Republic of Kenya

MINISTRY OF HEALTH

KENYA NATIONAL GUIDELINES FOR CARDIOVASCULAR DISEASES MANAGEMENT

DIVISION OF NON-COMMUNICABLE DISEASES
MINISTRY OF HEALTH

HAND BOOK

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Introduction

Cardiovascular dieases are the leading cause of death globally. In Kenya, approximately 25% of hospital admissions and 13% of deaths are due to CVD.
These guidelines are recommended for use by policy makers, program designers, implementers of NCD interventions, health care workers, community educators and teaching institutions.
All levels of care in our health system have a responsibility to reduce the burden of CVD and their interaction is demonstrated in the figure below.

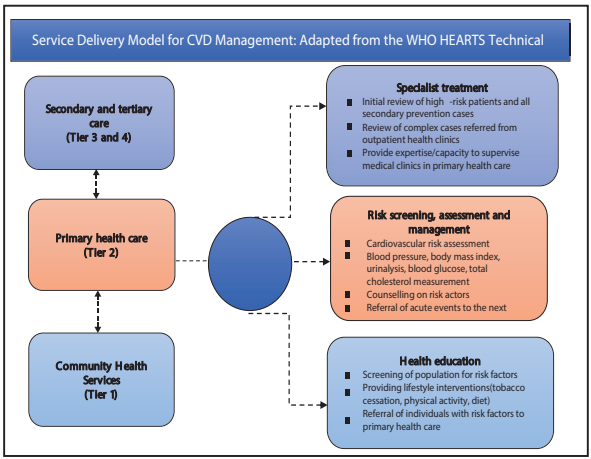


Figure 1: Service delivery model for CVD.
(Adapted from WHO, HEARTS Package)

Prevention of Atherosclerotic Cardiovascular Disease

Etiology and Risk Factors

The table and figure below summarise the risk factors and causation pathway for development of CVDs.

Table 1: Risk factors for CVD

Modifiable Risk factors	Non-modifiable Risk factors
• Tobacco use and exposure to tobacco smoke • Unhealthy diet • Overweight/ obesity, • Physical inactivity • Harmful use of alcohol, • Hypertension, • Diabetes and hyperlipidemia • Infections e.g. Rheumatic fever/heart disease, HIV	• Sex • Age • Race, • Family history

Causation pathway for CVD.

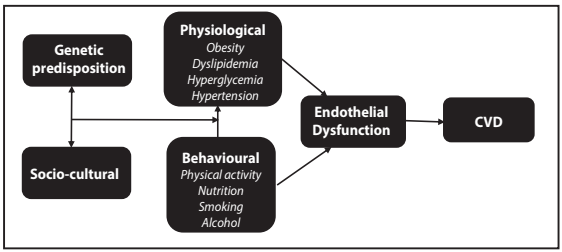


Figure 2. Causation pathway for CVD. Adapted from Stoner et al. 2014 (2)

What to assess and document for in cardiovascular risk assessment

The table below summarises the elements to assess for CVD

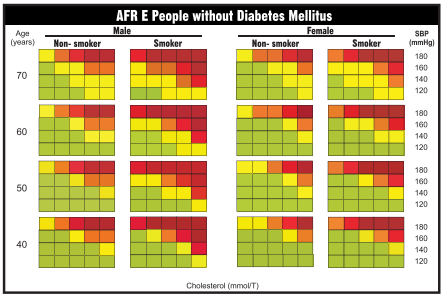
Table 2: Assessment for CVD

History	• Age • Gender • Ethnicity • Smoking status (if stopped smoking for <12 months, assess as a smoker)
Family history	• Premature coronary heart disease or ischemic stroke in a first -degree relative (father or brother <55 years, mother or sister <65 years) • Type 2 diabetes • Genetic lipid disorder
Past medical history	• Past history of CVD (MI, PCI, CABG, angina, ischemic stroke, TIA, peripheral vascular disease [PVD]) • Lipid disorder • Renal impairment (eGFR<60 if under age 75) • Diabetes • Atrial fibrillation
Measure	• Average of two sitting BP measurements – one sitting measurement if not above 160/95; two sitting measurements if the first is above 160/95 • BMI, waist circumference • Non-fasting lipid profile • Fasting Blood Glucose

* For asymptomatic Indo-Asians/Caucasians conduct CVD risk assessment at 30years for men and 35 years for women.

Risk assessment for CVD is crucial for all individuals. Below are tools for assessment of risk of developing a CVD in 10 years. The approach to management is guided by the risk level of the individual as shown in the table below.

WHO/ISH Risk Prediction Charts for AFRO E region.



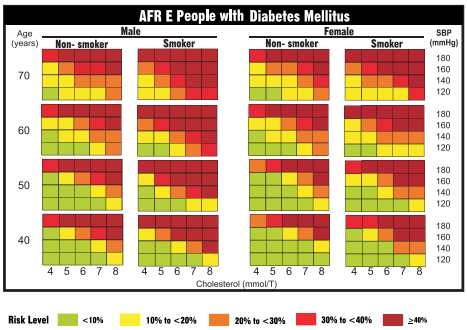


Figure 3: WHO/ISH CVD Risk Prediction Charts

Recommendations for prevention and care of CVD based on individual risk level (Adapted from WHO)

Table 3: Recommendations for prevention and care of CVD

10-year risk of cardiovascular event >30%	10-year risk of cardiovascular event 20–30%	10-year risk of cardiovascular event 10–20%	10-year risk of cardiovascular event <10%
● Individuals in this category are at veryhigh risk of fatal or non-fatal vascular events ● Monitor risk profile every 3–6 months	● Individuals in this category are at high risk of fatal or non-fatal vascular events ● Monitor risk profile every 3–6 months	● Individuals in this category are atmoderate risk offatal or non-fatalvascular events. ● Monitor risk profile every 6–12 months	● Individuals in this category are at lowrisk. Low risk does not mean“no” risk. ● Conservative management focusing on lifestyle Interventions is suggested
When resources are limited, individual counseling and provision of care may have to be prioritized according to cardiovascular risk.			

Principles of Non-Pharmacological Therapy

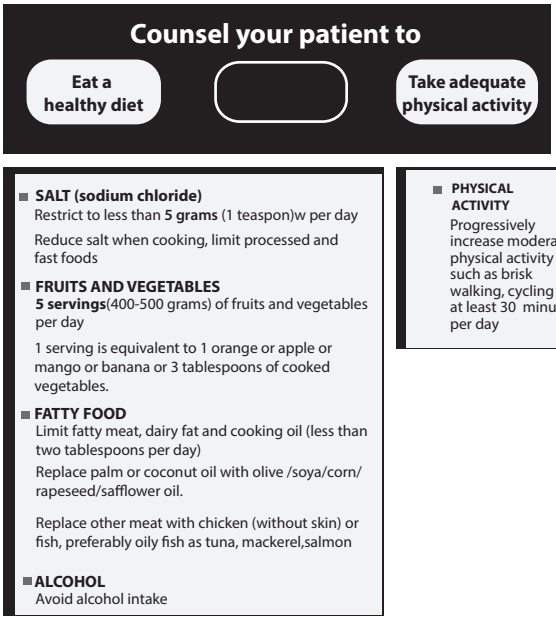
Non-pharmacological interventions are largely lifestyle interventions.Graded lifestyle advice is appropriate for everyone and needs to consider the individual's circumstances. Specific lifestyle interventions are based on a behavior counseling approach.

1. Nutrition and healthy diet

Total fat should not exceed 30% of total energy intake, with a shift away from saturated fats to unsaturated fats, and towards the elimination of industrial trans-fats.

Limit intake of refined sugars to less than 10% of total energy intake. A further reduction to less than 5% of total energy intake is suggested for additional health benefits.

Figure 4: Protocol for counselling on diet and physical activity



Recommendations for salt reduction

- Adults: consume less than 5 g (just under a teaspoon) of salt per day.
- Children: consume less than 3g of salt per day
- All salt that is consumed should be iodized or "fortified" with iodine, which is essential for healthy brain development in the fetus and young child and optimizing people's mental function in general.
- Avoid processed foods such as bread,crisps
- Avoid adding salt at the table while eating.

2. Physical Activity

Patients should be encouraged to engage in a variety of physical activities and to progressively increase their activity as tolerated. Individuals with a history of CVD should consult their healthcare provider before they undertake vigorous physical activity. Vigorous activity is generally not encouraged in people with impaired left ventricular function, severe coronary artery disease, recent MI, significant ventricular arrhythmias or stenotic valve disease. Moderate activity is however recommended under the guidance of a healthcare provider.

Recommendations (Adapted from WHO Physical Activity Fact Sheet)

- Children and adolescents aged 5-17 years should do at least 60 minutes of moderate to vigorous-intensity physical activity daily.
- Adults should do at least 150 minutes of moderate-intensity physical activity throughout the week.
- Those with poor mobility should perform physical activity to enhance balance and prevent falls, 3 or more days per week.
- Muscle-strengthening activities should be done involving major muscle groups, 2 or more days a week.

Table 5: Contraindications, Precautions and Indications to stop physical activity in patients with heart disease

Contraindications	Precautions	Indications to stop physical activity
● Unstable angina ● Symptoms (e.g chest discomfort, shortness of breath) on low activity ● Decompensated heart failure ● Severe aortic stenosis ● Uncontrolled hypertension (e.g. systolic BP≥180 mmHg; Diastolic BP ≥110 mmHg) ● Acute infection or fever ● Symptomatic uncontrolled diabetes (e.g. blood glucose <6mmol/L or >15mmol/L)	● All patients should be provided with clear advice on risks and benefits of physical activity, warm-up and cooldown, limiting physical activity to low-moderate intensity, appropriate footwear and clothing, and the importance of following their symptom (chest pain, diabetes) management plans ● Resting tachycardia/ arrhythmia	● Squeezing, discomfort or typical pain in the centre of the chest or behind the sternum, spreading to the shoulders, neck, jaw and/or arms ● Dizziness, lightheadedness or feeling faint, difficulty breathing,nausea, uncharacteristic excessive sweating ● Palpitations associated with feeling unwell, undue fatigue ● Shakiness, tingling lips, hunger, weakness or palpitations in people with diabetes

3. Weight Management

Overweight and /or obesity results from an energy imbalance between calories consumed and calories expended. Measurement of body mass index (BMI) is one way of assessing for overweight and obesity. The table below catogorises overweight and obesity.

Table 6: Definition of overweight and obesity

Population Group	Overweight	Obesity
Children <5 years	Weight-for-height > 2 standard deviations above the WHO Child Growth Standards median*	Weight-for-height > 3 standard deviations above the WHO Child Growth Standards median*
Children 5-19 years	BMI-for-age > 1 standard deviation above the WHO Growth Reference median*	BMI-for-age >2 standard deviations above the WHO Growth Reference median *
Adults	BMI 25-30	BMI > 30

*Refer to WHO growth reference charts and tables for children aged < 5years between 5–19 years

NB:

Waist circumference: this helps determine whether a patient has the metabolic syndrome or is at risk for type 2 diabetes. Risk is high when the measurement is >102 cm in men or >88 cm in women.

Recommendations for Weight Management

- Limit energy intake from total fats and sugars;
- Increase consumption of fruit and vegetables, as well as legumes, whole grains and nuts; (refer to Nutrition and healthy diet section)
- Engage in regular physical activity. (Refer to physical activity section)

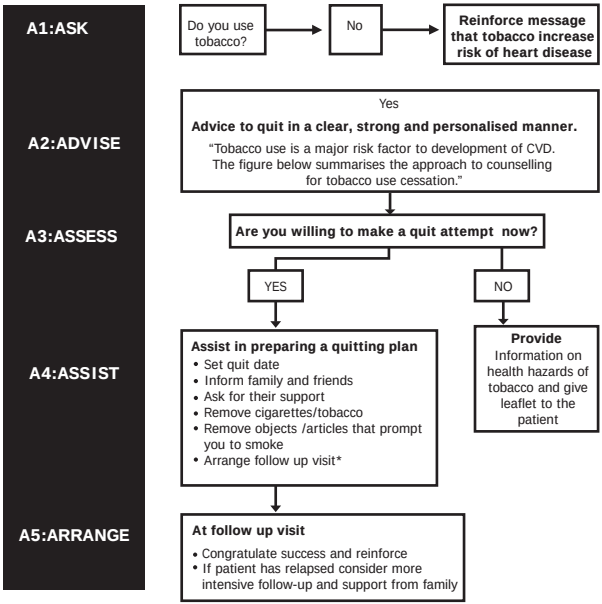
4. Tobacco Dependence Treatment, Cessation and Prevention

There are three main categories of interventions:

a. Brief advice by a healthcare professional
b. Behavioral support
c. Pharmacotherapy

Brief advice on Tobacco Cessation by a healthcare professional (5As)

Protocol for counselling on cessation of tobacco use the 5As approach



* Ideally second follow-up visit is recommended within the same month and every month thereafter for 4 months and evaluation after 1 year. If not feasible, reinforce counselling whenever the patient is seen for blood pressure monitoring.

Figure 7: Counselling on Cessation of Tobacco Use. Adapted from WHO (8)

Behavioural support

Behavioural support aims at changing thought processes and beliefs. If one changes the way they feel about tobacco use, a change in behaviour should follow. The healthcare provider helps the person to deal with negative feelings and assists the clients in setting realistic goals to avoid failure.

Behavioural strategies that can support a client to cope with the triggers and high-risk situations for tobacco use include:

- Face to face support
- Individual behavioural counselling
- Group behaviour therapy
- Telephone counselling or quit lines
- Self-help materials

The pharmacological interventions include:

1. Nicotine replacement therapies
    - Nicotine gums
    - Nicotine patches
    - Nicotine lozenges/sublingual tablets
    - Nicotine inhalers
    - Nicotine nasal spray
2. Non-Nicotine replacement therapies
    - Bupropion
    - Varenicline

For further details on behavioral and pharmacological interventions, refer to the Kenya National Guidelines for Tobacco Dependence Treatment and Cessation.

Principles of Pharmacological Therapy in CVD

Pharmacological management is largely disease-specific and is majorly handled in the subsequent chapters. Discussed below are general principles of lipid lowering and antiplatelet therapy.

a) Lipid Lowering Therapy

This therapy is given to patients depending on the individual's risk of developing CVD as per the risk assessment. However, in patients with diabetes, total Cholesterol (TC) ≥8 mmol/L or a TC: HDL-C ratio ≥8, lipid-lowering treatment is usually recommended irrespective of the combined CVD risk.

Lipid lowering for people with combined CVD risk between 10 % and 20 %

- For patients with combined CVD risk between about 10% and 20%, discuss the benefits (and risks) of initiating statins.
- Following lifestyle management, repeat lipid profile (non-fasting) to recalculate risk and use the results to inform shared treatment decision-making in 6–12 months.

Before a person starts medication, it is important to consider and exclude a treatable primary cause for a dyslipidemia e.g. high saturated fat diet and excessive alcohol consumption, hypothyroidism, diabetes, liver disease, nephrotic syndrome and steroid treatment.

For people with known cardiovascular disease and those with a combined cardiovascular risk >20%, statin treatment is strongly recommended.

Monitoring

Monitor non-fasting lipids every three to six months until the person is stable on their treatment regime and then no more than once a year. Measuring more frequently may mislead as the variation in day-to-day measurement may be greater than drift over time. The aim is to achieve a moderate reduction in LDL-C; no target is required for those with a combined risk ratio under 20 percent. Consider interactions of statins with other medications. Review with a pharmacist. Consider a simvastatin dose reduction for patients taking fibrates, systemic fusidic acid, colchicine or with renal impairment. Monitoring liver function tests with statin use is not considered necessary as the risk of liver toxicity appears negligible. Monitoring creatine kinase (CK) is not required in those who are asymptomatic. Check CK for unexplained muscle pain, tenderness or weakness. The risk of myopathy is usually dose-related and is increased in the elderly and with combination treatments. For muscle pain without CK rise, dose reduction or discontinuation may be required. With CK rise 3–10x normal with symptoms, dose reduction or discontinuation with regular weekly monitoring of symptoms and CK is appropriate. With CK rise >10x normal with symptoms, discontinue statin immediately.

b) Antiplatelet Therapy

- Aspirin and other antiplatelet agents are not generally recommended for people with a risk lower than 20 percent.
- Antiplatelet therapy is recommended for people with combined CVD risk over 20% but without established cardiovascular disease.
- Low-dose Aspirin (75-150 mg) can be considered for primary prevention in high-risk populations, taking into account the harms and benefits.
- Antiplatelet therapy for people with established cardiovascular disease
- Antiplatelet therapy is strongly recommended for people with established cardiovascular disease.

