Emergency Drug Guidelines

Kiribati Ministry of Health
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These guidelines have been adapted for Kiribati from the second edition of the Fiji Emergency Drug guidelines, 2007 with the kind permission of the National Drug and Therapeutics Committee, Fiji.

The Emergency drug guidelines overlap with some of the conditions already covered in the other booklets – particularly those in the cardiovascular, diabetes and respiratory guidelines. Some, like the management of cardiac arrest, are treated more fully in the Emergency Guidelines but the information in each set of guidelines is consistent.

Emergency Drug Guidelines were requested as a "stand-alone" booklet by the medical staff of Tungaru Hospital, Kiribati.

Contents

1 Cardiovascular Emergencies

1.1 Cardiac Arrest

1.1.1 Basic cardiac life support (BCLS)

Prompt and effective cardiopulmonary resuscitation (CPR) has been shown to increase survival after cardiac arrest. It should be begun as early as possible after the onset of cardiac arrest and continued with as little interruption as possible until the patient either recovers spontaneous circulation or a decision is made to cease the resuscitation efforts.

a. Call for help

Proper CPR requires at least two people. At least one other person is required to obtain the drugs and equipment needed for advanced cardiac life support.

b. Check for response

Assess the patient's conscious state quickly by shaking the patient and yelling his or her name. Loss of consciousness always accompanies cardiac arrest. Unconscious patients are unable to protect their own airway.

c. Airway

Look in the mouth for a foreign body or vomitus. These should be removed by a finger sweep. Listen for breath sounds. Noisy breath sounds are a sign of a partly obstructed airway. The absence of breath sounds may indicate complete airway obstruction.

Act to protect and maintain the airway. Perform appropriate procedures including suctioning, head tilt, chin lift, jaw thrust, and insertion of an oral airway. The correct size oral airway can be estimated by holding it against the side of the patients face - it should reach from the corner of the mouth to the ear lobe.

d. Breathing

Look for movement of the chest wall and listen to the lungs for breath sounds on both sides of the chest. Asymmetry of breath sounds may be a sign of a pneumothorax.

Act by ventilating the patient with a bag and mask. Be sure to use an appropriate size facemask that fits the patient's face. Mouth to mouth ventilation should be performed if a bag and mask are unavailable.

e. Circulation

Feel for the carotid or femoral pulse and listen for heart sounds. (The brachial pulse is often the easier to feel in small children)

If there is no palpable pulse, act by starting external cardiac massage. Cardiac massage should be performed on the lower 1/2 of the sternum, depressing it about 5 cm in adults and older children. In young children and babies it should be depressed about 1/4 of the distance between the front and the back of the chest. The rate should be 80 per minute in adults and 100 per minute in children and babies. The ratio of ventilations to compressions should be 1:5 in all ages if two people are performing resuscitation and 2:15 if one person is performing the resuscitation.

Start advanced cardiac life support as soon as possible.

1.1.2 Advanced cardiac life support (ACLS)

Cardiac arrest most commonly occurs due to life-threatening arrhythmias. The first step in ACLS is to determine what the cardiac rhythm is by attaching a cardiac monitor.
Cardiac arrest rhythms can be divided into three basic types:

- Pulseless ventricular tachycardia or ventricular fibrillation (VF)
- Asystole or severe bradycardia
- Pulseless ventricular activity

a. Pulseless ventricular tachycardia or ventricular fibrillation (VF)

Ventricular tachycardia without an adequate cardiac output should be treated as for ventricular fibrillation. The most important feature of the treatment of these arrhythmias is prompt **defibrillation**. Defibrillation is the only treatment that has been definitely shown to increase survival after cardiac arrest - it should be performed as early as possible.

The primary drug in the treatment of VF is adrenaline - all other drugs are of secondary importance.

- Provide basic cardiac life support as described above
- Defibrillate with 200 J), if no response then
- Defibrillate with 360 J, if no response then
- Defibrillate with 360 J
- Continue external cardiac massage
- Establish intravenous access
- Secure airway and continue to ventilate with maximum oxygen available
- Continue external cardiac massage
- **Give adrenaline 1 mg intravenous bolus (1 mL of 1:1000 or 10 mL of 1: 10 000)**

NOTE: Adrenaline dose should be followed by a 20 mL normal saline flush. Adrenaline may also be given down the endotracheal tube - the dose is 5 times the intravenous dose and it should be diluted in 10 ml of normal saline.

If still no response:

- Continue external cardiac massage
- **Give adrenaline 1 mg intravenous bolus (1 mL of 1:1000 or 10 mL of 1: 10 000)**

If still no response:

- Continue external cardiac massage
- Defibrillate at 360 J three times in succession

If no response has been achieved at this point, the chances of recovery are slight. Acidosis will certainly have occurred and may be treated with

- **Give 8.4% sodium bicarbonate (1 mmo per ml) 1 mmol per kg intravenously over 5-15 minutes**

Early administration of sodium bicarbonate is indicated in cases where arrhythmia is secondary to hyperkalemia, severe acidosis (e.g. due to renal failure) and overdose of tricyclic antidepressants.
Control of rhythm may be attempted with:

- **Lignocaine 1%, 75-100 mg intravenously over 1-2 minutes followed by 4 mg per minute for the next hour and decreasing to a maintenance dose of 1-2 mg per minute thereafter**

If patient is unresponsive or if Lignocaine is contraindicated:

- **Give high dose adrenaline, e.g. 5 mg intravenous bolus**

NOTES:

- If there is no spontaneous circulation 20 minutes after cardiac arrest then the chance of recovery is essentially zero.
- If sinus rhythm is restored the patient should be given Lignocaine 1 mg/kg intravenous bolus then commenced on a Lignocaine infusion.

- In children:
- Defibrillate at 2 J/kg then 4 J/kg
- **Give adrenaline 10 micrograms/kg (0.1 mL/kg of 1: 10 000 up to 1 mL)**

Ventricular tachyarrhythmias in special circumstances

Other drugs may be indicated in some special circumstances:

Digoxin Toxicity (see section 4.14) - Ventricular tachycardia in the presence of Digoxin toxicity may respond to phenytoin and magnesium sulphate. If defibrillation is necessary then 25 J may be all that is required. Higher defibrillation energies may induce ventricular fibrillation.

b. Asystole or severe bradycardia

Asystole has a very poor survival rate compared to VF. It is wise to make sure that the rhythm is indeed asystole by inspecting more than one lead on the ECG monitor. Very occasionally, VF may look like asystole in one of the ECG leads.

- Provide basic cardiac life support as described above
- Obtain intravenous access
- Secure airway and continue to ventilate with maximum oxygen available
- **Give adrenaline 1 mg IV bolus (1mL of 1:1 000 or 10mL of 1: 10 000)**
- Continue external cardiac massage

If no response:

- **Give atropine 3 mg IV bolus**
- Continue external cardiac massage

If no response:

- **Give adrenaline 5 mg IV bolus**
- Continue external cardiac massage

NOTES:
In children:

- **Give adrenaline 10 micrograms/kg (0.1 mL/kg of 1: 10 000 up to 1 mL) then 100 micrograms/kg**
- **Give atropine 50 micrograms/kg**

c. Pulseless ventricular activity (formerly called electromechanical dissociation [EMD])

This term refers to patients who have a cardiac rhythm other than VF, VT or asystole but without a detectable cardiac output. Most cases are due to severe and irreversible cardiac muscle dysfunction but occasionally pulseless ventricular activity may be due to a treatable cause.

Treatment is as for ventricular asystole with the addition of the need to exclude potentially reversible causes such as:

- Hypoxia
- Hypovolemia
- Hypothermia or hyperthermia
- Hypokalemia or hyperkalemia and metabolic acidosis
- Cardiac tamponade
- Tension pneumothorax
- Toxins, poisons, drugs
- Thrombosis – pulmonary or coronary

Tension pneumothorax

Insert an wide-bore IV cannula in the 2nd intercostal space in the mid-clavicular line on the side of the pneumothorax.

Hypovolaemia

- **Administer haemaccel 10 ml/kg intravenous bolus**

Severe hyperkalaemia or acidosis

- **Give 0.1 ml/kg of 10% calcium chloride (to a maximum dose of 5 ml) intravenous bolus and repeat in 5 minutes if necessary**

PLUS

- **Give 8.4% sodium bicarbonate 1 mmol/kg intravenous bolus**

Calcium channel blocker overdose or hypocalcaemia

- **Give 0.1 ml/kg of 10% calcium chloride (to a maximum of 5 ml) intravenous bolus** and repeat in 5 minutes if necessary. Massive doses may be required in calcium-channel blocker poisoning but this is a potentially reversible condition and resuscitation should be continued while repeated doses of calcium are given. Most poisoned patients have normal cardiac muscle and therefore the prognosis is much better than in those arresting after myocardial ischaemia

Beta-adrenergic antagonist overdose (see section 4.7)

- **Give glucagon 5 mg intravenous bolus**. Note: This is not available in the Kiribati EDL
- Obtain intravenous access
- Secure airway and continue to ventilate with maximum oxygen available.
- **Give adrenaline 1 mg IV bolus**
- Continue external cardiac massage
- **Give adrenaline 5 mg IV bolus**
- Continue external cardiac massage

1.1.3 Rapid sequence intubation

The aim of rapid sequence intubation is to obtain smooth and prompt control of the airway in emergency situations. All patients undergoing intubation in an emergency should be assumed to have a full stomach and so be at significant risk of aspiration. Properly prepared and checked equipment and appropriately trained staff are essential.

The steps of the procedure of rapid sequence intubation are:

a. Preoxygenation
b. Preparation
c. Sedation
d. Cricoid pressure
e. Paralysis
f. Intubation
g. Maintenance of sedation and paralysis

a. Pre-oxygenation

The patient should be pre-oxygenated with 100% oxygen via a bag and mask. This will increase arterial oxygen saturation to the maximum possible and also fill the lungs with oxygen providing a reservoir during intubation.

If the patient is breathing spontaneously then pre-oxygenate for three minutes (if time permits). Commence early while equipment is being checked and drugs drawn up - by the time the patient is ready to be intubated several minutes have usually gone by and the patient will be adequately pre-oxygenated.

If the patient is not breathing spontaneously (i.e. respiratory arrest) then ventilate with the bag and mask giving at least 3 large breaths prior to intubation.

b. Preparation

Make sure the following equipment is available and ready to use:

- suction catheter with adequate suction
- laryngoscope of the correct size with a light that works
- connectors for connecting the endotracheal tube and the ventilation bag
- an endotracheal tube with a balloon that stays inflated
- a flexible introducer or stylet for the endotracheal tube
- a 10 ml syringe to inflate the balloon
- the appropriate drugs (see below)
- a working intravenous line

c. Sedation

The choice of what drug to use for sedation depends upon the conscious state of the patient. Unconscious or semi-conscious patients will need very little sedation (or sometimes none at all). Awake patients will need much more sedation.

Several different drugs are available - it is best to use one with which you are familiar. All drugs are likely to cause hypotension to a lesser or greater degree.

i. Unconscious or semi-conscious:

- **Give diazepam 0.2 mg/kg intravenous bolus to a maximum of 10 mg**

OR

- **Give midazolam 0.2 mg/kg intravenous bolus to a maximum of 10 mg**

ii. Semi-conscious or conscious

- **Give ketamine 2 mg/kg intravenous bolus**

OR

- **Give midazolam 0.1 to 0.2 mg/kg intravenous bolus to a maximum of 15 mg**

PLUS

- **Give fentanyl 1 to 2 microgram/kg intravenous bolus**

OR

- **Give thiopentone 3-5 mg/kg intravenous bolus (use lowest dose)**

NOTE: Ketamine should not be used in patients who are at risk of raised intracranial pressure (e.g. meningitis, closed head injury). Thiopentone should not be used in patients who are hypotensive or hypovolaemic. Use the lower end of the dose ranges in the elderly or those who are hypotensive.

d. Cricoid pressure

As soon as the patient loses consciousness after sedation is given, firm pressure should be applied over the cricoid cartilage. This is to prevent regurgitation and aspiration of gastric contents. Cricoid pressure should be maintained until the patient has been intubated and the balloon of the endotracheal tube has been inflated.

e. Paralysis

It is not always necessary to paralyse a patient prior to intubation but it often makes the procedure quicker and easier. Deeply unconscious patients usually have little or no response to the stimulation of insertion of the laryngoscope and these are the patients in whom paralysis is optional. Most other patients should be paralysed prior to attempting intubation:

For ADULTS:

- **Give suxamethonium 1.5 mg/kg intravenous bolus**

OR

- **Give vecuronium 0.3 mg/kg intravenous bolus**

For CHILDREN:

- **Give suxamethonium 2 mg/kg intravenous bolus**

PLUS

- **Give atropine 20 mcg/kg intravenous bolus**

OR

- **Give vecuronium 0.2 mg/kg intravenous bolus**

NOTE: In general, suxamethonium is preferable to other non-depolarising drugs because of its more rapid onset and offset of action. Suxamethonium should not be used in the following circumstances:

- If the patient is markedly hyperkalaemic (serum potassium > 6 mmol/L)
- If the patient has suffered major burns (i.e. > 10% of body surface area 3rd degree burn) more than 3 days or less than 2 years previously
- In the presence of a chronic lower motor neurone disease (e.g. Guillain-Barre syndrome)

f. Intubation

This is an important skill that is best learnt through practice.

g. Maintenance of sedation and paralysis

The frequency and size of doses to maintain sedation and paralysis should be adjusted according to the patient's response.

i. Sedation

Bolus doses:

- **Give diazepam 0.1 to 0.2 mg/kg intravenous bolus every 2 hours**

OR

- **Give midazolam 0.1 to 0.2 mg/kg intravenous bolus every hour**

Infusion:

- **Give midazolam 0.05 mg/kg per hour via intravenous infusion and titrate infusion rate to patient response**

PLUS

- **Give morphine 0.05 mg/kg per hour via intravenous infusion and titrate infusion rate to patient response**

ii. Paralysis

- **Give vecuronium 0.1 mg/kg intravenous bolus every 40 minutes**

1.2 Cardiogenic Shock

Cardiogenic shock is defined as a state where the cardiac output is inadequate to maintain tissue perfusion. It is usually characterised by hypotension, compensatory peripheral vasoconstriction and signs of congestive cardiac failure. It should be distinguished from hypovolaemic shock (see section 6.5) and distributive shock (due to anaphylaxis or sepsis).

Cardiogenic shock has many different causes, the most common being myocardial infarction. Treatment of the underlying cause is essential. Administration of inotropic agents should be viewed only as a temporary measure while the underlying cause is reversed. Close monitoring in an intensive care unit is highly

desirable.

1.2.1 Maintain airway and breathing

The usual manoeuvres to maintain an adequate airway and adequate ventilation, up to and including endotracheal intubation should be used. All patients should at least receive high flow oxygen via face mask.

- **Give oxygen to maintain arterial oxygen saturation greater than 95%**

1.2.2 Optimise intravascular volume

Insertion of a central venous line allows accurate measurement of central venous filling pressures and also makes administration of inotropic agents safer. Correct anaemia with administration of blood or otherwise use boluses of normal saline to achieve an optimal central venous pressure. Note that patients with right ventricular infarction usually require a much central venous filling pressure (e.g. 30 mmHg) than other patients. If CVP is not available, examination of neck veins is an option.

- **Give 0.9% saline boluses of 100 ml intravenously to obtain an optimal central venous filling pressure**

1.2.3 Inotropic agents

The initial agent of choice in cardiogenic shock is dobutamine. However, this drug is not currently available on the Kiribati EDL

If hypotension remains a problem then adrenaline should be used. The addition of low dose dopamine may help to maintain urine output.

In ADULTS:

- **Give adrenaline 2 microgram/minute by intravenous infusion and increase rate by 1 to 2 microgram per minute every 5 minutes to a maximum of 20 microgram/minute**

PLUS if the urine output is less than 30 ml/hour

- **Give dopamine 2 microgram/kg per minute by intravenous infusion and increase to 5 microgram/kg per minute if necessary**

In CHILDREN:

- **Give dopamine 2 microgram/kg per minute by intravenous infusion and increase rate by 1 microgram/kg per minute every 5 minutes to a maximum of 20 microgram/kg per minute**

NOTE: Ideally, inotropic agents should be infused via a central venous line. Otherwise, a large peripheral vein (such as the femoral vein or the cubital veins) should be used. Dopamine in doses greater than 5 microgram/kg per minute acts as an inotropic agent but its side effects are greater than those of adrenaline.

