4th EDITION

the STROKE SOCIETY of the PHILIPPINES

Guidelines for the Prevention, Treatment and Rehabilitation of Brain Attack

A Project of the Stroke Society of the Philippines
Front Panel Painting;
“LIFE”
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Guidelines for the Prevention, Treatment and Rehabilitation of Brain Attack
STROKE: THINK GLOBALLY, ACT LOCALLY

Principles:

1. Stroke is a "brain attack"
   … needing emergency management, including specific treatment and secondary and tertiary prevention.

2. Stroke is an emergency
   … where virtually no allowances for worsening is tolerated.

3. Stroke is treatable
   … optimally, through proven, affordable, culturally acceptable and ethical means.

4. Stroke is preventable
   … in a manner that could be implemented across all levels of society.

The recommendations contained in this document are intended to merely guide practitioners in the prevention, treatment and rehabilitation of patients with stroke. In no way should these recommendations be regarded as absolute rules, since nuances and peculiarities in individual patients, situations or communities may entail differences in specific approaches. The recommendations should supplement, not replace, sound clinical judgments on a case-to-case basis.
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MESSAGE from the FOUNDING PRESIDENT

These *Guidelines for the Prevention, Treatment and Rehabilitation of Brain Attack* is the output of the seven Stroke Congresses on Brain Attack organized by the Stroke Society of the Philippines.

Aware of the many advances in research toward the prevention, treatment and rehabilitation of brain attack, the Stroke Society of the Philippines initiated the First Congress with the theme, “Thinking Globally, Acting Locally,” in October 1999. Since then, six more congresses have tackled the issue of organizing stroke services, and subsequently, intracerebral and subarachnoid hemorrhage.

We worked on the principles that stroke is preventable through ways that may be implemented across all levels of society; that stroke is a “brain attack” needing emergency management where no allowance for worsening is tolerated; and that in the Philippine setting, the treatment should be optimal through proven, affordable, culturally acceptable and ethical means.

With the panel of experts of the Stroke Society of the Philippines consisting of neurologists, internists, neurosurgeons, vascular surgeons and physiatrists, we worked with the practitioners in the field, identified by the Department of Health.

The *Guidelines* were subjected to close scrutiny by experts and practitioners in the field, whose recommendations and comments were embodied in the final output.

We realize that this is not a perfect document, but the Society is proud to present to our public these guidelines, which embody our best efforts to gather the latest, evidence-based data, and the opinion of experts in the Philippines.

We continue to update the *Guidelines* as new knowledge comes to the forefront. We dedicate the *Guidelines* to our patients, students, practitioners and our health workers, that we may in our small way, contribute to the vision of “Health for All” in our beloved country.

JOVEN R. CUANANG, MD
Founding President
Stroke Society of the Philippines
MESSAGE from the PRESIDENT

The three revisions and four editions of these Guidelines for the Prevention, Treatment and Rehabilitation of Brain Attack since 1999 reflect the dynamic evolution of the management of cerebrovascular diseases and its outcomes in the last seven years. The latest data from the most recent trials have been incorporated into the previous guidelines, resulting in these updated Guidelines edition. Furthermore, there is a newly added section: The Establishment and Operation of Stroke Units.

The working group of this fourth edition has strived hard to come out with this new document, a project and testament of the SSP of its commitment to our colleagues and to the fight against brain attack.

ABDIAS V. AQUINO, MD
President
Stroke Society of the Philippines
Guidelines for PRIMARY and SECONDARY PREVENTION of STROKE
STROKE PREVENTION

Preface to the Guidelines on Primary and Secondary Prevention of Stroke

- These practice guidelines provide an overview of the epidemiology and evidences associated with established and modifiable stroke risk factors, followed by recommendations for reducing stroke risk. These revised guidelines reflect current knowledge on primary and secondary stroke prevention.
- The strategy in developing these guidelines was to utilize information from several existing national consensus and evidence-based guidelines to highlight significant associations between a risk factor and stroke and how modifying the risk factor through treatment or lifestyle modification can improve outcome. This knowledge would lead to proper recommendations.
- The Stroke Prevention Writing Group members are active members of the Stroke Society of the Philippines and the Philippine Neurological Association invited by the committee chairs on the basis of each reviewer’s interest, training and previous work in the relevant topic areas. Members then updated the previous editions using recently published local data. The updated working paper was submitted for initial comments by the society members, and later to key opinion leaders and institutions.
- Each major topic first discusses epidemiology (Section A) of a risk factor and its association with stroke, then highlights clinical trials or interventions on the risk factor for preventing stroke (Section B). When evidence is available, a separate subsection (Section B1) discusses primary- and secondary-prevention trials. Section C states the recommendations based on evidences.
- When available, the strength of the recommendation are included and graded according to the American Heart Association (AHA)/American Stroke Association methods of classifying levels of certainty of the treatment effect and the class of evidence (Table 1).
- Recommendations considered the cost-effective treatment of drugs with established efficacy.
- These guidelines concentrated on modifiable risk factors: hypertension, diabetes, atrial fibrillation (AF) and other specific cardiac conditions, dyslipidemia, carotid artery stenosis, peripheral arterial disease, obesity, and lifestyle (exposure to cigarette smoke, excessive alcohol use, physical inactivity and unhealthy diet).
- Other less well-documented or potentially modifiable risk factors are recognized. These include metabolic syndrome, drug abuse, oral contraceptive use, sleep-disordered breathing, migraine headache, hyperhomocysteinemia, hypercoagulability, inflammation and infection. Future editions may highlight these topics.
Because most strokes are cerebral infarcts, these recommendations focus primarily on the prevention of ischemic stroke or transient ischemic attack (TIA).

Although the primary outcome of interest is the prevention of stroke, many recommendations reflect the evidence on the reduction of all vascular outcome after stroke, including stroke, myocardial infarction (MI) and vascular death.

For secondary stroke prevention, the aim is to provide comprehensive and timely evidence-based recommendations on the prevention of ischemic stroke among survivors of ischemic stroke or TIA.

| Class I | Conditions for which there is evidence for and/or general agreement that the procedure or treatment is useful and effective |
| Class II | Conditions for which there is conflicting evidence and/or a divergence of opinion about the usefulness/efficacy of a procedure or treatment |
| IIa | Weight of evidence or opinion is in favor of the procedure or treatment. |
| IIb | Usefulness/efficacy is less well established by evidence or opinion |
| Class III | Conditions for which there is evidence and/or general agreement that the procedure or treatment is not useful/effective and in some cases may be harmful |

Table 1. Classes and Levels of Evidence Used in AHA Recommendations

I. HYPERTENSION

Hypertension awareness, treatment and control remain low. Stroke mortality rates are predicted by the prevalence of hypertension. Yet compelling data show that first stroke can be prevented by blood pressure (BP) control, among others.1

A. Epidemiology: Hypertension is directly related to primary and secondary stroke risk. The higher the BP, the greater is the risk. Hypertension has a local prevalence of 17.4%2 and is the most important modifiable risk factor for stroke. The population attributable risk (PAR) of hypertension for stroke is high at around 25%.3 Hypertensive people are three to four times more likely to have a stroke than non-hypertensive people. Furthermore, both systolic and diastolic hypertensions are risk factors.
B. Risk Modification: Treatment of hypertension substantially reduces the risk of stroke. All classes of antihypertensive drugs are effective for BP control. A meta-analysis shows BP lowering confers a 30% to 40% stroke risk reduction. A 10 to 12 mmHg SBP reduction and a 5 to 6 mmHg DBP reduction confers relative reductions in stroke risk of 38%. The treatment of isolated systolic hypertension in the elderly decreases the risk for stroke by 36%. Furthermore, small BP reductions in a population may lead to substantial reductions in stroke risk: it is estimated that a population strategy to reduce systolic BP (SBP) by 2 mmHg will reduce stroke mortality by 6%. A 3-mmHg SBP reduction reduces risk by 8%. A 5-mmHg reduction reduces risk by 14%. Similar to other cardiovascular disorders, stroke reduction is progressive as BP is reduced to at least 115/75 mmHg.

B1. Primary Stroke Prevention

There is strong evidence that the control of high BP contributes to the prevention of stroke. The choice of antihypertensive agents should be individualized. BP reduction is generally more important than the specific agent used to achieve this goal.

Hypertension remains undertreated in the community, and programs to improve treatment compliance need to be developed and supported. The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC 7) provides a comprehensive, evidence based approach to the classification and treatment of hypertension (Table 2).

Table 2. Classification and management of BP for adults*

<table>
<thead>
<tr>
<th>BP Classification</th>
<th>SBP* mmHg</th>
<th>DBP* mmHg</th>
<th>Lifestyle Modification</th>
<th>Initial drug therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Without Compelling Indication</td>
</tr>
<tr>
<td>Normal</td>
<td>&lt;120</td>
<td>and</td>
<td>Encourage</td>
<td>No antihypertensive drug indicated.</td>
</tr>
<tr>
<td></td>
<td>&lt;80</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Prehypertension</td>
<td>120-139</td>
<td>or 80-89</td>
<td>Yes</td>
<td>Thiazide-type diuretics for most. May consider ACEI, ARB, BB, CCB, or combination.</td>
</tr>
<tr>
<td>Stage 1 Hypertension</td>
<td>140-159</td>
<td>or 90-99</td>
<td>Yes</td>
<td></td>
</tr>
</tbody>
</table>
Stage 2 Hypertension \( \geq 160 \text{ or } \geq 100 \) Yes Two-drug combination for most† (usually thiazide-type diuretic and ACEI or ARB or BB or CCB).

DBP, diastolic blood pressure; SBP, systolic blood pressure.

Drug abbreviations: ACEI, angiotensin-converting enzyme inhibitor; ARB, angiotensin-receptor blocker; BB, beta-blocker; CCB, calcium-channel blocker.

*Treatment determined by highest BP category.
†Initial combined therapy should be used cautiously in those at risk for orthostatic hypotension.
‡Treat patients with chronic kidney disease or diabetes to BP goal of <80 mmHg.

B2. Secondary Stroke Prevention

The overall decrease in stroke is related to the degree of BP lowering achieved. Meta-analyses of randomized controlled trials (RCTs) confirm an approximate 30% to 40% stroke reduction with BP lowering. Furthermore there is a continuous association of both SBP and DBP with the risk of ischemic stroke.

The JNC 7 stresses the importance of lifestyle modifications in the overall management of hypertension. SBP reductions have been associated with weight loss; diet rich in fruits, vegetables and low-fat dairy products; regular aerobic physical activity; and limited alcohol consumption.

Data on the relative benefits of specific antihypertensive regimens for secondary stroke prevention are largely lacking. A meta-analysis showed a significant reduction in recurrent stroke with diuretics and combined diuretics and angiotensin-converting enzyme (ACE) inhibitors (ACEIs), but not with beta-blockers (BBs) or ACEIs alone. Whether a particular class of antihypertensive drug or a particular drug within a given class offers an advantage in patients after ischemic stroke remains uncertain.

With regard to stroke risk reduction, there may be beneficial non-BP-lowering properties of certain classes of BP-lowering agents, particularly from ACEIs and angiotensin-receptor blockers (ARBs). Among hypertensive diabetics, the use of ACEIs or ARBs reduces the risk of major vascular events and stroke by 24%.

C. Recommendation:

C1. For primary prevention

Regular screening for hypertension (at least every 2 years in most adults and more frequently in minority populations and the elderly) and appropriate management (Class I, Level A), including dietary changes, lifestyle modification and pharmacological therapy as summarized in JNC 7, are recommended.

C2. For secondary prevention

Antihypertensive treatment is recommended for both prevention of recurrent stroke and of other vascular events in patients who have had an
ischemic stroke or TIA and are beyond the hyperacute period (Class I-A). Because this benefit extends to people with or without a history of hypertension, this recommendation should be considered for all ischemic stroke and TIA patients (Class IIa-B). The absolute target BP level and reduction are uncertain and should be individualized, but benefit has been associated with an average reduction of 10/5 mmHg, and normal BP levels have been defined as <120/80 mmHg by JNC 7 (Class IIa-B). BP should be adequately controlled in patients with hypertension. Physicians should check the BP of all patients at every visit. Patients with hypertension should be advised to monitor their BP at home.

Several lifestyle modifications have been associated with BP reductions and should be included as part of a comprehensive antihypertensive therapy (Class IIb-C).

The optimal drug regimen remains uncertain; however, the available data support the use of diuretics and the combination of diuretics and an ACEI (Class I-A). The choice of specific drugs and targets should be individualized on the basis of reviewed data and consideration of specific patient characteristics (e.g., extracranial cerebrovascular occlusive disease, renal impairment, cardiac disease or diabetes) (Class IIb-C). The Stroke Society of the Philippines supports the guidelines set forth by the Philippine Society of Hypertension and the JNC 7.

Bibliography

II. TRANSIENT ISCHEMIC ATTACK

Conventionally, a person is diagnosed with stroke if neurological symptoms persist more than 24 hours; otherwise, a focal neurological deficit lasting <24 hours was defined as a TIA. However, with modern brain imaging, infarctions can be detected even in patients with brief symptoms. The most updated definition of stroke used by clinical trials is either symptoms lasting >24 hours or an acute clinically relevant brain lesion on imaging in patients with rapidly vanishing symptoms. The proposed new definition of TIA is a “brief episode of neurological dysfunction caused by a focal disturbance of brain or retinal ischemia, with clinical symptoms typically lasting less than 1 hour, and without evidence of infarction.”

A. Epidemiology: Although TIA is most correctly considered a manifestation of cerebrovascular disease and not a stroke risk factor, it is an important predictor of future strokes. Reported 90-day stroke risk associated with TIA reaches 10.5%, and the highest risk is apparent in the first week. The risk is 4% to 8% in the first month, 12% to 13% in the first year, and 24% to 29% in 5 years. Patients with hemispheric TIA and carotid stenosis of more than 70% have a particularly poor prognosis, with a stroke rate of >40% in 2 years.

B. Risk Modification: The distinction between TIA and ischemic stroke has become less important in recent years because many preventive approaches are applicable in both. As the risk factors for ischemic stroke and TIA are the same, the evidences supporting that modification of a particular risk factor are found in the corresponding sections of these guidelines. Many clinical trials have demonstrated that antiplatelets reduce stroke risk after TIA or minor stroke by 18% to 41%. RCTs on antiplatelet drugs that reduce stroke, either alone or as part of a composite of vascular outcomes, include aspirin, dipyridamole, aspirin-dipyridamole combination, ticlopidine, cilostazol and clopidogrel. Although some studies limited subjects to those with minor strokes instead of TIA, it is reasonable to consider a similar prophylactic effect in TIA patients.

C. Recommendations: Efforts to increase public awareness and that of health workers regarding TIA and its significance should be maximized. Evaluation of TIA should be attempted to define cause and determine prognosis and treatment. TIA patients should be expeditiously evaluated for vascular and cardiac risk factors for stroke. Hypertension, hyperlipidemia, diabetes, carotid stenosis and other modifiable risk factors should be treated, as outlined in these guidelines. The cost and benefit of a drug should be considered when choosing an antiplatelet agent. Aspirin is the first choice unless contraindicated. Patients who developed stroke recurrence while on aspirin, or those who cannot tolerate or have contraindications to aspirin may be given clopidogrel,
combined aspirin and dipyridamole, cilostazol, or other antiplatelets with RCT evidence of benefit. Aspirin is not recommended for primary stroke prevention, as this has no evidence especially among men.

While there is evidence of benefit of combined aspirin and clopidogrel in coronary heart disease or post-revascularization patients, this combination is not recommended for stroke prevention.

Bibliography:

APPENDIX OF TIA MANAGEMENT FOR STROKE PREVENTION

Since the 3rd edition of these guidelines, there have been several important trials related to antiplatelets and anticoagulants. The 2006 recommendations of the American Heart Association and the American Stroke Association are outlined below:

ASA/AHA Recommendations for Antithrombotic Therapy for Noncardioembolic Stroke or TIA (Oral Anticoagulant and Antiplatelet Therapies)

- For patients with noncardioembolic ischemic stroke or TIA, antiplatelet agents rather than oral anticoagulation are recommended to reduce the risk of recurrent stroke and other cardiovascular events (Class I-A).
- Aspirin (50 to 325 mg/day), the combination of aspirin and extended-release dipyridamole, and clopidogrel are all acceptable options for initial therapy (Class IIa-A).
- Compared with aspirin alone, both the combination of aspirin and extended-release dipyridamole and clopidogrel are safe.
- The combination of aspirin and extended-release dipyridamole is suggested over aspirin alone (Class IIa-A).
- Clopidogrel may be considered over aspirin alone on the basis of direct-comparison trials. Insufficient data are available to make evidence-based
recommendations with regard to choices between antiplatelet options other than aspirin. Selection of an antiplatelet agent should be individualized based on patient risk factor profiles, tolerance, and other clinical characteristics (Class IIb-B).

- Addition of aspirin to clopidogrel increases the risk of hemorrhage and is not routinely recommended for ischemic stroke or TIA patients (Class III-A).
- For patients allergic to aspirin, clopidogrel is reasonable (Class IIa-B).
- For patients who have an ischemic cerebrovascular event while taking aspirin, there is no evidence that increasing the dose of aspirin provides additional benefit. Although alternative antiplatelet agents are often considered for noncardioembolic patients, no single agent or combination has been well studied in patients who have had an event while receiving aspirin.


III. DIABETES MELLITUS

A. Epidemiology: Diabetes mellitus (DM) is a serious public health problem in the Philippines. Estimated to affect 8% of the adult population worldwide, the local prevalence of DM (fasting blood sugar >125 mg/dL) according to the 2003 National Nutrition Health Survey is 4.6% (4.1% in males, 5% in females) while impaired fasting glucose is 3.2% (similar rates for males and females).

People with type 2 DM have both an increased risk of atherosclerosis and increased prevalence of atherogenic risk factors (i.e., hypertension, obesity and abnormal blood lipids). DM is a definite risk factor for stroke. Case-control studies of stroke patients and prospective epidemiological studies have confirmed an independent effect of DM on ischemic stroke, increasing risk by 1.8- to nearly 6-fold. DM is frequently encountered in stroke care, being present in 15% to 33% of patients with ischemic stroke. The local RIFASAF case-control study showed a 1.6-fold higher risk for stroke among those with DM. However, data supporting DM as a risk factor for recurrent stroke are sparse.

B. Risk Modification

B1. Primary Stroke Prevention

DM has microvascular and macrovascular complications. Intensive DM therapy delays the onset and slows down the progression of microvascular complications, such as retinopathy, nephropathy and neuropathy, but not macrovascular complications. Systematic review of RCTs on intensive insulin therapy (IIT) showed that IIT can decrease the occurrence of macrovascular events by up to 42%, including stroke, myocardial infarction (MI), angina and
claudication, among patients with type 1 DM. Sub-studies on diabetic patients included in drug trials show that the use of ACEIs and ARBs can reduce the combined outcome of MI, stroke and cardiovascular death by 21% to 33%. Similarly, ACEIs and ARBs decrease new-onset diabetes.

Primary stroke prevention guidelines have emphasized more rigorous BP control (target BP <130/80 mmHg) among both type 1 and type 2 diabetics. The American Diabetes Association (ADA) now recommends that all patients with diabetes and hypertension be treated with a regimen that includes either an ACEI or ARB.

Hyperlipidemia is a common comorbidity of diabetes. For any given cholesterol level, patients with diabetes have a greater frequency of cardiovascular events for which aggressive therapy of diabetic dyslipidemia is indicated, aiming for LDL<100 mg/dL or even up to 70 mg/dL among very high-risk groups. The use of statins among DM patients can reduce vascular events, including stroke. Addition of a statin for DM patients at high risk reduces stroke risk by 24%, and in those with one additional risk factor by 48%.

Among high-risk patients with type 2 DM, the thiazolidinedione, pioglitazone, seems to reduce the composite of all cause mortality, non-fatal stroke and MI, as well as reduce the need for insulin treatment.

**B2. Secondary Stroke Prevention**

Most of the available data on stroke prevention in DM patients pertain to primary prevention. However, glycemic control is consistently recommended in multiple guidelines of both primary and secondary prevention of stroke and cardiovascular disease. Among patients with type 2 DM with or without vascular events, such as stroke, multifactorial approaches involving intensive treatments to control hyperglycemia, hypertension, dyslipidemia and microalbuminuria reduce the risk of cardiovascular events. These approaches included behavioral measures and the use of a statin, ACEI, ARB and antiplatelet drugs, as appropriate. The beneficial role of antiplatelets among stroke patients with or without diabetes has been proven in many trials.

**C. Recommendation:** A long-term, intensified DM control, which includes behavioral and pharmacological modification to prevent microvascular and macrovascular complications, is recommended.

Rigorous BP and lipid control should be considered in patients with diabetes (Class IIa-B). A target BP of <130/80 mmHg (Class I-A) is recommended as part of a comprehensive risk-reduction program. An ACEI or ARB is preferred for DM patients. Adults with DM, especially those with additional risk factors, should be treated with a statin to lower the risk of a first stroke (Class I-A).