Aspirin contraindications

- Aspirin allergies/intolerance
- Active peptic ulceration
- Systolic BP >180
- Other major bleeding risks

Hypertension

Introduction

Hypertension is defined as persistently elevated, systolic and/or diastolic blood pressure (BP) of 140/90 mmHg or more in subjects aged 18 years and above

Table 7: Definition and classification of hypertension (2)

Category	Systolic		Diastolic
Optimal	<120	and	<80
Normal	120-129	and/or	80-84
High normal	130-139	and/or	85-89
Grade 1 hypertension	140-159	and/or	90-99
Grade 2 hypertension	160-179	and/or	100-109
Grade 3 hypertension	≥180	and/or	≥100
Isolated systolic hypertension	≥140	and	<90

(Adapted from the ESH/ISH guidelines)

NB: The class is determined by whichever of the readings is highest.

Diagnosis

The accurate diagnosis of hypertension depends on the accurate measurement of BP. BP readings can be taken in the clinic (office) or out of the office i.e. ambulatory BP measurement (ABPM) or home measurement. Figure 8 gives details of office/health facility measurement.

Allow patient to sit for 3–5 minutes before commencing measurement

The SBP should be first estimated by palpation to avoid missing the auscultatory gap

Take two readings 1–2 minutes apart. If consecutive readings differ by > 5 mm, take additional readings

At initial consultation measure BP in both arms, and if discrepant use the higher arm for future estimations

The patient should be seated, back supported, arm bared and arm supported at heart level Patients should not have smoked, ingested caffeine-containing beverages or food in previous 30 min

An appropriate size cuff should be used: a standard cuff (12 cm) for a normal arm and a larger cuff (15 cm) for an arm with a mid-upper circumference > 33 cm (the bladder within the cuff should encircle 80% of the arm)

Measure BP after 1 and 3 minutes of standing at first consultation in the elderly, diabetics and in patients where orthostatic hypotension is common

When adopting the auscultatory measurement use Korotkoff I (appearance) and V (Disappearance) to identify SBP and DBP respectively Take repeated measurements in patients with atrial fibrillation and other arthythmias to improve accuracy

Choice of cuff size in children
Newborns and premature infants: 4 × 8 cm
Infants: 6 × 12 cm
Older children: 9 × 18 cm

Figure 8: BP measurement procedure
Adapted from the South African hypertension practice guideline 2014 and AAFP 2005

The table below defines the cutoffs for office/clinic readings of BP and ambulatory readings of BP for diagnosing hypertension

Table 8: Diagnostic cutoffs for office and out of office BP measurements

Category	Systolic BP(mmHG)		Diastolic BP(mmHG)
Office BP	≥140	and	≥90
Ambulatory BP			
Daytime (or awake)	≥135	and/or	≥85
Nighttime (or asleep)	≥120	and/or	≥70
24-h	≥130	and/or	≥80
Home BP	≥135	and/or	≥85

(Adapted from the 2013 ESH/ESC Guidelines for the management of arterial hypertension)

NB:

White coat/ isolated office hypertension: It is persistently elevated BP in the clinic while BP is normal outside the clinic.

Masked/ isolated ambulatory Hypertension: BP is normal in the office and abnormally high out of the medical environment. It is usually associated with other risk factors, asymptomatic organ damage and increased risk of diabetes and sustained hypertension.

NB: These terms should only be used to define untreated patients.

Cardiovascular Disease Risk factor stratification for management of Hypertension

The approach for management of hypertension should be directed by the CVD risk of the individual. The table below describes the elements to note while assessing the CVD risk of a hypertensive individual. The below risk stratification tool is a guide on assessing the individual for existing or impending CVD and the action to take.

Table 9: Major risk factors, target-organ damage (TOD) and complications.

Major risk factors	TOD	Complications
• Levels of systolic and diastolic BP • Smoking • Dyslipidaemia: — total cholesterol > 5.1 mmol/l, OR — LDL > 3 mmol/l, OR — HDL men < 1 and women < 1.2 mmol/l • Diabetes mellitus • Men > 55 years • Women > 65 years • Family history of early onset of CVD: — Men aged < 55 years — Women aged < 65 years • Waist circumference: abdominal obesity: — Men ≥ 102 cm — Women ≥ 88 cm	• LVH: based on ECG • Microalbuminuria: albumin creatine ratio 3–30 mg/mmol preferably spot morning urine and eGFR > 60 ml/min	• Coronary heart disease • Heart failure • Chronic kidney disease: — macroalbuminuria > 30 mg/mmol — OR eGFR < 60 ml/min • Stroke or TIA • Peripheral arterial disease • Advanced retinopathy: — haemorrhages OR — exudates — papilloedema

Table 10: Cardiovascular Risk Stratification Tool

Other disease/risk factor history	BP				
	Normal **SBP 120-129** **Or DBP 80-84**	**High normal** **SBP 130-139** **Or DBP 85-89**	**Stage 1** **Mild hypertension** **SBP 140-159** **Or DBP 90-99**	**Stage 2** **Moderate hypertension** **SBP 160-179** **Or DBP 100-109**	**Stage 3** **Severe hypertension** **SBP >180** **Or DBP >110**
No other major risk factors	Average risk No BP intervention	Average risk No BP intervention	Low added risk • Lifestyle changes for several months • Then add BP drugs targeting <140/90	Moderate added risk • Lifestyle changes for several months • Then add BP drugs targeting <140/90	High added risk • Lifestyle changes • Immediate BP drugs targeting <140/90
1-2 major risk factors	Low added risk • Lifestyle changes • No BP intervention	Low added risk • Lifestyle changes • No BP intervention	Moderate added risk • Lifestyle changes for several months • Then add BP drugs targeting <140/90	Moderate added risk • Lifestyle changes for several months • Then add BP drugs targeting <140/90	Very high added risk • Lifestyle changes • Immediate BP drugs targeting <140/90
≥3 major risk factors or TOD or DM or metabolic syndrome	Moderate added risk • Lifestyle changes • No BP intervention	High added risk • Lifestyle changes • No BP intervention	High added risk • Lifestyle changes • BP drugs targeting <140/90	High added risk • Lifestyle changes • BP drugs targeting <140/90	Very high added risk • Lifestyle changes • Immediate BP drugs targeting <140/90
Symptomatic CVD, CKD stage ≥4 or diabetes with OD/RFs	High added risk • Lifestyle changes • No BP intervention	Very high added risk • Lifestyle changes • No BP intervention	Very high added risk • Lifestyle changes • BP drugs targeting <140/90	Very high added risk • Lifestyle changes • BP drugs targeting <140/90	Very high added risk • Lifestyle changes • Immediate BP drugs targeting <140/90

Adapted from the 2013 ESH/ECS Guidelines for the Management of arterial hypertension

Management of Hypertension

The overall aim of the treatment of hypertension is the adequate control of blood pressure and the control of other risk factors with the overall aim of reducing morbidity and mortality from the complications.

Non-pharmacologic/Lifestyle Modification

At every clinic visit, all patients should receive advice about lifestyle modification. Healthy lifestyle choices can reduce blood pressure and cardiovascular risk and reduce the dose and number of antihypertensive medications required.

Table 11: Lifestyle advice for hypertension

- Avoidance of alcohol
- Avoidance of all forms of tobacco
- Daily adequate physical exercise: Hypertensive patients should be advised to participate in at least 30 min of moderate-intensity dynamic aerobic exercise (walking, jogging, cycling or swimming) on 5–7 days per week
- Consumption of a healthy diet: Hypertensive patients should be advised to eat vegetables, lowfat dairy products, dietary and soluble fibre, whole grains and protein from plant sources, reduced in saturated fat and cholesterol, Fresh fruits. Avoid added salt and high salt food.

(Adapted from the ESH/ESC guidelines, 2013).

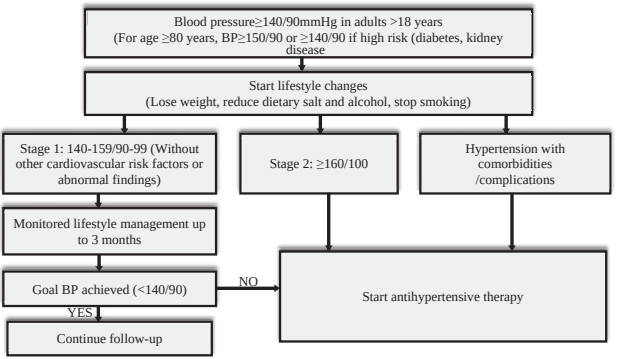


Figure 9: Threshold for treatment initiation

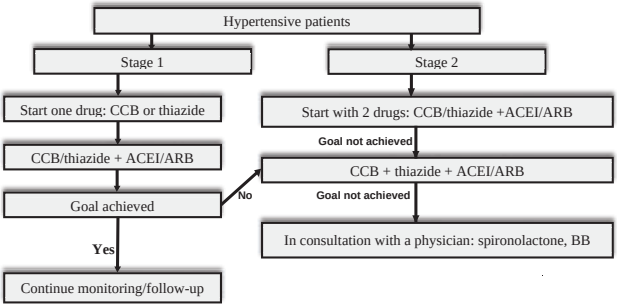


Figure 10: Choice of medications for lowering blood pressure

Combination therapy

Combination therapy including fixed dose combinations are recommended as it minimizes toxicity and therefore side effects as well as improves adherence to treatment. The recommended options for combinations therapy are shown in the figure beside:

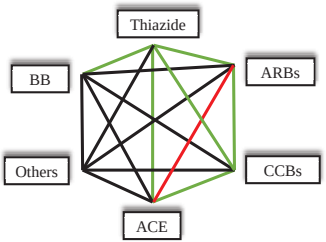


Figure 11: Choices for combination medications

Key: Green: Use, Red: Don't use, Black: consult a physician (Adapted from 2013 ESH/ESC Guidelines for the management of arterial hypertension).

The recommended dosages and common side effects of various antihypertensive medications is outlined below.

Class	Examples	Usual monotherapy	Maximum daily dose	Possible side effects
Long-acting CCB	Amlodipine	5 mg OD	10 mg OD	• Oedema • Fatigue • Headache • Palpitations
	Felodipine	5 mg OD	10 mg OD	
	Nifedipine	Retard tabs: 10-20 mg BD LA tabs: 30 mg OD	Retard tabs: 30 mg BD LA tabs: 90 mg OD	
Thiazide diuretic	Chlorthalidone	25 mg OD	50 mg OD	• Hypokalaemia • Hyponatraemia, • Hyperuricaemia • Hypocalciuria, • Hyperglycaemia • Rash • Dyslipidaemia
	Hydrochlorothiazide (HCTZ)	12.5 mg OD	25 mg OD	
Thiazide-like diuretic	Indapamide	2.5 mg OD	5 mg OD	
ACE inhibitor	Captopril	25-50 BD or TDS	50 mg TDS	• Cough (ACEI) • Hyperkalaemia • Increased serum creatinine • Angioedema
	Enalapril	5-20 mg daily in 1 or 2 divided doses	20 mg daily in 1 or 2 divided doses	
	Lisinopril	10 mg OD	40 mg OD	
	Perindopril	4 mg OD or 5 mg OD	8 mg OD or 10 mg OD	
	Ramipril	2.5 mg OD	10 mg OD	
Beta-blockers	Atenolol	25 mg OD	100 mg	
	Bisoprolol	2.5 mg OD	20 mg OD	
	Carvedilol	6.25mg BD	25 mg BD	
	Labetalol	100 mg BD	400 mg BD	
	Metoprolol succinate	25mg OD	100 mg OD	
	Nebivolol	5 mg OD	20 mg OD	
ARB	Candesartan	8 mg OD	32 mg OD	
	Irbesartan	150 mg OD	300 mg OD	
	Losartan	50 mg OD	100 mg OD	
	Telmisartan	40 mg OD	80 mg OD	
	Valsartan	80 mg OD	160 mg OD	

CCB: Calcium channel blocker; ACE: angiotensin converting enzyme; ARB: angiotensin receptor blocker
OD: administer once daily; BD: administer twice daily; TDS: administer 3 times daily

The table below outlines other less commonly used medications that prescribed by specialists depending on circumstances of the patient.

Table 13: Other anti-hypertensive medications

Other antihypertensive agents				
Class	**Examples**	**Usual monotherapy starting dose**	**Maximum daily dose**	**Possible side effects**
Centrally acting agents	Methyldopa	250mg BD or TDS	1000mg/day	• Angina • Orthostatic Hypotension • Gynaecomastia • Rash
	Clonidine	0.1mg BD	2.4mg/day	
	Phenoxybenzamine	0mg BD	40mg TDS	
Potassium sparing diuretics	Amiloride	5mg OD/ divided	10mg OD or divided dose	• Hyperkalaemia • Headache
	Triametrene	25mg OD or divided dose	100mg OD or divided dose	
Loop Diuretics	Torasemide	5 mg OD	20 mg OD	• Hyperuricaemia • Hypokalaemia
	Furosemide	20 mg OD	80mg OD or divided dose	
Vasodilators	Hydrallazine	25 mg BD or TDS	150 mg/day	Hypotension Palpitations
Alpha 1 Receptor Blocker	Prazosin	1mg BD-TDS	20mg/day	Hypotension, diarrhea, Tachycardia
	Terazosin	1mg OD	20mg/day	

Resistant hypertension

This is defined as BP ≥ 140/90 mmHg despite treatment with at least 3 drugs (including a diuretic) in adequate doses and after exclusion of false hypertension such as isolated office hypertension and failure to use large cuffs on large arms.

Table 14: Causes of resistant hypertension

Category	Possible causes	Interventions
Non-adherence to therapy	• Instructions not understood • Side effects • Cost of medication and/or cost of attending at healthcare centre • Lack of consistent and continuous primary care • Inconvenient and chaotic dosing schedules • Organic brain syndrome (e.g. memory deficit)	• Adherence counselling • Ensure family/social support mechanism for the patient • Tailor dosing schedules to individual patients
Volume overload	• Excess salt intake • Inadequate diuretic therapy • Progressive renal damage (nephrosclerosis)	• Counsel on low salt diet, optimize diuretic therapy, refer as appropriate
Associated conditions	• Smoking • Increasing obesity • Sleep apnoea • Insulin resistance/hyperinsulinaemia • Ethanol intake of more than 30 g (three standard drinks) daily • Anxiety-induced hyperventilation or panic attacks • Chronic pain • Intense vasoconstriction (Raynaud's phenomenon), arteritis	• Manage the associated condition
Identifiable causes of hypertension	• Chronic kidney disease • Renovascular disease • Primary aldosteronism • Coarctation • Cushing's syndrome • Phaeochromocytoma	• Investigate and/or refer
Pseudoresistance	• 'Whitecoat hypertension' or office elevations • Pseudohypertension in older patients • Use of regular cuff in obese patients	• Out of office BP measurement • Ensure proper BP measurement technique
Drug-related causes	• Doses too low • Wrong type of diuretic • Inappropriate combinations • Rapid inactivation (e.g. hydralazine)	• Review treatment plan
Drug actions	• Non-steroidal anti-inflammatory drugs (NSAIDs) • Sympathomimetics: nasal decongestants, appetite	• Remove offending drug, refer for specialist care

Follow-up for hypertensive patients on treatment

Initially, patients should be seen at 4 week intervals to assess antihypertensive efficacy, check for side effects and adjust medication as appropriate. The aim of therapy is to control BP without side effects. Patients should be advised to return earlier if they feel unwell or experience new symptoms (e.g., headache, persistent cough). Once goal BP has been achieved, the patient should be followed up every 4- 6 months.

NB:-in treating hypertension older age is defined as age greater or equal to 80 years.

What to do if goal BP is not achieved and when to step up therapy

i) Confirm that the patient is taking his/her medication as instructed (i.e., every day, the correct dose, the correct number of times per day, at the correct time of day). If necessary ask the spouse or another family member to confirm this information.

ii) If the patient has not been taking their medication as prescribed, determine reasons for this and address them appropriately.

iii) Ask about use of other prescribed medicines, over-the-counter medicines

Hypertensive crises and appropriate management and referral

Hypertensive emergencies: large elevations in SBP or DBP (>180mmHg or >120mmHg, respectively) associated with impending or progressive organ damage/dysfunction. They require immediate BP reduction (not necessarily to normal) to prevent or limit target organ damage. They must be managed on an inpatient with close monitoring and physician presence.

Hypertensive urgencies: are those situations associated with severe elevations in BP without progressive target organ dysfunction. The majority of these patients present as noncompliant or inadequately treated hypertensive individuals, often with little or no evidence of target organ damage. This can be managed in the outpatient setting by investigating the factors that may underlie the BP rise and dose adjustments as necessary.

