Dedicated To
Dr Nora Ahmad Miswan & Dr Madiha
Medical Specialist, Hospital Ampang
Thank you for your tireless dedication and support towards housemen
I will forever remember your kindness and valuable ward teachings.

Special Thanks
Dr Sharma, Dr Rosaida
Dr Sharifah, Dr Oon, Dr Jaideep, Dr Hana, Dr Ummi, Dr Najib, Dr Ng, Dr Yuha
Dr Ho Thian Hao, Dr Yap Chiou Han, Dr Yung Chen Tong, Dr Firdaus, Dr Chin, Dr Grace, Dr Melinda,
Dr Jakclyn Daniels, Dr Farah Diyana, Dr Ranjitha, Dr Rahman, Dr Raudhah, Dr Aravind

And all the nursing staff and sisters of the Medical Dept
SN Azilawati your dedication is an example to us all

This guide was written as a guide to aid new Medical HO and serves as an introduction and quick reference only.
Always refer to the CPG for full guidelines recommended by The Malaysian Health Ministry.

Dept Of General Internal Medicine HA
Wards : 6B (male ward) , 6C (female ward) , 6D (dengue ward)
Gastro : Daycare (scope)
MOPD: 2nd floor
CCU, ICU, HDW: 3rd floor
ED: 1st floor

HO places of duty
1) Wards
2) Gastro Team
3) ED
4) Periphery
5) CCU/ICU/HDW

Contents

Introduction to GIM
- General Clerking & reviews

Short Notes compilation

Appendix
- Quick Reference
- Charts
- Medication List

The GIM HO guide
Compiled by Dr Gerard Loh October 2012 - 1st Edition
*more citation needed, to be updated in the near future

A project by the House Officers Workshop
Log on to www.myhow.wordpress.com for more guides and also The HOW Medical guide part I (2010)
**General Clerking**
– cases are usually seen in ED and referrals in periphery, clerking is done in eHIS as `<Generic Progress Note>`

*Start by type of review:*

**<medical review in: red zone/yellow zone/peri etc >**
s/b Dr Ho (seen by MO/specialist)

* Referred for..

**Current Problem/General Complaints**

- **Age/Race/Sex**
- **co-morbid(s) (k/c/o)-** underlying disease, years diagnosed, medication, current follow up, other issues...
  
  eg: DM 7 years, on OHA (T MTF 500mg TDS), under f/up KK Ampang

- **Presented with...** (p/w or c/o)
  
  Eg: p/w Cough 1/7 – productive with yellowish sputum, minimal amount, no blood

- **Otherwise**
  
  NO fever, No UTI sx, no abdominal pain etc

- **Social Hx**
  
  Occupation, Marital status, smoking/alcohol etc

- **Family Hx**
  
  Strong hx of DM/HPT/Stroke/Ca/IHD
  
  eg: mother died of IHD, 80 years old with underlying HPT

**Physical Examination**

- **O/e:** Alert, conscious, non tachypnoic, hydration fair etc

- **V/s:** BP, PR, RR, SpO2, T

  Lungs bibasal crepitations
  
  CVS S1S2 DRNM
  
  No pedal edema

**Analysis/assessment:**

- **ECG:** SR, no acute ischaemic changes

**Lab:**

- **LINK relevant results**

**Imaging:** comment on CXR, CT brain…etc

- **CXR clear lung fields**

**Medication:**

- **Pt’s old meds….. T MTF 500mg TDS…etc**

**Management:** *management/ plan carried out in ED*

- **eg: In ED given crushed Aspirin 300mg stat...**

**Diagnosis:** *give an impression*

- **Imp: 1) CAP**

**Plan**

- Admit ward 6B acute, IV Rocephine 2g stat BD, etc

**Investigations:**

- septic workout, FBC/RP/LFT etc..
Writing an entry in ward
- Admission review – must be done for all patients, and presented to specialist on call
- AM/PM/On-call review
- Transfer Out summary → for patients who are being transferred out to another ward (6D)
- Transfer In summary → for patients transferred in from other wards

7/5/12
7.30 pm

<Admission Review>

\[\text{Dr Ho @ 830 pm}\]

\[\text{Type of Review: Admission/ AM/ PM/ On Call}\]

\[\text{seen by MO/Specialist}\]

75 years old / Malay/ gentleman
k/o/c/o: 1) HPT - (10 years) on 1 Perindopril 2mg OD, =up KK Ampants
2) DM (10 years) on OHA, =up KK Ampants
...etc

current Imp: 1) CAP

p/w: 1) Cough 3/7 - productive with yellowish sputum....

otherwise
no fever, no LOA/LOW...etc

Social Hx ....

F Hx....

On Arrival in ED:
\(\text{O/C: Alert, conscious}\)
\(\text{BP 120/30}\)
\(\text{PR 88}\)
\(\text{RR 20}\)
\(\text{SpO2 99% RA}\)
\(\text{T 38}\)
\(\text{DXT 5.6}\)

\(\text{ECG: SR, no acute ischaemic changes...}\)
\(\text{CXR: bilateral haziness}\)
\(\text{FBC: Hb... WCC 13}\)
\(\text{RD: .....}\)
\(\text{LFT:.....}\)

\(\text{lungs clear}\)
\(\text{CVS DRNM}\)
\(\text{PA Soft non tender}\)
\(\text{no pedal edema}\)
NOTE:
Documentation is very important medico-legally, so make sure everything is documented properly!

- S/T MO/specialist….D/w specialist…. Requests for blood or radiology imaging…etc MUST be documented
- Any events, other than ward round reviews should be documented as <Ad-hoc entry>
- <retrospective entry> may be used if you are unable to document during time of the event, hence you may come back and document it later.
- when seen by MO/specialist, <case and progress noted> is written, followed by progress, physical examination and plan.
GASTRO ENDOSCOPY NOTES
- Scopes are done in OGDS and it is the Gastro Team’s duty to fill in notes and carry out orders by specialists
- Specialist performing the scope will usually write their findings in a book (Elective (outpatient) / Emergency (inpatient) )
- These findings and plans need to be entered into the eHis under pt’s clinical notes: Upper/Lower GI endoscopy notes

Eg:

< Upper GI Endoscopy notes >

<table>
<thead>
<tr>
<th>Lead Surgeon</th>
<th>Dr Jaideep</th>
<th>Role: Specialist</th>
</tr>
</thead>
</table>

**Indication for scope**
Anemia/Variceal Assessment/Bleeding etc (select from droplist)

**Endoscope findings:**

**Esophagus**
Z line 37cm, variceal bleeding? Esophagitis? etc

**Stomach:**
antral gastritis? Ulcer? Forrest classification…etc

**Duodenum:**
D1 D2 normal, duodenitis? etc

**Pronto test:** *negative or positive (test for Helicobacter Pylori)*

**Plan:**
C Omeprazole 40mg BD 6/52…etc
ACS (Acute Coronary Syndrome)
3 Criteria:
i) Chest pain (retrosternal, central, radiating to limbs/jaw)
ii) ECG changes (ST depression/elevation)
iii) Cardiac biomarkers elevated (Trop T, CK, CK-MB)

Further classified to **Unstable Angina/NSTEMI and STEMI**

<table>
<thead>
<tr>
<th>Cardiac biomarkers</th>
<th>UA</th>
<th>NSTEMI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Not elevated</td>
<td></td>
<td>elevated</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>ECG ST/T changes</th>
<th>UA</th>
<th>NSTEMI</th>
</tr>
</thead>
<tbody>
<tr>
<td>ST depression</td>
<td></td>
<td>ST depression</td>
</tr>
<tr>
<td>T inversion</td>
<td></td>
<td>T inversion</td>
</tr>
</tbody>
</table>

**Unstable Angina** = Chest pain + transient ECG changes + no cardiac biomarkers

**Class**
1) New onset severe angina, no rest pain
2) Angina at rest within 1/12, but not within 48hrs
   (angina at rest, subacute)
3) Angina at rest (last >20mins) within 48 hrs
   (acute angina)

1) Secondary to extracardiac disease
   - increased O2 demand (fever, tachycardia)
   - reduced coronary flow (hypotension)
   - reduced O2 delivery (anemia, hypoxemia)
2) Primary – dev in absence of extracardiac disease
3) Post infarct – dev within 2/52 of acute MI

- Uncontrolled hypertension, anaemia, thyrotoxicosis, severe aortic stenosis, hypertrophic cardiomyopathy and other co-morbid conditions such as lung disease should be identified.
- Presence of left ventricular failure (hypotension, respiratory crackles or S3 gallop)
- Carotid bruits or peripheral vascular disease indicates extensive atherosclerosis and a higher likelihood of concomitant CAD.

**NSTEMI** = chest pain + ECG ST/T changes + elevated cardiac biomarkers

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**Presentation**

**Provisional Diagnosis**

**ECG**

**Cardiac Biomarkers**

**Final Diagnosis**

**Ischemic Chest Discomfort**

**Acute Coronary Syndrome**

**No ST Elevation**

**ST Elevation**

**Unstable Angina**

**NSTEMI**

**STEMI**

**Myocardial Infarction**
History
1) Chest pain – retrosternal/central, burning/pressing/crushing, radiating to jaw/limbs, pain score ?/10, occur @ time, a/w sweating? Nausea/vomiting, lethargy
2) a/w Dyspnoea – NYHA class?, a/w orthopnea / PND?
3) a/w Failure symptoms – SOB, leg swelling

Fam Hx
- mother/father with IHD/DM/HPT/Stroke/Ca?
- died @ what age due to what disease
- genetic diseases?

Social Hx
- smoker – pack years, alcohol consumer?
- marital status, children
- occupation
- allergic hx – drugs/food

Physical Examination

<table>
<thead>
<tr>
<th>O/E</th>
<th>Lungs</th>
<th>CVS</th>
<th>PA</th>
<th>SOB</th>
<th>Leg swelling</th>
<th>Tongue coated</th>
</tr>
</thead>
<tbody>
<tr>
<td>general consciousness, GCS full?</td>
<td>clear / coarse crepts?</td>
<td>S1S2, murmurs?</td>
<td>soft, non tender</td>
<td>hepato splenomegaly?</td>
<td>Ascites?</td>
<td>pedal oedema?</td>
</tr>
<tr>
<td>tachypnoic?</td>
<td>Failure sx?</td>
<td>any pneumonic changes?</td>
<td>Fluid overload?</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>anemia sx?</td>
<td>Dehydrated?</td>
<td>CXR: clear?</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Vitals on arrival</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>BP/HR</td>
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<tr>
<td>RR/SpO2</td>
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<td>T</td>
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</tbody>
</table>

Investigations:
ECG: Serial ECG (x3) Features suggestive of UA/NSTEMI are:
- Dynamic ST/T changes
- ST depression > 0.5 mm in 2 or more contiguous leads
- T-wave inversion – deep symmetrical T-wave inversion
- New/presumed new onset BBB (new LBBB = tx as STEMI)

CXR: clear? Any pneumonic changes? Fluid overload?
CE: Trop T (should be taken >6H after onset), CK/CK-MB, AST, LDH
FBC: Hb anemic?
RP: dehydration? AKI? K+ level

Risk stratification by TIMI score

<table>
<thead>
<tr>
<th>TIMI RISK SCORE for UA/NSTEMI</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>HISTORICAL POINTS</strong></td>
</tr>
<tr>
<td><strong>AGE ≥ 65</strong></td>
</tr>
<tr>
<td><strong>≥ 3 CAD risk factors</strong></td>
</tr>
<tr>
<td>(CHF, HTN, ?dm, ?htn, ?htn)</td>
</tr>
<tr>
<td>Known CAD (stenois ≥ 50%)</td>
</tr>
<tr>
<td>ASA use in past 7 days</td>
</tr>
<tr>
<td><strong>PRESENTATION</strong></td>
</tr>
<tr>
<td>Recent (≤24h) severe angina</td>
</tr>
<tr>
<td>↑ cardiac markers</td>
</tr>
<tr>
<td>ST deviation ≥ 0.5 mm</td>
</tr>
</tbody>
</table>

*Risk criteria: UA or NSTEMI defined as: ischemic pain at rest within past 24h, with evidence of CAD (ST segment deviation or troponin)
Management

Goals: relief of angina + prevent recurrence + prevent complications

ED: Crushed Aspirin 300mg stat + Plavix 300mg stat + S/L GTN 0.5mg stat

Admit to ward/CCU

<table>
<thead>
<tr>
<th>Plan:</th>
<th>CI for Beta blockers</th>
</tr>
</thead>
<tbody>
<tr>
<td>- T Aspirin 300mg crushed stat , 150mg OD</td>
<td>AV block</td>
</tr>
<tr>
<td>- T. Plavix 300mg stat, 75mg OD</td>
<td>Bronchial Asthma</td>
</tr>
<tr>
<td>- S/C Clexane (1mg/kg) BD or (*CKD- 1mg/kg OD if CrCl &lt; 30) OR S/C Arixtra 2.5mg OD (muslims) (*CKD- CI if CrCl &lt; 30)</td>
<td>Cardiogenic Shock</td>
</tr>
<tr>
<td>- S/L GTN 0.5mg (every 5 mins up to 3 doses) CI: hypotension</td>
<td>Decompensated LV dysfunction</td>
</tr>
<tr>
<td>- oxygen supplementation</td>
<td>Peripheral Vascular Disease</td>
</tr>
<tr>
<td>+ statins T. Lovastatin 20mg ON</td>
<td>beta-blockers/CCB/ACEi/ARB as indicated</td>
</tr>
</tbody>
</table>

Other alternatives

- T Ticlopidine 250mg BD ( SE: neutropenia, monitor wcc 3/12)
- T. Prasugrel 60mg stat, 10mg OD (for pt after angio and plan for PCI) CI: 75years, bleeding risk
- T. Ticagrelor 180mg stat, 90mg BD (short acting, use if need surgery)

Investigations:

- FBC/RP/LFT/Coagulation profile/electrolytes
- FBS/FSL
- serial CE(without Trop T) + Serial ECG

Risk stratification

- refractory angina, hemodynamically unstable = Revascularization/urgent coronary angiography
- intermediate/high risk = early invasive strategy (within 72hours)
- low risk = non invasive investigation as outpatient

Post hospital care

- Acute phase: 1-3/12 (risk of recurrence) close monitoring
- discharge with ASA+clopidogrel for 1/12 (optimal 9-12mo)
- beta blockers should be started unless contraindicated
- ECHO/EST as outpatient  referral to cardiac centre if indicated (Hospital Serdang)
- rehab programmes – smoking cessation, diet modification, exercise, lipids (LDL<2.0)
Acute STEMI = MI due to total occlusion of the coronary artery, impaired blood supply results in necrosis of heart muscle

Clinical Diagnosis
1) Chest pain – ischaemic type
2) Cardiac biomarkers elevated – indicating myocardial injury/necrosis
3) ECG changes – new onset ST elevation > 0.1 mV in 2 contiguous limb leads or V4-V6
   > 0.2mV in 2 contiguous precordial leads V1-V3
   - presumed new LBBB (use Sgarbossa’s criteria >3points = likely AMI)
      A) concordance ST elevation >1mm 5 points
      B) concordance ST depression >1mm V1-V3 3 points
      C) disconcordance ST elevation >5mm 2 points

History
1) Chest pain - retrosternal, >30mins, start @ time? (important for risk stratification)
   - severe crushing/pressing a/w sweating, nausea/vomiting, SOB + radiates to limbs/jaw
   - Occur at rest/activity , pain score ?/10
   * atypical sx – nausea/vomiting, weakness, light headedness with syncope, dizziness (common in diabetics and women)

other significant hx
- previous hx of IHD/PCI/CABG
- Risk factors for atherosclerosis
- prev TIA/CVA
- sx of peripheral vascular disease

Fam Hx : IHD/CVA/DM/HPT/Ca
Social : smoker/alcoholic, occupation, allergic hx

<table>
<thead>
<tr>
<th>O/E</th>
<th>Lungs : clear / coarse crepts?</th>
</tr>
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<tr>
<td>GCS full ?</td>
<td>CVS: S1S2, murmurs?</td>
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<td>Tachypnoeic? Failure sx?</td>
<td>PA: soft, non tender,</td>
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<td>Anemia sx? Dehydrated?</td>
<td>hepatosplenomegaly? Ascites?</td>
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<td>Vitals on arrival :</td>
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<td>tongue coated?</td>
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<tr>
<td>RR/SpO2</td>
<td>T</td>
</tr>
</tbody>
</table>
**Ix:** ECG: ST elevation? New LBBB (x3 to detect evolving changes)

<table>
<thead>
<tr>
<th>Location</th>
<th>Leads</th>
<th>ECG findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anteroseptal</td>
<td>V1 – V3</td>
<td>ST elevation, Q wave</td>
</tr>
<tr>
<td>Extensive anterior</td>
<td>V1 – V6</td>
<td>ST elevation, Q wave</td>
</tr>
<tr>
<td>Posterior</td>
<td>V7 – V8</td>
<td>T elevation, Q wave</td>
</tr>
<tr>
<td>Posterior</td>
<td>V1 – V2</td>
<td>ST depression, Tall R wave</td>
</tr>
<tr>
<td>Anterolateral</td>
<td>I, AVL, V5 – V6</td>
<td>ST elevation, Q wave</td>
</tr>
<tr>
<td>Inferior</td>
<td>II, III, AVF</td>
<td>ST elevation, Q wave</td>
</tr>
<tr>
<td>Right Ventricular</td>
<td>V4R, V5R</td>
<td>ST elevation, Q wave</td>
</tr>
</tbody>
</table>

blood supply of heart:
RCA = RV, AV node, SA node, Posterior –inferior LV, posterior septum
LCx = Lateral-inferior wall LV, posterior wall LV
LADA = ant wall LV, anterior septum

CE: Trop T (take >6hours), CK/CK-MB, LDH, AST elevation
CXR: TRO pneumothorax, aortic dissection, fluid overload etc

**Mx:** goal = Pain relief + early reperfusion + tx complications
medications same as UA/STEMI + assessment for reperfusion strategy

**Reperfusion strategy** (fibrinolytic therapy / PCI)

**Door-to-balloon time** = Arrival in ED until time of PCI (should be no more than 90mins)
**Door-to-needle time** = Arrival in ED until time of administration of fibrinolytic tx (<30mins)

Depends on:
**Time from onset of symptoms**
1) Early (within 3 hours) = both equally effective
   * PCI preferred if: Fibrinolysis CI, high risk pt, PCI time delay (door-to-balloon t – door-to-needle t <60mins)

2) Late (3-12 hours) = PCI is preferred (Door to Balloon = 90 minutes)
   * If Transfer, should be within 2hours (if more, fibrinolytic tx shld be given)

3) Very late (>12 hours) = PCI is recommended
   Both PCI/Fibrinolytic not recommended if asymptomatic/hemodynamically stable

**Fibrinolytic Therapy**
1) IV Streptokinase 1.5 mega Units in 100ml NS over 1 hour
2) IV Alteplase 15 mg bolus, 50mg over 30mins and 35mg over 60mins (higher rate of reocclusion, give Heparin 48hours)
3) Tenecteplase (metalyse)
   Regimen: *Tenecteplase (TNK-tPA) single i.v. bolus*
   - 30mg if < 60kg
   - 35mg if 60 to < 70kg
   - 40mg if 70 to < 80kg
   - 45mg if 80 to < 90kg
   - 50mg if >90kg

Heparin needs to be given for 48 hours

**CONTRAINDICATIONS**

<table>
<thead>
<tr>
<th>ABSOLUTE</th>
<th>RELATIVE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Active bleeding/aortic dissection</td>
<td>Risk of bleeding</td>
</tr>
<tr>
<td>Brain hemorrhage/AV malformation/neoplasm</td>
<td>Elevated BP</td>
</tr>
<tr>
<td>Stroke</td>
<td>Last 3/12 surgery</td>
</tr>
<tr>
<td></td>
<td>Active peptic ulcer/ASA</td>
</tr>
<tr>
<td></td>
<td>Traumatic CPR</td>
</tr>
<tr>
<td></td>
<td>Internal bleeding (UGIB/UTI) / INR &gt;2</td>
</tr>
<tr>
<td></td>
<td>Vascular puncture (non compressible)</td>
</tr>
<tr>
<td></td>
<td>Exposure to streptokinase &gt;5days within 12mo</td>
</tr>
</tbody>
</table>

**Indicator of successful reperfusion**
1) Resolution of chest pain
2) Return of ST elevation to isoleine or decrease by 50% (within 60-90mins)
3) Early peaking CK/CK-MB levels
4) Restoration of hemodynamic and electrical stability
Failed Fibrinolysis = persistent chest pain, ST elevation and hemodynamic instability
Mx: rescue PCI

Percutaneous Coronary Intervention
Intraaortic balloon pump

CABG (coronary artery bypass graft)
- from Radial artery/ saphenous vein/ internal thoracic artery
CCU care
1) CRIB at least 12 hours
2) Sedation
3) Prevention of valsala manoeuvre (constipation) → Syr lactulose
4) continuous ECG monitoring
5) Oxygen NPO2, maintain SpO2 >95%
6) Meds : ASA, plavix + BB/ACEi + Statins

Complications of MI
1) Arrhythmias – tacharythmias, VT, VPC, AF, AIVR
2) mechanical complications – wall rupture = new murmurs/diminished heart sounds, confirm with ECHO
3) RV infarction → hypotension + clear lung filed + raised JVP, (ST elevation Right precordial leads)
4) pericarditis → pain, worsening on deep inspiration, relieved when sitting and lean forward, +pericardial rub

Dressler’s syndrome (Pictured→)
= fever + chest pain, occurs 2-10wks after STEMI, immunologically mediated → tx with ASA 600mg tds