Among diabetic patients with TIA or stroke, glucose control is recommended to near-normoglycemic levels to reduce microvascular complications (Class I-A) and possibly macrovascular complications (Class IIb-B). The goal for hemoglobin A1c should be 7% (Class IIa-B).
**Bibliography**


**IV. ATRIAL FIBRILLATION**

**A. Epidemiology:** Non-valvular atrial fibrillation (NVAF) alone is associated with a three- to four-fold increase in stroke risk after adjustment for other vascular risk factors as shown in the Table 3.1
Table 3: Epidemiology of NVAF by Age Group\(^1-6\)

<table>
<thead>
<tr>
<th>Age group</th>
<th>Prevalence</th>
<th>PAR</th>
<th>RR</th>
<th>Risk reduction with treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>50-59 y</td>
<td>0.5%</td>
<td>1.5%</td>
<td>4.0</td>
<td>Adjusted-dose warfarin vs control: 62% (CI 48%-72%); 6 trials, n=2,900.</td>
</tr>
<tr>
<td>60-69 y</td>
<td>1.8%</td>
<td>2.8%</td>
<td>2.6</td>
<td>Aspirin vs placebo: 22% (CI 2%-38%); 6 trials, n=3,119.</td>
</tr>
<tr>
<td>70-79 y</td>
<td>4.8%</td>
<td>9.9%</td>
<td>3.3</td>
<td>Adjusted-dose warfarin vs aspirin: 45% (CI 29%-57%); 6 trials, n=4,025.</td>
</tr>
<tr>
<td>80-89 y</td>
<td>8.8%</td>
<td>23.5%</td>
<td>4.5</td>
<td></td>
</tr>
</tbody>
</table>

PAR, population-attributable risk; RR, relative risk.

The prevalence of ischemic stroke for those with NVAF but without prior TIA or stroke is 2% to 4% per year.\(^3,4\)

Table 3 also shows that the prevalence of NVAF increases with age. The mean age of NVAF patients is 75 years.\(^1,5\) Estimates of attributable risk reveal that about one quarter of strokes in the very elderly (≥80 years old) are due to NVAF.\(^1\) NVAF is also associated with increased mortality after adjustment for other vascular risk factors partly because resultant strokes are large and disabling.\(^7\)

**B. Risk Modification**

RCTs have established the value of antithrombotic therapies, particularly warfarin and aspirin, in reducing stroke risk in patients with NVAF (Table 3). Adjusted-dose warfarin reduces stroke by 45% compared with aspirin.\(^4\)

The absolute risk reduction from warfarin (from 4.5% for control down to 1.4% with treatment) means the prevention of 31 ischemic strokes each year for every 1,000 patients treated. Overall, warfarin is relatively safe, with a 1.3% annual rate of major bleeding compared with 1% for placebo or aspirin. The optimal international normalized ratio (INR) for stroke prevention in AF patients appears to be 2.0 to 3.0.

The absolute risk of stroke varies 20-fold among AF patients, according to age and associated vascular diseases. Several stroke risk-stratification schemes have been developed and validated.\(^5-10\) The 2001 American College of Cardiology (ACC)/AHA/European Society of Cardiology (ESC) guidelines recommended anticoagulation for AF patients >60 years old and have a history of hypertension, DM, coronary artery disease (CAD), impaired left ventricular (LV) systolic function, heart failure or prior thromboembolism, and those >75 years old.\(^11\) This stratification scheme had not been prospectively validated even though the individual factors are validated.

Since publication of the 2001 ACC/AHA/ESC guideline, the so-called CHADS\(_2\) stratification scheme has been proposed and validated.\(^8\) CHADS\(_2\) stands for Congestive heart failure (CHF), Hypertension, Age >75 years, DM, and prior Stroke or TIA. The CHADS\(_2\) score was derived from independent predictors of stroke risk in patients with NVAF as shown on Table 4. The score
gives 1 point each for CHF, hypertension, age >75 years, and DM; and 2 points for prior stroke or TIA. The score was validated in a large cohort study and in clinical trials.\textsuperscript{3,8,9} In this scheme, stroke risk of NVAF patients was reliably predicted as low (usually comprises half of patients), moderate (25% of patients) or high (25%).\textsuperscript{3} The validation study shows that patients with prior stroke or TIA and no other risk factors average 10.8 strokes per 100 patient-years, and that in the Stroke Prevention in Atrial Fibrillation (SPAF),\textsuperscript{12} patients with prior stroke or TIA without other risk factors had a stroke rate of 5.9%/year. Therefore, patients with stroke or TIA in the setting of AF should be treated with warfarin unless contraindicated.

LV dysfunction, left atrial size, mitral annular calcification (MAC), spontaneous echo contrast, and left atrial thrombus by echocardiography also predict increased thromboembolic risk.

Anticoagulation is particularly underused in elderly patients with NVAF.\textsuperscript{13} Although the attributable risk of stroke associated with AF increases with age,\textsuperscript{2} elderly (≥75 years old) AF patients have about twice the risk of serious bleeding complications during anticoagulation compared with younger patients. Nevertheless, anticoagulation is still warranted if their risk of ischemic stroke without warfarin is greater than their risk of bleeding. In addition to age, poorly controlled hypertension and concomitant aspirin or non-steroidal anti-inflammatory drug use confer higher bleeding risk during anticoagulation. Therefore, age alone is not a contraindication to anticoagulation of high-risk AF patients.

No data are available to address the question of when to initiate oral anticoagulation in an AF patient after a stroke or TIA. In general, initiation is recommended within 2 weeks of an ischemic stroke or TIA; however, for patients with large infarcts or uncontrolled hypertension, further delays may be appropriate. For AF patients with ischemic stroke or TIA despite therapeutic anticoagulation, no data indicate that either increasing the intensity of anticoagulation or adding an antiplatelet agent provides additional protection from future ischemic events.

Table 4: NVAF Risk Stratification and Treatment Recommendations: Risk Stratification by CHADS\textsubscript{2} Scheme

<table>
<thead>
<tr>
<th>CHADS\textsubscript{2} Score*</th>
<th>Risk Level</th>
<th>Stroke Rate (%/year)</th>
<th>Treatment Recommendations</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Low</td>
<td>1.0</td>
<td>Aspirin (75-325 mg/day)</td>
</tr>
<tr>
<td>1</td>
<td>Low-moderate</td>
<td>1.5</td>
<td>Warfarin INR 2.0-3.0 or aspirin (75-325 mg/day)†</td>
</tr>
<tr>
<td>2</td>
<td>Moderate</td>
<td>2.5</td>
<td>Warfarin INR 2.0-3.0†</td>
</tr>
<tr>
<td>3</td>
<td>High</td>
<td>5.0</td>
<td>Warfarin INR 2–3‡</td>
</tr>
<tr>
<td>≥4</td>
<td>Very high</td>
<td>&gt;7.0</td>
<td></td>
</tr>
</tbody>
</table>
CHF, hypertension, age >75 years, or diabetes=1 point. Stroke or TIA=2 points. All NVAF patients with prior stroke or TIA should be considered high risk and treated with anticoagulants; the CHADS2 scheme should be applied for primary prevention.

†Consider patient preferences, bleeding risk and access to good INR monitoring. For those with score=1, the number needed to treat with warfarin to prevent one stroke over 1 year is around 100. Excellent anticoagulation control is essential to achieve this benefit.

‡If patient is >75 years old, an INR target of 1.6 - 2.5 is recommended by some experts.

C. Recommendations: Antithrombotic therapy with warfarin or aspirin is recommended to prevent stroke in NVAF patients according to assessment of absolute stroke risk, estimated bleeding risk, patient preferences, and access to high-quality anticoagulation monitoring. For risk stratification and treatment recommendations, the CHADS2 scheme (Table 4) should be followed (Class I-A).

Warfarin (INR=2.0-3.0) is recommended for high-risk (>4% annual stroke risk) AF patients provided there are no clinically significant contraindications to oral anticoagulants (Class I-A).

For patients with ischemic stroke or TIA with persistent or paroxysmal (intermittent) AF, anticoagulation with adjusted-dose warfarin (INR=2.5 [2.0-3.0]) is recommended (Class I-A). In patients unable to take oral anticoagulants, aspirin 325 mg/day is recommended (Class I-A).

Bibliography
V. ACUTE MYOCARDIAL INFARCTION (WITH LEFT VENTRICULAR THROMBUS) AND CARDIOMYOPATHY

Acute MI with LV Thrombus

A. Epidemiology: Stroke or systemic embolism are less common among uncomplicated MI patients but can occur in up to 12% of patients with acute MI complicated by an LV thrombus. Acute MI is associated with up to 5% risk of ischemic stroke within 2 weeks. The rate is higher in those with anterior than inferior infarcts and may reach 20% in those with large anteroapical infarcts.\(^1\) The incidence of embolism is highest during the period of active thrombus formation in the first 1 to 3 months, yet the embolic risk remains substantial even beyond the acute phase in patients with persistent myocardial dysfunction, CHF or AF.

B. Risk Modification: An overview of trials on anticoagulation after MI has shown that INR of 2.5 to 4.8 may increase hemorrhagic stroke 10-fold, whereas INR below 2.0 may not be effective in preventing ischemic stroke. An INR range of 2.0 to 3.0 with a target of 2.5 is recommended. Two studies of MI patients (n=4,618) found that warfarin (INR=2.8-4.8) reduced ischemic stroke risk by 55% and 40%, respectively, compared with placebo, over 37 months.\(^2,3\)

Statins for secondary prevention in patients with established atherosclerosis (CAD, thrombotic cerebral stroke, peripheral arterial disease or prior revascularization) significantly reduced overall risk of stroke, total mortality, cardiovascular death, MI and revascularization when total cholesterol is \(\geq\)190 mg/dL or LDL is \(\geq\)100 mg/dL. Stroke Prevention by Aggressive Reduction in Cholesterol Levels study (SPARCL) showed that patients previously documented to have stroke or TIA and no history of coronary heart disease benefited from atorvastatin 80 mg in reducing fatal stroke and TIA.\(^4\)

C. Recommendations: Oral anticoagulation for MI patients is recommended if they have one or more of the following conditions: persistent AF, decreased LV function (e.g., ejection fraction [EF] 28%) or when LV thrombi are detected within several months after MI. Antiplatelets is not recommended to prevent a first stroke after an MI.

For patients with ischemic stroke or TIA due to acute MI in whom LV mural thrombus was identified by echocardiography or another form of cardiac imaging, oral anticoagulation is reasonable, aiming for an INR of 2.0 to 3.0 for at least 3
months and up to 1 year (Class IIa-B). Aspirin should be used concurrently for ischemic CAD during oral anticoagulant therapy in doses up to 160 mg/d (Class IIa-A). For patients with established atherosclerosis and total cholesterol \( \geq 190 \) mg/dL or LDL \( \geq 100 \) mg/dL, statins are recommended. Furthermore, adherence to the 2005 Clinical Practice Guidelines for the Management of Dyslipidemia in the Philippines is recommended.\(^5\)

For patients with stroke or TIA but without coronary heart disease, statin therapy should be administered to prevent recurrence of stroke and TIA.

**Cardiomyopathy**

**A. Epidemiology:** Two large studies found that the incidence of stroke is inversely proportional to EF.\(^6,7\) In the Survival and Ventricular Enlargement (SAVE) study, patients with EF of 29% to 35% (mean=32%) had a 0.8% stroke rate per year, whereas the yearly rate in those with EF \( \leq 28\% \) (mean=23%) was 1.7%. There was an 18% incremental increase in stroke risk for every 5% decline in EF. A retrospective analysis of data from the Studies of Left Ventricular Dysfunction (SOLVD) trial, which excluded patients with AF, found a 58% increase in risk of thromboembolic events for every 10% decrease in EF among women \((p=0.01)\) but no increased risk in men.\(^8\)

In patients with non-ischemic dilated cardiomyopathy, the rate of stroke appears similar to that associated with cardiomyopathy resulting from ischemic heart disease.

**B. Risk Modification:** Warfarin is sometimes prescribed to prevent cardioembolic events in patients with cardiomyopathy. However, no RCT has demonstrated the efficacy of anticoagulation. Considerable controversy surrounds the use of warfarin in patients with cardiac failure or reduced LVEF.\(^9,10\)

Warfarin appears to reduce the risk of ischemic stroke in patients with non-ischemic cardiomyopathy and in those with ischemic heart disease.\(^11\) Aspirin reduces the stroke rate by around 20%.\(^12\) Potential antiplatelet therapies used to prevent recurrent stroke include aspirin (50 to 325 mg/day), combined aspirin and extended-release dipyridamole (25mg/200 mg twice daily), and clopidogrel (75 mg daily).

**C. Recommendation:** For patients with ischemic stroke or TIA who have dilated cardiomyopathy, either warfarin (INR=2.0-3.0) or antiplatelet therapy may be considered for prevention of recurrent events (Class IIb-C).

**Bibliography**


VI. VALVULAR HEART DISEASE and PROSTHETIC HEART VALVES

A. Epidemiology

Annual rates of systemic thromboembolism (TE) in different valvular diseases are shown in Table 5:

Table 5: Incidence of systemic thromboembolism in valvular heart disease

<table>
<thead>
<tr>
<th></th>
<th>Alone (No AF)</th>
<th>With AF (vs without AF)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Prosthetic valve</td>
<td>20%</td>
<td>Increased</td>
</tr>
<tr>
<td>2. Rheumatic mitral regurgitation</td>
<td>7.7%</td>
<td>22%</td>
</tr>
<tr>
<td>3. Rheumatic mitral stenosis</td>
<td>1.5%-4.0%</td>
<td>Increased by 7-18x</td>
</tr>
<tr>
<td>4. Mitral valve prolapse</td>
<td>&lt;2%</td>
<td>Increased</td>
</tr>
<tr>
<td>5. Aortic valve</td>
<td>Not increased</td>
<td>Increased</td>
</tr>
</tbody>
</table>

Patients with paroxysmal or persistent AF and valvular heart diseases such as mitral stenosis are at highest risk for future embolic events.

B. Risk Modification: Antithrombotic therapy can reduce the likelihood of stroke and systemic embolism in patients with valvular heart disease. The rate of TE in patients with mechanical heart valves is 4.4 per 100 patient-years without antithrombotic therapy; 2.2 per 100 patient-years with antiplatelet drugs; and 1 per 100 patient-years with warfarin. With or without AF, all patients with mechanical heart valves require anticoagulation with target anticoagulation levels varying according to type and position of the valve, and the presence of other risk factors.
factors. The risk of TE in patients with native valvular heart diseases or mechanical or biological heart-valve prostheses must be balanced with the risk of bleeding. Nevertheless, because the frequency and permanency of consequences of TE events are usually greater than the outcome of hemorrhagic complications, anticoagulant therapy is generally recommended, particularly when associated with AF.\textsuperscript{5}

**Rheumatic Mitral Valve Disease**

**a. Epidemiology:** The annual rate of TE in rheumatic mitral regurgitation (MR) and stenosis (MS) without AF are 7.7% and 1.5% to 4% respectively. The presence of AF increases TE by 22% in MR patients and by seven- to 18-fold in MS patients.

Recurrent embolism occurs in 30% to 65% of patients with rheumatic mitral valve disease who have a history of a previous embolic event.\textsuperscript{6-8} Between 60% to 65% of these recurrences develop within the first year, most within 6 months.\textsuperscript{6,7}

**b. Risk Modification:** Although not evaluated in randomized trials, multiple observational studies have reported that long-term anticoagulant therapy effectively reduces the risk of systemic embolism in patients with rheumatic mitral valve disease.\textsuperscript{3,9} Long-term anticoagulant therapy in patients with MS who had left atrial thrombus identified by transesophageal echocardiography can result in the disappearance of the thrombus.\textsuperscript{10}

**c. Recommendations:** For patients with rheumatic mitral valve disease or prosthetic valve without prior stroke or TIA, oral anticoagulation with coumadin is recommended unless contraindicated.

For patients with ischemic stroke or TIA who have rheumatic mitral valve disease, whether or not AF is present, long-term warfarin therapy is reasonable, with a target INR of 2.5 (range; 2.0-3.0) (Class IIa-C).

Antiplatelet agents should not routinely be added to warfarin to avoid the additional bleeding risk (Class III-C). Aspirin 80 mg/day is suggested for patients with ischemic stroke or TIA with rheumatic mitral valve disease, whether or not AF is present, who have recurrent embolism while receiving warfarin (Class IIa-C).

**Mitral Valve Prolapse**

**a. Epidemiology:** Mitral valve prolapse (MVP) is the most common form of valve disease in adults.\textsuperscript{11} Thromboembolic phenomena have been reported in patients with mitral valve prolapse in whom no other source could be found.\textsuperscript{12-16} The annual rate of TE in those with MVP and no AF is less than 2%. AF increases TE risk.
b. Risk Modification: No randomized trials have addressed the efficacy of selected antithrombotic therapies for this subgroup of stroke or TIA patients. The evidence on the efficacy of antiplatelet agents for general stroke and TIA patients was used to reach these recommendations.

c. Recommendation: For patients with MVP who had ischemic stroke or TIA, antiplatelet therapy is reasonable (Class IIa-C).

Mitral Annular Calcification
a. Epidemiology: Although the incidence of systemic and cerebral embolism is not clear, thrombus has been found on heavily calcified annular tissue upon autopsy.17-22

b. Risk Modification: From observations and in the absence of randomized trials, anticoagulant therapy may be considered for patients with MAC and a history of TE.

c. Recommendations: For patients with ischemic stroke or TIA in whom MAC is not documented to be calcific, antiplatelet therapy may be considered (Class IIb-C).

For patients with MR due to MAC and without AF, antiplatelet or warfarin therapy may be considered (Class IIb-C).

Aortic Valve Disease
a. Epidemiology: Clinically detectable systemic embolism in isolated aortic valve disease is increasingly recognized because of microthrombi or calcific emboli.23 In an autopsy study of 165 patients with calcific aortic stenosis, systemic embolism was found in 31 patients (19%). In the absence of associated mitral valve disease or AF, systemic embolism in patients with aortic valve disease is uncommon. TE increases in patients with aortic valve disease.

b. Risk Modification: No randomized trials on selected patients with stroke and aortic valve disease exist.

c. Recommendation: For patients with ischemic stroke or TIA and aortic valve disease but no AF, antiplatelet therapy may be considered (Class IIb-C)

Prosthetic Heart Valves
b. Epidemiology: The annual percentage of occurrence of systemic TE in those with prosthetic heart valves is 20%. The risk increases with AF.

c. Risk Modification: A variety of mechanical heart valve prostheses are available for clinical use, all of which require antithrombotic prophylaxis. The most convincing evidence that oral anticoagulants are effective in patients with prosthetic heart valves comes from patients randomized to
treatment for 6 months with either warfarin in uncertain intensity or one of two aspirin-containing platelet-inhibitor regimens. In two randomized studies, concurrent treatment with dipyridamole and warfarin reduced the incidence of systemic embolism, and the combination of dipyridamole (450 mg/day) and aspirin (3.0 g/d) reduced the incidence of TE in patients with prosthetic heart valves. A randomized study of aspirin 1.0 g/day plus warfarin versus warfarin alone in 148 patients with prosthetic heart valves found a significant reduction of embolism in the aspirin-treated group. Another trial showed that the addition of aspirin 100 mg/day to warfarin (INR=3.0-4.5) improved efficacy compared with warfarin alone.

The ESC guidelines recommend anticoagulant intensity in proportion to the TE risk associated with specific types of prosthetic heart valves. For first-generation valves, an INR of 3.0 to 4.5 was recommended; an INR of 3.0 to 3.5 was recommended for second-generation valves in the mitral position, whereas an INR of 2.5 to 3.0 was advised for second-generation valves in the aortic position. The 2004 American College of Chest Physicians recommended an INR of 2.5 to 3.5 for patients with mechanical prosthetic valves, and 2.0 to 3.0 for those with bioprosthetic valves and low-risk patients with bileaflet mechanical valves (such as the St. Jude Medical device) in the aortic position. Similar guidelines have been promulgated conjointly by the ACC and the AHA.

**d. Recommendations:** For patients who have modern mechanical prosthetic heart valves, with or without ischemic stroke or TIA, oral anticoagulants should be administered to an INR target of 3.0 (range; 2.5-3.5) (Class I-B).

For patients with mechanical prosthetic heart valves who had an ischemic stroke or systemic embolism despite adequate therapy with oral anticoagulants, aspirin 75 to 100 mg/day in addition to oral anticoagulants and maintenance of the INR at 3.0 (range; 2.5-3.5) are reasonable (Class IIa-B).

For patients with ischemic stroke or TIA who have bioprosthetic heart valves with no other source of thromboembolism, anticoagulation with warfarin (INR=2.0-3.0) may be considered (Class IIb-C).

**Table 6. Summary of Recommendations for Patients With Cardioembolic Stroke or TIA**

<table>
<thead>
<tr>
<th>Risk Factor</th>
<th>Recommendation</th>
<th>Class/Level of Evidence</th>
</tr>
</thead>
<tbody>
<tr>
<td>AF</td>
<td>For patients with ischemic stroke or TIA with persistent or paroxysmal (intermittent) AF, anticoagulation with adjusted-dose warfarin (target INR=2.5 [2.0-3.0]) should be administered</td>
<td>Class I-A</td>
</tr>
<tr>
<td></td>
<td>In patients unable to take oral anticoagulants, aspirin 325 mg/day is recommended.</td>
<td>Class I-A</td>
</tr>
</tbody>
</table>
### Acute MI and LV thrombus
For patients with ischemic stroke caused by acute MI with LV mural thrombus identified by echocardiography or another form of cardiac imaging, oral anticoagulation is reasonable (INR=2.0-3.0 for at least 3 months up to 1 year).

<table>
<thead>
<tr>
<th>Aspirin up to 160 mg/day (preferably enteric-coated) should be used concurrently for patients with ischemic CAD during oral anticoagulant therapy.</th>
</tr>
</thead>
</table>

### Cardiomyopathy
For patients with ischemic stroke or TIA who have dilated cardiomyopathy, either warfarin (INR=2.0-3.0) or antiplatelet therapy may be considered to prevent recurrent events.

### MVP
For patients with MVP who have ischemic stroke or TIA, long-term antiplatelet therapy is reasonable.

### MAC
For patients with ischemic stroke or TIA and MAC not documented to be calcific, antiplatelet therapy may be considered.