1.3 Coronary Pain Syndromes

Pain attributable to coronary artery obstruction occurs in each of the three coronary pain syndromes – **stable angina, unstable angina and myocardial infarction. However, there are patients who are asymptomatic but have evidence of myocardial ischaemia.**

1.3.1 Stable angina

Angina pectoris is pain, usually felt in the central chest, which may radiate to the neck, both arms and occasionally, the back that occurs during exercise or emotional stress and is rapidly relieved by rest. Angina is stable if, for at least one month, it has been brought on by the same amount of exertion and is not accompanied by pain at rest – unless caused by emotional stress.

a. Acute attack

- **Glyceryl trinitrate 300-600 micrograms sublingually**

Repeat every 5 minutes if pain persists up to a maximum of three tablets. If pain persists, check that tablets are active (a tingling sensation if put on the tongue). If no response and tablets are of good quality, treat as for unstable angina. Patients should sit or lie down when first using glyceryl trinitrate because of the possibility of symptomatic hypotension. Glyceryl trinitrate should not be exposed to light.

b. Subsequent treatment

Patients should be on aspirin and will usually require further treatment to improve exercise tolerance. Initially, use:

- **Aspirin 100-150 mg orally daily**

AND

- **Atenolol 50-100 mg orally daily**

OR

- **Propranolol 40-80 mg orally daily**

The other drugs that can be considered in uncontrolled angina include:

- **Isosorbide dinitrate 10-40 mg orally three times daily**

To prevent the development of nitrate tolerance, there should be an interval of eight hours between the night dose and the first dose the next day.

- **Verapamil 40-120 mg orally 2-3 times daily**

OR

- **Nifedipine SR 20-40 mg orally daily**

Please note that the combination of a beta-blocker and Verapamil is contraindicated.

c. Use of glyceryl trinitrate as prophylaxis

Nitrates may be used prophylactically for any form of physical or emotional stress.

- **Glyceryl trinitrate 300-600 micrograms sublingually**

d. Refractory stable angina

Occasionally, patients will not respond to preventive treatment even if a combination of beta-blocker, calcium channel blocker (nifedipine) and nitrates is prescribed.

If pain persists despite addressing the modifiable risk factors and optimum drug therapy, it is recommended that patient be referred for further cardiac assessment with
a view to possible echocardiography, exercise stress test and coronary revascularization procedures.

1.3.2 Unstable angina

This coronary syndrome is characterised by anginal pain which is severe, of recent onset, or which has recently become abruptly worse. Angina occurring at rest or following recent myocardial infarction is also classified as unstable angina.

There is evidence that the reason for unstable angina is a sudden change in a previously stable plaque within an atheromatous coronary artery. Rupture of the endothelium over and around the plaque leads to vasoconstriction, platelet adhesion and an inflammatory response. If the vessel becomes completely occluded, a myocardial infarct will result. However, commonly, occlusion is not complete and the area around the plaque settles down over a period of a few weeks.

All patients diagnosed to be suffering from unstable angina should be referred for admission to an area where cardiac monitoring can be performed.
The most important distinction to make is between unstable angina and an acute myocardial infarction. The factors favouring an acute myocardial infarction include pain of more than 15-20 minutes duration;

pain not responsive to nitrates or requiring narcotics; systemic features such as pallor, sweating, vomiting and hypotension. If any or all of these are present, refer immediately for admission. An electrocardiogram (ECG) is critically important in making the diagnosis.

The aim of treatment in unstable angina is to relieve the pain and to modify the environment around the "active" plaque to reduce the likelihood of coronary artery occlusion. However, it should be borne in mind that chest pain might be secondary to other serious conditions like acute myocardial infarction, pericarditis, aortic dissection and pulmonary embolism.

For initial treatment:

- Oxygen therapy
- **Aspirin 150-300 mg orally stat**

AND

- **Morphine 2.5-10 mg intravenously as needed**

AND

- **Atenolol 50-100 mg orally daily**

OR

- **Propranolol 40-80 mg orally two to three times daily**

If pain persists and if the patient's hemodynamic status allows, ADD:

- **Nifedipine SR 20-40 mg orally twice daily**

AND, if required, ADD

- **Isosorbide dinitrate 10-40 mg orally three times daily**

If pain still persists, in addition, heparin should be given as follows:

- **Heparin 5,000 units by bolus dose followed by 1,000 units per hour by intravenous infusion**

Subsequent doses should be adjusted to keep the APTT (activated partial thromboplastin time) between 60 and 85 seconds. The APTT should be measured 6-hourly until stable, then daily.

Heparin will normally be required for at least three days and possibly longer depending on clinical response.

If symptoms persist despite all of the above treatment, cardiological intervention, if available, is required with a view to further investigation and revascularization.

1.3.3 Myocardial infarction

Complete occlusion of a coronary artery leads to the death of the cardiac muscle it supplies. Occlusion of a large, proximal vessel may cause myocardial ischaemia of such an extent that the patient dies rapidly of pump failure. Alternatively, a ventricular arrhythmia (tachycardia, fibrillation) may reduce cardiac output to such a drastic extent that, if the abnormal rhythm cannot be reversed, death is most likely.

Severity of pain by itself is a poor indicator of the extent of myocardial damage especially in a diabetic patient. Poor cerebral function, peripheral circulatory signs such as pallor, sweating and hypotension combined with extensive ECG changes with or without arrhythmias point to a large infarct.

The aims of immediate management are to:

- relieve pain
- achieve coronary reperfusion and minimise infarct size
- prevent and treat heart failure and shock
- allay the patient's anxiety

All patients with suspected myocardial infarction should be admitted to hospital and preferably to a unit where cardiac monitoring can be performed.

a. Immediate management

Unless the patient is very anxious, routine use of a sedative drug (e.g. diazepam) is not recommended.

- **Morphine 2.5-10 mg intravenously with repeat doses as necessary**

AND

- **Glyceryl trinitrate 600 micrograms sublingually with a repeat dose in 5 minutes if no response**

It should not be given in hypotension and if right ventricular infarction is suspected.

b. Limiting infarct size

- **Aspirin 300 mg chewed or dissolved before swallowing**
- **Oxygen 4-6 L per minute by mask**
- **Thrombolytic therapy – streptokinase**

The indications for thrombolytic therapy includes chest pain that has developed within the previous 12 (and preferably 6) hours with either ST segment elevation myocardial infarction (STEMI) or development of new left bundle branch block (LBBB).The difficulty with transport in Kiribati means that the only patients likely to be considered for thrombolysis are those living in south Tarawa.

Streptokinase

Administer streptokinase (STK) 1.5 million International Units (IU) by intravenous infusion over 30-60 minutes. If blood pressure falls as a result of the infusion, reduce the rate or stop briefly and restart at half the previous rate.

Streptokinase induces antibody formation that makes it unsuitable for use in subsequent episodes of coronary occlusion. It may also produce allergic symptoms (i.e. bronchospasm, angio-oedema, urticaria, flushing and musculoskeletal pain).

The contraindications to thrombolytic therapy are shown in Table 1.

Patients most likely to benefit from thrombolytic treatment are those presenting early with large anterior infarcts especially if complicated by heart failure. Those presenting after 24 hours have less chance of benefit and increased risk of cardiac rupture.

For mild or moderate allergic reactions to streptokinase:

- **Promethazine 25 mg intravenously**

OR

- **Hydrocortisone 100 mg intravenously**

Severe allergic reactions should be treated as for anaphylaxis. Give:

- **Adrenaline 1 in 1,000 solution, 0.5- 1 ml (0.5-1 mg) intravenously over 5 minutes**

Table 1. Contraindications to thrombolytic therapy

Absolute contraindications	**Relative contraindications**
Active internal bleeding	Previous peptic ulcer disease
Recent surgery, biopsy or trauma	Warfarin therapy
Prior cardiopulmonary resuscitation	Liver disease
Known bleeding disease (haemophilia, platelet disorders	Previous streptokinase therapy within the last four years
Recent or disabling stroke	Previous hypersensitivity to streptokinase
Neurosurgery within 6 months	Heavy pervaginal bleeding

A previous intracranial bleed Severe uncontrolled hypertension (a blood pressure greater than 180/110 mm) Hg during presentation) Aortic dissection Coma Oesophageal varices	Diabetic proliferative retinopathy Pregnancy

If response is poor, increase dose to:

- **Adrenaline 1 in 1,000 solution 2 to 5 ml (2-5 mg) intravenously over 5 minutes**

AND ADD

- **Promethazine 25 mg intravenously**

OR

- **Hydrocortisone 100 mg intravenously**

c. Management in the post-infarct period

i. Beta-blockers

- **Atenolol 25-100 mg orally daily**

OR

- **Propranolol 40-80mg orally two-three times daily**

The benefit persists long-term and beta-blockade should be continued **indefinitely.**

ii. Angiotensin converting enzyme inhibitors (ACEIs)

- **Enalapril 5-40 mg orally daily**

Outcome is improved after myocardial infarction with these agents. ACEIs should be started 24-48 hours after the acute episode in patients with a previous myocardial infarct, diabetes mellitus, hypertension, anterior infarct or evidence of persisting left ventricular dysfunction. Persistent hypotension and/or renal dysfunction are the only major contraindications.

iii. Antiplatelet agent

- **Aspirin 150-300 mg orally daily**

iv. "Statins" (hydroxymethylgutaryl CoA reductase inhibitor) drug

These compounds which inhibit one of the crucial steps in the biosynthesis of cholesterol have a limited and as yet not fully defined place in the primary prevention of cardiovascular disease (see text later). Recent large-scale trials have demonstrated a substantial role for them in the secondary prevention of coronary thrombosis and myocardial infarction. The benefits may not be fully explained by their lipid-lowering action so it is possible that an alternative mode of action may be involved. Survival benefits have been shown in patients with comparatively low, as well as elevated, total cholesterol at the outset.

The commonest adverse effect is reversible myalgia with elevated plasma creatine kinase levels and, rarely, rhabdomyolysis.

Simvastatin is available on the Kiribati EDL and there is no evidence that other "statins" provide any greater benefit

Thus a combination of lifestyle modification, and ongoing treatment with aspirin, beta blockade, a statin, and, in many cases, ACEIs has been justified by clinical trials of adequate and duration.

1.4 Cardiac Arrhythmias

Cardiac arrhythmias range from trivial ectopic beats to the life-threatening ventricular fibrillation. Whether or not an arrhythmia requires intervention depends largely on its capacity to make a significant impact on cardiac output.

In a patient whose myocardial function is already impaired (e.g. by a large infarct) a change from normal sinus rhythm to atrial fibrillation with a ventricular rate of 140 beats per minute may be sufficient to cause heart failure. By contrast, a young person with a normal myocardium may sustain a supraventricular tachycardia at the same rate for days without any evidence of cardiac decompensation.

The urgency for intervention and the nature of that intervention are dictated equally by the situation in which the arrhythmia occurs and by the nature of the arrhythmia itself.

1.4.1 Causes of cardiac arrhythmias

The common and/or important causes of arrhythmias are:

- ischaemic heart disease
- valvular heart disease
- cardiomyopathy
- hypoxia
- electrolyte disturbance – hypokalaemia, hyperkalaemia, hypocalcaemia, hypomagnesaemia
- endocrine – hyperthyroidism, phaechromocytoma(rare)
- drugs – Digoxin, tricyclic antidepressants
- congenital conduction abnormalities

1.4.2 Aims of treatment

In general, there are four aims in the treatment of cardiac arrhythmias:

- return the heart to normal sinus rhythm, if possible
- control the heart rate
- treat any associated risks (e.g. anticoagulant therapy in atrial fibrillation to prevent thrombo-embolism)
- treat the underlying cause

Most arrhythmias are benign and injudicious use of antiarrhythmic drugs can be harmful as many of them are proarrhythmic on their own.

1.4.3 Tachyarrhythmias

a. Atrial tachyarrhythmias

i. Sinus tachycardia

This implies a persistent heart rate over 100 per minute in a resting patient.

It usually has an underlying cause such as anxiety, thyroid overactivity or systemic illness. The first approach should be to identify and treat the underlying cause.

If no obvious underlying cause is apparent, treatment is generally not needed.

ii. Atrial premature complexes

Treatment is seldom required. If patient is symptomatic,

- **Atenolol 25-100 mg orally daily**

OR

- **Propranolol 40-80 mg orally two- three times daily**

iii. Paroxysmal supraventricular tachycardia (PSVT)

This occurs intermittently and sometimes can be converted to sinus rhythm by carotid sinus massage, by the Valsalva manoeuvre or by holding ice cold water in the mouth. If these are ineffective,

- **Verapamil 5 mg intravenously slowly; repeat if needed up to 15 mg**

If this is not available,

- **Digoxin 0.25-0.50 mg orally stat,**
  repeat same dose orally six hours later,
  followed by 0.25 mg orally six hours after the second dose, and
  followed by 0.25 mg orally six hours after the third dose and
  continue at 0.25 orally mg daily

If rapid control is needed, Digoxin may be given intravenously (see below under section on atrial fibrillation).However the distributive phase after either oral or intravenous use is exactly the same (around 6 hours) and therefore there is probably little to be gained from using it parenterally.

The maintenance Digoxin dose should be adjusted depending on the patient's renal function and serum potassium level.

Verapamil must never be given to a patient with a wide-complex undiagnosed tachycardia – QRS > 0.12 seconds. If there is any possibility that the rhythm is a ventricular tachycardia treat as for ventricular tachycardia.

iv. Prophylaxis for paroxysmal supraventricular tachycardia (PSVT)

A few patients may require prophylaxis if attacks are frequent. This may require electrophysiological investigation if available.

- **Atenolol 25-100 mg orally daily**

OR

- **Propranolol 40-80 mg orally three times daily**

v. Atrial flutter and fibrillation

Atrial flutter usually presents with a 2:1 atrioventricular block and a regular rate of 150 beats per minute. Atrial fibrillation presents with a similar rate which is however quite irregular. The aims of treatment are discussed below.

Control ventricular rate

This is only required if the ventricular rate is >100 per minute. The urgency to control the rate depends on the pre-existing ventricular rate.

Digitalization

- **Digoxin 0.5-1.0 mg orally, followed by 0.25-0.5 mg every 4-6 hours up to a maximum of 1.5-2.0 mg in the first 24 hours**

Maintenance treatment thereafter will require Digoxin 0.0625-0.5 mg daily depending on age, renal function and plasma Digoxin level, if available. **The intravenous route is rarely necessary because oral digititalization is just as effective.**

However, if rapid digitalization is needed and cannot be achieved with oral drug, Digoxin may be given intravenously. The total loading dosage is 0.5-1.5 mg. A loading dose of 0.5 mg in 20 ml of normal saline is given as an intravenous infusion for 20 minutes. The remaining dose is also given intravenously over 20 minutes at intervals of 4-6 hours depending on the response over a period of 24 hours. The total digitalizing dose will need to be reduced if the patient has had Digoxin in the preceding two-week period.

OR

- **Verapamil 5 mg intravenously up to 15 mg with careful monitoring of pulse and blood pressure**

For long-term control, Digoxin can be used. If the ventricular rate is not controlled, a beta-blocker can be added.

- **Atenolol 25-100 mg orally daily**
  OR
- **Propranolol 40-80 mg orally two-three times daily**

If beta-blockers are contraindicated,

- **Verapamil 40-80 mg orally three times daily**

Treatment of underlying cause

Whenever possible, the underlying cause should be identified and treated (e.g. hypokalaemia, thyrotoxicosis).

Reversal to sinus rhythm

For atrial fibrillation of recent onset, consideration should be given to convert it to sinus rhythm by electrocardioversion. Medical therapy with amiodarone or sotalol might be effective. In chronic AF, recent evidence suggests that rate control is just as effective as rhythm control.

Anticoagulant therapy

Unless contraindicated and impractical (i.e. poor patient compliance, difficulty in monitoring), anticoagulant therapy should be considered in every patient with chronic AF to prevent thromboembolic event. If Warfarin cannot be used for one reason or another, aspirin can be used as alternative but is not as effective. The risk of thromboembolism increases in patients with previous thromboembolism, mitral valve disease, heart failure, and hypertension and in older patients – especially women over the age of 75 years.

b. Ventricular arrhythmias

i. Premature ventricular ectopics including bigeminy

These are benign unless patients have underlying heart disease. If no obvious cause is found, the following measures are advisable:

- reduction coffee and tea intake
- cessation of smoking
- reduction alcohol intake

Drug treatment is not normally required but in symptomatic cases beta-blockade may be of value.

- **Atenolol 25-100 mg orally daily**

OR

- **Propranolol 40-80 mg orally two- three times daily**

ii. Ventricular tachycardia (VT)

Non-sustained ventricular tachycardia

In hospitals where ECG monitoring is possible, treat only prolonged episodes that cause cardiovascular haemodynamic instability.

- **Lignocaine, 1-1.5mg/kg (normally 75-100mg) intravenously over 1-2 minutes followed by 4 mg per minute intravenous infusion for a maximum of one hour then 1-2 mg per minute by intravenous infusion for 24 hours (see Appendix)**

Sustained ventricular tachycardia

- With haemodynamic stability
  Treatment is the same as for non-sustained ventricular tachycardia.