Management of complications
1) heart failure → O2, Diuretics, IV GTN, IV morphine , +inotropes
2) cardiogenic shock (BP<90/60, tissue hypoperfusion) → urgent PCI/CABG
3) wall ruptures → = surgical intervention
4) RV infarction = give IVD + inotropes , if fail to respond give afterload reducing agents (nitroprusside/PCI)
5) LV thrombus + arterial embolism → anticoagulation 3-6mo
6) DVT = prophylactic anticoag tx (s/c clexane 40mg OD)

Risk stratification
Poor prognosis → consider early coronary angiography
-Age >65, females, pervious MI, previous anterior MI, inferior MI with RV involvement, DM, persistent ischaemia, hypotension, heart failure, AF, new LBBB

Assess → to differentiate Scarred from Viable ischaemic myocardium (require revascularization)

1) LV fn – clinical, CXR, ECHO, cardiac MRI
2) presence of myocardial ischaemia → EST, Dobutamine ST , cardiac MRI

Rehabilitation care
1) smoking cessation
2) Diet
3) regular exercise
4) control hypertension and DM

Follow up
Target to treat
BP <130/80
Lipids LDL <2.6
Diabetic FBS 6.0 : HbA1C < 6.5%
others – Cardiac sx, CPR training, Psychosocial status

Table 7: Clinical and Haemodynamic Subsets in AMI

<table>
<thead>
<tr>
<th>KILLIP CLASS</th>
<th>CLINICAL FEATURES</th>
<th>APPROXIMATE PROPORTION OF PATIENTS WITH AMI (%)5</th>
<th>30 day - MORTALITY (%)5</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>No signs of LV failure</td>
<td>71</td>
<td>7</td>
</tr>
<tr>
<td>II</td>
<td>S3 gallop, bibasal crackles</td>
<td>23</td>
<td>20</td>
</tr>
<tr>
<td>III</td>
<td>Acute pulmonary oedema</td>
<td>3.7</td>
<td>39</td>
</tr>
<tr>
<td>IV</td>
<td>Cardiogenic shock</td>
<td>2.4</td>
<td>70</td>
</tr>
</tbody>
</table>

Flow chart 1: MANAGEMENT OF PATIENTS PRESENTING WITH STEMI

Chest Pain

ECG
Cardiac Biomarkers

Concomitant initial management includes:
- Sublingual GTN
- Continuous ECG monitoring
- Oxygen
- Aspirin
- Clopidogrel
- Analgesia

Assessment for reperfusion:
- Onset of symptoms:
  - < 3 hrs
  - 3-12 hours
  - >12 hours

Preferred option:
- Primary PCI** or Fibrinolytic
- Primary PCI***
- Medical Therapy + Anti thrombotics

Second option:
- Primary PCI*

Concomitant Therapy:
- Anti Thrombotics*
  - Blockers
- ACEI/ARB
- Statins
- Nitrates*
- Calcium antagonists *
Heart Failure

= syndrome characterized by SOB + fatigue + fluid retention supported by cardiac dysfn
= inability of heart to pump blood at rate to meet the body needs

**Common causes**: CHD, Hypertension, valvular heart disease

**Other causes of HF include**:  
severe anemia, Cor pulmonale  
Congenital heart disease  
large A-V shunts  
Viral myocarditis  
Acute rheumatic fever  
Hypertrophic cardiomyopathy  
peripartum cardiomyopathy  
Tachycardia induced cardiomyopathy

**Pericardial disease**: constrictive pericarditis, cardiac tamponade

**Toxic**: Alcohol, adriamycin, cyclophosphamide

**Endocrine**: thyroid disease, acromegaly, phaeochromocytoma

**Collagen vascular disease**: systemic lupus erythematosus, polymyositis, polyarteritis nodosa

**Acute decompensation precipitated by**:  
Acute myocardial infarction/ myocardial ischemia  
Arrhythmias (e.g. atrial fibrillation)  
Uncontrolled Blood Pressure  
Infections (e.g pneumonia)  
Non-compliance to medications

**Excessive fluid and salt intake**  
Anemia  
Development of renal failure  
Adverse effects of drug therapy (NSAIDs)

**Classification**  
Acute/Chronic HF  
Others – Right vs left, forward vs backward, diastolic vs systolic etc

**Risk**  
- atherosclerotic disease  
- HPT, DM, metabolic syndrome  
- Fam Hx cardiomyopathy  
- Thyroid disorders  
- renal disease

**Symptoms**  
- sudden severe SOB (AHF) / gradual SOB (CHF)  
- breathlessness, fatigue, leg swelling  
- Nocturia  
- cough with whitish sputum  
- confusion

**Signs** (congestion of systemic veins)  
- raised JVP  
- peripheral edema  
- hepatomegaly  
- others: tachycardia, creps, gallop rhythm

**Investigations**  
1) **ECG** – ischemia, MI, Left Atrium overload, LV hypertrophy, arrhythmias  
2) **CXR** – cardiomegaly, fluid overload picture  
3) **Blood** – anemia, kidney failure, liver failure, FBS/FSL  
4) **UFEME** – proteinuria, glucosuria  
5) **ABG** – hypoxemia, respiratory failure  
6) **ECHO/ EST/Angiogram as indicated**  
7) others: TFT, Cardiac biomarkers
Figure 1: Algorithm for the diagnosis of Heart Failure or LV dysfunction

1. Suspected Heart Failure because of symptoms/signs
   - ECG
   - Chest X-ray
   - Natriuretic peptides (where available)

2. Tests abnormal
3. Tests normal but clinical suspicion high
4. Tests normal but clinical suspicion low

5. Imaging by Echocardiography (Nuclear angiography or MRI where available)

6. Tests abnormal
7. Tests normal

8. Determine:
   - Underlying cause
   - Severity
   - Precipitating factors
   - Type of LV dysfunction (systolic/ diastolic)

9. Additional diagnostic tests where appropriate (e.g. coronary angiography)

10. Heart Failure or LV dysfunction unlikely. Consider other diagnosis such as:
    - coronary artery disease (angina equivalent),
    - pulmonary disease,
    - obesity

11. Treat accordingly
Acute Heart Failure
- Acute Pulmonary edema sx

Presentation of APO
Acute breathlessness, Anxiety, Ascites
Pink frothy sputum, PND, Panting
Odemea, Orthopnea

Physical Exam
- Confused
- Cold clammy limbs, cyanosis
- Tachypnoic, use accessory muscles
- Ausc: wheezing +crackles+ronchi
- VS: high BP, SpO2 < 85%

Ix
ECG – LA/LV hypertrophy, Acute MI or ischaemia
CXR – heart failure picture
Blood – Hb, electrolytes, urea, creatinine, cardiac markers,
ABG – respiratory failure

Mx:
- Adequate Oxygenation (SpO2 >95%)
- Face mask
- BiPAP/CPAP
- Intubation (persistent hypoxemia)
- Frusemide + morphine + nitrates
- Assess response to tx + BP
- Correct other underlying conditions

1) Preload Reduction (pulmonary venous return)
- IV GTN 10mcg/min
- IV Frusemide 40-100mg
- IV Morphine 3-5mg bolus + Maxolon 10mg

2) Afterload reduction (vascular resistance)
- ACE inhibitors
- ARB

3) Inotropes (if hypotensive)
- Dobutamine 2-5mcg/kg/min
- Dopamine 2mcg/kg/min
- Noradrenaline 0.02-1mcg/kg/min
- Milrinone 50mcg/kg bolus, 0.75mcg/kg/min

Cardiogenic Shock
- sBP <90mmHg, not improved with fluids
- Hypoperfusion-cold extremities, altered mental status, restless
- Reduced urine output (<20cc/hr)

Mx
- Inotropic support (high dose Dopa/Noradrenaline/Dobutamine)
- Pump failure ⇒ PCI
- Mechanical failure ⇒ ventricular septal rupture, acute MR ⇒ surgery
Flowchart I: Management of Acute Cardiogenic Pulmonary Edema

ACUTE CARDIOGENIC PULMONARY EDEMA

Oxygen
IV Diuretics

BLOOD PRESSURE

SBP ≥100mmHg

- Nitrates (caution in valvular stenosis)
- Morphine

? ARRYTHMIA

Treat accordingly

SBP <100mmHg

- dopamine 1st
- noradrenaline 2nd
- correct hypoxia/acidosis

SBP ≥100mmHg

SBP still<100mmHg

IMPROVED

NO IMPROVEMENT

** Oral Medications

- ↑ Diuretics, continuous infusions + combination of diuretics
- ↑ Nitrates
- low dose dopamine
- dobutamine

IMPROVED

NO IMPROVEMENT

** Oral Medications

- milrinone
- correct acidosis
- consider ventilation
- invasive monitoring
- IABP, VAD, heart transplant

NOTE:
Chronic Heart Failure

Measures
- Education → warning signs of HF, medications etc
- Diet and nutrition → salt restriction
- Lifestyle → smoking and alcohol cessation
- Exercise

Pharmacological Mx

<table>
<thead>
<tr>
<th>GROUP</th>
<th>Improvement</th>
<th>Side Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>1) Diuretics</td>
<td>Improves fluid retention</td>
<td>SE: Hypokalemia, dehydration</td>
</tr>
<tr>
<td>2) ACEi</td>
<td>Improves survival, delays progression in all classes * start low dose, monitor RP after 2/52</td>
<td>SE: Cough, renal insuff, angioedema, hyperK</td>
</tr>
<tr>
<td>3) ARB</td>
<td>ACEi intolerant, post MI</td>
<td></td>
</tr>
<tr>
<td>4) B blockers</td>
<td>Improves survival, delays progression in all classes</td>
<td></td>
</tr>
<tr>
<td>5) Digoxin</td>
<td>Indicated in HF with AF Start with 0.125mg – 0.25mg OD</td>
<td></td>
</tr>
</tbody>
</table>

Flowchart II: Optimizing Drug Therapy in CHF

1. **No**
   - (LVEF <40%)
   - ACEi
   - β-blockers

2. **Yes**
   - **ACEi / Diuretics**

3. **Clinical Improvement**
   - **No**
     - Add spironolactone
   - **Yes**
     - Continue with:
       - Diuretics – low maintenance dose
       - ACEi titrate to max tolerated dose
       - β-blockers

4. **Clinical Improvement**
   - **No**
     - Add
       - Digoxin
       - Consider combination with ARB
   - **Yes**
     - Continue with:
       - Diuretics
       - ACEi
       - spironolactone
       - β-blockers

5. **Clinical Improvement**
   - **No**
     - See Flowchart I (pg 12)
       - Loop diuretics + thiazides
       - short term parenteral positive inotropes
       - ? IABP ? VAD
       - ?Cardiac transplant
   - **Yes**
     - Continue with:
       - Diuretics
       - ACEi
       - spironolactone
       - digoxin
       - ARB
       + β-blockers
STROKE

CVA = Clinical syndrome characterized by rapidly dev sx or signs of focal/global loss of cerebral fn, with sx lasting >24hrs or leading to death, with no apparent cause rather than that of vascular origin

Transient Ischaemic Attack (TIA) = Sx lasting <24hrs, due to inadequate blood supply as a result of thrombosis/embolism

Classification
1) Ischaemic Stroke
2) Hemorrhagic Stroke

Evaluation
1) Neurological deficit
2) site of lesion- location, type, cause
3) Complications and prognosis

Clinical features according to circulation
Anterior and Posterior circulation

<table>
<thead>
<tr>
<th>1) Anterior Circulation (Carotid artery)</th>
<th>2) Posterior Circulation (vertebro basilar system)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Middle cerebral artery</td>
<td>Cortical blindness</td>
</tr>
<tr>
<td>Aphasia</td>
<td>Ataxia, vertigo</td>
</tr>
<tr>
<td>Hemiparesis/plegia</td>
<td>Homonymous hemianopia</td>
</tr>
<tr>
<td>Homonymous Hemianopia</td>
<td>Dysarthria</td>
</tr>
<tr>
<td>Parietal lobe ➔ astereognosis, agraphaesthesia, impaired 2 point discrimination, sensory and visual inattention left-right dissociation, acalculia</td>
<td>Diplopia</td>
</tr>
<tr>
<td></td>
<td>Dysphagia</td>
</tr>
<tr>
<td></td>
<td>Hemiparesis/sensory loss contralateral to CN palsy</td>
</tr>
<tr>
<td></td>
<td>Cerebellar signs</td>
</tr>
</tbody>
</table>

Anterior Cerebral artery ➔ weakness LL>UL

Lesion of Right Middle Cerebral Artery

- Lesion of the right hemisphere
- Poor sensory and visual attention
- Acalculia
- Homonymous hemianopia
- Left sided hemiplegia
Causes
1) Ischaemic stroke
   a) atherothromboembolism
   b) penetrating artery disease (Intracranial small vessel disease)
   c) Cardiogenic embolism

Risk
Vascular risk factors associated with increased risk of stroke:

<table>
<thead>
<tr>
<th>NON-MODIFIABLE</th>
<th>MODIFIABLE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>High Blood Pressure (systolic and diastolic)</td>
</tr>
<tr>
<td>Sex</td>
<td>Cigarette smoking</td>
</tr>
<tr>
<td>Ethnicity / Race</td>
<td>Diabetes mellitus</td>
</tr>
<tr>
<td>Family history of stroke</td>
<td>Atrial Fibrillation</td>
</tr>
<tr>
<td></td>
<td>Coronary heart disease</td>
</tr>
<tr>
<td></td>
<td>Hyperlipidaemia</td>
</tr>
<tr>
<td></td>
<td>Obesity &amp; physical inactivity</td>
</tr>
<tr>
<td></td>
<td>Raised Homocysteine levels</td>
</tr>
<tr>
<td></td>
<td>High dietary salt intake</td>
</tr>
<tr>
<td></td>
<td>Heavy alcohol consumption</td>
</tr>
<tr>
<td></td>
<td>Previous stroke</td>
</tr>
</tbody>
</table>

Physical Examination
1) GCS: EVM
2) Orientation to time/place/person: Name? How old are you? Where are you? What time is it now? Who is this?

![The Glasgow Coma Scale](image)
Neurological Examination

1) Power = against resistance
2) Tone = intact, flaccid
3) Reflexes = intact/hyper/hyporeflexia
4) Sensations = intact/absent/reduced/paraesthesia
5) Pupils = bilaterally reactive to light, same size
   (diff size = indicates ICB —)
6) Gag reflex = if absent, perform swallowing test, if fail must insert Ryle’s Tube (feeding)

<table>
<thead>
<tr>
<th>Power</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>5</td>
<td>Able to move + against full resistance</td>
</tr>
<tr>
<td>4</td>
<td>Able to move + against moderate resistance</td>
</tr>
<tr>
<td>3</td>
<td>Able to move + without resistance</td>
</tr>
<tr>
<td>2</td>
<td>Able to move with force of gravity eliminated</td>
</tr>
<tr>
<td>1</td>
<td>Muscle contraction seen/felt on palpation</td>
</tr>
<tr>
<td></td>
<td>Insufficient to produce joint motion</td>
</tr>
<tr>
<td>0</td>
<td>NO muscle contraction seen or felt</td>
</tr>
</tbody>
</table>

Reflexes

Babinski Reflex

Normal plantar response
Extensor plantar response (Babinski sign)
<table>
<thead>
<tr>
<th>CN</th>
<th>NERVE</th>
<th>FUNCTION</th>
<th>TYPE</th>
<th>TEST</th>
</tr>
</thead>
<tbody>
<tr>
<td>CN I</td>
<td>Olfactory</td>
<td>Special Sensory: Small</td>
<td>Sensory</td>
<td>Ask patient to identify small (e.g., coffee/perfume)</td>
</tr>
<tr>
<td>CN II</td>
<td>Optic</td>
<td>Special Sensory: Sight</td>
<td>Sensory</td>
<td>Assess vision in each eye (Snellen chart/Rosebush chart)</td>
</tr>
<tr>
<td>CN III</td>
<td>Occulomotor</td>
<td>Somatic Motor: Superior, Medial, Inferior Rectus, Inferior Oblique</td>
<td>Motor</td>
<td>Check pupil constriction, eye movement</td>
</tr>
<tr>
<td>CN IV</td>
<td>Trochlear</td>
<td>Somatic Motor: Superior Oblique</td>
<td>Motor</td>
<td>Assess patient's ability to look downward and inward</td>
</tr>
<tr>
<td>CN V</td>
<td>Trigeminal</td>
<td>Somatic Motor: Face</td>
<td>Both</td>
<td>Motor: assess patient's ability to clench jaw</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Somatic Motor: Mastiication, Tensor Tympani, Tensor Palatini</td>
<td>Sensory</td>
<td>assess facial response to touch</td>
</tr>
<tr>
<td>CN VI</td>
<td>Abducens</td>
<td>Somatic Motor: Lateral Rectus</td>
<td>Motor</td>
<td>Assess lateral deviation of eye</td>
</tr>
<tr>
<td>CN VII</td>
<td>Facial</td>
<td>Somatic Sensory: Posterior External Ear Canal</td>
<td>Both</td>
<td>Motor: assess patient's ability to smile, frown, and elevate eyebrows, Assess symmetry</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Special Sensory: Taste (Anterior 2/3 Tongue)</td>
<td>Sensory</td>
<td>Sensory: check taste on anterior 2/3 of the tongue</td>
</tr>
<tr>
<td>CN VIII</td>
<td>Vestibulocecal</td>
<td>Special Sensory: Auditory/Balance</td>
<td>Sensory</td>
<td>Rinne/Weber Nystagmus</td>
</tr>
<tr>
<td>CN IX</td>
<td>Glossopharyngeal</td>
<td>Somatic Sensory: Posterior 1/3 Tongue, Middle Ear</td>
<td>Both</td>
<td>Motor: ask patient to swallow. Check gag reflex</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Visceral Sensory: Carotid Body/Sinus</td>
<td>Sensory</td>
<td>Sensory: check taste on posterior 1/3 of tongue</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Somatic Motor: Stylopharyngeal</td>
<td>Both</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Visceral Motor: Parotid Gland</td>
<td>Both</td>
<td></td>
</tr>
<tr>
<td>CN X</td>
<td>Vagus</td>
<td>Somatic Sensory: External Ear</td>
<td>Both</td>
<td>Check symmetry of soft palate and uvula</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Visceral Sensory: Acoustic Arch/Body</td>
<td>Both</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Special Sensory: Taste over epiglottis</td>
<td>Both</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Somatic Motor: Soft Palate, Pharynx, Larynx (Vocalization and Swallowing)</td>
<td>Both</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Visceral Motor: Bronchocoughstion, Peristalsis, Erythrocardia, Vomiting</td>
<td>Both</td>
<td></td>
</tr>
<tr>
<td>CN XI</td>
<td>Accessory</td>
<td>Somatic Motor: Trapezius, Sternocleidomastoid</td>
<td>Motor</td>
<td>Ask patient to shrug shoulder against resistance</td>
</tr>
<tr>
<td>CN XII</td>
<td>Hypoglossal</td>
<td>Somatic Motor: Tongue</td>
<td>Motor</td>
<td>Ask patient to stick out tongue</td>
</tr>
</tbody>
</table>

---

**The anatomy of impairment**

- **Parietal lobe:** inability to attend to more than one object at a time, inability to name an object, inability to locate the words to write (graphia), problems with reading (alexia), difficulty distinguishing left from right, difficulty doing mathematics (dyscalculia), lack of awareness of body parts and/or surrounding space, difficulty with visual attention, difficulty with visual hallucinations.

- **Frontal lobe:** loss of simple movement of various body parts (aparaxia), inability to plan a sequence of complex movements needed to complete multisteped tasks, such as making coffee (sequencing), loss of spontaneity in interacting with others, loss of flexibility in thinking, persistence of a single thought (preservation), failure to focus on a task (attending), mood changes (emotionality), changes in social behavior, changes in personality, difficulty with problem solving, inability to express language (Broca's aphasia).

- **Occipital lobe:** defects in vision (visual field cuts), difficulty locating objects in the environment, difficulty identifying colors (color agnosia), hallucinations, visual illusions, or incorrectly seeing objects.

- **Temporal lobe:** difficulty recognizing local objects, inability to recognize the movement of an object (movement agnosia), difficulty reading and writing, difficulty swallowing food and water (dysphagia), difficulty organizing or perceiving the environment, problems with balance and movement, dizziness and nausea (vertigo), sleeping difficulties (insomnia, sleep apnea).

- **Brain stem:** difficulty swallowing food and water (dysphagia), difficulty understanding spoken words (Wernicke's aphasia), disturbance with selective attention to the patient and others, difficulty with identification and verbalization about objects, short-term memory loss, interference with long-term memory, increased or decreased interest in sexual behavior, inability to categorize objects, persistent looking (right side damage can cause this), increased aggressive behavior.
Carotid bruit = due to stenosis of carotid artery, may be cause of CVA
Lightly apply the bell of the stethoscope over the course of the carotid artery, from the base of the neck to angle of the jaw, during full expiration.
Motor Neuron Weakness

<table>
<thead>
<tr>
<th>Location</th>
<th>Upper Motor Neurons</th>
<th>Lower Motor Neurons</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Cerebral cortex</td>
<td>Brain stem + Spinal cord</td>
</tr>
<tr>
<td>Sx</td>
<td>Pyramidal weakness</td>
<td>3A- Atonia, Areflexia, Atrophy</td>
</tr>
<tr>
<td></td>
<td>- LL Flexors</td>
<td>- impulses arrive at patho LMN → Fasciculations/Fibrillations</td>
</tr>
<tr>
<td></td>
<td>- UL Extensors</td>
<td>Babinski upgoing</td>
</tr>
<tr>
<td></td>
<td>Spasticity, clasp-knife</td>
<td>Babinski reflex absent</td>
</tr>
<tr>
<td></td>
<td>Increased Deep tendon reflex</td>
<td></td>
</tr>
<tr>
<td>manifestations</td>
<td>CVA, brain Ca, cervical spine injury</td>
<td>Diabetic neuropathy, poliomyelitis, spinal cord injury, multiple sclerosis</td>
</tr>
</tbody>
</table>

Other Investigations
1) ECG – look for AF
2) Blood – routine blood, +thrombophilia screening (young ICB)
Imaging

CT Brain

Figure 3. Normal Brain Symmetry

A normal brain is symmetrical. Abnormal masses or hemorrhage may deviate brain structures across an imaginary line dividing the brain, creating "midline shift." The degree of midline shift is more important acutely than the exact etiology of the shift, since shift is an indication of threatened subfalcine herniation and may require surgical intervention. © 2007 Joshua Broder.