Table 15: Hypertensive emergencies and urgencies (10)

Emergency	Management options
Acute ischemic stroke	Antihypertensive therapy is **not routinely recommended**for patients with acute stroke and HTN.
Acute Intracerebral Hemorrhage	BP lowering when the SBP is **>200mmHg** or the DBP is**>110mmHg.** If signs of **increased ICP**, maintain **MAP** just **below 130mmHg** (or **SBP <180mmHg**) for first 24 hours after onset. Patients **without increased ICP**, maintain **MAP < 110mmHg** (or **SBP < 160mmHg**) for first 24 hours after symptoms onset.
Subarachnoid Hemorrhage-	Maintain **SBP <160mmHg** until the aneurysm is treated or cerebral vasospasm occurs. Oral **nimodipine** is used to **prevent delayed ischemic neurological deficits**, but it is **NOT indicated** for treating acute hypertension.
Aortic dissection	**Immediately** reduce the **SBP < 110mmHg** and maintain it at this level unless signs of end-organ hypo perfusion are present. Preferred treatment includes a combination of; a) narcotic analgesics (morphine sulphate), b)β-blockers (labetalol, esmolol) or calcium channel blockers (verapamil, diltiazem); **Avoid β-blockers** if there is aortic valvular regurgitation or suspected cardiac tamponade.
Acute Coronary Syndrome	Treat if **SBP>160 mmHg** and/or **DBP >100 mmHg**. Reduce BP by **20-30%**of baseline. Thrombolytics are **contraindicated** if BP is**>185/100 mmHg**. Preferred medications include **β-blockers**& **Nitroglycerin**
Acute Heart Failure	Treatment with vasodilators (in addition to diuretics) for **SBP ≥140 mmHg**. **IV or sublingual nitroglycerin** is the preferred agent.
Preeclampsia/ eclampsia	Prepartum and intrapartum: **SBP** should be **< 160 mmHg** and **DBP <110 mm Hg** If the **platelet count is <100,000 cells/mm, S**BP should be maintained below **150/100mmHg.** Patients with eclampsia or preeclampsia should also be loaded with **IV Magnesium sulphate 4gm**diluted in 100mL NS over 15 mins then with an **infusion of 2gm/hr** to avoid seizures. **Preferred medications**-Hydralazine,Labetalol, Nifedipine **Medications to avoid** -Nitroprusside, ACEIs, Esmolol

(Adopted from Emergency care algorithms, Kenya, 2017)

Ischemic heart disease

Ischaemic heart disease (IHD), also known as coronary heart disease/coronary artery disease is the eventual manifestation of myocardial dysfunction following occlusion of the coronary arteries by cholesterol plaque.

Presentation of IHD

Patients with coronary artery disease can present acutely or insidiously with symptoms over months to years.

1. Stable disease

Stable angina is defined as myocardial ischaemia on exertion and relieved by rest in the absence of cardiomyocyte necrosis. The Canadian Cardiovascular Society Classification is usually used to grade angina as

Table 16: Canadian Cardiovascular Society Classification of Angina

Class I	**Angina with strenuous or prolonged exertion**
Class II	**Angina on walking or climbing stairs rapidly,walking/stair climbing after meals, during the first few hours after awakening, walking more than 2 blocks on level or climbing more than one flight of ordinary stairs at a normal pace and conditions**
Class III	**Angina on walking 1 to 2 blocks on level or climbing one flight of stairs at normal pace and conditions**
Class IV	**Angina at rest**

Unstable angina is defined as myocardial ischaemia at rest or minimal exertion in the absence of cardiomyocyte necrosis. Unstable angina can be classified according to Braunwald as shown below: -

Table 17: Braunwald classification of angina severity

Class/severity	Description
I	**New onset severe angina, no rest pain**
II	**Angina at rest within preceding month, but not past 48 hours**
III	**Angina at rest within the preceding 48 hours**

2. **Acute Coronary Syndrome or Acute myocardial infarction (MI)** defines cardiomyocyte necrosis that is consistent with acute myocardial ischaemia.

3. **Heart failure**
Chronic ischemic heart disease may lead to heart failure.

4. **Arrhythmia**
Some patients may present with VT/VF commonly and less commonly with
supraventricular arrhythmia such as atrial fibrillation. VF usually signifies acute disease whereas VT signifies scar in a previously infarcted territory.

5. **Cardiac arrest and sudden cardiac death**
Patients with severe disease affecting the left main stem or severe disease with a single remaining vessel may experience sudden cardiac death.

Assessment for IHD

Given broad spectrum of IHD, the presentation is quite varying. Therefore, a high index of suspicion is demanded from the clinician. Patients with chest pain and suspected angina should have full history and examination performed as part of their initial evaluation. This should include family history of coronary artery disease and sudden death.

Stable angina
The table below outlines the prediction tool developed by Diamond and Forrester.

Table 18: Angina risk prediction tool

Criteria **A: Sub-sternal chest pain with rest**	**B: Exertional chest pain**	**C: Chest pain relieved**
Interpretation		
A: Typical angina (All 3 criteria)	**Age**	**Sex**
		Male / **Female**
	30-39	Intermediate / Intermediate
	40-49	High / Intermediate
	50-59	High / Intermediate
	60-69	High / High
B: Atypical angina (2 criteria)	30-39	Intermediate / Low
	40-49	Intermediate / Low
	50-59	Intermediate / Intermediate
	60-69	Intermediate / Intermediate
C: Non-anginal chest pain (1 criteria)	30-39	Low / Low
	40-49	Intermediate / Low
	50-59	Intermediate / Low
	60-69	Intermediate / Intermediate
D: No criteria present	Risk is low for both men and women	
Interpretation		
	Risk	**Action**
	Low	Investigate for non-cardiac causes
	Intermediate	Perform/refer for stress testing
	High	Perform/refer for coronary angiogram

The following dynamic stress tests should be performed under supervision by an experienced cardiologist:

- Exercise ECG
- Exercise stress echocardiogram
- Dobutamine stress echo-cardiogram
- Nuclear Myocardial perfusion

Patients that have negative tests should be evaluated for other causes of chest pain.

Patients with borderline tests should be offered a CT coronary angiogram

Diagnosis of acute IHD

Acute IHD presents as acute coronary syndrome (ACS). The diagnosis of ACS is dependent of on 3 variables that include chest pain, ECG changes and elevated cardiac biomarkers. These can be utilized to score the patient to direct action.

Table 19: Heart Score for possible ACS

Parameter	Category	Points
History	Highly suspicious	2
	Moderately suspicious	1
	Slightly/Non- suspicious	0
ECG	Significant ST deviation	2
	Non-specific repolarization	1
	Normal	0
Age (Years)	≥65	2
	46-64	1
	≤45	0
Risk factors	≥3 risk factors or history of CAD	2
	1 or 2 risk factors	1
	No risk factors	0
Troponin levels	≥3× normal limit	2
	>1 to <3× normal limit	1
	≤ normal limit	0
Risk factors: DM, current/recent 91 month) smoker, HTN, hyperlipidaemia, family history of CAD and obesity		
Interpretation and action		
Score	**Action**	
0-3	Discharge home	
4-6	Admit for observation	
7-10	Perform/refer for invasive strategies	

ECG

In patients with chest pain the ECG should be performed within 10 minutes of presentation.

Biomarkers for ACS

The gold standard biomarker for the diagnosis of ACS is cardiac troponin. Most assays now use the highly sensitive troponin I and T.

Blood for troponin should be drawn immediately after the ECG is performed. Usually it takes approximately 4 hours after onset of symptoms before a rise in troponin can be elicited in the peripheral blood.

It is recommended that all patients should have troponin performed at the time of presentation, and if the initial test is negative and the patients has suspicious symptoms another test should be repeated in 4 hours. A rise in troponin usually suggests a coronary cause of chest pain. A negative troponin in two serial tests and a normal ECG should trigger evaluation for non-cardiac causes of symptoms but where the index of suspicion remains high, non-invasive evaluation should be considered.

Differential Diagnosis of Acute Chest Pain

The table below outlines the other possible diagnosis of acute chest pain and the diagnostic elements.

Table 20: Differential diagnosis for acute chest pain

Aortic dissection	Tearing pain radiating to the back	New murmur, bruits, unequal pulses	CXR,CT-Angiogram,Echo
ACS	Pressure-like pain radiating to the arms/face, diaphoresis, dysponea, risk factors	Evidence of heart failure	ECG, biochemical markers
Pulmonary embolism	Sudden onset, pleuritic pain, dyspnoea, risks for DVT	Tachypnea, tachycardia, DVT	CXR, V/Q scan, CT angiogram, pulmonary angiogram
Esophageal rupture	Constant severe retrosternal/epigastric pain, inciting event	Mediastinal rub/crunch	CXR
Pneumothorax	Pleuritic pain, dyspnea	Diminished breath sounds over hemi-thorax	CXR
Pneumonia	Cough, fever, dyspnea, pleuritic pain	Abnormal breath sounds, fever, hypoxia, tachypnea	CXR
Pericarditis	Positional ache, dyspnea	Rub	ECG, CXR, sonogram
GI causes	Associated abdominal/ GERD symptoms	Abdominal tenderness, guarding	Amylase, lipase, KUB, ultrasound
Musculoskeletal causes	Pain increased with minimal muscular activity or movement	Chest wall tenderness to palpation	Normal

Adapted from Green G, Hill P. Approach to chest pain and possible myocardial ischemia. Emergency medicine: A comprehensive study guide. New York: McGraw Hill; 2000: 341-352

On admission, the following tests should be considered

a. OGTT/FBG
b. HBA1c
c. Lipid profile
d. Thyroid screen
e. HIV

Other tests should be performed depending on the complexity of the patient as guided by the clinical team. These include blood gas analysis in patient with severe dyspnea and lactate levels in patients with hypotension and shock.

Treatment of IHD

Acute Disease

Acute IHD presents as ACS. This is usually STEMI, NSTEMI or UA as defined above

The following are recommendations to institutions that offer care for patients with ACS

1. There should be clear point of contact for patients and health care professionals where help can be obtained immediately
2. There should be ambulances that are appropriately equipped to support ACLS care to patients with ACS
3. The ambulances should be equipped with an ECG machine
4. There should be a system that coordinates ambulances to enhance patient care
5. The triage system in the hospital should identify chest pain as one of the symptoms whose care is expedited
6. There should exist a link with cardiologist to enhance diagnostic capability and management of patients with acute coronary syndromes
7. There should exist a protocol for care for patients with ACS including care pathways, care bundles and care teams to expedite care for patients with ACS
8. There needs to have a collaborative effort that includes hospitals and cardiologists to improve care and outcomes for ACS patients

In the hospital

1. Patients with ACS should be evaluated in the acute room of the health facility
2. These patients should be connected to the ECG monitor and vitals assessed every 15 minutes.
3. There should be resuscitation trolley in all hospitals that take care of STEMI patients
4. There should be resuscitation teams in all hospitals that take care of STEMI patients
5. ECG should be performed and interpreted within 10 minutes of arrival to the hospital
6. ACS patients should managed by the EMERGENCY team in consultation with an attending cardiologist
7. The following tests should be performed on patients with ACS
    a. TBC
    b. Renal function and electrolytes
    c. RBS
    d. Portable CXR
8. On admission, the following tests should be considered
    a. OGTT/FBG
    b. HBA1c
    c. Lipid profile
    d. Thyroid screen
    e. HIV
9. Other tests should be performed depending on the complexity of the patient as guided by the clinical team. These include blood gas analysis in patient with severe dyspnea and lactate levels in patients with hypotension and shock.
10. Pain should be managed with morphine or morphine derivatives such as fentanyl.
    The recommended doses are as follows: -
    Morphine – 2.5-5mgmg iv or SC
    Fentanyl 25mcg -100mcg iv
    Caution is given in patients with respiratory distress where these drugs can cause respiratory failure and therefore airway support should be available

11. Drugs to avoid: These include NSAIDS and steroids. These have been shown to have deleterious effects in patients with ACS
12. Fluids: Patients with ACS should not be given fluids without a clear indication. This may precipitate heart failure in the vulnerable patient. Fluids are useful in a subset of patients with inferior MI and RV infarction. Consult the cardiologist on this.

STEMI

The treatment for STEMI underscore the need for system preparedness, anticipation and collaboration amongst different entities and institutions. Therefore, the patient must be identified early, diagnosis made swiftly and treatment/transfer offered immediately.
The following are recommended for patients with STEMI

1. There should a care box for STEMI patients that consists of the following drugs
    a. Aspirin 300mg orally stat
    b. Clopidogrel 300mg stat
    c. Enoxaparin 1mg/kgsc stat
2. Patient should be connected to ECG monitors immediately on arrival to the health facility
3. Oxygen therapy should be considered to those patients with saturation of less than 92% at room air. Routine oxygen utilization is not recommended
4. Intravenous access should be obtained as soon as possible once the patient is in the health facility
5. Thrombolysis should be administered within 30 minutes of diagnosis being made after consultation with the attending cardiologist.

STEMI should be treated in consultation with an interventional cardiologist. Where the cardiologist is not accessible, immediate transfer should be done to a facility with a cardiologist after administering oxygen as appropriate, aspirin and clopidogrel and heparin (LMWH).

NSTEMI

Patients with NSTEMI will require the same treatment as patients with STEMI, only that they do not require thrombolysis as it has not been shown to be beneficial.
The following recommendations are made:

1. There should a care box for NSTEMI patients that consists of the following drugs
    a. Aspirin 300mg orally stat
    b. Clopidogrel 300mg stat
    c. Enoxaparin 1mg/kg sc stat
2. Patient should be connected to ECG monitors immediately on arrival to the health facility
3. Oxygen therapy should be considered to those patients with saturation of less than 92% at room temperature. Routine oxygen utilization is not recommended
4. Intravenous access should be obtained as soon as possible once the patient is in the health facility
5. Prompt referral to a center with a cardiologist.

Unstable angina

1. There should a care box for NSTEMI patients that consists of the following drugs
    a. Aspirin 300mg orally stat
    b. Clopidogrel 300mg stat
    c. Enoxaparin 1mg/kg sc stat
2. Patient should be connected to ECG monitors immediately on arrival to the health facility
3. Oxygen therapy should be considered to those patients with saturation of less than 92% at room. Routine oxygen utilization is not recommended
4. Intravenous access should be obtained as soon as possible once the patient is in the health facility
5. An attending cardiologists should be informed immediately to guide care in the patient.

Treatment for chronic IHD

The purpose of treatment for chronic stable IHD is geared towards alleviation of symptoms and prevention of death
Treatment is therefore dependent on making of correct diagnosis using coronary angiography. The medical management of stable IHD consist of the following

1. Statin
2. Aspirin
3. Short and Long acting nitrates
4. Beta blockers
5. If these are insufficient then the following can be considered for

symptom control.

a. Nicoradil
b. Ranolazine
c. Trimetazidine
d. Procorolan

These patients should undergo coronary angiography to confirm diagnosis and determine extend and severity of disease.
Further treatment depends on severity of disease but medical therapy applies to all patients. This is guided by a cardiologist on an ongoing process.

Complications of myocardial infarction

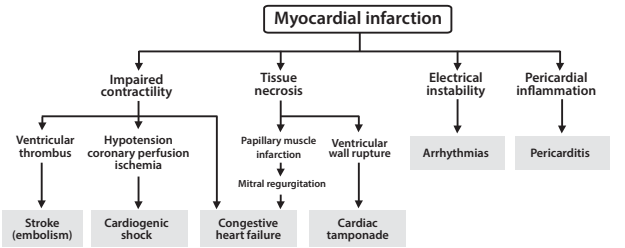


Figure 12: Common complications of IHD

Heart Failure

HF is a clinical syndrome which may be caused by varied underlying conditions including hypertension, valvular disease including rheumatic heart disease, coronary artery disease or primary myocardial disease. It has typical symptoms, which may be accompanied by signs resulting from abnormality in cardiac structure and function, resulting in a reduced cardiac output and/or elevated intra-cardiac pressures (1).

Functional classification of heart failure

The New York Heart Association (NYHA) Functional Classification is based on the degree of limitation of the patient's day-to-day activities of living and is a strong predictor of hospitalization and mortality. The American College of Cardiology/American Heart Association (ACC/AHA) staging is an important tool to identify patients at risk of developing HF.

The TACC/AHA tool below is useful to detect individuals at risk of developing HF. All individuals with DM, HTN or other cardiovascular risk factor should be assessed regularly on the risk of developing HF.

Table 21: NYHA classification of HF

NYHA Functional Classification	
None	
I	No limitations of physical activity. No HF symptoms
I	No limitations of physical activity. No HF symptoms
II	Slight limitations of physical activity. Comfortable at rest, but ordinary results in HF symptoms
III	Marked limitation of physical activity. Comfortable at rest, but less than ordinary activity causes HF symptoms
IV	Unable to carry on any physical activity without HF symptoms

Table 22: TACC/AHA Classification of HF

Class	Objective Assessment
A	No objective evidence of cardiovascular disease. No symptoms and no limitation in ordinary physical activity.
B	Objective evidence of minimal cardiovascular disease. Mild symptoms and slight limitation during ordinary activity. Comfortable at rest.
C	Objective evidence of moderately severe cardiovascular disease. Marked limitation in activity due to symptoms, even during less-than-ordinary activity.Comfortable only at rest.
D	Objective evidence of severe cardiovascular disease. Severe limita Experiences symptoms even while at rest.