- With haemodynamic instability ("pulseless VT")
  The treatment for this condition is immediate intervention by defibrillation. Maintenance of sinus rhythm after electrocardioversion requires drug therapy:

- **Lignocaine 1-1.5 mg/kg (normally 75-100 mg) intravenously over 1-2 minutes followed by 4 mg per minute intravenous infusion for a maximum of one hour then 1-2 mg per minute by intravenous infusion for 24 hours (see Appendix)**

If maintenance treatment is required:

- **Atenolol 25-100 mg orally daily**
  OR
- **Propranolol 40-80 mg orally two- three times daily**

Amiodarone is an alternative but is not currently in the Kiribati EDL

iii. Torsades de pointes

This is a rare, polymorphic ventricular tachycardia in which the QRS axis is constantly shifting (turning, "torsade"). Patients usually have a prolonged QTc (greater than 0.45 seconds) on the ECG. The rhythm is particularly prone to occur as a result of drug therapy including treatment with tricyclic antidepressants, phenothiazines, erythromycin and ketoconazole. Any drug suspected of causing the arrhythmia should be stopped immediately.

Patients should be managed in hospital with ECG monitoring. No consensus exists about the most effective treatment. Lignocaine can be effective.

- **Lignocaine for i.v. use, 75-100 mg intravenously over 1-2 minutes followed by 4 mg per minute for a maximum of one hour. Maintenance infusion thereafter of 1-2 mg per minute by intravenous infusion (see Appendix)**

Alternatively,

- **Magnesium sulphate 50%, 2 g intravenously over 10-15 minutes followed, if necessary, by 0.5-0.75 g per hour by intravenous infusion for 12-24 hours**

DO NOT use amiodarone (if available) to treat this arrhythmia as it may provoke it.

iv. Ventricular fibrillation (see under cardiac arrest)

v. Ventricular asystole

Institute CPR.

- **Adrenaline 1mg (1 ml of a 1:1,000 solution) intravenously and repeat at 5 minute intervals until the return of spontaneous circulation is achieved**

- **Atropine 3 mg intravenously with a saline flush of 20 ml**

vi. "Pulseless" ventricular activity

Treatment is as for ventricular asystole with the addition of the need to exclude potentially reversible causes such as:

- hypoxia
- hypovolaemia
- hypothermia or hyperthermia
- hypokalaemia or hyperkalaemia and metabolic disorders
- cardiac tamponade
- tension pneumothorax
- toxins, poisons, drugs
- thrombosis – pulmonary or coronary

vi. Cardiac arrest

This is due to ventricular tachycardia, fibrillation, asystole or "pulseless" ventricular activity.

On the assumption that no immediate ECG diagnosis can be made of the underlying rhythm, immediately:

- Institute and continue cardio-pulmonary resuscitation (CPR).
- Defibrillate at 200 joules and, if no response, twice more at 360 joules (for children: 4 joules per kg).
- Secure airway and ventilate at maximum oxygen percentage achievable.
- Obtain an ECG tracing while maintaining CPR.
- Give adrenaline 1 mg (1 ml of a 1:1,000) as an intravenous bolus followed by 20 ml saline flush.
- Repeat defibrillation at 360 joules three times in succession.
- Repeat intravenous adrenaline. If venous access cannot be obtained in order to administer adrenaline, give adrenaline 5mg (5 ml of a 1:1,000 solution) diluted to 10 ml of normal saline may be given through the endotracheal tube.
- Repeat defibrillation at 360 joules on three successive occasions.

If no response has been achieved at this point, the chances of recovery are slight. Acidosis will certainly have occurred and may be treated with:

- **Sodium bicarbonate 8.4% (1 mmol per ml) 1 mmol per kg intravenously over 5-15 minutes**

Sodium bicarbonate is also indicated in cases where arrhythmia is secondary to hyperkalaemia.

Control of rhythm may be attempted with:

- **Lignocaine 1%, 75-100 mg intravenously over 1-2 minutes followed by 4 mg per minute for the next hour and decreasing to a maintenance dose of 1-2 mg per minute thereafter (see Appendix)**

However, the mainstay of management remains effective CPR followed by urgent defibrillation. The primary drug in emergency treatment is adrenaline.

1.4.4 Bradyarrhythmias

a. Sinus bradycardia

Treat only if symptomatic. Exclude hypothyroidism, pituitary failure and drugs (e.g. beta-blockers, Digoxin, and Verapamil).

If intervention is required:

- **Atropine 0.6-1.8 mg intravenously and repeat as needed**

b. Atrioventricular block

Drugs (Digoxin, beta-blockers or Verapamil) may be the cause and should be withheld if this appears to be the case.

i. First degree AV block

There is prolonged PR interval on ECG. This requires no treatment.

ii. Second degree AV block

There are two types.

Wenckebach phenomenon (Mobitz type I)

In this type of AV block, there is successive prolongation of the PR interval followed by a dropped beat and the whole cycle repeats.

Mobitz type II

There is a fixed ratio between the atrial and ventricular contractions in this type of arrhythmia, e.g. 2:1 or 3:1.

Generally, both types of AV block do not require treatment. Rarely, pacing may be required in Mobitz type II AV block.

iii. Third degree heart block

This may be an acute and potentially spontaneously reversible complication of, for example, an acute **anterior or inferior** myocardial infarction. In centres where cardiac pacing is possible, this is the treatment of choice.

If pacing is not available give:

- **Isoprenaline 20 micrograms intravenously,**
  repeat according to clinical response and
  follow with an infusion of 1-4 micrograms per minute or
  occasionally higher in patients who have been on beta-blockers (see Appendix)

There is anecdotal evidence for the efficacy of ephedrine, Salbutamol and theophylline in maintaining response if the block has responded to isoprenaline.

The treatment of choice for chronic heart block is permanent cardiac pacing.

iv. Sinoatrial block and sick sinus syndrome

These conditions require pacemaker therapy if persistent.

1.5 Acute Pulmonary Oedema

Acute pulmonary oedema is a medical emergency that requires prompt treatment. Oxygen, morphine, vasodilators and diuretics should be used. If the patient becomes hypotensive (systolic blood pressure <90 mmHg) then treat as for cardiogenic shock (see section 1.2).

1.5.1 Maintain airway and give oxygen

Give high flow oxygen via a face mask. Some patients with severe pulmonary oedema may require intubation and mechanical ventilation. The use of continuous positive airway pressure (CPAP) via mask is very useful if available.

1.5.2 Positioning

Sit the patient upright. This reduces the intrathoracic blood volume and improves ventilation of the lungs.

1.5.3 Bronchodilators

Fluid in the airways often causes bronchospasm, which worsens the effect of pulmonary oedema:

- **Give Salbutamol 5 mg via nebuliser (using oxygen) and repeat in 30 minutes if necessary**

1.5.4 Morphine

Narcotics reduce anxiety and dyspnea and may also cause pulmonary vasodilation

- **Give morphine 2.5 to 5 mg intravenously every 5 minutes to a maximum of 15 mg**

NOTE: Use the lower dose of morphine in the elderly and those patients with a lower body weight.

1.5.5 Vasodilators

Glyceryl trinitrate and isosorbide dinitrate cause vasodilation and may also improve myocardial blood supply:

- **Apply glyceryl trinitrate 2% paste 2 cm topically**

OR

- **Give glyceryl trinitrate tabs 600mcg sublingually and repeat every 15 minutes if necessary**

1.5.6 Diuretics

Intravenous frusemide has a beneficial vasodilatory action as well as being a powerful diuretic:

- **Give frusemide 40 mg intravenous bolus**

NOTE: Patients with renal impairment may require larger doses of frusemide (up to 250 mg). It is best to titrate repeat doses according to the patient's response. If the urine output is inadequate 30 minutes after the first dose then give a further dose of 80 mg intravenously. Doses greater than 40 mg should be given slowly over 5 to 10 minutes to avoid damage to the inner ear.

1.5.7 Inotropes

Failure to respond to the above treatment may require addition of dobutamine(if available) or dopamine as for cardiogenic shock (see section 1.2). Most patients needing inotropes will also need intubation and mechanical ventilation. Persistent hypoxia (oxygen saturation less than 90%) despite treatment is an indication for intubation.

1.6 Hypertensive Emergency

This is seldom needed but may be required in hypertensive encephalopathy, acute ehypertensive heart failure, dissecting aneurysm and phaeochromocytoma. Patients with these conditions should be admitted to the hospital and monitored. The aim is to reduce blood pressure within 60-90 minutes.

While the blood pressure may respond to oral agents (as above), initial parenteral treatment may be needed:

- **Hydrallazine 5 mg bolus intravenously (IV) over 5-10 minutes and repeated every 20 minutes up to a maximum of 20 mg followed by intravenous infusion of hydrallazine (see Appendix)**

OR

- **Labetalol (100 mg per 20 ml); initial dose of 20-40 mg given intravenously over 1-2 minutes and repeated at intervals of 5-10 minutes until 200 mg have been given. Alternatively, labetalol may be given as a continuous intravenous infusion at a rate of 2 mg per minute (see Appendix)**

After initial stabilisation, the patient should be transferred to oral treatment for maintenance.

The practice of opening a nifedipine 10 mg capsule and giving it sublingually is **not supported** as emergency treatment. It delivers an uncertain dose and most of the effect occurs as a result of absorption of the swallowed drug. **On occasions in older patients unexpected rapid falls in blood pressure have resulted in stroke or myocardial infarction.**

Please note that in some situations, urgent reduction with intravenous drugs over a short period of time is not recommended (e.g. severe, uncomplicated essential hypertension; severe hypertension postoperatively in a patient suffering from pain; severe asthma). In such situations, oral antihypertensive drugs can be used and blood pressure reduction can be achieved in 48-72 hours.

2 Respiratory Emergencies

2.1 Asthma

Asthma is a common respiratory disease in both adults and children. Severity is estimated by clinical assessment, measurement of peak expiratory flow rate and by pulse oximetry. All patients with moderate or severe asthma should be given oxygen. Patients with severe asthma should be managed in an intensive care unit if possible and may occasionally require intubation and mechanical ventilation.

2.1.1 Treatment in adults

a. Oxygen

- Give oxygen 6-8 l/min via face mask if moderate or severe asthma

b. Beta-adrenergic agonists

Use beta-adrenergic agonists to reverse bronchospasm. In very severe asthma intravenous Salbutamol may be useful in addition to nebulised.

- **Give Salbutamol 5 mg by nebuliser with oxygen and repeat every 30 minutes if necessary (or give continuously in severe asthma)**

OR

- **Give Salbutamol by puffer using spacer (up to 50 puffs) if nebulisers are not available**

PLUS if very severe

- **Give Salbutamol 5 microgram/kg intravenously (to a maximum of 250 microgram) over one minute then commence an infusion at 5 microgram/kg per hour**

NOTE: Continuous nebulised Salbutamol is probably as effective as intravenous Salbutamol.

c. Anticholinergics

These agents have a synergistic effect with beta-adrenergic agonists. In severe asthma consider the use of ipratropium.
(At the time of writing this drug is **not** available in Kiribati)

- **Give ipratropium bromide 250-500 μg by nebuliser and repeat every 4 hours if necessary**

d. Corticosteroids

Steroids reduce inflammation of the airways. Their effects are delayed for at least 4 hours but they are important to prevent relapse. Oral steroids are as effective as those given parenterally in most patients.

- **Give hydrocortisone 200 mg intravenously then 100 mg 6-hourly**

OR

- **Give prednisolone 40 mg orally daily**

e. Other drugs

Aminophylline provides no additional benefit to optimal doses of beta-adrenergic agonists. It has a number of undesirable side effects including seizures, ventricular tachycardia, hypokalaemia and vomiting. Routine use in asthma is not recommended. However, it may be of benefit in patients with severe asthma who require hospitalisation. A loading dose is given to patients who are not taking oral theophylline:

- **Give aminophylline 5 mg/kg (to a maximum of 250 mg) intravenously over 5 minutes**
- Followed an infusion at a dose of 0.6-0.9 mg/kg/min

Adrenaline does not appear to have any advantage over Salbutamol. It may be used as a last resort or when intravenous access is not available:

- **Give 1:1000 adrenaline 0.5 - 1 mL intramuscularly or subcutaneously**

NOTE: Adrenaline may be given down the endotracheal tube - the dose is 5 times the intravenous dose and it should be diluted in 10 ml of normal saline.

2.1.2 Treatment in children

a. Oxygen

- Give oxygen via face mask to all children with asthma.

b. Beta-adrenergic Agonists

Use beta-adrenergic agonists to reverse bronchospasm. In very severe asthma intravenous Salbutamol may be useful in addition to nebulised.

- **Give Salbutamol 2.5 mg by nebuliser with oxygen to children 5 years of age or under, or**

give 5 mg by nebuliser to children over 5 years and repeat every 30 minutes if necessary (or give continuously in severe asthma)

PLUS if very severe

- **Give Salbutamol 5 microgram/kg intravenously (to a maximum of 250 microgram) over one minute then commence an infusion at 5 microgram/kg per hour**

NOTE: Intravenous Salbutamol may be more effective than continuous nebulised in young children with severe asthma.

c. Anticholinergics

These agents have a synergistic effect with beta-adrenergic agonists (Not available in Kiribati)

- **Give ipratropium bromide 0.25 mg by nebuliser and repeat every 4 hours if necessary**

d. Corticosteroids

Steroids reduce inflammation of the airways. Their effects are delayed for at least 4 hours but they are important to prevent relapse. Oral steroids are as effective as those given parenterally in most patients.

- **Give hydrocortisone 1 - 4 mg/kg intravenously to a maximum of 200 mg then every six hourly**

OR

- **Give dexamethasone 0.2 mg/kg intravenously or intramuscular to a maximum of 8 mg**

OR

- **Give prednisolone 1 mg/kg orally to a maximum of 50 mg daily**

e. Other Drugs

Aminophylline provides no additional benefit to optimal doses of beta-adrenergic agonists. It has a number of undesirable side effects including seizures, ventricular tachycardia, hypokalaemia and vomiting. Routine use in asthma is not recommended. However it may be of benefit in patients with severe asthma.

A single dose is given to patients who are not taking oral theophylline:

- **Give aminophylline 5 mg/kg (to a maximum of 250 mg) intravenously over 5 minutes**

Adrenaline does not appear to have any advantage over Salbutamol. It may be used in severe asthma as a last resort or when intravenous access is not available:

- **Give 1:1000 adrenaline 0.1 ml/kg intramuscularly or subcutaneously to a maximum of 0.5 ml**

2.2 Exacerbation of Chronic Obstructive Airways Disease

Exacerbation of chronic obstructive airways disease (COAD) is a common problem in emergency medicine. The response of COAD to treatment is generally slower than that of asthma and most patients require admission.

2.2.1 Oxygen

It is essential that oxygen be given to maintain oxygen saturation greater than 92%. Although administration of oxygen can cause an elevation in arterial carbon dioxide levels in a few patients, this is far less of a problem than hypoxia itself. For mildly hypoxic patients oxygen via an intranasal catheter will be sufficient while those with more severe hypoxia may require oxygen via a face mask. Use the lowest flow rate necessary to maintain an adequate arterial oxygen saturation.

CAUTION: In patients with C02 retention, oxygen saturation should be maintained between 90 – 95%.

2.2.2 Bronchodilators

- **Give Salbutamol 5 mg via nebuliser every 2 to 4 hours**

2.2.3 Corticosteroids

Oral and parenteral routes are equally effective except in the sickest patients.

- **Give hydrocortisone 200 mg intravenously every 6 hours**

OR

- **Give prednisolone 40 mg orally daily**

2.2.4 Antibiotics

Antibiotics should be given when infection is the underlying cause of the exacerbation.

- **Give amoxycillin 500 mg orally or ampicillin 500mg intravenously every 8 hours**

OR if penicillin sensitive

- **Give erythromycin 500 mg orally every 6 hours**

2.3 Croup

Croup is a viral infection of the upper airway which affects children from the ages of 6 months to 3 years. It is characterised by fever, a harsh cough, a hoarse voice and stridor. Children who have stridor while at rest or who have signs of respiratory distress (i.e. suprasternal retraction, tachypnea, restlessness) should be admitted. Pulse oximetry is useful – an oxygen saturation of 93% or less while breathing air is also an indication for admission. Most cases of croup however are mild and self-limited.

2.3.1 Mild croup

These patients will have stridor only with exertion or crying and no signs of respiratory distress. Avoid exposure to cold air. Give paracetamol for fever.

- **Give paracetamol 20 mg/kg every 4 hours**

2.3.2 Moderate croup

These patients will have stridor at rest and some signs of respiratory distress but oxygen saturation should be greater than 90% on air.