Figure 4. Air-filled Spaces

These are the normal locations and appearances of air-filled spaces when viewed on "brain windows." Air-filled spaces are normally black on both "brain" and "bone windows."

A. Maxillary sinuses
B. Mastoid air cells
C. Ethmoid sinuses
D. Sphenoid sinus
E. Frontal sinus

Figure 5. Normal CSF Spaces

In a normal brain, the basilar cisterns (A, arrow) are patent (sometimes referred to as the "smile sign"). The lateral ventricles are open but not enlarged (B). Sulci are visible but not excessive (C). © 2007 Joshua Broder.
Subdural hematomas are most often the result of shearing of dural veins from blunt trauma. They usually have a crescent shape and may cross suture lines as they lie within the dura and are thus free to extend along the brain surface, rather than being restricted by the tethering of the dura to the calvarium at sutures.

This subdural hematoma (arrowheads) demonstrates several classic features:
- Crescent shape
- Crosses suture lines (A)
- Mass effect with midline shift (B)
- Elevated ICP, with small ventricles (C)
- No visible sulci (D)

Subarachnoid hemorrhage appears white on non-contrast CT. In this case of diffuse subarachnoid hemorrhage, note the presence of subarachnoid blood filling the sulci, as well as extending into the cisterns, Sylvian fissures, and even lateral ventricles.

A. Blood in basilar cistern
B. Blood in Sylvian fissure
C. Blood in posterior horns of lateral ventricles
D. Blood in sulci

Classic features of epidural hematoma (arrowheads) are visible:
- Lens-like or biconvex disc shape
- Temporal location, with associated depressed temporal bone fracture (A)
- Does not cross suture lines (B - expected location of suture)
- Mass effect with midline shift (C)
- "Swirl sign" – heterogeneous appearance suggesting active bleeding (D)
- Elevated ICP, with small ventricles (E) and no visible sulci (F)

Acute intraparenchymal hemorrhage appears white on CT. Hemorrhage in the patient's left frontal region is creating mass effect with midline shift. The left lateral ventricle has been effaced.

A calcified mass in the right occipital region must be differentiated from acute hemorrhage. Calcifications are extremely bright white on brain windows – as white and dense as bone. On bone windows, they remain visible, while hemorrhage does not.
Figure 14. Calcifications

Calcification of the choroid plexus (A) is a frequent incidental finding which may resemble punctate intraparenchymal hemorrhage. Clues are a bright white density (equal to that of bone), location in the posterior horns of the lateral ventricle, and frequent bilaterality. This patient also has a calcified meningioma (B). Meningiomas are common benign neoplasms which may become quite large. A well-circumscribed rounded appearance and calcification are common. © 2007 Joshua Broder.

Figure 15. Masses

This image shows a mass with surrounding vasogenic edema (arrowheads). Neoplasms are frequently associated with vasogenic edema, named for the putative cause, which is abnormal vessels that allow extravasation of edema fluid. This form of edema appears hypodense, like an ischemic infarct, but is not restricted to a vascular territory. An abscess might appear similar. © 2007 Joshua Broder.

Figure 16. Gray And White Matter

A left MCA distribution stroke, day two (A) and day four (B) after symptom onset. Early ischemic changes may be visible within three hours of symptom onset. The rate of progression of CT findings may depend on the degree of ischemia or infarction and thus may vary between patients. © 2007 Joshua Broder.

This shows normal gray-white matter differentiation. Myelinated regions (white matter) have a greater fat content than unmymelinated regions (gray matter). As a consequence, white matter is lower density and appears darker on CT. When ischemia renders this interface less discrete, the CT appearance is called loss of gray-white differentiation. © 2007 Joshua Broder.

Figure 17. Progression Of Ischemic Hypodensity Over Days

A left MCA distribution stroke, day two (A) and day four (B) after symptom onset. Early ischemic changes may be visible within three hours of symptom onset. The rate of progression of CT findings may depend on the degree of ischemia or infarction and thus may vary between patients. © 2007 Joshua Broder.
**Figure 18. Cerebral Atrophy**

In cerebral atrophy, all CSF spaces become prominent. The basilar cisterns (A, arrow) are open, and the lateral ventricles are enlarged (B). Sulci (C) are equally prominent, helping to distinguish this condition from hydrocephalus. © 2007 Joshua Broder.

---

**Figure 19. Hydrocephalus**

In hydrocephalus, the basilar cisterns (A, arrow) are effaced, as are the sulci (C). The lateral and third ventricle are enlarged (B). © 2007 Joshua Broder.

---

**Figure 20. Cerebral Edema**

In cerebral edema, the basilar cistern (A, arrow) becomes effaced. The lateral ventricles become compressed and slit-like (B), or even completely effaced. Sulci (C) become effaced. © 2007 Joshua Broder.
**Acute Management**

1) **Oxygen and Airway support**
2) **Mobilization - physiotherapy**

3) **BP** – mild hypertension is desirable: **160-180/90-100**, sudden decrease in BP may result in hypoperfusion
   - treat if >220/120
   - Med: IV Labetalol 10-30mg bolus, then IVI 1-3mg/min or T Captopril 6.25-12.5mg

4) **Glucose** – treat hyper/hypoglycaemia accordingly

5) **Nutrition** – swallow test/gag reflex

**Swallowing test**
1) Feed pt with spoon full/syringe of water x 10
   - Observe for: coughing/choking, drooling, gurgling sound

   If PASS = Allow orally (start with clear sips of fluid)

   If FAIL = Insert Ryle’s Tube (for nasogastric feeding)

6) **Others**:
   - Fever → antipyretics
   - Infection → antibiotics
   - raised ICP → IV mannitol 0.25-0.5g/kg (20mins)

**REPERFUSION of Ischaemic brain**
- Must have stroke unit with specialist in stroke mx (not in Hosp Ampang), available neuroimaging tests, able to manage ICB

IV THROMBOLYSIS with **rt-PA 0.9mg/kg** (max 90mg) *** Recommended within 4.5hrs of onset ***
Dose: Give 10% Bolus followed by IVI over 1hr

<table>
<thead>
<tr>
<th><strong>Indication</strong></th>
<th><strong>Contraindication</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>Neuro Deficit (not minor/isolated, no clearing spontaneously) Onset 4.5hrs</td>
<td>Anticoagulant use – oral anticoagulant (PT &gt;15, INR &gt;1.7)</td>
</tr>
<tr>
<td>No CI for Thrombolytics</td>
<td>Heparin in prev 48H (prolonged APTT)</td>
</tr>
<tr>
<td>BP &lt;185/110</td>
<td>Plt &lt;100k</td>
</tr>
<tr>
<td>CT brain normal/minimal change</td>
<td>Head injury/stroke past 3/12</td>
</tr>
<tr>
<td>Family understand risk/benefits</td>
<td>Surgery 2/52</td>
</tr>
<tr>
<td></td>
<td>Arterial puncture at non compressible site past 21/7</td>
</tr>
<tr>
<td></td>
<td>BP &gt;185/110</td>
</tr>
<tr>
<td></td>
<td>Seizure at onset of CVA</td>
</tr>
<tr>
<td></td>
<td>Glucose hypo/hyperG &lt;2.7 &gt;22.2</td>
</tr>
<tr>
<td></td>
<td>GIT/urinary bleeding past 24/7</td>
</tr>
<tr>
<td></td>
<td>Recent MI</td>
</tr>
<tr>
<td></td>
<td>Isolated neuro def – ataxia, sensory loss, dysarthria, weakness</td>
</tr>
</tbody>
</table>

- Neurological deficit may resolve over 3/12, in which after it may become permanent

**Primary Prevention**
1) Age >55, Fam hx of stroke → Aspirin 100mg EOD for women >65yo
2) Hypertension → tx if BP >140/90 (target DM 130/80)
3) Smoking and alcohol cessation
4) Post menopausal HRT therapy → reduces risk of stroke 31% (according to progestin study)
5) DM → Try to keep HbA1c <6%
6) Atrial Fibrillation → warfarin therapy if indicated
7) Hyperlipidemia → start statins, keep LDL <2.6,
Secondary Prevention

1) Antiplatelet therapy
   a) Aspirin → 75-325mg daily
   b) Ticlopidine → 250mg BD
   c) Clopidogrel → 75mg OD
   d) Trifusal → 600mg OD (new recommendation)
   e) Cilostazol → 100mg BD (new recommendation but under review)

2) Anticoagulation (Ind: Atrial Fibrillation)
   - May be commenced within 2-4 days after pt neurological and hemodynamically stable
   a) T warfarin
      Start 5mg OD 3/7 then check INR and taper accordingly (target INR 2.5 (2-3))
   b) Dabigatran etexilate (150mg BD) new recommendation, does not require INR monitoring

CHADS2 score | CHA2DS2 –VASc (new recommendation)
--- | ---
+1 Congestive Heart failure hx? | +1 Congestive Heart failure hx?
+1 Hypertension | +1 Hypertension
+1 Age >75 | * Age 65yo +1 | >75yo + 2
+1 DM hx | +1 DM hx
+2 Stroke sx or TIA | +2 Stroke sx or TIA or thromboembolism hx
| +1 Vascular disease His (MI, PAD, aortic plaque)
| +1 Female

CHADS2 criteria | Points
--- | ---
Previous stroke or TIA | 2
Age ≥ 75 years | 1
Hypertension | 1
Diabetes mellitus | 1
Heart failure | 1

Stroke risk score | Recommended therapy
--- | ---
High 2–6 | Warfarin (INR 2–3)
Moderate 1 | Warfarin or aspirin
Low 0 | Aspirin 100–300 mg daily

0 Low
1 Low moderate
>2 moderate-high → start anticoagulation tx

CI warfarin:
Bleeding – GIT, ICB, aneurysm, retinopathy
Liver disease-alcoholic hepatitis
Endocarditis (bacterial)
Elevated uncontrolled BP
Dementia, with likely poor compliance

Counselling for warfarin
1) Requires frequent visits to INR clinic for blood taking (every 3 days) until optimal dose determined
2) Must be compliant to dose and time
3) Consequences: bleeding tendencies, bruises, melena, hemorrhage

3) Anti-hypertensive → ACEi is recommended or ARB
4) Lipid lowering
5) DM good control
6) Cessation of smoking

HAS-BLED score → indicates risk of bleeding
Hypertension
Abnormal renal/liver fn
Stroke
Bleeding
Labile INR
Elderly >65yo
Drugs/Alcohol – ASA, NSAIDs etc

<table>
<thead>
<tr>
<th>HAS-BLED score</th>
<th>n</th>
<th>Bleeds, n</th>
<th>Bleeds/100 patients*</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>798</td>
<td>9</td>
<td>1.13</td>
</tr>
<tr>
<td>1</td>
<td>1286</td>
<td>13</td>
<td>1.02</td>
</tr>
<tr>
<td>2</td>
<td>744</td>
<td>14</td>
<td>1.88</td>
</tr>
<tr>
<td>3</td>
<td>187</td>
<td>7</td>
<td>3.74</td>
</tr>
</tbody>
</table>

Surgical intervention
1) Carotid Endarterectomy
2) Carotid Angioplasty and stenting
3) Intracranial Angioplasty and stenting
Stroke Management Algorithm

Symptoms & signs suggestive of Stroke
Symptoms & signs persist > 1 hour

Acute Care
Urgent Clinical Evaluation
Urgent brain CT
Blood tests
ECG

Ischaemic Stroke
Infarction

Specific Stroke therapy
Thrombolytic therapy (if no contraindications,
Antiplatelet therapy

Haemorrhagic Stroke
(ICH / SAH)
Brain CT shows haemorrhage

Neurosurgical Evaluation & Treatment

Acute Stroke Care
Stroke Unit (if available)
Airway, Breathing, Circulation
Hydration
Blood Pressure monitoring
Neurological Status monitoring
Anticipate & treat complications
Begin Rehabilitation

Neurorehabilitation
Multidisciplinary Team Approach
Proper Positioning
Early mobilization
Physiotherapy
Occupational therapy
Speech therapy
Treat spasticity
Treat depression

Further Investigations
Establish Stroke subtype and underlying cause
Cardio & Cerebrovascular Risk Assessment

Education
Patient & Caregiver

Secondary Prevention
Antiplatelet therapy
Treat risk factors
Treat specific underlying cause
Therapeutic lifestyle modification (new recommendation)
**Dengue Fever**
- Infection caused by Dengue virus, mosquito born (aedes aegypti/albopictus)
- 4 serotypes: DEN 1,2,3,4.

**Incubation**
4-7 days (3-14 days)

**Clinical course**

I) **Febrile phase** – sudden onset FEVER 2-7 days
+ facial flushing, skin erythema, myalgia, arthralgia, headache, retoorbital pain
+ nausea and vomiting
* hepatomegaly/hepatitis are more suggestive of DHF
* earliest abnormality is NEUTROPENIA with positive history of neighbourhood dengue

II) **Critical phase** – day3-5 of illness, usually late febrile phase or around defervesence, lasts 24-48hours
- Condition either improves or worsens depending on capillary permeability -->DHF/DSS
DHF- rapid drop in temperature, with increase in capillary permeability
Plasma leakage = raised HCT + hemodynamic instability

III) **Recovery phase**
- After 24-48hrs defervesence , plasma leakage stop
  = reabsorption of extracellular fluid
- signs: return of appetite, improved general condition,
  GIT sx abate, hemodynamic status normalizes, Diuresis

**Warning signs**
1) Abdominal pain
2) Persistent Vomiting
3) Clinical fluid accumulation
4) mucosal bleeding
5) restlessness lethargy
6) Tender enlarged liver
7) Lab- increased HCT , decreased plt

Decreased Plt + increased HCT
Enlarged tender Liver
Nausea , persistentVomiting
GIT (abdominal) pain
Unrest, lethargy
Erythema (bleeding mucosal)
Fluid accm

**DHF criteria (DHFP)**
1) Decrease Plt (Thrombocytopenia <100 K)
2) Hemorrhagic tendencies = (i) tourniquet test (ii) petechiae (iii) mucosal bleed (iv) hematemesis/melena)
3) Fever 2-7days
4) Plasma leakage = (i) rise/decrease HCT >20% from baseline (ii) pleural effusion/ascites

### Dengue Shock Syndrome

<table>
<thead>
<tr>
<th>All 4 DHF criteria present + below</th>
<th>WHO classification: Grade</th>
</tr>
</thead>
<tbody>
<tr>
<td>1) Consciousness - altered</td>
<td>I – Fever + non specific sx (+tourniquet test or easy bruising)</td>
</tr>
<tr>
<td>2) Pulse volume = weak, thread</td>
<td>II- Spontaneous bleeding + (I)</td>
</tr>
<tr>
<td>3) HR : tachycardia</td>
<td>III- Circulatory failure( compensated DSS sx)</td>
</tr>
<tr>
<td>4) Pulse Pressure - narrowing</td>
<td>IV –profound shock - undetectable BP/pulse (decompesated DSS)</td>
</tr>
<tr>
<td>5) CRT = prolonged</td>
<td></td>
</tr>
<tr>
<td>6) BP- Hypotensive (diastolic raised), postural hypotension</td>
<td></td>
</tr>
<tr>
<td>7) Limbs: cold clammy</td>
<td></td>
</tr>
<tr>
<td>8) Respiration = tachynoic</td>
<td></td>
</tr>
<tr>
<td>9) Urine output = decrease</td>
<td></td>
</tr>
</tbody>
</table>

* **Notification** – within 24hours by telephone and ; form within 1 week
## Table 1.4 Normal Circulation vs Compensated vs Decompensated / Hypotensive Shock

<table>
<thead>
<tr>
<th>Normal Circulation</th>
<th>Compensated shock</th>
<th>Decompensated / Hypotensive shock</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clear consciousness</td>
<td>Clear consciousness – shock can be missed if you do not touch the patient</td>
<td>Change of mental state – restless, combative or lethargy</td>
</tr>
<tr>
<td>Brisk capillary refill time (&lt;2 sec)</td>
<td>Prolonged capillary refill time (&gt;2 sec)</td>
<td>Mottled skin, very prolonged capillary refill time</td>
</tr>
<tr>
<td>Warm and pink extremities</td>
<td>Cool extremities</td>
<td>Cold, clammy extremities</td>
</tr>
<tr>
<td>Good volume peripheral pulses</td>
<td>Weak &amp; thready peripheral pulses</td>
<td>Feeble or absent peripheral pulses</td>
</tr>
<tr>
<td>Normal heart rate for age</td>
<td>Tachycardia</td>
<td>Severe tachycardia with bradycardia in late shock</td>
</tr>
<tr>
<td>Normal blood pressure for age</td>
<td>Normal systolic pressure with raised diastolic pressure</td>
<td>Hypotension/unrecordable BP</td>
</tr>
<tr>
<td></td>
<td>Postural hypotension</td>
<td></td>
</tr>
<tr>
<td>Normal pulse pressure for age</td>
<td>Narrowing pulse pressure</td>
<td>Narrowed pulse pressure (&lt;20 mmHg)</td>
</tr>
<tr>
<td>Normal respiratory rate for age</td>
<td>Tachypnoea</td>
<td>Metabolic acidosis/ hyperpnoea/ Kussmaul’s breathing</td>
</tr>
<tr>
<td>Normal urine output</td>
<td>Reduced urine output</td>
<td>Oliguria or anuria</td>
</tr>
</tbody>
</table>

**Figure 1.4 Suggested dengue case classification and levels of severity**

**DENGUE ± WARNING SIGNS**

- **with warning signs**
- **without**

**SEVERE DENGUE**

1. Severe plasma leakage
2. Severe haemorrhage
3. Severe organ impairment

**CRITERIA FOR DENGUE ± WARNING SIGNS**

- **Probable dengue**
  - Live in/ travel to dengue endemic area.
  - Fever and 2 of the following criteria:
    - Nausea, vomiting
    - Rash
    - Aches and pains
    - Tourniquet test positive
    - Leukopenia
    - Any warning sign
  - Laboratory-confirmed dengue (important when no sign of plasma leakage)

- **Warning signs**
  - Abdominal pain or tenderness
  - Persistent vomiting
  - Clinical fluid accumulation
  - Mucosal bleed
  - Lethargy, restlessness
  - Liver enlargement > 2cm
  - Laboratory: Increase in HCT concurrent with rapid decrease in platelet count

**CRITERIA FOR SEVERE DENGUE**

- **Severe plasma leakage leading to:**
  - Shock (DSS)
  - Fluid accumulation with respiratory distress

- **Severe bleeding**
  - as evaluated by clinician

- **Severe organ involvement**
  - Liver: AST or ALT > 1000
  - CNS: Impaired consciousness
  - Heart and other organs

*requiring strict observation and medical intervention*
History
1) Fever how many days? Last taken T PCM?
2) Alarm signs
3) Mental state
4) Urine output
5) relevant hx – fogging, recent travel, jungle trekking, swimming in waterfall, high risk behaviour etc

Physical
1) GCS
2) Hydration
3) Hemodynamics – skin, cold/warm limbs, CRT, pulse volume, BP, PR, pp
4) Respiration: tachypnoea, effusion
5) PA: abdominal tenderness? Ascites?Hepatomegaly
6) bleeding manifestations (tourniquet test)

Ix:
1) FBC – neutropenia, HCT rising, Plt decreasing
2) LFT – AST elevation > ALT (DHF)
3) Dengue serology Tests:
   a) Dengue IgM – taken ASAP when suspected, then repeat Day 7 (seroconversion)
   b) sero surveillance – taken for statistics purposes, before Day 5

Diagnosis of DENGUE
1) Phase of illness (Febrile/Critical/recovery)
2) Hydration and Hemodynamics
   (in shock or not)

Management
Hydration
5-7ml/kg/hr – 1-2hours
3-5ml/kg/hr – 2-4hours
2-3ml/kg/hr – adjust and taper
* according to clinical response and HCT

Compensated Shock
1) Obtain HCT level before fluid resus \(\rightarrow\) IVD 5-10ml/kg/hr x 1 Hour
2) repeat: FBC/HCT/BUSE/LFT/RBS/CoAg/ Lactate/Bicarb / GXM
   - check HCT if no improvement repeat IVD 5-10ml/kg/hr (up to 2 cycles, if no improvement change to colloids)
* If HCT decrease, consider occult bleeding \(\rightarrow\) Tx PC
* If persistent shock after x 3 cycles, consider other causes of shock = sepsis, cardiogenic shock
  * adjust fluids clinically, avoid overload = ascites/pleural effusion/APO

Decompensated shock
1) Obtain HCT level before fluid resus
2) IVD 10-20ml/kg/hr give over 15-30mins then repeat Ix: FBC/HCT/BUSE/LFT/RBS/CoAg/ Lactate/Bicarb / GXM
3) Check HCT if no improvement repeat 2nd bolus 10-20ml/kg/hr 30-60mins then repeat HCT,
   3rd Bolus 10-20ml/kg/hr over 1 hour (with colloids)
* if persistent shock after 3x fluid resus, other causes of shock must be considered \(\rightarrow\) bleeding, sepsis, cardiogenic
* if after fluid resus HCT decrease, consider Tx with packed cell

Mx of bleeding
1) Gum bleeding \(\rightarrow\) Tranexamic acid oral gargle TDS, monitor Hb
2) Occult bleed \(\rightarrow\) when HCT drop without clinical improvement despite fluid resus, blood tx with PC is recommended