Diagnosis of Heart Failure

The table below outlines the symptoms and signs of HF.

Table 23: Symptoms and signs of HF

Symptoms	Signs
Typical	**More specific**
• Breathlessness • Orthopnea • Paroxysmal nocturnal dyspnea • Reduced exercise tolerance • Fatigue, tiredness, increased time to recover after exercise • Ankle swelling	• Elevated jugular venous pressure • Hepatojugular reflex • Third heart sound (gallop rhythm) • Lateral displaced apical impulse
• Less typical symptoms	• Less specific
• Nocturnal cough • Wheezing • Bloated feeling • Loss of appetite • Confusion (especially in the elderly) • Depression • Palpitations • Dizziness • Syncope • Bendopnea (dyspnea on bending over)	• Weight gain(>2kg/week) • Cardiac murmur • Peripheral edema (ankle, sacral, scrotal) • Pulmonary crackles • Dullness on percussion at the lung bases (pleural effusion) • Tachycardia • Irregular pulse • Tachypnoea • Cheyne-Stokes respiration (late) • Hepatomegaly/Ascites • Cold extremities • Oliguria • Narrow pulse pressure

Diagnostic Criteria

Below is a simplified distinction between HF-REF and HF-PEF.

The diagnosis of HF-REF requires three conditions to be satisfied:
1. Symptoms typical of HF
2. Signs typical of HF
3. Reduced LVEF
The diagnosis of HF-PEF requires four conditions to be satisfied:
1. Symptoms typical of HF
2. Signs typical of HF
3. Normal or only mildly reduced LVEF and LV not dilated
4. Relevant structural heart disease (LV hypertrophy/LA enlargement) and or/or diastolic dysfunction (see Sectoin 4.1.2)

HF=heart failure; HF-PEF = heart failure with 'preserved' ejection fractions; HF-REF= heart failure and a reduced ejection fraction; LA = left atrial; LV = left ventricular; LVEF = left ventricular ejection fraction.
[a]Signs may not be present in the early stages of HF (especially in HF-PEF) and in patients treated with diuretics.

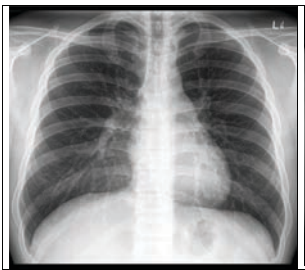
Figure 14: Distinction of diagnosis between HF-REF and HF-PEF

1. Clinical history – History of common risk factors (HTN, RHD or CAD), family history of cardiac disease or SCD. Exposition to cardiotoxicity (alcohol, drugs/radiation). Typical symptoms Shortness of breath on exertion. Good response to diuretic therapy.
2. Physical examination – On auscultation: Bilateral crackles, third heart sound, murmurs. Signs of RV failure: Elevated JVP, hepatomegaly/ascites or bilateral pedal oedema.
3. 12-lead ECG
4. Echocardiography
5. (Natriuretic peptides: NTproBNP or BNP) – useful in HFpEF when no clear echocardiographic signs of HF have been established.

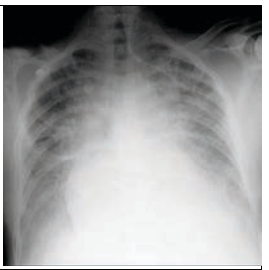
Investigations

Chest radiograph

Important radiographic abnormalities associated with HF include pulmonary vascular redistribution, cardiomegaly, pleural effusions and interstitial oedema. However in clinical practice the chest x-ray will more often be used to rule out differential diagnoses like pneumonia, COPD or pulmonary TB as illustrated below. In children cardiomegaly/-cardiac shadow.



Normal chest radiograph.



Chest radiograph of a patient with heart failure.

- Note the enlarged cardiac shadow(cardiomegaly),increased vascular markings, blunting of the costo-phrenic and cardio-phrenic angles.

Figure 15: Chest radiograph showing normal and abnormal findings

Electrocardiogram (ECG)

A normal ECG makes the diagnosis of HF highly unlikely. However, ECG alone cannot be used to confirm the diagnosis of HF. Major features on the ECG seen in patients with HF include poor r-wave progression, axis deviation, atrial fibrillation, bundle branch blocks, left or right ventricular hypertrophy and pathological Q-waves.

Echocardiography

Echocardiography is the Gold standard test for characterizing cardiac dysfunction in HF as it: -

- Determines systolic LV performance through determining left ventricular ejection fraction (LVEF), cardiac output, chamber quantification and regional wall motion abnormalities.
- Determines end-diastolic LV filling pressures for assessment of diastolic dysfunction and systolic pulmonary artery pressure to diagnose pulmonary hypertension.
- identifies clinically important valvular and pericardial disease.

Laboratory investigations

Other laboratory investigations are required both to assist in the diagnosis of HF, evaluate for underlying aetiologies, and to monitor patients' clinical status during therapeutic interventions. **At baseline these should include:**

- Natriuretic peptides (BNP or NT-proBNP): Are useful in both diagnosis with very high sensitivity for ruling out HF at the lowest cut off point.
- Full blood count: HF due to, or aggravated by, anaemia or infections; HF associated anaemia which is associated with poorer prognosis.
- Urinalysis: proteinuria due to nephropathy/nephritic syndrome, or red blood cells/casts due to glomerulonephritis.
- Serum urea, electrolytes and creatinine (UECs): renal dysfunction, a major determinant of disease progression. (baseline)
- Serum albumin: rule out oedema secondary to low serum albumin in nephrotic syndrome(baseline)
- Lipid profile, thyroid function tests (TFTs): reveal potential cardiovascular or thyroid disease as a cause of HF. (baseline)

- Liver function tests: may indicate liver dysfunction in liver failure or disease progression in liver congestion. (baseline)
- Fasting blood sugar or HbA1c: Type 2 diabetes mellitus is more common in HF.
- Thyroid Function test,
- Blood cultures: Are useful in suspected infectious endocarditis.

Management

Acute Care
This is indicated for decompensated or exacerbation of chronic HF (NYHA III and IV).
Treatment involves:

1. Oxygen
During decompensation, oxygen administration in semi-recumbent position will relieve symptoms of dyspnea and increase tissue oxygen delivery. On occasions, oxygen therapy may have independent beneficial effects, for example in myocardial ischemia if SpO2<90%.

2. Diuretics
Loop diuretics, such as furosemide, are the mainstay medications. Intravenous dosage plays a critical role in acute management due to the occurrence of gut wall edema that limits absorption of oral medication. Short-term thiazide diuretics (including metolazone) may be used in instances of furosemide resistance to augment diuresis.
Caution must be exercised to avoid excessive diuresis leading to hypovolaemia and its consequences (acute renal failure, and electrolyte imbalance).
Regular assessment of hydration status and monitoring of UECs are therefore necessary.

3. Vasodilators
Nitrates are predominantly venodilators and often relieve symptoms of pulmonary congestion, particularly at night when the heart is exposed to increased filling pressures due to the recumbent position.
Long-term vasodilators, such as ACEIs and ARB's can be continued, increased or added through- out the treatment period, particularly if blood pressure is elevated.

4. Beta-blockers
These should not be commenced or increased during the acute decompensation episode, as the acute effect of these agents at a time of fluid overload may worsen clinical status.

5. Inotropic agents
Dobutamine, norepinephrine and dopamine and other inotropic agents have not been shown to decrease mortality and should only be used when there is protracted hypotension.

Discharge planning, the vulnerable period and follow up
Discharge planning should commence as soon as the patient's condition is stable.
Self-Care and Symptom recognition

- Provide educational material that helps the patient understand their condition and related symptoms.
- Sodium and fluid management with 3 to 6-montly UECs and preferably weekly/ daily weight is a proven cost-effective way of avoiding undue hospitalization for worsening HF, hyponatremia and reduces mortality.
- Patients should weigh themselves once to twice a week, preferably daily and at the same time of day.
- Patient should seek medical help with worsening symptoms such as weight gain, shortness of breath, peripheral edema, dizziness or fainting episodes.
- Strict fluid restriction in patients with mild to moderate heart failure does not appear to confer clinical benefit. However, patients should be advised to avoid excessive fluid intake (recommended daily fluid intake less than 1.5day) including fruits.

Pharmacotherapy
This is dependent on the LVEF:

- HF with reduced ejection fraction (HFrEF): HF with LVEF<40%. (From a clinical perspective, it is reasonable to include patients with LVEF 40 – 50% in this group).
- HF with preserved ejection fraction (HFpEF): HF with LVEF ≥ 50%.

Pharmacotherapy of HFrEF

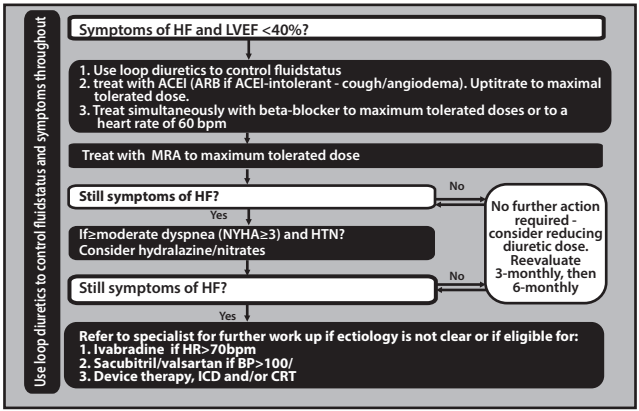


Figure 16: HF algorithm for symptomatic HFrEF. Modified from the HFA/ESC Guidelines 2016(1). Symptomatic = NYHA Class II-IV.

Physical activity/Cardiac Rehabilitation

- Exercise training improves exercise tolerance, health-related quality of life and HF hospitalization rates in patients with HF.
- Ideally, the exercise program should be supervised by cardiac rehabilitation team and individualized to every patient. If this is not possible, patients are encouraged to participate in activities of daily living with regular follow-up by primary health provider.

Table 24: Contraindications to exercise in heart failure

Absolute contraindications
• NYHA class IV • Progressive worsening of exercise tolerance or shortness of breath at rest or on exertion over the prior 3-5 days; • Significant ischaemia at rest; • Uncontrolled diabetes; • Acute systemic illness or fever; • Recent embolism; • Thrombophlebitis; • Active pericarditis or myocarditis; • Severe aortic stenosis; • Regurgitant valvular heart disease requiring surgery; • Myocardial infarction within previous 3 weeks; • New-onset atrial fibrillation

Recommended Pharmacotherapy for patients with heart failure and normal/preserved LVEF (LVEF ≥50%)

Treatment should be individualized targeting the underlying cause, most often controlling risk factors such as hypertension, obesity, obstructive sleep apnea (OSA) in order to lower left ventricular end-diastolic pressures.
If no high-level echocardiography is available, these patients should be referred to a cardiologist for further work up.

The tables below detail out the medications used to manage HF and the recommended dosage.

Table 25: Pharmacotherapy dosage for disease-modifying drugs in HF with reduced ejection fraction

	Starting dose	Target dose
ACEI		
Enalapril	2.5 mg b.id.	10 – 20 mg b.i.d.
Ramipril	1.25 mg b.i.d.	5mg b.i.d.
Lisonopril	2.5 – 5 mg o.d.	20 – 35 mg o.d.
Beta-blockers		
Bisoprolol	1.25 mg o.d.	10 mg o.d.
Carvedilol	3.125 mg b.i.d.	25 mg b.i.d.
Metoprolol Succinate	12.5 – 25 mg b.i.d.	200 mg o.d.
Nebivolol*	1.25 – 2.5 mg o.d.	10 mg o.d.
ARBs		
Candesartan	4 mg o.d.	32 mg o.d.
Losartan	50mg o.d.	150mg o.d.
Valsartan	40 mg o.d.	160 mg o.d.
MRAs		
Eplerenone	25 mg o.d.	50 mg o.d.
Spironolactone	12.5mg o.d.	25 – 50 mg o.d.
If-channel inhibitor		
Ivabradine	5 mg b.d.	7.5 mg b.d.
ARNI		
Sacubitril/valsartan	49/51 mg b.i.d.	97/103 mg b.i.d.

Table 26: Pharmacotherapy dosages for diuretics used in HF.

Diuretics	Starting dose	Usual dose
Loop diuretics		
Furosemide	20 – 40 mg	40 – 240 mg o.d.
Torasemide	5 – 10 mg	10 – 20 mg o.d.
Thiazides		
Hydrochlorthiazide	25 mg	12.5 – 100 mg o.d.
Metolazone	2.5 x 2/week	2.5 – 10 mg o.d.
Others		
Hydralazine/ISD(M)N	25/10 mg t.d.s.	50/20 mg t.d.s.
Digoxin	0.0625 mg o.d	0.125 mg o.d.

Table 27: Clinical consideration when using HF medication.

Drug class	Clinical use considerations
Loop diuretics	Use as first line therapy for immediate volume and symptom control in the acute decompensated patient. Monitor renal function and electrolytes (including calcium and magnesium) frequently especially with IV administration. Overly aggressive diuresis may cause renal dysfunction or hyperkalaemia. Titrate doses based on fluid status. Monitor weight closely (daily if inpatient and at each visit if outpatient).
ACE-I/ARB	May all cause renal dysfunction with hyperkalaemia. Monitor UECs 3 – 6 monthly. Hold if creatinine increases more than 30% from baseline, or if K+ >5.5 mmol/L, and re-start at a lower dose once K drops < 5. Titrate to target doses, but watch out for hypotension which may worsen kidney dysfunction.
ACE-I	ACEI intolerance may lead to a dry cough or angioedema. The most common cause of cough in HF, however, remains cardiac decompensation.
Beta-blockers	May cause bradycardia and cardiac decompensation. Consider holding or reducing dose if HR <50 bpm. Wait 2 – 3 weeks between titrations. Start at lowest dose. Do not give if in acute. decompensation. Reduce, but don't stop entirely during admissions for worsening HF.
MRAs	May cause hyperkalaemia or creatinine rise. Monitor UECs 3-6 monthly. Hold if K+ >5.5 mmol/L and re-start at a lower dose once K drops < 5. Hyperkalaemia more likely in renal dysfunction, older age, or patients also on ACE-I/ARB. Counsel patients to reduce intake of foods with high K+ e.g. bananas and avocados. Spironolactone may cause gynecomastia and such patients can be switched to eplerenone which is a selective MRA blocker and has less androgen side effects.
If-channel inhibitors	Use only if heart rate >70 bpm despite maximally tolerated beta-blocker dose. May precipitate atrial fibrillation. Do not use in atrial fibrillation.
Digoxin	Use primarily in atrial fibrillation when beta-blocker is contraindicated. Digoxin toxicity may occur at high doses or at normal doses in elderly patients or those with renal dysfunction. Digoxin therapy in conjunction with electrolyte abnormalities may lead to malignant arrhythmias. Correct K+, Mg+ and Ca++ for patients on Digoxin. Use max maintenance doses of 0.125 mg per day. In low body weight patients, elderly and those with eGFR < 60, consider lower doses of 0.125mg every 48-72 hours. Digoxin use in sinus rhythm can be attempted in

Guidelines to the diagnosis and management of paediatric heart failure

Paediatric Heart Failure (PHF) is defined broadly as the failure of the heart to supply blood to either systemic or pulmonary circulation at an appropriate rate of flow, or to receive venous return at an inappropriate filling pressure, resulting in adverse effects on the heart, the circulation, and the patient.

Causes of paediatric heart failure

Table 28: Cardiac malformations leading to heart failure

Shunt Lesions

- Ventricular septal defect
- Patent ductus arteriosus
- Aortopulmonary window
- Atrioventricular septal defect
- Single ventricle without pulmonary stenosis
- Atrial septal defect (rare), Total/Partial Anomalous Pulmonary Venous Connection & coronary artery anomalies

Valvular Regurgitation

- Mitral regurgitation
- Aortic regurgitation

Inflow Obstruction

- Cor triatriatum
- Pulmonary vein stenosis & congenital Mitral stenosis

Outflow Obstruction

- Aortic valvular, subvalvular and supravalvular stenosis
- Aortic coarctation

Table 29:Sources of heart failure with a structurally normal heart

Primary Cardiac

- Cardiomyopathy
- Myocarditis
- Myocardial infarction
- Acquired valve disorders- rheumatic fever and rheumatic heart disease
- Hypertension
- Kawasaki syndrome
- Arrhythmia (bradycardia or tachycardia)

Non-Cardiac

- Anaemia
- Sepsis
- Hypoglycaemias
- Diabetic ketoacidosis
- Hypothyroidism
- Other endocrinopathies
- Arteriovenous fistula
- Renal failure
- Muscular dystrophies

The table below lists the types and causes of cardiomyopathy that results in HF in children.