- **Give oxygen to maintain an oxygen saturation greater than 93%**

PLUS

- **Give dexamethasone 0.6 mg/kg intramuscularly as a single dose**

2.3.3 Severe croup

These patients will have signs of marked respiratory distress plus hypoxia or cyanosis. Admission to an intensive care unit is desirable and intubation may be necessary.

- **Give oxygen to maintain an oxygen saturation greater than 93%**

PLUS

- **Give dexamethasone 0.6 mg/kg intramuscularly as a single dose**

PLUS

- **Give nebulised adrenaline, 0.5 ml/kg of 1: 1000 solution or 0.05 ml/kg of a 1% solution diluted with saline to a volume of 2.5 ml**

NOTE: Patients who fail to respond to nebulised adrenaline may require endotracheal intubation. Nebulised adrenaline provides only temporary relief of airway obstruction lasting 1 to 2 hours. Patients should be closely observed after this period for recurrence of obstruction.

2.4 Epiglottitis

Epiglottitis is a medical emergency and failure to provide prompt treatment may be fatal. It is due to infection of the epiglottis with Haemophilus influenzae bacteria. Epiglottitis mainly affects children between the ages of 3 and 8 years but is occasionally seen in adults as well. It is characterised by fever,

inspiratory and expiratory upper airway noises, a severe sore throat, dysphagia and drooling. The patient usually looks very unwell.

There is a very high risk of acute airway obstruction. All patients should be referred immediately to an anaesthetist and admitted to an intensive care unit. Attempting to view the throat or otherwise upsetting the child may cause airway obstruction and should be avoided. Keep the patient sitting up.

- **Give ceftriaxone 100 mg/kg stat then 50mg/kg intravenously daily**

OR

- **Give chloramphenicol 40 mg/kg stat then 25mg/kg intravenously daily**

2.5 Oxygen Therapy

Oxygen is essential for human metabolism and lack of oxygen is generally fatal within 5 to 6 minutes. Oxygen has almost no adverse effects in the acute situation and should not be withheld if there is any suggestion of it being needed. The indications for oxygen therapy are:

- Cardiac or respiratory arrest
- Hypoxia of any cause
- Cardiac failure
- Myocardial infarction
- Shock of any cause
- Carbon monoxide poisoning

Oxygen therapy should be monitored with pulse oximetry and blood gas estimation if available. Aim to achieve an oxygen saturation of at least 95%. Humidification of oxygen is not necessary.

2.5.1 Methods of oxygen delivery

a. Intranasal catheters

These provide a low concentration of oxygen of between 25 and 40%. They should be used with an oxygen flow rate of between 1 and 4 litres/minute (1 – 2L/min in children). Higher flow rates cause drying of the nasal mucosa and are uncomfortable. They should only be used in patients with mild hypoxia or cardiac failure or myocardial ischaemia. They do not provide a high enough oxygen concentration for patients with significant hypoxia, carbon monoxide poisoning, shock or cardiac arrest.

b. Plastic face masks

These provide oxygen concentrations of between 35 and 70%. The oxygen flow rate should be set between 4 and 15 litres/minute. Do not use face masks with an oxygen flow rate less than 4 litres/minute. This method of oxygen delivery is suitable for patients with moderate hypoxia or shock.

c. Tight Fitting Face Masks (e.g. Laerdal, CPAP masks)

These devices can provide oxygen concentrations close to 100%. They should be used in patients with severe hypoxia or with cardiac arrest.

2.5.2 Adverse effects of oxygen

Patients with chronic obstructive airway disease and elevated carbon dioxide levels may occasionally have a hypoxia-dependent respiratory drive. In these patients, the administration of oxygen causes hypoventilation and an increase in the carbon dioxide level. Although this may cause problems it is far less dangerous than hypoxia itself. In the emergency situation, it is important that hypoxia is corrected – problems with carbon dioxide retention can be handled later. Do not hesitate to give oxygen to hypoxic patients with chronic obstructive airway disease.

Administration of 100% oxygen sometimes causes pulmonary toxicity but this only occurs after 24 hours and therefore is not a problem in the emergency situation.

NOTE: If arterial blood gases are available, then they should be measured before the commencement of oxygen to establish the baseline.

3 Neurologic Emergencies

3.1 Seizures

There are numerous causes of epileptic seizures. In adults the majority occur in known epileptics with idiopathic epilepsy whilst in children febrile convulsions are a common cause. However, it is important to exclude less common, reversible and serious causes of seizures such as hypoglycaemia, hyponatraemia, hypocalcaemia, eclampsia, drug overdose, meningitis or intracranial haemorrhage.

Most seizures are self-limited and brief. Emergency drug treatment is only necessary if the seizures are prolonged (>5 minutes) or recurrent. Initial treatment is gentle restraint of the patient in the left lateral position and administration of high flow oxygen via face mask.

3.1.1 Treatment in adults

- **Give diazepam 5 mg intravenous bolus and repeat every 2 minutes to a maximum dose of 20 mg. This dose may be repeated 30 minutes later if necessary**

OR if there is no intravenous access

- **Give diazepam 0.5 mg/kg per rectum (use intravenous solution)**

PLUS if seizures persist

- **Give phenytoin 15 mg/kg via intravenous infusion over 20 minutes**

NOTE: If seizures persist despite diazepam and phenytoin, THEN diazepam and thiopentone infusions should be considered.

a. Diazepam infusion

- **Give diazepam 80 –100mg in normal saline to achieve a rate of 2mg/hour**

Patient must be carefully monitored for respiratory depression.

b. Thiopentone infusion

The patient should be intubated before the thiopentone infusion is begun:

- **For induction give thiopentone 5 mg/kg intravenous bolus**

THEN

- **Commence thiopentone infusion at a rate of 50 mg/hour**

3.1.2 Treatment in children

- **Give diazepam 0.1 mg/kg intravenous bolus and repeat every 2 minutes to a maximum dose of 0.4 mg/kg**

OR if there is no intravenous access

- **Give diazepam 0.5 mg/kg per rectum**

PLUS if seizures persist

- **Give phenytoin 15 mg/kg via intravenous infusion over 20 minutes**

OR

- **Give phenobarbitone 10 mg/kg IV or IM which may be repeated hourly up to maximum of another two doses, followed by a maintenance dose of 5mg/kg daily**

NOTE: If seizures persist despite diazepam and have lasted more than 20 minutes then the patient should be intubated and a thiopentone infusion begun.

- **For induction give thiopentone 5 mg/kg intravenous bolus**

THEN

- **Commence thiopentone infusion at a rate of 1 mg/kg per hour**

3.2 Migraine

Migraines are recurrent, often unilateral, throbbing headaches associated with nausea, photophobia and sometimes visual disturbances. The diagnosis is usually fairly obvious but it is important to consider other causes of headache (e.g. meningitis, subarachnoid haemorrhage, cerebral haemorrhage) if there are atypical features.

3.2.1 Treatment in adults

Patients should rest in a quiet dark room after treatment.

- **Give metoclopramide 10 mg intramuscularly**

OR

- **Give prochlorperazine 5mg orally or 12.5 mg intramuscularly**

THEN 15 minutes later

- **Give aspirin 900 mg orally**

PLUS

- **Give paracetamol 1.5 g orally**

OR

- **Give paracetamol 1 g rectally**

NOTE: In severe cases, consider admission for a trial of narcotics.

3.2.2 Treatment in children

Paracetamol alone is usually sufficient along with rest in a quiet dark room.

- **Give paracetamol 20 mg/kg orally or rectally**

3.3 Oculogyric Crisis

Oculogyric crisis is an acute focal dystonic reaction to neuroleptic agents and anti-emetic drugs such as metoclopramide. It most commonly affects young women. It is characterised by involuntary deviation of the eyes upward often with torticollis (spasm of the neck muscles).

- **Give benztropine 40 microgram/kg (to a maximum of 2 mg) orally or intramuscularly**

NOTE: A further oral dose of benztropine should be given 6 hours later to prevent recurrence.

3.4 Tetanus

Tetanus is a severe life-threatening disease caused by the toxin producing bacteria *Clostridia tetani*. It usually follows local wound contamination in an improperly immunized individual. Clinical features include muscle rigidity (esp. trismus), painful muscle spasms, fever, labile hypertension and abnormalities of cardiac rhythm. Patients should be managed in an intensive care unit if possible.
With the advent of routine immunization in children, this condition is very rare in Kiribati

3.4.1 Airway and breathing

Give high flow oxygen via face mask. Patients with respiratory muscle involvement will need intubation, muscle paralysis and ventilation.

3.4.2 Tetanus immune globulin

This substance neutralizes circulating toxin. Large doses are required:

- **Give tetanus immune globulin 4000 units intravenously over 30 minutes**

3.4.3 Wound debridement

Aggressive wound debridement is essential.

3.4.4 Antibiotics

Penicillin or metronidazole are effective:

- **Give benzylpenicillin 100,000 units/kg (maximum dose 2.4 million units) intravenously every 4 hours**

OR if penicillin sensitive

- **Give metronidazole 7.5 mg/kg (maximum dose 500 mg) intravenously every 8 hours**

3.4.5 Muscle spasms

Morphine and diazepam are used to control muscle spasms. Very large doses may be required.

3.5 Acute Bacterial Meningitis

In adults, *Streptococcus pneumoniae* is the most likely organism. *Haemophilus influenzae* and *Neisseria meningitides* are less common. Cerebrospinal fluid (CSF) microscopy and culture are vital in directing antibiotic therapy. Therefore, a lumbar puncture and blood culture should be performed as soon as possible. Caution is required with lumbar puncture if the patient is in coma, has signs of increased intracranial pressure or has focal neurological signs. A computed tomography (CT) scan of the head is preferred before lumbar puncture in such cases if facilities are available.

Bacterial meningitis is a medical emergency and antibiotic therapy should not be delayed if there is difficulty in obtaining a CSF sample. In such cases, empirical therapy should be started immediately.

In rural areas or where there is a delay in transferring patient to a major hospital, if meningitis is suspected, antibiotics should be started immediately, either with:

- **Penicillin G 4 megaunits intravenously/intramuscularly, 6-hourly**

OR

- **Ceftriaxone 2 g intravenously as a single dose (if available)**

Penicillin, chloramphenicol and ceftriaxone have proven to be effective in the treatment of meningitis. Chloramphenicol in oral doses achieves good CSF penetration.

Dexamethasone has been found to be useful in children. Recent literature suggests that it has a role in the management of bacterial meningitis in adults and is to be given just before the first antibiotic dose.

- **Dexamethasone 10 mg intravenously just before the first dose of antibiotic followed by 10 mg 6-hourly intravenously for 4 days**

3.5.1 Empirical therapy

- **Penicillin G 1.8 g (3 megaunits) intravenously 4-hourly for 10 days**

PLUS

- **Chloramphenicol 750 mg – 1 g intravenously 6-hourly for 10 days**

In patients hypersensitive to penicillin:

- **Chloramphenicol alone**

OR

- **Ceftriaxone 4 g intravenously daily in one or two divided doses**

Change to appropriate regimen once the organism and susceptibility result is available. If no organism is identified, continue empirical therapy for a total of 10 days.

3.5.2 Specific therapy where organism is known or strongly suspected

a. *Pneumococcal/Neisseria meningitidis* meningitis

- **Penicillin G 1.8 g (3 megaunits) intravenously 4-hourly**

In penicillin hypersensitive patients:

- **Ceftriaxone 4 g intravenously daily in two divided doses**

Pneumococcal meningitis is to be treated for 10-14 days. Some very ill patients may require treatment for 21 days. Meningitis due to *Neisseria meningitidis* usually requires treatment for 7 days only.

NOTE: At the end of penicillin therapy, *rifampicin 10 mg/kg/dose (up to 600 mg) orally 12-hourly for 4 doses* should be given to eradicate nasopharyngeal carriage in cases of meningococcal meningitis. This treatment should also be given to all close contacts.

b. Gram negative bacterial meningitis and cryptococcal meningitis

Consultation is advisable. Generally, *for* gram-negative meningitis other than *H. influenzae*, a combination of ceftriaxone and gentamicin is useful and the treatment is for 21 days.

4 Poisoning and Overdoses

Poisoning may occur with both chemicals (e.g. insecticides) or with therapeutic drugs, many of which can be toxic in overdose. Poisoning and overdose may or may not be life-threatening, depending on the type and amount of substance ingested. Treatment is most often supportive only and care should be taken that any intervention does not worsen the situation. Below are the steps to be taken in most cases of poisoning.

4.1 General principles

4.1.1 Resuscitation (see section 2.1)

- Rapidly assess the airway, breathing and circulation
- Maintain the airway if necessary
- Administer oxygen
- Obtain intravenous access
- Give intravenous fluids if the patient is hypotensive

NOTE: Oxygen should be avoided unless absolutely necessary in patients with paraquat poisoning as it may increase toxicity. Patients who are unable to protect their airway should be intubated BEFORE insertion of a nasogastric tube and administration of activated charcoal.

4.1.2 Gastric decontamination

The best method of removal of ingested poisons is with activated charcoal.

- **Give activated charcoal 1 g/kg (to a maximum of 50 g) orally or via nasogastric tube**

In children:

- **Give activated charcoal 15 – 30g if under 12 years old and 50 – 60g if over 12 years of age**

NOTE: Ipecac syrup has no role in the treatment of poisoning. Gastric lavage should be performed only in exceptional circumstances such as recent ingestion of large doses of paracetamol. Gastric lavage is

contraindicated in ingestion of hydrocarbons, caustics and corrosives.

Administration of activated charcoal is the easiest, safest and most effective method of decontamination of the gut in almost all situations. In paraquat poisoning, Fullers Earth (if available) should be substituted for activated charcoal. Activated charcoal does not effectively absorb hydrocarbons, anticholinesterase insecticides, heavy metals or acids and alkalis but it is unlikely to cause harm in these situations and may still be given, especially if there is doubt about exactly what the patient has ingested.

4.1.3 Supportive care

Continued observation and the provision of oxygen, intravenous fluids and airway support as required. Knowledge of the pharmacologic effects of the substance ingested allows anticipation of possible problems.

4.1.4 Specific antidotes

Antidotes to a number of drugs exist and their use are described in the sections below.

4.1.5 Psychiatric evaluation

Self-administered overdoses are more often expressions of distress due to emotional crises than true suicide attempts. All patients should be assessed for suicidal intent and treated appropriately. Appropriate counselling should be given prior to discharge.

4.2 Treatment of Specific Poisons

4.2.1 Opiates (e.g. codeine, heroin, pethidine, morphine, methadone)

These drugs cause depression of conscious state and hypoventilation. Particular attention should be paid to the maintenance of the airway and adequate ventilation. The specific antidote naloxone is highly effective.

- **Give naloxone 0.4 mg intravenously or intramuscularly and repeat in 5 minutes if necessary to a maximum of 2 mg**

NOTE:

- Failure to respond to a dose of 2 mg of naloxone is an indication that the depression of conscious state is not due to opiate ingestion alone - other possible causes should be considered
- Naloxone has a short half-life and further IM doses may be required after 1-2 hours
- Naloxone may induce pulmonary oedema

4.2.2 Paracetamol

Overdose of this drug is common and can be fatal. Initial symptoms are mild with nausea, vomiting and sometimes abdominal pain. Hepatic failure and death may follow in days to weeks. The minimum toxic dose is 150 mg/kg and almost all patients who ingest more than 350 mg/kg will develop hepatic failure. Acetylcysteine is the specific antidote and if given within 8 hours will completely prevent hepatic damage. It is still useful when given from 8 to 48 hours after the ingestion.

Assessment of the risk of hepatic damage is done using the Rumack-Matthew nomogram which relates serum paracetamol levels to time since ingestion. If a paracetamol level is available within 8 hours of ingestion then withhold acetylcysteine until a toxic level is confirmed. If a paracetamol level will not be available within 8 hours of ingestion then acetylcysteine should be commenced immediately. **If there is any doubt about the time of ingestion or if paracetamol levels are not available, then acetylcysteine should be given regardless.**

- **Give acetylcysteine 150 mg/kg intravenously over 15 minutes**

THEN

- **Give acetylcysteine 50 mg/kg intravenously over 4 hours**

THEN

- **Give acetylcysteine 100 mg/kg intravenously over 16 hours**

NOTE: Acetylcysteine may cause severe bronchospasm in some individuals. If this occurs the infusion should be ceased and Salbutamol administered. Hydroxycobalmin has been used as an alternative antidote.