<table>
<thead>
<tr>
<th>Among patients with MR due to MAC, without AF, antiplatelet or warfarin therapy may be considered.</th>
</tr>
</thead>
</table>

### Aortic valve disease
For patients with ischemic stroke or TIA and aortic valve disease who do not have AF, antiplatelet therapy may be considered.

### Prosthetic heart valves
For patients with ischemic stroke or TIA who have modern mechanical prosthetic heart valves, oral anticoagulants are recommended, with an INR target of 3.0 (range; 2.5-3.5).

<table>
<thead>
<tr>
<th>For patients with mechanical prosthetic heart valves who had an ischemic stroke or systemic embolism despite adequate therapy with oral anticoagulants, aspirin 75 to 100 mg/day in addition to oral anticoagulants maintained at INR of 3.0 (range; 2.5-3.5) is reasonable.</th>
</tr>
</thead>
</table>

| For patients with ischemic stroke or TIA who have bioprosthetic heart valves with no other source of TE, anticoagulation with warfarin (INR=2.0-3.0) may be considered. |

Bibliography
VII. CHOLESTEROL

A. Epidemiology: Epidemiological and observational studies have not shown a definite correlation between serum cholesterol levels and the incidence of stroke.\textsuperscript{1,2} According to the Asia Pacific Cohort Studies Collaboration,\textsuperscript{3} the relationship between cholesterol and stroke risk is more complex, with a stronger positive association with ischemic stroke and a weaker negative association with hemorrhagic stroke. However, this trend in hemorrhagic stroke was not seen in the HPS and combined data from the Long-term Intervention with Pravastatin in Ischaemic Disease study (LIPID) and the Cholesterol and Recurrent Event study. Furthermore, low cholesterol is common in patients with weight loss, severe handicap, or severe and chronic illness, which may be confounding factors for the demonstrated trend between hemorrhagic stroke and low total cholesterol.

B. Risk Modification

B1. Primary Stroke Prevention

A meta-analysis of 13 lipid-lowering trials prior to statin use showed no change in risk for total stroke.\textsuperscript{4} With the advent of statins, a meta-analysis of CARE, LIPID, HPS, the Scandinavian Simvastatin Survival Study (4S), the Prospective Study of Pravastatin in the Elderly at Risk study (PROSPER), the Antihypertensive and Lipid-Lowering Treatment to Prevent Heart Attack Trial (ALLHAT), the Kyushu Lipid Intervention Study (KLIS), the GREek Atorvastatin and Coronary-heart-disease Evaluation (GREACE), and the Anglo-Scandinavian Cardiac Outcomes Trial (ASCOT) showed that the overall relative risk reduction is 21% (ARR=9%).\textsuperscript{5} The greater the LDL reduction, the greater the intima-media thickness and stroke risk reduction.\textsuperscript{6}

Statins conferred an important and large relative reduction in cardiovascular events including stroke among hypertensive patients who are not conventionally deemed dyslipidemic.\textsuperscript{7} Pretreatment with statins seem to reduce clinical severity in patients with stroke, especially among diabetics.\textsuperscript{8,9}

B2. Secondary Stroke Prevention

HPS showed a 19% reduction in major vascular events, but the decrease was due to reduction in coronary events and not in stroke recurrence.\textsuperscript{10}

The Stroke Prevention with Aggressive Reduction of Cholesterol Levels (SPARCL), a large-scale statin RCT in patients with a history of stroke or TIA without CAD, and have mildly elevated lipid levels (mean LDL=133 mg/dL).\textsuperscript{11} The trial showed that atorvastatin 80 mg/day significantly reduced stroke by 16% in this patient subgroup.
Statins are likely to reduce stroke risk by stabilizing and/or repressing plaque. Statins also have pleiotropic (anti-inflammatory, antioxidant) effects that further justify their use in the primary and secondary prevention of cerebrovascular disease. Statins have a favorable safety profile and are not associated with increased hemorrhagic stroke or cancer risk.

C. Recommendations

C1. Primary Stroke Prevention

Therapeutic lifestyle changes are recommended as an essential modality in clinical management. These include smoking cessation, weight management, regular physical activity and adequate BP monitoring and control. For patients at any level of cardiovascular risk, especially those with established atherosclerosis, a low-fat, low-cholesterol diet is recommended for life.

High-risk hypertensive patients and those with CAD should be treated with lifestyle measures and a statin, even with normal LDL levels (Class I-A).

Adults with diabetes, especially those with additional risk factors, should receive statins to lower the risk of a first stroke (Class I-A).

Patients with coronary artery disease and low HDL may be treated with weight reduction, increased physical activity, smoking cessation, and possibly niacin or fibrates (Class IIa-B).

C2. Secondary Stroke Prevention

Statins are recommended in patients with coronary heart disease or symptomatic atherosclerotic disease to lower cholesterol levels to LDL<100 mg/dL (<70 mg/dL for very high-risk persons) (Class I-A).

Patients with ischemic stroke or TIA presumed to be due to atherosclerosis but without preexisting indications for statins are reasonable candidates for statin treatment to reduce the risk of vascular events (Class IIa-B).

Patients with ischemic stroke or TIA and low HDL may be considered for treatment with niacin or fibrates (Class IIb-B).

Lastly, adherence to the statements of the 2005 Clinical Practice Guidelines for the Management of Dyslipidemia in the Philippines is recommended. These are:

1. To reduce the overall cardiovascular risk, all patients, regardless of their present morbid condition or risk profile, should be advised on the need for the following:
   - Smoking cessation;
   - Weight management;
   - Regular physical activity; and
   - Adequate blood pressure monitoring and control.
2. For patients at any level of cardiovascular risk, especially those with established atherosclerosis, a low-fat, low-cholesterol diet is recommended for life.

3. In poorly nourished and elderly patients, correction of nutritional deficiencies can be achieved even with a low-fat, low-cholesterol diet.

4. For low-risk patients without evidence of atherosclerosis, drug therapy is not recommended, regardless of lipid levels. Risk factors include hypertension, familial hypercholesterolemia, left ventricular hypertrophy, smoking, family history of premature CAD, male sex, age >55 years, proteinuria, microalbuminuria, BMI ≥25. Low risk patients have <3 risk factors. The presence of familiar hypercholesterolemia warrants treatment even without other risk factors.

5. For patients without established atherosclerosis but with ≥3 risk factors and total cholesterol ≥190 mg/dL or LDL ≥100 mg/dL, statins may be recommended.

6. For diabetic patients without evidence of atherosclerosis and with total cholesterol ≥190 mg/dL or LDL ≥100 mg/dL, statins are recommended.

7. Fibrates may be recommended as an alternative to statins in diabetic patients with HDL ≤35 mg/dL and LDL ≤90 mg/dL.

8. For patients with established atherosclerosis and total cholesterol ≥190 mg/dL or LDL ≥100 mg/dL, statins are recommended.

9. Fibrates may be recommended as an alternative to statins if HDL ≤35 mg/dL and LDL ≤90 mg/dL.

10. In patients without risk factors and history or symptoms of established atherosclerosis, screening for lipid levels is not recommended.

11. In patients without established atherosclerosis but with ≥3 risk factors, lipid profile may be recommended.

12. In patients with established atherosclerosis or diabetes, the use of lipid profile for screening is recommended.

Bibliography

VIII. CAROTID STENOSIS

A. Epidemiology: Extracranial carotid artery disease accounts for 15% to 20% of all ischemic strokes. Individuals with carotid stenosis often have more widespread atherosclerotic disease with a high prevalence of coronary heart disease and claudication. The stroke risk due to carotid artery stenosis is determined primarily by symptom status and is related to lesion severity. Patients with symptomatic severe carotid stenosis have an annual stroke risk of 13% to 15%, compared with 1% to 2% in those with no history of prior stroke or TIA or those with asymptomatic lesions. In addition, echoluent or ulcerated plaques, hypertension and progressive lesions are associated with increased risk of neurological events.
B. Risk Modification

B1. Primary Stroke Prevention

The role of carotid endarterectomy (CEA) in asymptomatic cases requires special consideration. Among patients with asymptomatic carotid artery stenosis of 60% to 99% enrolled in the Asymptomatic Carotid Artery Stenosis Study (ACAS), CEA combined with best medical treatment reduced the 5-year ipsilateral-stroke risk from 11% to 5.1% (RRR=53%).\(^5\) The Asymptomatic Carotid Surgery Trial (ACST) supports the results of ACAS, showing a small but definite reduction in the risk of stroke with surgery among patients with at least 60% stenosis (5-year stroke risk of 11.8% in the medical arm compared with 6.4% in the combined CEA and medical treatment arm).\(^6\) For asymptomatic patients to benefit from surgery, there should be an exceptionally low perioperative complication rate (< 3%).\(^5,7\)

Neither ACAS nor ACST showed increasing benefit from surgery with increasing degree of asymptomatic stenosis within the 60%-to-99% range.\(^5,6,8\)

B2. Secondary Stroke Prevention

Among symptomatic patients with 70% stenosis or greater but without near occlusion, combined CEA and medical treatment provide up to 16% absolute-risk reduction or 61% relative-risk reduction in ipsilateral and perioperative stroke over medical treatment alone (over 5 years).\(^4,9,11\)

There was a trend toward benefit with surgery at 2 years (ARR=5.6 %) among patients with near-total carotid occlusion, but this was seen only for in the short term (-1.7% over 5 years).\(^9\)

CEA was harmful for symptomatic patients with less than 30% stenosis. It had no effect among patients with 30% to 49% stenosis, and was of marginal benefit in patients with 50% to 69% stenosis. Greater benefit was seen in men, those ≥75 years old, those with hemispheric symptoms (compared with those with transient monocular blindness) and those who were randomized within 2 weeks of a TIA or a non-disabling ischemic stroke.\(^4,9,11\)

Meta-analysis of five completed or terminated RCTs comparing endovascular treatment and CEA (Carotid and Vertebral Artery Transluminal Angioplasty Study [CAVATAS], Kentucky, Leicester, Wallstent, Stenting and Angioplasty with Protection in Patients at High Risk for Endarterectomy [SAPPHIRE]) found no difference in the odds of death or any stroke at 30 days and at one year between the two groups.\(^12\) As yet, there is no evidence on the long-term efficacy of angioplasty and stenting available from any of the studies. Several international trials such as the Carotid Revascularization Endarterectomy versus Stenting Trial (CREST),\(^13\) International Carotid Stenting Study (ICSS),\(^14\) Stent-protected Percutaneous Angioplasty of the Carotid versus Endarterectomy (SPACE)\(^15\) and Endarterectomy versus Angioplasty in patients with severe Symptomatic Stenosis (EVA-3s)\(^16\) are awaiting completion.
C. Recommendations: Antiplatelets, statins and modification of stroke risk factors for all patients with carotid artery disease (Class I-C).

At present, mass screening for high-grade asymptomatic carotid stenosis is not cost-effective. However it is reasonable to do screening using non-invasive tests (e.g., carotid duplex) in patients at risk for significant carotid disease, such as those who survived a stroke, or those who have carotid bruit, peripheral vascular disease, and/or CAD. It is also reasonable to consider CEA for patients with asymptomatic stenosis of >60% if the patient has a life expectancy of at least 5 years and the perioperative risk can be reliably documented to be <3% (Class I-A).

CEA combined with optimal medical management is recommended for patients with recent TIA or stroke and ipsilateral severe carotid artery stenosis (70%-99%) if perioperative risk of <6% can be attained (Class I-A).

For symptomatic patients with 50% to 69% stenosis, CEA is recommended depending on patient-specific factors, such as age, gender, comorbidities and severity of initial symptoms (Class I-A). When the degree of stenosis is <50%, there is no indication for CEA (Class III-A).17,18

Since benefit from CEA is dependent on the degree of stenosis, measurement must be accurate and reliable. In deciding for surgical intervention, the North American Symptomatic Carotid Endarterectomy Trial (NASCET) method of angiographically defining the degree of stenosis is recommended (i.e., \% stenosis = \{1-\[diameter of stenosis/diameter of distal internal carotid artery\]} x 100%).19

In patients with concomitant carotid and coronary artery disease, available data at this time are insufficient to declare superiority of timing CEA either before or simultaneous with coronary-artery bypass grafting (CABG).

Unless results of ongoing studies are reported, carotid angioplasty and stenting (CAS) remains to be a second option to CEA. The endovascular approach is favored in certain cases (e.g., stenosis is difficult to access surgically; restenosis after CEA or medical conditions exist that greatly increase the risk of surgery) (Class IIb-B). CAS is reasonable when performed by operators with established periprocedural morbidity and mortality rates of <6% (Class IIb-B).18

Bibliography
IX. INTRACRANIAL STENOSIS

A. Epidemiology: Atherosclerotic intracranial stenoses are responsible for ischemic stroke in 5% to 10% of Caucasian patients, and in up to 33% of Asian, Hispanic and African patients. Other risk factors for intracranial atherosclerosis include age, hypertension, smoking, diabetes, lipid disorders and metabolic syndrome.

The annual risk of stroke among patients with symptomatic intracranial stenosis ranges from 3% to 15% (approximate annual values are: 7.6% for the carotid siphon, 7.8% for the middle cerebral artery [MCA], 2%-7% for the vertebral artery and 11% in the basilar artery). In contrast, asymptomatic MCA stenosis appears to have a benign prognosis with a low risk of ipsilateral stroke (1.4% annually) in medically treated Caucasian patients.
B. Risk Modification: There is currently no data available.

Among patients with stroke or TIA caused by a 50% to 99% stenosis of a major intracranial artery, the Warfarin-Aspirin Symptomatic Intracranial Disease (WASID) study showed that warfarin does not confer an advantage over aspirin in terms of stroke reduction (2-year ischemic stroke rate of 19.7% for aspirin vs. 17.2% for warfarin). In the Trial of cilOstazol in Symptomatic intracranial arterial Stenosis (TOSS), adding cilostazol to aspirin was superior to aspirin monotherapy in preventing progression of intracranial arterial stenosis. Continued trials are warranted to confirm the efficacy of cilostazol in preventing progression and further vascular events in patients with symptomatic intracranial arterial stenosis. The EC-IC Bypass Trial failed to show clinical benefit of revascularization procedure (extracranial-intracranial anastomosis) in patients with atherosclerotic disease of the carotid artery and MCA. Bypass-patency rate was 96%, but fatal and non-fatal stroke occurred more frequently and earlier among those randomized to surgery.

Single-center experiences suggest that intracranial angioplasty and/or stenting can be performed with a high degree of technical success. Acceptable anatomical and clinical results of up to 6 months were obtained in small groups of medically refractory patients with strokes attributable to intracranial stenosis enrolled in the Stenting of Symptomatic Atherosclerotic Lesions in the Vertebral or Intracranial Arteries (SSYLVIA) and WingSpan trials. Further studies are needed in larger cohorts with longer follow-up periods to fully determine the effectiveness of interventional catheter-based procedures for intracranial stenosis.

C. Recommendation: Patients with intracranial stenosis should be counseled regarding optimal medical therapy (antiplatelets, statins) and aggressive management of stroke risk factors.

Further studies are warranted to evaluate short and long-term efficacy of angioplasty and or stenting in patients with hemodynamically significant intracranial stenosis (>50%) and symptoms despite medical therapy. (Class IIb-C)

Bibliography

X. SMOKING

A. Epidemiology

A1. Prevalence

Asian countries have the highest prevalence of smoking in the world: 72% in Korean men, 63% in Chinese men and 58% in Japanese men.¹ The 2003 NNHeS survey showed that the prevalence of smoking in Filipinos is 56.3% in males and 12.1% in females.² Two national surveys on Filipino adolescents revealed that one in five adolescents current smoking, males having a higher rate than females (37.3% vs. 6.3%).³

A2. Smoking as Stroke Risk Factor
A meta-analysis of 32 studies estimated the relative risk for cerebral infarct to be 1.9 for smokers versus nonsmokers.\(^5\) Case control and prospective studies have shown that cigarette smoking is an independent predictor of stroke, with a dose-response relationship affecting both men and women.\(^6\)\(^-\)\(^8\) The Framingham Heart Study showed that the relative risk of stroke in heavy smokers (>40 cigarettes/day) was twice that of light smokers (<10 cigarettes/day), and the risk increased with the number of cigarettes smoked.\(^9\) The large cohort study of U.S. male physicians showed heavy smokers (>20 cigarettes/day) had a relative risk of 2.71 for total nonfatal stroke, and 1.46 for fatal stroke.\(^10\)

Studies also suggest a dose-response relationship between pack-years of smoking and carotid-artery intima-media wall thickness.\(^11\) Smoking only one cigarette increases heart rate, blood pressure and cardiac index, and decreases arterial distensibility.\(^12\) In addition, both active and passive smoke are associated with the development of atherosclerosis.\(^13\)

Stroke also increases the risk of hemorrhagic stroke. In men, current smokers of <20 cigarettes/day had relative risks of 1.65 for total hemorrhagic strokes, 1.60 for intracerebral hemorrhage (ICH), and 1.75 for subarachnoid hemorrhage (SAH) compared to those who never smoked.\(^14\) Current smokers of >20 cigarettes/day had relative risks of 2.36 for total hemorrhagic stroke, 2.06 for ICH and 3.22 for SAH.

In women, current smokers of <15 cigarettes/day had a relative risks of 1.93 for total hemorrhagic stroke, 2.5 for ICH and 1.70 for SAH.\(^15\) Women who smoked >15 cigarettes/day had relative risks of 3.29 for total hemorrhagic stroke, 2.67 for ICH and 4.02 for SAH.

A3. Smoking Potentiates Effects of Other Stroke Risk Factors

A synergistic effect on the risk of cerebral infarction exists between the use of oral contraceptives (OC) and smoking. With nonsmoking women who do not use OC as reference group, the odds for cerebral infarction were 1.3 times greater for women who smoked but did not use OC, 2.1 times greater for nonsmoking OC users, and 7.2 times greater for OC users who smoked.\(^16\)

A similar impact on hemorrhagic stroke is observed. The odds for hemorrhagic stroke were 1.6 times greater for women smoked but did not use OC, 1.5 times greater for nonsmoking OC users, and 3.7 times greater for OC users who smoked.\(^17\)

A4. Environmental Tobacco Smoke and Passive Smoking

Like outdoor air pollution, the effects of second-hand smoke are large and rapid. Several studies suggest that environmental tobacco smoke is a substantial risk factor for stroke, with risk approaching the doubling found seen with active smoking.\(^18\) Passive smoking may exert detrimental effects on vascular homoeostasis. Cohort studies showed an elevated prevalence of stroke among women nonsmokers living with husbands who smoked, and prevalence increased with increasing intensity and duration of husbands’ smoking.\(^19\)
B. Risk Modification: Both the Framingham Heart Study and the Nurses Health Study showed that 5 years after cessation of smoking, risk ratios normalized.\textsuperscript{9,20} However, another study showed that the risk reduction was dependent on the quantity of cigarettes smoked before stopping: light smokers (<20 cigarettes/day) reverted back to normal values but heavy smokers retained twice the incidence of stroke as non smokers.\textsuperscript{21} Switching to pipe or cigar smoking confers little benefit, emphasizing the need for complete cessation of smoking.\textsuperscript{22} A combination of nicotine replacement therapy, social support and skills training seems to be the most effective approach to smoking cessation.\textsuperscript{23,24}

C. Recommendations: Smoking cessation for all current smokers is recommended (Class I-C).\textsuperscript{25} Nonsmokers are also advised not to start smoking. Exposure to environmental tobacco smoke should be minimized (Class IIa-C). Republic Act No. 9211 or The Tobacco Regulation Act of 2003 should be implemented to protect the populace from hazardous products, promote the right to health and instill health consciousness. Effective behavioral and pharmacological treatments should be advised and encouraged for nicotine dependence (Class IIa-B).

Bibliography

XI. EXCESSIVE ALCOHOL

A. Epidemiology: There is a direct, dose-dependent effect of the consumption of alcohol on the risk of hemorrhagic stroke but the association with ischemic stroke varies with different studies.\(^1\)-\(^4\) Most studies suggest a J-shaped association between alcohol and ischemic stroke: a protective effect with light to moderate drinking, and an elevated risk with heavy consumption.\(^5\)-\(^8\) While the protective effect of light consumption alcohol is evident among Caucasians, this is not evident among Asians.\(^2\)-\(^4\),\(^6\)-\(^9\),\(^11\) Moderate alcohol consumption decreased risk of ischemic stroke in a multiethnic population.\(^12\) Heavy alcohol use, either daily or in binges, is related to excess of stroke risk.\(^13\)

B. Risk Modification: Alcohol consumption of up to two drinks per day was protective against ischemic strokes in Caucasians, Blacks and Hispanics, but consumption above five drinks per day increased the risk of ischemic stroke.\(^14\)

C. Recommendations: Moderate intake of alcohol in those who drink alcohol and have no health contraindications to its use. Consumption of alcohol, up to 30 mL (or 28 grams) of ethanol per day, equivalent to 60 mL or two jiggers of 100-proof whiskey, one glass of wine (240 mL) or two bottles of beer (720 mL), or two drinks per day for men and one drink per day for non-pregnant women, may reduce the risk of ischemic stroke (Class IIb-C).

Patients with ischemic stroke or TIA who are heavy drinkers should eliminate or reduce their consumption of alcohol (Class I-A).

Those who do not customarily drink alcohol should not be encouraged to do so.