ICU care
Ind: persistent shock, respiratory support (mech ventilation), significant bleeding, encephalopathy/encephalitis

Discharge criteria (GO BACK LA)
1) General condition improves
2) Organ dysfn recovered
3) Bleeding episodes resolved
4) Afibrile >48hours
5) Clear lungs- pleural effusion/ascites
6) Kencing (good urine output)
7) Lab-Plt rising, Hct Stable
8) Appetite returns
ALGORITHM A - FLUID MANAGEMENT IN COMPENSATED SHOCK

COMPENSATED SHOCK
(systolic pressure maintained but has signs of reduced perfusion)
- Fluid resuscitation with isotonic crystalloid 5 - 10 ml/kg/hr over 1 hour
- FBC, HCT, before and after fluid resuscitation, BUSEC, LFT, RBS, PT/APTT, Lactate/HCO₃, GXM

IMPROVEMENT

YES
- IV crystalloid 5 - 7ml/kg/hr for 1 - 2 hours, then:
  - reduce to 3 - 5 ml/kg/hr for 2 - 4 hours;
  - reduce to 2 - 3 ml/kg/hr for 2 - 4 hours
- If patient continues to improve, fluid can be further reduced
- Monitor HCT 4 - 6 hourly
- If the patient is not stable, act according to HCT levels:
  - if HCT increases, consider bolus fluid administration or increase fluid administration
  - if HCT decreases, consider transfusion with fresh whole blood
- Consider to stop IV fluid at 48 hours of plasma leakage / defervescence

NO
- Check HCT
- HCT ↑ or high
  - Administer 2nd bolus of fluid
  - 10-20 ml/kg/hr for 1 hr
- HCT ↓
  - Consider significant occult/overt bleed
  - Initiate transfusion with fresh blood (whole blood/packed cell)

IMPROVEMENT

YES
- If patient improves, reduce to 7-10 ml/kg/hr for 1 - 2 hours
- Then reduce further

NO
DECOMPENSATED SHOCK

- Fluid resuscitation with 20 ml/kg/hr isotonic crystalloid or colloid over 15 – 30 minutes
- Try to obtain a HCT level before fluid resuscitation
- FBC, HCT, before and after fluid resuscitation, BUSEC, LFT, RBS, PT/APTT, Lactate/HCO3, GXM

IMPROVEMENT

YES

Review 1st HCT

HCT↑ or high

Administer 2nd bolus of fluid (colloid)
10-20 ml/kg over ½ to 1 hour

NO

HCT↓

Consider significant occult/overt bleed
Initiate transfusion with fresh blood
(whole blood/packed cell)

IMPROVEMENT

Repeat 2nd HCT

HCT↑ or high

Administer 3rd bolus of fluid (colloid)
10-20 ml/kg over 1 hour

NO

HCT↓

Repeat 3rd HCT

IMPROVEMENT

YES

- Crystallloid/colloid 10ml/kg/hr for 1 hour, then continue with:
  - IV crystallloid 5 - 7ml/kg/hr for 1-2 hours;
  - reduce to 3 - 5 ml/kg/hr for 2-4 hours;
  - reduce to 2 - 3 ml/kg/hr for 2-4 hours

- If patient continues to improve, fluid can be further reduced

- Monitor HCT 4 hourly or more frequent as indicated

- If the patient is not stable, act according to HCT levels:
  - if HCT increases, consider bolus fluid administration or increase fluid administration;
  - if HCT decreases, consider transfusion with fresh whole blood

- Consider to stop IV fluid at 48 hours of plasma leakage / defervescence
### Diabetis Mellitus

**Sx = Polyphagia, Polydipsia, Polyuria, weight loss, fatigue, poor wound healing (majority are asymptomatic)**

#### History

<table>
<thead>
<tr>
<th>Specific symptoms</th>
<th>Polyuria, Polydipsia, Polyphagia, Weight loss, Nocturia, Hyperglycaemia, Malaise, Fatigue, Altered vision</th>
</tr>
</thead>
<tbody>
<tr>
<td>Predisposition to diabetes</td>
<td>Age over 35, Family history, Ethnic group, Overweight, Physical inactivity, Hypertension, Obstetric history of large babies or Gestational diabetes, Medication causing hyperglycaemia, Autoimmune disease (personal and/or family history of other autoimmune diseases e.g. hypo or hyperthyroidism)</td>
</tr>
<tr>
<td>Risk factors for complications</td>
<td>Personal or family history of CVD, Smoking, Hypertension, Dyslipidaemia</td>
</tr>
<tr>
<td>General symptoms review</td>
<td>Cardiovascular symptoms, Neurological symptoms, Bladder and sexual dysfunction, Foot and toe problems, Recurrent infections (especially urinary and skin)</td>
</tr>
<tr>
<td>Lifestyle issues</td>
<td>Smoking, Alcohol, Occupation, Eating and physical activity</td>
</tr>
</tbody>
</table>

#### Examination

<table>
<thead>
<tr>
<th>Weight/waist</th>
<th>BMI = weight (kg) divided by height² (m²), WC</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiovascular</td>
<td>Blood pressure (lying and standing), Peripheral neck and abdominal systemic vessels</td>
</tr>
<tr>
<td>Eye</td>
<td>Visual acuity (with corrected vision), Cataract, Retinopathy (examine with pupils dilated)</td>
</tr>
<tr>
<td>Feet</td>
<td>Sensation and circulation, Skin condition, Pressure areas, Interdigital problems, Abnormal bony architecture</td>
</tr>
<tr>
<td>Peripheral Nerves</td>
<td>Tendon reflexes, Sensation, touch (e.g. with 10G monofilament), vibration (e.g. with 128Hz tuning fork)</td>
</tr>
</tbody>
</table>

#### Investigations

<table>
<thead>
<tr>
<th>Baseline</th>
<th>Urinalysis: albumin, microalbuminuria, Renal profile: plasma urea and creatinine, Lipids: Low density lipoprotein (LDL) cholesterol, HDL cholesterol, Triglycerides, Glycaemia: FPG, HbA1c</th>
</tr>
</thead>
<tbody>
<tr>
<td>Others</td>
<td>ECG, Thyroid function tests if there is a family history or clinical suspicion</td>
</tr>
</tbody>
</table>
Oral Glucose Tolerance Test

<table>
<thead>
<tr>
<th></th>
<th>Fasting</th>
<th>Random</th>
</tr>
</thead>
<tbody>
<tr>
<td>Venous Plasma Glucose</td>
<td>≥ 7.0 mmol/L</td>
<td>≥ 11.1 mmol/L</td>
</tr>
</tbody>
</table>

In the symptomatic individual, one abnormal glucose value is diagnostic.

In the asymptomatic individual, 2 abnormal glucose values are required.

Algorithm 1: Screening for type 2 diabetes mellitus at primary care level – with symptoms

WITH SYMPTOMS

Venous Plasma Glucose

- Fasting
  - <7.0
    - OGTT
  - ≥7.0
- Random
  - ≥11.1
    - OGTT
  - <11.1

Type 2 Diabetes Mellitus

* All values in mmol/L. Capillary whole blood reading is 12% lower than venous plasma glucose.

**Oral Glucose Tolerance Test**

<table>
<thead>
<tr>
<th>Category</th>
<th>0-hour</th>
<th>2-hour</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>&lt; 6.1*</td>
<td>&lt; 7.8</td>
</tr>
<tr>
<td>IFG</td>
<td>6.1* – 6.9</td>
<td>-</td>
</tr>
<tr>
<td>IGT</td>
<td>-</td>
<td>7.8 – 11.0</td>
</tr>
<tr>
<td>DM</td>
<td>≥ 7.0</td>
<td>≥ 11.1</td>
</tr>
</tbody>
</table>

Ix: FBS/FSL/HbA1C/RP/LFT/UFEME
Table 3: Targets for Type 2 Diabetes Mellitus

<table>
<thead>
<tr>
<th>Glycaemic Control</th>
<th>Levels</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fasting</td>
<td>4.4 – 6.1 mmol/L</td>
</tr>
<tr>
<td>Non-Fasting</td>
<td>4.4 – 8.0 mmol/L</td>
</tr>
<tr>
<td>HbA1c</td>
<td>&lt;6.5 %</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Lipids</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Triglycerides</td>
<td>≤1.7 mmol/L</td>
</tr>
<tr>
<td>HDL cholesterol</td>
<td>≥1.1 mmol/L</td>
</tr>
<tr>
<td>LDL cholesterol</td>
<td>≤2.6 mmol/L</td>
</tr>
<tr>
<td>Exercise</td>
<td>150 mins/week</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Blood Pressure</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal Renal Function</td>
<td>≤130/80 mmHg</td>
</tr>
<tr>
<td>Renal Impairment/Gross Proteinuria</td>
<td>≤125/75 mmHg</td>
</tr>
</tbody>
</table>

**Treatment**

1) **Lifestyle Modification** (3 months, to achieve control HbA1c <6.5, FBS <6mmol)
   - Diet modification (medical nutrition therapy)
   - Physical Activity

2) **Medication**
   i) Monotherapy OHA: Metformin (HbA1c 6.5-8%, FBS 6-10)
   ii) combination of OHA

<table>
<thead>
<tr>
<th>Monotherapy OHA</th>
<th>HbA1C</th>
<th>FBS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Metformin</td>
<td>6.5-8</td>
<td>6-10</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Combination of OHA</th>
<th>HbA1C %</th>
<th>FBS</th>
</tr>
</thead>
<tbody>
<tr>
<td>OHA + Insulin</td>
<td>&gt;10%</td>
<td>&gt;13</td>
</tr>
</tbody>
</table>

**Groups**

1) **Biguanides (eg: Metformin)**
   MOA: reduces hepatic glucose production  
   CI: impaired renal fn (Cr >150, CrCl <30), liver cirrhosis, CCF, recent MI, chr resp dis (any dis that cause lactate accumulation)  
   Dose: start 500mg OD up to 500mg TDS, Max 1g BD

2) **Secretagogues** - sulfanyureas  
   MOA: increases insulin secretion  
   CI: renal impairment, elderly >65yo  
   SE: hepatitis, SiADH  
   Dose: Glicazide start 40mgOM -Max 160mg BD , Glibenclamide start 2.5mg OM, Max 10mg BD

**Rarely used**
- AGIs – Acarbose  
- Dipeptidyl peptidase 4 (DPP4) – Sitagliptin 
- Thiazolidinediones (TZDs) – Rosiglitazone, Pioglitazone

- Refer to appendix for full list of doses and drugs
INSULINOTHERAPY:
1) 0.5U/kg/day (then 0.1U/kg premeal + 0.2U/kg bedtime)

2) Changing from s/s to insulin basal bolus
   - Add up 24 hours sliding scale Units or start by
   - Total daily insulin/4 = ⅔ Actrapid, ¼ Insulatard
   - For BD insulin (mixtard) = Total insulin = 2/3 AM, 1/3 ON

---

**INSULIN INITIATION AND OPTIMISATION**

<table>
<thead>
<tr>
<th>Insulin Regimen</th>
<th>Starting Dose</th>
<th>Dose Optimisation</th>
<th>Optimal Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Basal</td>
<td>10 units or 0.2U/kg at bedtime (0.1 units/kg if higher risk for hypos)</td>
<td>Adjust insulin doses after 3 consecutive BG values obtained (every 3 – 7 days) Refer to (*)</td>
<td>0.2 – 0.3 units/kg in lean patients 0.4 – 0.6 units/kg in most patients Up to 0.7 units/kg in obese patients</td>
</tr>
<tr>
<td>Premixed</td>
<td>Once daily: 10 units or 0.2U/kg at pre-dinner Twice daily: 10 units or 0.2U/kg at pre-breakfast and pre-dinner (0.1 units/kg if higher risk for hypos)</td>
<td>Adjust insulin doses after 3 consecutive BG values obtained (every 3 – 7 days) Refer to (*) Pre-breakfast BG determine pre-dinner premixed dose adjustment Pre-breakfast BG determine pre-breakfast premixed dose adjustment</td>
<td>Total daily dose of 0.5 – 1.0 units/kg in most patients (Maybe more than 1.0 units/kg/day in obese, insulin resistant patients)</td>
</tr>
<tr>
<td>Prandial</td>
<td>6 units or 0.1units/kg for each meal with short-acting or rapid-acting analogue.</td>
<td>Adjust insulin doses after 3 consecutive BG values obtained (every 3 – 7 days) Refer to (*) Adjust the dose of prandial insulin of the preceding meal (e.g. if pre lunch BG is high, adjust pre-breakfast prandial insulin)</td>
<td>Prandial dose for each meal will vary according to carbohydrate content and amount. Dose should ideally not exceed 0.5U/kg/dose.</td>
</tr>
<tr>
<td>Basal Bolus</td>
<td>Prandial Insulin: 6 units or 0.1U/kg before each meal Basal insulin: 10 units or 0.20U/kg at bedtime</td>
<td>Refer to Prandial Section Refer to Basal Section Aim for normal pre-breakfast BG first by adjusting the dose of bed-time basal insulin before adjusting the prandial (bolus) insulin dose.</td>
<td>Generally basal insulin would contribute 50% of total daily insulin dose and prandial insulin would contribute remaining 50% (distributed over three main meals). Refer to Prandial Section &amp; Basal Section</td>
</tr>
</tbody>
</table>

(*) - < 4 mmol/L (≥ 1 value) — reduce dose by 2 units
- 4-6 mmol/L (all values) — maintain current dose
- > 6 mmol/L (≥1 value, no hypos) — increase by 2 units
Diabetic Ketoacidosis
= state of absolute/relative insulin insufficiency
Hyperglycemia (>14mmol/L ) +
Metabolic Acidosis (pH<7.3, Bicarb <15mmol/L)
Ketonemia/ketonuria

Precipitated by:
1) Infection, sepsis 2) Dehydration
3) Trauma 4) surgery 5) stress factors

Lab Investigations
FBC: WCC raised
RP: K raised, Na decrease/N, Urea & Cr raised
UFEME: ketones elevated
Urine ketones high
VBG: Metabolic acidosis, pH <7.3, Bicarb <15

Mx:
1) correction of fluid loss/hypovolemia/dehydration
DKA regime→NS 0.9% 1 L x 1hr then x 2 hr, x 4hr, x 6hr, x8hr
*monitor BUSE tds
* when DXT <15 , alternate with DS

2) electrolyte imbalance
a) HypoK+: correct if K <5.3, maintain levels at 4-5
1g KCl in 0.5L, adjust accordingly if,

<table>
<thead>
<tr>
<th>K+</th>
<th>Withhold insulin, add 40mmol/L</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;3</td>
<td></td>
</tr>
<tr>
<td>3-4</td>
<td>30</td>
</tr>
<tr>
<td>4-5</td>
<td>20</td>
</tr>
<tr>
<td>&gt;5</td>
<td>None</td>
</tr>
</tbody>
</table>
* monitor BUSE TDS, ECG, urine output

b) Na: if high, change to 0.45% NS

3) hyperglycemia: start IVI insulin sliding scale 0.1U/kg/hr
* not to decrease DXT >5 per hr (cerebral edema) maintain DXT at 8-12
when DXT < 15, halve the insulin rate, IVD change to dextrose containing solution (D5%/ DS)
* Change to s/c insulin when acidosis resolves, urine ketones neg/1+, plasma ketone <1mmol

Changing to insulin basal bolus: add up 24hours sliding scale Units
Total daily insulin/4 = ¼ Actrapid, ¼ Insulatard
For BD insulin = Total insulin = 2/3 AM, 1/3 ON

4) acidosis
Bicarbonate therapy after adequate hydration if arterial pH <6.9 + severe hyperK with ECG changes
8.4% NaHCO3 100ml in H2O 400ml + 20mmol KCL over 2hrs until Venous pH >7.0, if pH still <7.0, rpt q2Hr till ph 7.0
* w/o for hypoglycaemia, cerebral edema, pulmonary edema, vascular thrombosis, hypoK

Hyperglycaemic Hyperosmolar State (HHS)
= Relative insulin insufficiency resulting in marked hyperglycemia but not/minimal ketone formation
Hyperglycemia >33mmol/L + pH >7.3 , Bicarb .18 , no ketones

Calculation:
Effective serum Osmolality = 2(Na +K) + RBS + Urea = > 320mmol/L
Total Osmolality = 2(Na) + RBS + urea = >330mmol/L
Anion gap = Na – (Cl+bicarb)

Mx almost same as DKA, requires more hydration
* maintain DXT 12-16mmol, when DXT < 15, halve the insulin rate, IVD change to D5%
* CVL may be inserted to aid fluid replacement assessment (fluid deficit usually ~9L)
Hypertension = persistent elevation of systolic BP >140/90

<table>
<thead>
<tr>
<th>Category</th>
<th>Systolic (mmHg)</th>
<th>Diastolic (mmHg)</th>
<th>Prevalence in Malaysia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Optimal</td>
<td>&lt;120</td>
<td>&lt;80</td>
<td>32%</td>
</tr>
<tr>
<td>Prehypertension</td>
<td>120-139</td>
<td>80-89</td>
<td>37%</td>
</tr>
<tr>
<td>Hypertension</td>
<td>140-159</td>
<td>90-99</td>
<td>20%</td>
</tr>
<tr>
<td>Stage 1</td>
<td>160-179</td>
<td>100-109</td>
<td>8%</td>
</tr>
<tr>
<td>Stage 2</td>
<td>≥180</td>
<td>≥110</td>
<td>4%</td>
</tr>
</tbody>
</table>

The classification is based on the average of two or more readings taken at two or more visits to the doctor. When SBP and DBP fall into different categories, the higher category should be selected to classify the individual’s BP.

**Evaluation**
1) Exclude secondary causes (table 2)
2) Look for any Target organ damage ➔
3) Identify other CVS risks (table 3)

**History**
1) Duration of elevated BP (if known)
2) Sx of secondary causes
3) Sx of Target Organ Damage
4) Co morbid: DM, renal dis, gout etc
5) Fam Hx: IHD, HTN, CKD, dyslipidemia
6) Social: diet, alcohol, smoker, caffeine
7) Med hx: OTC drugs, herbal

**O/E:**
General: alert, conscious
BP * 2 or more separated by 2 mins supine/seating and standing for 1 min (resting, no caffeine or rest)

**other exams:**
- Fundoscopy
- Neuro: sx of CVA
- Carotid/abdominal bruit, aneurysm
- endocrine disorders

**Investigation:**
Lab: FBC/RP/LFT/FBS/FSL/uric acid/UFEME
ECG + CXR

**Management:**
1) Lifestyle changes
2) exercise
3) Antihypertensives

**Target BP**
< 65 yo: <140/85
Diabetics: <130/80
Kidney disease <125/75

**Table 2. Secondary causes of hypertension**

<table>
<thead>
<tr>
<th>Secondary causes of hypertension</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sleep apnoea</td>
</tr>
<tr>
<td>Drug-induced or drug-related</td>
</tr>
<tr>
<td>Chronic kidney disease</td>
</tr>
<tr>
<td>Primary aldosteronism</td>
</tr>
<tr>
<td>Renovascular disease</td>
</tr>
<tr>
<td>Chronic steroid therapy and Cushion syndrome</td>
</tr>
<tr>
<td>Phaeochromocytoma</td>
</tr>
<tr>
<td>Acromegaly</td>
</tr>
<tr>
<td>Thyroid or parathyroid disease</td>
</tr>
<tr>
<td>Coarctation of the aorta</td>
</tr>
<tr>
<td>Takayasu Arteritis</td>
</tr>
</tbody>
</table>

**Organ system**

<table>
<thead>
<tr>
<th>Organ system</th>
<th>Manifestations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiac</td>
<td>Left ventricular hypertrophy, coronary heart disease, heart failure</td>
</tr>
<tr>
<td>Cerebrovascular</td>
<td>Transient ischaemic attack, stroke</td>
</tr>
<tr>
<td>Peripheral vasculature</td>
<td>Absence of one or more major pulses in extremities (except dorsalis pedis) with or without intermittent claudication</td>
</tr>
<tr>
<td>Renal</td>
<td>GFR &lt;60 ml/min/1.73 m², proteinuria (1+ or greater), microalbuminuria (2 out of 3 positive tests over a period of 4-6 months)</td>
</tr>
<tr>
<td>Retinopathy</td>
<td>Haemorrhages or exudates, with or without papilloedema</td>
</tr>
</tbody>
</table>

**Table 3. Cardiovascular risk factors**

<table>
<thead>
<tr>
<th>Major risk factors</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypertension</td>
</tr>
<tr>
<td>Cigarette smoking</td>
</tr>
<tr>
<td>Central obesity (waist circumference &gt;90 cm for men, &gt;80 cm for women)</td>
</tr>
<tr>
<td>Physical inactivity</td>
</tr>
<tr>
<td>Dyslipidaemia</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
</tr>
<tr>
<td>Microalbuminuria</td>
</tr>
<tr>
<td>Estimated GFR &lt;60 ml/min</td>
</tr>
<tr>
<td>Age (&gt;55 years for men, &gt;65 years for women)</td>
</tr>
<tr>
<td>Family history of premature cardiovascular disease (men &lt;55 years or women &lt;65 years)</td>
</tr>
</tbody>
</table>