4.2.3 Anticholinesterases (e.g. insecticides)

This would be a rare poison to be used in Kiribati as there is little crop-growing pasture
These substances cause severe cholinergic effects including vomiting, diarrhoea, bradycardia, hypotension, hypersalivation, bronchospasm, urinary incontinence, muscle weakness, constricted pupils and pulmonary oedema. Poisoning may occur with skin exposure or inhalation, as well as with oral ingestion. The specific antidote is atropine and very large doses may be required. Atropine will not reverse muscle weakness so intubation and mechanical support of ventilation may be required. **Pralidoxime**, which is an acetylcholinesterase reactivator, may also be useful.

a. Treatment in adults

- **Give atropine 3 mg intravenously every 5 minutes until the patient develops sinus tachycardia (heart rate up to 100 beats/min) (there is no maximum dose - 20 or 30 mg may be required)**

PLUS

- **Pralidoxime 1 g intravenously over 30 minutes and repeat every 12 hours if symptoms persist**

b. Treatment in children

- **Give atropine 1.2 mg intravenously every 5 minutes until the patient develops sinus tachycardia (heart rate of up to 120 beats/min)(there is no maximum dose)**

PLUS

- **Pralidoxime 20 mg/kg intravenously over 30 minutes and repeat every 12 hours if symptoms persist**

NOTE: Further doses of atropine may needed for 24 to 48 hours after exposure.

4.2.4 Aliphatic hydrocarbons (e.g. kerosene, petroleum)

Hydrocarbons cause irritation of the gastro-intestinal tract, with common symptoms being abdominal pain, vomiting and diarrhoea. Their most dangerous toxic effects occur when they are aspirated into the lungs causing a chemical pneumonitis. This may occur either during the primary ingestion or when the patient subsequently vomits. **Hydrocarbons are not absorbed by activated charcoal so this should not be given.** Patients who have any signs or symptoms of aspiration pneumonitis (e.g. cough, dyspnea, wheeze) should be given oxygen and admitted for observation. Patients with severe vomiting and diarrhoea may need intravenous hydration. There is no specific antidote for these chemicals.

4.2.5 Alkali ingestion (e.g. bleach)

Gastric decontamination is not indicated in alkali ingestion and vomiting should be avoided. There is no specific antidote; the treatment is supportive only.

Ingestion of an alkaline substance causes damage to the oropharynx and oesophagus. Household bleach (5% sodium hypochlorite) is not a very strongly alkaline substance and is unlikely to cause serious injury. These patients need only symptomatic treatment with intravenous fluids and admission for observation. Stronger alkalis such as drain cleaner may cause severe chemical burns the complications of which include airway obstruction and oesophageal or gastric perforation. These patients should be admitted for rehydration and upper gastro-intestinal endoscopy to determine the extent of the damage.

4.2.6 Oral anticoagulants (e.g. Warfarin, rat poison)

Overdose of these substances causes prolongation of the prothrombin time and increased risk of bleeding.

Patients who have active bleeding or who are at high risk of developing bleeding (e.g. post-operative) should be actively treated. Vitamin K reverses the effect of oral anticoagulants over 12 to 24 hours whereas fresh frozen plasma provides immediate replacement of coagulation factors. In patients not at immediate risk, ceasing Warfarin temporarily until the prothrombin time is in the therapeutic range is all that is required. The potency of rat poisons vary: some may require large doses of vitamin K over several weeks.

a. Treatment in adults

- **Give vitamin K 10 mg intramuscularly daily**

PLUS if necessary (i.e. active bleeding or at high risk because of for example known active peptic ulcer)

- **Give fresh frozen plasma 2 units intravenously and repeat as necessary to a maximum of 8 units using repeated measurement of the prothrombin time as a guide to therapy**

b. **Treatment in children**

- **Give vitamin K 0.3 mg/kg (maximum 10 mg) intramuscularly daily**

PLUS if necessary

- **Give fresh frozen plasma 20 ml/kg (maximum 2 units) intravenously and repeat as necessary using repeated measurement of the prothrombin time as a guide to therapy**

4.2.7 Beta-adrenergic antagonists (e.g. propranolol, atenolol)

Beta-blocker overdose causes bradycardia, AV node block and hypotension, sometimes complicated by bronchospasm, congestive cardiac failure and confusion. Hypoglycaemia may also occur in children. These overdoses may be fatal and patients with significant toxic effects may need a central venous line, ECG monitoring and monitoring in an intensive care unit (if available).

a. Treatment in adults

- **Give adrenaline infusion 10 micrograms/minute and increase by 5 micrograms/minute every 2 minutes until the systolic blood pressure is >90 mmHg, to a maximum of 100 micrograms/minute**

b. Treatment in children

- **Give adrenaline infusion 0.5 micrograms/kg per minute and increase by 5 micrograms/minute every 2 minutes until the systolic blood pressure is >90 mmHg, to a maximum of 100 micrograms/minute**

OR

- **Give isoprenaline infusion 0.5 - 10 micrograms/kg/min**

Glucagon has an inotropic and hyperglycaemic action independent of adrenoreceptors stimulation. Logically it is a better choice as an antidote but currently is not available in Kiribati

4.2.8 Iron

Overdose of iron initially causes vomiting, diarrhoea, abdominal pain and sometimes haematemesis. After a variable quiescent period during which these gastro-intestinal symptoms resolve, the patient may develop shock and hypoglycaemia plus cardiac, hepatic and renal failure. The specific antidote is desferrioxamine but supportive care including intravenous fluid and glucose (if necessary) is important as well. Iron is not well absorbed by activated charcoal.

The following patients should receive desferrioxamine:

- All symptomatic patients
- All patients who have iron tablets visible on a plain abdominal X-ray
- All patients in whom the serum iron level (if available) is greater than 350 microgram/dl (90 micromol/l)

- **Give desferrioxamine 15 mg/kg per hour by intravenous infusion continued until the patient is asymptomatic (usually 12 to 24 hours)**

4.2.9 Benzodiazepines (e.g. diazepam)

These substances are very safe in overdose generally causing only drowsiness. Supportive care and observation is usually all that is necessary.

4.2.10 Nonsteroidal anti-inflammatory dugs (e.g. indomethacin, ibuprofen, aspirin)

Overdose of these drugs causes nausea, vomiting, abdominal pain and drowsiness. Treatment is supportive only.

4.2.11 Phenytoin

In overdose, phenytoin causes cerebellar dysfunction (nystagmus, ataxia, dysarthria, nausea and vomiting) plus confusion, coma and paradoxically, seizures. Treatment is essentially supportive. Diazepam should be used to control seizures.

4.2.12 Aspirin

This commonly used drug can be highly toxic in overdose. The clinical features include gastro-intestinal (nausea, vomiting, haematemesis), neurologic (confusion, coma, seizures) and metabolic (fever, tachypnea and hypokalaemia). Metabolic acidosis and hypoglycaemia may occur in children. Cardiac failure and acute respiratory distress syndrome are uncommon complications. The toxic dose is greater than 150 mg/kg. All symptomatic patients should be admitted and treated as follows:

- **Give 0.9% saline (or 0.3% saline with 3% dextrose in children) intravenously at a rate necessary to maintain a urine output greater than 2 ml/kg per hour**

PLUS

- **Give sodium bicarbonate 1 mmol/kg intravenously every 4 hours to maintain a urine pH greater than 7.5**

PLUS

- **Give potassium chloride 0.25 mmol/kg intravenously over at least one hour, every 4 hours to maintain serum potassium levels > 4 mmol/l**

NOTE: Frequent measurement of urine output, urine pH and serum potassium should be performed (e.g. every 2 to 4 hours). Larger or smaller doses of sodium bicarbonate and potassium chloride than those listed above may be required.

4.2.13 Carbon Monoxide (e.g. car exhaust)

This odourless and colourless gas competes with oxygen for the binding sites on the haemoglobin molecule. Toxic effects include headache, nausea, confusion, coma, seizures and cardiac arrhythmias. Treatment for symptomatic patients is with 100% oxygen for at least 12 hours.

4.2.14 Digoxin

Poisoning with this drug may be acute (usually intentional self-poisoning) or chronic (gradual accumulation in a patient taking Digoxin for therapeutic reasons). Patients with significant toxicity always complain of anorexia, nausea and vomiting. Other clinical features include headache, diarrhoea, visual disorders, confusion and coma. Acute poisoning causes marked hyperkalaemia whereas chronic toxicity cases are often hypokalaemic. Digoxin toxicity has been known to cause just about every type of cardiac arrhythmia from complete heart block to ventricular tachycardia. In addition to the usual supportive care, complications should be treated as follows:

a. Ventricular tachycardia

- **Give phenytoin 15 mg/kg intravenously, infused no faster than 50 mg/min**

PLUS

- **Give magnesium sulphate 50 mg/kg intravenously (maximum dose 5 g) over 5 minutes**

THEN if arrhythmia persists

- **Give Lignocaine 1 mg/kg intravenous bolus**

NOTE: Use cardioversion only as a last resort as it may induce intractable ventricular fibrillation. If it is absolutely necessary then use low energies (e.g. 25 J in an adult).

b. Bradyarrhythmias

- **Give atropine 10 microgram/kg intravenous bolus and repeat in 5 minutes if necessary**

Calcium should not be given to patients with Digoxin toxicity.

c. Hyperkalaemia (serum potassium > 5 mmol/l)

i. Treatment in adults

- **Give short-acting insulin 10 units intravenous bolus**

PLUS at the same time

- **Give 50 ml of 50% glucose intravenously over five minutes**

PLUS

- **Give sodium bicarbonate 100 mmol intravenously over five minutes**

ii. Treatment in children

- **Give short-acting insulin 0.1 units/kg intravenous bolus**

PLUS at the same time

- **Give 50% glucose 2 ml/kg intravenously over 5 minutes**

PLUS

- **Give sodium bicarbonate 2 mmol/kg intravenously over 5 minutes**

NOTE: Serum glucose should be monitored hourly over the next 4 hours. The above treatment for hyperkalaemia may be repeated in 2 hours if necessary.

4.2.15 Barbiturates (e.g. phenobarbitone)

In overdose, barbiturates can cause severe central nervous system depression with coma, hypoventilation and hypotension. Patients with significant ingestions will need intensive supportive care. There is no specific antidote.

4.2.16 Theophylline

Theophylline poisoning may occur because of deliberate ingestion of an overdose or due to gradual accumulation of the drug in those taking it for therapeutic reasons. Toxicity affects several organ systems:

- Cardiovascular – supraventricular and ventricular tachycardia, atrial fibrillation
- Gastrointestinal – nausea, vomiting and diarrhoea
- Neurologic – agitation, confusion, seizures
- Metabolic – hypokalaemia, hyperglycaemia

Seizures may be resistant to treatment with benzodiazepines, and intubation and sedation with barbiturates may be required. Hypokalaemia should be treated with intravenous potassium replacement (see page x), while tachyarrhythmias usually respond to beta-adrenergic antagonists.

For Supraventricular or Ventricular Tachycardia:

- **Give propranolol 0.5 mg intravenous bolus and repeat every 2 minutes up to a maximum of 10 mg**

Note: Propranolol may be contraindicated in patients with asthma.

4.2.17 Chloroquine

This drug is highly toxic in overdose causing hypotension and cardiac arrhythmias. Treatment involves adrenaline and very large doses of diazepam. Most patients will require intubation and management in an intensive care unit (if available).

- **Give adrenaline 0.25 microgram/kg per minute via intravenous infusion and increase rate by 5 microgram per minute until systolic blood pressure is greater than 90 mmHg**

PLUS

- **Give diazepam 2 mg/kg intravenously over 30 minutes**

4.2.18 Verapamil

The minimum toxic oral dose of Verapamil in an adult is about 1 g. Its main effects are upon the heart where it causes hypotension, bradycardia and heart block. The specific antidote is calcium. In addition, bradycardia may require treatment with atropine (see section 1.5) and hypotension may require inotropes (see section 1.2).

- **Give 5 ml of 10% calcium chloride (equals about 1 g of elemental calcium) intravenously over 5 minutes. Further doses may be required according to response and should be continued repeatedly if each dose produces a brief improvement in the cardiovascular status**

4.2.19 Tricyclic antidepressants (e.g. amitriptyline, doxepin, imipramine)

These agents are very dangerous in overdose. The most important toxic effects are on the cardiovascular system (hypotension and ventricular tachycardia), and the central nervous system (coma and seizures). Toxicity is greatly enhanced in the presence of acidosis. Respiratory acidosis due to depressed conscious state may occur and should be promptly treated with intubation and ventilation. Seizures and hypotension may cause metabolic acidosis and also should be promptly treated. All patients should be observed, preferably with cardiac monitoring, for at least 6 hours. Patients who exhibit no signs of toxicity at that time (including sinus tachycardia or QRS widening > 0.12s) can be discharged safely.

a. Seizures

- **Give diazepam 0.1 mg/kg intravenous bolus and repeat in 5 minutes if necessary**

NOTE: Phenytoin is not effective in this situation. If seizures persist despite 2 doses of diazepam then the patient requires intubation and sedation with barbiturates.

b. Hypotension

- **Give 0.9% saline 10 ml/kg intravenous bolus**

THEN if hypotension persists

- **Give sodium bicarbonate 1 mmol/kg intravenous bolus and repeat in 5 minutes if necessary**

NOTE: If hypotension persists despite the above measures then inotropes may be necessary (see section 1.2).

c. Ventricular tachycardia

- **Give sodium bicarbonate 1 mmol/kg intravenous bolus and repeat in 5 minutes if necessary**

THEN if arrhythmia persists

- **Give Lignocaine 1 mg/kg intravenous bolus**

NOTE: If ventricular arrhythmias persist or the patient becomes unstable then treat with synchronized cardioversion (see section 1.1).

4.2.20 Phenothiazines (e.g. chlorpromazine, haloperidol, promethazine, fluphenazine, thioridazine, trifluoperazine)

Drugs in this class cause sedation, hypotension and occasionally torsade de pointes (see section 1.5) in overdose. Anticholinergic symptoms such as dry mouth, sinus tachycardia and urinary retention may occur especially with chlorpromazine and thioridazine. Complications of therapeutic doses include oculogyric crisis (see section 3.3). Treatment is mainly supportive.

4.2.21 Lithium

Acute lithium overdose mainly affects the gastro-intestinal system (nausea, vomiting and diarrhoea) and the central nervous system (tremor, hyperreflexia, ataxia, confusion, seizures and coma). Treatment is supportive and includes hydration with adequate amounts of 0.9% saline intravenously to maintain a good diuresis and enhance lithium excretion.

4.2.22 Ciguatera poisoning

This syndrome is caused by ingestion of certain reef fish (especially *teingo* in Kiribati). It is characterised by nausea and vomiting and dysthesias (especially abnormal temperature perception when placing the hands in water). Other symptoms include headache, myalgia, diarrhoea, tremor, itching and sweating. On examination patients often have a mild sinus bradycardia. Severity varies greatly. Treat dehydration as described in section 6.5.

NOTE: Patient may present with ECG changes that may mimic ischaemic heart disease. For bradycardia and vomiting:

- **Give atropine 10 mcg/kg intravenously or intramuscularly to a maximum of 1.2 mg**

If symptoms have been present for less than 24 hours then mannitol is often useful. Patients must be adequately rehydrated with 0.9% saline prior to administration of mannitol.

- **Give 20% mannitol 1g/kg (200 mLs) intravenously over 30 minutes**
- **Consider promethazine 25mg intramuscularly or 20mg orally for symptomatic treatment**

4.2.23 Paraquat poisoning

Rapid treatment is essential within 4 hours and certainly no longer than twelve hours. However, treatment must not be withheld because a longer period has elapsed.

The prognosis is always poor, with death occurring from pulmonary fibrosis and general system failure usually within a few days.

Steps:

- Give stomach washout
- Give *120g fuller's earth* (2x60 g containers) mixed with 800 mL of water orally or by gastric tube (within four hours of ingestion if possible). If fuller's earth is not available, activated charcoal may be used
- Following this give as a purgative 200 mL (for an adult) of *20% mannitol orally or via a nasogastric tube*
- Repeat administration of fuller's earth and mannitol until the stools are seen to contain Fullers earth. This may take between 4 to 6 hours after the administration of the purgative
- Close clinical and bio-chemical monitoring is necessary
- Delay the use of oxygen (at least 48 hours) as it enhances the toxicity of paraquat
- General supportive measures may include:
  - Use of antibiotics
  - Care of mouth and throat ulcers.

4.3 Poisons Information

You may contact:

New Zealand National Poisons Centre

Office 9am-5pm weekdays:	Tel	643 479 7248
24 hour emergency:	Tel	643 474 7000
	Fax	643 477 0509
	e-mail	poisons@otago.ac.nz

Address: **National Poisons Centre**
Department of Preventive and Social Medicine
University of Otago
POB Box 913
Dunedin
NEW ZEALAND

5 Endocrine Emergencies

5.1 Diabetic Ketoacidosis

Diabetic ketoacidosis (DKA) is a life-threatening complication in patients mostly suffering from type 1 diabetes mellitus. It usually occurs in the setting of an intercurrent infective illness or of missed insulin doses. It is characterised by hyperglycaemia, ketosis and acidosis. Except for mild cases, patients should be preferably managed in an intensive care unit. DKA can be the first presentation of an undiagnosed type 1 diabetes mellitus.