Bibliography


XII. PERIPHERAL ARTERIAL DISEASE

Peripheral arterial disease (PAD) is characterized by arterial stenosis and occlusion of the peripheral arterial bed. It can be symptomatic or asymptomatic. Symptomatic PAD ranges from intermittent claudication (IC) to chronic limb ischemia. Regardless of symptomatology, PAD is an indicator of diffuse systemic atherosclerosis. Risk factors include smoking, DM, dyslipidemia, hypertension and hyperhomocysteinemia, which considerably and frequently overlap and coexist with coronary and cerebrovascular disease. There are numerous reports on the increased risk of MI, stroke and cardiovascular death in patients with PAD.1,2

A. Epidemiology: The prevalence of PAD is highly age dependent. Using objective testing with ankle-brachial index (ABI) in a U.S. population showed the prevalence was 2.5% in people aged <60 years old, while among the 60- to 69-year age group the prevalence is 8.3%, and is 18.8% in those >70 years old.3 There is a 20% to 60% increased risk for MI and a two- to six-fold increased risk of death due to coronary artery events in PAD patients.4-8 The risk of stroke is increased by approximately 40%. In the Atherosclerosis Risk in Communities (ARIC) study, men with PAD were four to five times more at risk of stroke and TIA than those without PAD.8 In addition, all-cause mortality rate is 61.8% after 10 years in men with PAD compared with 16.9% in unaffected men.6 The corresponding mortality rates for women were 33.3% and 11.6%, respectively. The increase in total mortality was due to a sharp increase in cardiovascular mortality, which persisted even
after adjusting for pre-existing CAD and cerebrovascular disease at baseline. The risk was proportional to the severity of PAD.

Local studies reported that 2% of Filipinos aged 55 years and older have IC, and approximately 5% have PAD upon ABI confirmation. In a study on Filipino patients aged 40 years or older and confined in the intensive care unit for heart attack, stroke or type 2 DM, 30% had silent PAD. The 2003 NNHeS reported a PAD prevalence of 1.6% among Filipinos aged 20 years and above.

B. Risk Modification: In lower-extremity PAD, adverse cardiovascular events may be reduced with lifetime modification or elimination of risk factors, such as cigarette smoking, diabetes mellitus, dyslipidemia and hypertension. Exercise and a non-atherogenic diet are strongly advised

B1. Smoking Cessation
No prospective RCTs have shown the effects of smoking cessation on cardiovascular events. Only observational studies have shown that the risk of death, MI and limb loss is greater in individuals who continue to smoke than those who stop smoking.

B2. Diabetes Mellitus
It is still unclear whether blood glucose control decreases the risk of adverse cardiovascular events in those with lower-extremity PAD. Analysis of the Diabetes Control and Complication Trial (DCCT) showed that the use of intensive insulin therapy on type 1 DM patients only reduced risk of IC, peripheral revascularization and amputation by 22%, which was not statistically significant. The 10-year United Kingdom Prospective Study (UKPDS) showed that aggressive treatment (using sulfonylureas or insulin) in type 2 DM patients reduced the risk of MI by 16% (borderline significance) compared with conventional treatment, but did not reduce the risk of death or stroke.

B3. Dyslipidemia
Treatment of dyslipidemia in patients with systemic atherosclerosis can reduce future adverse cardiovascular events. In the HPS, which included 6,748 PAD patients, there was a 25% reduction of cardiovascular events in the simvastatin-treated group.

B4. Hypertension
In PAD patients, antihypertensive treatment may diminish perfusion to the limb and exacerbate symptoms of limb ischemia. However, most patients do not experience any worsening of symptoms with appropriate antihypertensive therapy needed to reduce risk of cardiovascular events.

The use of beta-blockers has been controversial in the treatment of PAD patients. However, a meta-analysis of 11 placebo-controlled studies in patients with PAD showed that beta-blockers did not adversely affect walking capacity.

The Heart Outcomes Prevention Evaluation (HOPE), which included 4,051 PAD patients randomized to ramipril or placebo, reported risk reduction for MI,
stroke or vascular death by 25%, similar to that achieved in the overall study population.\textsuperscript{18}

**C. Recommendations:** Individuals with risk factors for PAD, regardless of whether they are symptomatic and asymptomatic, should be identified and ABI measured. Therapeutic interventions to diminish the increased risk of MI, stroke and death may be given (Class I-B). Treatment used in the management of atherosclerotic conditions, such as CAD and cerebrovascular disease, as recommended in the other sections of these guidelines, may reduce the risk of stroke.

Those with PAD who smoke should be advised to quit. Smoking cessation interventions, including behavior modification therapy or nicotine replacement therapy, should be offered (Class I-B).

Reducing HbA\textsubscript{1c} to <7% through tight glycemic control may reduce microvascular complications and improve cardiovascular outcomes (Class IIa-C).

Statins are indicated for all patients with PAD (Class I-B), and treatment should aim to reduce serum cholesterol to <70mg/dL when at very high risk of ischemic events (Class IIa-B). Treatment with fibric acid derivatives can be useful for PAD patients with low HDL, normal LDL and elevated triglycerides (Class IIa-C).

Antihypertensive treatment should aim to reduce BP to <140/90 mmHg, or <130/80 mmHg for those with DM or chronic renal disease (Class I-A). Beta-blockers are effective and are not contraindicated in PAD patients (Class I-A). It is reasonable to use ACEIs for those with symptomatic PAD to reduce cardiovascular risk (Class IIa-B). ACEIs may be considered for asymptomatic PAD patients to reduce cardiovascular risk (Class IIb-C).

Antiplatelets are indicated to reduce risk of MI, stroke and vascular death in PAD patients (Class I-A). Daily aspirin 75 to 325 mg is considered safe and effective in reducing the risk of MI, stroke or vascular death (Class I-A). Clopigrel 75 mg/day is recommended as an alternate to (Class I-B). Oral anticoagulation therapy with warfarin is not indicated to reduce the risk of adverse cardiovascular events in patients with PAD (Class III-C).

**Bibliography**

XIII. PHYSICAL INACTIVITY

A. Epidemiology: Physical inactivity is a growing public health problem that may have a major impact on the prevalence of atherothrombotic cardiovascular disease in the coming decades. The relative risk of cardiovascular disease associated with physical inactivity ranges from 1.5 to 2.4, a risk increase similar to those observed with high blood cholesterol, high BP or cigarette smoking.\(^1\) Physical inactivity is a risk factor for stroke, DM, colon and breast cancer, obesity, hypertension, osteoporosis and depression.\(^2\) Local data shows the odds ratio for stroke associated with physical inactivity is 1.23.\(^7\)

B. Risk modification
B1. Primary Stroke Prevention

Physical activity is defined as any bodily movement produced by skeletal muscles that result in energy expenditure beyond resting expenditure. Physical activity reduces stroke risk in both genders and across all racial/ethnic and age groups (OR; 0.37). The Framingham Heart Study, the Honolulu Heart Program, and the Oslo Study have shown the protective effect of physical activity for men. There seems to be a graded linear relation between the volume of physical inactivity and total stroke. The Physicians’ Health Study showed a lower total stroke risk associated with vigorous exercise (five times a week or more) among men (RR; 0.86). The Harvard Alumni Study showed a decrease in total stroke risk in men who were highly physically active (RR; 0.82).

For women, the Nurses’ Health Study and the Copenhagen City Heart Study showed an inverse association between level of physical activity and stroke incidence. Physical activity (in sports, leisure time or at work) also reduced risk of ischemic strokes, in particular.

The protective effect of physical activity may be partly mediated by BP reduction, improvement of lipid profiles (reduces triglyceride, increases HDL, and decreases LDL:HDL ratios), improvement of glucose homeostasis and insulin sensitivity, and improvement in body composition and weight.

Other benefits of physical activity include reduction in blood coagulability, improvement of coronary blood flow, augmentation of cardiac function, enhancement of endothelial function, improvement of autonomic tone, and reduction of systemic inflammation.

B2. Secondary Stroke Prevention

The cardiac response to acute exercise among stroke survivors has been documented in some studies. Stroke patients achieve significantly lower maximal workloads, heart rate and BP responses than control subjects during progressive exercise testing to volitional fatigue. Furthermore, stroke survivors are often deconditioned and predisposed to a sedentary lifestyle that limits performance of activities of daily living, increases the risk for falls, and may contribute to a heightened risk for recurrent stroke and cardiovascular disease. Thus, the major rehabilitation goals for the stroke patient are: (1) to prevent complications of prolonged inactivity; (2) to decrease recurrent stroke and cardiovascular events, and; (3) to increase aerobic fitness.

Stroke patients can increase their cardiovascular health and fitness with regular aerobic exercise. In a RCT of 42 hemiparetic stroke survivors, vigorous aerobic exercise training three times per week for 10 weeks significantly improved peak oxygen consumption and workload, submaximal exercise, BP response, exercise time and sensorimotor function. In a study of 35 stroke patients with multiple comorbidities who underwent 12 weeks of a 1-hour/day, 3-days/week exercise program of combined cardiovascular, strength and flexibility training, the exercise group had significant gains in peak oxygen uptake, strength and improvements in body composition compared with controls. In a RCT involving 88 men with CAD and disability, two-thirds of whom were stroke survivors, a 6-month home exercise training program significantly increased peak
left ventricular ejection fraction and HDL, and decreased resting heart rate and total serum cholesterol.\textsuperscript{35}

C. Recommendations

C1. Primary Stroke Prevention

Increased physical activity is associated with a reduction in stroke risk, and is recommended (Class I-B). It is important to recognize that physical education in school may form the starting point for an active lifestyle later in life. At least 30 minutes of moderate-intensity aerobic exercise on most days of the week (preferably all days) is recommended as part of a healthy lifestyle (Class IIa-B). Healthy people should be advised to choose enjoyable activities that fit into their daily routine, preferably for 30 to 45 minutes, four to five times weekly, at an intensity to maintain 60% to 80% of the average maximum heart rate. Additional benefits are gained from vigorous-intensity activity.\textsuperscript{36}

C2. Secondary Stroke Prevention

Exercise for stroke patients is recommended and should be tailored to individual needs and limitations. In general, aerobic training at 40% to 70% of peak oxygen consumption or heart rate reserve may be advised to stroke survivors.\textsuperscript{37} Continuous or accumulated aerobic training for 20 to 60 minutes daily, three to seven days a week, depending on the patient’s level of fitness, is advised.

Persons with known or suspected cardiovascular, respiratory or neurological disorders should consult a physician before beginning or significantly increasing physical activity. Adaptive programs for post-stroke patients depending on neurological deficits are recommended.\textsuperscript{38}

Bibliography

XIV. OBESITY
A. Epidemiology: The World Health Organization defined “overweight” as having a body mass index (BMI) >25 kg/m² and “obesity” with a BMI >30 kg/m² for the general population. However, the National Institute for Health and Clinical Excellence (NICE) recommends that overweight or obesity among Asian adults should be set at 23 and 27.5 kg/m² respectively.

Waist circumference (WC) is positively correlated with disease risk, and is one of the most practical measurements for assessing abdominal fat mass (central obesity). For Asians, the cutoff points are 90 cm (35 inches) for males and 80 cm (32 inches) for females.

Another measure of abdominal fat deposition is the waist-hip ratio (WHR), defined as the waist circumference divided by the hip circumference. Elevated WHR is defined as >0.95 in males and >0.85 in females.

In the Philippines, the prevalence of obesity based on BMI has increased from 4.6% to 5.0% between 1998 to 2003. Obesity is increasingly being recognized as a modifiable risk factor for cardiovascular disease, particularly ischemic heart disease. Primary prevention studies documenting the specific impact of obesity on stroke have varied results. In men, findings from the Physicians’ Health Study have shown that an increasing BMI is associated with a steady increase in ischemic stroke, independent of hypertension, diabetes and cholesterol. Among women, data are inconsistent, with some studies showing association, while others, none.

Several studies suggest that abdominal obesity, rather than general obesity, is more related to stroke risk. In the Northern Manhattan Study, a significant and independent association between abdominal obesity (elevated WHR) and ischemic stroke was found in all racial/ethnic groups, whereas BMI did not show any significant association with ischemic stroke. Furthermore, persons with elevated BMI or WHR have increased carotid artery intima-media thickness and cross-sectional intima-media area, which are two preclinical predictors of atherosclerosis.

B. Risk Factor Modification

B1. Primary Stroke Prevention

Epidemiological studies indicate that increased body weight and abdominal fat are directly associated with stroke risk. Weight reduction is recommended because it lowers BP (Class I-A) and may thereby reduce the risk of stroke. Modest weight loss (e.g., 10% of the initial body weight over 6 months) is realistic and attainable. It is better to maintain a moderate loss over the long term than to achieve a greater weight loss that cannot be maintained.

B2. Secondary Stroke Prevention
Although no study has demonstrated that weight reduction will reduce stroke recurrence in patients who have suffered a previous stroke or TIA, losing weight significantly improves BP, fasting glucose values, serum lipids and physical endurance.\textsuperscript{13} Because obesity is a contributing factor to other risk factors associated with recurrent stroke, promoting weight loss and maintenance of a healthy weight is important. Exercise and a diet rich in fruits and vegetables can help control weight and reduce the risk of stroke, MI and death.\textsuperscript{14,15}

**C. Recommendation:** Weight reduction should be considered for all overweight patients to maintain the following goals: \(\text{BMI}=18.5 \text{ to } 22.9 \text{ kg/m}^2\); \(\text{WHR} \leq 0.95\) in males and \(\leq 0.85\) in females; and \(\text{WC} \leq 90 \text{ cm (35 inches)}\) in males and \(\leq 80 \text{ cm (32 inches)}\) in females.\textsuperscript{16}

Gradual and moderate weight loss is encouraged for overweight and obese patients. Clinicians should encourage weight management as early as childhood through an appropriate balance of caloric intake, physical activity, exercise and behavioral counseling.

<table>
<thead>
<tr>
<th>Classification</th>
<th>BMI (kg/m(^2))</th>
<th>Risk of comorbidities</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Waist circumference</td>
</tr>
<tr>
<td></td>
<td></td>
<td>&lt;90 cm (men)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>&lt;80 cm (women)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(\geq 90) cm (men)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(\geq 80) cm (women)</td>
</tr>
<tr>
<td>Underweight</td>
<td>&lt;18.5</td>
<td>Low*</td>
</tr>
<tr>
<td>Normal range</td>
<td>18.5-22.9</td>
<td>Average</td>
</tr>
<tr>
<td>Overweight (at risk)</td>
<td>23-24.9</td>
<td>Increased</td>
</tr>
<tr>
<td>Obese I</td>
<td>25-29.9</td>
<td>Increased</td>
</tr>
<tr>
<td>Obese II</td>
<td>(\geq 30)</td>
<td>Severe</td>
</tr>
</tbody>
</table>

* But increased risk of other clinical problems

**Bibliography**


XV. DIET

A. Epidemiology: Although dietary factors may be risk factors for stroke, their role is poorly defined. Homocysteine may be associated with stroke and is associated with deficiency of dietary intake of folate, vitamins B6 and B12 (observed in case-control studies but not clearly in prospective studies). In ecological and some prospective studies, a higher level of sodium intake is associated with an increased risk of stroke. Higher potassium intake is also associated with reduced stroke risk in prospective studies. However, several methodological limitations, particularly difficulties in estimating dietary electrolyte intake, hinder risk assessment and may lead to false-negative results in observational studies.

B. Risk Modification: Prospective studies show that increased fruit and vegetable consumption is associated with a dose-related reduced stroke risk. Fruits and vegetables may contribute to stroke prevention though antioxidant mechanisms or elevation of potassium levels. Increase sodium intake is associated with hypertension, and reduction in salt consumption may significantly lower BP and reduce stroke mortality.

The 2006 AHA Guidelines recommend a well-balanced diet containing ≥5 servings of fruits and vegetables per day to reduce stroke risk. The DASH diet, which emphasizes fruit, vegetables and low-fat dairy products and is reduced in saturated and total fat, also lowers BP and is recommended (Class I- A).
C. Recommendations: While awaiting more definitive data, reducing intake of sodium and increasing intake of potassium to help lower BP is recommended (Class I-A). The recommended sodium intake is ≤2.3 g/day (100 mmol/day), and the recommended potassium intake is ≥ 4.7 g/day (120 mmol/day).

It seems prudent to limit excess saturated fat and to replace vitamins B6 and B12 and folate when such deficiencies are identified

A diet rich in fruits and vegetable is advised (Class IIb-C).

Bibliography:

APPENDIX OF DIET MODIFICATION FOR STROKE PREVENTION

The FNRI-DOST and the Nutritionists-Dietitians Association of the Philippines (NDAP) provide a simple diet guide that clinicians can in advising patients on dietary fat modification. However, patients requiring intensive dietary interventions for whatever reason or condition should be referred to a nutritionist/dietitian for individualized counseling.

Simple Dietary Plan for Fat Modification (2000)
The Biomedical Nutrition Research Division, FNRI-DOST, and NDAP

Some pointers to observe in planning meals:
1. Choose freely from fruits, vegetables, cereals, root crops, bread, dried beans and nuts.
2. Eat fish as main dish at least three times a week.
3. May eat chicken meat as a substitute to fish at least three to four times a week.
4. For other kinds of meat, use lean parts and prepare as boiled, baked, broiled, or roasted. Trim off any visible fat.
5. Use evaporated filled milk or skimmed milk instead of whole milk and avoid whole milk products such as cheese, butter, cream, etc. Use margarine made with allowed vegetable oil.
6. Use unsaturated fats and oils such as corn oil, soybean oil, peanut butter, etc.
7. Limit eggs to only three per week.
8. Avoid rich desserts such as cakes, pastries, cookies, pies, ice cream and chocolates.
9. Always read the nutrition labels of packaged/processed foods.

### Food selection guide

<table>
<thead>
<tr>
<th>Food group</th>
<th>Allowed</th>
<th>Restricted/avoided</th>
</tr>
</thead>
</table>
| Fats and oils                                        | *In prescribed amounts:* Olive, canola, corn, soybean, palm, sunflower and peanut oils. Coconut oil. | • Fats and oils from animal foods, butter. Hydrogenated vegetable oils (e.g., margarine, lard, shortening, spread)  
• Meat and chicken fat drippings used for sauces, bacon fat, “chicharon” |
| Meat, fish, poultry, eggs, milk, dry beans           | *Eat frequently***: Fish (fresh, frozen or canned in water, tomato or vinegar); chicken breast without skin or fat. Dried beans, lentils, fresh or frozen sweetpeas; “vege-meat”, tokwa, taho, tofu & other bean products;  
*Eat occasionally****: Very lean, well-trimmed cuts of beef, pork, veal, lamb; crabmeat, shrimp without head; whole eggs up to 3 pieces per week, eggwhite as desired, may be cooked in allowed fat; Skimmed milk or low fat milk or cheese | • Fish roe, crabfat “aligui” shrimp head, oyster, clams.  
• Fatty meats: cold cuts, canned or frozen meats, sausages.; fatty poultry with skin; internal organs (liver, kidney, heart, tripe, sweetbreads)  
• Whole milk/cow’s milk and cheese made from whole milk |
| Vegetable                                            | All vegetables prepared without fat or with allowed fats only.  
*Eat frequently***: Green leafy and yellow vegetables (they are good sources of beta-carotene, vitamin C, calcium, iron and dietary fiber among others) | Buttered, creamed, fried vegetables in restricted fats or cooked with fatty meat and sauces. |
| Fruit                                                | All fruits; adjust fat allowance when using avocado.  
*Eat frequently***: Vitamin C-rich fruits and deep colored fruits | Avocado in moderation (due to its high fat content) |
| Rice, corn, rootcrops, noodles, bread and cereals    | All cereals, roots/tubers, certain noodles/pasta, wheat bread, “pan de sal” except those restricted  
*Eat frequently***: Oatmeal, cold cereals, corn and sweet potato | • Croissants, muffins, crackers, biscuits, waffles, pancakes, doughnut, rolls made with whole egg, butter, margarine or fat of unknown composition  
• Fresh mami or miki noodles  
• Potato chips, french fries, popcorn |
<table>
<thead>
<tr>
<th>Desserts</th>
<th>Fat-free/low-fat/light dessert. Fresh or canned fruits in light syrup only. Plain cakes with no icing (angel or sponge cakes), meringue, yogurt, sherbet</th>
<th>Rich dessert especially those made with cream, butter, solid shortening, lard, whole egg, chocolate cookies and pies made from cream fudge, ice cream; pastillas from whole milk, yema</th>
</tr>
</thead>
<tbody>
<tr>
<td>Soups</td>
<td>Fat-free broths made from meat or chicken stock. Soups prepared with skimmed/low-fat milk.</td>
<td>Cream soups, fatty broth or stock</td>
</tr>
</tbody>
</table>
| Beverage | • Coffee (not more than 3 cups black), decaffeinated coffee, tea, carbonated beverages in moderation.  
• Alcoholic drinks: not more than 1 jigger for women and not more than 2 jiggers for men | Soda fountain beverages such as milk shake, malted milk and chocolate drinks.  
Alcoholic drinks in moderation. |
| Miscellaneous | • Nuts (peanuts, walnut, almond, cashew, pili, etc.) preferably boiled, roasted/baked, consume in moderation.  
• Nondairy cream in moderation  
• Spices and seasonings in moderation. Sauce made with allowed fats and skimmed milk, vinegar, pickles, mustard, catsup, banana sauce. | • Sauces and gravies with restricted fats or milk; regular mayonnaise  
• Butter-dipped foods.  
• Packed dinners or “instant foods” of unknown fat content. |

*Eat frequently – at least 4 to 5 times a week.  
**Eat occasionally – at most, once a month.*
APPENDIX TO STROKE PREVENTION

Independent Risk Factors for Stroke Among Filipinos (RIFASAF data)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Odds Ratio</th>
<th>95% Confidence Interval</th>
<th>P-Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypertension</td>
<td>6.01</td>
<td>4.48-8.05</td>
<td>0.000</td>
</tr>
<tr>
<td>Diabetes</td>
<td>1.60</td>
<td>1.10-2.32</td>
<td>0.014</td>
</tr>
<tr>
<td>Atrial Fibrillation</td>
<td>1.91</td>
<td>0.51-7.19</td>
<td>0.337</td>
</tr>
<tr>
<td>Myocardial Infarction</td>
<td>4.67</td>
<td>1.18-18.52</td>
<td>0.029</td>
</tr>
<tr>
<td>Rheumatic Heart Disease</td>
<td>3.69</td>
<td>1.05-12.99</td>
<td>0.042</td>
</tr>
<tr>
<td>Smoking</td>
<td>1.36</td>
<td>1.00-1.86</td>
<td>0.053</td>
</tr>
<tr>
<td>Snoring</td>
<td>3.37</td>
<td>2.49-4.58</td>
<td>0.000</td>
</tr>
<tr>
<td>Stress</td>
<td>1.69</td>
<td>1.25-2.29</td>
<td>0.001</td>
</tr>
<tr>
<td>Frequent Alcohol Intake</td>
<td>1.75</td>
<td>1.14-2.70</td>
<td>0.011</td>
</tr>
</tbody>
</table>

Statistical model derived using multiple regression analysis (Strata v. 5.0).
The odds ratio estimates the risk of a patient for stroke if the variable (risk factor) is resent compare with a similar person without the risk factor.

