorial
   - Cerebral edema and/or mass effect ☐ Yes ☐ No
   - Intraventricular extension of hemorrhage ☐ Yes ☐ No
   - Hydrocephalus ☐ Yes ☐ No
5. ICP, if available: _____ mm Hg (1 mm H_2O = 0.07 mm Hg)

Actions

1. Consider admitting the patient to the ICU or stroke unit for close monitoring. Assess GCS on admission and perform serial GCS assessments.
2. Determine Hunt and Hess or WFNS grading and refer to neurosurgeon.
3. Arrange for 4–vessel catheter angiogram or CT angiography.
4. If there is neurological deterioration from baseline, repeat brain CT scan to rule out rebleed, acute or worsening hydrocephalus, acute or worsening cerebral edema, or infarction from vasospasm. (*See also sections on SAH Medical Management and Worsening after Stroke*)
5. If acute hydrocephalus is present on the brain CT scan:
   a. An external ventricular drain should be inserted.
   b. Keep the ICP between 15–25 mm Hg (200–350 mm H_2O) to avoid risk of rebleeding due to relative increase in transmural pressure across the aneurysm wall after ventriculostomy.
   c. Regularly assess the need for permanent shunt.
6. Depending on the co-morbid conditions, pre-stroke functional status, and size, location, and the configuration of the aneurysm or vascular malformation, choose the most suitable method of securing the aneurysm or AVM, i.e. craniotomy and clipping/obliteration versus

endovascular intervention versus conservative management. Radiosurgery is also an option for vascular malformation.

7. Once the aneurysm is secured, the SBP may be allowed up to 200 mm Hg. Monitor the patient for up to 2 weeks for evidence of vasospasm. Suspect vasospasm if neurological deterioration is noted with rising intracranial arterial flow velocity on transcranial Doppler, and:
   - Start triple H therapy. (*See section on SAH Medical Management*)
   - Consider endovascular techniques such as angioplasty or intra-arterial papaverine if triple H fails to reverse neurological deficit from vasospasm.

Evidence

1. Hydrocephalus (HCP) is seen on initial CT in 15–20% of SAH cases. Patients with poor grade SAH (WFNS 4–5) with HCP may be symptomatic of it and should undergo a ventriculostomy. Permanent shunt will be required by 20% of all patients with ruptured aneurysm and by up to 50% of those with acute HCP following SAH.
2. Risk of vasospasm is associated with increasing subarachnoid blood seen on CT.
3. Mortality from rebleeding after a SAH is > 70%. Risk of rebleeding is 4.1% within the first day, 1.5% per day from day 2 to 14 (cumulative 19%), and 50% in 6 months. Thereafter, the risk is 3% per year. Risk increases with worse Hunt and Hess grade.
4. For ruptured aneurysm deemed suitable for both endovascular and surgical treatment, coiling is associated with better outcome and this option should be offered. The benefit of early survival in the endovascular group is maintained for up to 7 years. Overall rates of late rebleeding for both groups are low but more common in the endovascular group. The risk of seizure is higher in the surgical group.

Suggested Readings

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5. International Subarachnoid Hemorrhage Aneurysm Trial (ISAT) Collaborative Group. (2005) International Subarachnoid Hemorrhage aneurysm trial of neurosurgical clipping versus endovascular coiling in 2143 patients with ruptured intracranial aneurysms. *Lancet* **366**: 809–817.
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7. van der Schaaf I, Algra A, Wermer M, *et al*. (2005) Endovascular coiling versus neurosurgical clipping for patients with aneurysmal subarachnoid haemorrhage. *Cochrane Database of Systematic Reviews*, Issue 4. Art. No.: CD003085. DOI: 10.1002/14651858.CD003085.pub2.

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BLOOD SUGAR IN ACUTE STROKE

Dr. Rajinder Singh

Goal

To maintain strict euglycemia so as to optimize favourable stroke outcomes.

Patient Data Needed

1. History of pre-existing diabetes mellitus and HbA1c if available
2. If the patient is diabetic, note current medications for diabetes mellitus
3. BSL on admission = _____ mmol/L or _____ mg/dl
4. Stroke subtype and severity (NIHSS score)

Actions

1. Preferably use only normal saline (0.9% NaCl) infusions as maintenance fluids — avoid use of dextrose infusions unless the patient is hypoglycemic.
2. Aim to maintain the BSL between 4 to 7 mmol/L (70–140 mg/dl).
3. In patients with mild strokes and BSL on admission < 20 mmol/L (< 400 mg/dl), SC soluble insulin (SI) may be used according to a sliding scale with 6 hourly capillary blood glucose monitoring. (*See Insert 1 in this section*).
4. Start 1500 Cal DM diet for diabetic patients with no dysphagia.
5. Use the Glucerna enteral formula for diabetic patients on nasogastric tube feeding.
6. For patients with large strokes (MCA or brainstem), consider strict blood sugar control by IV SI infusion, with hourly capillary blood glucose monitoring preferably in a Stroke Unit or ICU. (*See Insert 2 in this section — Note: Infusion protocols vary among institutions*).
   May convert to SC SI sliding scale once patient has stabilized and started on enteral feeds.
7. Whenever BSL > 20 mmol/L (> 400 mg/dl), treat as for diabetic emergency.

Evidence

1. Approximately one-third of patients with stroke have hyperglycemia detected upon admission.
2. A markedly elevated blood glucose concentration forecasts a poor outcome after acute ischemic stroke.
3. Persistent hyperglycemia during the first 24 h predicts expansion of stroke and poor outcomes.
4. Hyperglycemia may be a manifestation of underlying diabetes or it may be a secondary stress reaction.
5. Very strict blood glucose control between 4.4 to 6.1 mmol/L has been shown to dramatically reduce the acute mortality in patients receiving intensive care

by 42%.

Selected Readings

1. Baird TA, Parsons MW, Barber PA, *et al*. (2002) The influence of diabetes mellitus and hyperglycemia on stroke incidence and outcome. *J Clin Neurosci* **9**: 618–626.
2. Bruno A, Levine SR, Frankel MR, *et al* and NINDS r-tPA Stroke Study Group. (2002) Admission glucose levels and clinical outcomes in the NINDS r-tPA stroke trial. *Neurology* **59**: 669–674.
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7. Gray CS, Hildreth AJ, Sandercock PA, *et al* for the GIST Trialists Collaboration. (2007) Glucose-potassium-insulin infusions in the management of post-stroke hyperglycaemia: The UK Glucose Insulin in Stroke Trial (GIST-UK). *Lancet Neurol* **6**: 397–406.

BLOOD SUGAR IN ACUTE STROKE

Insert 1. Sample Regular Insulin Sliding Scale

Blood Sugar Level (BSL)		Low Insulin Resistance	Intermediate Insulin Resistance	High Insulin Resistance
mmol/L	mg/dL			
≤ 4	≤ 70	Follow hypoglycemia protocol (see below) and call doctor		
4.1–7	71–140	0 units	0 units	0 units
7.1–10	141–180	0 units	2 units	4 units
10.1–13	181–240	2 units	4 units	6 units
13.1–16	241–300	4 units	6 units	8 units
16.1–19	301–340	6 units	8 units	10 units
19.1–22	341–400	8 units	10 units	12 units
> 22	> 400	10 units & call doctor	12 units & call doctor	14 units & call doctor

- Omit bedtime regular insulin dose due to risk of overnight hypoglycemia. Bedtime insulin may be considered for patients who are eating or on bolus tube feed. If regular insulin is given at bedtime, administer only half of indicated dose and recheck BSL at 0300.
- May need to decrease doses in patients with renal failure because insulin may not be as rapidly cleared.
- May need to increase doses for patients who are septic or treated with steroids because of relative insulin resistance.
- Mild hyperglycemia is better than hypoglycemia.
- Do not discharge patients on insulin sliding scale. Determine appropriate outpatient regimen before discharge.
- For BSL ≤ 4 mmol/L (≤ 70 mg/dl), follow Hypoglycemia Protocol:

  A. If able to take orally or has NGT:

  1. Give 15–20 grams of fast acting carbohydrate, e.g.
     a. ½ cup fruit juice
     b. ¾ cup regular soda
     c. 3 or 4 tablets of glucose
     d. 1 cup low fat milk
     e. Crackers
     f. 3 tsp honey or corn syrup
  2. Check BSL every 15 min until > 4 mmol/L (> 70 mg/dl).
  3. Consider increasing dietary carbohydrates.

B. If unable to take orally:
   1. Give 25 ml of D_{50} IV push.
   2. Check BSL every 15 min until > 4 mmol/L (> 70 mg/dl).
   3. Consider changing IV fluid to D_5-containing.

BLOOD SUGAR IN ACUTE STROKE

*Insert 2. Sample Insulin Infusion Protocol**

— Treatment setting (e.g. Stroke Unit, ICU) must be equipped to monitor patients closely for hypoglycemia.

1. Discontinue all previous orders for insulin and oral hypoglycemics.
2. Start dextrose-containing fluid (e.g. D_5NS) at a maintenance rate (50–100 ml/h). Use $D_{10}NS$ or similar IV fluid solution for fluid-restricted patients. Consider adding potassium 20 mEq/L.
3. Prepare standard insulin solution of 25 units regular insulin in 250 ml NS (1 unit/10 cc). Flush the first 50 cc through the tubing before connecting to the patient.
4. Check blood sugar level (BSL) before starting the insulin infusion.
5. Start the insulin infusion rate as follows when BSL ≥ 100 mg/dl (> 5.5 mmol/L).
   - 1.0 unit/h for patients who were previously diet-controlled, on oral hypoglycemics only, or who take < 30 units of insulin/day at home.
   - 1.5 units/h for patients taking ≥ 30 units of insulin/day at home.
6. Monitor blood sugar level (BSL) by glucometer
   - Check BSL every hour until stable (range 100–200 mg/dl or 5.5–11 mmol/L) for two consecutive readings. Then check BSL every 2 h.
   - If the BSL has changed by > 100 mg/dl (> 5.5 mmol/L) from previous reading, recheck BSL prior to adjusting insulin dose to verify accuracy.
   - Resume hourly checking if BSL > 200 mg/dl (> 11 mmol/L).
7. Adjusting the infusion rate according to BSL:

< 80 mg/dl (< 4.5 mmol/L)	Stop infusion and Call House Officer
**Do not restart insulin infusion until BSL > 100 mg/dl (> 5.5 mmol/L)*	
80–120 mg/dl (4.5–6.5 mmol/L)	Decrease drip by 0.5 unit/h
121–180 mg/dl (6.6–10 mmol/L)	No change in drip rate
181–250 mg/dl (10.1–13.5 mmol/L)	Increase drip by 0.5 unit/h
> 250 mg/dl (> 13.5 mmol/L)	Bolus 5 units regular insulin and increase drip by 0.5 unit/h.

8. Call doctor for BSL < 80 mg/dl (< 4.5 mmol/L) or > 400 mg/dl (> 22 mmol/L):
   - If BSL is 60–80 mg/dl (3.5–4.5 mmol/L), stop the infusion and check BSL every 15 min.
   - If BSL < 60 mg/dl (< 3.5 mmol/L), stop insulin infusion and give 50 ml D_{50} IV push; check BSL every 15 min and repeat D_{50} until BSL > 100 mg/dl (> 5.5 mmol/L). The doctor should reassess and consider reducing the insulin infusion rate.
   - If BSL > 400 mg/dl, the doctor should reassess and consider increasing the insulin infusion rate.
9. Adjust the infusion rate if there is any change in parenteral or enteral feedings (started or stopped).

10. When converting to SC insulin, give dose 30 min prior to discontinuing insulin infusion.

**Based on the UCSF Adult Insulin Infusion Orders, 2001. http://www.mdtext.com/diabetes/diabetes22/ figures22/pdfdoc1.pdf*

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FLUID AND NUTRITION IN ACUTE STROKE

Dr. Carlos Chua and Dr. Maria Cristina Z. San Jose

Goals

To maintain vital bodily functions and fluid-electrolyte balance for optimal hemodynamic stability and cerebral perfusion, and to provide adequate early nutritional support for prevention of complications and maximization of recovery.