5.1.1 General considerations

Diabetic ketoacidosis (DKA) usually occurs in Type 1 diabetics and is infrequent in Kiribati. The diagnostic features include:

- vomiting
- abdominal pain
- Kussmaul's breathing- deep, sighing respiration similar to that after exercise
- dehydration
- ketotic breath
- mental confusion progressing to coma

It is necessary to test urine for moderate to large ketone bodies. Arterial blood gas is desirable if facilities are available.

DKA might be the first presentation in an unknown type 1 diabetic.

The common precipitating factors of DKA include:

- history of omission of insulin
- drugs, e.g. corticosteroids
- sepsis
- acute coronary event
- recent trauma
- pregnancy

5.1.2 Management

Management should be undertaken urgently in the nearest health care facility.

a. Airway and breathing

All patients should be given oxygen via a face mask. In patients who are drowsy and vomiting, insertion of

a nasogastric tube is recommended to limit regurgitation and aspiration.

b. Intravenous Fluids

Moderate to severe dehydration is always present in DKA. Initially fluid resuscitation should be with large volumes of normal saline. When the blood glucose falls to below 15 mmol/l then the fluid should be changed to 5% dextrose or dextrose-saline.

Administer intravenous infusion of normal saline as follows:

One litre for 30 minutes
One litre for one hour
One litre for 2 hours
One litre for 4 hours

Further infusion should be administered according to clinical assessment of the patient. In children, a paediatrician should be consulted and appropriate fluid management should be administered.

Once the blood sugar is ≤12 mmol/L, change intravenous fluid to either dextrose saline or dextrose 5%.

c. Insulin

- **Intravenous bolus dose of 10 units short-acting insulin followed by short-acting insulin intravenously 4 units/hour either by direct intravenous administration or by using an infusion pump.**

If venous access cannot be established, give:

- **Short-acting insulin intramuscularly 8 units per hour**

Blood sugar should be measured every hour and insulin doses adjusted. Insulin doses can be halved when blood glucose reaches ≤12 mmol/l. Thereafter, insulin can be change to multiple-dose ("QID") insulin regimen subcutaneously followed by twice-daily dosing. If infusion pumps are not available use the microset intravenous giving set used in paediatrics to achieve the required infusion rate.

d. Electrolytes

i. Potassium

Insulin takes glucose and potassium into the cells and their respective serum concentrations fall. A safe and cautious approach is to initiate supplementary intravenous potassium at a rate of no more than 10-20 mmol/hour once insulin and fluids have been started and when **renal function and urinary output** have been assessed as satisfactory.

Measure serum potassium along with serum sodium every 4-6 hours.

ii. Bicarbonate

Sodium bicarbonate should not be given routinely. It is only given when the blood pH is less than 7.0. In such cases, *infuse 50 mmol of sodium bicarbonate over one hour*.

e. Treatment of underlying cause

Treat the underlying cause especially infections.

f. Other measures

An indwelling catheter should be inserted to monitor urine output. Other measures that may be required are: oxygen therapy and insertion of nasogastric tube if paralytic ileus develops.

On recovery, every patient with DKA should be re-educated about avoidance of the complication and the recognition of early warning signs and symptoms.

5.1.3 Special considerations in children (but always contact a paediatrician)

Rehydration is critical. The degree of dehydration should be assessed as follows:

Mild (3% or less) - just clinically detectable.
Moderate (around 6%) - easily detected, reduced skin turgor, poor capillary return.
Severe (10%) - poor perfusion, rapid pulse, reduced blood pressure.

Normal saline is the recommended intravenous fluid for rehydration.

Deficits should be replaced gradually (over 24-48 hours) and **not with rapid infusion** as is appropriate for adults. Tables to guide the rate of fluid replacement according to body weight and degree of dehydration are available at paediatric units of respective divisional hospitals.

5.2 Hyperosmolar, hyperglycaemic state

This is a relatively uncommon event usually occurring as a dramatic presenting feature or as a complication of type 2 diabetes.

It presents with a history of thirst, polyuria and progressive impairment of consciousness commonly in a patient who is 60 years or older. It differs from DKA in that patients in hyperosmolar, hyperglycaemic state do not develop ketoacidosis.

Investigations reveal very high blood glucose, usually higher than 30 mmol/L, the serum sodium is often elevated and the calculated serum osmolality >320 mOsm/l.[1]

The treatment is similar to that in DKA (see above).

Intravenous isotonic saline, low dose intravenous insulin (4-6 units per hour by infusion) and careful attention to serum potassium concentrations are the central strategies. Careful monitoring is required as in DKA.

On recovery, the patient may not need long-term insulin therapy. After an initial period of stabilisation with insulin, most patients with type 2 diabetes who present in a hyperosmolor, hyperglycaemic state can be controlled with oral hypoglycaemic drugs combined with diet.

5.3 Adrenal insufficiency

Adrenal insufficiency is most often due to sudden cessation of long term corticosteroid treatment (10 mg prednisolone/prednisone or greater daily generally for more than 2 weeks). Other causes include Addison's disease, adrenal tumours and meningococcal septicaemia. Clinical features are non-specific and include anorexia, nausea, vomiting, lethargy and postural hypotension. Blood chemistry usually reveals **hyperkalaemia, hyponatraemia** and an elevated urea level. Hypoglycaemia commonly occurs.

5.3.1 Treatment in adults

a. Intravenous fluids

Use normal saline to correct hyponatraemia and dehydration:

- **Give 0.9% saline 1000 ml intravenously over 1 hour**

THEN

- **Give 0.9% saline 1000 ml intravenously over 2 hours**

THEN

[1] Serum osmolality = 2 (Na + K) + urea (mmol/L) + blood sugar (mmol/L).

- **Give 0.9% saline 1000 ml intravenously over 4 hours and repeat as necessary**

b. Corticosteroids

- **Give hydrocortisone 200 mg intravenous bolus then give 100 mg every 6 hours**

5.3.2 Treatment in children

a. Intravenous fluids

Use normal saline to correct hyponatraemia and dehydration:

- **Give 0.9% saline 20 ml/kg intravenously over 1 hour**

THEN

- **Give 0.9% saline intravenously at the rate necessary to correct the estimated fluid deficit plus maintenance requirements over the next 24 hours**

b. Corticosteroids

- **Give hydrocortisone 3 mg/kg intravenous bolus then 1 mg/kg every 6 hours**

If the patient is usually taking steroids, and the usual dose is known, give four times the dose the patient is usually taking.

NOTE: See section 6.5 for calculation of paediatric fluid deficits. Treat hypoglycaemia as described in section 5.4.

5.4 Hypoglycaemia

Hypoglycaemia presents as:

- sweating, tremor, tachycardia and pallor from adrenal and sympathetic activity triggered by the low blood glucose and/or
- hunger, mental confusion, coma and seizures

The factors that precipitate hypoglycaemia include:

- high insulin dose
- high doses of sulphonylureas
- presence of renal failure
- liver disorder
- missed meals
- hormonal disturbances
- vigorous physical activity

Patients should be treated urgently.

If the patient is conscious and able to swallow, give a sugary food or drink followed by foods that are absorbed longer, e.g. crackers.

If the patient is unable to swallow or unconscious at home, give sugar paste or honey into the mouth and transfer immediately to the nearest health care facility for intravenous glucose therapy. At the health care facility, if the patient is unconscious or unable to swallow:

- **Give dextrose 50% 50 ml intravenously followed by continuous intravenous infusion of 5% dextrose for up to 24 hours**

Hypoglycaemia in the elderly, particularly as a consequence of accumulation of sulphonylurea in the plasma, may be difficult to reverse and may recur for several days after stopping the drug.

NOTE: Oral hypoglycaemics have long half lives particularly in renal failure (e.g. 12 hours for

glibenclamide) so relapse of hypoglycaemia may occur if these drugs have been taken in overdose. Admission for observation may be needed. Although the sympathetic signs of hypoglycaemia resolve within minutes of the administration of glucose, the neuroglycopenic features may take up to 30 minutes to resolve completely. Glucagon will not reverse hypoglycaemia if the hepatic glycogen stores are exhausted (e.g. starvation, alcoholism).

5.5 Thyroid Storm

Thyroid storm is diagnosed by the presence of prominent signs of hyperthyroidism plus fever and central nervous system dysfunction (confusion, coma, seizures). It is a **rare** medical emergency requiring prompt intensive treatment.

5.5.1 Airway and breathing

Give high flow oxygen via a face mask. Unconscious patients may need intubation.

5.5.2 Intravenous fluids

Obtain intravenous access. Dehydrated or shocked patients should be resuscitated with 0.9% saline.

5.5.3 Beta-adrenergic antagonists

These drugs antagonise the peripheral effects of thyroid hormone. Cardiac monitoring is desirable.

- **Give propranolol 0.5 mg intravenous bolus every 2 minutes to a maximum of 10 mg using control of tachycardia (pulse <100 beats/minute) as an endpoint**

5.5.4 Antithyroid drugs

- **Give carbimazole 100 mg via nasogastric tube then 20 mg every 8 hours**

5.6 Myxedema coma (Hypothyroid crisis)

Hypothyroid crisis occurs in patients with longstanding hypothyroidism who are exposed to an extra physiological stress that causes decompensation. The stress is usually an intercurrent illness such as pneumonia or stroke. Treatment should be directed at the underlying cause of the decompensation as well as at the hypothyroidism itself. This condition has a very high mortality. Clinically these patients have signs and symptoms of longstanding hypothyroidism plus hypotension, hypothermia, confusion, coma and seizures. Biochemical abnormalities include hyponatraemia and hypoglycaemia.

5.6.1 Airway and breathing

Oxygen should be supplied via a face mask. Intubation and ventilation will be required for comatose patients.

5.6.2 Intravenous fluids

Despite their oedematous appearance, most patients have intravascular fluid depletion. This should be corrected by careful administration of 0.9% saline intravenously. Monitoring of central venous pressure and urine output is essential.

5.6.3 Corticosteroids

Most patients have a co-existing adrenal insufficiency as part of pituitary failure.

- **Give hydrocortisone 200 mg intravenously stat then 100 mg intravenously 6-hourly**

This should be given before the thyroid hormone.

5.6.4 Thyroid hormone

Replacement of thyroid hormone is the definitive treatment for hypothyroidism but it should be done

slowly over several days/weeks.

- **Give thyroxine 5 microgram/kg via nasogastric tube as a single dose then 50 microgram per day rising to 100 microgram daily after 7 days**

NOTE: The dose may need to be reduced in patients suffering from ischemic heart disease as too rapid replacement therapy may precipitate angina or even myocardial infarct.

5.7 Phaeochromocytoma

Phaeochromocytoma is a catecholamine-producing tumour usually located in the adrenal glands. These tumours cause paroxysmal swings in the blood pressure along with sweating, palpitations and headache. Definitive treatment is surgical excision of the tumour but in the emergency situation control of the blood pressure is important.

- **Give labetalol 0.2 mg/kg intravenously and repeat every 10 minutes OR infuse labetalol until the paroxysm ends or the blood pressure is stable**

Definitive treatment involves locating the tumour, controlling the response to the excess of circulating catecholamines (combined alpha and beta blockade) and careful surgical removal.

6 Fluid and Electrolyte Emergencies

6.1 Hyperkalaemia

The organ principally affected by hyperkalaemia is the heart. It can lead to heart block, bradycardia, ventricular fibrillation and asystole. In all cases the cause of the hyperkalaemia should be established and corrected if possible. The treatments below are mostly temporary measures to lower the serum potassium level. A 12-lead ECG and monitoring is desirable.

6.1.1 Mild hyperkalaemia (serum potassium <6.0 mmol/l)

This is best treated with diuresis and administration of an ion-exchange resin. Give fluids and frusemide as required to promote urine flow. This may not initially be possible in patients with acute oliguric renal failure. If indicated:

- **Give resonium 1 g/kg (to a maximum of 30 g) orally or rectally diluted in 50 to 100 ml of water**

6.1.2 Moderate to severe hyperkalaemia (serum potassium >6.0 mmol/l)

Treat as for mild hyperkalaemia and consider giving insulin-dextrose and/or sodium bicarbonate.

a. Treatment in adults

- **Give short-acting insulin 10 units intravenous bolus**

PLUS

- **Give 50 ml of 50% dextrose or glucose intravenously over five minutes**

For persistent hyperkalemia, consider giving:

- **Sodium bicarbonate 100 mmol intravenously over 5 minutes**

For intractable hyperkalemia attributable to renal failure, consider peritoneal dialysis.

b. Treatment in children

- **Give neutral or short-acting insulin 0.1 units/kg intravenous bolus**

PLUS at the same time

- **Give 50% dextrose or glucose 2 ml/kg intravenously over 5 minutes**

PLUS

- **Give sodium bicarbonate 2 mmol/kg intravenously over 5 minutes**

NOTE: Serum glucose should be monitored hourly over the next 4 hours. The above treatment for hyperkalaemia may be repeated in 2 hours if necessary.

If hyperkalemia is accompanied with cardiac arrhythmias, give: calcium.

- **10% calcium chloride 0.2 ml/kg (to a maximum of 10 ml) intravenously over 5 minutes**

Calcium should not be given to patients with Digoxin toxicity.

6.2 Hypokalaemia

Hypokalaemia (serum potassium less than 3.5 mmol/l) causes muscle weakness and cardiac arrhythmias. Levels less than 3.0 mmol/l may be life-threatening. Patients with pre-existing cardiac disease (especially ischaemic heart disease and those on Digoxin therapy) may be at increased risk of cardiac arrhythmias when the serum potassium is less than 4.0 mmol/l.

Intravenous potassium is irritant to veins and may cause cardiac arrhythmias if administered too quickly. It should be given no faster than 0.25 mmol/kg per hour (not more than 20 mmol/hr) except in dire emergencies. For mild deficiencies, oral potassium replacement is acceptable. One 600 mg tablet of potassium chloride contains about 8 mmol of potassium. Intravenous replacement should be combined with oral replacement in moderate or severe deficiency states.

- **Give potassium chloride 0.25 mmol/kg over at least one hour and repeat as necessary. Dilute in 0.9% saline or 5%**

6.3 Hypercalcaemia

Hypercalcaemia causes nausea, vomiting, lethargy, confusion, coma and cardiac arrhythmias (usually heart block). There are a variety of causes. Initial treatment is with intravenous rehydration and frusemide. The aim should be to produce a daily urine volume of about 6 litres in an adult. Close monitoring of fluid status is important especially in elderly patients to avoid over or under-hydration. Subsequent treatment depends upon the cause and may include mithromycin, calcitonin, and corticosteroids. Expert advice should be sought.

Treatment of hypercalcaemia should be based on the ionized calcium rather than the total calcium level. The normal total serum calcium level is 2.1 to 2.6 mmol/l but the ionized fraction will vary with blood pH and serum albumin levels.

- **Give frusemide 0.5 mg/kg intravenously every 4 hours**

PLUS

- **Give 0.9% saline 15 ml/kg intravenously every 4 hours**

NOTE: Adjustment of the frusemide dose may be necessary to avoid dehydration or fluid overload.

6.4 Hypocalcaemia

Hypocalcaemia may cause confusion, seizures, neuromuscular irritability, cardiac failure, heart block and ventricular fibrillation. Symptoms generally do not appear until the serum calcium is less than 2.0 mmol/l.

- **Give 10% calcium chloride 0.1 ml/kg (to a maximum of 5 ml) intravenously over 5 minutes and repeat in 30 minutes if necessary**

PLUS if symptoms fail to resolve

- **Give magnesium sulphate 0.1 mmol/kg intravenously over 5 minutes**

NOTE: Calcium levels should be corrected to account for changes in protein level. The corrected calcium = patient's serum calcium value + [(40 – albumin) x 0.04)].

6.5 Fluid resuscitation

The basic principles of fluid resuscitation are:

- Accurate assessment of fluid deficits
- Replacement with a fluid that approximates the composition of the fluid that was lost
- Continuous reassessment of hydration status

6.5.1 Assessing fluid deficits

Estimation of a fluid deficit is mainly via clinical assessment. Measurement of the blood urea may also be useful but elevation is usually delayed and only occurs with moderate to severe dehydration. Signs of dehydration include reduced tissue turgor, dry skin and mucous membranes, tachycardia, mild fever and reduced urine output. Hypotension and acidosis are late signs and signal the development of hypovolaemic shock.

6.5.2 Selection of replacement fluid

The basic principle is to replace the deficit with a similar fluid. In most cases the fluid lost is similar to the extracellular fluid, containing lots of sodium and chloride. Potassium losses vary. Sometimes fluid with a low sodium content is lost causing a hypernatraemic dehydration. On other occasions sodium rich fluids are lost causing a hyponatraemic dehydration.