Philippine Prevalence for Atherosclerosis Risk Factors (≥20 years old)
(2003 National Nutrition Health Survey)

<table>
<thead>
<tr>
<th>Risk Factor</th>
<th>Basis</th>
<th>Prevalence (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypertension</td>
<td>BP or history</td>
<td>17.4</td>
</tr>
<tr>
<td>Diabetes</td>
<td>FBS &gt;125 mg/dL or history or use of anti-diabetes medication</td>
<td>4.6</td>
</tr>
<tr>
<td>Stroke</td>
<td>History</td>
<td>1.4</td>
</tr>
<tr>
<td>Hypercholesterolemia</td>
<td>TC ≥240 mg/dL</td>
<td>8.5</td>
</tr>
<tr>
<td>Current Smoking (M/F)</td>
<td>History</td>
<td>56.3 / 12.1</td>
</tr>
<tr>
<td>Obesity: BMI</td>
<td>BMI ≥30</td>
<td>4.8</td>
</tr>
<tr>
<td>Obesity: Waist Hip Ratio (M/F)</td>
<td>1.0 for men; 0.85 for women</td>
<td>12.1 / 54.8</td>
</tr>
<tr>
<td>Angina Pectoris</td>
<td>History or questionnaire</td>
<td>12.1</td>
</tr>
<tr>
<td>Peripheral Arterial Disease</td>
<td>Questionnaire or ankle-brachial index</td>
<td>8.9</td>
</tr>
</tbody>
</table>

Guidelines for ACUTE STROKE TREATMENT
ACUTE STROKE TREATMENT

Definition of “stroke”
Sudden onset of focal neurological deficit lasting more than 24 hours due to an underlying vascular pathology

Table 8: Definition of stroke severity

<table>
<thead>
<tr>
<th>TIA and MILD STROKE</th>
<th>MODERATE STROKE</th>
<th>SEVERE STROKE</th>
</tr>
</thead>
<tbody>
<tr>
<td>TIA: Deficits resolve within 24 hours but usually lasts less than 1 hour without evidence of acute infarction on neuroimaging</td>
<td>Awake patient with significant motor and/or sensory and/or language and/or visual deficit or Disoriented, drowsy or stuporous patient, but with purposeful response to painful stimuli</td>
<td>Comatose patient with non-purposeful response, decorticate, or decerebrate posturing to painful stimuli or Comatose patient with no response to painful stimuli</td>
</tr>
<tr>
<td>Alert patients with any of the following: Mild pure motor weakness of one side of the body, defined as: can raise arm above shoulder, has clumsy hand, or can ambulate without assistance Pure sensory deficit Slurred but intelligible speech Vertigo with incoordination (e.g., gait disturbance, unsteadiness or clumsy hand) Visual field defects alone Combination of (a) and (b)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

See Guidelines for TIA and Mild Stroke See Guidelines for Moderate Stroke See Guidelines for Severe Stroke
| Management Priorities | Ascertain clinical diagnosis of stroke or TIA (history and physical exam are very important)  
> Exclude common stroke mimickers (Appendix I)  
> Provide basic emergent supportive care (ABCs of resuscitation)  
> Monitor neuro-vital signs, BP, MAP, RR, temperature, pupils  
> Perform stroke scales (NIHSS, GCS) (Appendix II)  
> Monitor and manage BP; treat if SBP > 220 or DBP > 120 or MAP > 130 (Appendix III).  
> Precautions:  
> Avoid precipitous drop in BP (BP not > 20% of baseline MAP) (Appendix III). Do not use rapid-acting sublingual agents; when needed, use easily titratable IV or oral antihypertensive medication  
> Ensure appropriate hydration. If IVF is needed, use 0.9% NaCl |
| --- | --- |
| Emergent Diagnostics | • Complete blood count (CBC)  
> Blood sugar (CBG, HGT or RBS)  
> Electrocardiogram (ECG)  
> PT/PTT  
> Plain CT scan of the brain as soon as possible; computation of hematoma volume (Appendix IV) |
| Early Specific Treatment | Ischemic Hemorrhagic  
> Non-cardioembolic (Thrombotic, Lacunar)  
> Cardioembolic (Appendix V) |
CT Scan Confirmed
(Appendix V)

<table>
<thead>
<tr>
<th>Aspirin 160-325 mg/day start as early as possible and continue for 14 days (for secondary prevention, see under “Delayed Management and Treatment”)</th>
<th>Consider anticoagulation with IV heparin or SQ low molecular-weight heparin (LMWH) (Appendix VI) or Aspirin 160-325 mg/day (if anticoagulation is not possible or contraindicated)</th>
<th>Early neurology and/or neurosurgeon consult for all ICH is recommended</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neuroprotection (Appendix V-D)</td>
<td>Neuroprotection (Appendix V-D)</td>
<td>Monitor and maintain BP: MAP 110-130 mmHg (lower limit preferred) (Appendix III)</td>
</tr>
<tr>
<td>Early rehabilitation once stable within 72 hours</td>
<td>Early rehabilitation once stable within 72 hours</td>
<td>Neuroprotection (Appendix V-D)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Early rehabilitation once stable within 72 hours</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Give anticonvulsants only if with seizures</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Steroids are not recommended</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Monitor and correct metabolic parameters</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Correct coagulation / bleeding abnormalities</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Follow recommendations for neurosurgical intervention (Appendix VII)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>For aneurysmal SAH, refer to next chapter.</td>
</tr>
</tbody>
</table>

**TIA**

<p>| Aspirin 160-325 mg/day | If crescendo TIA (multiple events within hours, increasing severity and duration of deficits), consider anticoagulation with IV heparin or SQ LMWH | |</p>
<table>
<thead>
<tr>
<th>CT Scan Not Available</th>
</tr>
</thead>
<tbody>
<tr>
<td>No specific emergent drug treatment recommended</td>
</tr>
<tr>
<td>Neuroprotection (Appendix V-D)</td>
</tr>
<tr>
<td>Consult a neurologist or neurosurgeon</td>
</tr>
<tr>
<td>Early supportive rehabilitation</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Place of Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Admit to Hospital (Stroke Unit)</td>
</tr>
<tr>
<td>Urgent Outpatient Work-up</td>
</tr>
<tr>
<td>1. Stroke onset within 48 hours</td>
</tr>
<tr>
<td>2. Patients requiring any specific active intervention, such as:</td>
</tr>
<tr>
<td>BP control, monitoring and stabilization</td>
</tr>
<tr>
<td>Cardiac stabilization, including AF, CHF, acute MI</td>
</tr>
<tr>
<td>Hydration</td>
</tr>
<tr>
<td>Anticoagulation, if cardioembolic</td>
</tr>
<tr>
<td>3. Rapidly worsening deficits</td>
</tr>
<tr>
<td>4. Recurrent TIA within the past 2 weeks, especially those with increasing severity and duration of deficits, cardiac arrhythmia, or carotid bruit</td>
</tr>
<tr>
<td>1. Single TIA more than 2 weeks</td>
</tr>
<tr>
<td>2. Transient monocular blindness alone</td>
</tr>
<tr>
<td>3. Stable mild strokes &gt;48 hours from ictus not requiring specific active intervention</td>
</tr>
<tr>
<td>Advise immediate re-consult or admission if there is worsening of deficit</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Delayed Management</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ischemic</td>
</tr>
<tr>
<td>Thrombotic/Lacunar</td>
</tr>
<tr>
<td>Cardioembolic</td>
</tr>
<tr>
<td>Hemorrhagic</td>
</tr>
<tr>
<td>Control of risk factors</td>
</tr>
<tr>
<td>-------------------------</td>
</tr>
<tr>
<td>Antiplatelets (aspirin, ticlopidine, dipyridamole, extended-release dipyridamole + aspirin combination, clopidogrel, cilostazol) <em>(Appendix VIII)</em></td>
</tr>
<tr>
<td>Carotid ultrasound: If this reveals &gt;70% stenosis, refer to neurologist/neurosurgeon/vascular surgeon for decision-making regarding CEA or stenting</td>
</tr>
<tr>
<td>Recommend transcranial Doppler (TCD) to document intracranial stenosis</td>
</tr>
</tbody>
</table>
GUIDELINES FOR MODERATE STROKE

Management Priorities

- Ascertain clinical diagnosis of stroke (history and physical exam are very important)
- Exclude common stroke mimickers (Appendix I)
- Basic emergent supportive care (ABCs of resuscitation)
- Neuro-vital signs, BP, MAP, RR, temperature, pupils
- Perform stroke scales (NIHSS, GCS) (Appendix II)
- Monitor and manage BP; treat if SBP > 220 or DBP > 120 or MAP > 130 (Appendix III).
- Precaution: Avoid precipitous drop in BP (not > 20% of baseline MAP) (Appendix III). Do not use rapid-acting sublingual agents; when needed use easily titratable IV or oral antihypertensive medication.
- Identify comorbidities (cardiac disease, diabetes, liver disease, gastric ulcer, etc.)
- Recognize and treat early signs and symptoms of increased ICP (Appendix IX)
- Ensure appropriate hydration. If IVF is needed, use 0.9% NaCl

Emergent Diagnostics

- CBC
- CBG, HGT or RBS
- PT/PTT
- Serum Na⁺ and K⁺
- ECG
  - Plain CT scan of brain as soon as possible;
  - Computation of hematoma volume (Appendix IV)

Early Specific Treatment

<table>
<thead>
<tr>
<th>Ischemic</th>
<th>Hemorrhagic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Non-cardioembolic (Thrombotic, Lacunar)</td>
<td>Cardioembolic (Appendix V)</td>
</tr>
<tr>
<td>CT scan confirmed</td>
<td>Early neurology and/or neurosurgical consult for all ICH is recommended</td>
</tr>
<tr>
<td>-------------------</td>
<td>-------------------------------------------------------------------------</td>
</tr>
<tr>
<td>(Appendix V)</td>
<td>Monitor and maintain BP: MAP 110-130 mmHg (lower limit preferred)</td>
</tr>
<tr>
<td></td>
<td>Neuroprotection (Appendix V-D)</td>
</tr>
<tr>
<td></td>
<td>Give anticonvulsants only if with seizures</td>
</tr>
<tr>
<td></td>
<td>Steroids are not recommended</td>
</tr>
<tr>
<td></td>
<td>Monitor and correct metabolic parameters</td>
</tr>
<tr>
<td></td>
<td>Correct coagulation/bleeding abnormalities</td>
</tr>
<tr>
<td></td>
<td>Follow recommendations for neurosurgical intervention (Appendix VII)</td>
</tr>
<tr>
<td></td>
<td>Early rehabilitation once stable</td>
</tr>
<tr>
<td></td>
<td>For aneurysmal SAH, refer to next chapter.</td>
</tr>
</tbody>
</table>

- **CT scan confirmed (Appendix V)**
- If within 3 hours of stroke onset, consider IV recombinant tissue plasminogen activator (rtPA) and refer to specialist.
- If within 6 hours of stroke onset and in specialized centers, consider intra-arterial (IA) thrombolysis.
- Aspirin 160-325 mg/day, start as early as possible.
- Neuroprotection (Appendix V-D).
- Early supportive rehabilitation.

- If within 3 hours of stroke onset, consider IV rtPA and refer to specialist.
- If within 6 hours of stroke onset and in specialized centers, consider IA thrombolysis.
- Aspirin 160-325 mg/day, start as early as possible.
- If source of embolism can be demonstrated, consider early anticoagulation.
- Neuroprotection (Appendix V-D).
- Early supportive rehabilitation.
- If infective endocarditis is suspected, give antibiotics and do not anticoagulate.

- Early neurology and/or neurosurgical consult for all ICH is recommended.
- Monitor and maintain BP: MAP 110-130 mmHg (lower limit preferred).
- Neuroprotection (Appendix V-D).
- Give anticonvulsants only if with seizures.
- Steroids are not recommended.
- Monitor and correct metabolic parameters.
- Correct coagulation/bleeding abnormalities.
- Follow recommendations for neurosurgical intervention (Appendix VII).
- Early rehabilitation once stable.
- For aneurysmal SAH, refer to next chapter.
## CT scan not available

<table>
<thead>
<tr>
<th>Likely Ischemic</th>
<th>Likely Hemorrhagic</th>
</tr>
</thead>
<tbody>
<tr>
<td>No specific emergent drug treatment recommended</td>
<td>Refer to neurologist/neurosurgeon for further diagnostic work-ups and/or subsequent surgery</td>
</tr>
<tr>
<td>Neuroprotection <em>(Appendix V-D)</em></td>
<td>Neuroprotection <em>(Appendix V-D)</em></td>
</tr>
<tr>
<td>Refer to neurologist</td>
<td>Early supportive rehabilitation</td>
</tr>
<tr>
<td>Early supportive rehabilitation</td>
<td>Early supportive rehabilitation</td>
</tr>
</tbody>
</table>

## Place of Treatment

Hospital – Intensive Care Unit or Stroke Unit

## Delayed Management and Treatment (Secondary Prevention)

### Ischemic

#### Thrombotic/Lacunar

- Control of risk factors
- Antiplatelets (aspirin, ticlopidine, dipyridamole, extended-release dipyridamole + aspirin combination, clopidogrel, cilostazol) *(Appendix VIII)*
- Carotid ultrasound: If this reveals >70% stenosis, refer to neurologist/neurosurgeon/vascular surgeon for decision-making regarding CEA or stenting
- Recommend TCD to document intracranial stenosis

#### Cardioembolic

- Echocardiography and/or cardiology consult
- INR 2.0 (1.6 – 2.5)
- If age <75 and PT/INR available, anticoagulation with coumadin (target INR: 2-3)
- If age >75, aspirin 80-325 mg/day or coumadin (target INR: 2.0 [1.6 – 2.5])

### Hemorrhagic

- Long-term strict BP control and monitoring
- Consider CT angiography, MRA, or 4-vessel angiography in suspected cases of aneurysm, AV malformation or vasculitis
GUIDELINES FOR SEVERE STROKE

**Management Priorities**

- Ascertain clinical diagnosis of stroke (history and physical exam are very important)
- Exclude common stroke mimickers *(Appendix I)*
- Basic emergent supportive care (ABCs of resuscitation)
  - Neuro-vital signs, BP, MAP, RR, temperature, pupils
  - Perform stroke scales (NIHSS, GCS) *(Appendix II)*
- Monitor and manage BP; treat if SBP > 220 or DBP > 120 or MAP > 130 *(Appendix III)*.

**Precautions:**

- Avoid precipitous drop in BP (not > 20% of baseline MAP) *(Appendix III)*. Do not use rapid-acting sublingual agents; when needed, use easily titratable IV or oral antihypertensive medication *(Appendix IIIB)*.

**Identify comorbidities** (cardiac disease, diabetes, liver disease, gastric ulcer, etc.)

**Recognize and treat early signs and symptoms of increased ICP** *(Appendix IX)*

**Ensure appropriate hydration. If IVF is needed, use 0.9% NaCl**

<table>
<thead>
<tr>
<th>Emergent Diagnostics</th>
<th>Ischemic</th>
<th>Hemorrhagic</th>
</tr>
</thead>
<tbody>
<tr>
<td>CBC</td>
<td>Serum Na⁺ and K⁺</td>
<td></td>
</tr>
<tr>
<td>CBG, HGT or RBS</td>
<td>ECG</td>
<td></td>
</tr>
<tr>
<td>PT/PTT</td>
<td>Plain CT scan of brain; computation of hematoma volume <em>(Appendix IV)</em></td>
<td></td>
</tr>
<tr>
<td><strong>CT scan confirmed</strong> (Appendix V)</td>
<td><strong>Non-cardioembolic</strong> (Thrombotic)</td>
<td><strong>Cardioembolic</strong> (Appendix V)</td>
</tr>
<tr>
<td>-----------------------------------</td>
<td>-----------------------------------</td>
<td>---------------------------------</td>
</tr>
<tr>
<td>May give aspirin 160-325 mg/day</td>
<td>May give aspirin 160-325 mg/day</td>
<td>1. Mannitol 20% 0.5 - 1 g/kgBW q 4-6 hours for 3-7 days</td>
</tr>
<tr>
<td>In posterior circulation strokes within 6 hours of onset, consider IA thrombolysis and refer to specialist</td>
<td>In posterior circulation strokes within 6 hours of onset, consider IA thrombolysis and refer to specialist</td>
<td>2. Neuroprotection (Appendix V-D)</td>
</tr>
<tr>
<td>Neuroprotection (Appendix V-D)</td>
<td>Neuroprotection (Appendix V-D)</td>
<td>Neurosurgery consult if:</td>
</tr>
<tr>
<td>If cerebellar infarct, consult neurosurgeon as soon as possible</td>
<td>If cerebellar infarct, consult neurosurgeon as soon as possible</td>
<td>Patient not herniated; bleed located in putamen, pallidum, cerebellum; family is willing to accept consequences of irreversible coma or persistent vegetative state and goal is reduction of mortality (Appendix VII)</td>
</tr>
<tr>
<td>Early supportive rehabilitation</td>
<td>Early supportive rehabilitation</td>
<td>2. ICP monitoring is contemplated and salvage surgery is considered</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Early supportive rehabilitation</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>CT scan not available</strong></th>
<th><strong>No specific emergent drug treatment recommended</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Neuroprotection (Appendix V-D)</td>
</tr>
<tr>
<td></td>
<td>Refer to neurologist</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Place of Treatment</strong></th>
<th><strong>Intensive Care Unit</strong></th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th><strong>Delayed Management and Treatment</strong></th>
<th><strong>Discuss prognosis with relatives of the patient in most compassionate manner</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Ischemic</strong></td>
<td>Hemorrhagic</td>
</tr>
<tr>
<td><strong>Thrombotic</strong></td>
<td><strong>Cardioembolic</strong></td>
</tr>
<tr>
<td>Control of risk factors</td>
<td>Echocardiography and/or cardiology consult</td>
</tr>
<tr>
<td>------------------------</td>
<td>------------------------------------------</td>
</tr>
<tr>
<td>Antiplatelets (Aspirin, ticlopidine, dipyridamole, extended-release dipyridamole + aspirin combination, clopidogrel or cilostazol) (Appendix VIII)</td>
<td>If age &lt;75 and PT/INR available, anticoagulation with coumadin (target INR=2.0-3.0)</td>
</tr>
<tr>
<td>If age &gt;75, aspirin 80-325 mg/day or coumadin with target INR 2.0 (1.6-2.5)</td>
<td></td>
</tr>
</tbody>
</table>
APPENDIX I

Differential Diagnoses of Stroke

A. The presence of any of the following should alert the physician to consider conditions other than stroke:
   - Gradual progressive course and insidious onset
   - Pure hemifacial weakness including forehead (Bell’s palsy)
   - Trauma
   - Fever prior to onset of symptoms
   - Recurrent seizures
   - Weakness with atrophy
   - Recurrent headaches (migraine, tension-type headache)

B. Conditions that mimic stroke in the emergency department (according to decreasing frequency):
   1. Seizures
   2. Systemic infection
   3. Brain tumor
   4. Toxic-metabolic
   5. Positional vertigo
   6. Cardiac
   7. Syncope
   8. Trauma
   9. Subdural hematoma
   10. Herpes encephalitis
   11. Transient global amnesia
   12. Dementia
   13. Demyelinating disease
   14. Cervical spine fracture
   15. Myasthenia gravis
   16. Parkinsonism
   17. Hypertensive encephalopathy
   18. Conversion disorder

Bibliography


APPENDIX II

Stroke Scales

I. Glasgow Coma Scale

<table>
<thead>
<tr>
<th>Category</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Eye Opening</strong></td>
<td></td>
</tr>
<tr>
<td>Spontaneous</td>
<td>4</td>
</tr>
<tr>
<td>To speech</td>
<td>3</td>
</tr>
<tr>
<td>To pain</td>
<td>2</td>
</tr>
<tr>
<td>None</td>
<td>1</td>
</tr>
<tr>
<td><strong>Best Motor Response</strong></td>
<td></td>
</tr>
<tr>
<td>Obey</td>
<td>6</td>
</tr>
<tr>
<td>Localizes</td>
<td>5</td>
</tr>
<tr>
<td>Withdraws</td>
<td>4</td>
</tr>
<tr>
<td>Abnormal flexion (decorticate)</td>
<td>3</td>
</tr>
<tr>
<td>Abnormal extension (decerebrate)</td>
<td>2</td>
</tr>
<tr>
<td>None</td>
<td>1</td>
</tr>
<tr>
<td><strong>Best Verbal response</strong></td>
<td></td>
</tr>
<tr>
<td>Oriented</td>
<td>5</td>
</tr>
<tr>
<td>Confused</td>
<td>4</td>
</tr>
<tr>
<td>Inappropriate words</td>
<td>3</td>
</tr>
<tr>
<td>Incomprehensible sounds</td>
<td>2</td>
</tr>
<tr>
<td>None</td>
<td>1</td>
</tr>
</tbody>
</table>