Table 30: Types and causes of cardiomyopathies associated with paediatric heart failure

Dilated cardiomyopathy (systolic dysfunction)	Hypertrophic cardiomyopathy Diastolic dysfunction	Restrictive cardiomyopathy
- **Inflammatory** (viral myocarditis like HIV related and influenza, protozoal like Chagas disease and rickettsia) **Endocrine/metabolic** (hypothyroidism, diabetic, excessive catecholamine, rickets& hypocalcaemia, - **Nutritional deficiency** (kwashiorkor, beriberi, carnitine deficiency) **- Drugs** (doxorubicin, adriamycin) Barth syndrome -Neuromuscular disorder (i.e., Becker dystrophy, Duchenne dystrophy) -Familial DCM	Pompe's diseases Noonan syndrome Maternal diabetes -Mitochondrial diseases -Familial hypertrophic cardiomyopathy and	Endomyocardial fibrosis Idiopathic restrictive cardiomyopathy

Clinical manifestation of paediatric heart failure

Clinical presentation is uniquely related to age:

- **Newborns** – Clinical recognition during this period requires a high index of suspicion with subtle features of:
  - Tachypnoea- RR> 50b/min and tachycar- dia- HR> 150/min
  - Hepatomegaly commonly encountered.
  - Duct dependent pulmonary circulation or right sided lesions present with deep cyanosis and acidosis
  - Duct dependent systemic (left sided lesions) present with heart failure, hypotension and shock.

- **Infants**
  - Respiratory
  - Feeding difficulties characterised by prolonged feeding time> 20minutes, decreased volume intake, irritability with feeding, vomiting after feeds, refusal to feed and excessive sweating (diaphoresis).
- **Older children& adolescence.**
  - Fatigue
  - Shortness of breath
  - Tachypnea
  - Exercise intolerance
  - Abdominal pain
  - Oedema
  - Increased metabolic demands

At any age, unequal pulses in upper and lower limb should be looked for in a child with unexplained heart failure. The severity of heart failure in children must be classified according to Ross modified classification table 4 which recognises the functional classification with increasing severity

Table 31: The Ross Classification of symptomatic severity in paediatric heart failure

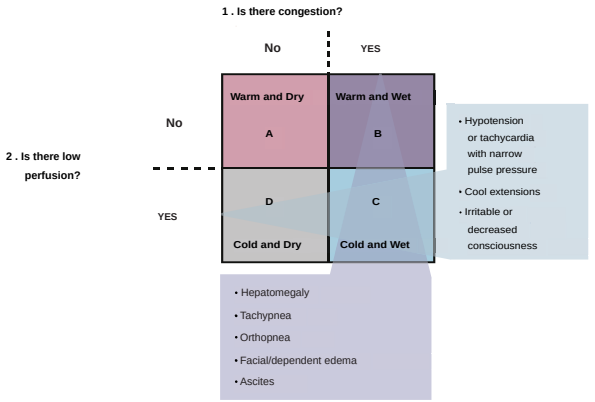
Class of symptoms	Symptoms noted on history
I	Asymptomatic
II	Infants: mild tachypnoea or diaphoresis with feeding; no growth failure Older children: dyspnoea on moderate exertion
III	Infants: marked tachypnoea or diaphoresis with feeding; growth failure Older children: dyspnoea on mild or minimal exertion
IV	Tachypnoea, diaphoresis or respiratory distress at rest

Diagnostic Approach

Clinical Examinations

This involves stepwise guided examinations

Step 1: Identify abnormal clinical findings based on abnormal perfusion or increased fluid congestion as depicted in figure 5.4



Step 2: Rapidly determine the hemodynamic status (Figure 17)

Step 3: Identify any reversible causes of heart failure
- Assessment of electrolyte (Na+ K, Cl, Ca) glucose acid base status, urea creatinine, thyroid hormone, hepatic transaminase
- Screening for hypoxia and sepsis should be done in new-born with HF severe anaemia

Step 4: Investigate for specific underlying disease

Chest Radiography – This is indicated as a first line investigation for children with suspected heart disease to

a) Assess for heart size- Cardiomegaly on chest X ray is highly predictive of ventricular dilatation

b) Check the typical heart shapes like egg on side for transposition of great arteries, wall to wall heart in cardiomyopathy and Ebstein's anomaly, snowman's sign and figure of eight in total anomalous pulmonary venous return

c) Signs of heart failure- Kerley- B lines and pleural effusions

Electrocardiography- Can confirm the tachycardia and type of rhythm, chamber enlargement, and myocardial infarction patterns

Echocardiography - Most useful and widely available and low cost. It should be prioritised.

- It provides immediate data on cardiac morphology and structure, assessment of right and left heart contractile and filling functions
- Assessment of pulmonary pressures
- Provides a guide to appropriate therapy
- Serial echocardiography is useful in surveillance of disease progression and to the response to therapy

Cardiac catheterization – indicated for:

- Accurate diagnosis of complex congenital heart disease
- Accurate evaluation of pressure gradients in patients with complex valve disease and multiple obstructive lesions
- Evaluation of hemodynamic parameter in children with late referral shunt lesions for pulmonary and systemic resistance
- Useful in evaluation of grown up unoperated and palliated patients with congenital heart disease

Biomarkers - Brain natriuretic peptide (BNP) or NT-proBNP levels are useful in distinguishing heart failure from respiratory or other non-cardiac diseases and should be used as a confirmatory test in acute evaluation of paediatric heart failure

Metabolic and genetic testing- Recommended for children with unexplained cardiomyopathy (dilated, hypertrophic or non-compaction type

Drug Therapy in Paediatric Heart Failure

Drug treatment in PHF aims to decrease of pulmonary wedge pressures, increase of cardiac output and the improvement of end organ perfusion and finally to delay of disease progression.

Scenario 1: No structural heart disease with left ventricular systolic dysfunction such as dilated cardiomyopathy
Digoxin – it is useful in symptomatic patients with left and/ or right ventricular systolic dysfunction. Rapid digitalization is not indicated when using digoxin for heart failure but can be used in tachyarrhythmias to slow the heart rate. The oral dose is twice daily for children under 10 years and once daily for children over 10yrs at (8-10ug/kg/day)

ACE inhibitors - they decrease afterload by antagonising the renin angiotensin/ aldosterone system. Should be started at low dose with up titration to the target dose. Captopril is the first choice in infants staring dose 0.1mg/kg/dose gradually increase to 0.5-1mg/kg/dose three times per day, maximum dose 2mg/kg/dose.
Enalapril is useful in older children at a dose 0.1-0.5mg/kg /dose twice daily.

Children treated with ACEIs should be watched for deterioration in renal function and hypotension. Other adverse effects include cough and angioedema. Angiotensin receptor blockers are generally reserved for those children with systemic ventricular systolic dysfunction who would benefit from renin-angiotensin-aldosterone system blockade but are intolerant of ACEIs.

Beta blockers- the addition of beta blocker to above therapy may be useful in patients with left ventricular systolic dysfunction. Low dose therapy should be started with progressive upward titration.
Carvedilol started at **0.05mg/kg/dose** twice daily increased to **0.4-0.5mg/kg/dose** twice daily.
Metoprolol 0.1-0.2mg/kg dose twice daily to be escalated to 1mg twice daily

Scenario 2: Congenital Heart Disease: Volume overload such as large ventricular septal defects (VSDs), patent ductus arteriosus, or endocardial cushion defects

a) Loop diuretics such as furosemide, is recommended for patients with HF and signs and symptoms of congestion. An initial starting dose of **0.5-1 mg/kg** intravenously or orally every 6-12 hours, is safe and effective with a maximum dose of **4mg/kg/day**

b) Patients who are unresponsive to loop diuretics alone may benefit from addition of a thiazide agent

c) Aldosterone antagonist (spironolactone) therapy is used in children with chronic systolic HF. The starting dose of spirono lactone is **1mg/kg/day**, and the target maximum dose is **2 mg/kg/day.** Male gynecomastia can occur with spironolactone requiring discontinuation.

Scenario 3: Congenital Heart Disease: Pressure Overload

Includes critical aortic stenosis, severe pulmonary stenosis, coarctation of aorta.

In the new-born period presents as duct dependent lesions and requires prostaglandins to maintain the ductus as a bridge to catheter interventions. Prostaglandin E1 is given by intravenous infusion starting 0.05-0.1ug/kg/min and once desired dose achieved can be reduced to 0.05 to 0.01ug/kg

Table 32: Drugs used in paediatric heart failure.

Drugs	Routes of administration	Doses
Furosemide	Oral	1-2 mg/kg q6-12h
Furosemide	Intermittent bolus	0.5-2 mg/kg q6-12h
Furosemide	Continuous infusion	0.1-0.4 mg/kg/h
Captopril	Oral	0.3-2 mg/kg q8h
Enalapril	Oral	0.05-0.25 mg/kg q12h
Losartan	Oral	0.5-1.5 mg/kg/d
Carvedilol	Oral	0.05 mg/kg/d q12h
Metoprolol	Oral	0.25 mg/kg/d q12h
Spironolactone Oral		0.5-1.5 mg/kg q12h
Nitroglycerin	Continuous infusion	0.5-10 mg/kg/min
Nitroprusside	Continuous infusion	0.5-4 mg/kg/min
Hydralazine	Intermittent bolus	0.1-0.2 mg/kg every 4-6 h
Hydralazine	Oral	0.3-1 mg/kg/d in q8 -12h
Digoxin	Oral	5-10 mg/kg/d
Dobutamine	Continuous Infusion	2.5-10 mg/kg/min
Epinephrine	Continuous Infusion	0.01 - 0.1 mg/kg/min
Epinephrine	Intermittent bolus	0.01 mg/kg
Milrinone	Continuous Infusion	0.5 -1 mg/kg/min
Levosimendan	Continuous Infusion	0.05 -0.2 mg/kg/min

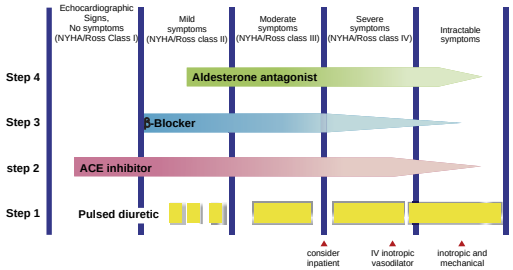


Figure 18: Stepwise introduction of medical therapy in heart failure

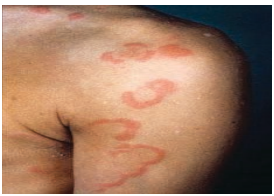
Rheumatic Fever and Rheumatic Heart Disease

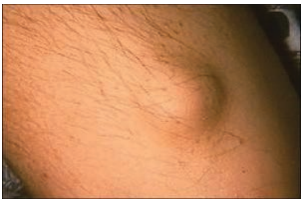
Rheumatic Heart Disease (RHD) is a chronic heart condition caused by acute rheumatic fever (ARF)

Clinical Manifestations

RF causes joint pains, fever, skin changes and sometimes abnormal movements. In most cases the heart also becomes inflamed during RF. However, when other symptoms of RF resolve, changes to the heart valves persist. Further features are shown in the table below:

Table 33: Manifestations of Acute Rheumatic Fever

Manifestation	Description
Carditis	Is the only manifestation that has the potential for long -term complications. It usually manifests as a pancarditis involving the endocardium, myocardium and pericardium. It presents as a new murmur, cardiac enlargement, congestive heart failure, pericardial friction rub, and/or pericardial effusion
Arthritis	Inflammation of the synovial membranes of several joints characterized by swelling, redness, warmth and pain. Usually presents as polyarthritis which is migratory in nature. Mostly affects the larger joints, including the knee, ankles and elbows and wrists. It rapidly improves on NSAIDS. Usually runs a self-limited course lasting ≈4 weeks Monoarthritis may be a presenting feature in high-risk populations
Sydenham's chorea	Chorea in ARF is characterized by purposeless, involuntary, nonstereotypical movements of the trunk or extremities. It often is associated with muscle weakness and emotional lability.
Erythema marginatum	Distinctive rash marked by the presence of pink rings in the torso or upper(proximal) parts of the body; can appear and disappear within minutes 

Subcutaneous nodules	Small painless lumps under the skin. Usually present over the elbows, wrists, knees, ankles, achilles tendon, occiput and posterior spinal processes of the vertebrae. These are uncommon, and represent severe carditis. 
Minor manifestations	
Clinical	Fever, polyarthralgia
Laboratory	Elevated acute phase reactants (erythrocyte sedimentation rate or leukocyte count)
Electrocardiogram	prolonged P-R interval
Supporting evidence of a preceding streptococcal infection within the last 45 days	
— elevated or rising antistreptolysin-O or other streptococcal antibody, or — a positive throat culture, or — rapid antigen test for group A streptococci, or — recent scarlet fever	

Diagnosis

The diagnosis of ARF is usually guided by the Jones criteria and the more recent World Health Organization (WHO) criteria. The clinical diagnosis of carditis usually depends on detecting: (i). myocarditis (ii). Pericarditis, and (iii). Valve regurgitation. Carditis as a major manifestation of ARF has been a clinical diagnosis based on the auscultation of typical murmurs that indicate mitral or aortic valve regurgitation at either valve or both valves.

To increase sensitivity of ARF diagnosis current evidence now support the use of echocardiography/doppler as part of the diagnostic criteria for confirmation of the presence of carditis in patients with suspected ARF. Accordingly, in the Revised Jones Criteria for the diagnosis of ARF in the era of Doppler Echocardiography recommends: -

i. Echocardiography with Doppler should be performed in all cases of confirmed and suspected ARF.
ii. It is reasonable to consider performing serial echocardiography/ Doppler studies in any patient with diagnosed or suspected ARF even if documented carditis is not present on diagnosis.
iii. Echocardiography/Doppler testing should be performed to assess whether carditis is present in the absence of auscultatory findings, particularly in moderate-to high-risk populations and when ARF is considered likely.
iv. Echocardiography/Doppler findings not consistent with carditis should exclude that diagnosis in patients with a heart murmur otherwise thought to indicate rheumatic carditis.

Diagnostic criteria for Rheumatic Fever and RHD

Any 1 of the following can serve as evidence of a preceding GAS infection, as per a recent AHA statement:

1. Increased or rising anti-streptolysin O titer or other streptococcal antibodies (anti-DNASE B). A rise in titer is better evidence than a single titer result.
2. A positive throat culture for group A β-hemolytic streptococci.
3. A positive rapid GAS carbohydrate antigen test in a child whose clinical Presentation suggests a high pretest probability of streptococcal pharyngitis.

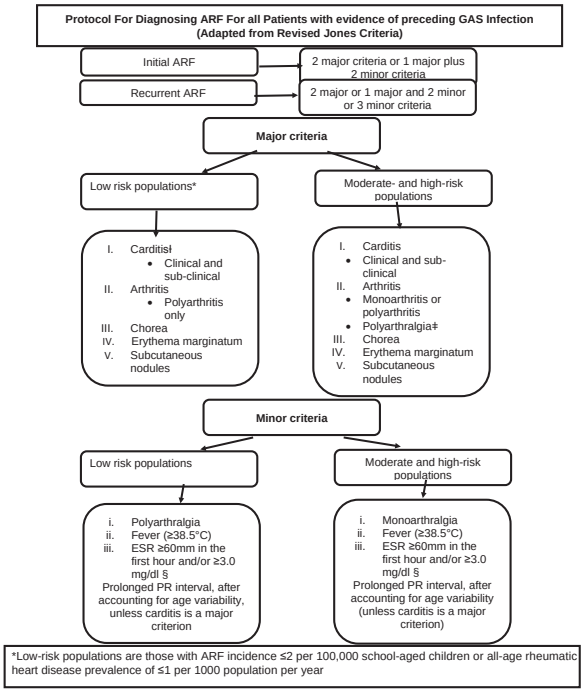


Figure 19: Diagnosis strategy for acute rheumatic fever with Echocardiography findings. *Subclinical carditis can be considered. Alt - alternative; ARF - acute rheumatic fever; echo - echocardiography; GAS - group A streptococcal; neg - negative

Differential diagnosis

Table 34: Differential diagnosis of Arthritis, Carditis and Chorea

Arthritis	Carditis	Chorea
· Septic arthritis (including gonococcal) · Connective tissue and other autoimmune diseases such as juvenile idiopathic arthritis · Viral arthropathy · Reactive arthropathy · Lyme disease · Sickle cell anaemia · Infective endocarditis · Leukaemia or lymphoma · Gout and pseudogout · Poststreptococcal reactive arthritis · Henoch-Schonlein purpura	· Physiological mitral regurgitation · Mitral valve prolapse · Myxomatous mitral valve · Fibroelastoma · Congenital mitral valve disease · Congenital aortic valve disease · Infective endocarditis · Cardiomyopathy · Myocarditis, viral or idiopathic · Kawasaki disease	· Drug intoxication · Wilson disease · Tic disorder · Choreoathetoid cerebral palsy · Encephalitis · Familial chorea (including · Huntington disease) · Intracranial tumor · Lyme disease · Hormonal · Metabolic (e.g. Lesch-Nyhan, hyperalaninemia, ataxia teleangiectasia) · Antiphospholipid syndrome · Autoimmune: Systemic lupus erythematosus, systemic vasculitis · Sarcoidosis · Hyperthyroidism

Management of Acute Rheumatic Fever

Rheumatic carditis is primarily a valvulitis rather than a myocarditis.