No matter what fluid has been lost, the initial treatment for severe dehydration is administration of normal saline. After initial resuscitation, subsequent fluid therapy should be based on measured levels of plasma sodium and potassium.

6.5.3 Reassessment

Ongoing reassessment is necessary, particularly of urine output. The best sign of adequate hydration is a urine output exceeding 0.5 ml/kg per hour.

6.5.4 Hypovolaemic shock

- **Give 0.9% saline 10 ml/kg intravenous bolus and repeat if necessary**

Consider the use of *plasma expanding solution such as haemmacel or gelofusin*

6.5.5 Maintenance requirements each 24 hours

These maintenance requirements are averages only. The actual fluid requirements will vary from patient to patient and with factors such as the presence or absence of fever, and ongoing losses.

a. Adults

Water	-	40 ml/kg
Sodium	-	2 mmol/kg
Potassium	-	2 mmol/kg

b. Children

Water	-	100 ml/kg for the first 10 kg of body weight then 50 ml/kg for the next 10 kg of bodyweight then 20 ml/kg thereafter
Sodium	-	2 to 3 mmol/kg
Potassium	-	2 mmol/kg

7 Miscellaneous Emergencies

7.1 Anaphylaxis

Allergic reactions may be triggered by a variety of factors including drugs (e.g. penicillin), foods (e.g. shellfish), insect stings and chemicals. There is a wide spectrum of severity ranging from a harmless skin rash (urticaria), to potentially fatal airway obstruction (laryngeal oedema) and full-blown anaphylaxis (hypotension, bronchospasm). Anaphylaxis is much more common in adults than children.

The mainstays of treatment are oxygen, adrenaline and intravenous fluid. Steroids may prevent relapse and anti-histamines provide some relief of urticarial itch but these drugs do nothing for the life-threatening features of acute severe anaphylaxis.

7.1.1 Treatment in adults

a. Airway and breathing

Administer high flow oxygen via face mask. Administer a bronchodilator.

- **Give Salbutamol 5 mg via nebuliser**

b. Adrenaline

If severe (hypotension or severe bronchospasm or stridor or hypoxia):

- **Give adrenaline 0.5 mg intramuscularly and repeat in 5 minutes if required**

OR

If less severe (systolic blood pressure greater than 90 mmHg, mild bronchospasm, no stridor and no hypoxia):

- **Give adrenaline 100 mcg intravenously each minute until symptoms subside**

c. Intravenous fluids

- **Give 0.9% saline 250 ml intravenous bolus and repeat if necessary**

NOTE: Large volumes of intravenous fluid may be necessary to maintain an adequate blood pressure in severe anaphylaxis.

d. Corticosteroids

- **Give hydrocortisone succinate 200 mg intravenously THEN 100 mg six hourly**

OR

- **Give dexamethasone 8 mg intramuscularly**

OR

- **Give prednisolone 50 mg orally daily**

NOTE: All patients with significant anaphylaxis should be observed for 24 hours as relapse may occur.

e. Anti-histamines

- **Give promethazine 25 mg intramuscularly followed by either 25mg intramuscularly or 20mg orally three times daily**

f. Other Issues

- Proper documentation of all cases
- Patient and relatives should be educated to avoid subsequent episodes

- Patient may be provided with a medic alert bracelet
- Some patients may require adrenaline syringes for home use

7.1.2 Treatment in children

a. Airway and breathing

Administer high flow oxygen via a face mask. Bronchodilators reduce bronchospasm.

- **Give Salbutamol 2.5 mg via nebuliser in children 5 years of age or under, for children older than 5 years give Salbutamol 5mg via nebuliser**

b. Adrenaline

- **Give adrenaline 10 mcg/kg mg IV over 1 minute and repeat in 5 minutes if required**

NOTE: If intravenous access is not available then adrenaline may be given via the intramuscular route.

- **Give adrenaline 10 mcg/kg intramuscularly**

c. Intravenous fluids

- **Give 0.9% saline 10 ml/kg bolus intravenously and repeat as necessary**

d. Corticosteroids

- **Give hydrocortisone succinate 4 mg/kg intravenously**

OR

- **Give dexamethasone 0.2 mg/kg intramuscularly**

OR

- **Give prednisolone 1 mg/kg orally**

NOTE: All patients with significant anaphylaxis should be observed for 24 hours as relapse may occur.

e. Anti-histamines

- **Consider the use of promethazine**

f. Other Issues

- Proper documentation of all cases
- Patient and relatives should be educated to avoid subsequent episodes
- Patient may be provided with a medic alert bracelet
- Some patients may require adrenaline syringes for home use

7.2 Pre-eclampsia

Pre-eclampsia is a poorly understood condition that affects women in the second half of pregnancy. It is characterised by hypertension (blood pressure > 140/90) and proteinuria. Other features include oliguria, peripheral oedema, pulmonary oedema, hepatitis, thrombocytopenia and haemolysis. Neurologic features include headaches, visual disturbances, hyperreflexia and seizures. When seizures occur the condition is called **eclampsia**.

Mild pre-eclampsia can be treated with bed rest only. Severe pre-eclampsia however is a very serious disorder which poses a significant risk to both the mother and foetus. Definitive treatment is delivery of the foetus and early consultation with an obstetrician is essential. Signs of severity include a blood pressure of more than 160/110 mmHg or the development of neurologic symptoms or pulmonary oedema or oliguria.

7.2.1 Treatment of severe pre-eclampsia

a. Airway and breathing

Give high flow oxygen via a face mask and obtain intravenous access.

b. Hypertension

Mild elevations in blood pressure often respond to magnesium sulphate. If the blood pressure remains high then it should be gently lowered over several hours to a level of 140/90 mmHg.

- **Give hydrallazine 5 mg intravenously and repeat every 20 minutes to a maximum of 40 mg**

c. Seizures

i. Seizure prevention

Magnesium sulphate reduces the blood pressure and helps prevent seizures.

- **Give magnesium sulphate 4g intravenously over 10 minutes then commence an infusion at a rate of 2g/hour**

NOTE: It is important to monitor magnesium levels during treatment. Clinically, hypermagnaesaemia causes loss of deep tendon reflexes and a reduction in the respiratory rate. If the patient becomes hyporeflexic or the respiratory rate falls below 8 per minute then the infusion should be ceased temporarily. If biochemical estimation of the serum magnesium level is available then this should be measured frequently, aiming for a level of between 2 and 4 mmol/l. Magnesium is superior to phenytoin which was previously used for seizure prophylaxis.

ii. Seizure management

If seizures occur despite administration of magnesium sulphate then conventional anti-convulsants should be used.

- **Give diazepam 5 mg intravenous bolus and repeat every 5 minutes to a maximum of 20 mg**

PLUS if seizures persist

- **Give phenytoin 15 mg/kg intravenously over 20 minutes**

NOTE: If seizures persist despite adequate levels of magnesium sulphate and phenytoin then the patient will require intubation and ventilation and the administration of a thiopentone infusion.

d. Fluid balance

Insertion of a urinary catheter to monitor urine output is essential. A central venous catheter may also be useful. Careful administration of crystalloid solutions is necessary to maintain urine output while avoiding pulmonary and cerebral oedema associated with over-hydration.

7.3 Septic Shock

Septic shock is a serious complication of bacterial septicaemia. Hypotension in this condition is a result of a combination of abnormal vasodilation, hypovolaemia and impaired cardiac function. **It has a very high mortality and requires intensive treatment, preferably in an intensive care unit.** The most important aspect of management of septic shock is of course treatment of the infection with antibiotics.

Other supportive therapy may include treatment of gastrointestinal haemorrhage, renal failure and disseminated intravascular coagulation (DIC).

7.3.1 Maintain airway and breathing

The usual manoeuvres to maintain an adequate airway and adequate ventilation, up to and including endotracheal intubation should be used. All patients should at least receive high flow oxygen via face mask.

- Give oxygen to maintain an arterial oxygen saturation greater than 95%

7.3.2 Optimise intravascular volume

Insertion of a central venous line is useful as it allows accurate measurement of central venous filling

pressures and also makes administration of inotropic agents safer. Correct anaemia with administration of blood or otherwise use boluses of normal saline to achieve an optimal central venous pressure.

- **Give 0.9% saline boluses of 100 ml intravenously to obtain an optimal central venous filling pressure**

7.3.3 Inotropic agents

The inotropes most useful in septic shock are dopamine and adrenaline. These agents increase cardiac output and act as vasoconstrictors. They should only be used once patients are well hydrated.

a. Treatment in adults

- **Give dopamine 2 microgram/kg per minute by intravenous infusion and increase rate by 1 to 2 microgram/kg per minute every 5 minutes to a maximum of 20 microgram/kg per minute**

OR

- **Give adrenaline 2 microgram/minute by intravenous infusion and increase rate by 1 to 2 microgram per minute every 5 minutes to a maximum of 20 microgram/minute**

OR

- **Add 3mg of adrenaline in 100ml of normal saline and infused at a rate 5 – 10 macro drops/min using an IV chamber**

b. Treatment in children

- **Give dopamine 2 microgram/kg per minute by intravenous infusion and increase rate by 1 microgram/kg per minute every 5 minutes to a maximum of 20 microgram/kg per minute**

NOTE: Ideally, inotropic agents should be infused via a central venous line. Otherwise a large peripheral vein (such as the femoral vein or the cubital veins) should be used.

7.4 Acute Psychosis

The acutely confused and agitated patient can present a diagnostic and management problem. Often a combination of temporary sedation and gentle physical restraint is necessary to allow the patient to be examined and assessed. However, it is essential that the underlying cause of the confusion is found and treated. Sedation is not a treatment in itself.

Important and treatable causes of confusion include hypoxia, hypercapnia, drug overdoses, head injuries, infections, hyponatraemia and many other medical problems. Sedated patients should be closely observed.

- **Give haloperidol 2.5 to 10 mg intramuscularly and repeat in 10 minutes if necessary**
- **With the first dose give promethazine 50mg by i.m. injection**

OR

- **Give diazepam 5- 10mg intravenously and repeat 30 minutes later if required-this may require restraining the patient and may not be as appropriate as intramuscular injection**

OR

- **Give midazolam 2.5 to 5 mg intramuscularly or intravenously and repeat in 5 minutes if necessary**

NOTE: Use the lower doses in the elderly or those with body weights less than 50 kg.

8 Emergency Drugs

This section is intended as a brief guide to some of the drugs mentioned in this book. It is by no means exhaustive and does not cover all contraindications or dosage alterations in special situations. Information about these areas should be obtained elsewhere. The dosages below are guides only. Drug therapy should always be adjusted to the individual patient situation.

8.1 Local Anaesthetics

Local anaesthetic agents are used to provide anaesthesia by local wound infiltration, nerve blocks or regional techniques. Side-effects from systemic absorption include seizures and cardiac arrhythmias. Avoid accidental intravenous administration by aspirating for blood prior to injection.

Lignocaine is the agent of choice for local wound infiltration. A concentration of 2% should be used for digital nerve blocks and any other area where a smaller volume of anaesthetic is desirable. The 1% solution is suitable for local infiltration of most wounds.

Lignocaine with adrenaline should not be used on the extremities i.e. nose, fingers, toes, ears or penis. The addition of adrenaline helps control bleeding and also extends the duration of anaesthesia.

8.2 Sedatives and Induction Agents

These drugs are used to depress the conscious state either for sedation or general anaesthesia. They should be used with great care as unconscious patients are unable to protect their airway and because of the risk of causing hypoventilation and hypotension.

Ketamine has anaesthetic and analgesic properties and is less likely to produce hypotension than other sedative agents. It also has a mild bronchodilator action which makes it useful when anaesthetising patients with asthma. Adverse effects include increased salivation, laryngospasm, raised intracranial pressure and unpleasant hallucinations after recovery of consciousness. Ketamine should not be used in patients at risk of raised intracranial pressure (e.g. head injuries, meningitis) or in patients with ischaemic heart disease. It should not be given unless personnel and facilities are available to protect the patient's airway.

Ketamine should be given as an intravenous bolus. The usual dose is 2 mg/kg although some patients may require larger amounts. Onset of action is within 60 seconds and duration is about 20 minutes.

Thiopentone is a short acting barbiturate. Its main adverse effects relate to cardiorespiratory depression. It should be used with great care in patients who are hypotensive and should not be given unless personnel and facilities are available to protect the patient's airway. It is a potent anticonvulsant and is the drug of choice for anaesthetising patients with status epilepticus.

Thiopentone should be given as an intravenous bolus. The usual dose is 3 to 5 mg/kg. The lower dose should be used in the elderly. Onset of action is about 30 seconds and its effects last 5 to 10 minutes.

Midazolam is a short acting benzodiazepine. It has powerful amnestic properties and produces less cardiorespiratory depression than thiopentone. It is a fairly safe and useful anaesthetic induction agent in the critically ill and is also used to sedate children and agitated patients.

Midazolam can be given intramuscularly or as an intravenous bolus. In children, intranasal midazolam is a useful premedication prior to suturing. The onset of action of intravenous midazolam is 1 to 2 minutes and the effects of a single dose last from 60 to 90 minutes. Intranasal midazolam has its maximal effect within 10 minutes and lasts up to 2 hours.

In ADULTS: The intravenous dose is 2.5 to 15 mg. The dose should be titrated to its effect. Smaller doses are usually required for the elderly, whereas alcoholics may require higher doses. The intramuscular dose is 5 to 10 mg.

In CHILDREN: The intravenous dose is 0.15 to 0.3 mg/kg up to 0.5 mg/kg. The intranasal dose is 0.2 to 0.4 mg/kg (to a maximum of 5 mg), slowly dropped into alternate nostrils over 15 seconds.

8.3 Anticholinergics

Anticholinergic drugs block the effects of acetylcholine at muscarinic receptors. The most commonly used anticholinergic agent is atropine. This drug is used for the treatment of bradycardia due to increased vagal tone, to block the cholinergic effects of drugs such as suxamethonium in children, and to reverse some of the adverse effects of anticholinesterase poisoning. Ipratropium is used in the treatment of asthma and benztropine is useful in the treatment of oculogyric crisis.

Atropine should be used with care in patients with ischaemic heart disease as it may cause a marked sinus tachycardia. The usual adult dose is 0.6 mg intravenously as a bolus, repeated in 5 minutes if necessary. A dose of 3 mg will produce complete blockade of muscarinic cholinergic receptors in an adult.

Much larger doses are used in anticholinesterase poisoning. Atropine may be given via the endotracheal tube in an emergency. Use twice the normal intravenous dose and dilute in 10 ml of 0.9% saline. The paediatric dose is 20 microgram/kg (to a maximum of 0.5 mg). The onset of action is within 5 minutes and the duration of action is 2 to 4 hours.

Benztropine is used in the treatment of oculogyric crisis. Overdose of benztropine can cause central anticholinergic syndrome (confusion, hallucinations). The usual dose in adults is 1 to 2 mg orally or intramuscularly. Children should be given 20 microgram/kg. Its duration of action is shorter than most of the drugs that cause oculogyric crisis so a repeat oral dose should be given four hours after the initial dose.

8.4 Opioid Analgesics

Opioid agents are mainly used for their analgesic and sedative actions. The main side effects of these drugs are respiratory depression, hypotension and nausea. In addition, morphine can cause spasm of the biliary tree and ureter so it should not be given to patients with biliary or ureteric colic.

NOTE: The use of parenteral opioid drugs is not recommended in patients with chronic or recurrent painful conditions such as migraine or back pain due to the risk of addiction.

Morphine is usually used in the treatment of acute myocardial infarction and pulmonary oedema. As an analgesic, it may produce less dysphoria than pethidine. The usual dose in adults is 2.5 mg intravenous bolus repeated every few minutes to a maximum of 15 mg. The usual dose in children is 0.05 mg/kg given intravenously every five minutes to a maximum of 0.2 mg/kg. The duration of action is about 3 hours.

Pethidine is mainly used as an analgesic. This is a highly addictive drug even after a few doses. The best method of administration is incremental intravenous boluses of 0.5 mg/kg every 5 minutes to a maximum of 3 mg/kg. Alternatively, a dose of 2 mg/kg may be given intramuscularly. The duration of action is 2 to 3 hours.

Fentanyl is a short acting narcotic used to sedate patients prior to painful procedures or intubation (often in combination with midazolam). The usual dose of fentanyl is 1 microgram/kg. The duration of action is 30 to 40 minutes.

8.5 Anti-emetics

Anti-emetic drugs are used for the temporary relief of nausea and vomiting.

Metoclopramide (brand name: Maxolon) should not be given to children less than 16 years of age due to the high incidence of acute dystonic reactions. It should also not be given to patients with bowel obstruction. Metoclopramide is useful in the treatment of migraine and may also help the passage of calculi in renal colic. The usual dose is 10 mg by intravenous bolus or intramuscular injection. Males weighing more than 70 kg may require 15 or 20 mg. Females with low body weight or the elderly should be given 5 mg initially.