Total score=15

II. National Institutes of Health (NIH) Stroke Scale

<table>
<thead>
<tr>
<th>Items</th>
<th>Scale Definition</th>
</tr>
</thead>
</table>
| Ia. 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, or totally unresponsive, flaccid, areflexic |
| (LOC)                          |                                                                                                                                                  |
| Ib. LOC Questions             | 0 = Answers both questions correctly  
1 = Answers one question correctly  
2 = Answers neither question correctly |
| Ic. 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. 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 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) |
| 5. Motor (Arm)  
5 a. Left arm  
5 b. Right arm | 0 = No drift; limb holds 90 (or 45) degrees for full 10 seconds  
1 = Drifts; 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 up 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 or joint fusion; explain |
| 6. Motor (Leg)  
6 a. Right leg  
6 b. Left leg | 0 = No drift; leg holds 30-degree position for full 5 seconds  
1 = Drifts; 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 or joint fusion; explain |
| 7. Limb ataxia | 0 = absent  
1 = Present in one limb  
2 = Present in two limbs  
9 = Amputation or joint fusion; explain |
| 8. Sensory | 0 = Normal; no sensory loss  
1 = Mild to moderate sensory loss; patient feels pinprick is less sharp or 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 or total sensory loss; patient is not aware of being touched in the face, arm or leg |
9. Best Language

<table>
<thead>
<tr>
<th>Score</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>No aphasia</td>
</tr>
<tr>
<td>1</td>
<td>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 on provided material difficult</td>
</tr>
<tr>
<td>2</td>
<td>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 the burden of communication</td>
</tr>
<tr>
<td>3</td>
<td>Mute, global aphasia; no usable speech or auditory comprehension</td>
</tr>
</tbody>
</table>

10. Dysarthria

<table>
<thead>
<tr>
<th>Score</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Normal</td>
</tr>
<tr>
<td>1</td>
<td>Mild to moderate; patient slurs at least some words and at worst, can be understood with some difficulty</td>
</tr>
<tr>
<td>2</td>
<td>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</td>
</tr>
<tr>
<td>9</td>
<td>intubated or other physical barrier; explain</td>
</tr>
</tbody>
</table>

11. Extinction & Inattention

<table>
<thead>
<tr>
<th>Score</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>No abnormality</td>
</tr>
<tr>
<td>1</td>
<td>Visual, tactile, auditory, spatial or personal inattention or extinction to bilateral simultaneous stimulation in one of the sensory modalities</td>
</tr>
<tr>
<td>2</td>
<td>Profound hemiattention or hemi-inattention to more than one modality. Does not recognize own hand or orients to only one side of space</td>
</tr>
</tbody>
</table>

Total score=42

III. Modified Rankin Scale

<table>
<thead>
<tr>
<th>Description</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>No symptoms at all</td>
<td>0</td>
</tr>
<tr>
<td><strong>No significant disability</strong> despite symptoms; able to carry out all usual duties and activities</td>
<td>1</td>
</tr>
<tr>
<td>Slight disability; unable to carry out all previous activities but able to look after own affairs without assistance</td>
<td>2</td>
</tr>
<tr>
<td>Moderate disability; requiring some help but able to walk without assistance</td>
<td>3</td>
</tr>
<tr>
<td>Moderately severe disability; unable to walk without assistance and unable to attend to own bodily needs without assistance</td>
<td>4</td>
</tr>
<tr>
<td>Severe disability; bedridden, incontinent and requiring constant nursing care and attention</td>
<td>5</td>
</tr>
</tbody>
</table>
Bibliography


APPENDIX III

Blood Pressure Management

A. BP management in Acute Ischemic Stroke

1. Use the following definitions:
   Cerebral Perfusion Pressure (CPP) = MAP – ICP
   \[ MAP = \frac{2 \times \text{diastolic} + \text{systolic}}{3} \]

2. Check if patient is in any condition that may increase BP such as pain, stress, bladder distention or constipation, which should be addressed accordingly.

3. Allow “permissive hypertension” during the first week to ensure adequate CPP but ascertain cardiac and renal protection
   a. Treat if SBP >220 or DBP >120 or MAP >130
   b. Defer emergency BP therapy if MAP is within 110-130 or SBP=185-220 mmHg or DBP=105-120 mmHg, unless in the presence of:
      ▪ Acute MI
      ▪ Congestive heart failure
      ▪ Aortic dissection
      ▪ Acute pulmonary edema
      ▪ Acute renal failure
      ▪ Hypertensive encephalopathy

4. Treat with small doses of IV antihypertensives patients who are potential candidates for rtPA therapy who have persistent elevations in SBP >185 mmHg or DBP >110 mmHg. Maintain BP just below these limits.

5. Use the following locally available intravenous anti-hypertensives in acute stroke:

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dose</th>
<th>Onset of Action</th>
<th>Duration of Action</th>
<th>Availability /Dilution</th>
<th>Stability</th>
<th>Adverse Reactions</th>
<th>Action</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nicardipine</td>
<td>1-15 mg/hour</td>
<td>5-10 mins</td>
<td>1-4 hours</td>
<td>(10 mg/ 10 ml amp ); 10 mg in 90 ml NSS/D5W</td>
<td>1 to 4 hours</td>
<td>Tachycardia, headache, flushing, dizziness, somnolence, nausea</td>
<td>Inhibits calcium ion from entering slow channel, producing coronary, vascular, smooth muscle relaxation &amp; vasodilatation</td>
</tr>
<tr>
<td>Drug</td>
<td>Dosage and Administration</td>
<td>Duration</td>
<td>Side Effects</td>
<td>Mechanism of Action</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>------------</td>
<td>-------------------------------------------------------------------------------------------</td>
<td>----------</td>
<td>---------------------------------------------------------------------------------------------------------------------------------------------------</td>
<td>-------------------------------------------------------------------------------------</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hydralazine</td>
<td>IV push 10-20 mg/dose q 4-6 hours as needed, may increase to 40 mg/dose</td>
<td>10-20 mins</td>
<td>3-8 hours</td>
<td>25 mg/mL amp; 25 mg/tab</td>
<td>4 days</td>
<td>Tachycardia, flushing, headache, vomiting, increased angina</td>
<td>Direct vasodilatation of arterioles &amp; decreased systemic resistance</td>
</tr>
<tr>
<td>Labetalol</td>
<td>5 mg IV push over 2 mins, repeat with incremental dose of 10, 20, 40, 80 mg until desired BP is achieved or a total dose of 300 mg has been administered</td>
<td>2-5 mins</td>
<td>2-4 hours</td>
<td>5 mg/ml in 40 ml vial; 250 mg in 250 mL NSS/D5W</td>
<td>72 hours</td>
<td>Orthostatic hypotension, drowsiness, dizziness, lightheadedness, dyspnea, wheezing &amp; bronchospasm</td>
<td>Alpha- &amp; beta-blocker. Beta-adrenergic blocking activity is 7x &gt; than alpha-adrenergic blockers. Produces dose-dependent ↓ in BP without significant ↓ in HR or cardiac output</td>
</tr>
<tr>
<td>Esmolol</td>
<td>0.25-0.5 mg/kg IV push 1-2 mins followed by infusion of 0.05 mg/kg/min. If there is no response, repeat 0.5 mg/kg bolus dose &amp; ↑ infusion to 0.10 mg/kg/min. Maximum infusion rate=0.30 mg/kg/min</td>
<td>2-10 mins</td>
<td>10-30 mins</td>
<td>100 mg/10 ml vial; 2,500 mg in 250 mL D5W/NSS</td>
<td>48 hours</td>
<td>Hypotension, bradycardia, AV block, agitation, confusion, wheezing / bronchoconstriction, phlebitis</td>
<td>Short-acting beta-adrenergic blocking agent. At low doses, has little effect on beta2 receptors of bronchial &amp; vascular smooth muscle</td>
</tr>
</tbody>
</table>

B. Blood pressure management in Acute Hypertensive ICH

Maintain MAP<130, but not lower than 110 mmHg
- Sustained hypertension may alter cerebral autoregulation, promote progression of bleed and increase edema
- Hypotension may result in cerebral hypoperfusion especially in the setting of increased intracranial pressure (ICP)
- Absence of penumbra allows for more aggressive BP management

Bibliography


APPENDIX IV

Neuroimaging (CT and MRI)

A. Hyperacute or Acute Ischemic Stroke

- Plain CT scan of the head is the initial neuroimaging study of choice in acute stroke. The main objective is to exclude hemorrhagic stroke and stroke mimickers. A plain study obviates the need to wait for creatinine result.
- CT scan is 100% sensitive in documenting intracranial hemorrhage (ICH) and 96% sensitive in documenting subarachnoid hemorrhage (SAH).
- Cerebral infarcts are often not documented within 3 hours from stroke onset. However, 60% have “early” infarct signs when viewed very closely:
  1. Dense MCA sign
  2. Obscuration of the lentiform nucleus
  3. Loss of the gray-white interphase along the lateral insula (Insular ribbon sign)
  4. Effacement of the sulci
- In cases of neurologic deterioration, large infarcts or suspected hemorrhagic conversion, a follow-up plain CT of the head is recommended.
- MRI has the technical advantage of documenting small lesions or those located in the brainstem or posterior fossa.
- MRI can detect early infarction as early as 90 minutes using Diffusion Weighted Imaging (DWI).
- Despite the superior diagnostic yield of MRI over CT, MRI is not recommended as routine evaluation of patients with acute ischemic stroke. It is more expensive, time-consuming and less readily available.

B. Intracerebral Hemorrhage

- CT can accurately document the exact location of the hemorrhage and the presence of mass effect, ventricular extension and hydrocephalus.
- In hypertensive ICH, a repeat plain CT scan after 24 hours of ictus is recommended especially in cases showing clinical deterioration to document hematoma enlargement and/or development of hydrocephalus.

**Computation of Hematoma Volume (Kothari method)**

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 with hemorrhage X slice thickness (in cm)
Count slice as 1 if size of hematoma is >75% of largest diameter
Count slice as 0.5 if size of hematoma is 25-75% of largest diameter
Disregard slice if size of hematoma is <25% of largest diameter

- In suspected cases of AV malformation, aneurysm or tumor bleed, a contrast CT of the head may be warranted

C. Subarachnoid Hemorrhage

- Plain CT of the head is strongly recommended as the initial procedure for diagnosis.
- The diagnostic yield of CT goes down from 92% within the first 24 hours to 50% within 7 days of onset.

Bibliography


APPENDIX V

Early Specific Treatment For Ischemic Stroke

A. Thrombolytic therapy
   - Patients treated with IV recombinant tissue plasminogen activator (rtPA) within 3 hours of stroke onset are at least 30% more likely to have minimal or no disability at 3 months.
   - Streptokinase has no role in acute thrombolysis for ischemic stroke.

National Institute of Neurological Disorders and Stroke (NINDS) rTPA guidelines

1. Dose of rtPA is 0.9 mg/kg (maximum 90 mg). Ten percent of total dose is given as IV bolus, the rest as infusion over 60 minutes.
2. rtPA is recommended within 3 hours of onset of ischemic stroke. The benefit of IV rtPA for acute ischemic stroke beyond 3 hours from onset of symptoms is not established. IV rtPA is not recommended when the time of onset of stroke cannot be ascertained reliably, including strokes recognized upon awakening.
3. Thrombolytic therapy is not recommended unless the diagnosis is established by a physician with expertise in diagnosing stroke and CT of the brain is assessed by physicians with expertise in reading this imaging study. If CT demonstrates early changes of a recent major infarction such as sulcal effacement, mass effect, edema or possible hemorrhage, thrombolytic therapy should be avoided.
4. Thrombolytic therapy cannot be recommended for patients with any of the following (NINDS Study):
   a. Current use of oral anticoagulants or PT>15 seconds (INR>1.7)
   b. Use of heparin in the previous 48 hours or prolonged PTT>1.5x normal
   c. Platelet count <100,000 mm$^3$
   d. Another stroke or a serious head injury within the previous 3 months
   e. Major surgery within the preceding 14 days
   f. Sustained pretreatment SBP>185 mmHg or DBP>110 mmHg (when aggressive treatment necessary to lower BP)
   g. Rapidly improving neurological signs
   h. Mild, isolated neurological deficits, such as ataxia alone, sensory loss alone, dysarthria alone, or minimal weakness
   i. Prior ICH
   j. Blood glucose <50 mg/dL or > 400 mg/dL
   k. Seizure at onset of stroke
   l. Gastrointestinal or urinary bleeding within preceding 21 days
   m. Recent myocardial infarction (within the previous 3 months)
5. Thrombolytic therapy should not be given unless emergent ancillary care and the facilities to handle bleeding complications are readily available.

6. Caution is advised before giving rtPA to persons with severe stroke (NIH Stroke Scale Score >22)

7. Because the use of thrombolytic drugs carries the real risk of major bleeding, whenever possible the risks and potential benefits of rtPA should be discussed with the patient and his or her family before treatment is initiated.

8. Patients given rtPA should not receive antiplatelets or anticoagulants within 24 hours of treatment.

B. Antithrombotic therapy

1. International Stroke Trial (IST)
   - Multicenter randomized clinical trial of 19,435 patients
   - Regimen: Aspirin 300-325 mg/day vs. no aspirin
     Heparin SC vs. no heparin
     5,000 units bid or 12,500 units bid
   - Started within 48 hours of stroke onset for 14 days or until discharge
   - Results:
     Aspirin
     - Fewer recurrent stroke within 14 days
     - Fewer deaths and dependency at 6 months
     Heparin
     - No benefit even at 6 months
     - If used should not exceed 5,000 units bid

2. Chinese Acute Stroke Trial (CAST)
   - 21,106 patients randomized
   - Aspirin 160 mg/day vs. placebo
   - Started within 48 hours of stroke onset
   - Results:
     Risk of recurrent stroke or vascular death:
     Aspirin 5.3%
     Placebo 5.9%  (p=0.03)

3. Meta-analysis on low molecular-weight heparin (LMWH) and heparinoids in acute ischemic stroke involving 2,855 patients has shown that treatment was associated with significant reduction in venous thromboembolism (DVT and pulmonary embolism). LMWH has no significant effect on reducing death and disability at 6 months. Symptomatic ICH was not significantly increased.
C. Neuroprotection

1. Neuroprotective Interventions: The 5 “H” Principle

Avoid hypotension, hypoxemia, hyperglycemia or hypoglycemia and hyperthermia (fever) during acute stroke in an effort to "salvage" the ischemic penumbra

Avoid Hypotension
- Aggressive BP lowering is detrimental in acute stroke. Manage hypertension as per recommendation (Appendix III)

Avoid Hypoxemia
- Routine oxygenation in all stroke patients is not warranted
- Maintain adequate tissue oxygenation (target $O_2$ saturation >95%)
- Do arterial blood gases (ABG) determination or monitor oxygenation via pulse oximeter
- Give supplemental oxygen if there is evidence of hypoxemia or desaturation
- Provide ventilatory support if upper airway is threatened or sensorium is impaired or ICP increased.

Avoid hypoglycemia or hyperglycemia
- Hyperglycemia can increase the severity of ischemic injury (causes lactic acidosis, increases production of free radicals, worsens cerebral edema and weakens blood vessels), whereas hypoglycemia can mimic a stroke
- Prompt determination of blood glucose should be done in all stroke patients
- Ensure tight glycemic control at 80-110 mg/dL
- Avoid glucose-containing (D5) IV fluids. Use isotonic saline (0.9% NaCl)

Avoid Hyperthermia
- Fever in acute stroke is associated with poor outcome possibly related to increased metabolic demand, increased free radical production and enhanced neurotransmitter release.
- For every 1°C increase in body temperature, the relative risk of death or disability increases by 2.2.
- Search for the source of fever.
- Treat fever with antipyretics and cooling blankets.
- Maintain normothermia.

D. Neuroprotectants
Neuroprotectants are drugs that:

- Protect against excitotoxins and prolong neuronal survival
- Block the release of glutamate, free radicals, inflammatory cytokines, and the accumulation of intracellular calcium cations.

Several neuroprotective drugs have reached phase III clinical trials, but most had negative or disappointing results except for citicoline. Data-pooling analysis on four trials involving 1,652 patients with ischemic stroke show that treatment with citicoline within the first 24 hours increases the probability of global recovery (NIHSS, mRS, BI) by 30% at 3 months.

CDP–choline helps increase phosphatidylcholine synthesis and inhibition of phospholipase A2 within the injured brain during ischemia.

Several phase III clinical trials (e.g. SAINT II, FAST-MAG) are currently underway.

Bibliography


APPENDIX VI

Anticoagulation In Acute Cardioembolic Stroke

A. Cardioembolic sources

<table>
<thead>
<tr>
<th>High Risk</th>
<th>Low or Uncertain Risk</th>
</tr>
</thead>
<tbody>
<tr>
<td>AF (valvular or non-valvular)</td>
<td>Mitral valve Prolapse</td>
</tr>
<tr>
<td>Rheumatic mitral stenosis</td>
<td>Mitral annular calcification</td>
</tr>
<tr>
<td>Prosthetic heart valves</td>
<td>Patent foramen ovale (PFO)</td>
</tr>
<tr>
<td>Recent MI</td>
<td>Atrial septal aneurysm</td>
</tr>
<tr>
<td>LV/LA thrombus</td>
<td>Calcific aortic stenosis</td>
</tr>
<tr>
<td>Atrial myxoma</td>
<td>Mitral valve strands</td>
</tr>
<tr>
<td>Infective Endocarditis</td>
<td></td>
</tr>
<tr>
<td>Dilated cardiomyopathy</td>
<td></td>
</tr>
<tr>
<td>Marantic endocarditis</td>
<td></td>
</tr>
</tbody>
</table>

B. Indications and contraindications for anticoagulation in patients with cardioembolic stroke

<table>
<thead>
<tr>
<th>Probably Indicated</th>
<th>Contraindicated</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intracardiac thrombus</td>
<td>Bleeding diathesis</td>
</tr>
<tr>
<td>Mechanical prosthetic valve</td>
<td>Non-petechial intracranial hemorrhage</td>
</tr>
<tr>
<td>Recent MI</td>
<td>Recent major surgery or trauma</td>
</tr>
<tr>
<td>CHF</td>
<td>Infective endocarditis</td>
</tr>
<tr>
<td>Bridging measure for long term anticoagulation</td>
<td></td>
</tr>
</tbody>
</table>

C. When considering anticoagulation in acute cardioembolic stroke, the benefits of anticoagulation in reducing early stroke recurrence should be weighed against the risk of hemorrhagic transformation. The latter is higher in patients with large infarction, severe strokes or neurological deficits and uncontrolled hypertension.

D. How to anticoagulate

1. Requirements for IV anticoagulation of patients with cardiogenic source of embolism:
   a. Heparin sodium in D5W
   b. Infusion pump, if available
   c. Activated partial thromboplastin time (aPTT) or clotting time

2. Procedure:
   a. Start intravenous infusion at 800 units heparin/hour ideally using infusion pump. IV heparin bolus is not recommended.
b. Perform aPTT as often as necessary, every 6 hours if need, to keep aPTT at 1.5-2.5x the control. Risk for major hemorrhage, including intracranial bleed, progressively increases as aPTT exceeds 80 seconds.

c. Infusion may be discontinued once oral anticoagulation with coumadin has reached therapeutic levels or once antiplatelet medication is started for secondary prevention.

To date, there has been no trial directly comparing efficacy of unfractionated heparin vs LMWH in patients with acute cardioembolic stroke. LMWH has the advantage of ease of administration and does not require aPTT monitoring.

**Bibliography**


APPENDIX VI

Early Specific Treatment of Hypertensive Intracerebral Hemorrhage

A. Medical Treatment for all ICH:
   The goals are to prevent complications and careful manage BP.
   a. Maintain MAP <130, but not lower than 110 mmHg
   b. Manage increased ICP accordingly (see Appendix IX)
   c. Start anticonvulsants only if with seizures
      • The incidence of seizures is higher in ICH, especially in lobar hematomas.
      • The role of prophylactic anticonvulsants in deep hemorrhages is unclear. It is justified to withhold anticonvulsants until clinically indicated.
   d. Prevent and treat respiratory complications. Endotracheal intubation is performed in patients to provide airway protection and in those in coma or with respiratory failure.
   e. Prevent and treat infections.
   f. Maintain adequate nutrition.
   g. Ensure proper fluid and electrolyte balance; maintain normothermia and normoglycemia.
   h. Rehabilitate early once stable.
   i. Practice bedsore precautions.
   j. Deep-vein thrombosis and pulmonary embolism prophylaxis should be instituted (use antiembolic stockings or intermittent pneumatic compression devices)

B. Surgical Treatment
   Its role depends on the size, extent and location of the hematoma, and patient factors.
   a. There is evidence of increase in hematoma size by 33% within 24 hours of stroke onset in 38% of cases.
   b. Considerations for surgical intervention:

   Non-surgical candidates
   • Patients with small hemorrhages (<10 mL) or minimal neurological deficits
   • Patients with GCS<5 except those who have cerebellar hemorrhage and brainstem compression
   • Patients with hematoma volume > 85 mL

   Candidates for immediate surgery
   • Patients with cerebellar hemorrhage >3 cm who are neurologically deteriorating or have brainstem compression and hydrocephalus from ventricular obstruction
• Patients with bleed associated with a structural lesion such as an aneurysm, AV malformation or cavernous angioma if there is a chance for good outcome and the vascular lesion is surgically accessible
• Clinically deteriorating young patients with moderate or large lobar hemorrhage.
• Ventricular drainage for patients with intraventricular hemorrhage with moderate to severe hydrocephalus.