- The mainstay of treatment is supportive
- Bed rest, decreased physical activity, high nutrition
- Medical management
- Anti-inflammatory treatment with steroids in severe cases. The ubiquitous use of anti-inflammatory agents is thought to have masked many cases of ARF. There is still no convincing evidence that this changes the course nor natural history of the disease
- Symptomatic treatment of cardiac failure using anti-failure medication is required in severe cases. In the worst case scenario, surgery may need to form part of acute treatment
- Adequate penicillin for eradication for GAS

Management of RF/RHD involves four main steps:-

i. Primary prevention- eradication of streptococci and prevention of new infections
ii. Anti-inflammatory treatment
iii. Supportive treatment and management of complications
iv. Secondary prevention- prevention of recurrent attacks

The figure below summarizes the opportunities for prevenstion of acute rheumatic fever and heart disease.

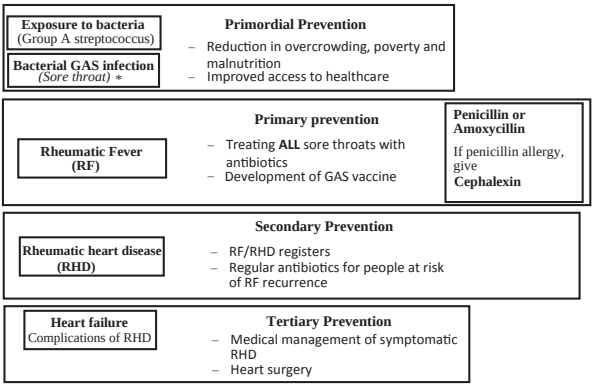


Figure 20: Opportunities for intervention for RF and RHD

Treatment of streptococcal pharyngitis/Antimicrobial therapy

The goal of therapy is eradication of the pharyngeal streptococcal infection which is mandatory to avoid chronic repetitive exposure to streptococcal antigens. Ideally, two throat cultures should be performed before starting antibiotics. However, antibiotic therapy is warranted even if the throat cultures are negative. Antibiotic therapy does not alter the course, frequency and severity of cardiac involvement (3).

Table 35: Medications for streptococcal pharyngitis

Agent	Pediatric dosage	Adult Dosage	Route of administration	Duration
Benzathine penicillin	<27kg: 0.6 MU STAT	1.2 MU stat	IM	
Amoxycillin	Mild to moderate pharyngitis12.25 mg/kg BDOr 10mg/kg TDSSevere pharyngitis22.5 mg/kg BDOr 13.3 mg/kg TDS	500mg TDS	PO	10 days
Erythromycin ***(if penicillin allergies)***	30-50mg/kg per day in 2-4 divided doses	500mg QID	PO	10 days
Azithromycin		500mg OD	PO	5 days

Anti-inflammatory therapy

Salicylates are the preferred agents, although other nonsteroidal agents are probably equally efficacious. Steroids are also effective but should probably be reserved for patients in whom salicylates fail.

Table 36: Priorities in managing ARF

Admission to hospital
Admit all patients suspected to have ARF
Confirmation of the diagnosis
Observation prior to anti-inflammatory treatment; Paracetamol (first line) or codeine for fever or joint pain **Investigations**
Treatment
All cases **Single-dose IM BPG (preferable) or 10 days' oral penicillin V (IV not needed; oral erythromycin if allergic to penicillin)**
Arthritis and fever **Paracetamol (first line) or codeine until diagnosis confirmed** **Aspirin, naproxen or ibuprofen once diagnosis confirmed, if arthritis or severe arthralgia present** **Mild arthralgia and fever may respond to paracetamol alone** **Influenza vaccine for children receiving aspirin during the influenza season (autumn/winter)**
Chorea **No treatment for most cases** **Carbamazepine or valproic acid if treatment necessary**
Carditis/heart failure **Bed rest, with mobilization as symptoms permit** **Urgent echocardiogram** **Antifailure medication** • **diuretics/fluid restriction for mild to moderate failure** • **ACE inhibitors for more severe failure, particularly if AR present. Glucocorticoids optional for severe carditis (consider treating for possible opportunistic infections)** • **digoxin, if AF present** **Valve surgery for life - threatening acute carditis (rare)**
Long - term preventive measures
First dose of secondary prophylaxis **Notify case to ARF/RHD register, if available** **Contact local primary care staff to ensure follow-up** **Referral to a medical specialist** **Provide culturally - appropriate education to patient and family** **Arrange dental review and ongoing dental care to reduce risk of endocarditis**

ACE, angiotensin converting enzyme; AF, atrial fibrillation; AR, aortic regurgitation; BPG, benzathine penicillin G; IM, intramuscular; IV, intravenous

Treatment of Acute Rheumatic Fever

The table below lists the medications, indications dosage and duration of treatment for ARF.

Table 37: Medications Used In Management Of Acute Rheumatic Fever In Children

Medication	Indication	Regimen	Duration
BPG, im	Treat streptococcal infection	900 mg (1,200,000 U) ≥20kg 450 mg (600,000 U) <20kg	Single dose
Or phenoxymethylpenicillin po (penicillin V)	Initial treatment of streptococcal infection	Child: 250 mg, bd Adolescents and adults: 500 mg/bd	10 days
Or erythromycin ethyl succinate, po **(only if allergic to penicillin)**	Initial treatment of streptococcal infection	Child: 20 mg/kg up to 800 mg, bd Adult: 800 mg, bd	10 days
Or Erythromycin, po **(only if allergic to penicillin)**	Initial treatment of streptococcal infection	Child: 12.5 mg/kg upto 500 mg, bd Adult: 500 mg, bd	10 days
Paracetamol, po	Arthritis or until diagnosis confirmed Arthralgia	60 mg/kg/day (max 4 g) given 4-6 doses/day; may increase to 90 mg/kg/day, if needed, under medical supervision	Until symptoms relieved or NSAID started
Aspirin, po	Arthritis or severe arthralgia (when ARF diagnosis confirmed)	Begin with 50-60 mg/kg/day, increasing, if needed, up to 80-100 mg/kg/day (4-8 g/day in adults) given in 4-5 doses/day If higher doses required, reduce to 50-60 mg/kg/day when symptoms improve, and cease when symptom free for 1-2 weeks Consider ceasing in the presence of acute viral illness, and consider influenza	Until joint symptoms relieved

Naproxen, po	Arthritis or severe arthralgia (when ARF diagnosis confirmed)	10-20 mg/kg/day (max 1250 mg) given, bd	As for aspirin
Ibuprofen, po	Arthritis or severe arthralgia (when ARF diagnosis confirmed)	30 mg/kg/day (max 1600 mg) given tds	As for aspirin
Prednisone or prednisolone, po	Severe carditis, heart failure, pericarditis with effusion	1-2mg/kg/day (max. 80 mg); if used > 1 week, taper by 20-25% per week	Usually 1-3 weeks
Frusemide, po/iv (can also be given im)	Heart failure	Child: 1-2mg/kg stat, then 0.5-1mg/kg/dose 6-24 hourly(max 6mg/kg/day Adult: 20-40mg/dose, 6-24 hourly, up to 250-500 mg/day	Until failure controlled and carditis improved
Spironolactone,po	Heart failure	1-3mg/kg/day (max 100-200 mg/day) in 1-3 doses; round dose to multiple of 6.25mg (1/4 of a tablet)	As for frusemide
Enalapril. Po	Heart failure	Child: 0.1 mg/kg/day in 1-2 doses, increased gradually over 2 weeks to max of 1 mg/kg/day in 1-2 doses Adult: initial dose 2.5 mg daily, maintenance dose 10-20mg daily (max 40mg)	As for frusemide
Captopril, po	Heat failure	Child: initial dose 0.1 mg/kg/dose. Beware of hypotension. Increase gradually over 2 weeks to 0.5-	As for frusemide
Valproic acid, po	Severe chorea (may affect salicylate metabolism)	Usually 15-20mg/kg/day (can increase to 30mg/kg/day) given tds	As for carbamazepine

Bd - twice daily; BPG - benzathine penicillin G; im - intramuscular; iv - intravenous; NSAID - non-steroidal anti-inflammatory drug; po - per oral; tds - three times daily

Guidelines for Secondary Prophylaxis

Length of time for secondary prophylaxis depends on a number of factors including

- Age at first diagnosis of ARF (or RHD)
- Severity of disease
- If carditis was present with first ARF
- Time (years) since last ARF illness
- Ongoing risk factors (e.g. level of poverty)
- If medication is received regularly

World Health Organisation guidelines for secondary prophylaxis duration:

Disease Classification	Duration of secondary prophylaxis
ARF (no carditis)	Minimum of 5 years after last ARF, or Until age 18 years (whichever is longer)
Mild-moderate RHD (or healed carditis)	Minimum of 10 years after last ARF, or Until age 25 years (whichever is longer)
Severe RHD and after Surgery	Continue for life

Below are guidelines on how to institute secondary prohylaxis for ARF.
In pregnant patients, penicillin prophylaxis should continue for the duration of pregnancy to prevent recurrent ARF. There is no evidence of teratogenicity. Erythromycin is also considered safe in pregnancy.

Table 38: Selection of therapy for secondary prevention of rheumatic fever

Agent	Dose	Mode of administration
Benzathine Penicillin	Patients weighing 27 kg (60 lb) or less: 600,000 units IM every 4 weeks Patients weighing more than 27 kg: 1,200,000 units IM every 4 weeks	IM
Penicillin V	250 mg BD	PO
Azithromycin ***(if penicillin allergies)***	250mg OD	PO
Erythromycin	250mg BD	PO

Chronic Rheumatic Heart Disease

Chronic RHD is sequelae of poorly-managed or undiagnosed RF. It evolves over 2-10 years following repeated episodes of RF. The hallmark of this condition is the development of valvular heart lesions.

Clinical presentation

The various presentations of chronic RHD are summarized in table below.

Table 39: Valvular lesions in chronic RHD with recommendation

.l	Symptoms	Signs	Recommendation
Mitral stenosis	-Fatigue -Dyspnoea -Palpitation -Cough Hemoptysis Edema Ascites Chest pain	Atrial fibrillation -Mitral facies (flushed cheeks) -Crepitations -Diastolic murmur -Loud P2	Maintain airway, breathing and circulation. Then refer to a physician/ Paediatrician for definitive diagnosis and management plan.
Mitral regurgitation	-Fatigue -Cough -Palpitation -Edema -Ascites -Dyspnoea	-Atrial fibrillation -Cardiomegaly -Apical pansystolic murmur -Crepitations -Signs of RHF (edema, Ascites,hepatomegaly)	Maintain airway, breathing and circulation. Then refer to a physician/ paediatrician for definitive diagnosis and management plan.
Aortic regurgitation	-if mild to moderate it is asymptomatic -Palpitation -Breathlessness -Angina	-Large volume or collapsing pulse -Femoral Bruit (doroziez's sign) -Capillary pulsation (quincke's) -Bounding periphery pulse -Head nodding (demusset's signs) -Murmurs sytolic murmurs (soft mid diastolic murmur (Austin flint murmur)	Maintain airway, breathing and circulation. Then refer to a physician/paediatrician for definitive diagnosis and management plan.

Aortic Stenosis	Mild to moderate –asymptomatic -Dyspnoea -Angina -Exertion syncope -Angina -Episodes of acute pulmonary oedema	-Injection systemic murmur -Slow carotid pulse -Narrow pulse pressure -Thrusting apex beat -Crepitations	Maintain airway, breathing and circulation. Then refer to a physician/paediatrician for definitive diagnosis and management plan.
Tricuspid regurgitation/ Tricuspid Stenosis	Edema Ascites Exercise intolerance→ Angina (rare; due to RV overload and strain) Symptoms of heart failure if is underlying cause	- S3 gallop - Jugular venous distention with a prominent V wave -In some patients, a pansystolic murmur -Diminished peripheral pulse volume secondary to impaired forward blood flow; -Right ventricular heave and S 4 gallop that increases with inspiration -Ascites -Peripheral edema -Cachexia and jaundice -Atrial fibrillation -Pulmonary rales, if TR is associated with LV dysfunction or MS	Maintain airway, breathing and circulation. Then refer to a physician/paediatrician for definitive diagnosis and management plan.

Infective Endocarditis

Definition: Infective endocarditis (IE) is defined as an infection of the endocardial surface of the heart, which may include one or more heart valves, the mural endocardium, or a septal defect.

Risk factors: Cardiac conditions at high risk of endocarditis for which prophylaxis should be considered prior to a high-risk procedure include: **a.** Patient with a prosthetic valve; **b.** patients with previous episode of IE; **c.** Patients with any type of cyanotic CHD; **d.** Any type of CHD repaired with prosthetic material

Classification of IE: Five main types

i. Native valve endocarditis (NVE) . Caused by RHD and cyanotic CHD. Organisms include streptococcus spp(70%) ans staphylococcus spp (25%)
ii. Prosthetic valve endocarditis (PVE) . Main organisms are Staph. Aureus, Corynebacterium, HACEK spp
iii. Intravenous drug abuse (IVDA) endocarditis
iv. Nosocomial/healthcare-associated endocarditis: Due to intravascular devices (e.g. central lines/catheters, peripheral intravenous catheters) or Intracardiac devices (e.g. pacemakers)

The table below summarizes the signs and symptoms of Infective Endocarditis

Table 40: Signs and symptoms of IE

Category	Signs/symptoms
Septic signs	Fever, rigors, night sweats, weight loss anaemia, splenomegaly, finger clubbing
Cardiac lesions	Any new murmur or a changing existing murmur,congestive cardiac failure
Immune complex deposition/vasculitis	Hematuria; glomerulonephritis, roth spots, splinter hemorrhages, Osler's nodes
Embolic phenomena	Abscesses in organs e.g brain, heart, kidney,spleen,gut or skin (Janeway lesions)

Investigations

i. Blood cultures (At least three sets are taken at 30-min intervals)
ii. FBC (anaemia, leukocytosis)
iii. ESR(elevated in 90% cases)
iv. Urinalysis (proteinuria, microscopic hematuria),
v. Echocardiography (visible vegetations, abscesses),
vi. 2-D Doppler ultrasonography- vegetations, valvular thrombi
vii. Other studies: Chest radiography(pulmonary pyogenic lesions), Head CT scan (CNS manifestations)

Diagnosis: Use the Modified Duke Criteria

Modified Duke Criteria for infective endocarditis

Major Criteria

Major blood culture criteria

- Two blood cultures positive for organisms typically found in patients with IE
- Blood cultures persistently positive for typical organisms from cultures drawn more than 12 hours apart
- Three or more separate blood cultures drawn at least 1 hour apart

Major echocardiographic criteria

- Echocardiogram positive for IE (intracardiac vegetation, myocardial abscess, dehiscence of a prosthetic valve
- New-onset valvular regurgitation

Minor criteria

- Predisposing heart condition or intravenous drug use
- Fever of 38°C (100.4°F) or higher
- Vascular phenomenon(embolic, vasculitis)
- Immunological phenomenon
- Positive blood culture results not meeting major criteria
- Echocardiogram results consistent with IE but not meeting major echocardiographic criteria

How to diagnose: 2 major, I major and 3 minor, or 5 minor criteria

Management

Emergency treatment (stabilize the patient): Oxygen therapy, treatment of CCF; hemodialysis

Antibiotic therapy: Mainstay of management for IE. Treatment is essentially always parenteral; oral therapy is less desirable.