Prochlorperazine (brand name: Stemetil) is also useful for the treatment of vertigo as well as nausea and vomiting. It should not be given to children less than 16 years of age. The oral dose is 5 mg 8-hourly. The intramuscular or intravenous dose is 12.5 mg every 8 hours.

Promethazine (brand name: Phenergan) is a weaker antiemetic than prochlorperazine and is more sedating. It can be given intravenously, intramuscularly or orally. The usual dose is 0.5 mg/kg.

8.6 Corticosteroids

Although very useful in the treatment of asthma, anaphylaxis and many other conditions, the beneficial effects of these drugs are delayed for several hours at least. They should be used with care in patients with diabetes or peptic ulcer disease. All agents have similar anti-inflammatory effects but differ in their mineralocorticoid potency. Their mineralocorticoid (or aldosterone-like) effects may be undesirable and include sodium retention, oedema and hypokalaemia.

Equivalent anti-inflammatory doses are:

100 mg hydrocortisone = 25 mg prednisolone = 4 mg dexamethasone

Hydrocortisone has marked mineralocorticoid effects. It should be given intravenously over 5 minutes. The usual dose is 3 mg/kg every 8 hours.

Dexamethasone has virtually no mineralocorticoid effects. It can be given intramuscularly or intravenously. The usual dose is 0.1 mg/kg every 8 hours.

Prednisolone has moderate mineralocorticoid effects. It is given orally. The usual dose is 1 mg/kg daily to a maximum of 50 mg.

8.7 Anti-epileptics

The first line drug in the treatment of epilepsy is diazepam. Phenytoin is useful for the treatment of idiopathic epilepsy but is less effective for seizures due to other causes. Barbiturates such as phenobarbitone are powerful anti-epileptics but also cause cardiorespiratory depression and marked depression of conscious state often necessitating intubation and ventilation.

Diazepam is a safe and effective agent for the termination of seizures. It may be given intravenously or rectally. The oral route is too slow in an emergency, whereas the intramuscular route is painful and unpredictable. The onset of action when given intravenously is 1 to 2 minutes, whereas the rectal route may take 5 to 10 minutes to have its full effect. The usual intravenous dose is 0.1 mg/kg repeated every 5 minutes if required. The usual rectal dose is 0.5 mg/kg.

Phenytoin must be given by slow intravenous injection. The infusion rate should not exceed 50 mg per minute in adults or 1 mg/kg per minute in children. The drug should be diluted in 0.9% saline only (not 5% dextrose) so that the concentration is no greater than 5 mg/ml. Rapid infusion of concentrated solutions may cause hypotension. The usual loading dose is 15 mg/kg intravenously.

8.8 Antiarrhythmics

8.8.1 Vaughan-Williams classification system

Antiarrhythmics are usually divided into groups using the Vaughan-Williams classification system.

Class I

These drugs reduce the conduction velocity of the myocardium. They are further subdivided according to their effect on the action potential duration:

1A. Quinidine

These drugs prolong the action potential duration. None of them are currently used in the emergency treatment of arrhythmias.

1B. Lignocaine, Phenytoin

These drugs shorten the action potential duration. Lignocaine is the drug of first choice in the treatment of ventricular tachycardia. Phenytoin is used in the treatment of ventricular tachycardia due to Digoxin toxicity.

Lignocaine should be given intravenously over one minute in a dose of 1 mg/kg. A further 0.5 mg/kg may be given after 5 minutes if necessary. Lignocaine has a very short half life so the intravenous bolus should be followed by an infusion at a rate of 40 microgram/kg per minute. Adverse effects include confusion, coma, seizures and heart block but are not often encountered. Elderly patients may require lower infusion rates.

Phenytoin is given intravenously in a dose of 5 mg/kg over 5 minutes.

Class II

This class comprises the beta-adrenergic antagonists. **Propranolol** is occasionally used to delay conduction through the AV node in the treatment of supraventricular tachycardia or atrial fibrillation. It also may be of benefit in the treatment of ventricular tachycardia due to theophylline overdose. Propranolol should not be used in patients with left ventricular failure, asthma or bradyarrhythmias. The dose should be titrated according to effect. Give 10 microgram/kg intravenously every 2 minutes to a maximum of 100 microgram/kg.

Class III

Drugs in this class share the effect of prolonging the action potential duration. **Amiodarone** is used in the treatment of both ventricular and atrial arrhythmias. At least some of its therapeutic effects are often delayed for up to 24 hours. Amiodarone is not a negative inotrope and is well tolerated by patients with cardiac failure. Intravenous administration is occasionally associated with hypotension due to vasodilatation. The usual loading dose of amiodarone is 5 mg/kg given intravenously over 30 minutes.

Class IV

This class includes the calcium channel antagonists. They act to depress sinus node automaticity and AV node conduction. These drugs are powerful negative inotropes and should be used with great care in patients with impaired left ventricular function.

Verapamil should not be given to children less than two years of age. It should not be given to patients with left ventricular failure, bradycardia or hypotension. If the systolic blood pressure falls below 95

mmHg then ephedrine should be given. The effectiveness of vagal manoeuvres is increased following the administration of Verapamil. The usual dose in adults is 1 mg intravenous boluses every minute to a maximum of 10 mg.

8.8.2 Other antiarrhythmics

Digoxin is used to control the ventricular rate in atrial fibrillation. It is contraindicated in atrial fibrillation associated with Wolff-Parkinson-White syndrome. The main advantage Digoxin has over other drugs available to control conduction through the AV node, is the fact that it is not a negative inotrope. Most patients with atrial fibrillation have significant underlying cardiac disease and often tolerate poorly the myocardial depressant effects of Verapamil and propranolol, making Digoxin the safest choice. The effects of Digoxin are increased in the presence of hypokalaemia, hypothyroidism, hypomagnesaemia and hypercalcaemia. Digoxin should not be given to patients with bradycardia. The maximum therapeutic effects of Digoxin are delayed by 6 to 24 hours after administration. The usual loading dose is 10 microgram/kg given intravenously over 20 to 30 minutes.

8.9 Anti-hypertensives

Several different drugs are available for the management of hypertensive emergencies.

Glyceryl trinitrate is predominantly a direct acting venodilator. At higher doses (i.e. at infusion rates greater than 100 microgram per minute) it is an arteriodilator as well. Its onset of action is almost immediate and it has a half life of less than 5 minutes and for this reason it is best given as an infusion. The anti-hypertensive effects of sublingual or topical glyceryl trinitrate are unpredictable. Side effects include sinus tachycardia, nausea and headache.

Hydrallazine is a direct acting arteriodilator. It should not be used in patients with ischaemic heart disease. Side effects include nausea, tachycardia and headache. Peak effects are not seen for 10 to 20 minutes after intravenous injection and it has duration of action of 4 to 8 hours. The usual dose in adults is 10 mg intravenously every 20 minutes to a maximum of 50 mg.

Nifedipine is an arterial dilator with unpredictable effects when given orally or sublingually and **should not be used in hypertensive emergencies**.

Labetalol, a combined alpha and beta-receptor blocking drug has been requested for the Kiribati EDL. It is for intravenous use only in hypertensive emergencies

8.10 Inotropic Agents

Inotropic agents are used in the treatment of cardiogenic and distributive shock. All should be infused via a large vein.

Adrenaline is an alpha and beta adrenergic agonist. It causes an increase in cardiac output and heart rate plus vasoconstriction. It is given by infusion into a large vein at a rate of 1 to 70 microgram per minute titrated to effect.

Dopamine has similar effects to adrenaline but produces more tachycardia at higher doses. It may preferentially enhance renal blood flow at rates less than 5 microgram/kg per minute.

Dobutamine (not currently available in Kiribati) has positive chronotropic and inotropic effects which are balanced by a mild degree of vasodilation so that myocardial oxygen demand is generally not increased. Dobutamine is the inotrope of choice in patients with myocardial ischaemia. The usual dose range is 2 to 20 microgram/kg per minute.

8.11 Diuretics

Frusemide is a potent loop diuretic used in the treatment of fluid overload. Its main side effects are hypokalaemia and fluid depletion. Damage to the inner ear may occur with too rapid intravenous injection. Frusemide is ineffective in the acute treatment of hypertension and should not be used except as an adjunct to other more powerful drugs.

Intravenous frusemide has an onset of action within 5 minutes, a peak effect at about 30 minutes, and duration of action of 2 hours. Dosage varies according to renal function; most patients without renal impairment will have a significant diuresis after 40 mg given intravenously. Doses in excess of 250 mg may be required to diurese patients with severe renal failure.

Frusemide should not be given intravenously at a rate faster than 40 mg per minute. The absorption of intramuscular frusemide is unpredictable and this route of administration should not be used.

8.12 Muscle Relaxants

Muscle relaxants are divided into two groups, depolarising and non-depolarising (or reversible).

Suxamethonium is a depolarising muscle relaxant. It is given as an intravenous bolus and has its maximal effect within 60 seconds. The duration of paralysis is usually about 5 minutes. Some patients with an atypical plasma cholinesterase enzyme will be paralysed for much longer. The usual dose is 1 to 1.5 mg/kg in adults and 2 mg/kg in children. Suxamethonium is contra-indicated in the presence of hyperkalaemia, lower motor neurone diseases and between 3 days and 2 years after major burns. In the absence of these contra-indications suxamethonium is the drug of first choice for muscle relaxation in rapid sequence intubation.

Vecuronium is a non-depolarising muscle relaxant. Given as an intravenous bolus it has its onset in about 3 minutes and lasts 20 to 30 minutes. The usual dose is 0.1 mg/kg.

8.13 Neuroleptics

Haloperidol is the safest neuroleptic to use for sedation. It may be given intramuscularly or intravenously. The usual intramuscular dose in adults is 2.5 mg repeated every 5 minutes to a maximum of to 10 mg.

Chlorpromazine is more likely to cause hypotension than haloperidol.

8.14 Anti-asthma Drugs

Salbutamol is a beta-adrenergic agonist. It is best given in the inhaled form (either via an inhaler or a nebuliser). Oral Salbutamol is ineffective and should not be used. Intravenous Salbutamol may be used for severe asthma where marked airway obstruction may prevent the inhaled form of the drug from reaching the distal airways. The main side-effects of Salbutamol are sinus tachycardia, tremor and anxiety.

Nebulised Salbutamol may be given as often as necessary, even up to continuously. It is not possible to overdose a patient with nebulised Salbutamol. The usual dose is 5 mg in adults and older children, and 2.5 mg in children less than 5 years old. The dose should be diluted up to 2 ml using 0.9% saline but may be given neat.

Intravenous Salbutamol is given in a dose of 5 microgram/kg (up to a maximum of 250 microgram) over 1 to 2 minutes and repeated once 15 minutes later if necessary.

Corticosteroids are discussed in section 8.6.

Aminophylline is a xanthine derivative that has been used for many years in the treatment of asthma. However it is a weak bronchodilator and has no additional benefit over optimal doses of Salbutamol. It has a very narrow therapeutic margin. Adverse effects include ventricular tachycardia, seizures and hypokalaemia. Its use should be restricted to severe asthma.

8.15 Intravenous Fluids

0.9% saline contains 154 mmol/l of sodium chloride. It is essentially isotonic and iso-osmolar, and is distributed to the extracellular fluid space. It is the fluid of first choice in the treatment of hypovolaemia. The usual dose is 10 ml/kg as an intravenous bolus repeated if necessary. 0.9% saline contains too much saline to be used as the sole maintenance fluid, although it may be alternated with 5% dextrose.

5% dextrose contains 50 g/l of dextrose. It is distributed to the total body water space and is thus not suitable for emergency rehydration. Although it can be used as the sole maintenance fluid in the short term, prolonged administration of 5% dextrose alone may cause hyponatraemia, especially in children.

3% dextrose with 0.3% saline contains 51 mmol/l of sodium chloride and 30 g/l of dextrose. Its primary use is as a maintenance fluid (with potassium) in children. It may be suitable for rehydration of patients with mild or moderate dehydration.

Hartmanns solution contains a mixture of ions similar to that of the extracellular fluid. It may be substituted for 0.9% saline except in the presence of hyperkalaemia or alkalosis. It contains 140 mmol/l of sodium, 109 mmol/l of chloride, 29 mmol/l of bicarbonate, 5 mmol/l of potassium and 2 mmol/l of calcium.

Plasma volume expanding solution (e.g. Haemmacel, Gelofusin): Colloids can be used for patients with hypovolaemic shock in association with crystalloid solutions.

8.16 Tetanus Prophylaxis

Regular immunisation with tetanus toxoid is the best way to prevent death due to tetanus. Children should receive doses of tetanus toxoid at 2, 4 and 6 months of age then booster doses at 5 years. Thereafter, booster doses of tetanus toxoid should be given every 10 years.

Patients presenting with a skin wound should be treated as described below. A non-immune patient is one who has never received a full course of tetanus toxoid injections. Tetanus prone wounds include puncture wounds, contaminated or infected wounds and crush wounds.

8.16.1 Non-immune patient with tetanus prone wound

- **Give tetanus toxoid 0.5 ml intramuscularly and complete course (with repeat tetanus toxoid injections at 6 weeks and 6 months)**

PLUS

- **Give tetanus immune globulin 250 units intramuscularly at a different site than that of the tetanus toxoid injection**

8.16.2 Non-immune patient with clean wound

- **Give tetanus toxoid 0.5 ml intramuscularly and complete course (with repeat tetanus toxoid injections at 6 weeks and 6 months)**

8.16.3 Immune patient with tetanus prone wound

If more than 5 years since last tetanus toxoid booster then

- **Give tetanus toxoid 0.5 ml intramuscularly**

8.16.4 Immune patient with clean wound

If more than 10 years since last tetanus toxoid booster then

- **Give tetanus toxoid 0.5 ml intramuscularly**

8.17 Drugs Used in Cardiac Arrest

Adrenaline is a powerful endogenous catecholamine. The pharmacologic doses used in cardiac arrests far exceed the amounts usually produced by the adrenal glands. Adrenaline has both alpha and beta adrenergic agonist effects. It stimulates myocardial contraction, increases the heart rate and raises the blood pressure. Its most dangerous adverse effect is induction of ventricular arrhythmias, an effect which is far more likely when the myocardium is sensitized to catecholamines. This occurs with myocardial ischaemia (which adrenaline can also induce by increasing myocardial work), and with overdoses of drugs such as amphetamines and cocaine. In the setting of a cardiac arrest, the induction of ventricular arrhythmias is obviously not a problem and a large intravenous bolus doses should be given. However, in situations other than cardiac arrest, such as anaphylaxis or asthma, adrenaline should be used with great care to avoid worsening the patient's condition. In these circumstances, adrenaline is best given intravenously in small carefully titrated doses.

The absorption of adrenaline given by the subcutaneous or intramuscular routes is unpredictable especially in shock states. In ventricular fibrillation or asystole adrenaline should be given in doses of 10 to 100 micrograms/kg by intravenous bolus. Administration should be by a central line if already present or by a large peripheral vein and followed by a 20 ml 0.9% saline flush to ensure it rapidly reaches the central circulation. If there is no intravenous access then adrenaline can be given via the endotracheal tube. When given by this route it should be diluted in 10 ml of 0.9% saline and the dose should be 5 times the intravenous dose. There is no role for intracardiac injection of adrenaline (or any other drug).

Sodium bicarbonate is used to treat the metabolic acidosis associated with cardiac arrest. Its effectiveness has not been proven and it has many potential adverse effects. Most authorities recommend that it be given in prolonged cardiac arrests (i.e. those greater than 10 minutes in duration). It should be given earlier in the presence of acute renal failure, hyperkalaemia or tricyclic antidepressant overdose. The usual dose is 1 mmol/kg intravenously over 1 to 2 minutes.

Lignocaine is recommended for the treatment of ventricular fibrillation and ventricular tachycardia. Its effectiveness has not been proven but it is unlikely to be harmful. The usual dose is 1 mg/kg intravenously given over 1 minute. Lignocaine has a short therapeutic half-life so a successful bolus dose should be followed by a Lignocaine infusion. Lignocaine may be given via the endotracheal tube if intravenous access is unavailable. Twice the intravenous dose is used and it should be diluted in 10 ml of 0.9% saline.

Atropine is used in the treatment of asystole and severe bradycardia. It acts to block the effects of the vagus nerve on the heart. A dose of 3 mg in an adult produces complete atropinization, blocking all the cholinergic receptors. The main adverse effect of atropine is to produce a sinus tachycardia which may be harmful in the presence of ischaemic heart disease. The usual total dose of atropine used in cardiac arrest is 40 microgram/kg which may be given as a single bolus or in several divided doses a few minutes apart.