Patient Data Needed

1. Weight: _____ kg
2. Temperature: _____ °C
3. Signs of dehydration and malnutrition

Actions

1. If IV fluid is required to maintain a balanced fluid status, use isotonic saline (0.9% NaCl).
   - — Correct dehydration promptly, if present.
   - — Hospitalized patients usually require 30–35 ml/kg/day. If with fever, add 250 ml per day for each °C above 37°C.
   - — Avoid glucose-containing or hypotonic solutions unless medically necessary.
   - — Once able, shift to oral or enteral water supplementation.
2. Assess the patient's nutritional status at baseline and periodically thereafter.
   - — Check for physical signs of malnutrition e.g. generalized muscle wasting, angular stomatitis, cheilosis, raw swollen tongue, easily plucked thin dyspigmented hair.
   - — Nutritional supplements may be used if the patient is judged to be undernourished on admission or have deteriorating nutritional status in the hospital.
3. Compute the patient's energy, protein, and electrolyte requirements. Most patients will require a total of 30 kcals of energy and 0.8–1 g of protein per kg body weight per day. Refer to a dietician for more defined estimates of nutritional requirements.
4. Assess for any difficulty in swallowing using the dysphagia screening test and perform bedside WST. (*See Insert 1 in this section*)
5. If the patient has signs of dysphagia, has altered sensorium, failed WST, or is unable to consume sufficient quantities of fluid or food orally, insert a NGT for feeding and administration of medications.
   - — Choose type of feeding formula by considering formula characteristics (e.g. adequacy of nutrients, osmolality) and patient-specific factors (e.g. concomitant diseases, electrolyte balance). (*See Insert 2 in this section*)
   - — Start tube feeding early within 48–72 h. (*See Insert 3 in this section*)
   - — Perform videoflouroscopic examination or fiberoptic laryngoscopy if indicated.
   - — Consider PEG placement in patients who will require prolonged NGT feeding of more than 2–3 weeks.

6. Consider parenteral nutrition only if the enteral route is not feasible.
7. For patients with moderate to severe stroke, use H2 blockers or proton pump inhibitors to prevent gastrointestinal bleeding and aspiration pneumonitis.
8. Monitor intake and output regularly. Include mannitol (if given) and IV or NGT flushes in the computation of total fluid intake.
9. Monitor regularly for complications of enteral nutrition support such as diarrhea, constipation, mucosal damage, aspiration, and metabolic abnormalities.

Evidence

1. Hypotonic solutions and overhydration may worsen cerebral edema. Hyperglycemia promotes lactic acidosis, increases production of free radicals, worsens cerebral edema, and weakens blood vessels.
2. Malnutrition is a risk factor for complications, mortality, and prolonged hospitalization.
3. Routine oral nutritional supplementation for stroke patients who are well-nourished on admission is not associated with improved outcome.
4. A simple bedside swallowing assessment is a reliable technique to screen patients who are at risk for aspiration and who may need a more thorough evaluation.
5. Early enteral tube feeding within the first few days of admission is associated with reduction in mortality among dysphagic stroke patients compared to those fed only after 7 days. Among patients who cannot take orally, NGT feeding may be the chosen route during the first 2–3 weeks, while PEG is superior to NGT if a prolonged use of tube feeding is needed.
6. H2 blockers and proton pump inhibitors substantially reduce aspiration risk by reducing gastric volume and increasing pH.

Selected Readings

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3. The FOOD Trial Collaboration. (2003) Poor nutritional status on admission predicts poor outcomes after stroke. *Stroke* **34**: 1450–1456.
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FLUID AND NUTRITION IN ACUTE STROKE

Insert 1. Assessment for Dysphagia and Swallowing

Dysphagia Screen

- ☐ Drooling, excessive secretions
- ☐ Coughing or choking while eating
- ☐ Regurgitation through nose, mouth, or tracheostomy tube
- ☐ Pocketing of food in cheek, under tongue, or on hard palate
- ☐ Poor tongue control
- ☐ Facial weakness
- ☐ Significantly slurred speech
- ☐ Wet "gurgly" voice after eating or drinking or frequent throat clearing
- ☐ Hoarse voice
- ☐ Delay or absence of laryngeal (Adams' apple or thyroid cartilage) elevation.

Note: The presence of gag reflex does not guarantee intact swallowing reflex.

*Bedside 3 oz Water Swallowing Test (WST)**

- Ensure that any of the above signs and symptoms is absent.
- Do not perform WST if the patient is drowsy, if stroke is suspected to be in the brainstem, or stroke is bilateral.
- Ask the patient to drink from a cup containing 3 oz (90 mL) of water without interruption.
- Rate as abnormal if coughing occurs during or within 1 min after completion or a wet-hoarse voice quality is observed post-swallowing.

**Alternatively, may use water swallowing test method described in Insert 2 of section on Basic Admitting Orders and Stroke Clinical Pathway.*

Reference

DePeppio Kl, Hollas MA, Redding MJ, *et al*. (1992) Validation of the 3 oz water swallow test for aspiration following stroke. *Arch Neurol* **49**: 1259–1261.

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Insert 2. Guide to Choosing Enteral Formula

Enteral Formula Categories

Category	Subcategory	Characteristics	Indications
Polymeric	Standard	Similar to average diet	Normal digestion
	High nitrogen	Protein > 15% of total kcal	Catabolism Wound Healing
	Caloric dense	1.2 to 2 kcal/ml	Fluid restriction Electrolyte abnormality
	Fiber containing	Fiber 10 to 15 g/L	Regulation of bowel function
Monomeric	Partially hydrolyzed Elemental Peptide-based	One or more nutrients are hydrolyzed. Composition varies	Impaired digestive & absorptive capacity
Disease specific	Renal	Low protein, low electrolyte content	Renal failure
	Hepatic	High branched chain AA; low protein, low electrolyte content	Hepatic encephalopathy
	Pulmonary	Higher % of calories from fat	Acute respiratory distress syndrome
	Diabetic	Low carbohydrate (CHO)	Diabetes mellitus
	Immune enhancing	Arginine, glutamine, omega 3 FA, antioxidants	Immune dysfunction Metabolic stress

Summary of Commercially Available Formulae (from Dietetic and Food Services of Changi General Hospital, compliments of Ms. Ling Ping Sing, *et al.*)

Name	Kcal/ml	Prot g/L	Fat g/L	CHO g/L	Fibre g/L	Na mmol	K mmol	H_2O %	mosm/kgH_2O
				STANDARD					
Osmolite	1.06	37.1	34.7	151.1	0	27.8	26.2	84.1	300
Ensure	1.06	37.2	25.7	169	0	36.7	40	84.4	555
Ensure (powder)	1.06	37.6	37.6	143	0	36.7	40	84.5	470
Ensure LIFE (powder)	1.0	36.9	32.5	135.7	0	36.3	39.9	84.8	350

(*Continued*)

(*Continued*)

Name	Kcal/ml	Prot g/L	Fat g/L	CHO g/L	Fibre g/L	Na mmol	K mmol	H_2O %	mosm/kgH_2O
Isocal	1.06	34	44	135	0	23	34	84.0	300
Isocal (powder)	1.0	32.5	42	125	0	21.7	32.5	84.5	iso-tonic
Jevity	1.06	44.2	34.6	154.4	14.4	40.4	40.1	83.3	300
Jevity Plus	1.2	55.5	39.3	171.5	12.0	58.7	47.4	80.5	450
Resource Standard	1.06	38	25	170	0	40.4	37.9	84	430
DIABETIC									
Glucerna	1.0	42	54.4	81.9	14.3	40.4	40	85.3	355
GlucernaSR (powder)	0.93	46.5	33.8	122.9	11.8	38.7	40	71.2	NA
Nutren DM (powder)	1.0	38.1	44.2	111.7	15.2	37.8	32.3	71.7	350
Resource Diabetic	1.06	63	47	100	12.8	50.9	45.4	84.7	300
CALORIC DENSE									
Ensure Plus	1.5	62.5	49	202	0	52.2	21.3	76.9	690
Enercal Plus (powder)	1.5	58	50	204	0	47.8	48.1	75.0	500
Pulmocare	1.5	62.4	93.2	105.5	0	56.9	50.3	78.5	475
Resource Plus	1.5	55	46	220	0	57	49.7	76.6	600
Resource 2.0	2.0	84	88.6	219	0	34.9	38.8	70.0	790
RENAL									
Suplena	2.0	30	95.8	255.7	0	33.9	28.7	71.3	600
Nepro	2.0	70	95.8	222.8	0	36.7	27	70	665
ELEMENTAL									
Alitraq	1.0	52.3	15.5	165	0	43.5	30.8	84.6	575
Peptamen (Peptide-based)	1.0	39.8	38.7	123	0	21.7	32	90.0	375
SINGLE NUTRIENT									
Name	**Kcal/scoop**	**Prot g/L**	**Fat g/L**	**CHO g/L**	**Fibre g/L**	**Na mmol**	**K mmol**	**H_2O %**	**mosm/kg H_2O**
Promod (powder)	28	5	0.6	0.67	0	0.65	1.7	0.6	NA
Propass (powder)	30	6	0.5	NA	0	0.65	NA	NA	NA

FLUID AND NUTRITION IN ACUTE STROKE

Insert 3. Guideline to Enteral Formula Tube Feeding

- Preferably, initiate feeding by continuous infusion using a volumetric pump to establish digestive tolerance. Start at 25–50 mL/h with gradual increments of 20–25 mL every 4–12 h until the desired volume is reached. May later shift to intermittent or bolus feeding.
- Intermittent feeding is best tolerated when no more than 250–400 mL is given over 20–30 min using gravity drip (either by syringe or delivery bag) or infusion pump every 3–6 h.
- Bolus feeding can be administered by a syringe bolus (volume of no more than 300–400 mL per feeding given over 10 min or less) every 4–6 h. Watch for nausea, abdominal discomfort, fullness, or cramping.
- Always check for tube patency and placement and residuals prior to each feeding.
  - — If the gastric residual is ≥ 200 mL, readminister the residual and withhold feeding for 1–4 h. Then recheck the residual and if excessive residual persists, reduce the rate of administration or begin drug therapy to stimulate gastric emptying.
  - — If the gastric residual is < 200 mL, readminister the residual and add in formula feeding to make up total of the scheduled desired volume.
- Refer to a dietician for review of the fomula's composition and the administration rate.

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REHABILITATION, PHYSICAL THERAPY, OCCUPATIONAL THERAPY, AND SPEECH THERAPY

Dr. Sherry Young

Goal

To optimize functional recovery of the stroke patient by limiting disability and handicap.

Patient Data Needed

1. Types of impairment and functional deficit the patient has, e.g. hemiparesis leading to unsafe ambulation, neglect leading to problems with dressing and bathing, etc.
2. Barthel Index and mRS scores (*See Inserts 1 and 2 in this section*)

Actions

1. Refer the patient to a PT (physiotherapist or physical therapist) within 24 h for assessment and treatment if the patient has loss of gross motor function, e.g. walking.
2. Refer patient to an OT (occupational therapist) within 24 h for assessment and treatment if the patient has loss of functional skills, e.g. dressing, bathing, driving.
3. Refer the patient to a ST (speech therapist) within 24 h for early assessment of swallowing difficulties and/or problems with communication.
4. Refer the patient to a Rehabilitation Medicine specialist, the specialist who focuses not only on disease management but also on amelioration of disability and handicap, prevention of complications, functional prognostication, and management of young and/or complex stroke cases in a stroke rehabilitation unit.
5. Assess the patient's clinical status, needs for inpatient or community-based rehabilitation, and progress. (*See Inserts 3,4, and 5 in this section*)

Evidence

1. Post-stroke rehabilitation provided by specialized multidisciplinary stroke units enables patients to maintain both short- and long-term functional improvements, when compared with general rehabilitation unit.
2. Early rehabilitation therapy should be provided as soon as the patient's medical status is stable.
3. The use of therapeutic exercises and task-oriented training help with the functional recovery of stroke patients.
4. Faster recovery and earlier discharge from the hospital can result from intense therapies delivered in a short periods of time within the post-stroke rehabilitation process.

Selected Readings

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7. Australian National Stroke Foundation. (2005) *Clinical Guidelines for Stroke Rehabilitation and Recovery*.
8. Bates B, Choi JY, Duncan PW, *et al*. (2005) Veterans Affairs/Department of Defense Clinical Practice Guideline for the Management of Adult Stroke Rehabilitation Care: Executive Summary. *Stroke* **36**: 2049–2056.