All other patients may benefit from surgery
• Patients with basal ganglia or thalamic hemorrhage
• Patients with GCS >4
• Patients with supratentorial hematoma with volume >30 cc

Bibliography
Academy of Filipino Neurosurgeons Guidelines on the Management of Hypertensive ICH


APPENDIX VIII

Antiplatelets for Secondary Stroke Prevention

A. Aspirin

1. Antiplatelet Trialist’s Collaboration
   - 65 trials involving 60,196 patients with symptomatic atherosclerosis (e.g., unstable angina, MI, TIA, stroke)
   - Aspirin 50-1,500 mg/day vs control
   - 23% odds reduction on composite outcome of MI, stroke or vascular death
   - Highest RRR was seen in the low (75-150 mg) and medium dose (160-325 mg) groups

2. Mini-meta-analysis on aspirin among patient with prior stroke or TIA
   - 10 trials involving 6,171 patients with prior TIA or non-disabling stroke
   - Aspirin reduced the odds for the cluster of stroke, MI or vascular death by 16%
   - No difference in RRR for low (<100 mg), medium (300-325 mg) and high doses (>900 mg) of aspirin

B. Ticlopidine

1. Canadian American Ticlopidine Study (CATS)
   - 1,072 patients with recent thromboembolic stroke
   - Ticlopidine 250 mg bid vs placebo
   - 30.2% risk reduction on composite outcome of MI, stroke, vascular death over placebo

2. Ticlopidine Aspirin Stroke Study (TASS)
   - 3,069 patients with recent TIA/cerebral infarction
   - Ticlopidine 250 mg bid vs aspirin 1,300 mg od
   - 12% risk reduction vs aspirin for stroke or death at 3 years

C. Clopidogrel

1. Clopidogrel vs Aspirin in Patients at Risk of Ischemic Events (CAPRIE)
   - 19,185 patients with prior stroke, MI or PAD
   - Clopidogrel 75 mg/day vs aspirin 325 mg/day
   - 8.7% RRR vs aspirin for combined endpoint of stroke, MI and vascular death

2. Clopidogrel and Aspirin combination (Management of Atherothrombosis with Clopidogrel in High-Risk Patients with TIA or Stroke [MATCH])
   - 7,599 patients with prior stroke or TIA and additional risk factors
   - Clopidogrel-aspirin 75 mg/75 mg vs clopidogrel 75 mg
   - No significant difference in composite outcome of ischemic stroke, MI, vascular death or rehospitalization secondary to ischemic events
3. Clopidogrel for High Atherothrombotic Risk and Ischemic Stabilization, Management and Avoidance (CHARISMA)
   - 15,603 patients with either clinically evident cardiovascular disease or multiple risk factors
   - Clopidogrel 75 mg with low-dose aspirin (75-162 mg) vs low-dose aspirin only
   - Overall, clopidogrel/aspirin combination was not significantly more effective than aspirin alone in reducing rate of MI, stroke or vascular death
   - Suggestion of benefit with combination treatment in patients with symptomatic atherothrombosis

D. Cilostazol
   Cilostazol Stroke Prevention Study (CSPS)
   - 1,095 patients with cerebral infarction in the past 6 months
   - Cilostazol 100 mg bid vs placebo
   - 41.7% RRR for recurrent stroke

E. Dipyridamole and aspirin combination
   1. European Stroke Prevention Study (ESPS 2)
      - 6,602 patients with recent TIA or stroke randomized to placebo, aspirin 25 mg bid, extended-release dipyridamole 200 mg bid, or aspirin 25 mg + ER-dipyridamole 200 mg bid
      - Aspirin better than placebo; dipyridamole better than placebo; combination treatment better than either agent alone.
      - 37.8% risk reduction for stroke with combination therapy over placebo
      - No increased risk of major bleeding with combination treatment

   2. European/Australasian Stroke Prevention in Reversible Ischemia Trial (ESPRIT Trial)
      - 2,739 patients with recent TIA or minor stroke of arterial origin randomized to aspirin 30-325 mg/day or aspirin 30-325 mg od + dipyridamole 200 mg bid
      - 20% risk reduction for composite outcome of stroke, MI, vascular death with combination therapy
      - No increased risk of major bleeding with combination therapy

Bibliography


APPENDIX IX

Management of Increased Intracranial Pressure

A. Signs and symptoms of increased ICP
   1. Deteriorating level of sensorium
   2. Cushing’s triad
      i. Hypertension
      ii. Bradycardia
      iii. Irregular respiration
   3. Anisocoria

B. Management options for increased ICP

   General
   1. Control agitation and pain with short-acting medications, such as NSAIDS and opioids.
   2. Control fever. Avoid hyperthermia.
   3. Control seizures if present. May treat with phenytoin with a loading dose of 18-20 mg/kg IV then maintained at 3-5 mg/kg. Status epilepticus should be managed accordingly.
   4. Strict glucose control between 80-110 mg/dL
   5. No dextrose-containing IVF. Hyperglycemia may extend ischemic zone (penumbra) and further cause cerebral edema
   6. Use stool softeners to prevent straining.

   Specific
   1. Elevate the head at 30 to 45 degrees to assist venous drainage.
   2. Give osmotic diuretics: Mannitol 20% loading dose at 1 g/kg, maintenance dose at 0.5-0.75 mg/kg) to decrease intravascular volume and free water.
   3. Lost fluids must be replaced. Hypertonic saline is an option and has the advantage of maintaining an effective serum gradient for a prolonged period with lower incidence of rebound intracranial hypertension. Aim for serum osmolarity=310 mOsm/L. (Serum osmolarity = 2 (Na) + Glucose/18 + BUN /2.8)
   4. Hyperventilate only in impending herniation by adjusting tidal volume and pCO2 between 25 to 30. This maneuver is usually effective only for approximately 6 hours. Otherwise maintain normal pCO2 between 35 and 40.
   5. Carefully intubate patients with GCS 8 or less, or those unable to protect the airway.
   6. Do CSF drainage in patients with intraventricular hemorrhage (IVH) or hydrocephalus.
   7. Use barbiturates if all other measures fail. Available locally is thiopental (loading dose=10 mg/kg, maintenance dose titrated at 1-12 mg/kg/hour continuous infusion to achieve burst suppression pattern in EEG)
8. Consider surgical evacuation for mass lesions.
9. Consider decompressive hemicraniectomy in cases of malignant middle cerebral artery infarcts

C. Sedatives and Narcotics Available Locally

<table>
<thead>
<tr>
<th>Drugs</th>
<th>Usual Dose</th>
<th>Onset of Action</th>
<th>Duration of Effect</th>
<th>Comments</th>
<th>Availability/Dilution</th>
</tr>
</thead>
<tbody>
<tr>
<td>Midazolam</td>
<td>0.025-0.35 mg/kg</td>
<td>1 to 5 min</td>
<td>2 hours</td>
<td>Unpredictable sedation</td>
<td>15 mg/3 mL amp; 5 mg/5 mL amp; 50 mg in 100 mL NSS/D5W</td>
</tr>
<tr>
<td>Diazepam</td>
<td>0.1-0.2 mg/kg</td>
<td>Immediate</td>
<td>20 to 30 minutes</td>
<td>Sedation can be reversed with flumazenil (0.2-1 mg at 0.2 mg/min at 20 min interval, max dose 3 mg in one hour)</td>
<td>10 mg/2 mL amp; 50 mg in 250 mL NSS/D5W</td>
</tr>
<tr>
<td>Propofol</td>
<td>5-50 ug/kg/min</td>
<td>&lt;40 secs</td>
<td>10 to 15 min</td>
<td>Expensive</td>
<td>(10 mg/mL) 100 mL vial (premixed)</td>
</tr>
<tr>
<td>Ketorolac</td>
<td>50-100 mg IV</td>
<td>1 hour</td>
<td>6 to 8 hours</td>
<td>NSAID</td>
<td>30 mg/mL amp</td>
</tr>
<tr>
<td>Tramadol</td>
<td>50-100 mg IV</td>
<td>1 hour</td>
<td>9 hours</td>
<td>Centrally acting synthetic analgesic compound not chemically related to opiates but thought to bind to opioid receptors and inhibit reuptake of NE and serotonin</td>
<td>50 mg/2 mL amp; 100 mg/2 mL amp</td>
</tr>
<tr>
<td>Fentanyl</td>
<td>50-100 ug/hour</td>
<td>1-2 mins</td>
<td>&gt;60 min</td>
<td>Can be easily reversed with naloxone (0.4-2 mg IVP; repeat at 2-3 min intervals, max dose 10 mg) * 110x more potent than morphine</td>
<td>100 ug/2 mL; 2,500 ug in 250 mL NSS/D5W</td>
</tr>
<tr>
<td>Morphine</td>
<td>2.5 mg/hour</td>
<td>5 mins</td>
<td>&gt;60 min</td>
<td>Opioid</td>
<td>10 mg/mL gr 1/6; 16 mg/mL gr 1/4</td>
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</tbody>
</table>

**Bibliography**

Special Section: Guidelines for the
MANAGEMENT OF ANEURYSMAL SUBARACHNOID HEMORRHAGE
MANAGEMENT OF ANEURYSMAL SUBARACHNOID HEMORRHAGE

I. DIAGNOSIS OF SUBARACHNOID HEMORRHAGE (SAH)
   Clinical – may present with sudden, severe headache (thunderclap headache), loss of consciousness or adult-onset seizures.
   Neurological examination – signs of meningeal irritation (i.e., neck rigidity), altered or decreased level of consciousness, CN III or VI nerve palsy.
   Patients may or may not have focal neurological deficits

   Emergent referral to a neurologist/neurosurgeon and transfer to a facility with capabilities of managing acute stroke are recommended.

II. NEURODIAGNOSTIC EXAMINATIONS
   1. Non-contrast cranial CT scan should be done and interpreted immediately. Hyperdense blood in the basal cisterns is usually diagnostic, but parenchymal clot in the temporal or basal frontal, and intraventricular hemorrhage are also suggestive of an underlying aneurysm.

   2. Lumbar tap with CSF analysis in the absence of focal neurological signs is an option in the following cases:
      - Cranial CT scan is negative
      - Cranial CT scan is unavailable
      - Special circumstances (e.g., issues with CT scan cost)
      Multiple specimens (at least 3 tubes) should be collected to rule out traumatic tap. Opening pressures should be measured.

   3. Cerebral angiogram is the gold standard in determining the cause of SAH. Early angiography should be performed in all cases, whether poor- or good-grade SAH. If the initial angiogram is negative, a repeat angiogram should be performed after 2 weeks.

   4. Good-quality CT angiogram and MR angiography are other options.

III. SAH GRADING
   1. Hunt and Hess Classification is recommended for the clinical grading of SAH.

   Table 9: Hunt and Hess Classification

<table>
<thead>
<tr>
<th>Grade</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Asymptomatic, or mild headache, slight nuchal rigidity</td>
</tr>
<tr>
<td>2</td>
<td>Moderate to severe headache, nuchal rigidity, no neurologic deficit other than cranial nerve palsy</td>
</tr>
<tr>
<td>3</td>
<td>Drowsiness, confusion, or mild focal signs</td>
</tr>
<tr>
<td>4</td>
<td>Stupor, moderate to severe hemiparesis, possibly early</td>
</tr>
<tr>
<td>Grade</td>
<td>Description (Blood on CT)</td>
</tr>
<tr>
<td>-------</td>
<td>---------------------------------------------------------------</td>
</tr>
<tr>
<td>1</td>
<td>No subarachnoid blood detected</td>
</tr>
<tr>
<td>2</td>
<td>Diffuse or vertical layers &lt;1 mm thick*</td>
</tr>
<tr>
<td>3</td>
<td>Localized clot or vertical layer &gt;1 mm thick*</td>
</tr>
<tr>
<td>4</td>
<td>Intracerebral or intraventricular clot with diffuse or no SAH</td>
</tr>
</tbody>
</table>

**Vertical layer** refers to blood in the subarachnoid spaces including in the interhemispheric fissure and cisterns

2. Fisher grading may be used as a guide in considering therapeutic options.

**Table 10: Fisher Grading**

IV. GENERAL SYMPTOMATIC TREATMENT

1. Absolute bed rest in a quiet, comfortable environment
2. Cardiac monitoring and frequent assessment of neuro-vital signs
3. Soft diet for alert patients, nasogastric-tube (NGT) feedings if with impaired consciousness
4. Analgesics, including opiates for headache. Avoid aspirin and other NSAIDs
5. Paracetamol and cooling blankets, if febrile
6. Maintenance of euglycemia
7. Sedatives, if agitated
8. Antiemetics, as needed for nausea and vomiting
9. Stool softeners

V. EARLY SPECIFIC TREATMENT

1. Nimodipine: A systematic review showed a significant reduction in poor outcomes with calcium antagonists for SAH. The evidence rests mainly on one large trial with oral nimodipine. It is uncertain whether nimodipine acts through neuroprotection or through reduction of the frequency of vasospasm, or both. Nimodipine 60 mg every 4 hours by mouth or NGT for 3 weeks is recommended.

2. Anticonvulsants: Short-term anticonvulsants are recommended for patients with documented seizures in the acute phase of SAH. Although no randomized trial has proven that prophylactic anticonvulsants in SAH is effective, they can be considered in patients with significant cortical damage, thick cisternal clot, parenchymal hemorrhage, or those in coma. Phenytoin is the recommended anticonvulsant, given as a 15 mg/kg IV loading dose followed by 3 to 5 mg/kg/day in divided doses.

3. Steroids: Corticosteroids have no proven role and are not recommended for use in SAH.
4. Antifibrinolytic agents are not recommended. Although they reduce the risk of rebleeding, they are associated with a higher rate of cerebral ischemia.
5. Manage increased ICP
6. BP management: Although the best antihypertensive agent and BP remains unsettled, IV nicardipine to a target SBP<150 in the pre-operative phase is reasonable.

VI. PREVENTION AND MANAGEMENT OF VASOSPASM
1. Monitoring: Serial transcranial Doppler (TCD) is recommended for the diagnosis and monitoring of vasospasm.
2. Maintenance of euvolemia: Evidence on the use of blood volume expansion alone or in combination with induced hypertension and hemodilution (triple H therapy) in the prophylaxis and management of secondary ischemia (vasospasm) following aneurysmal SAH is lacking.
3. Endovascular angioplasty (chemical +/- mechanical) is an effective way of managing vasospasm. Intervention has to be early before clinical signs suggesting irreversible infarction (i.e., hemiplegia) are present.
4. Treatment strategies undergoing current investigation include intravenous magnesium sulfate and statins.

VII. TREATMENT OF SAH
Excluding the aneurysm from the circulation is the main goal of treatment. Obliteration the aneurysm can be achieved through surgical clipping or endovascular coiling.

VIII. TIMING OF SURGERY
1. Definitions:
   Early surgery is surgery performed within 72 hours from ictus
   Late surgery is surgery performed more than 3 days from ictus.
2. Indications:
   a. Early, immediate surgery is recommended for good- to moderate-grade (Hunt and Hess I-III) aneurysmal SAH patients to minimize the chance of a devastating rebleed.
   b. For poor grade patients (Grade IV-V), early surgery is recommended in the presence of:
      – Hematoma
      – Hydrocephalus

   Surgery may be delayed in the presence of:
   – Ischemia or infarction
   – Severe angiographic vasospasm
– Casted ventricles
– Diffuse SAH (Fisher Grade III) Complex aneurysm on angiography.

C. A maximum cut-off age for early surgical management (for the elderly) is not recommended in the absence of organ failure.

IX. COILING
1. Can be performed early in both good- and poor-grade patients.
2. Reduces the rebleed rate for poor-grade patients who would otherwise be treated conservatively.
3. Vasospasm is not a contraindication and can be dealt with endovascularly during coiling.
4. Can be performed under local anesthesia if needed.

X. WHERE TO ADMIT
SAH patients should be admitted at the Stroke Unit or Intensive Care Unit. In the absence of an ASU/ICU, patients may be placed in a quiet, regular room with very close monitoring.

XI. NURSING ISSUES:
SOLICITUDE, THOUGHTFULNESS, PROTECTIVENESS
1. Acute Phase
   a. Care must be exercised to prevent further ICP increase
   b. There should be close monitoring of fluid status and for possible secondary cardiac, respiratory or metabolic insults.
   c. Persistent headache, deteriorating level of consciousness, other signs of increased ICP and development of focal neurological deficits should be recognized for urgent referral to a neurologist or neurosurgeon.

2. Convalescent Phase
   a. Presence of sensory or perceptual alterations, motor deficits, and impaired verbal communication and physical mobility should be addressed in nursing care.
   b. Feelings of depression should be monitored. Emotional support and encouragement should be provided.
   c. Upon discharge, patients and relatives should be educated on continuity of care, medication intake and follow-up

XII. REHABILITATION
1. Supportive rehabilitation is done initially in the pre-operative phase.
2. Early rehabilitation is recommended for all SAH patients in the post-operative period.

Bibliography


Guidelines for STROKE REHABILITATION
STROKE REHABILITATION

I. ACUTE POST-STROKE REHABILITATION

Patient with stroke during acute phase

Obtain medical history and physical examination. Initial assessment of complications, impairment and rehabilitation needs including NIHSS, GCS, MRS, FIMS/Barthel Index.

Initiate secondary prevention and prevention of complications.

Acute post-stroke patient assessed for rehabilitation services.

Obtain medical history and physical examination. Determine nature and extent of rehabilitation needs and services based on stroke severity, functional status and social support.

MILD STROKE

Go to II, III or IV.

MODERATE STROKE

Go to II.

SEVERE STROKE

Go to II.

A. Initial Brief Assessment

Assessment for complications and prior and current impairment:
1. Risk factors for recurrent stroke and coronary heart disease
2. Medical comorbidities (DM, hypertension, increase ICP, re-bleed, re-stroke)
3. Consciousness and cognitive status
4. Brief swallowing assessment
5. Skin assessment and pressure ulcers
6. Mobility and need for assistance of movement
7. Deep-vein thrombosis (DVT) risk assessment

B. Assessment of Rehabilitation Needs
1. Prevention of complications: swallowing problems, skin breakdown, DVT, bowel and bladder dysfunction, malnutrition, pain, contractures, SHS/CRP, pulmonary.
2. Assessment of impairments: communication impairments, motor impairment, cognitive deficits, visual and spatial deficiency, psychological or emotional deficits, sensory deficits.
3. Psychosocial assessment and family or caregivers support
4. Assessment of function (e.g., functional independence measure or FIM).
5. Financial support.
II. INPATIENT REHABILITATION

Post-stroke patient in inpatient rehabilitation

Determine level of care based on medical status, cognitive and motor function, social support, and access to care and services.

Discuss rehabilitation program with patient and family. Determine and document treatment plan

Initiate rehabilitation programs and interventions.

Reassess progress, future needs and risks with team.

Is patient progressing toward treatment goals?

NO

Address treatment adherence and barriers to improvement. If medically unstable, refer to acute services. If with other health factors, refer to other health services.

Severe stroke and/or maximum dependence, or poor prognosis for functional recovery?

YES

Educate patient, family, caregiver about future plans and home therapy. Discharge patient to the home or community. Go to IV.

NO

Continue inpatient rehabilitation.

YES

Is patient ready for community living?

NO

Go to IV.

YES

Go to III.
A. Reassessment of Rehabilitation Progress

1. General (medical status)
2. Functional status (FIM, etc.): Mobility, activities of daily living (ADL) and instrumental ADLs, communication, nutrition, cognition, mood/affect/motivation, sexual function
3. Family support: Resources, caretaker, transportation
4. Patient and family adjustment
5. Reassessment of goals
6. Risk for recurrent cerebrovascular events
III. OUTPATIENT REHABILITATION

Post-stroke patient ready for home

Does patient need outpatient rehabilitation services?

YES

Reassess progress, rehabilitation interventions and optimal environment for outpatient rehabilitation.

Discuss shared decision regarding rehabilitation program and treatment plan with patient and family. Continue secondary prevention.

Continue rehabilitation intervention at nearby center or hospital, or use home rehabilitation services.

Did patient plateau or achieve optimal function?

YES

NO

Reassess periodically.

NO

Go to IV.

NO

Go to IV.
A. Assessment of Discharge Environment
   1. Functional needs
   2. Motivation and preferences
   3. Intensity of tolerable treatment: Equipment, duration
   4. Availability and eligibility
   5. Transportation
   6. Home assessment for safety
IV. COMMUNITY–BASED REHABILITATION

Post-stroke patient ready for community living

Does patient need community-based rehabilitation services?

YES

Determine optimal environment for community-based rehabilitation.

Discuss shared decision regarding rehabilitation program and treatment plan with patient and family.

Continue rehabilitation intervention with patient and family/caregiver education.

Did patient plateau or achieve optimal function?

YES

NO

Reassess periodically.

NO

YES

Discharge to the home or community setting. Arrange primary-care follow-up. Provide home rehabilitation program.