Table 41: Antibiotic treatment of inective endocarditis due to streptococcus group

Antibiotic	Dosage and route	Comments
Strains penicillin - susceptible (MIC ≤ 0.125 mg/L) oral and digestive streptococci		
Standard treatment: 4-week duration		
Penicillin G Or Cefriaxone	12-18 million U/day i.v either in 4-6 doses or continuously 2g/day i.v. or i.m. in 1 dose **Pediatric doses** Penicillin G 200,000 U/kg/day i.v. in 4-6 doses Cefriaxone 100 mg/kg/day i.v. or i.m. in 1 dose	Preferred in patients. 65 years or with impaired renal or VIII (Vestibulocochlear) cranial nerve functions. 6-week therapy recommended for patients with PVE
Standard treatment: 2-week duration		
Penicillin G or Ceftriaxone combined with Gentamicin	12–18 million U/day i.v. either in 4–6 doses or continuously 2 g/day i.v. or i.m. in 1 dose 3 mg/kg/day i.v. or i.m. in 1 dose **Paediatric doses** Penicillin G and ceftriaxone as above Gentamicin 3 mg/kg/day i.v. or i.m. in 1 dose or 3 equally divided doses	Only recommended in patients with non-complicated NVE with normal renal function.
In beta-lactam allergic patients[j]		
Vancomycin	30 mg/kg/day i.v. in 2 doses **Paediatric doses:** Vancomycin 40 mg/kg/day i.v. in 2 or 3 equally divided doses	6-week therapy recommended for patients with PVE
Strains relatively resistant to penicillin (MIC 0.250–2 mg/l)[k]		
Standard treatment		
Penicillin G or	24 million U/day i.v. either in 4– 6 doses or continuously	6-week therapy recommended for patients with PVE

Antibiotic prophylaxis in high-risk patients:

- Dental procedures requiring manipulation of the gingival or periapical region of the teeth or perforation of the oral mucosa
- Before placement of a prosthetic valve, pacemaker or other intracardiac devices

Regimen for Prophylaxis

Table 42: Recommended prophylaxis for high-risk dental procedres in high-risk patients

Situation	Antibiotic	Single-dose 30-60 minutes before procedure	
		Adults	**Children**
No allergy to penicillin	**Amoxicillin** **or** **Ceftriaxone**	**2g orally or i.v.** **1 g i.v.**	**50mg/kg orally** **50 mg/kg i.v.**
Allergy to penicillin	**Clindamycin**	**600 mg orally or i.v.**	**20 mg/kg orally or i.v.**

Congenital Heart Disease in Children and Adults

Definition: A range of birth defects that affect the normal workings of the heart. It may be referred to as a structural anomaly of the heart or great vessels that is or could be of functional significance.

Aetiology: Conditions occurring in pregnancy: infections (Toxoplasmosis, Rubella, Parvovirus B19, Herpes, Varicella, Syphilis, Cytomegalovirus), Chromosomal abnormalities (e.g Downs syndromes), Phenylketonuria, Alcohol abuse, Anti-convulsion medications (phenytoin sodium, benzodiazepines), Acne medications (topical retinoid and isotretinoin), Organic solvents (nail polish/glue), poorly controlled diabetes)

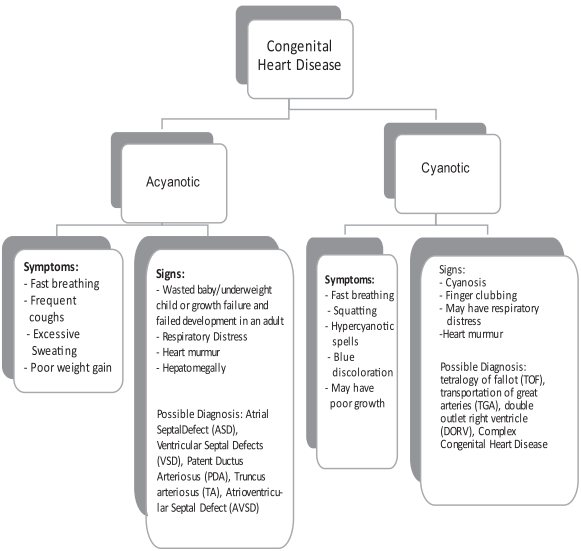


Figure 21: Classification of congenital heart disease

Recommended action when a potential case is detected is to refer to level 4 facility to see Pediatrician for definitive diagnosis and management plan.

Further Recommendations

i. **Infant screening for CHD.**
All infants should be subjected to a simple screening process as follows:
a. Midwife to auscultate the babies heart with a stethoscope
b. Pulse oximetry – see below protocol

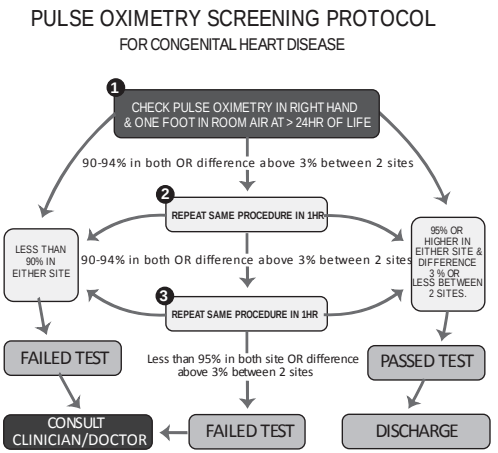


Figure 21: Protocol for screening CHD in neaonates

Babies suspected to have CHD should be reviewed by a clinician before discharge.

2. Counselling of the patient and family by health care provider should include education on:

a. Endocarditis prophylaxis measures where necessary(see section on IE)

b. Recommendations for physical exertion based on the patient's ability,underlying haemo-dynamics, and the risk of acute decompensation/arrhythmias.

c. Contraception and D information
All women with CHD should receive pre-pregnancy counselling for assessment of fetal risk, risk of prematurity or low birth weight in the offspring, review of medications that may be harmful to the fetus, appropriate management of antico agulation, and discussion of potential maternal complications should be done before pregnancy.
Health providers managing pregnant patients with CHD should have a plan for management of labor and the postpartum period that includes consideration of the appropriate response to potential complications.

d. Advice on healthy lifestyle (smoking cessation, weight loss/ maintenance, hypertension/lipid screening).

Venous Thromboembolism

Venous thromboembolism(VTE) constitutes two serious medical conditions, Deep Venous Thrombosis(DVT) and Pulmonary Embolism(PE). Missed diagnosis or delayed treatment can lead to death or long-term complications like pulmonary hypertension and post-thrombotic syndrome(1).

Risk factors

Virchow proposed three major pathophysiologic determinants of VTE: venous stasis, endothelial injury and hypercoagulability.

Table 43: Classification of Risk Factors of VTE

Strong	Moderate	Weak
Fracture (hip or leg)	Arthroscopic knee surgery	Bed rest >3 days
Hip or knee replacement	Central venous lines	Immobility due to sitting (e.g. prolonged car or air travel)
Major general surgery	Chemotherapy	Increasing age
Major trauma	Congestive heart or respiratory failure	Laparoscopic surgery (e.g. cholecystectomy)
Spinal cord injury	Hormone replacement therapy	Obesity
	Malignancy	Pregnancy/ antepartum
	Oral contraceptive therapy	Varicose veins
	Paralytic stroke	
	Pregnancy/, postpartum	
	Previous venous thromboembolism	
	Thrombophilia	

Diagnosis of DVT

1. Clinical Signs and Symptoms

DVT most commonly develops in the leg veins, but can occasionally also occur in the upper extremities, especially if there is an in-dwelling central venous catheter or a hemodialysis catheter. Common symptoms of proximal lower limb DVT include:

- Sudden onset of unilateral leg pain or tenderness of the thigh or calf.
- Leg swelling (oedema)
- Skin that feels warm to the touch

2. Clinical Probability Testing in suspected DVT

There are many scoring systems to assist diagnosis, but many are difficult to implement. The Wells' Score is a simplified prediction tool for DVT to help the busy clinician working in limited resource settings.

Table 44: Wells' Score for suspected DVT

Clinical features	Score
Active cancer (treatment within last 6 months or palliative)	1 point
Paralysis, paresis, or recent plaster immobilization of leg	1 point
Recently bed-ridden for >3d or major surgery in last 12wks	1 point
Local tenderness along distribution of deep venous system	1 point
Entire leg swollen	1 point
Calf swelling >3cm compared with asymptomatic leg (measured 10cm below tibial tuberosity)	1 point
Pitting edema (greater in the symptomatic leg)	1 point
Collateral superficial veins (non-varicose)	1 point
Previously documented DVT	1 point
Alternative diagnosis** at least as likely as DVT	-2 points
Note: In patients with symptoms in both legs, the more symptomatic leg is used ***≥2 points = DVT likely:*** Perform ultrasound; if positive, treat as DVT. If ultrasound is unequivocal, do D-dimer test. If D-dimer positive and ultrasound negative, repeat ultrasound in 1 week. If both D-dimer and ultrasound negative, DVT excluded ***<2 point = DVT unlikely:*** Perform D-dimer (where available). If negative, DVT excluded. If positive, proceed to Ultrasound (if ultrasound negative, DVT excluded; if positive, treat as DVT).	

** An alternative diagnosis may include: superficial phlebitis, post-thrombotic syndrome, cellulitis, muscle strain or tear, leg swelling in paralyzed limb, venous insufficiency, edema due to CCF or cirrhosis, external venous obstruction (e.g. due to tumor), lymphangitis or lymphedema, popliteal (Baker's) cyst, hematoma, pseudo aneurysm or knee abnormality

3. Imaging in DVT

The gold standard test for diagnosing lower or upper extremity DVT is venous compression ultrasonography (CUS). Failure to compress the vein is diagnostic of DVT.

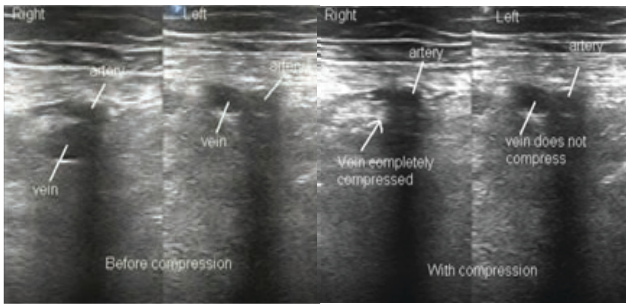


Figure 22: Ultrasonography images of femoral vessels before and with compression

Alternative imaging using CT, MRI and contrast venography are reserved for imaging segments that are not easily assessed by CUS, e.g. pelvic veins, or subclavian vein DVT.

4. Laboratory testing in DVT

Plasma D-dimer is a non-specific marker of fibrin lysis anywhere in the body. It may be elevated in VTE but also in many other conditions, including Myocardial Infarction (MI), heart failure, infection, surgery, aortic dissection and pregnancy. The D-dimer test is very useful in the evaluation of outpatients or in casualty setting in patients with suspected VTE. The D-dimer test may be positive but CUS shows no evidence of proximal DVT. In this subgroup of patients, it is advised to repeat CUS in 7 days to rule out DVT

5. Diagnostic algorithm for suspected DVT

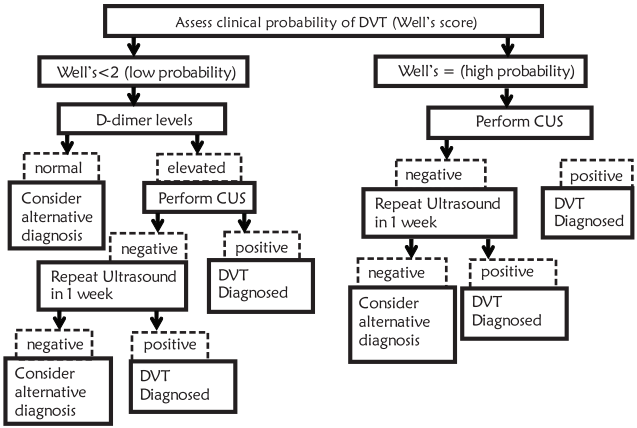


Figure 23: Algorithm for diagnosis of suspected DVT); Source: BMJ Evidence Center

Diagnosis of Acute Pulmonary Embolism

1. Clinical Presentation

The clinical presentation of acute PE may vary widely and the signs and symptoms are often non-specific. Common features include dyspnea, cyanosis or fainting in massive acute PE. Large thrombi obstruct the pulmonary arterial tree and cause hemodynamic and gas exchange abnormalities, resulting in hypotension, hypoxemia and increased Right Ventricular afterload. Clinical features may include the following:-

- Unexplained sudden onset of dyspnea
- Lightheaded sensation or syncope
- Pleuritic chest pain
- Pulse≥ 100 beats/minute
- Heart murmur of tricuspid regurgitation, loud P2
- Distended neck veins.

2. Assessment of clinical probability in acute PE

The combination of clinical findings and the use of prediction rules can help to classify patients with suspected PE into categories of probability that correspond to an increasing actual prevalence of confirmed PE on further testing.

Table 45: Probability tool for possible PE

Clinical Feature	Score
Previous PE or DVT	1
Pulse ≥ 100 beats/minute	1
Surgery or immobilization within the past 4 weeks	1
Hemoptysis	1
Active cancer	1
Clinical signs of DVT	1
Alternative diagnosis less likely than PE	1
Clinical Probability	
PE likely	≥2
PE unlikely	0-1

3. Radiologic imaging in Acute PE

CT pulmonary angiography is recommended in outpatients with an elevated plasma D-dimer and in patients with a high clinical probability.

4. Laboratory Testing in suspected Acute PE

- Plasma D-dimer testing (combined with clinical probability assessment) should be carried out in patients with suspected acute PE in the absence of shock. Not for patients with high clinical probability or in hospitalized patients.
- Cardiac biomarker testing (NT-Pro BNP or Troponin) may be useful in risk stratification of severity of acute PE in patients with hypotension. Poorer prognosis if elevated.

6. Predicting early mortality in acute PE

Features indicating higher 30-day mortality in acute PE:

- Shock
- Pulmonary Embolism Severity Index(PESI) Class
- Signs of right ventricular dysfunction on Echocardiography or CT imaging
- Elevated cardiac biomarkers

Prevention of Venous Thromboembolism

The use of VTE prophylaxis should be considered in all hospitalized patients. Patients should be assessed for VTE risk for all medical and surgical patients, in order to identify at-risk patients in whom VTE prophylaxis can commence.

1. Pharmacological Thrombo-prophylaxis

Recommendations for hospitalized patients at risk of VTE :-

- Enoxaparin 40mg SQ OD
- Unfractionated Heparin 5000 SQ BD
- Fondaparinux (where available) 2.5mg SQ OD

2. Mechanical Thromboprophylaxis

Graduated compression stockings/Anti-embolism stockings and intermittent pneumatic compression devices (where available) for at-risk patients who are NOT candidates for pharmacological thromboprophylaxis due to high risk of bleeding pre-operatively, intra-operatively and post operatively. Mechanical prophylaxis is effective when used in combination with early ambulation.

Standards for the use of anti-embolism stockings (15)

- Use correctly fitting stocking
- Use stockings that provide graduated compression and produce a calf pressure of 14–15 mmHg
- Encourage patients to wear them day and night until they no longer have significantly reduced mobility
- Remove stockings daily for hygiene purposes and to inspect skin condition.
- In patients with a significant reduction in mobility, poor skin integrity or any sensory loss, inspect the skin two or three times per day, particularly over the heels and bony prominences
- Discontinue use if there is marking, blistering or discoloration of the skin, particularly over the heels and bony prominences, or if the patient experiences pain or discomfort.
- If suitable, offer intermittent pneumatic compression (IPC) device as an alternative.

Standards for Intermittent Pneumatic Compression

- Proper fitting of the device
- Compression pressure and cycling time: IPC pressure of 120–140mmHg applied to foot and calf with a one-second delay, and at a frequency of three to four impulses per minute

Contraindications to mechanical prophylaxis

- Peripheral artery disease, including history of peripheral arterial bypass grafting
- Severe peripheral neuropathy or other cause of sensory impairment
- Allergy to stocking material
- Massive leg edema or pulmonary edema from congestive cardiac failure
- Local skin or soft-tissue condition, including recent skin graft, fragile skin, gangrene, severe dermatitis and cellulitis
- Extreme deformity of the leg, or unusual leg shape or size preventing correct fit
- Documented DVT

VTE Treatment and Patient Management

i. Anticoagulation

Mainstay of therapy. It is administered sequentially as follows:

- Initial anticoagulation in patients with acute DVT and PE
- Long-term anticoagulation (usually up to 3 months)
- Extended- treatment (beyond 3 months in select patients)

Table 46: Medications used in VTE treatment

Drug option	Dosage	Comments
Low molecular weightHeparin (LMWH)	Enoxaparin 1 mg/kg SQ BD**Renal Dose:**Reduce dose in patients with severe renal insufficiency (GFR< 30ml/min): 1 mg/kg SQ OD	No routine lab monitoring required in hospital Safe and effective as UFH when startingtherapy. Preferred over
Fondaparinux	**Wt < 50kg**: 5mg SQ OD x 5 days **Wt 50-100 kg**: 7.5mg SQ OD x 5d **Wt >100kg**: 10mg	Should NOT be used in renal impairment
Unfractionated heparin(UFH)	**Intravenous (IV) infusion regimen**: Heparin 60IU/Kg IV bolus THEN infuse at a rate of 18 units/kg/h. Check APTT at 6h, aim for APTT of 60-85 sec or APTT ratio of 1.5–2.5.Measure APTT daily or 10h after dose change **Subcutaneous regimen**-17,500IU SQ BD	UFH is recommended in patients with a CrCl<30 mL/min with dose adjustment based on APTT.
Warfarin	**Recommended starting dose**: 5 mg PO OD Initiate on day 1 of parenteral anticoagulation therapy; do daily INR **Typical maintenance dose**: 2 to 10 mg PO ODIndividualize dosage according to the patient's INR. **Target INR**: 2.5 (range: 2 to 3) **Renal Dose**: No dosage adjustment necessary for patients with renal failure	**Drug-drug interactions:** Warfarin has many drug interactions: antimicrobials(antibiotics,anti-TBs, antifungals), ARVs, CVD drugs,steroids, anticonvulsants, contraceptives etc. *Consult with pharmacist before prescribing medications* **Contraindications**: Peptic ulcers, bleeding disorders, severe HTN, pregnancy **Caution**: Elderly; those with past GI bleeds.**Dietary restrictions:** Warfarin has many food interactions. *Consult a nutritionist.*
DirectOral AntiCoagulants (DOACs)	• Rivaroxaban 15 mg BD as a loading dose for 21days followed by 20 mg OD. • Dabigatran: 150mg BD after 5 days of parenteral therapy	Mode of action: Factor Xa inhibitors(rivaroxaban and apixaban) Direct thrombin inhibitors (dagibatran)

2. Duration of Anticoagulation Therapy in VTE

The importance of anticoagulation therapy in patients with VTE is to prevent recurrence. Vitamin K agonists(VKAs) are used in most cases, while LMWH are preferred in patients with VTE and active cancer. The DOACs have been utilized as an alternative to VKA for long term treatment of VTE.