REHABILITATION, PHYSICAL THERAPY, OCCUPATIONAL THERAPY, AND SPEECH THERAPY

Insert 1. The Barthel Index (BI)

The BI is a simple index of independence to score the ability of a patient with a neuromuscular or musculoskeletal disorder to care for self. It may be the most widely studied and used disability index globally. There are several modified versions.

It includes 10 items and they are scored differently according to a weighted scoring system that assigns points based on independent or assisted performance.

BI Items and Scoring Weights

Items	With Help	Independent
1. Feeding (If food needs to be cut up = help)	5	10
2. Moving from wheelchair to bed and return (including sitting up in bed)	5–10	15
3. Personal toilet (wash face, comb hair, shave, clean teeth)	0	5
4. Getting on and off toilet, (handling clothes, wipe, flush)	5	10
5. Bathing self	0	5
6. Walking on level surface (or, if unable to walk, propel wheelchair)	10 0	15 5[a]
7. Ascend and descend stairs	5	10
8. Dressing (including tying shoes, fastening fasteners)	5	10
9. Controlling bowels	5	10
10. Controlling bladder	5	10

[a]Score only if unable to walk

A person scoring 100 is continent, feeds himself, dresses himself, gets up out of bed and chair, bathes himself, walks at least a block, and can ascend and descend stairs. This does not mean that he is able to live alone; he may not be able to cook, keep house and meet the public, but he is able to get along without attendant care.

Reference

Mahoney FI, Barthel DW. (1965) Functional evaluation: The Barthel Index. *Maryland State Med* **14**: 61–65.

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Insert 2. Modified Rankin Scale (*mRS*)

mRS measures independence rather than performance of specific tasks and incorporates physical as well as mental adaptation to neurological deficits. The scale gives an impression of whether the patients can look after themselves and represents handicap rather than disability. The mRS not only allows comparison between patients with different neurological deficits but also relates to the person's previous activities.

Grade	Description
0	No symptoms at all
1	No significant disability despite symptoms; able to carry out all duties and activities
2	Slight disability; unable to carry out all previous activities, but able to look after own affairs without assistance
3	Moderate disability; requiring some help, but able to walk without assistance
4	Moderately severe disability; unable to walk without assistance and unable to attend to own bodily needs without assistance
5	Severe disability; bedridden, incontinent and requiring constant nursing care and attention
6	Dead

The original Rankin scale did not include Grade 0.

References

Rankin J. (1957) Cerebral vascular accidents in patients over the age of 60. II. Prognosis. *Scot Med J* **2**: 200–215.

Van Swieten JC, Koudstaal PJ, Visser MC, *et al.* (1988) Interobserver agreement for the assessment of handicap in stroke patients. *Stroke* **19**: 604–607.

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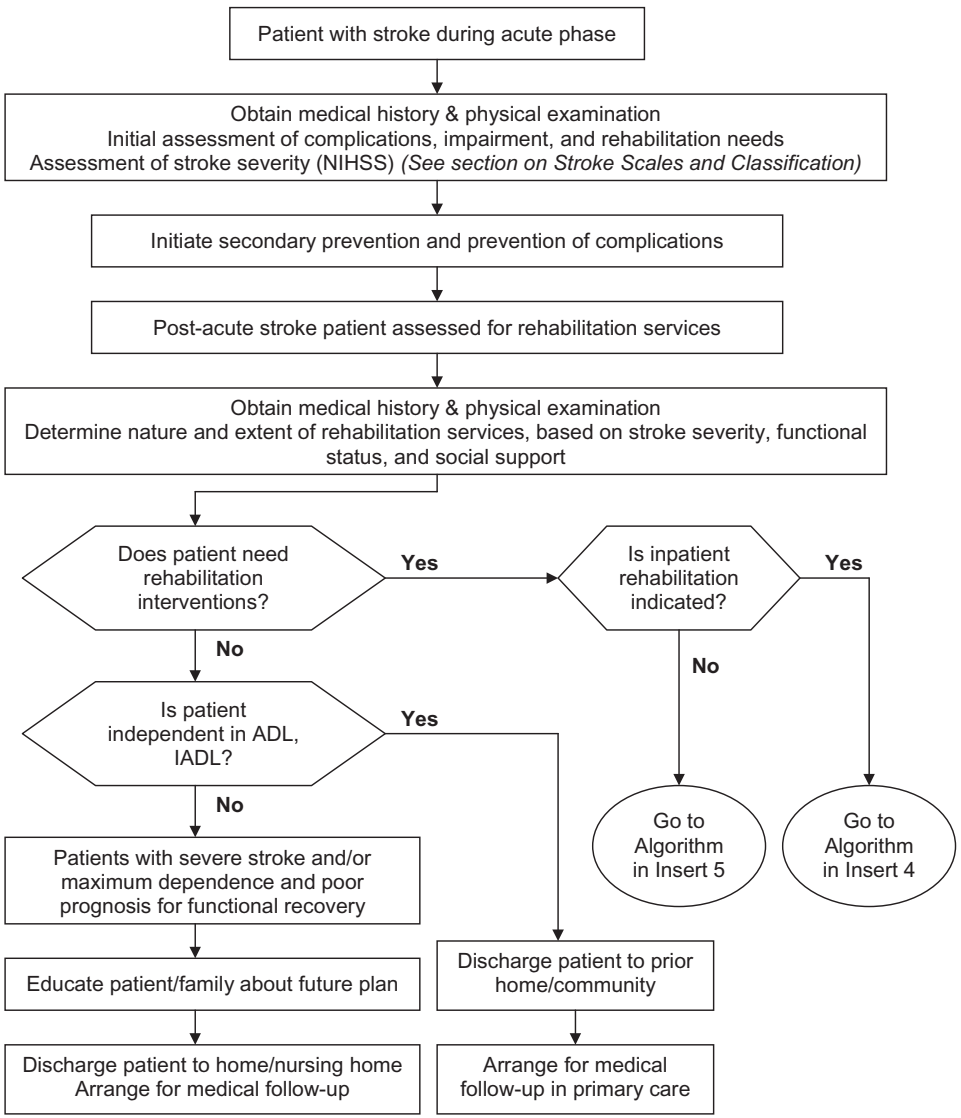
REHABILITATION, PHYSICAL THERAPY, OCCUPATIONAL THERAPY, AND SPEECH THERAPY

Insert 3. Algorithm for Initial Assessment for Rehabilitation of Stroke Patient



Reference

Duncan PW, Zorowitz R, Bates B, *et al*. (2005) Management of adult stroke rehabilitation care: A clinical practice guideline. *Stroke* **36**: 100–143.

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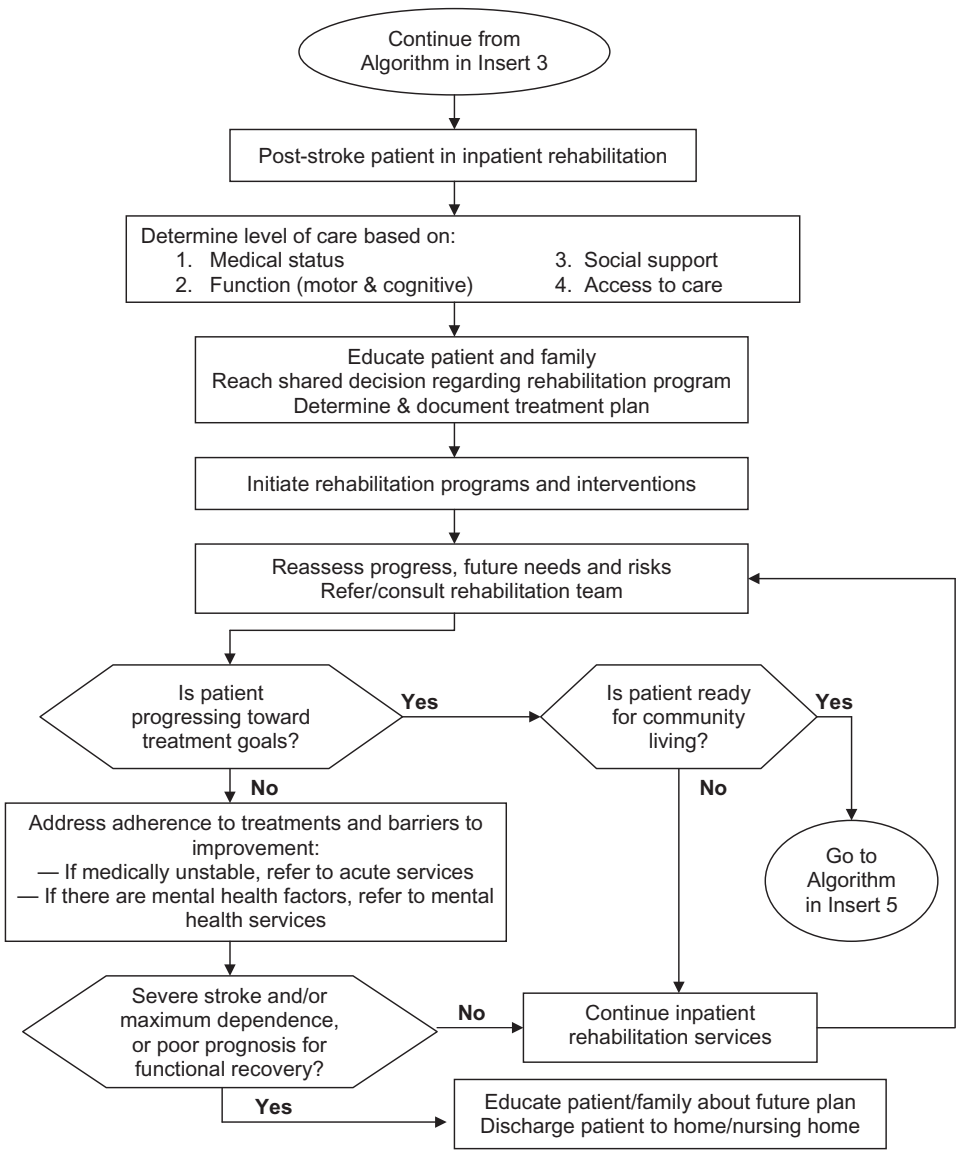
REHABILITATION, PHYSICAL THERAPY, OCCUPATIONAL THERAPY, AND SPEECH THERAPY

Insert 4. Algorithm for Inpatient Rehabilitation of Stroke Patient



Reference

Duncan PW, Zorowitz R, Bates B, *et al*. Management of adult stroke rehabilitation care: A clinical practice guideline. *Stroke* **36**: 100–143.