**Target Organ Damage**

<table>
<thead>
<tr>
<th>Heart</th>
<th>Left ventricular hypertrophy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Angina or prior myocardial infarction</td>
<td></td>
</tr>
<tr>
<td>Prior coronary revascularisation</td>
<td></td>
</tr>
<tr>
<td>Heart failure</td>
<td></td>
</tr>
<tr>
<td>Brain</td>
<td>Stroke or transient ischemic attack</td>
</tr>
<tr>
<td>Chronic kidney disease</td>
<td></td>
</tr>
<tr>
<td>Peripheral arterial disease</td>
<td></td>
</tr>
<tr>
<td>Retinopathy</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Grade</th>
<th>Classification</th>
</tr>
</thead>
<tbody>
<tr>
<td>Grade I</td>
<td>Mild generalized retinal arteriolar narrowing or sclerosis</td>
</tr>
<tr>
<td>Grade II</td>
<td>Definite focal narrowing and arteriovenous crossings</td>
</tr>
<tr>
<td>Moderate to marked sclerosis of the retinal arterioles</td>
<td></td>
</tr>
<tr>
<td>Exaggerated arterial light reflex</td>
<td></td>
</tr>
<tr>
<td>Grade III</td>
<td>Retinal hemorrhages, exudates and cotton wool spots</td>
</tr>
<tr>
<td>Sclerosis and atrophic lesions of retinal arterioles</td>
<td></td>
</tr>
<tr>
<td>Grade IV</td>
<td>Severe grade III and papilloedema</td>
</tr>
</tbody>
</table>
HPT + DM
Target BP: <130/80
Drug of choice:
1) ACEi (CVS + renal protective)
2) ARB (if x tolerate ACEi, proteinuria +)
3) + Diuretics / CCB / BB (if monotherapy inadequate or contraindicated)

HPT + non diabetic renal dis
Target BP: proteinuria <1g/24H: <130/80; proteinuria >1g/24H: <120/75
Mx
1) ACEi (anti-proteinuric effect) = drug of choice
   * ACEi is not contraindicated in renal insufficiency but close monitoring of Creatinine rqd
   Check after 2 weeks of initiation, If >30% increase frm baseline, stop ACEi

2) Loop diuretics: for fluid overload, preferred if Cr > 200 (thiazides will be ineffective)
3) Salt and fluid restriction
4) Non dihydropyridine CCB (verapamil/deltiazem) + anti proteinuric effect

HPT + Heart Disease
Left ventricle Hypertrophy
1) ARB

Coronary Heart Dis
1) Beta Blockers = reduce O2 demand
2) ACEi
3) long acting CCB

Congestive Heart Failure
1) Diuretics – aldosterone antagonist
2) Beta Blockers
3) ACEi/ARB

HPT + Stroke
1) CCB (better primary prevention)
2) ACEi/ARB + Diuretics (secondary prevention)
   * Acute stroke = do not start antihpt unless with Hpt emergency >220/110

<table>
<thead>
<tr>
<th>Group</th>
<th>Pros</th>
<th>Adverse Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>ACEi</td>
<td>+ anti proteinuric effect</td>
<td>Dry Cough, angioedema, renal artery Stenosis, fetal/neonatal mortality</td>
</tr>
<tr>
<td></td>
<td>I: DM, renal dis, CHD,CHF, stroke</td>
<td></td>
</tr>
<tr>
<td>ARB</td>
<td>+ reduce mortality in heart failure/LVH</td>
<td>Renal artery stenosis</td>
</tr>
<tr>
<td>Beta Blockers</td>
<td>+ reduce effort angina, tachyarrhythmias, previous MI</td>
<td>Obs airway dis, severe PVD, heart block</td>
</tr>
<tr>
<td>CCB</td>
<td>+ primary prevention of stroke, coronary heart dis</td>
<td>Tachycardia, headache, flushing, constipation, pedal edema</td>
</tr>
<tr>
<td>Diuretics</td>
<td>Congestive HF, elderly</td>
<td>Increase cholesterol, glucose, uric acid decrease K, Na, Mg</td>
</tr>
</tbody>
</table>

*refer to appendix for full drugs list and doses

Revision: the Korotkoff sound

<table>
<thead>
<tr>
<th>Phase</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Silence</td>
</tr>
<tr>
<td>2</td>
<td>A tapping sound</td>
</tr>
<tr>
<td>3</td>
<td>A soft swishing sound</td>
</tr>
<tr>
<td>4</td>
<td>A crisp sound</td>
</tr>
<tr>
<td>5</td>
<td>A blowing sound</td>
</tr>
<tr>
<td></td>
<td>Silence</td>
</tr>
</tbody>
</table>

Cuff pressure / mmHg
Systolic pressure
Diastolic pressure
Hypertensive Crisis = elevation of diastolic BP >120, with or without Target Organ damage

Classification
1) Hypertensive Urgency = elevation of BP but without signs of TOD
2) Hypertensive Emergency = elevation of BP with signs of TOD

<table>
<thead>
<tr>
<th>Sx of TOD</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Brain</td>
<td>Stroke/TIA, seizure, coma, severe headache</td>
</tr>
<tr>
<td>Eyes</td>
<td>Retinopathy, papilloedema</td>
</tr>
<tr>
<td>Heart</td>
<td>LVH, Angina/MI, heart failure</td>
</tr>
<tr>
<td>Kidney</td>
<td>CKD</td>
</tr>
<tr>
<td>Limbs</td>
<td>Peripheral arterial disease</td>
</tr>
</tbody>
</table>

Mx
- Rapid reduction of BP (within minutes to hours) in asymptomatic severe hypertension or hypertensive urgencies is best avoided as it may precipitate ischaemic events.

1) Hypertensive Urgency
- BP should be repeated after 30mins bed rest
- aim for reduction by 25% over 24 hours, not <160/90
- recommended to use Oral drugs:

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dose</th>
<th>Onset of action</th>
<th>Duration</th>
<th>Frequency</th>
</tr>
</thead>
<tbody>
<tr>
<td>Captopril</td>
<td>25 mg</td>
<td>0.5</td>
<td>6</td>
<td>1 - 2 hrs</td>
</tr>
<tr>
<td>Nifedipine</td>
<td>10 - 20 mg</td>
<td>0.5</td>
<td>3 - 5</td>
<td>1 - 2 hrs</td>
</tr>
<tr>
<td>Labetalol</td>
<td>200 - 400 mg</td>
<td>2.0</td>
<td>6</td>
<td>4 hrs</td>
</tr>
</tbody>
</table>

2) Hypertensive Emergency
- BP needs to be reduced rapidly
- possible complications: acute HF, hypertensive encephalopathy, SAH, acute renal failure
- aim for reduction by 25% over 3-12 hours

<table>
<thead>
<tr>
<th>Drugs</th>
<th>Dose</th>
<th>Onset of action</th>
<th>Duration</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sodium nitroprusside</td>
<td>0.25 - 10 μg/kg/min</td>
<td>seconds</td>
<td>1 - 5 min</td>
<td>Caution in renal failure</td>
</tr>
<tr>
<td>Labetalol</td>
<td>IV bolus 50 mg (at least 1 minute) repeating if necessary at 5 minute intervals up to a max of 200 mg then 2 mg/min IVI</td>
<td>≤5 min</td>
<td>3 - 6 hrs</td>
<td>Caution in heart failure</td>
</tr>
<tr>
<td>Nitrites</td>
<td>5 - 100 μg/min</td>
<td>2 - 5 min</td>
<td>3 - 5 min</td>
<td>Preferred in acute coronary syndromes and acute pulmonary edema</td>
</tr>
<tr>
<td>Hydralazine</td>
<td>IV 5-10 mg maybe repeated after 20 - 30 minutes IVI 200-300 mcg/min initially. Maintenance 50-150 mcg/min</td>
<td>10 - 20 min</td>
<td>3 - 8 hrs</td>
<td>Caution in acute coronary syndromes, cerebrovascular accidents and dissecting</td>
</tr>
<tr>
<td>Nicardipine</td>
<td>IV bolus 10-30 mcg/kg over 1 minute IVI 2-10 mcg/kg/min</td>
<td>3 - 10 min</td>
<td>1 - 4 hrs</td>
<td>Caution in acute heart failure and coronary</td>
</tr>
<tr>
<td>Esmolol</td>
<td>IV bolus1-2 min 250-500 mcg/kg over 1 min IVI 50-200 mcg/kg/min for 4 min. May repeat sequence</td>
<td>3 - 10 min</td>
<td></td>
<td>Operative and tachyarrhythmias</td>
</tr>
</tbody>
</table>
FIGURE 1. Algorithm for the Management of Hypertension

BLOOD PRESSURE
(Repeated Readings)

SBP = 120 - 159 mmHg
AND/OR
DBP = 80 - 99 mmHg

Assess global cardiovascular risk
(refer to Table 4 and Table 5)

Medium/High/Very High

Drug Treatment

Low

3-6 monthly follow-up with advice on non-pharmacological management

SBP < 140 mmHg
AND/OR
DBP < 90 mmHg

6-monthly follow-up

SBP 140-149 mmHg
AND/OR
DBP 90-94 mmHg

3-6 monthly follow-up and assessment

SBP ≥ 150 mmHg
AND/OR
DBP ≥ 95 mmHg

Drug treatment
Bronchial Asthma

**Asthma pathogenesis**
- Attacks of SOB/wheezing
- Spasms bronchus
- Treatable/reversible
- Hypersensitivity of airway
- Mucous hypersecretion
- Allergic/Atopic cause

**Sx:**
- Cough worsening at night/early morning
- Aggravated by allergen/triggers
- PEFR increase >15% after SABA

**Criteria (GINA)**
- Activity Limitation
daytime
- Symptoms anytime
- Test – lung fn PEF <80%
- Inhaler (inhaler) rescue
- Malam symptoms
- Attacks-exacerbation

**Classification by GINA**

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Controlled</th>
<th>Partly Controlled</th>
<th>Uncontrolled</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(All of the following)</td>
<td>(Any measure present in any week)</td>
<td>Three or more features of partly controlled asthma present in any week</td>
</tr>
<tr>
<td>Daytime symptoms</td>
<td>None (twice or less/week)</td>
<td>More than twice/week</td>
<td></td>
</tr>
<tr>
<td>Limitations of activities</td>
<td>None</td>
<td>Any</td>
<td></td>
</tr>
<tr>
<td>Nocturnal symptoms/awakening</td>
<td>None</td>
<td>Any</td>
<td></td>
</tr>
<tr>
<td>Need for reliever/rescue treatment</td>
<td>None (twice or less/week)</td>
<td>More than twice/week</td>
<td></td>
</tr>
<tr>
<td>Lung function (PEF or FEV)</td>
<td>Normal</td>
<td>&lt;80% predicted or personal best (if known)</td>
<td></td>
</tr>
<tr>
<td>Exacerbations</td>
<td>None</td>
<td>One or more/year*</td>
<td>One in any week†</td>
</tr>
</tbody>
</table>

* Any exacerbation should prompt review of maintenance treatment to ensure that it is adequate.
† by definition, an exacerbation in any week makes that an uncontrolled asthma week.
‡ Lung function is not a reliable test for children 5 years and younger.

---

**Consider asthma if any of the following signs or symptoms are present:**
- Wheezing = high-pitched whistling sounds when breathing out (A normal chest examination does not exclude asthma).
- History of any of the following:
  - cough, worse particularly at night/early morning
  - recurrent difficulty in breathing
  - recurrent wheeze
  - recurrent chest tightness

*Note: Eczema, hay fever, or a family history of asthma or atopic diseases is often associated with asthma.*

- Symptoms occur or worsen at night/early morning, awakening the patient
- Symptoms occur or worsen in the presence of:
  - exercise
  - animals
  - pollen
  - aerosol chemicals
  - dust mites (in mattress, pillows, upholstered furniture, carpets)
  - respiratory tract infection
  - smoke (tobacco, wood)
  - changes in temperature
  - drugs (aspirin, beta blockers)
  - strong emotional expression (laughing or crying hard)

- Reversible and variable airflow limitation as measured by a peak expiratory flow (PEF) meter in any of the following ways:
  - □ PEF increases more than 15% 15 to 20 minutes after inhaling a short-acting beta2-agonist, or
  - □ PEF varies more than 20% from morning measurement upon arising to measurement 12 hours later in patients who are taking a bronchodilator (more than 10% in patients who are not taking a bronchodilator), or
  - □ PEF decreases more than 15% after 6 minutes of running or exercise
### Classification of Asthma Severity Before Treatment

<table>
<thead>
<tr>
<th></th>
<th>Symptoms</th>
<th>Night time Symptoms</th>
<th>PEF</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Severe Persistent</strong></td>
<td>Daily Frequent exacerbations Limitation of physical activity</td>
<td>Frequent</td>
<td>≤ 60% predicted Variability &gt; 30%</td>
</tr>
<tr>
<td><strong>Moderate Persistent</strong></td>
<td>Daily Daily use of beta₂-agonist Exacerbations affect activity and sleep</td>
<td>&gt; 1 time a week</td>
<td>&gt; 60% - &lt; 80% predicted Variability &gt; 30%</td>
</tr>
<tr>
<td><strong>Mild Persistent</strong></td>
<td>≥ 1 time a week but &lt; 1 time a day Exacerbations may affect activity and sleep</td>
<td>&gt; 2 times a month</td>
<td>≥ 80% predicted Variability 20-30%</td>
</tr>
<tr>
<td><strong>Intermittent</strong></td>
<td>&lt; 1 time a week Brief exacerbations Asymptomatic and normal PEF between exacerbations</td>
<td>≤ 2 times a month</td>
<td>≥ 80% predicted Variability &lt; 20%</td>
</tr>
</tbody>
</table>

### Management Approach Based On Control

**For Children Older Than 5 Years, Adolescents and Adults**

- **Level of Control**
  - Controlled
  - Partly controlled
  - Uncontrolled
  - Exacerbation

- **Treatment Action**
  - Maintain and find lowest controlling step
  - Consider stepping up to gain control
  - Step up until controlled
  - Treat as exacerbation

- **Treatment Steps**
  - Step 1: Asthma education
  - Step 2: Environmental control
  - Step 3: As needed rapid-acting β₂-agonist
  - Step 4: Controller options
  - Step 5: As needed rapid-acting β₂-agonist

**Controller options***

- Low-dose ICS
- Leukotriene modifier **
- Low-dose ICS plus leukotriene modifier
- Low-dose ICS plus sustained release theophylline
- Low-dose ICS plus high-dose ICS

**As needed rapid-acting β₂-agonist**

- Select one
- Select one or more
- Add one or both
- Low-dose ICS plus medium-dose ICS
- Medium-dose ICS plus low-dose ICS
- Medium-dose ICS plus high-dose ICS
- Leukotriene modifier
- Ant- IgE treatment
- Oral glucocorticosteroid (lowest dose)
### TREATMENT OF ADULT ASTHMA

Preferred treatments are in bold print
Patient education is essential at every step

<table>
<thead>
<tr>
<th></th>
<th>Long-Term Preventive</th>
<th>Quick-Relief</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>STEP 4</strong></td>
<td><strong>Severe Persistent</strong></td>
<td></td>
</tr>
<tr>
<td>Daily medications:</td>
<td>• Inhaled corticosteroid*, 800-2000 mcg, and</td>
<td>• Short-acting bronchodilator: *inhaled beta₂-agonist as needed for symptoms</td>
</tr>
<tr>
<td></td>
<td>• Long-acting bronchodilator: either inhaled long-acting beta₂-agonist and/or sustained-release theophylline, and/or oral long acting beta₂-agonist, and</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Oral corticosteroid long term</td>
<td></td>
</tr>
<tr>
<td><strong>STEP 3</strong></td>
<td><strong>Moderate Persistent</strong></td>
<td></td>
</tr>
<tr>
<td>Daily medications:</td>
<td>• Inhaled corticosteroid*, 500-1000 mcg AND, if needed</td>
<td>• Short-acting bronchodilator: *inhaled beta₂-agonist as needed for symptoms</td>
</tr>
<tr>
<td></td>
<td>• Long-acting bronchodilator: either inhaled long-acting beta₂-agonist, sustained-release theophylline, or oral long acting beta₂-agonist (Inhaled long-acting beta₂-agonist may provide more effective symptom control when added to low-medium dose steroid compared to increasing the steroid dose)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Consider adding anti-leukotriene, especially for aspirin-sensitive patients and for preventing exercise-induced bronchospasm</td>
<td></td>
</tr>
<tr>
<td><strong>STEP 2</strong></td>
<td><strong>Mild Persistent</strong></td>
<td></td>
</tr>
<tr>
<td>Daily medications:</td>
<td>• Either Inhaled corticosteroid*, 200-500 mcg, or cromoglycate or sustained-release theophylline or anti-leukotrienes</td>
<td>• Short-acting bronchodilator: *inhaled beta₂-agonist as needed for symptoms</td>
</tr>
<tr>
<td><strong>STEP 1</strong></td>
<td><strong>Intermittent</strong></td>
<td></td>
</tr>
<tr>
<td></td>
<td>• None needed</td>
<td>• Short-acting bronchodilator: *inhaled beta₂-agonist as needed for symptoms, but less than once a week</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Inhaled beta₂-agonist or cromoglycate before exercise or exposure to allergen</td>
</tr>
</tbody>
</table>

\[
\frac{\text{PEF (max)} - \text{PEF (min)}}{\text{PEF (max)}} \times 100 = \text{_____%}
\]

* Variability should be maintained <10%
**Acute Exacerbations of BA (AEBA)**

Secondary to: URTI/ CAP/ allergens

<table>
<thead>
<tr>
<th>Mild</th>
<th>severe</th>
<th>Very severe</th>
<th>Life Threatening</th>
</tr>
</thead>
<tbody>
<tr>
<td>Persistent cough</td>
<td>breathless when talking talks in phrases</td>
<td>breathless at rest talks in words</td>
<td>central cyanosis</td>
</tr>
<tr>
<td>Increased chest tightness</td>
<td>loud wheeze</td>
<td>loud wheeze</td>
<td>exhaustion</td>
</tr>
<tr>
<td>Breathless when walking</td>
<td>pulse rate 100-120/min respiratory rate 25-30 PEF between 50 to 75 SpO2 91-95% (on room air)</td>
<td>pulse rate &gt; 120/min respiratory rate &gt; 30 • PEF &lt; 50% SpO2 &lt; 90% (on room air)</td>
<td>confusion or unconsciousness</td>
</tr>
<tr>
<td>Normal speech</td>
<td></td>
<td></td>
<td>or convulsion</td>
</tr>
<tr>
<td>Moderate wheeze on auscultation, often end expiratory only</td>
<td></td>
<td></td>
<td>Fleeble respiratory effort</td>
</tr>
<tr>
<td>Pulse rate &lt; 100/min</td>
<td></td>
<td></td>
<td>silent chest on auscultation</td>
</tr>
<tr>
<td>Respiratory rate &lt; 25</td>
<td></td>
<td></td>
<td>Bradycardia or hypotension</td>
</tr>
<tr>
<td>PEF &gt; 75%</td>
<td></td>
<td></td>
<td>Fleeble respiratory effort</td>
</tr>
<tr>
<td>• SpO2 &gt; 95% (on room air)</td>
<td></td>
<td></td>
<td>• PEF &lt; 30% (&lt; 100L/min)</td>
</tr>
</tbody>
</table>

**Management of Chronic Asthma**

**Aims**
1. Abolish day and night symptoms
2. Restore long term airway fn
3. Prevent acute attacks
4. Prevent mortality

**Assessment**
- Identify and avoid TRIGGER factors
- Assess SEVERITY and monitor RESPONSE to tx
- EDUCATE – patient and family

**Medications**

1) **Anti-inflamatory**
   a) **Corticosteroids** = Inhaled: MDI Beclomethasone, Budesonide, Fluticasone
   Oral: prednisolone 30-60mg OD
   IV Hydrocortisone 200mg stat, 100mg tds
   b) **Cromones** – Sodium cromoglycate
   c) **Antileukotrienes** – Montelukast 10mg OD

2) **Short acting bronchodilators**
   a) SABA – MDI Salbutamol 200mcg PRN, Terbutaline, Fenoterol
   b) Anticholinergics – Ipatropium bromide
   c) Methylxanthines -Theophylline

3) **Long acting bronchodilators** (LABA)→used in combination with CS-Salmeterol/Fluticasone, Budesonide/formoterol
   a) **LABA**
   Inhaled: Formoterol, Salmeterol
   Oral: bumberal, Salbutamol SR, Terbutaline SR, Clenbuterol
   b) Methylxanthines – Theophylline (nuelin 250mg BD)
Assess asthma severity clinically and with PEF

Mild acute asthma
PEF > 75% predicted/best

Give inhaled or nebulised beta₂-agonist

Observe for 60 mins. If PEF > 75% and clinically improved, discharge patient

Moderate to severe acute asthma
PEF < 75% predicted/best

Give oxygen > 40%
Give nebulised beta₂-agonist or multiple puffs of MDI via a large spacer in combination with inhaled ipratropium

Give oral prednisolone 30-60 mg or i.v. hydrocortisone 100-200 mg stat

Give i.v. aminophylline 250 mg slowly over 20 mins or i.v. terbutaline/salbutamol 0.25 mg over 10 mins. AND ADMIT patient to ICU. Do not give bolus aminophylline if patient is on oral theophylline

Poor response
PEF < 50% with deteriorating or persistent symptoms and signs

Admit patient

Life threatening acute asthma
PEF < 30% predicted/best

Give oxygen > 40%
Give nebulised beta₂-agonist or multiple puffs of MDI via a large spacer in combination with inhaled ipratropium

Give oral prednisolone 30-60 mg or i.v. hydrocortisone 100-200 mg stat

Give i.v. aminophylline 250 mg slowly over 20 mins or i.v. terbutaline/salbutamol 0.25 mg over 10 mins. AND ADMIT patient to ICU. Do not give bolus aminophylline if patient is on oral theophylline