Arrange primary-care follow-up. Provide home rehabilitation program.
A. Assessment of Discharge Environment
   1. Functional needs
   2. Motivation and preferences
   3. Intensity of tolerable treatment: Equipment, duration
   4. Availability and eligibility
   5. Transportation
   6. Home assessment for safety
   7. Maximal patient functioning
Guidelines for the
NURSING MANAGEMENT of STROKE PATIENTS
## I. PREVENTIVE CARE

<table>
<thead>
<tr>
<th>General Objective</th>
<th>Specific Objectives</th>
<th>Process</th>
<th>Outcome</th>
</tr>
</thead>
</table>
| 1. Nurses will provide preventive care through health education activities based on identified learning needs. | Provide information on stroke, risk factors, lifestyle modification and regular medical check-ups.     | 1. A nurse will implement a health education program (HEP) approved or published by a duly accredited or recognized health agency.  
2. A nurse will implement the HEP appropriate for the people’s level of understanding.  
3. A nurse will use appropriate, available teaching material.  
4. A nurse will actively participate in fora on health education on stroke prevention. | 1. People understand risk factors and show interest in modifying lifestyle.  
2. Incidence of stroke decreases.  
3. Awareness increased.  
4. Use of published materials increase participation in stroke prevention fora. |
| 2. Nurses will actively identify patients with risk factors.                       | Identify people who are at risk for developing stroke.                                                  | 1. The nurse will implement assessment based on established guidelines on stroke risk factors and will use a risk-assessment nursing framework.  
2. The nurse will appropriately refer identified people high risk for stroke.  
3. The nurse will report and document identified people high risk for stroke. | 1. Early detection, referral and management of identified at-risk people.  
2. Risk identification implemented in a standardized manner.  
3. Contribute factual, accurate data due to existing |
<table>
<thead>
<tr>
<th>General Objective</th>
<th>Specific Objectives</th>
<th>Process</th>
<th>Outcome</th>
</tr>
</thead>
</table>
| 3. Nurses will be actively involved in HEP regarding lifestyle modification. | Identify, promote and participate in available programs regarding lifestyle modification. | 1. The nurse will identify agencies in the community that have programs for lifestyle modification for stroke prevention.  
2. The nurse will recommend available programs for lifestyle modifications.  
3. The nurse will facilitate referral to appropriate community or health care agency regarding lifestyle modification. | 1. Increased awareness of available facilities and programs regarding lifestyle modification.  
2. Increased adherence to lifestyle modification. |

**II. CURATIVE CARE**

<table>
<thead>
<tr>
<th>General Objective</th>
<th>Specific Objectives</th>
<th>Process</th>
<th>Outcome</th>
</tr>
</thead>
</table>
| 1. Nurses will promptly identify patient’s needs by performing proper health assessment with emphasis on neurological assessment techniques. | 1. Promptly identify and prioritize patient’s needs.  
2. Use and perform proper neurological assessment techniques. | 1. Nurses will assess patients comprehensively using current and acceptable neurological assessment tools.  
2. Nurses will correlate patient’s history with signs and symptoms.  
3. Nurses will identify priority patient needs based on assessment.  
4. Nurses will prioritize and facilitate series of diagnostic examinations according to stroke guidelines and will have nursing responsibilities | 1. Early needs identification and prioritization.  
2. Immediate initiation of management.  
3. Early transfer and admission to hospital with stroke or intensive care unit. |
before, during and after the procedure, including patient safety and informed consent.

| 2. Nurses will provide quality nursing care based on the identified patient needs in collaboration with other member of the health team, utilizing a holistic approach. | Plan and manage nursing care based on patient’s condition, needs and priorities:  
- a. Physiologic care  
- b. Safe Measures  
- c. Comfort Measures  
- d. Therapeutic environment  
- e. Prevention of complications and infections  
- f. Spiritual and psychosocial care | 1. Nurses will implement emergency nursing measures if needed.  
2. Nurses will closely monitor, document and report neuro-vital signs.  
3. Nurses will provide safety measures, such as  
   a. Aspiration precautions  
   b. Fall precautions  
   c. Use of restraints  
   d. Seizure precautions  
4. Nurses will provide comfort measures, such as:  
   a. Linen changes  
   b. Personal hygiene  
   c. Turning  
   d. Proper positioning  
   e. Range of motion (ROM)  
5. Nurses will provide a therapeutic environment  
   a. Proper ventilation and lighting  
   b. Minimize noise  
   c. Proper orientation to time, place and person  
   d. Provisions of window murals in every room  
6. Nurses will prevent complications and possible infections by:  
   a. Establishing patent airway  
   b. Monitoring and maintaining BP  
   c. Observing and | 1. Early identification and assessment of disease progression  
2. No incidence of falls and aspirations.  
3. No bedsores, contractures and muscular atrophy.  
5. Complications and infections prevented  
6. Psycho-emotional and spiritual upliftment  
7. No medication errors; medications adhere to.  
8. Self expression using alternative means, if |
providing catheter and tube care
d. Monitoring input and output
e. Monitoring and prevention of increased ICP
f. Nutrition and hydration

7. Nurses will provide spiritual and psychosocial care:
   a. Alleviation of anxiety by encouraging verbalization of feelings
   b. Guidance in identifying positive coping mechanisms
   c. Respect of patient’s beliefs and culture
   d. Facilitation of patient’s spiritual needs

8. Nurses will apply the principles of Bioethics in the Practice of Nursing Care.

9. Nurses will administer medications observing the 10 R’s.

10. Nurses will establish alternative means of communication if necessary.

11. Nurses will assess patient’s capabilities in performing ADLs and assist in identifying alternative means.

9. Patient’s independent functions maximized; disabilities correctly addressed.

10. Cooperation and active participation of clients and significant others.

11. Complete, accurate records

12. Early medical intervention

13. Utilization of other health and community resources.

III. REHABILITATIVE AND PROMOTIVE CARE
<table>
<thead>
<tr>
<th>General Objective</th>
<th>Specific Objectives</th>
<th>Process</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Nurses will focus on early rehabilitation and discharge planning.</td>
<td>1. Assist the patient towards maximum functional capacity. 2. Discuss the care plan with the patient and significant others. 3. Involve the patient's family and significant others in decision making and the care plan.</td>
<td>1. Nurses will initiate rehabilitation upon admission. 2. Nurses will assist the patient in performing ADLs in collaboration with other health team members. 3. Nurses will educate patient on alternative, physiologically safe sexual practice (as indicated). 4. Nurse will include significant others in providing specific nursing care, such as provisions of hygiene, nutrition, turning, positioning, pulmonary toile, ROM exercises, and other care. 5. Nurses will ensure good compliance to medications and provide options for compliance to outpatient follow-up 6. The nurse will collaborate with the family and significant others in the care plan.</td>
<td>1. Performance of simple ROM exercises and ADLs by patient with minimal or no supervision. 2. Maintenance of sexual function. 3. Performance of simple nursing procedures by significant others with minimal or no supervision from nurses. 4. Compliance to treatment regimen and adherence to outpatient follow-up 5. Active participation of patient and family in care plan.</td>
</tr>
<tr>
<td>2. Nurses will assist in sustaining and maintaining patient's healthy, productive lifestyle.</td>
<td>1. Provide guidelines for home care. 2. Guide patient in lifestyle modification based on identified risk</td>
<td>1. Nurses will provide a discharge care plan containing the following: a. Activity and exercise b. Medication regimen c. Symptoms needing referral d. Prescribed diet e. Medical follow-up</td>
<td>1. Adherence of patient and family to prescribed discharge care plan. 2. Compliance to alternative</td>
</tr>
<tr>
<td>Factors</td>
<td>Schedule</td>
<td>Lifestyle</td>
<td></td>
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<tr>
<td>3. Assist patient in accepting and adapting to disability.</td>
<td>f. Special care to be provided</td>
<td>3. Motivation and stimulation of patient’s interest in self-enhancing activities.</td>
<td></td>
</tr>
<tr>
<td>2. Nurses will facilitate referrals to community resources.</td>
<td></td>
<td>4. Maximal patient potential.</td>
<td></td>
</tr>
<tr>
<td>3. Nurses will identify appropriate lifestyle modification suited to the patient’s current status.</td>
<td></td>
<td>5. Active participation of family members.</td>
<td></td>
</tr>
<tr>
<td>4. Nurses will involve patient in diversion activities that will enhance self-esteem.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. Nurses will involve family member in the care plan.</td>
<td></td>
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</tbody>
</table>
Guidelines for the ESTABLISHMENT and OPERATION of STROKE UNITS
I. THE STROKE CENTER
A. Major Aspects of Acute Stroke Care in Stroke Center

1. **Acute Stroke Teams:** Hospital-based stroke teams should be available round-the-clock, seven days a week in order to evaluate within 15 minutes any patient who may have suffered a stroke.

2. **Written Care Protocols:** The availability of written protocols is key in reducing time to treatment and treatment complications.

3. **Emergency Medical Services:** Emergency medical services (EMS) are vital in the rapid transport and survival of stroke patients.

4. **Emergency Department:** The emergency department staff should be trained in diagnosing and treating stroke and have good lines of communication with both EMS and the acute stroke team.

5. **Stroke Unit:** Where patients can receive specialized monitoring and care.

6. **Neurosurgical Services:** These should be provided to stroke patients within two hours of when the services are deemed necessary.

7. **Support of the Medical Organization:** The facility and its staff, including administration, should be committed to the Stroke Unit.

8. **Neuroimaging:** There must be capability to perform an imaging study within 25 minutes of the physician’s order. A physician should evaluate the image within 20 minutes of completion.

9. **Laboratory Services:** Standard laboratory services should be available round-the-clock, seven days per week at a Primary Stroke Center.

10. **Outcomes/Quality Improvement:** Primary Stroke Centers should have a database or registry for tracking the type and number of stroke patients seen, their treatments, timelines for treatments, and some measurements of patient outcome.

11. **Educational Programs:** The professional staff should receive at least eight hours per year of continuing medical education credits. In addition to professional education, the Stroke Center should plan and implement at least two annual programs to educate the public about stroke prevention, diagnosis and availability for emergency treatment.

B. Definition of a Stroke Unit
A Stroke Unit is a hospital unit that cares for stroke patients exclusively or almost exclusively, with specially trained staff and a multidisciplinary approach to treatment and care.\(^1\)

C. Characteristics of a Stroke Unit

Organization
- Coordinated multidisciplinary team care
- Nursing integration with multidisciplinary care
Involvement of caregivers in rehabilitation process

Specialization
- Medical and nursing interest
- Expertise in stroke and rehabilitation

Education
- Education and training program for staff, patients and caregivers

D. Goals of a Stroke Unit
1. Improve chances of survival
2. Reduce disability
3. Shorten length of hospital stay
4. Shorten length of rehabilitation

E. Types of Stroke Units

E1. Acute Admission Units:
1. Intensive Care Units – dedicated stroke unit with facilities such as ventilators and intensive invasive and non-invasive monitoring equipment. The units focus on the very acute care for a selected group of acute stroke patients and have little or no focus on rehabilitation.

2. Acute stroke unit – dedicated stroke units that accept patients acutely but discharge them early (within 7 days) and have no or at best a modest focus on rehabilitation. The units usually do not have intensive care facilities, but usually have facilities for non-invasive monitoring of vital signs.

3. Combined acute/rehabilitation stroke unit – dedicated stroke units which accept stroke patients acutely for acute treatment combined with early mobilization and rehabilitation for an average period of at least one to two weeks.

4. Mixed acute units – units that treat stroke patients and patients with other diagnoses. The units accept patients acutely. Some have a program of care similar to acute stroke units while others have a program similar to a combined unit.

E2. Delayed admission unit
1. Rehabilitation stroke unit – dedicated units that accept patients after a minimum delay of seven days after stroke onset. The units focus on rehabilitation.

2. Mixed assessment/rehabilitation unit – wards or units which have an interest and expertise in the assessment and rehabilitation of disabling illness, but do not exclusively manage stroke patients.

E. Effects of Stroke Unit Care on Recovery
Analysis on Cochrane Data Base involving 23 trials showed significant reduction of death (OR; 0.88), death or dependency (OR; 0.75) and death or institutionalization (OR; 0.77) when patients were treated in a stroke unit compared with those treated in general wards.²

Two trials evaluated the long-term effects of stroke unit care. On the 5-year follow-up, admission in combined acute/rehabilitation stroke units reduced death (OR; 0.59, NNT=9), death or dependency (OR; 0.36, NNT=6) and death or institutionalization (OR; 0.48, NNT=9). Ten-year follow-up of patients admitted in combined acute/rehabilitation stroke units similarly showed a reduction in death (OR; 0.45), death or dependency (OR; 0.45) and death or institutionalization (OR;0.42).³⁻⁵

Patients admitted in a rehabilitation stroke unit even after a minimum delay of seven days post-stroke resulted in reduced death (OR; 0.66, NNT=10) and death or dependency (OR; 0.83, NNT=90).⁶

The stroke unit benefits stroke patients of both sexes, all ages, and those with mild, moderate or severe strokes.²,⁷

Comparing the different stroke unit models, the unit with the strongest evidence of benefit is the combined acute/rehabilitation stroke-unit model, and to some extent the dedicated rehabilitation stroke unit.²

II. STROKE UNIT ORGANIZATION

A. The Stroke Unit:
Basic Equipment:
1. 4 to 8 beds
2. Cranial computerized tomography (available 24 hours)
3. Angiography (available 24 hours)
4. Ultrasound (continuous-wave, TC Duplex, transthoracic echocardiogram; transesophageal echocardiogram)
5. Monitoring (RR, Respiration, Holter, O₂ saturation)
6. Emergency laboratory

Monitoring:
1. Basic – Holter, blood pressure, O₂ saturation, respiration, temperature
2. Special – Transcranial Doppler, embolus detection, electroencephalography, central breathing patterns (sleep apnea)

B. Tasks
1. Admission within the unstable phase (in general, <24 hours)
2. Monitoring of vital and neurological parameters
3. Immediate diagnosis (etiology, pathogenesis)
4. Immediate treatment and secondary prevention
5. In general, length of stay not longer than seven days
C. Patient Selection

1. Indications for Admission to the Stroke Unit
   a. Acute stroke (< 24 hours)
   b. Awake, somnolent patient
   c. Symptoms fluctuating or progressive
   d. TIA with high stroke risk (non-valvular AF, stenosis)
   e. Vital parameters unstable
   f. Thrombolysis, Anticoagulation
   g. New investigational treatment or procedure

2. Admission to Acute Stroke Unit Not Indicated
   a. Patients with severe consciousness impairment (should be admitted to intensive care unit instead)
   b. Severely disabled patients by previous strokes
   c. Very old patients or those with multiple comorbidities

3. Patients with the following should be admitted to the intensive care unit instead of the acute stroke unit:
   a. Stupor and coma
   b. Central respiratory disorders requiring artificial ventilation
   c. Space-occupying cerebral infarctions with risk of herniation
   d. Severe cardiopulmonary insufficiency
   e. Hypertensive-hypervolemic treatment

D. The Stroke Team

1. Personnel
   a. Medical doctors
   b. Nurses
   c. Physiatrists
   d. Occupational therapists
   e. Speech pathologist
   f. Nutritionists
   g. Social workers

2. Personnel with special interest in stroke are medical doctors or other paramedical people who:
   a. Have undergone continuing education on stroke and other related activities or subspecialties on stroke
   b. Have been attending at least one national or international meeting on stroke in a year
   c. Have undergone stroke fellowship or preceptorship training on stroke
   d. Is a member or officer of a national or international organization devoted to stroke
III. HOSPITALS IN THE PHILIPPINES WITH ACUTE STROKE UNITS

Table 11.

<table>
<thead>
<tr>
<th>Hospitals</th>
<th>Stroke Unit Type</th>
<th>Contact Number</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Metro Manila</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>East Avenue Medical Center</td>
<td>Mixed acute units</td>
<td>9280611 loc.503</td>
</tr>
<tr>
<td>Jose Reyes Memorial Medical Center</td>
<td>ASU</td>
<td>7119491 loc 262</td>
</tr>
<tr>
<td>Makati Medical Center</td>
<td>ASU</td>
<td></td>
</tr>
<tr>
<td>Manila Adventist Medical Center</td>
<td>Mixed acute units</td>
<td>5259191 loc 324</td>
</tr>
<tr>
<td>Manila Doctors Hospital</td>
<td>ASU</td>
<td>5243011</td>
</tr>
<tr>
<td>Manila Central University</td>
<td>Mixed acute units</td>
<td>3672031 loc 1127</td>
</tr>
<tr>
<td>Philippine General Hospital</td>
<td>ASU</td>
<td>5218450 loc 2406</td>
</tr>
<tr>
<td>Philippine Heart Center</td>
<td>Mixed acute units</td>
<td>9252401 loc 2483</td>
</tr>
<tr>
<td>San Juan de Dios Medical Center</td>
<td>Mixed acute units</td>
<td>8319731 loc 1226</td>
</tr>
<tr>
<td>St. Luke’s Medical Center</td>
<td>ASU</td>
<td>7230101 loc 7399</td>
</tr>
<tr>
<td>Sto. Tomas University Hospital</td>
<td>ASU</td>
<td>7313001 loc 2368</td>
</tr>
<tr>
<td>The Medical City</td>
<td>Mixed acute units</td>
<td>6356789 loc 6281</td>
</tr>
<tr>
<td><strong>Luzon</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mt. Carmel Diocesan General Hospital, Lucena</td>
<td>Mixed acute units</td>
<td>042-7102576</td>
</tr>
<tr>
<td>Lorma Medical Center, San Fernando, La Union</td>
<td>Mixed acute units</td>
<td>072-700-0000</td>
</tr>
<tr>
<td>Lucena United Doctors Hospital</td>
<td>ASU</td>
<td>042-3736161</td>
</tr>
<tr>
<td><strong>Cebu</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cebu Doctors Hospital</td>
<td>ASU</td>
<td>032-2555555</td>
</tr>
<tr>
<td>Chong Hua Hospital</td>
<td>Mixed acute units</td>
<td>032-2541461</td>
</tr>
</tbody>
</table>

ASU, acute stroke unit.

IV. RECOMMENDATIONS

Stroke patients should be treated in stroke units (Level I). Admission to stroke unit decreases death, dependency and institutionalization.

Stroke units should provide coordinated multidisciplinary care provided by medical, nursing and therapy staff who specialize in stroke care (Level I).

Bibliography


**STRATEGY FOR IMPLEMENTATION OF GUIDELINES**

To effectively implement the guidelines set forth in the previous sections, it is recommended that Stroke Centers be established in every region. Stroke Centers shall be designated according to levels as follows:

<table>
<thead>
<tr>
<th>STROKE CENTER LEVEL</th>
<th>REQUIREMENTS</th>
<th>ACTIVITIES</th>
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</thead>
<tbody>
<tr>
<td>I</td>
<td>Basic: &lt;br&gt; Physician/Municipal Health Officer &lt;br&gt; Optional: &lt;br&gt; Nurse/Midwife &lt;br&gt; Barangay Health Worker &lt;br&gt; Facilities: &lt;br&gt; Municipal Health Clinic</td>
<td>Stroke prevention and public education &lt;br&gt; Recognition of stroke &lt;br&gt; Acute treatment of TIA and mild stroke &lt;br&gt; Referral of moderate and severe strokes to Levels II or III centers &lt;br&gt; Referral to Levels II or III centers for diagnostic tests &lt;br&gt; Rehabilitation &lt;br&gt; Secondary prevention of stroke and follow-up visits</td>
</tr>
<tr>
<td>II</td>
<td>Basic: &lt;br&gt; Neurologist (If not available, other physicians with special training in stroke) &lt;br&gt; Neurosurgeon &lt;br&gt; Stroke nurse &lt;br&gt; Radiologist &lt;br&gt; Physiatrist &lt;br&gt; Facilities*: &lt;br&gt; CT scan &lt;br&gt; Electrocardiogram &lt;br&gt; Laboratory &lt;br&gt; Stroke Team/Unit &lt;br&gt; Operating Room</td>
<td>Stroke prevention and public education &lt;br&gt; Recognition of stroke &lt;br&gt; Acute treatment of TIA, mild, moderate, and severe strokes &lt;br&gt; Referral of complicated strokes to Level III centers &lt;br&gt; Referral to Level III centers for further diagnostic tests &lt;br&gt; Rehabilitation &lt;br&gt; Secondary prevention of stroke and follow-up visits</td>
</tr>
</tbody>
</table>
III Basic:
- Neurologist
- Neurosurgeon
- Stroke nurse
- Neuroradiologist
- Vascular surgeon
- Cardiologist
- Neurosonologist
- Physiatrist

Stroke prevention and public education
Recognition of stroke
Acute treatment of TIA, mild, moderate, and severe strokes
Rehabilitation
Secondary prevention of stroke and follow-up visits
Training of stroke personnel
Research in stroke

Facilities:
- CT scan, MRI
- Cardiac diagnostic services (including ECG, Doppler, echocardiogram)
- Laboratory
- Angiography
- Stroke Unit
- Rehabilitation Unit
- Operating Room

### LEVELS OF STROKE CARE

<table>
<thead>
<tr>
<th>STROKE CENTER LEVEL</th>
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<tbody>
<tr>
<td>I</td>
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<tr>
<td>TIA or Mild Stroke</td>
</tr>
<tr>
<td>Moderate Stroke</td>
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<tr>
<td>Severe Stroke</td>
</tr>
</tbody>
</table>
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Dr. Rhoderick Casis         Dr. Gerardo Legaspi

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Dr. Maria Cristina San Jose
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SSP Annual Conventions

1st Philippine Congress on Brain on Brain Attack
Theme: Thinking Globally, Acting Locally
October 1-2, 1999
Manila Midtown Hotel

2nd Philippine Congress on Brain Attack
Theme: Organizing Stroke Services
Year 2001

3rd Philippine Congress on Brain Attack
Theme: Intracerebral Hemorrhage (ICH) and Subarachnoid Hemorrhage (SAH)
August 2002

4th SSP Biennial Convention
Theme: Ugaliing Tingnan, Ating Kalusugan, Upang Brain Attack ay Maiwasan
August 20-22, 2003
Bethel Guest House, Dumaguete City

5th SSP Annual Convention
Theme: Emerging Diagnostic Modalities & Therapeutic Interventions in Acute
Brain Attack
August 19-21, 2004
Taal Vista Hotel, Tagaytay City

6th SSP Annual Convention
Theme: SSP Goes to the Community
August 18-20, 2005
Fort Ilocandia Hotel, Laoag City

7th SSP Annual Convention
Theme: SSP goes to Mindanao: Empowering the Community for Optimal Stroke
Care
August 21-23, 2006
The Marco Polo Hotel, Davao City
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