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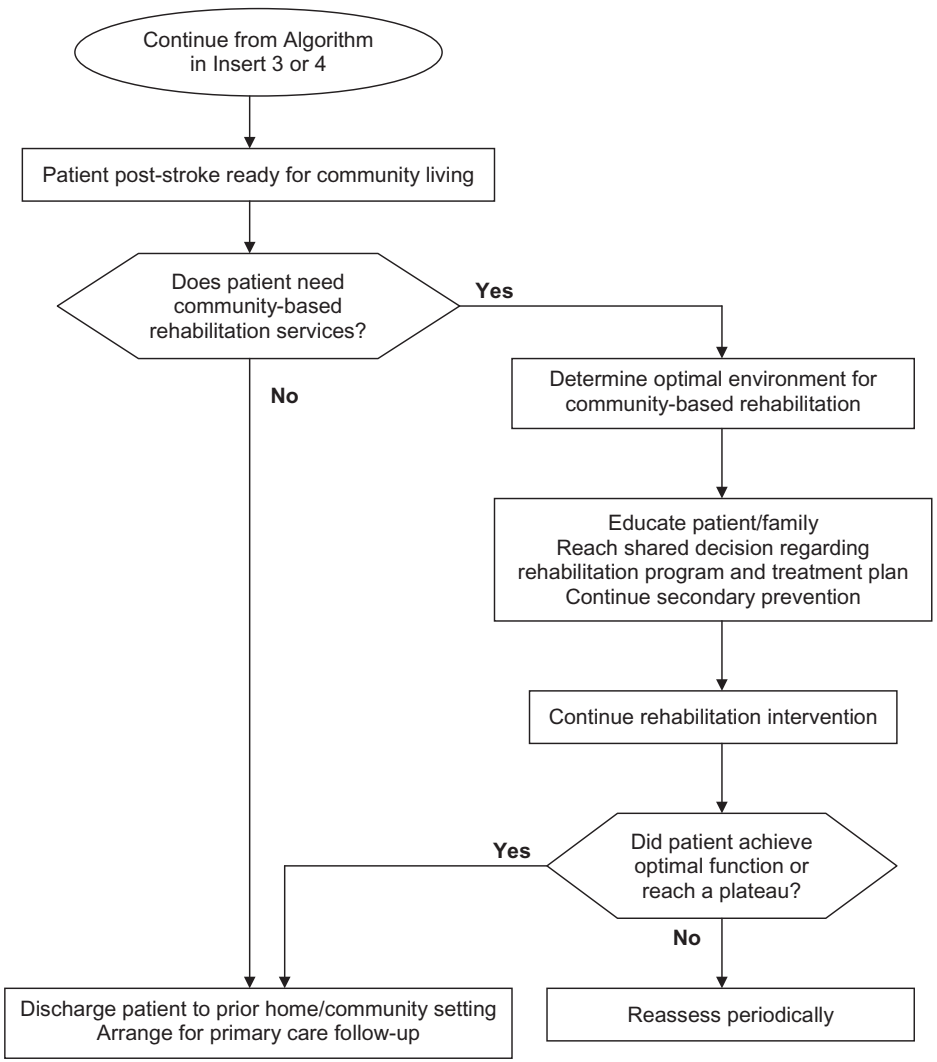
REHABILITATION, PHYSICAL THERAPY, OCCUPATIONAL THERAPY, AND SPEECH THERAPY

Insert 5. Algorithm for Community-Based Rehabilitation of Stroke Patient



Reference

Duncan PW, Zorowitz R, Bates B, *et al*. (2005) Management of adult stroke rehabilitation care: A clinical practice guideline. *Stroke* **36**: 100–143.

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NOTES

WORSENING AFTER STROKE

Dr. Robert N. Gan and Dr. Lee Kim En

Goal

To detect and treat underlying cause of clinical worsening after an acute stroke.

Patient Data Needed

1. GCS and NIHSS (*See section on Stroke Scales and Classifications*)
2. Stroke subtype diagnosis: ☐ Cerebral Infarct ☐ Intracerebral Hemorrhage ☐ Subarachnoid Hemorrhage
3. BP: _____ / _____ mm Hg
4. RR: _____ / min
5. Temperature: _____ °C
6. Oxygen saturation: _____ %
7. Capillary blood sugar: _____ mmol/L or _____ mg/dL
8. Symptoms: ☐ Chest pain ☐ Diaphoresis ☐ Dyspnea/tachypnea
9. Seizures noted ☐ Yes ☐ No
10. Sedation given ☐ Yes ☐ No

Actions

1. Urgently work up patients who exhibit deterioration on serial GCS and/or NIHSS.
2. Check for oversedation.
3. Treat seizures with anticonvulsant, if no contraindication. Adjust the dose if the patient has hepatic or renal insufficiency.
4. Give supplemental oxygen if the oxygen saturation is < 93%. Ensure airway.
5. Correct hypoglycemia or hyperglycemnia. (*See section on Blood Sugar in Acute Stroke*)
6. Treat fever and infection. (*See section on Fever and Infection*)
7. Treat severe hypertension or correct hypotension according to stroke subtype. (*See sections on Blood Pressure in Acute Ischemic Stroke, Acute ICH: Medical and BP Management, and Acute SAH: Medical Management*)
8. If the patient complains of chest pain, diaphoresis, or dyspnea/tachypnea, consider acute myocardial infarction and pulmonary embolism. (*See sections on Deep Venous Thrombosis/Pulmonary Embolism and Myocardial Infarction*)
9. Perform a brain imaging (CT scan or MRI) as soon as possible to rule out:
   - ☐ Worsening cerebral edema
   - ☐ Progression or recurrence of cerebral infarct
   - ☐ Hemorrhagic conversion of cerebral infarct
   - ☐ Expansion/extension of intracerebral hemorrhage

- ☐ Development of hydrocephalus
- ☐ Rebleeding of subarachnoid hemorrhage
- ☐ Infarction from vasospasm in subarachnoid hemorrhage

10. Manage increasing intracranial pressure if present. (*See section on Increased ICP*)
11. Facilitate diagnostic tests and consider "escalation" of treatment according to stroke subtype. (*See sections on TIA and Acute Ischemic Stroke, Acute ICH, or Acute SAH*)

Evidence

1. As much as 40% of patients with acute ischemic stroke experience clinical deterioration or progression of neurological deficits. Total anterior circulation and lacunar strokes are major groups showing progressive motor deficit.
2. Up to a quarter of patients with intracerebral hemorrhage, particularly those who present within 24 h of onset, have subsequent enlargement of the hematoma.
3. Fever, infection, hypoglycemia, hyperglycemia, hypotension, hypoxemia, and sedating drugs worsen neurological status after a stroke.
4. Paradoxically, early improvement in stroke deficit is a risk factor for subsequent deterioration.
5. Augmenting cerebral blood flow may improve/prevent worsening. More studies are needed to determine the role of anticoagulant in progressing stroke.

Selected Readings

1. Johnston SC, Leira EC, Hansen MD, Adams HP Jr. (2003) Early recovery after cerebral ischemia and risk of subsequent neurological deterioration. *Ann Neurol* **54**: 439–444.
2. Aslanyan S, Weir CJ, Johnston SC, Lees KR. (2004) Poststroke neurological improvement within 7 days is associated with subsequent deterioration. *Stroke* **35**: 2165–2170.
3. Tei H, Uchiyama S, Ohara K, *et al*. (2000) Deteriorating ischemic stroke in 4 clinical categories classified by the Oxfordshire Community Stroke Project. *Stroke* **31**: 2049–2054.
4. Nakamura K, Saku Y, Ibayashi S, *et al*. (1999) Progressive motor deficits in lacunar infarction. *Neurology* **52**: 29–33.
5. Steinke W, Ley SC. (2002) Lacunar stroke is the major cause of progressive motor deficits. *Stroke* **33**: 1510–1516.
6. Fujii Y, Takeuchi S, Sasaki O, *et al*. (1998) Multivariate analysis of predictors of hematoma enlargement in spontaneous intracerebral hemorrhage. *Stroke* **29**: 1160–1166.
7. Brott T, Broderick J, Kothari R, *et al*. (1997) Early hemorrhage growth in patients with intracerebral hemorrhage. *Stroke* **28**: 1–5.

NOTES

NOTES

INCREASED INTRACRANIAL PRESSURE

Dr. Epifania V. Collantes and Dr. Ester S. Bitanga

Goal

To reduce intracranial pressure to prevent herniation and irreversible brain injury.

Patient Data Needed

1. Stroke subtype diagnosis: ☐ Cerebral Infarct ☐ Intracerebral Hemorrhage ☐ Subarachnoid Hemorrhage
2. Location of stroke: ☐ Hemispheric ☐ Cerebellum
3. Serial GCS scores
4. BP, HR, RR
5. Pupillary size
6. ICP, if available: _____ mm Hg (1 mm H_2O = 0.07 mm Hg)
7. MAP $= \frac{SBP + (2 \times DBP)}{3} =$ _____ mm Hg

Actions

1. Suspect increasing ICP if the serial GCS is downtrend or if the patient exhibits at least two features of Cushing's triad (hypertension, bradycardia, bradypnea). A worsening GCS with an asymmetric pupillary size is transtentorial herniation until proven otherwise.
2. Check the ICP, if direct measurement through implanted monitoring device is available. A normal ICP is 3–15 mm Hg (50–200 mm H_2O).
3. Ensure adequate oxygenation and prevent hypercapnia.
4. Use only isotonic fluids, e.g. normal saline or lactated ringer's solution.
5. Elevate head to 20° to 30° from horizontal. Keep head midline to avoid venous obstruction.
6. Avoid coughing and straining.
7. Maintain normothermia.
8. If a direct ICP measurement is available, keep cerebral perfusion pressure (CPP) > 70 and < 110 mm Hg by using therapy to reduce the ICP and manipulation of the BP, where

   $$CPP = MAP - ICP.$$

   (*See also section on Blood Pressure in Acute Ischemic Stroke*)
9. Use osmotherapy, hyperventilation, and other measures to reduce the ICP if needed. Do not use them "prophylactically." (*See Insert 1 in this section*)
10. Do not give steroids.
11. Relieve pain and agitation. Use small doses of narcotics or sedation enough to relieve agitation or attain a quiet state but not to obscure neurological monitoring.

Agent	Dose
Sedative-hypnotic	
Propofol	0.6 to 6 mg/kg/h
Midazolam	0.05 to 0.1 mg/kg/h
Sedative-analgesic	
Morphine sulfate	2 to 5 mg every 1–4 h
Fentanyl	0.5 to 3.0 μg/kg/h
Sufentanil	0.1 to 0.6 μg/kg/h

12. Refer to a neurosurgeon for possible surgical intervention.
    a. Evacuation of hematoma, particularly in the posterior fossa/cerebellum (*See section on Surgery for Acute ICH*)
    b. Hemicraniectomy for "malignant" MCA infarction
    c. Suboccipital craniectomy for massive cerebellar infarction
    d. Ventriculostomy for intraventricular hemorrhage, cerebellar infarction with obstruction of the 4th ventricle (in association with posterior fossa decompression), or subarachnoid hemorrhage with the hydrocephalus.

Evidence

1. Head elevation to 30° reduces ICP by reducing jugular and cerebral venous pressure and enhancing venous outflow without significantly lowering CPP, CBF and cardiac output.
2. Mild to moderate hypothermia reduces cerebral metabolic rate leading to reduction in $CMRO_2$, CBF, ICP and glutamate release. Moderate hypothermia can help to control critically elevated ICP in severe edema after MCA stroke.
3. CPP < 70 mm Hg triggers vasodilatation and ICP elevation. CPP > 110 mm Hg increases cerebral perfusion, increasing the ICP.
4. Respiratory alkalosis from hyperventilation lowers the ICP by cerebral vasoconstriction.
5. Steroid is not recommended as management for an increased ICP.
6. Decompressive surgery for malignant MCA infarction within 48 h reduces mortality.

Selected Readings

1. Mayer SA, Chong JY. (2002) Critical care management of increased intracranial pressure. *Intensive Care Med* **17**: 55–67.
2. Feldman Z, Kanter MJ, Robertson CS, *et al*. (1992) Effects of head elevation on intracranial pressure, cerebral perfusion pressure and cerebral blood flow in head injured patients. *J Neurosurg* **76**: 207–211.
3. Rosner MJ, Rosner SD, Johnson AH. (1995) Cerebral perfusion pressure: Management protocol and clinical results. *J Neurosurg* **83**: 949–962.
4. Bardutzky J, Schwab S. (2007) Antiedema therapy in Ischemic Stroke. *Stroke* **38**: 3084–3094.
5. Vahedi K, Juettler E, Hofmeijer J, *et al*. (2007) Early decompressive surgery for malignant infarction of middle cerebral artery: A pooled analysis of three randomized controlled trials. *Lancet Neurol* **6**: 215–222.

INCREASED INTRACRANIAL PRESSURE

Insert 1. Medical Management of Increased ICP

Osmotherapy

1. Mannitol 20%
   - — Loading dose: 1 to 1.5 mg/kg
   - — Subsequent dose: 0.25 to 1 gm/kg every 4 to 6 h
   - — May combine with furosemide (10 mg every 2 to 8 h) to maintain an osmotic gradient
   - — Monitor Na and K and creatinine regularly.
   - — Adjust the dose according to:
     a. Clinical status
     b. ICP measurement, if available
     c. Maintaining hyperosmolar state of 300–320 mOsm/L (measured)
     d. Keeping osmolal gap (measured serum osmolality — computed serum osmolality) between 10 to 20 mOsm/L.

     $$\textit{Computed Serum Osm} = 2 \times [\textit{Na (mmol/L)} + \textit{K}^{*}\ \textit{(mmol/L)}] + \frac{\textit{Glucose (mg/dL)}}{18} + \frac{\textit{BUN (mg/dL)}}{2.8}$$

     **K is optional*
     Normal computed serum osmolality is 285–295 mOsm/L.
2. Hypertonic saline solutions
   - — 75 ml of 10% saline over 15 min
   - — 100 ml of 75 g/L NaCl and 60 g/L hydroxyethyl starch solution over 15 min.