Good response
PEF > 75% and clinically improved

Incomplete response
PEF 50-75% with persistent symptoms and signs

Repeat nebulised beta₂-agonist and ipratropium
Observe for 60 mins

Observe for 60 mins. If PEF > 75% discharge patient

PEF > 75%, discharge

PEF < 75%, admit

NB: Before discharge ensure that patient’s treatment plan is reviewed, the medicine is adequate, the inhaler technique is correct, the appointment for review is given, and patient is advised to return if condition deteriorates. If patient was given steroids, continue with oral prednisolone for 7-14 days⁸¹-⁸⁴.
COPD
= chronic inflammation and structural changes of respiratory airway resulting from repeated injury and repair due to inhaled cigarette smoke /noxious particles
= characterized by increase Neutrophils, macrophages and CD8 lymphocytes (different cell mediations as compared to BA)

Patho=mucus hypersecretion + expiratory airflow limitation, small airway collapse causing air trapping and hyperventilation, gas exchange abnormalities, progressive pulmonary hypertension

Table 3-1 Risk Factors for COPD

<table>
<thead>
<tr>
<th>Genes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Exposure to particles</td>
</tr>
<tr>
<td>Tobacco smoke</td>
</tr>
<tr>
<td>Organic and inorganic occupational dusts</td>
</tr>
<tr>
<td>Indoor air pollution from heating and cooking with biomass in poorly ventilated dwellings</td>
</tr>
<tr>
<td>Outdoor air pollution</td>
</tr>
<tr>
<td>Lung growth and development</td>
</tr>
<tr>
<td>Oxidative stress</td>
</tr>
<tr>
<td>Gender</td>
</tr>
<tr>
<td>Age</td>
</tr>
<tr>
<td>Respiratory infections</td>
</tr>
<tr>
<td>Socioeconomic status</td>
</tr>
</tbody>
</table>

Figure 3-2: Vicious Circle Hypothesis

Symptoms
- Dyspnoea – progressive SOB, which later may interfere with daily activities
Airflow limitation = small airway disease (bronchiolitis) + lung parenchymal destruction (emphysema)
- measured by spirometry:

<table>
<thead>
<tr>
<th>COPD stage</th>
<th>Severity</th>
<th>Post-bronchodilator spirometric values</th>
<th>Symptoms that may be present</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Mild</td>
<td>FEV₁/FVC &lt; 0.70 FEV₁ ≥ 80% predicted</td>
<td>Chronic cough and sputum production may be present. At this stage, the individual is usually unaware that his or her lung function is abnormal.</td>
</tr>
<tr>
<td>II</td>
<td>Moderate</td>
<td>FEV₁/FVC &lt; 0.70 50% ≤ FEV₁ &lt; 80% predicted</td>
<td>Dyspnoea typically on exertion, cough and sputum production sometimes also present. This is the stage at which patients usually seek medical attention because of chronic respiratory symptoms or an exacerbation of COPD.</td>
</tr>
<tr>
<td>III</td>
<td>Severe</td>
<td>FEV₁/FVC &lt; 0.70 30% ≤ FEV₁ &lt; 50% predicted</td>
<td>Greater dyspnoea, reduced exercise capacity, fatigue, and repeated exacerbations that almost always have an impact on the patient's quality of life.</td>
</tr>
<tr>
<td>IV</td>
<td>Very severe</td>
<td>FEV₁/FVC &lt; 0.70 FEV₁ &lt; 30% predicted or FEV₁ &lt; 50% predicted plus chronic respiratory failure</td>
<td>Respiratory failure may lead to cor pulmonale with signs which include elevation of the jugular venous pressure and pitting ankle oedema. At this stage, quality of life is markedly impaired and exacerbations may be life-threatening.</td>
</tr>
</tbody>
</table>

FEV₁: forced expiratory volume in one second; FVC: forced vital capacity; respiratory failure: arterial partial pressure of oxygen (PaO₂) less than 8.0 kPa (60 mmHg) with or without arterial partial pressure of CO₂ (PaCO₂) greater than 6.7 kPa (50 mmHg) while breathing air at sea level.
Assessment of symptoms
1) Dyspnoea – progressive, persistent, gradually interferes with daily activities
2) Cough – initially intermittent, then daily with chronic sputum production
3) Wheezing and chest tightness

History
1) Smoking
2) Occupational and environmental exposure to lung irritants
3) Family hx

Physical Examination
1) airflow limitation – barrel chest, loss of cardiac/liver dullness, prolonged expiration, reduced breath sounds, ronchi

Ix
1) Spirometry – no improvement post bronchodilator
2) CXR – hyperinflation, flattened diaphragm, increased lung volume
3) ABG – if FEV1 < 40% or Spo2 <92%, clinical signs of respiratory failure/cor pulmonale → may require continuous ventilation
4) FBC
5) ECG – pulmonary hypertension
Pharmacotherapy

Short Acting
1) Short Acting B2 Agonists – MDI salbutamol 200mcg, Fenoterol 200mcg, Terbutaline 500mcg PRN
2) Short acting Anti Cholinergics – MDI Ipratropium Bromide 40mcg QID

Long Acting
1) LABA – MDI Salmeterol 50mcg BD, Formoterol 9mcg BD
2) LAAC – Tiotropium 18mcg OD

Inhaled Corticosteroids (ICS)
MDI Budesonide 400mcg BD
MDI Fluticasone 500mcg BD

Combinations
MDI Combivent = Salbutamol + Ipratropium Bromide (SABA + SAAC)
MDI Seretide = fluticasone propionate/salmeterol (ICS + LABA)

Methyloxanthines – Theophylline 125-300mg BD

Corticosteroids
IV hydrocortisone 100mg QID 1/7
T Prednisolone 30mg OD 5/7

LTOT (long term Oxygen therapy)
Indications
1) PaO2 <55mmHg or Sa O2 <88%, with/without hypercapnia
2) Pa O2 55-60mmHg, SaO2 89% + pulm hpt, peripheral edema (CHF), polycythemia

Surgical intervention
1) Lung volume reduction surgery
2) Bullectomy
3) Lung transplantation
**AECOPD**

Exacerbation – sustained worsening of baseline dyspnoea, cough (sputum) that is beyond normal day to day variations

**Causes** – smoking, pneumonia, URTI, environmental factors, non compliance to medication

**Sx** – dyspnoea, cough and production of sputum, confusion, lethargy

**Physical exam**
1) Vital signs – T, RR, PR, BP
2) Poor prognosis – confusion, reduced conscious level, cachexia, respirator distress, cyanosis
3) Co morbids – CVS, DM, lung ca

**Ix**
1) ABG
2) Sputum C&S
3) CXR
4) ECG
5) FBC, LFT, RP

---

**Hospital Management**
- Controlled supplemental oxygen therapy to maintain PaO₂ > 8 kPa or SpO₂ > 90% without worsening hypercapnia or precipitating acidosis
- Inhaled short-acting bronchodilators from pMDI via a spacer device or nebuliser
- Consider intravenous aminophylline if inadequate response to inhaled short-acting bronchodilators
- Systemic corticosteroids for 7-14 days
- Antibiotics (if appropriate)
- Monitor fluid balance and nutrition
- Consider subcutaneous heparin
- Closely monitor condition of the patient
- Consider invasive or non-invasive ventilation
Tuberculosis

1) Extra pulmonary TB – TB lymphadenitis, pleural effusion, genitourinary, bones/joints, military TB, meningitis TB
2) Pulmonary TB – most common manifestation

- smear positive = AFB > 2 (+) or AFB x1(+) with CXR picture+ or AFB x1(+) with Mycobact C&S (+)
- smear negative = AFB x3 (-) but CXR picture+

PTB Symptoms and signs
- Cough persisting > 2 weeks
- Productive cough with blood streak
- LOA + significant LOW
- Fever
- Dyspnoea, night sweats, chest pain, hoarse voice

Investigations
- CXR – lesions in apical and posterior segments upper lobe, cavitation,
- ESR raised, FBC - monocytosis
- Mantoux Test – using 2Tuberculin units 0.1ml (POSITIVE = induration >10mm)
- Sputum AFB(x3) and Mycobacterium C&S

Terms:
1) New case = no prior tx for tb
2) Relapse/Reactivation = previously declared cured after completed tx
3) Chronic= remain smear + despite tx

Treatment
1st Line HRZSE = Isoniazid (H), Rifampicin (R), Pyrazinamide (Z), Streptomycin (S), Ethambutol (E)

Regimens:

<table>
<thead>
<tr>
<th>Category I</th>
<th>New Case</th>
</tr>
</thead>
<tbody>
<tr>
<td>(i) Intensive phase: 2H+RZ or 2EH+RZ or 2HRZ (2 months of daily doses)</td>
<td></td>
</tr>
<tr>
<td>(ii) Continuation phase: 4H+R2 or 4S+H+R2 or 4H+R or 4H+R2 or 4S+H+R2 (Duration may be extended for severe forms of extra pulmonary tuberculosis and immune compromised patients).</td>
<td></td>
</tr>
</tbody>
</table>

* The number preceding the treatment regimen refers to the treatment duration in months.

** The subscript below the drug symbol refers to the frequency of doses per week.

<table>
<thead>
<tr>
<th>Category II</th>
<th>Relapse, Treatment failure, Treatment after Interruption</th>
</tr>
</thead>
<tbody>
<tr>
<td>(i) Send Mycobacterium tuberculosis culture and sensitivity (MTB C&amp;S) (Rapid culture method if available)</td>
<td></td>
</tr>
<tr>
<td>(ii) Do not initiate standard therapy</td>
<td></td>
</tr>
<tr>
<td>(iii) Refer to chest physician or physician in charge of chest clinic</td>
<td></td>
</tr>
<tr>
<td>(iv) Subsequent drug regimen based on sensitivity results and clinical response.</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Category III</th>
<th>Chronic Case</th>
</tr>
</thead>
<tbody>
<tr>
<td>(i) Refer to chest physician or physician in charge of chest clinic.</td>
<td></td>
</tr>
</tbody>
</table>

6.2.4 Drug interactions

6.2.4.1 Isoniazid
Isoniazid tends to raise plasma concentrations of anti-epileptic drugs such as phenytoin and carbamazepine by inhibiting their metabolism in the liver. The absorption of isoniazid is impaired by aluminium hydroxide.

6.2.4.2 Rifampicin
Rifampicin induces liver enzymes, and may increase the dosage requirements of drugs metabolised in the liver. These include protease inhibitors, cyclosporin, corticosteroids, oral contraceptive pill, oral hypoglycaemic agents, oral anticoagulants, phenytoin, cimetidine, theophylline and digitals glycosides.

Biliary excretion of radiocontrast media and Sulphobromophthalein sodium may be reduced and microbiological assays for folic acid and vitamin B12 disturbed.

6.2.4.3 Streptomycin
Other ototoxic or nephrotoxic drugs should not be administered to patients receiving streptomycin. These include other aminoglycoside antibiotics: amphotericin B, cephalosporin, ethacrynic acid, cyclosporin, cisplatin, frusemide and vancomycin. Streptomycin may potentiate the effect of neuromuscular blocking agents administered during anaesthesia.
4.2.4 Flow chart for recommended 24 weeks (w) / 6 months (m) treatment regimen (adult)

<table>
<thead>
<tr>
<th>Visit</th>
<th>Duration</th>
<th>Regimen</th>
<th>Investigation</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>0 w (0 m)</td>
<td>2SHRZ / 2EHRZ</td>
<td>Baseline investigation FBC, RFT, LFT, RBS, HIV, Sputum AFB D/S, culture</td>
</tr>
<tr>
<td>2.</td>
<td>8 w (2 m)</td>
<td>4SHR R</td>
<td>sputum AFB D/S sputum MTB C&amp;S if smear positive CXR</td>
</tr>
<tr>
<td>3.</td>
<td>8 w (2 m)</td>
<td>Continue Rx</td>
<td>Continue Rx sputum AFB D/S CXR</td>
</tr>
<tr>
<td>4.</td>
<td>8 w (2 m)</td>
<td>Completion of Rx 24 w (6 m)</td>
<td>sputum AFB D/S CXR</td>
</tr>
<tr>
<td>5.</td>
<td>24 w (6 m)</td>
<td></td>
<td>Follow up sputum AFB D/S CXR</td>
</tr>
</tbody>
</table>

\[ E = Ethambutol \quad FBC = Full\ blood\ count \quad RBS = \text{random\ blood\ sugar} \]
\[ H = Isoniazid \quad LFT = \text{Liver\ function\ test} \quad HIV = \text{anti-HIV\ antibody} \]
\[ R = \text{Rifampicin} \quad RFT = \text{Renal\ function\ test} \quad \text{MTB = Mycobacterium tuberculosis} \]
\[ S = \text{Streptomycin} \quad D/S = \text{Direct\ smear} \quad \text{C&S = culture\ and\ sensitivity\ test} \]
\[ Z = \text{Pyrazinamide} \quad Rx = \text{Treatment} \]
\[ W = \text{week} \quad M = \text{month} \]

Note: (•) Recommended to be done where facilities are available

- DOTS (Directly Observed Treatment) → Observed taking of medication to make sure pt is compliant
- TB wallet – All new cases that are referred to IPR must have a TB wallet, needs to be filled by HO
Kidney Failure
Compiled by Dr Ong Lip Kent

AKI is a rapid loss of kidney function

1) Prerenal
- low blood volume
- low blood pressure
- heart failure
- renal artery stenosis

2) Intrinsic
- Glomerulonephritis
- acute tubular necrosis (ATN)
- acute interstitial nephritis (AIN)

3) Postrenal (surgical case)
- benign prostatic hyperplasia
- kidney stones
- obstructed urinary catheter
- bladder stone
- bladder, ureteral or renal malignancy.

Classic laboratory findings in AKI

<table>
<thead>
<tr>
<th>Type</th>
<th>UO (ml/kg/hr)</th>
<th>US (mg/dl)</th>
<th>Fe (mg/dl)</th>
<th>BUN/Cr (mg/dl)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prerenal</td>
<td>&gt;500</td>
<td>&lt;10</td>
<td>&lt;1%</td>
<td>&gt;20</td>
</tr>
<tr>
<td>Intrinsic</td>
<td>&lt;350</td>
<td>&gt;20</td>
<td>&gt;2%</td>
<td>&lt;15</td>
</tr>
<tr>
<td>Postrenal</td>
<td>&lt;350</td>
<td>&gt;40</td>
<td>&gt;4%</td>
<td>&lt;15</td>
</tr>
</tbody>
</table>

RIFLE

- Risk: Increased Cr x 1.5 or GFR decreases >25%
- Injury: Increased Cr x 2 or GFR decreases >50%
- Failure: Increased Cr x 3 or GFR decreases >75% or Cr > 4 mg/dl (with acute rise of ≥ 0.5 mg/dl)
- Loss: Persistent ARF (complete loss of renal function for > 4 weeks)
- ESRD: End Stage Renal Disease

AKIN

- Stage 1: Increased Cr x 1.5 or Cr ≥ 2 mg/dl (≥ 0.3 mg/dl)
- UO < 0.5 ml/kg/hr x 6 hr

- Stage 2: Increased Cr x 2
- UO < 0.5 ml/kg/hr x 12 hr

- Stage 3: Increased Cr x 3 or Cr ≥ 4 mg/dl (with acute rise of ≥ 0.5 mg/dl)
- UO < 0.3 ml/kg/hr x 24 hr
- Anuria x 12 hr

Patients who receive renal replacement therapy (RRT) are considered to have met the criteria for stage 3 irrespective of the stage that they are in at the time of commencement of RRT.
CKD is an irreversible loss of renal function for at least three months and poses a major public health problem.

Who should be screened?
 Patients with diabetes mellitus and/or hypertension should be screened at least yearly for chronic kidney disease  o Age >65 years old
 o Family history of stage 5 CKD or hereditary kidney disease
 o Structural renal tract disease, renal calculi or prostatic hypertrophy
 o Opportunistic (incidental) detection of haematuria or proteinuria
 o Chronic use of non-steroidal anti-inflammatory drugs (NSAIDs) or other nephrotoxic drugs
 o Cardiovascular disease (CVD)
 o Multisystem diseases with potential kidney involvement such as systemic lupus erythematosus

Screening method
 1) Proteinuria

Factors Increases protein excretion Decreases protein excretion
• Strenuous exercise
• Poorly controlled DM
• Heart failure
• UTI
• Acute febrile illness
• Uncontrolled hypertension
• Haematuria
• Menstruation
• Pregnancy

2) Hematuria

3) Renal function (RP)

Equations for estimation of renal function (suggest to use online calculators/apps)

i. MDRD eGFR = 175 x serum Cr^{-1.154} \times \text{age}^{0.203} \times \text{constant}  
   [\text{constant} = 1.212 \text{ [if black]} \text{ or } 0.742 \text{ [if female]} ]

* where GFR is expressed as ml/min/1.73m2 of body surface area and sCr is expressed in mg/dl

ii. CKD-epi eGFR (Chronic Kidney Disease Epidemiology Collaboration)
   - complexed formula calculation, suggest to use online app

iii. Cockcroft-Gault Creatinine Clearance
   CrCl (ml/min) = \frac{140 - \text{age (yrs)}}{\text{body weight (kg)}} \times \text{sCr (μmol/l)} \times \text{constant}  
   [\text{constant} = 1.23 \text{ in male or 1.04 in female}]

4) Renal tract US (US KUB)
identifies obstructive uropathy, renal size and symmetry, renal scarring and polycystic disease.

Indications for renal ultrasound in patients with CKD:
• a rapid deterioration of renal function (eGFR >5 ml/min/1.73m2 within one year or 10 ml/min/1.73m2 within five years)
• visible or persistent non-visible haematuria
• symptoms or history of urinary tract obstruction
• a family history of polycystic kidney disease and age over 20 years
• stage 4 or 5 CKD
• when a renal biopsy is required

CKD classification:

<table>
<thead>
<tr>
<th>Stages of CKD</th>
<th>Stage GFR (ml/min/1.73m2)</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>≥90</td>
<td>Normal or increased GFR, with other evidence of kidney damage</td>
</tr>
<tr>
<td>2</td>
<td>60 – 89</td>
<td>Slight decrease in GFR, with other evidence of kidney damage</td>
</tr>
<tr>
<td>3A</td>
<td>45 - 59</td>
<td>Moderate decrease in GFR, with or without other evidence of kidney damage</td>
</tr>
<tr>
<td>3B</td>
<td>30 - 44</td>
<td>Severe decrease in GFR, with or without other evidence of kidney damage</td>
</tr>
<tr>
<td>4</td>
<td>15 – 29</td>
<td>Established renal failure</td>
</tr>
<tr>
<td>5</td>
<td>&lt;15</td>
<td>Established renal failure</td>
</tr>
</tbody>
</table>
A patient with chronic kidney disease (CKD) and any of the following criteria should be referred to a nephrologist/physician:

- Heavy proteinuria (urine protein ≥1 g/day or urine protein: creatinine ratio (uPCR) ≥0.1 g/mmol) unless known to be due to diabetes and optimally treated
- Haematuria with proteinuria (urine protein ≥0.5 g/day or uPCR ≥0.05 g/mmol)
- Rapidly declining renal function (loss of glomerular filtration rate/GFR >5 ml/min/1.73m² in one year or >10 ml/min/1.73m² within five years)
- Resistant hypertension (failure to achieve target blood pressure despite three antihypertensive agents including a diuretic)
- Suspected renal artery stenosis
- Suspected glomerular disease
- Suspected genetic causes of CKD
- Pregnant or when pregnancy is planned
- Estimated GFR <30 ml/min or serum creatinine >200 μmol/L
- Unclear cause of CKD.