Hyperventilation

1. Increase ventilation rate at a constant tidal volume (12 to 14 mL/kg) in mechanically ventilated patients
2. Target pCO_2 levels of 25 to 35 mm Hg. Lower levels may be detrimental
3. Reduction of cerebral blood flow is almost immediate. Peak ICP reduction may take up to 30 min.

Others

1. Muscle relaxants
   - — Consider pretreatment with a bolus of a muscle relaxant or lidocaine before airway suctioning.
   - — Neuromuscular paralysis with nondepolarizing agents (e.g. vecuronium, pancuronium) prevents increases in intrathoracic and venous pressure associated with coughing, straining, suctioning, or “bucking” the ventilator.
2. Hypothermia: Cool the core body temperature to 32–33°C

References

Roper AH, Rockhoff MA.(1993) Physiology and clinical aspects of raised intracranial pressure. *Neurology and Neurosurgical Intensive Care, 3rd edn*. Philadelphia: Lippincott-Raven, pp. 11–28.

Broderick JP, Adams HP Jr, Barsan W, MD, *et al*. (1999) Guidelines for the management of spontaneous intracerebral hemorrhage. A statement for healthcare professionals from a special writing group of the Stroke Council, American Heart Association. *Stroke* **30**: 905–915.

Schwarz S, Schwab S, Bertram M. (1998) Effects of hypertonic saline hydroxyethyl solution and mannitol in patients with increased ICP after stroke. *Stroke* **29**: 1550–1555.

Schwarz S, Giorgiadis D, Aschoff A. (2002) Effects of hypertonic saline in patients with raised ICP after stroke. *Stroke* **33**: 136–140.

Schwab S, Schwarz S, Spranger M, *et al*. (1998) Moderate hypothermia in the treatment of patients with severe MCA infarction. *Stroke* **29**: 2461–2466.

NOTES

NOTES

FEVER AND INFECTION

Dr. Marlie Jane Mamauag

Goal

To detect and treat fever and infection early to improve outcome after stroke.

Patient Data Needed

1. Body temperature: _____ °C (aural or axillary)
2. Hospitalization day: _____
3. BP: _____ / _____ mm Hg
   Heart rate: _____ /min Respiratory rate: _____ /min
4. Symptoms of respiratory, urinary, abdominal problems
5. Presence of (signs of):

☐ Cardiac murmur	☐ Wound and skin infection
☐ Rales/crackles/"creps"	☐ Pressure sores/bed sores
☐ Urinary bladder distention	☐ Calf swelling
☐ "Cloudy urine"	☐ Abdominal tenderness
☐ Phlebitis	☐ Pharyngitis

Actions

1. Consider as fever if temperature is ≥ 37.8°C (or if temperature is ≥ 37.5°C on two separate occasions).
2. Check complete (full) blood count (CBC/FBC), ESR, C-reactive protein, electrolytes, creatinine, urea (BUN), urinalysis (urine FEME), and blood culture.
3. Correct dehydration.
4. Perform chest X-ray. Send sputum for grams stain and culture if patient has pulmonary signs and symptoms.
5. Check urine culture if the patient has urinary signs and symptoms or abnormal urinalysis (urine FEME).
6. Consider infective endocarditis in patients who present with stroke, fever, and cardiac murmur.
7. Remove the intravenous catheter if not needed or if phlebitis is present. Change the intravenous site at least every three days.
8. Institute wound care for skin breakdown and pressure sores.
9. Start empirical antibiotics empirically for the suspected infection. Consider hospital-acquired infection if fever starts after day 2 of hospitalization.
10. Give antipyretic medication (e.g. paracetamol) every 4 h, if necessary. Consider cooling devices if the fever does not respond to antipyretic medication.
11. Change current antibiotics if the fever does not lyse in the next 48 to 72 h.
12. If calf swelling is present, consider DVT. (*See section on Deep Vein Thrombosis and Pulmonary Embolism*)

13. If no obvious source of infection is found, consider work up for less common and often unrecognized sources of fever, including:
    — abdominal (e.g. cholecystitis, intraabdominal abscess, etc.)
    — gynecological (e.g. pelvic abscess, vaginal infection, etc.)
    — musculoskeletal (e.g. osteomyelitis, etc.)
    — other infections (e.g. fungal, tuberculous, parasitic, viral, anaerobic etc.)
    — auto-immune conditions and vasculitides.
    — endocrine (e.g. thyroid, adrenal, etc.)
    — neoplasm (e.g. solid, haematological, etc.)
    — drug fever (e.g. beta-lactam antibiotic, phenytoin, etc.).

Evidence

1. Fever in the setting of acute stroke is associated with poor neurological outcome (increased risk of morbidity and mortality).
2. Acute infections increase the risk of stroke, particularly during the first 3 days of infection.
3. The effectiveness of prophylactic use of acetaminophen or antibiotics after a stroke to improve neurological outcome is not yet established.

Selected Readings

1. Adams HP Jr, del Zoppo G, Alberts MJ, *et al*. (2007) Guidelines for the early management of adults with ischemic stroke. *Stroke* **38**: 1655–1711.
2. Azzimondi G, Bassein L, Nonino F, *et al*. (1995) Fever in acute stroke worsens prognosis: A prospective study. *Stroke* **26**: 2040–2043.
3. Ginsberg MD, Busto R. (1998) Combating hyperthermia in acute stroke: A significant clinical concern. *Stroke* **29**: 529–534.
4. Hajat C, Hajat S, Sharma P. (2000) Effects of poststroke pyrexia on stroke outcome: A meta-analysis of studies in patients. *Stroke* **31**: 410–414.
5. Wang Y, Lim LL, Levi C, *et al*. (2000) Influence of admission body temperature on stroke mortality. *Stroke* **31**: 404–409.
6. Kammersgaard LP, Jorgensen HS, Rungby JA, *et al*. (2002) Admission body temperature predicts long-term mortality after acute stroke: The Copenhagen Stroke Study. *Stroke* **33**: 1759–1762.
7. Lindsberg PJ, Grau AJ. (2003) Inflammation and infection as risk factors for ischaemic stroke. *Stroke* **34**: 2158–2532.
8. Chamorro A, Horcajada JP, Obach V, *et al*. (2005) The early systemic prophylaxis of infection after stroke study — A randomized clinical trial. *Stroke* **36**: 1495–1500.

NOTES

NOTES

DEEP VEIN THROMBOSIS AND PULMONARY EMBOLISM

Dr. Ruth Marie R. Divinagracia

Goal

To prevent, recognize, and manage DVT and/or subsequent PE.

Patient Data Needed

1. Duration of immobilization of the patient
2. Contraindication to anticoagulation
3. Clinical features of DVT (e.g. leg swelling, pain on palpation of deep vein) or PE (e.g. dyspnea, tachypnea, pleuritic pain, hemoptysis)
4. Vital signs (heart rate, blood pressure, respiratory rate, temperature)
5. History of previous DVT/PE or malignancy

Actions

1. Give DVT prophylaxis to all immobilized stroke patients who are at high risk.
   — If anticoagulation is feasible, prophylax with unfractionated Heparin at 5000 units subcutaneously (SC) BID or LMWH once daily (e.g. enoxaparine 0.4 ml SC, fraxiparine 0.3 ml SC or dalteparin 5000 iu SC).
   — If anticoagulation is contraindicated (e.g. hemorrhagic stroke, possible hemorrhagic conversion of a ischemic stroke, systemic bleeding), use Intermittent Pneumatic Compression (IPC) device with or without graduated compression stockings.
2. Assess the probability of "clinical DVT" if suspected. (*See Insert 1 in this section*)
   — Perform a Venous Duplex Scan/compression ultrasound of the lower extremities:
   If (+), treat according to #4 below.
   If (–) but clinical suspicion is high, perform CT angiography or MRI of the pelvis and lower extremities.
3. Assess the probability of "clinical PE" if suspected. (*See Insert 2 in this section*)
   — Consider an initiation of treatment pending the diagnostic evaluation. (see #4 below)
   — Follow the management algorithm for suspected PE. (*See Insert 3 in this section*)
4. Treat DVT or PE
   — If anticoagulation is not contraindicated, treatment options are:
      a. Start IV heparin to achieve an aPTT 1.5–2 times the control value. Load with heparin 5000 units by an intravenous bolus (if not contraindicated) and start infusion at 800 units/h. Check the aPTT every 6 h until the aPTT is stabilized. Adjust the drip as needed. Monitor the platelets every 3 to 4 days. (*See also Inserts 1 and 2 in section on TIA and Acute Ischemic Stroke: Anticoagulation*)
      b. Give LMWH (e.g. enoxaparine 1 mg/kg sc BID or fraxiparine 85 iu/kg SC BID). No aPTT monitoring is required. Monitor platelets every 3 to 4 days.
      - Warfarin can be started on day 1 or 2 by instituting the estimated daily maintenance dose. Overlap therapy for 3–5 days or until prothrombin time (PT) INR has become therapeutic (INR 2.0 to 3.0).

- Anticoagulation should be continued for 3 to 6 months.
- Closely monitor for any bleeding.

— If anticoagulation is contraindicated, insert an IVC filter device.

- Retrievable and "permanent" IVC filters are available. If anticoagulation may be started within 4 weeks of the acute DVT or PE, a retrievable filter should be considered.
- Place filter percutaneously under local anesthesia into the IVC below the level of the renal veins.

Evidence

1. Pulmonary embolism is the third leading cause of cardiovascular mortality in North America. In large autopsy series, PE-related death among hospitalized patients has remained constant at 15%. Patients with stroke are at high risk due to immobilization as well as presence of comorbidities.
2. Prophylaxis with unfractionated heparin or low molecular weight heparin reduces DVT, subsequent PE, and PE-associated deaths. Despite no large trials, IPC appears to be as effective as unfractionated heparin in preventing DVT.
3. Venous duplex scan is easy, sensitive and specific as initial test for DVT. CT pulmonary angiography has better accuracy than Ventilation-Perfusion Scan.
4. D-Dimer as initial screening test for suspected PE has high negative predictive value. A normal D-Dimer should make the clinician think of another diagnosis other than PE. An elevated D-Dimer does not mean the patient has PE but means that PE definitely should be considered as a diagnosis.
5. Placement of IVC filter reduces PE from DVT, although no large trials have shown filter effectiveness for prevention of recurrent PE and PE fatality. Long-term complications of "permanent" IVC filters include insertion site DVT, filter migration, and IVC-related obstruction/venous insufficiency.
6. Duration of treatment for DVT/PE can be up to 3 months (for reversible or time-limited risk factor e.g. patient able to ambulate within that time) or 6–12 months (for continuing risk factor e.g. bed bound).

Selected Readings

1. Fedullo PF, Tapson VF. (2003) The evaluation of suspected pulmonary embolism. *N Engl J Med* **349**: 1247–1256.
2. Geerts WH, Heit JA, Clagett GP, *et al*. (2001) Prevention of venous thromboembolism. *Chest* **119** (Suppl 1): 132S-175S
3. Ramzi DW, Leeper KV. (2004) DVT and pulmonary embolism: Part II. Treatment and prevention. *Am Fam Phys* **69**: 2841–2848.
4. Wells PS, Anderson DR, Roger M, *et al*. (2003) The evaluation of D-dimer in the diagnosis of suspected deep-venous thrombosis. *NEJM* **349**: 1227–1235.
5. Qaseem A, Snow V, Barry P, *et al*. (2007) Current diagnosis of venous thromboembolism in primary care: A clinical practice guideline from the AAFP and the ACP. *Ann Int Med* **146:** 454–458.
6. PREVAIL Investigators. (2007) The efficacy and safety of enoxaparin versus unfractionated heparin for the prevention of venous thromboembolism after acute ischaemic stroke: An open-label randomised comparison. *Lancet* **369**: 1347–1355.