**Uremic symptoms:**

- Neural and muscular
  - Fatigue
  - Peripheral neuropathy
  - Decreased mental acuity
  - Seizures
  - Anorexia
  - Nausea
  - Decreased taste and smell
  - Cramps
  - Sleep disturbance
  - Coma

- Endocrine and metabolic
  - Amenorrhea
  - Sexual dysfunction
  - Reduced body temperature
  - Altered levels of amino acids
  - Bone disease by hyperphosphatemia, hyperparathyroidism, and vitamin D deficiency
  - Reduced basal metabolic rate
  - Insulin resistance
  - Increased muscle protein catabolism

- Other
  - Itching
  - Hiccups
  - Granulocyte and lymphocyte dysfunction
  - Platelet dysfunction

---

### The Uremic Syndrome

**Symptoms**

- Fatigue, metallic taste, drowsiness/insomnia, anorexia, nausea/vomiting, delirium/seizures

**Signs**

- General appearance - thin (anorexia), irritability
- Skin - grey skin, bruises, pruritus
- Vitals – dypnea, hypertension
- Face – pallor, uremic frost, uremia smell
- Neck – elevated JVP
- Chest – crackles, pleural effusions
- Heart - CHF, pericardial friction rub
- MSK - Bone pain, muscle weakness, paresthesias, twitching, cramps
- Heme: uremic platelets, anemia

(c) 2007, Lesley A. Stevens, M.D.
**Treatment:**

1) **Medications**
- Avoidance of substances that are toxic to the kidneys, called nephrotoxins.
  - NSAIDs such as ibuprofen,
  - Iodinated contrasts such as those used for CT scans,
  - Many antibiotics such as gentamicin

2) Serial serum creatinine measurements
3) Monitoring of urine output - insertion of a urinary catheter
4) Diuretics such as frusemide (provided patient still has urine output)
5) Renal Replacement Therapy (RRT)

Types:
- Peritoneal dialysis
- Hemodialysis
- CVVH (Continuous Venovenous Hemofiltration)
- SLED

**Indication for HD:**
- Acidosis (bicarb <10, pH <7.2)
- Electrolyte – hyperkalemia (K >6 despite given lytic cocktail x3, with ECG changes)
- Intoxication
- Overload of fluid (pulmonary edema)
- Uremia (Urea >30, or presence of any uremic symptoms)

**Types of catheter for HD:**
- Femoral catheter – change every 2 weekly, NOT to be discharge with this catheter
- Intrajugular catheter – change monthly
- Permanent catheter – change yearly
- AVF (Arterio-venous fistula) – done by vascular surgeon **spare the non dominant hand upon referral for patient with ESRF**

**Patient with ESRF need to be counselled for RRT**
- Inform about the kidney problem – urine output will be poor and eventually anuria
- The need of RRT to prevent complication
- Social support – dialysis center, transport, financial problem
Appendix

Formulae

**HHS**
- Effective serum Osmolality = 2(Na + K) + RBS + Urea
- Total Osmolality = 2(Na) + RBS + urea

**Electrolytes**
- Corrected serum Na = 0.3 (RBS-5.5) + Na
- Corrected serum Ca = 0.025 (40-Albumin)

**Urine output** (/kg/hr) = total output ÷ IBW ÷ Hours

**Cockcroft-Gault Formula**

\[
\text{Creatinine Clearance} = \frac{[140 - \text{Age (years)}] \times \text{Weight (kg)}}{0.812 \times \text{s. creatinine (mcmol/L)}}
\]

**PEF**

\[
\text{PEF (max)} - \text{PEF (min)} \times 100 = \frac{\text{___________ % PEF (max)}}{\text{PEF}}
\]

**Smoking pack years**

= twenty cigarettes smoked everyday for one year
= \frac{\text{Cigarettes per day} \times \text{years}}{20}

Investigations to send

**CSF**
1) CSF FEME, Biochem, Cytology
2) C&S
3) India Ink (yeast)
4) Latex agglutination
5) Cryptoccal Antigen
6) AFB
7) Mycobacterium C&S
8) Viral Study
+ Random Blood Glucose

**Peritoneal Fluid**
1) body fluid FEME
2) Biochem
3) C&S
4) SAAG (Serum albumin ascites gradient
5) Cytology

**Pleural Fluid**
1) Cytology
2) Biochemistry
3) FEME
4) C&S
5) AFB
6) Mycobacterium C&S
7) Fungal C&S
### Table 1. CURB-65 Scoring

<table>
<thead>
<tr>
<th>Clinical Feature</th>
<th>Points</th>
</tr>
</thead>
<tbody>
<tr>
<td>Confusion (defined as a Mental Test Score of ≤ 8, or disorientation in person, place, or time)</td>
<td>1</td>
</tr>
<tr>
<td>Uremia blood urea: &gt;7 mmol/L (~19 mg/dL)</td>
<td>1</td>
</tr>
<tr>
<td>Respiratory rate: ≥ 30 breaths/minute</td>
<td>1</td>
</tr>
<tr>
<td>Blood pressure: systolic: &lt; 90 mm Hg or diastolic: ≤ 60 mm Hg</td>
<td>1</td>
</tr>
<tr>
<td>Age ≥ 65 years</td>
<td>1</td>
</tr>
<tr>
<td>Total points</td>
<td></td>
</tr>
</tbody>
</table>

### Table 2. Treatment Options Based on CURB-65 Score

<table>
<thead>
<tr>
<th>Score</th>
<th>Group</th>
<th>Treatment Options</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 or 1</td>
<td>Group 1; mortality low (1.5%)</td>
<td>Low risk; consider home treatment</td>
</tr>
<tr>
<td>2</td>
<td>Group 2; mortality intermediate (9.2%)</td>
<td>Consider hospital-supervised treatment (either short-stay inpatient or hospital-supervised outpatient)</td>
</tr>
<tr>
<td>≥3</td>
<td>Group 3; mortality high (22%)</td>
<td>Manage in hospital as severe pneumonia; consider admission to intensive care unit, especially with CURB-65 score of 4 or 5</td>
</tr>
</tbody>
</table>

### Pleural Tap:

**Ix:** Cytology, Biochemistry, C&S (AFB, Mycobacterium, Fungal)

**Light's Criteria:** Exudative Effusions will have at least one of the following:

- Pleural fluid protein / Serum protein >0.5
- Pleural fluid LDH / Serum LDH >0.6
- Pleural fluid LDH > 2/3 * Serum LDH Upper Limit of Normal
Cardiology

TIMI Risk score
- used to determine risk at 14 days of: all-cause mortality, new or recurrent MI, or severe recurrent ischemia requiring urgent revascularization.

Time: Age >65
Incidence of severe angina >2x/24hrs
Medication: used ASA in past 1/52
Increased Cardiac Markers
Risk factors >3
IHD with CAD (stenosis >50%)
ST changes >0.5mm

CHAD score - calculates stroke risk for AF

<table>
<thead>
<tr>
<th>CHADS2 score</th>
<th>CHA2DS2 -VASc</th>
</tr>
</thead>
<tbody>
<tr>
<td>+1 Congestive Heart failure hx?</td>
<td>+1 Congestive Heart failure hx?</td>
</tr>
<tr>
<td>+1 Hypertension</td>
<td>+1 Hypertension</td>
</tr>
<tr>
<td>+1 Age &gt;75</td>
<td>* Age 65yo +1</td>
</tr>
<tr>
<td>+1 DM hx</td>
<td>+1 DM hx</td>
</tr>
<tr>
<td>+2 Stroke sx or TIA</td>
<td>+2 Stroke sx or TIA or thromboembolism hx</td>
</tr>
<tr>
<td>+1 Vascular disease His (MI, PAD, aortic plaque)</td>
<td>+1 Female</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>CHADS2 criteria</th>
<th>Points</th>
</tr>
</thead>
<tbody>
<tr>
<td>Previous stroke or TIA</td>
<td>2</td>
</tr>
<tr>
<td>Age ≥ 75 years</td>
<td>1</td>
</tr>
<tr>
<td>Hypertension</td>
<td>1</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>1</td>
</tr>
<tr>
<td>Heart failure</td>
<td>1</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Stroke risk score</th>
<th>Recommended therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>High 2–6</td>
<td>Warfarin (INR 2–3)</td>
</tr>
<tr>
<td>Moderate 1</td>
<td>Warfarin or aspirin</td>
</tr>
<tr>
<td>Low 0</td>
<td>Aspirin 100–300 mg daily</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Risk score</th>
<th>Death or MI</th>
<th>Death or MI or urgent revasc</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>1</td>
<td>5</td>
</tr>
<tr>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>2</td>
<td>4</td>
<td>7</td>
</tr>
<tr>
<td>3</td>
<td>5</td>
<td>12</td>
</tr>
<tr>
<td>4</td>
<td>6</td>
<td>19</td>
</tr>
<tr>
<td>5</td>
<td>7</td>
<td>26</td>
</tr>
<tr>
<td>6</td>
<td>8</td>
<td>41</td>
</tr>
</tbody>
</table>

Table 2. Classifications Of Heart Failure.

American Heart Association Classification

<table>
<thead>
<tr>
<th>Class</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stage A</td>
<td>Patients are at high risk for heart failure but have not developed structural heart disease and have no symptoms.</td>
</tr>
<tr>
<td>Stage B</td>
<td>Patients have developed structural heart disease but have not (yet) developed symptoms.</td>
</tr>
<tr>
<td>Stage C</td>
<td>Patients with past or current heart failure symptoms in association with structural damage to the heart.</td>
</tr>
<tr>
<td>Stage D</td>
<td>Patients with end-stage or terminal heart failure requiring specialized treatment strategies.</td>
</tr>
</tbody>
</table>

New York Heart Association Classification

<table>
<thead>
<tr>
<th>Class</th>
<th>Functional status</th>
<th>Symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>No limitation</td>
<td>Asymptomatic during usual daily activities</td>
</tr>
<tr>
<td>II</td>
<td>Slight limitation</td>
<td>Mild symptoms (dyspnea, fatigue, or chest pain) with ordinary daily activities</td>
</tr>
<tr>
<td>III</td>
<td>Moderate limitation</td>
<td>Symptoms noted with minimal activity</td>
</tr>
<tr>
<td>IV</td>
<td>Severe limitation</td>
<td>Symptoms at rest</td>
</tr>
</tbody>
</table>

Killip Classification

<table>
<thead>
<tr>
<th>Class</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Killip class I</td>
<td>No evidence of pulmonary congestion or shock.</td>
</tr>
<tr>
<td>Killip class II</td>
<td>Mild pulmonary congestion (patients had rales up to 50% of each lung field) or an isolated S3 gallop.</td>
</tr>
<tr>
<td>Killip class III</td>
<td>Pulmonary edema (rales more than 50% up).</td>
</tr>
<tr>
<td>Killip class IV</td>
<td>Hypotension and evidence of shock.</td>
</tr>
</tbody>
</table>
ECG Localization of infarct

Anterior - Small V3-V4
- Extensive V2-V5
Anteroseptal V1-V3
Anterolateral V4-V6, I, AVL
Lateral I, AVL, V5-V6
Inferior II, III, AVF
Posterior V1-V2

The Glasgow Coma Scale

Vocal 5
- What's your name?
- my name is Gerard
- ALERT, ORIENTED
- Who am I??
- CONFUSED, yet COHERENT
- Fire rojak shoes
- INCOHERENT, JUMBLED
- "U-coo-uh-uh-uh-bike"
- INCOMPREHENSIBLE SOUNDS
- .......... NO SOUND

Motor 6
- Raise your LH
- Obey Commands
- Painful stimuli (rub chest)
- Hand moves towards stimuli
- Hand withdraw from pain
- Painful stimuli
- A. Abnormal flexion (decreases rigidity)
- B. Extension present (decreases rigidity)
- no movement
FORREST CLASSIFICATION

IA Spurting Blood
IB Oozing Blood

II A Exposed Vessel
II B Adherent Clot
II C Hematin on ulcer base

Acute hemorrhage
• Forrest I a (Spurting hemorrhage)
• Forrest I b (Oozing hemorrhage)

Signs of recent hemorrhage
• Forrest II a (Visible vessel)
• Forrest II b (Adherent clot)
• Forrest II c (Hematin on ulcer base)

Lesions without active bleeding
• Forrest III (Lesions without signs of recent hemorrhage)

Gerard Loh
THE CXR

Positions
1) PA – xray shot from back
2) AP – xray shot from front to back (usually supine), heart appears more enlarged

Presenting a radiograph
1) This is a CXR of ___, a ___ years old, gentleman/lady.
2) Taken in PA/AP/supine/erect/sitting, taken with good inspiration and penetration
3) Comment on the components:
   - normal CXR
     i) Trachea – central, not deviated
     ii) Mediastinum – not displaced, contours and hilar normal
     iii) Lungs – clear (black), no pneumothorax
     iv) Diaphragm – no free air under diaphragm
     v) Bones and soft tissue - normal

ABCDEF of Left ventricular failure

- Alveolar oedema (bat’s wings)
- Kerley B lines (interstitial oedema)
- Cardiomegaly
- Dilated prominent upper lobe vessels
- Effusion (pleura)
Chest Tube safe triangle

Identify the "safe triangle" for drain placement, as demarcated by the outer border of the pectoralis major, the anterior border of the latissimus dorsi and a horizontal line that meets the nipple anteriorly. In general, the drain should be sited in the 4th or 5th intercostal space within this triangle.

Pleural tap

Patient sitting upright and leaning on table

Fluid pushes on left lung

Pleural space filled with excess fluid

Fluid collects in bag or syringe
## Antihypertensives

<table>
<thead>
<tr>
<th>ARBs</th>
<th>Starting Dose</th>
<th>Maximum Daily Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gandesartan</td>
<td>8 mg od</td>
<td>16 mg od</td>
</tr>
<tr>
<td>Irbesartan</td>
<td>150 mg od</td>
<td>300 mg od</td>
</tr>
<tr>
<td>Losartan</td>
<td>50 mg od</td>
<td>100 mg od</td>
</tr>
<tr>
<td>Telmisartan</td>
<td>20 mg od</td>
<td>80 mg od</td>
</tr>
<tr>
<td>Valsartan</td>
<td>80 mg od</td>
<td>160 mg od</td>
</tr>
<tr>
<td>Olmesartan</td>
<td>20 mg od</td>
<td>40 mg od</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>ACEIs</th>
<th>Starting Daily Dose</th>
<th>Maximum Daily Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Captopril</td>
<td>25 mg bd</td>
<td>50 mg tds</td>
</tr>
<tr>
<td>Enalapril</td>
<td>2.5 mg od</td>
<td>20 mg bd</td>
</tr>
<tr>
<td>Fosinopril</td>
<td>10 mg od</td>
<td>40 mg od</td>
</tr>
<tr>
<td>Lisinopril</td>
<td>5 mg od</td>
<td>80 mg od</td>
</tr>
<tr>
<td>Perindopril</td>
<td>2 mg od</td>
<td>8 mg od</td>
</tr>
<tr>
<td>Quinapril</td>
<td>2.5 mg od</td>
<td>40 mg bd</td>
</tr>
<tr>
<td>Ramipril</td>
<td>2.5 mg od</td>
<td>10 mg od</td>
</tr>
<tr>
<td>Imidapril</td>
<td>2.5 mg od</td>
<td>10 mg od</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>β-blockers</th>
<th>Starting Dose</th>
<th>Maximum Daily Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acetbutolol</td>
<td>200 mg bd</td>
<td>400 mg bd</td>
</tr>
<tr>
<td>Atenolol</td>
<td>50 mg od</td>
<td>100 mg od</td>
</tr>
<tr>
<td>Betaxolol</td>
<td>10 mg od</td>
<td>40 mg od</td>
</tr>
<tr>
<td>Bisoprolol</td>
<td>5 mg od</td>
<td>10 mg od</td>
</tr>
<tr>
<td>Metoprolol</td>
<td>50 mg bd</td>
<td>200 mg bd</td>
</tr>
<tr>
<td>Propranolol</td>
<td>40 mg bd</td>
<td>320 mg bd</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>CGBs</th>
<th>Starting Dose</th>
<th>Maximum Daily Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amlodipine</td>
<td>5 mg od</td>
<td>10 mg od</td>
</tr>
<tr>
<td>Diltiazem</td>
<td>30 mg tds</td>
<td>60 mg tds</td>
</tr>
<tr>
<td>Diltiazem SR</td>
<td>90 mg bd</td>
<td>90 mg bd</td>
</tr>
<tr>
<td>Diltiazem R</td>
<td>100-200 mg od</td>
<td>100-200 mg od</td>
</tr>
<tr>
<td>Felodipine</td>
<td>2.5 mg od</td>
<td>10 mg od</td>
</tr>
<tr>
<td>Isradipine</td>
<td>1.5 mg bd</td>
<td>2.5 mg bd</td>
</tr>
<tr>
<td>Lacidipine</td>
<td>2 mg od</td>
<td>6 mg od</td>
</tr>
<tr>
<td>Lercanidipine</td>
<td>10 mg od</td>
<td>20 mg od</td>
</tr>
<tr>
<td>Nicardipine</td>
<td>10 mg tds</td>
<td>20 mg tds</td>
</tr>
<tr>
<td>Nifedipine</td>
<td>10 mg tds</td>
<td>30 mg tds</td>
</tr>
<tr>
<td>Nifedipine SR</td>
<td>30 mg od</td>
<td>120 mg od</td>
</tr>
<tr>
<td>Verapamil</td>
<td>80 mg bd</td>
<td>240 mg tds</td>
</tr>
<tr>
<td>Verapamil CR</td>
<td>200 mg od</td>
<td>200 mg bd</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Diuretics</th>
<th>Starting Dose</th>
<th>Maximum Daily Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chlorothiazide</td>
<td>250 mg od</td>
<td>500 mg od</td>
</tr>
<tr>
<td>Hydrochlorothiazide</td>
<td>25 mg od</td>
<td>200 mg od</td>
</tr>
<tr>
<td>Chlorothalidone</td>
<td>50 mg od</td>
<td>200 mg od</td>
</tr>
<tr>
<td>Amlodipine/hydrochlorothiazide 5 mg/50 mg</td>
<td>1 tablet od</td>
<td>4 tablet od</td>
</tr>
<tr>
<td>Indapamide SR</td>
<td>1.5 mg od</td>
<td>1.5 mg od</td>
</tr>
<tr>
<td>Indapamide</td>
<td>2.5 mg od</td>
<td>2.5 mg od</td>
</tr>
<tr>
<td>Triamterene/hydrochlorothiazide 50 mg/25 mg</td>
<td>1 tablet bd</td>
<td>2 tablets bd</td>
</tr>
</tbody>
</table>
### Hypertensive Crisis

#### Table 10. Oral treatment for hypertensive urgencies

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dose</th>
<th>Onset of action (hr)</th>
<th>Duration (hr)</th>
<th>Frequency (prn)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Captopril</td>
<td>25 mg</td>
<td>0.5</td>
<td>6</td>
<td>1 - 2 hrs</td>
</tr>
<tr>
<td>Nifedipine</td>
<td>10 - 20 mg</td>
<td>0.5</td>
<td>3 - 5</td>
<td>1 - 2 hrs</td>
</tr>
<tr>
<td>Labetalol</td>
<td>200 - 400 mg</td>
<td>2.0</td>
<td>6</td>
<td>4 hrs</td>
</tr>
</tbody>
</table>

#### Table 11. Treatment options for hypertensive emergencies

(References are from Malaysia Index of Medical Specialties (MIMS), 108th edition, 2007)

<table>
<thead>
<tr>
<th>Drugs</th>
<th>Dose</th>
<th>Onset of action</th>
<th>Duration</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sodium nitroprusside</td>
<td>0.25 - 10 µg/kg/min IV bolus</td>
<td>seconds</td>
<td>1 - 5 min</td>
<td>Caution in renal failure</td>
</tr>
<tr>
<td></td>
<td>50 mg (or at least 1 minute)</td>
<td>≤ 5 min</td>
<td>3 - 6 hrs</td>
<td>Caution in heart failure</td>
</tr>
<tr>
<td></td>
<td>repeating if necessary at 5 minute intervals to a max of 200 mg then 2 mg/min IV I</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Labetalol*</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Hydralazine*</td>
<td>10 - 20 min</td>
<td>3 - 8 hrs</td>
<td>Caution in acute coronary syndromes and acute pulmonary oedema</td>
</tr>
<tr>
<td></td>
<td>Nitrate</td>
<td>2 - 5 min</td>
<td>3 - 5 min</td>
<td>Preferred in acute coronary syndromes, cerebrovascular accidents and dissecting</td>
</tr>
<tr>
<td></td>
<td>Nicardipine</td>
<td>5 - 10 min</td>
<td>1 - 4 hrs</td>
<td>Caution in acute heart failure and coronary</td>
</tr>
<tr>
<td></td>
<td>Esmolol</td>
<td>3 - 10 min used in perioperators and tachy-arrhythmias</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*References are from Malaysia Index of Medical Specialties (MIMS), 108th edition, 2007*
### OHA

**Biguanides**

<table>
<thead>
<tr>
<th>Formulation</th>
<th>Minimum Dose</th>
<th>Maximum Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Metformin 500mg tablet</td>
<td>Initial dose 500mg OD&lt;br&gt;Usual dose 500mg TDS&lt;br&gt;The side effects can be further reduced by taking it with food</td>
<td>Maximum dose 1000mg BD</td>
</tr>
<tr>
<td>Metformin retard 850 mg tablet (slow release formulation)</td>
<td>Initial dose 850mg OD&lt;br&gt;Usual dose 850mg BD</td>
<td>Maximum dose 1700mg OM / 850 mg ON</td>
</tr>
<tr>
<td>Metformin extended release 500mg tablet</td>
<td>Initial dose 500mg OD</td>
<td>Maximum dose 2000mg OD</td>
</tr>
<tr>
<td>Glibenclamide and metformin fixed dose combination&lt;br&gt;1.25mg / 250mg tablet&lt;br&gt;2.5mg / 500mg tablet&lt;br&gt;5mg / 500mg tablet</td>
<td>Initial dose one 1.25mg / 250mg tablet OD or BD</td>
<td>Maximum dose two 5mg / 250mg tablets BD</td>
</tr>
</tbody>
</table>

#### Secretagogues

<table>
<thead>
<tr>
<th>Formulation</th>
<th>Minimum dose</th>
<th>Maximum dose</th>
<th>Duration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glibenclamide 5mg tablet</td>
<td>2.5mg OM</td>
<td>10mg BD</td>
<td>Long</td>
</tr>
<tr>
<td>Glibenclamide and Metformin Fixed Dose Combination&lt;br&gt;1.25mg / 250mg tablet&lt;br&gt;2.5mg / 500mg tablet&lt;br&gt;5mg / 500mg tablet</td>
<td>Initial dose one 1.25mg / 250mg tablet OD or BD</td>
<td>Maximum dose two 5mg / 500mg tablets BD</td>
<td>Long</td>
</tr>
<tr>
<td>Gliclazide 60mg tablet</td>
<td>40mg OM</td>
<td>160mg BD</td>
<td>Medium</td>
</tr>
<tr>
<td>Gliclazide MR 30mg tablet</td>
<td>30mg OM</td>
<td>120mg OM</td>
<td>Long</td>
</tr>
<tr>
<td>Glipizide 5mg tablet</td>
<td>2.5mg OM</td>
<td>10mg BD</td>
<td>Medium</td>
</tr>
<tr>
<td>Glibimepride 2mg / 3mg tablet</td>
<td>1mg OM</td>
<td>6mg OM</td>
<td>Long</td>
</tr>
</tbody>
</table>