DEEP VEIN THROMBOSIS AND PULMONARY EMBOLISM

Insert 1. Wells' Prediction Rule For Diagnosing Deep Venous Thrombosis

Clinical Feature	Points
Active cancer (treatment ongoing, within previous 6 months, or palliative)	1
Paralysis, paresis, or recent plaster immobilization of the lower extremities	1
Recently bedridden > 3 days or major surgery within 12 weeks requiring general or regional anesthesia	1
Localized tenderness along the distribution of the deep venous system	1
Entire leg swollen	1
Calf swelling 3 cm larger than asymptomatic side (measured 10 cm below tibial tuberosity)	1
Pitting edema confined to the symptomatic leg	1
Collateral superficial veins (nonvaricose)	1
Alternative diagnosis at least as likely as deep venous thrombosis	–2

In patients with symptoms in both legs, the more symptomatic leg is used

< 1	Low clinical probability
1 to 2	Intermediate clinical probability
≥ 3	High clinical probability

Reference

Wells PS, Anderson DR, Bormanis J, *et al.* (1997) Value of assessment of pretest probability of deep-vein thrombosis in clinical management. *Lancet* **350:** 1795–1798.

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DEEP VEIN THROMBOSIS AND PULMONARY EMBOLISM

Insert 2. Wells' Clinical Model for Suspecting Pulmonary Embolism

Clinical Features	Points
Clinical signs of DVT — swelling, pain with palpation of deep vein	3.0
Heart rate > 100 beats per min	1.5
Immobilization ≥ 3 consecutive days or surgery within 4 weeks	1.5
Previous proven DVT or PE	1.5
Hemoptysis	1.0
Malignancy	1.0
Alternative diagnosis less likely than PE	3.0

< 2.0	Low clinical probability
2 to 6	Intermediate clinical probability
> 6.0	High clinical probability

Reference

Wells PS, Anderson DR, Rodger M, *et al*. (2001) Excluding pulmonary embolism at the bedside without diagnostic imaging: Management of patients with suspected pulmonary embolism presenting to the emergency department by using a simple clinical model and d-dimer. *Ann Intern Med* **135**: 98–107.

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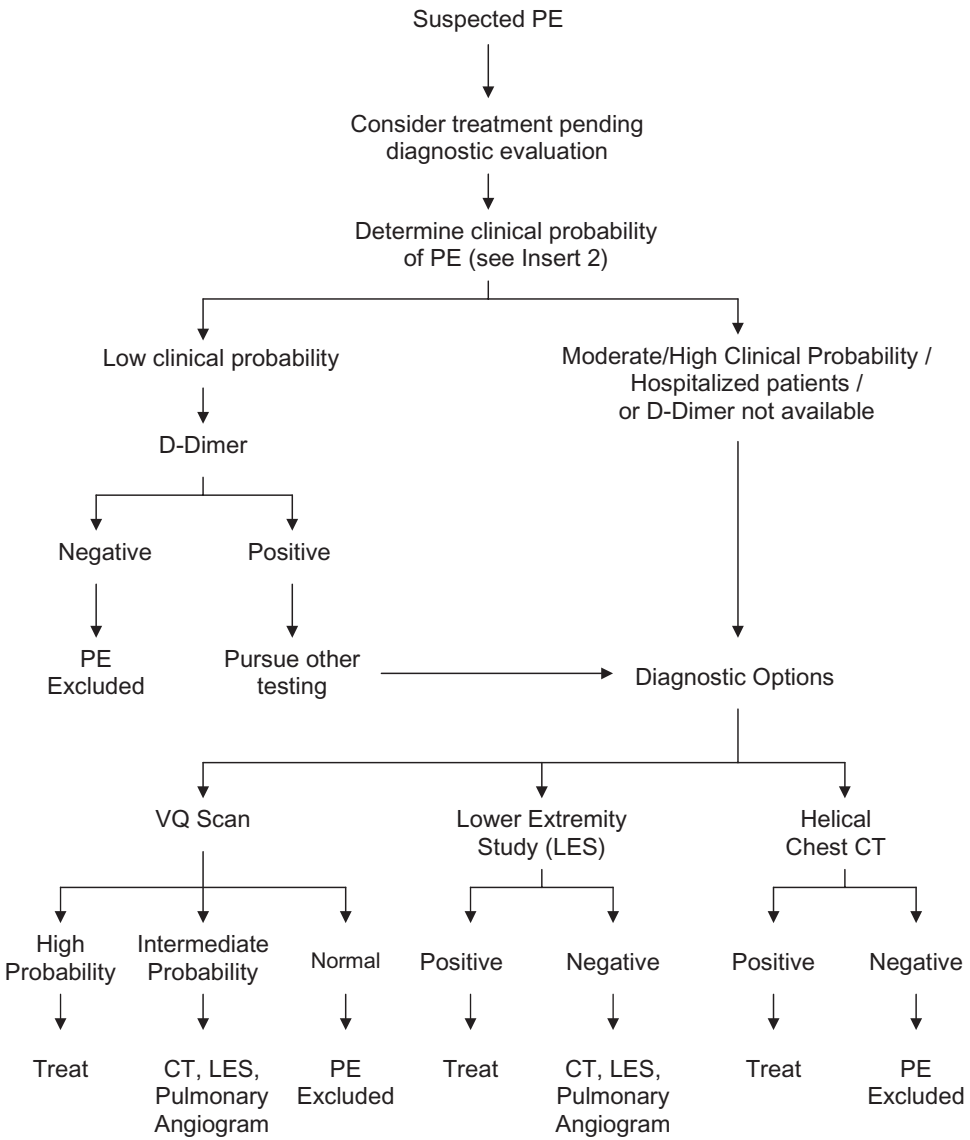
Insert 3. Management Algorithm for Suspected PE



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MYOCARDIAL INFARCTION IN ACUTE STROKE

Dr. Bernard PL Chan

Goal

To recognize co-existing acute MI and its mimics (i.e. dissecting thoracic aortic aneurysm, stress cardiomyopathy) in the setting of acute stroke and to initiate appropriate management in consultation with the cardiologist.

Patient Data Needed

1. Clinical features ☐ chest pain ☐ dyspnea ☐ diaphoresis
   ☐ hypotension ☐ arrhythmia or abnormal heart rate
2. Major contraindications to:
   All antithrombotics — hemorrhagic stroke, suspicion of dissecting aortic aneurysm
   High-dose heparin — large infarct size, uncontrolled hypertension

Actions

1. Carefully examine pulses and review the admission ECG and chest X-ray, as acute MI and dissecting aortic aneurysm can be painless. (*See Insert 1 in this section*)
2. If the patient has the above clinical features or abnormalities on ECG or chest X-ray:
   — measure cardiac enzymes including troponin I or T
   — do serial ECG and troponin if initial determinations are non-diagnostic
   — refer to the cardiologist.
3. Start continuous ECG monitoring and pulse oxymetry with frequent monitoring of neurological and cardiovascular status.
4. When appropriate, start low-dose nitrate, beta-blocker, and ACE inhibitor for management of angina, BP and left ventricular (LV) dysfunction. Avoid excessive reduction in BP that may adversely affect cerebral perfusion.
5. Transfer patients with poor cardiac output, hypotension, or frequent arrhythmias to the coronary care unit for intensive hemodynamic monitoring and access to defibrillation.
6. Perform transthoracic echocardiography to assess LV ejection fraction and to look for regional wall motion abnormality (RWMA), focal or global hypokinesia, and other potential source of cardioembolism, such as LV thrombus.
   — If an aortic dissection is suspected, consider CT scan of the chest/aorta or transesophageal echocardiography and refer to a cardiothoracic surgeon.
7. Consider myocardial reperfusion with primary percutaneous coronary intervention (PCI) in selected patients with stable stroke but severe acute MI.
8. If there is no contraindication, start aspirin 150 to 325 mg loading dose then 75 to 100 mg per day. Additional antithrombotic treatments include clopidogrel and heparin, and should be decided after careful consideration of the following:
   a. Adequacy of management of cerebrovascular and coronary ischemia
   b. Risk of intracerebral hemorrhage (related to cerebral infarct size and BP)
   c. Risk of recurrent cardioembolic stroke (e.g. presence of AF, RWMA, LV dysfunction, LV thrombus)

d. Specific indications (e.g. coronary stent).

— The following may be used in selected patients to reduce the risk of cerebral hemorrhage: omit the loading dose of clopidogrel when used in combination with aspirin, use low-dose instead of high-dose heparin, use low molecular weight instead of unfractionated heparin, and limit the dose of heparin used during PCI.

9. "Stress cardiomyopathy", characterized by chest pain, dyspnea, mild elevation in troponin, ECG changes that evolve over time, and echocardiographic evidence of RWMA and LV dysfunction beyond a single coronary artery territory, frequently requires supportive therapy for transient LV dysfunction.

Evidence

1. Stroke patients are at increased risk of developing MI and vice versa due to common underlying vascular risk factors and frequent co-existence of symptomatic or asymptomatic coronary and cerebrovascular diseases. Acute MI, either as cause of cardioembolic stroke or as complication of stroke, substantially worsens outcome.
2. About 4% of patients with acute ischemic stroke die from cardiac causes within 3 months (peak around day 14) and are usually preceded by one or more serious cardiac adverse events (acute MI, heart failure, ventricular tachycardia or fibrillation) occurring mostly between 2 to 3 days after stroke onset.
3. Stress cardiomyopathy may rarely complicate acute stroke, particularly subarachnoid hemorrhage, and is believed to be due to myocardial stunning caused by local release of catecholamines from cardiac nerves. Post-menopausal women and patients with right insular stroke are at increased risk. Although complete recovery in cardiac function is expected in the most patients, incidence of sudden cardiac death secondary to autonomic derangement and arrhythmias may be increased.
4. In acute ischemic stroke complicated by acute MI, rTPA is usually contraindicated because of the very short 3-h time window after stroke onset. Even when rTPA can be given within this time constraint, the lower dose used for acute stroke is suboptimal for acute MI. Conversely, in acute ischemic stroke patients with preceding acute MI, rTPA is often contraindicated as late treatment > 24 h after acute MI is associated with risks of cardiac tamponade and myocardial rupture.

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MYOCARDIAL INFARCTION IN ACUTE STROKE

Insert 1. Checklist of changes to look for on admission ECG and chest X-ray

On ECG look for:

— Ischemic or repolarization changes

- ☐ ST elevation
- ☐ ST depression
- ☐ T wave inversion
- ☐ flattened T wave
- ☐ peaked T wave
- ☐ U waves
- ☐ QTc prolongation
- ☐ Q waves

— Arrhythmias

- ☐ atrial fibrillation
- ☐ supraventricular ectopies/arrthythmia
- ☐ ventricular ectopies/arrhythmias

On chest X-ray, look for:

- ☐ cardiomegaly
- ☐ mediastinal widening
- ☐ pulmonary edema

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ANTITHROMBOTICS: ANTIPLATELETS AND ANTICOAGULANT

Dr. Robert N. Gan, Dr. N. Venketasubramanian Ramani, and Dr. Tan Kay Sin

Goal

To prevent ischemic stroke, other vascular event, and death in high-risk patients.

Patient Data Needed

1. Diagnosis of ischemic stroke or TIA ☐ Yes ☐ No
   TOAST classification (*See section on Diagnostic Tests for TOAST Classification*)
   - ☐ Large-artery atherosclerosis
   - ☐ Small artery occlusion (lacune)
   - ☐ Cardioembolism
   - ☐ Stroke of undetermined etiology
   - ☐ Stroke of other determined etiology, specify _____
2. If there is no ischemic stroke or TIA but there is atrial fibrillation, evidence of:
   - ☐ Heart failure or ejection fraction ≤ 35%
   - ☐ Hypertension
   - ☐ Age ≥ 75 years
   - ☐ Diabetes mellitus
3. Contraindication to antiplatelet or anticoagulant:
   - ☐ Allergy to _____
   - ☐ Bleeding risk, e.g. gastrointestinal, etc.
   - ☐ Non-compliant
   - ☐ Unsafe

Actions

1. Follow guideline on choosing long-term antiplatelet or anticoagulation. (*See Insert 1 in this section*)
2. Avoid combining anticoagulation and antiplatelet unless there is a strong indication to do so, e.g. concurrent coronary artery disease with or without stent, recurrent ischemic stroke despite adequate anticoagulation for cardioembolic stroke, etc.
3. For patients requiring long-term anticoagulation:
   - — Start warfarin (coumadin) at 5 mg per day for first 1 or 2 days with subsequent dosing based on INR.
   - — Monitor INR every 1 or 2 days after the second or third dose until therapeutic range is achieved and maintained for at least 2 consecutive days, then reduce INR testing to 2 or 3 times weekly for 1 to 2 weeks, then less often, depending on the stability of INR results.
   - — In patients older than 75 years, target a lower INR of 2.0–2.5.
   - — In patients with protein C or S deficiency, start parenteral anticoagulation, before or at the same time as oral anticoagulation to prevent early transient hypercoagulable state when warfarin (coumadin) is started (*See also section on TIA and Acute Ischemic Stroke: Anticoagulation*)
   - — In patients with contraindication to anticoagulation, consider antiplatelet therapy.
   - — In patients whose underlying reason for cardioembolism has been corrected, e.g. intracardiac clots secondary to MI resolved on subsequent echo, transient AF due to thyrotoxicosis, pulmonary embolism, MI, or recent surgery, consider switching oral anticoagulation to antiplatelet therapy.
4. For patients requiring long-term antiplatelet therapy, options include:
   a. aspirin 75 to 325 mg once a day
   b. cilostazol 100 mg twice a day
   c. clopidogrel 75 mg once a day

d. combination aspirin 25 mg + dipyridamole 200 mg twice a day (slow-release)
e. dipyridamole 200 mg twice a day (slow-release)
f. ticlopidine 250 mg twice a day.
   — Review contraindication to specific antiplatelet when deciding on regimen.
   — For ticlopidine, monitor complete blood count every 2 weeks for the first 3 months.
   — For dipyridamole, may titrate dose up over several days to avoid developing headache.

5. Regularly monitor patients for signs of bleeding.

Evidence

1. Long-term antiplatelet therapy reduces the risk of serious vascular events (recurrent stroke, myocardial infarction or vascular death) following an ischemic stroke or TIA by 22%; 36 serious vascular events will be avoided over 2 years per 1000 patients with previous stroke or transient ischaemic attack.
2. Aspirin is the most widely studied antiplatelet drug, with doses of 75–150 mg daily being at least as effective as higher daily doses. Other antiplatelet agents proven beneficial in randomized clinical trials include ticlopidine, clopidogrel, cilostazol, and dipyridamole (alone or in combination with aspirin).
3. The long-term combination of aspirin and clopidogrel is not superior to either aspirin or clopidogrel alone in preventing vascular events in high-risk patients and increases the risk of hemorrhagic complications. A trial comparing combination aspirin-dipyridamole to clopidogrel is currently underway.
4. Warfarin is no better than aspirin in non-cardioembolic ischemic stroke and may increase the risk of bleeding.
5. Patients with AF have a stroke risk of 4.5% per year. The risk is higher (8%) in patients with recent stroke/TIA, age > 75, diabetes, hypertension and congestive heart failure. Warfarin use reduces annual stroke rate from 4.5% to 1.4%.
6. In patients with AF and TIA or minor stroke, the risk of death, myocardial infarction, stroke, and systemic embolism is 19%/year. Anticoagulation reduces the annual risk to 8% (stroke risk reduced from 12% to 4%/year) while aspirin reduces risk to 15%. Risk of major bleeding for anticoagulation is 2.8%/year and aspirin 0.9%/year.

Selected Readings

1. Antithrombotic Trialists' Collaboration. (2002) Collaborative meta-analysis of randomised trials of antiplatelet therapy for prevention of death, myocardial infarction, and stroke in high risk patients. *BMJ* **324**: 71–86.
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ANTITHROMBOTICS: ANTIPLATELETS AND ANTICOAGULANTS

Insert 1. Choice of Long-Term Antiplatelet or Anticoagulation Therapy

Condition	Choice of Antithrombotic Therapy
Not TIA or ischemic stroke	
(–) Atrial fibrillation	None
(+) Atrial fibrillation	
CHADS2 score* < 2	Antiplatelet
CHADS2 score* ≥ 2	Anticoagulation (INR 2.0–3.0) long-term
Ischemic Stroke/TIA (TOAST classification**)	
Atheroclerotic	Antiplatelet
Lacunar	Antiplatelet
Cardioembolic	
Infective endocarditis	None
Persistent/Paroxysmal AF	Anticoagulation (INR 2.0–3.0) long-term
Rheumatic mitral valve disease	Anticoagulation (INR 2.0–3.0) long-term
Bioprosthetic heart valve	Anticoagulation (INR 2.0–3.0) long-term
Mechanical heart valve	Anticoagulation (INR 2.5–3.5) long-term
Acute MI with LV thrombus	Anticoagulation (INR 2.0–3.0) for 3 to 12 mos then switch to antiplatelet
Dilated cardiomyopathy	Antiplatelet or anticoagulation
Aortic valvular disease	Antiplatelet
Mitral valve prolapse	Antiplatelet
Patent foramen ovale	
(–) DVT	Antiplatelet
(+) DVT	Anticoagulation (INR 2.0–3.0) for 3 to 6 mos then switch to antiplatelet
Other determined cause	
Extracranial arterial dissection	Anticoagulation (INR 2.0–3.0) for 3 to 6 mos then switch to antiplatelet
Cerebral venous sinus thrombosis	Anticoagulation (INR 2.0–3.0) for 3 to 6 mos then switch to antiplatelet
Hyperhomocysteinemia	Antiplatelet + consider folate, vitamin B6 and B12 supplements
Hypercoagulable state	Anticoagulation (INR 2.0–3.0)
Antiphospholipid antibody positive	
(–) APL syndrome***	Antiplatelet
(+) APL syndrome***	Anticoagulation (INR 2.0–3.0)
Undertermined cause	Antiplatelet

* See Insert 2

** See section on Diagnostic Tests for TOAST Classification

*** Multiple organ venous or arterial occlusive disease, miscarriages, livedo reticularis, etc.

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ANTITHROMBOTICS: ANTIPLATELETS AND ANTICOAGULANTS

Insert 2. CHADS2 Scoring for Risk Stratification in Patients with Atrial Fibrillation

Clinical Features	Score
Congestive heart failure or left ventricular ejection fraction of ≤ 35%	1
Hypertension	1
Age ≥ 75	1
Diabetes mellitus	1
Stroke or TIA	2
Total Score*	

*Score ≥ 2 indicates moderate to high risk of thromboembolic event

CHADS2 Score	Adjusted Stroke Rate (%/year) (95% CI)
0	1.9 (1.2 to 3.0)
1	2.8 (2.0 to 3.8)
2	4.0 (3.1 to 5.1)
3	5.9 (4.6 to 7.3)
4	8.5 (6.3 to 11.1)
5	12.5 (8.2 to 17.5)
6	18.2 (10.5 to 27.4)

References

Gage BF, van Walraven C, Pearce L, *et al*. (2004) Selecting patients with atrial fibrillation for anticoagulation: Stroke risk stratification in patients taking aspirin. *Circulation* **110**(16): 2287–2292.

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BLOOD PRESSURE AND ANTIHYPERTENSIVES

Dr. Lee Sze Haur

Goal

To maintain BP at ≤ 130/80 mm Hg for secondary stroke prevention.

Patient Data Needed

1. BP
2. Co-morbid medical conditions, e.g.
   - ☐ Congestive heart failure
   - ☐ Renal impairment
   - ☐ Ischemic heart disease

Actions

1. If there are no contraindications, commence anti-hypertensive therapy about 2 weeks after the onset of the acute stroke.
2. Start treatment immediately if the patient is suffering from hypertensive encephalopathy, aortic dissection, acute renal failure, acute pulmonary edema, acute myocardial infarction, or severe hypertension greater than 220/120 mmHg.
3. Lower the BP promptly to less than 130/80 mm Hg within 3 months of starting treatment.
4. Effective anti-hypertensive medications include ACEI, ARB, thiazide diuretics, beta-blockers (BB), and calcium channel blockers (CCB).
5. In patients with concomitant congestive heart failure or renal impairment, consider using ACEI and ARB. In patients with ischemic heart disease, consider using BB.
6. Recommend a low salt diet.
7. For patients with poor BP control, recommend BP check at home 2 or 3 times a week using a home BP monitoring device.

Evidence

1. Secondary prevention trials in patients with recent stroke or TIA, who were either hypertensive or normotensive, show that lowering of BP reduces the relative risk of recurrent stroke by 28% over a period of 2 to 4 years.
2. The target BP for secondary stroke prevention in high-risk patients is < 130/80 mm Hg.
3. The time to starting anti-hypertensive therapy after acute stroke is variable as there is no good evidence for guidance. Some clinicians delayed treatment by 1 to 2 weeks.
4. High BP in two-thirds of patients with acute stroke falls spontaneously to baseline (pre-stroke) levels by 2 weeks after the onset of stroke.

5. Acute reduction of BP during the acute stage of stroke may worsen neurological outcome.
6. The benefit of lowering BP can be seen as early as 3 months after starting therapy.
7. Reduction in dietary salt intake helps to lower BP.

Selected Readings

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2. Guidelines Committee. (2003) 2003 European Society of Hypertension-European Society of Cardiology guidelines for the management of arterial hypertension. *J Hypertens* **21**: 1011–1053.
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8. Frost CD, Law MR, Wald NJ. (1991) By how much does dietary salt reduction lower blood pressure? I: Analysis of observational data within populations. *BMJ* **302**: 815–819.

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CHOLESTEROL

Dr. Maricar P. Yumul and Dr. N Venketasubramanian Ramani

Goal

To reduce cholesterol level to the target value for secondary stroke prevention.

Patient Data Needed

1. Ischemic stroke or TIA ☐ Yes ☐ No
2. Fasting Lipid Profile:
   - Total cholesterol: _____ mg/dL (_____ mmol/L)
   - HDL-cholesterol: _____ mg/dL (_____ mmol/L)
   - LDL-cholesterol: _____ mg/dL (_____ mmol/L)
   - Triglyceride: _____ mg/dL (_____ mmol/L)
3. Co-existing vascular risk factors, e.g.
   - ☐ CAD
   - ☐ Diabetes mellitus
   - ☐ Hypertension
   - ☐ Smoking

Actions

1. Advise patient on a reduced saturated fats and cholesterol diet, weight loss, physical activity, and smoking cessation. (*See section on Lifestyle Risk Factor Control*)
2. Refer to a nutritionist or dietician as necessary.
3. If the patient had an ischemic stroke or TIA:
   — start statin even if cholesterol levels are normal, unless there is contraindication.
   — consider niacin or gemfibrozil if HDL level is low.
4. Control other co-existing vascular risk factors in the patient. (*See sections on Blood Pressure and Antihypertensives, Diabetes Mellitus, and Lifestyle Risk Factor Control*)
5. Monitor the lipid panel about every 3 to 6 months. Include alanine transaminase, creatinine and/or creatine (phospho)kinase, if necessary.
6. Target lowering LDL level to < 100 mg/dL (< 2.6 mmol/L).
   — Target LDL level of < 70 mg/dL (< 1.8 mmol/L) if the patient is very high risk with multiple risk factors, i.e. CAD, diabetes, smoking, triglyceride level > 200 mg/dL (> 2.3 mmol/L), HDL level < 40 mg/dL (< 1.0 mmol/L), acute coronary syndrome.

Evidence

1. There is a strong positive relationship between cholesterol and ischemic stroke and a weaker negative association between cholesterol and hemorrhagic stroke.

2. A meta-analysis of trials on statins shows an overall relative risk reduction of 21% for stroke.
3. Greater LDL reduction is associated with greater stroke risk reduction.
4. A large-scale trial (SPARCL) of statin in patients with a history of stroke or TIA without CAD and have mildly elevated lipid levels showed significant stroke reduction of 16%.
5. Statins appear to have pleiotropic effects and may reduce stroke risk by several mechanisms, including plaque stabilization and anti-inflammatory effect, other than merely lowering lipid levels.

Selected Readings

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6. Welch KMA. (2004) Statins for the prevention of cerebrovascular disease: The rationale for robust intervention. *Eur Heart J* **6** (Suppl C): C34–C42.

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DIABETES MELLITUS

Dr. Abdias Aquino

Goal

To maintain blood sugar levels (BSL) to as close to normal as possible without significant hypoglycemia.