**Dosage of Antidiabetic Agents in Renal Failure**

<table>
<thead>
<tr>
<th>Generic Name</th>
<th>Usual Dose</th>
<th>Dose adjustment in renal failure</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mild (GFR 60 - 90ml/min)</td>
<td>Moderate (GFR 30 - 60ml/min)</td>
</tr>
<tr>
<td>Sulphonylureas</td>
<td>Avoid</td>
<td>Avoid</td>
</tr>
<tr>
<td>Glibenclamide</td>
<td>5mg od – 10mg bd</td>
<td>25-50%</td>
</tr>
<tr>
<td>Gliclazide</td>
<td>80mg od – 160mg bd</td>
<td>50-100%</td>
</tr>
<tr>
<td>Glibimepride</td>
<td>1mg od – 4mg od</td>
<td>100%</td>
</tr>
<tr>
<td>Glipizide</td>
<td>2.5mg od – 5mg od</td>
<td>100%</td>
</tr>
<tr>
<td>Others</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Acarbose</td>
<td>25mg tds – 100mg tds</td>
<td>50-100%</td>
</tr>
<tr>
<td>Exenatide</td>
<td>5mg od – 10 mg od</td>
<td>100%</td>
</tr>
<tr>
<td>Insulin</td>
<td>Variable</td>
<td>100%</td>
</tr>
<tr>
<td>Metformin</td>
<td>500mg bd – 1g bd</td>
<td>50%</td>
</tr>
<tr>
<td>Insulin Preparation</td>
<td>Onset of Action</td>
<td>Peak Action</td>
</tr>
<tr>
<td>---------------------</td>
<td>----------------</td>
<td>-------------</td>
</tr>
<tr>
<td><strong>Fast Acting</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rapid Analogue Aspart (NovoRapid) Lispro (Humalog)</td>
<td>5 – 15 minutes</td>
<td>1 – 2 hours</td>
</tr>
<tr>
<td>Human Regular Actrapid Humulin R</td>
<td>30 – 60 minutes</td>
<td>2 – 4 hours</td>
</tr>
<tr>
<td><strong>Intermediate Acting</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Human NPH Insulin Insulatard Humulin N</td>
<td>1 – 2 hours</td>
<td>4 – 8 hours</td>
</tr>
<tr>
<td><strong>Long Acting</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Basal Long Acting Analogue Glargine Detemir</td>
<td>1 – 2 hours</td>
<td>Flat</td>
</tr>
<tr>
<td><strong>Premixed Insulins</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mixtard 30/70 Humulin 30/70</td>
<td>Bi Phasic onset and peak</td>
<td>10 – 16 hours</td>
</tr>
<tr>
<td>B/Asp 30/70 Humalog mix 25/75</td>
<td></td>
<td></td>
</tr>
<tr>
<td>AGENT</td>
<td>DOSING REGIMEN</td>
<td></td>
</tr>
<tr>
<td>-------------------</td>
<td>--------------------------------------------------------------------------------</td>
<td></td>
</tr>
</tbody>
</table>
| **UFH**           | **Loading Dose:**  
| **UA/NSTEMI**     | - Empirical loading dose: 5000-10000 IU or  
|                   | - Weight adjusted loading dose:  
|                   |   - Not receiving GPIIb/IIIa inhibitors: 70-100 IU/kg  
|                   |   - Receiving GPIIb/IIIa Inhibitors: 50-70 IU/kg  
|                   | **Duration of therapy:** 2-8 days\(^{35-37}\)  
| **During PCI**    | **Further doses if procedure is > 1 hour may be by:**  
|                   | - Empirical weight adjusted doses:  
|                   |   - Not receiving GPIIb/IIIa inhibitors: 50 IU/kg  
|                   |   - Receiving GPIIb/IIIa Inhibitors: 50 IU/kg  
|                   | - Guided by ACT monitoring  
|                   |   - Not receiving GPIIb/IIIa inhibitors maintain ACT: 250-300 secs  
|                   |   - Receiving GPIIb/IIIa Inhibitors maintain ACT: 200 secs  

| **Enoxaparin**    | **Initial** 30 mg IV bolus and then 15 minutes later by:  
| **UA/NSTEMI**     | sc 1.0 mg/kg every 12 hours if age less than 75 years  
|                   | sc 0.75 mg/kg every 12 hours if age 75 years and above  
|                   | **Duration of therapy:** 2-8 days\(^{36-37}\)  
| **During PCI**    | **Depends on prior enoxaparin use:**  
|                   | - No prior use: 0.5-0.75 mg/kg IV bolus  
|                   | - Prior use within 8 hours of PCI: no additional dose  
|                   | - Prior use between 8-12 hours of PCI: 0.3 mg/kg IV. Supplemental UFH may also be given during PCI  

| **Bivalirudin**   | **0.1 mg/kg bolus and 0.25 mg/kg/hour Infusion**  
| **UA/NSTEMI**     | **Depends on prior bivalirudin/UFH use:**  
| **During PCI**    | - Prior treatment with bivalirudin: additional 0.5 mg/kg bolus and increase infusion rate to 1.75 mg/kg/hour  
|                   | - Prior treatment with UFH: wait 30 mins then 0.75 mg/kg bolus and infusion of 1.75 mg/kg/hour  
|                   | - No prior treatment: 0.75 mg/kg bolus and infusion of 1.75 mg/kg/hour  

| **Fondaparinux** | **2.5 mg sc daily for 8 days or duration of hospitalization\(^{38,39}\)**  
| **UA/NSTEMI**    | **If used during PCI, additional 50-60 IU/kg UFH is recommended.**  
| **During PCI**   |
Table 3: Recommended dosages of Nitrates in UA/NSTEMI*

<table>
<thead>
<tr>
<th>Compound</th>
<th>Route</th>
<th>Dosage</th>
<th>Time of Onset</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nitroglycerine, Glyceryl</td>
<td>Sublingual</td>
<td>0.3 - 0.6 mg, can repeat up to 3 times at 5 minute</td>
<td>2 minute</td>
</tr>
<tr>
<td>trinitrate</td>
<td>Intravenous</td>
<td>5 – 200 µg/min*</td>
<td>1 minute</td>
</tr>
<tr>
<td></td>
<td>GTN Spray</td>
<td>0.4 – 0.8 mg per metered dose, no more than 3</td>
<td>2 minute</td>
</tr>
<tr>
<td></td>
<td></td>
<td>sprays at 5 minute intervals</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Transdermal</td>
<td>2.5 – 20 mg over 12 hours on, then 12 hours off</td>
<td>1 – 2 hours</td>
</tr>
<tr>
<td>patch</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Isosorbide dinitrate</td>
<td>Intravenous</td>
<td>2 – 12 mg / hour</td>
<td>1 minute</td>
</tr>
<tr>
<td></td>
<td>Oral</td>
<td>10 – 20 mg, 2 – 3 times daily</td>
<td>30 – 60 minutes</td>
</tr>
<tr>
<td>Isosorbide mononitrate</td>
<td>Oral (LA)</td>
<td>30-60 mg daily, (max 120 mg)</td>
<td></td>
</tr>
</tbody>
</table>

*The dose of IV nitrates should be titrated every 5 – 10 minutes until symptoms and/or ischaemia is relieved and the desired haemodynamic response is obtained.

Table 6: Dosages of Anti-thrombotics in CKD*

<table>
<thead>
<tr>
<th></th>
<th>LOADING DOSE</th>
<th>MAINTENANCE DOSE</th>
</tr>
</thead>
<tbody>
<tr>
<td>UFH</td>
<td>No change</td>
<td>No change</td>
</tr>
<tr>
<td>Enoxaprin</td>
<td>30 mg IV</td>
<td>1 mg/kg sc every 24 hours if CrCl &lt; 30 ml/min</td>
</tr>
<tr>
<td>Fondaparinux</td>
<td>Avoid if Cr Cl &lt; 30 ml/min</td>
<td>Avoid if Cr Cl &lt; 30 ml/min</td>
</tr>
<tr>
<td>Eptifibatide</td>
<td>180 mcg/kg</td>
<td>IV Infusion 1.0 mcg/kg/min if Cr Cl &lt; 50 ml/min</td>
</tr>
<tr>
<td>Tirofiban</td>
<td>IV infusion 0.4 mcg/kg/min for 30 mins</td>
<td>IV infusion 0.05 mcg/kg/min if Cr Cl &lt;30 ml/min</td>
</tr>
</tbody>
</table>

CVA

Regimen for Treatment of Acute Ischaemic Stroke with Intravenous rtPA

1. Infuse 0.5mg/kg maximum of 90 mg over 60 minutes with 10% of the dose given as a bolus dose over 1 minute.
2. Admit the patient to an intensive care unit or a stroke unit for monitoring.
3. Perform neurological assessments every 15 minutes during the infusion of rt-PA and every 30 minutes for the next 6 hours and then every hour until 24 hours from treatment.
4. If the patient develops severe headache, acute hypertension, nausea or vomiting discontinue the infusion if agent is still being administered and obtain a CT scan of brain.
5. Measure blood pressure every 15 minutes for the first 2 hours, every 30 minutes for the next 6 hours and then every hour until 24 hours from treatment.
6. Increase blood pressure measurements if a systolic blood pressure >180mmHg or diastolic blood pressure >105mmHg is recorded. Administer anti-hypertensive medications to maintain blood pressure at or below these levels.
7. Delay placement of nasogastric tubes, indwelling bladder catheters or intra-arterial pressure catheters.
8. Avoid antiplatelet drugs for the first 24 hours after administration of rt-PA.
<table>
<thead>
<tr>
<th><strong>Table II: Drugs Commonly Used in AHF</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Route of Admin</strong></td>
</tr>
<tr>
<td><strong>Diuretics</strong></td>
</tr>
<tr>
<td>Frusemide</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td><strong>Vasodilators</strong></td>
</tr>
<tr>
<td>Nitroglycerin</td>
</tr>
<tr>
<td>Nitroprusside</td>
</tr>
<tr>
<td><strong>Sympathomimetics</strong></td>
</tr>
<tr>
<td>Dobutamine</td>
</tr>
<tr>
<td>Dopamine</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>Noradrenaline</td>
</tr>
<tr>
<td><strong>Phosphodiesterase-3- Inhibitors</strong></td>
</tr>
<tr>
<td>Milrinone</td>
</tr>
</tbody>
</table>
### Table V: Diuretics Used in Heart Failure

<table>
<thead>
<tr>
<th>Route of Administration</th>
<th>Usual Daily Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>LOOP DIURETICS</strong></td>
<td></td>
</tr>
<tr>
<td>Frusemide</td>
<td>IV / Oral</td>
</tr>
<tr>
<td>Bumetanide</td>
<td>IV / Oral</td>
</tr>
<tr>
<td></td>
<td>20 – 80mg</td>
</tr>
<tr>
<td></td>
<td>0.5 – 2mg</td>
</tr>
<tr>
<td><strong>THIAZIDES</strong></td>
<td></td>
</tr>
<tr>
<td>Hydrochlorothiazide</td>
<td>Oral</td>
</tr>
<tr>
<td>Chlorothiazide</td>
<td>Oral</td>
</tr>
<tr>
<td></td>
<td>25 – 50mg</td>
</tr>
<tr>
<td></td>
<td>250 – 500mg</td>
</tr>
<tr>
<td><strong>ALDOSTERONE ANTAGONISTS</strong></td>
<td></td>
</tr>
<tr>
<td>Spironolactone</td>
<td>Oral</td>
</tr>
<tr>
<td>Eplerenone</td>
<td>Oral</td>
</tr>
<tr>
<td></td>
<td>12.5mg – 50mg</td>
</tr>
<tr>
<td></td>
<td>25mg – 50mg</td>
</tr>
</tbody>
</table>

### Table VI: Recommended doses of ACEI used in HF

<table>
<thead>
<tr>
<th>ACEI</th>
<th>Initiating Dose</th>
<th>Target Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Captopril</td>
<td>6.25 mg tid</td>
<td>50 mg tid</td>
</tr>
<tr>
<td>Enalapril</td>
<td>2.5 mg bid</td>
<td>10 mg bid</td>
</tr>
<tr>
<td>Lisinopril</td>
<td>2.5 mg daily</td>
<td>20 mg daily</td>
</tr>
<tr>
<td>Quinapril</td>
<td>2.5-5 mg daily</td>
<td>20 mg bid</td>
</tr>
<tr>
<td>Perindopril</td>
<td>2 mg daily</td>
<td>8 mg daily</td>
</tr>
<tr>
<td>Ramipril</td>
<td>1.25 - 2.5 mg daily</td>
<td>5 mg daily</td>
</tr>
<tr>
<td>Fosinopril</td>
<td>10 mg daily</td>
<td>20 mg daily</td>
</tr>
</tbody>
</table>

### Table VIII: Recommended doses of ARB in HF

<table>
<thead>
<tr>
<th>ARB</th>
<th>Initiating Dose</th>
<th>Target Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Losartan</td>
<td>25 mg daily</td>
<td>50 – 100 mg daily</td>
</tr>
<tr>
<td>Valsartan</td>
<td>40 mg daily</td>
<td>80 – 160 mg bid</td>
</tr>
<tr>
<td>Candesartan</td>
<td>4 mg daily</td>
<td>16 – 32 mg daily</td>
</tr>
</tbody>
</table>

### Table IX: Recommended doses of β-Blockers used in HF

<table>
<thead>
<tr>
<th>β-Blockers</th>
<th>Initiating Dose</th>
<th>Target Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Carvedilol</td>
<td>3.125 mg daily</td>
<td>25 mg bid</td>
</tr>
<tr>
<td>Bisoprolol</td>
<td>1.25 mg daily</td>
<td>10 mg daily</td>
</tr>
<tr>
<td>Metoprolol succinate CR*</td>
<td>12 5 – 25 mg daily</td>
<td>200 mg daily</td>
</tr>
</tbody>
</table>
4.2.3 Anti-tuberculosis drugs and the recommended dosages

4.2.3.1 1st line anti-tuberculosis drugs

<table>
<thead>
<tr>
<th>1st line drug</th>
<th>Daily dosage</th>
<th>Biweekly dosage</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>mg/kg</td>
<td>max (mg)</td>
</tr>
<tr>
<td>Isoniazid (H)</td>
<td>5 - 8</td>
<td>300</td>
</tr>
<tr>
<td>Rifampicin (R)</td>
<td>10 - 15</td>
<td>600</td>
</tr>
<tr>
<td>Pyrazinamide(Z)</td>
<td>20 - 40</td>
<td>1500</td>
</tr>
<tr>
<td>Ethambutol (E)</td>
<td>15 - 25</td>
<td>1200</td>
</tr>
<tr>
<td>Streptomycin(S)</td>
<td>15 - 20</td>
<td>1000</td>
</tr>
</tbody>
</table>

Note: For patients more than 65 years of age, the dose of streptomycin should not exceed 750mg.

12.2.1.1 Second line drugs

2nd line anti-tuberculosis drugs

<table>
<thead>
<tr>
<th>2nd line drug</th>
<th>Recommended dosage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amikacin/kanamycin</td>
<td>15mg/kg/day 5x/week</td>
</tr>
<tr>
<td>Ofloxacin</td>
<td>400 - 600mg/day</td>
</tr>
<tr>
<td>Ciprofloxacin</td>
<td>750 - 1500mg/day</td>
</tr>
<tr>
<td>Cycloserine</td>
<td>15mg/kg/day</td>
</tr>
<tr>
<td>Enfumycin</td>
<td>15mg/kg/day 5x/week</td>
</tr>
<tr>
<td>Capreomycin*</td>
<td>15mg/kg/day 5x/week</td>
</tr>
<tr>
<td>Clarithromycin</td>
<td>500mg bd</td>
</tr>
<tr>
<td>Azithromycin</td>
<td>500 mg daily</td>
</tr>
<tr>
<td>Para amino salicylic acid</td>
<td>12 - 16gm/day</td>
</tr>
<tr>
<td>Ethionamide</td>
<td>15 - 20mg/kg/day</td>
</tr>
<tr>
<td>Clofazimine</td>
<td>100 - 300mg/day</td>
</tr>
<tr>
<td>Thiaccetazone*</td>
<td>160mg/day</td>
</tr>
</tbody>
</table>

* Not used in Malaysia.
2. Helicobacter Pylori INFECTION
(Ref. P. Maifertheimer et al. GUT 2007. 56:772-781)

- Peptic ulcer disease (including complicated PUD)
- MALtoma
- Atrophic gastritis
- After gastric cancer resection
- Patient who are first-degree relatives of patients with gastric cancer
- Non-ulcer dyspepsia
- Naïve NSAID users
- Chronic NSAID users
- Long term PPI therapy
- Immune Thrombocytopenic Purpura
  and iron deficiency anaemia

  *Proton Pump Inhibitors (PPI)
  e.g. Omeprazole, Pantoprazole, Lansoprazole, Rabeprazole,
  Esomeprazole PO q12h for 7 days

**PLUS**
Clarithromycin 500mg PO q12h for 7 days

**PLUS**
Metronidazole 400mg PO q12h for 7 days

OR
Amoxicillin 1g PO q12h for 7 days

**PPI, e.g.**
Omeprazole 20mg PO q12h

**PLUS**
Amoxicillin 1g PO q12h

OR
Tetracycline 500mg PO q8h

**PLUS**
Metronidazole 400mg PO q8h for 10 days

- First choice therapy recommended
  in areas with <15-20% Clarithromycin resistance
- Bismuth-based quadruple therapy
  for 7-10 days may be used at second choice therapy if available.
- Third choice or rescue treatment
  should be based on antibiotic susceptibility testing

* Dosages:
  Omeprazole 20mg q12h
  Pantoprazole 40mg q12h
  Lansoprazole 30mg q12h
  Rabeprazole 20mg q12h
  Esomeprazole 20mg q12h

B. LOWER RESPIRATORY TRACT INFECTIONS

1. Community Acquired Pneumonia (CAP)

a. No comorbidity

<table>
<thead>
<tr>
<th>Streptococcus Pneumoniae</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mycoplasma Pneumoniae</td>
</tr>
</tbody>
</table>

- No recent antibiotic therapy
  EES 800mg PO q12h for 1 week
  OR
  Amoxicillin 500mg PO q8h for 1 week

- Recent Antibiotic Therapy
  Treat as b (Presence of comorbidity or History of recent antibiotic therapy) as below

  Azithromycin 500mg PO q24h for 3 days
  OR
  EES 800mg PO q12h for 1 week

b. Presence of comorbidity or History of recent antibiotic therapy (2 months)

<table>
<thead>
<tr>
<th>Streptococcus Pneumoniae</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mycoplasma Pneumoniae</td>
</tr>
<tr>
<td>Haemophilus Influenzae</td>
</tr>
</tbody>
</table>

- β-lactam/β-lactamase inhibitors, e.g.
  Amoxicillin/Clavulanic 825mg PO q12h for 1 week
  OR
  Amoxicillin/Sulbactam 375mg PO q12h for 1 week
  OR
  Doxycycline 100mg PO q12h for 1 week

- Levofloxacin 500mg PO q24h for 1 week

Conservative use of quinolone is recommended to minimize resistant pathogen. Use when patients failed first line regimens or allergic to alternative.
<table>
<thead>
<tr>
<th>Infection/Condition &amp; Likely Organism</th>
<th>Suggested Treatment</th>
<th>Alternative</th>
<th>Comments</th>
</tr>
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<tbody>
<tr>
<td>Moderate &amp; Severe CAP (not requiring mechanical ventilation) <em>Streptococcus Pneumoniae</em> <em>Mycoplasma Pneumoniae</em> <em>Haemophilus Influenzae</em> <em>Klebsiella Pneumoniae</em> <em>Legionella</em> <em>Staphylococcus Aureus</em> Other Gram Negative Bacilli - Enterobacter - Escherichia Coli</td>
<td>Azithromycin 500mg IV/PO q24h OR Firoxymycin 500mg IV q6IV/FFS 800mg PO q12h</td>
<td>Levofloxacin 500mg IV/PO q24h for 1 week</td>
<td>Empirical therapy for melioidosis should be considered if patient has diabetes melitus Conservative use of quinolone is recommended to minimize resistant pathogen. Use when patients failed first line regimen or allergic to alternative</td>
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### Pseudomonas Infection

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<td><strong>PLUS</strong> 3rd gen. Cephalosporins, e.g. Ceftriaxone 1-2g IV q24h OR β-lactam/β-lactamase Inhibitors, e.g. (Amoxycillin/Clavulanate OR Ampicillin/ Sulbactam) Duration: 1 week</td>
<td><strong>PLUS</strong> Ciprofloxacin 500mg IV q12h for 1 week OR Celespine 2g IV q12h for 1 week</td>
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<tr>
<td>Piperacillin/Tazobactam 4.5g IV q8h for 1 week OR Celespine 2g IV q12h for 1 week</td>
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### Infection/Condition & Likely Organism

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<td>3rd gen. Cephalosporins, e.g. Ceftriaxone 2g IV q24h</td>
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<tr>
<td><strong>PLUS</strong> Metronidazole 500mg IV q8h followed by 400mg PO q6h for 4-6 weeks</td>
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<tr>
<td>Cefazolin 2g IV q6h for 10-14 days Claxacillin 2g IV q4-6h for 2-4 weeks</td>
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</table>

### 2. Lung Abscess

Organisms likely to be involved are anaerobes (34%), Gram positive cocci (25%), *Klebsiella Pneumoniae* (25%), *S. Milleri* (16%), *Moraxella* (3%).

If suspect melioidosis

**Staphylococcus Aureus** (e.g. *IVD*)

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### 3. Empyema

Always investigate as per pleural effusion. Drainage via chest tube required. Tuberculosis must be excluded

**Empyema**

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If *Anaerobes isolated/suspected: Enterobacteriaceae Bacteroides sp.*

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<td><strong>PLUS</strong> Metronidazole 500mg IV q8h</td>
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If *Staphylococcus Aureus Isolated*

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β-lactam/β-lactamase inhibitors, e.g. (Amoxycillin/Clavulanate 1.2g IV q6h OR Ampicillin/Sulbactam 1.5g IV q12h)

Vancomycin 1g IV q12h (if MRSA suspected)
Another Project by Gerard Loh, member of the House Officers Workshop

Other Publications
The Ortho HO guide
The O &G HO guide

This compilation is not affiliated with Hospital Ampang and does not necessarily reflect the management and care by the staff. This is a sole project by housemen to aid housemen during their medical posting. The author will not be held responsible for any mishaps caused by following the suggested management. Always refer to the Malaysian Clinical Practice Guidelines for concise management and protocols to aid your practice.