Patient Data Needed

1. Self-monitored BSL pre- and post-meals
2. HbA1c every 3 months
3. Co-existing vascular risk factors, e.g.
   - ☐ Hypertension
   - ☐ Dyslipidemia
   - ☐ Smoking
   - ☐ Obesity

Actions

1. Advise diabetic patients to monitor BSL before meals and/or 2 h post-meals.
2. Provide instructions on use of home blood sugar monitoring device. Refer to a pharmacist or nurse clinician if necessary.
3. Ideal glycemic goals should be:
   a. Preprandial BSL < 110 mg/dL (< 6.1 mmol/L) and 2-h postprandial BSL < 140 mg/dl (< 7.8 mmol/L)
   b. HbA1c ≤ 6.5%.
4. Advise patients on diet, weight loss, physical activity, and smoking cessation. (*See section on Lifestyle Risk Factor Control*)
5. Refer to a nutritionist or dietician as necessary.
6. Use oral anti-diabetic agents, if needed and not contraindicated, to achieve glycemic goals.
7. Scheduled subcutaneous insulin injection may be necessary and advantageous to the patient, if glycemic goals are not met with oral agents.
8. Aggressively address and treat other co-existing risk factors, i.e. maintaining blood pressure < 130/80 mm Hg, LDL < 70 mg/dL, HDL > 50 mg/dL, and triglyceride < 150 mg/dL. (*See sections on Hypertension and Cholesterol*)

Evidence

1. Cohort studies show that diabetic patients are 1.5 to 3 times more likely to have stroke than non-diabetics.
2. Although studies have not conclusively shown that tight control of serum glucose reduces the risk of stroke *per se*, tight sugar control leads to a reduction in microvascular complications like retinopathy, nephropathy, and neuropathy.
3. Tight blood pressure control among diabetics reduces stroke risk.

Selected Readings

1. American Diabetes Association: Standards of medical care in diabetes. (2006) *Diabetes Care* **29**(Suppl 1): S4–S42.
2. American Association of Clinical Endocrinologists. (2002) *Endocrine Pract* **8**(Suppl 1): 40–82.
3. Diabetes Control and Complications Trial Research Group. (1993) The effect of intensive treatment of diabetes on the development and progression of long-term complications in insulin-dependent diabetes mellitus. *N Engl J Med* **329**: 977–986.
4. European Diabetes Policy Group. (1999) *Diabet Med* **16**: 716–730.
5. UK Prospective Diabetes Study Group. (2000) Association of glycaemia with macrovascular and microvascular complications of type 2 diabetes (UKPDS 35): Prospective observational study. *BMJ* **321**: 405–412.

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LIFESTYLE RISK FACTOR CONTROL

Dr. Ma. Cristina Z. San Jose and Dr. Carlos Chua

Goals

To prevent stroke recurrence and other cardio-vascular events and to improve general well being by addressing modifiable lifestyle-related risk factors

Patient Data Needed

1. Height, Weight, BMI, Waist-hip ratio
2. Number of pack-years of smoking (number of packs/day X number of years)
3. Alcohol consumption (amount, type, frequency)
4. Patient's preferences in diet and physical activity
5. Residual disability and limitations from stroke

Actions

1. Encourage cigarette smokers to quit.
   - — Consider behavioral, pharmacological treatments and/or referral to smoking cessation clinic for nicotine dependence.
   - — Advise to avoid environmental (passive) smoking.
2. Habitual heavy drinkers should eliminate alcohol intake or reduce drinking to not more than 2 servings per day (e.g. 2 bottles of beer, 1–2 glasses of wine, or 2 jiggers of whiskey per day). Those who do not customarily drink alcohol should not be encouraged to do so.
3. Measure weight, height, waist circumference (WC), and compute for BMI (weight kg/height m^2).
   - — Provide behavioral counseling and encourage weight reduction for all overweight patients by decreasing caloric intake and increasing physical activity to achieve the following goals:

BMI	18–23
Waist-Hip Ratio	< 0.95 for men, < 0.85 for women
WC	< 90 cm (35 in) for men; < 80 cm (32 in) for women.

   - — Consider pharmacological and or surgical intervention in obese patients, if necessary.
4. Provide prescription for exercise tailored to the stroke patient's needs, limitations, and preferences (on what can be sustained). If able, advise to perform continuous or cumulative moderate-intensity aerobic exercise (e.g. brisk walking, jogging, ballroom dancing, swimming) for at least 30 min daily or on most days of the week. Recommend supervised adaptive exercise regimen to patients with disability.
5. Encourage a healthy diet that is low in salt (< 2.3 gms sodium or < 5–6 gms NaCl) and saturated fat and rich in fruits and vegetables. If needed, replace Vitamin B6, B12 and folate deficiencies.

Evidence

1. Cigarette smoking, both active and environmental (passive) is an independent risk factor for stroke for both men and women. Subjects who stopped smoking considerably reduce their risk of stroke.
2. Heavy alcohol drinking increases the risk of stroke. The protective effect of light to moderate alcohol consumption on the risk of ischemic stroke observed among Caucasians is not evident among Asians.
3. Stroke risk can be reduced with regular leisure-time physical activity. Sedentary lifestyle resulting from co-morbidities, disability and emotional barriers puts stroke survivors at high risk for recurrent stroke and cardiovascular disease. Aggressive rehabilitation and structured aerobic exercise program for patients can help increase independence in activities of daily living, optimize functional capacity, and improve multiple cardiovascular risk factors.
4. Increase body weight and abdominal fat are associated with stroke risk. Weight reduction promotes favorable effects in established stroke risk factors such as lowering BP, improving fasting blood sugar and serum lipids and physical endurance.
5. A diet high in sodium and saturated fat is associated with increased risk of stroke probably through its association with stroke risk factors like hypertension and dyslipidemia. Adequate intake of potassium and increased fruit and vegetable consumption are protective.

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CAROTID ENDARTERECTOMY AND ANGIOPLASTY/STENTING

Dr. Peter Rivera

Goal

To reduce the risk of subsequent stroke in patients with carotid stenosis who may benefit most from surgical or endovascular intervention.

Patient Data Needed

1. Side of carotid stenosis ☐ Left ☐ Right
2. Diagnosed with ischemic stroke or TIA ☐ Yes ☐ No
   If stroke or TIA, laterality of stroke or TIA
   ☐ Left, anterior circulation ☐ Right, anterior circulation
   ☐ Posterior circulation or brainstem
3. Modified Rankin Scale (mRS) (*See section on Rehabilitation, PT, OT, and ST*)

Actions

1. Treat all patients with ischemic stroke with best medical management (e.g. anti-platelets, control of risk factors, etc.), whether or not for CEA/CAS.
2. If the stroke is disabling (e.g. mRS is ≥ 4), continue the best medical treatment alone. Reconsider CEA/CAS if the functional status improves later on.
   If the stroke is non-disabling (e.g. mRS is < 4), determine if the stenosis is "symptomatic" by presence of symptoms directly attributable to the carotid artery of concern:
   a. Consider carotid stenosis as "symptomatic" if *all* of the following are fulfilled:
      — Ischemic stroke or TIA
      — Anterior circulation syndrome
      — Carotid stenosis on same side as the ischemic stroke or TIA.
   b. Consider carotid stenosis as "asymptomatic" if *any* of the following:
      — Not ischemic stroke or TIA, e.g. intracerebral hemorrhage
      — Posterior circulation syndrome
      — Carotid stenosis is contralateral to side of ischemic stroke or TIA.
3. Determine the patient's periprocedural morbidity and mortality risk for CEA or CAS. Also take into consideration the surgeon's operative risk record.
4. If carotid stenosis was reported as "severe" or > 50% by non-invasive modality (ultrasound, MRA, or CTA), consider catheter angiography (with endoluminal surface rendering and 3D views if possible) for accurate measurement of degree of diameter stenosis by NASCET criteria. (*See Insert 1 in this section*)
5. Recommend the following and refer to surgeon accordingly:
   a. For symptomatic severe (70–99%) stenosis and periprocedural risk < 6%, recommend CEA.
   b. For symptomatic high moderate (50–69%) stenosis and periprocedural risk < 6%, may recommend CEA but best medical therapy is also an option.

   c. For symptomatic < 50% stenosis, recommend best medical management
   d. For symptomatic occluded carotid, role of EC-IC bypass is still under investigation but may be considered if there is evidence of poor cerebrovascular reserve by TCD vasoreactivity testing or SPECT-acetazolamide scan.
   e. For asymptomatic > 60% stenoses, CEA is controversial. May consider CEA if periprocedural risk < 3%.
6. Do intervention soon after the event when the patient becomes neurologically stable to derive maximal benefit.
7. Consider referral to endovascular interventionist if the patient is high surgical risk with symptomatic severe stenoses. The role of CAS in asymptomatic severe stenosis and moderate stenosis, whether asymptomatic or not, is unclear.

Evidence

1. Patients with symptomatic severe stenosis (70–99%) benefit most from CEA with the two-year stroke risk decreasing from 26% to 7% with absolute risk reduction (ARR) of 19% and number needed to treat (NNT) of 6.
2. Patients with symptomatic high moderate stenosis (50–69%) may benefit from CEA but the benefit is muted (ARR = 5.3%, NNT = 19).
3. There is no additional benefit achieved for patients who are symptomatic with mild and low moderate stenosis (< 50%) who undergo CEA.
4. For asymptomatic severe stenosis, CEA reduces annual risk of stroke from ≈2% to ≈1% per year for the next five years. The periprocedural risk should be < 2.7%.
5. Reported risk from angiography is ≤ 1.2%.
6. CAS currently lacks level-1 evidence but may be an option in severe carotid stenosis with coexisting medical conditions that potentially increase the risk posed by CEA.

Selected Readings

1. North American Symptomatic Carotid Endarterectomy Trial Collaborators. (1991) Beneficial effect of carotid endarterectomy in symptomatic patients with high-grade stenosis. *N Eng J Med* **325**: 445–453.
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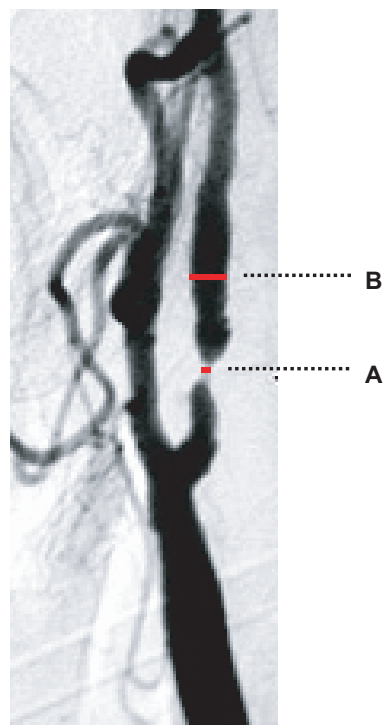
3. Executive Committee for the Asymptomatic Carotid Atherosclerosis Study. (1995) Endarterectomy for asymptomatic carotid stenosis. *JAMA* **273**: 1421–1428.
4. Barnett HJ, Meldrum HE, Eliasziw M, *et al*. (2002) The appropriate use of carotid endarterectomy. *CMAJ* **166**: 1169–1179.
5. Yadav JS, Wholey MH, Kuntz RE, *et al* for the SAPPHIRE Investigators. (2004) Protected carotid-artery stenting versus endarterectomy in high-risk patients. *N Eng J Med* **351**: 1493–1501.
6. MRC Asymptomatic Carotid Surgery Trial (ACST) Collaborative Group. (2004) Prevention of disabling and fatal strokes by successful carotid endarterectomy in patients without recent neurological symptoms. *Lancet* **363**: 1491–1502.
7. Derdeyn CP. (2007) Carotid stenting for asymptomatic carotid stenosis — Trial it. *Stroke* **38** (Suppl 2): 715–720.

CAROTID ENDARTERECTOMY AND ANGIOPLASTY/STENTING

Insert 1. NASCET Method of Computing Degree of Carotid Stenosis

$$\text{Diameter Stenosis} = 1 - \left(\frac{A}{B}\right) \times 100\%$$

where: A = narrowest diameter of residual lumen
B = diameter of ICA lumen distal to the bulb where walls are parallel



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