Table 47: Recommendations for duration of Anticoagulation after VTE

Scenario	Duration of anticoagulation
Patients with VTE secondary to a transient(reversible) risk factor	3 months orally
Patients with unprovoked VTE	At least 3 months orally
Patients with a second episode of unprovoked VTE	Indefinite duration* (guided by a physician)

**The risk-benefit ratio of continuing extended anticoagulation should be reassessed at regular intervals.*

Antidotes

- If UFH overdose: stop infusion. If there is bleeding, administer protamine sulfate in consultation with a physician.
- Warfarin: In case of any major bleed (including intracranial hemorrhage), stop warfarin. Give prothrombin complex concentrate 50units/kg (where available) in consultation with a Physician.
- If unavailable, give Fresh Frozen Plasma (FFP) (15mL/kg). Also give 5–10mg vitamin K IV slowly.

Important considerations

- Monitoring of bleeding- take note of gastrointestinal, brain, skin and urological bleeding
- Dosing, especially in renal disease
- Special groups-Pregnancy, HIV, elderly and children, sickle cell disease (refer to appropriate experts)
- Precautions: Drug interactions when on antiplatelets; warfarin and its interactions
- Follow-up- with regards to the efficacy of anticoagulation and recurrence of VTE

Stroke

Stroke: Rapidly developing episode of focal or at times global loss of cerebral functions with symptoms lasting more than 24 hours or leading to death, causing damage to the brain tissue.
Transient Ischemic Attack (TIA): a focal neurological or visual deficit caused by interruption in blood supply to the brain (or retina) in which all symptoms resolve within 24 hours.
Pathophysiology: Strokes can either be :-
Ischemic: Caused by occlusion of cerebrovascular blood supply thrombosis in a (brain artery) or embolism from a major artery e.g. carotid). Form 80-87% of all strokes
Hemorrhagic: Rupture of a weakened vessel or defects in coagulation leading to bleeding into the brain(intracerebral) or subarachnoid space. The blood accumulates and compresses the surrounding brain tissue. Form 12-20% of all strokes

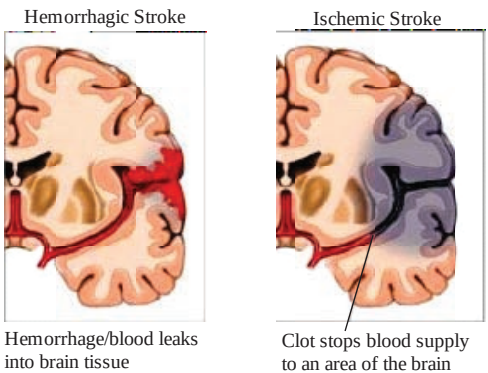


Figure 23: Types of stroke lesions

Table 48: Risk factors for stroke

Risk factors for ischemic stroke	Risk factors for hemorrhagic stroke
Non-modifiable • Advanced age • Race • Male sex • Previous history of stroke **Modifiable** • Hypertension (the most important) • Diabetes mellitus • Cardiac disease – Valvular heart diseases, HF, Arrthythmias • Tobacco use • Excessive alcohol use • Hypercholesterolemia • TIAs • Obesity/Sedentary lifestyle • Oral contraceptive use • Sickle cell disease • HIV infection	• Hypertension • Alcohol use • Advanced age • Coagulopathies • Eclampsia • Arteriovenous malformation (AVM), aneurysms, and other vascular malformations (venous and cavernous angiomas) • Dural sinus thrombosis • Cerebral amyloidosis • Vasculitis • Intracranial neoplasm • Use of illicit drugs (e.g. cocaine, other sympathomimetic drugs) • Anticoagulant therapy • Thrombolytic therapy

Primary prevention of stroke: Prevent stroke in patients with no such history

- Optimise treatment for DM, hypertension, obesity and dyslipidemia
- Behavioural modification: tobacco/alcohol cessation, physical activity, diet

Secondary prevention of stroke: Prevent recurrent stroke to reduce brain injury and disability

A: Antiaggregants (aspirin, clopidogrel, extended-release Dipyrida mole,Ticlopidine) and anticoagulants (Apixaban, Dabigatran, Edoxaban, Rivaroxaban, Warfarin)

B: Blood pressure–lowering medications

C: Cessation of cigarette smoking, cholesterol-lowering medications, carotid revascularization

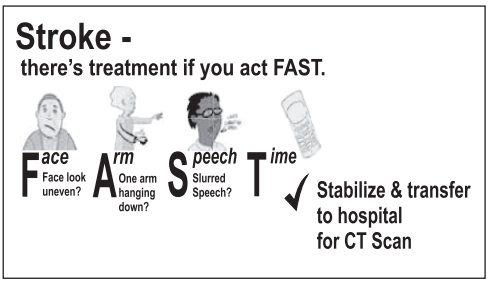
D: Diet

Low-dose aspirin (75 mg daily) or Clopidogrel (75 mg daily) should be prescribed after ischaemic stroke or TIA for secondary prevention of recurrent vascular events.

Diagnosis

TIME IS BRAIN –Permanent damage to brain tissue occurs very quickly and therefore to minimize the level of disability following stroke, treatment must be initiated as soon as possible

Below is a simplified criteria for detecting stroke.



Evaluation for stroke

Examination	Components
History	Abrupt onset of extremity weakness, hemisensory disturbance, visual disturbance, abnormal speech, facial droop, abnormal gait or posture, dizziness and loss of balance, sudden decrease in level of consciousness. *No historical feature distinguishes ischemic from hemorrhagic stroke, although nausea, vomiting, headache, and sudden change in level of consciousness are more common in hemorrhagic strokes.*
Physical examination	Assessment of ABCs, Vital signs (BP, Temp, Pulses) General exam: head and neck (signs of trauma or seizure activity e.g contusions, tongue lacerations), carotid disease (bruits), jugular venous distention Cardiac exam: myocardial ischemia, valvular conditions, irregular rhythm, CCF Neurological exam: GCS or strole scale, cranial nerves, motor function

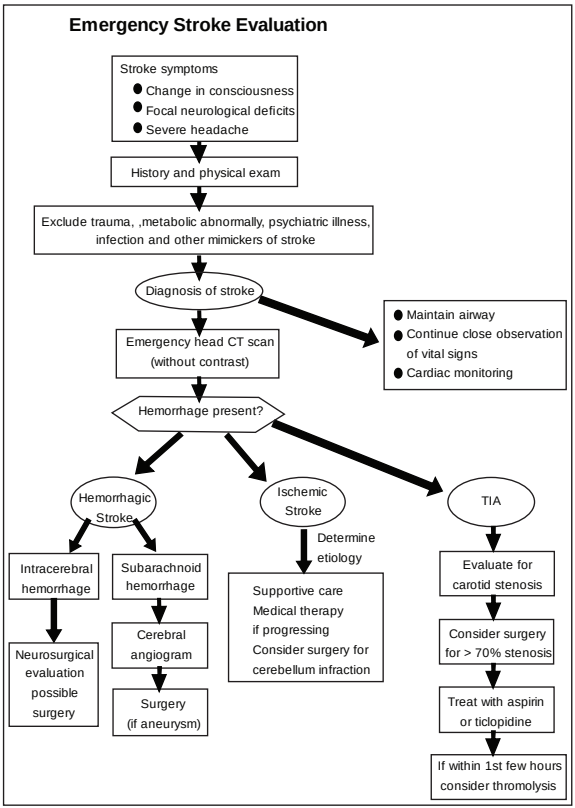


Figure 24: Protocol for diagnosis of stroke

Management of stroke

Step by Step Procedure	
i.	Manage ABCs
ii.	Monitor BP and other vitals every 15 min for the first hr and hourly thereafter
iii.	Gain large bore intravenous access
iv.	Oxygen (as required O2 saturation 92%)
v.	Assess for hypoglycemia or hyperglycemia
vi.	Take blood sample for lab analysis (refer above)
vii.	Maintain *Nil per oral* (NPO) and insert NGT to prevent aspiration
viii.	Get CT scan of the brain as soon as possible
ix.	Admit patient or organise for Referral to closest appropriate facility capable of treating acute stroke
x.	Alert receiving Hospital/Emergency Department

Management of ischemic stroke

Thrombolysis	Stable stoke patients within 4.5 hrs of onse:Treat with 0.9 mg/kg (up to maximum 90 mg) intravenous rt-PA(Alteplase). ***Intra-arterial thrombolysis should only be carried out by an appropriately trained interventional neuro-radiologist.***
Antiplatelet Agents	Aspirin 75mg or clopidogrel 75mg daily started immediately where thrombolysis is not available.
Anticoagulants	Anticoagulant therapy is recommended in cardio-embolic event. Refer to ischemic heart disease section for details.
Statins	Statins should be prescribed to patients who have had an ischaemic stroke, irrespective of cholesterol level.

Management of hemorrhagic stroke

Management depends on the cause and severity of the bleeding.
i. Basic life support
ii. Nil by mouth and NGT insertion to prevent aspiration pneumonia
iii. Control of bleeding, seizures, blood pressure (BP), and intracranialpressure, are critical
iv. Medications used in the treatment of acute stroke include the following:
- **Anticonvulsants** - To prevent seizure recurrence
- **Antihypertensive agents** - To reduce BP and other risk factors of CVD
- **Osmotic diuretics** - To decrease intracranial pressure in the subarachnoid space A potential treatment for hemorrhagic stroke is surgical evacuation of the hematoma.

Supportive management: Early mobilization; adequate hydration (to reduce risk of DVT), input-output monitoring; infection prevention; DVT and PE prophylaxis
Long-term care: Psychosocial support, palliative care, pain management, physiotherapy, occupational therapy, speech therapy, bed sore management e.t.c.

Special considerations in management

i. Careful monitoring of treatment since drug toxicities are common.
ii. Slow, careful titration of medications.
iii. Avoid atenolol in adults over 60 years of age, unless they have coronary artery disease.
iv. Avoid short-acting calcium channel blockers, e.g. Sublingual nifedipine
v. Compelling indications for hypertension treatment

Cardiovascular diseases in Elderly persons

Elderly persons are at a higher risk of developing CVD and is associated with higher rates of mortality among this population. Special considerations must be applied when managing CVD in the elderly.

Table 48: Compelling indications for antihypertensive medications in the elderly patient

Compelling Indication	Recommendation
Congestive Heart Failure	Use ACE Inhibitors or ARB's as first-line agents. B-blockers are also beneficial
Myocardial Infarction	Beta-blockers and/or ACEi (or ARB's) should be considered as first-line agents. If an anti-anginal is necessary, the use of CCB can be considered.
Nephropathy	Use ACEi (or ARBs) when the serum creatinine is >1.5 mg/dL or the 24-hour urine protein is > 1 gram.
Gout	Avoid thiazide diuretics in patients with gout.
Hyperlipidemia	Use calcium channel blockers or ACEi
Erectile dysfunction	Use chlorthalidone with caution

Table 49: Hypertension treatment indications and targets

Treatment targets in the elderly patient over 80 years		
	BP levels	
	SBP	**DBP**
Treatment indication	≥150 mmHg	≥ 90 mmHg
Treatment targets	<150 mmHg	< 90 mmHg
Treatment in the elderly patient 60-79 years		
	BP levels	
	SBP	**DBP**
Treatment indication	≥140 mmHg	≥ 90 mmHg
Treatment targets	<140 mmHg	< 90 mmHg
Treatment in the elderly patient over 60 years, with diabetes		
	BP levels	
	SBP	**DBP**
Treatment indication	≥135 mmHg	≥ 85 mmHg
Treatment targets	<135 mmHg	< 85 mmHg

Cardiovascular disease in diabetes

Common cardiovascular diseases in diabetes: Hypertension, heart disease (LVH, coronary artery disease, heart failure) and stroke

The management of CVD in diabetes follows the principles outlined in previous sections of this document with the exception of Hypertension

Table 50: Hypertension treatment target for various categories of diabetic patients

Hypertension treatment in patients with diabetes mellitus		
	BP levels	
	SBP	**DBP**
Treatment indication	≥140	≥ 90
Treatment targets	<140	< 85
Hypertension treatment in diabetic patients with established cardiovascular disease		
	BP levels	
	SBP	**DBP**
Treatment indication	≥140	≥ 90
Treatment targets	<140	< 90
Hypertension treatment in patients with diabetes, and renal impairment (serum creatinine >133µmol/L), GFR <60 and microalbuminuria)		
	BP levels	
	SBP	**DBP**
Treatment indication	≥140	≥ 90
Treatment targets	<140	< 90

Special considerations

- Monitor serum creatinine and potassium once a year and more frequently if there is evidence of renal impairment.
- Diuretics in large doses inhibit insulin release
- Betablockers may blunt or mask symptoms of hypoglycaemia and exacerbate peripheral vascular disease
- Dyslipidaemias may be worsened by betablockers and diuretics
- Impotence and postural hypotension may be precipitated or aggravated by alpha blockers and centrally acting agents (e.g.methyldopa).
- ACEI may induce hyperkalaemia, renal failure, a persistent cough
- Avoid sublingual antihypertensives and hydrallazine that have the potential to dramatically and catastrophically reduce blood pressure which can cause renal injury.

Table 51: Vascular protection checklist for patients with diabetes mellitus

Acronym	Parameter	Remarks
A	HbA1C	Optimal glycemic control (usually ≤7%)
B	BP	Optimal bloodpressure control (<140/90)
C	Cholesterol	LDL ≤2.0 mmol/L if decided to treat
D	Drugs to protect the heart(regardless of baseline BP or LDL	**A** – ACEi or ARB **S** – Statin **A** – ASA if indicated ; Use in patients with established CVD or with additional CV risk factors (Age over 40,HTN,Dyslipidemia)
E	Exercise/Eating healthily	Regular physical activity, achieve and maintain healthy body weight
S	Smoking cessation	

Cardiovascular diseases in People Living with HIV/AIDS (PLHIV)

HIV infection and its treatment increases risk of developing the following conditions:

Dyslipidemia

- LPV-r and EFV exacerbate dyslipidemia; consider change to a more-friendly drug(e.g. ATV/r or DTG) before initiating lipid lowering agents
- Consider statins if not achieved treatment targets despite lifestyle modification

Chronic kidney disease

- Management depends on cause. Consult a specialist
- Substitute TDF if CrCl<50 ml/min (except in patients with HIV/HBV coinfection
- Avoid nephrotoxic drugs (aminoglycosides, NSAIDs)
- All NRTIs except ABC require dose adjustments for renal impairment (NNRTIs and PIs and INSTIs do nor require dose adjustment)

Hypertension

- Metabolism of CCBs is induced by EFV: consider higher starting doses and monitor closely
- PIs inhibit metabolism of CCBs: monitor closely for excess BP reduction and reduce the dose of the CCBs
- Patients on PIs (e.g. LPV/r or ATV/r) may experience oedema, dizziness, fatigue and orthostatic hypotension within the first week of initiation of CCB therapy. Counsel patient accordingly Hypertension and TB and/or diabetes
- Refer to a facility with the capacity to monitor for potential drug interactions

The table below summarizes the approach to management of CVD in setting of chronic kidney disease.

Table 52: Mangement of CVD in CKD

CVD condition	Management	Caution in CKD
Coronary artery disease and MI	Aspirin, clopidogrel, ARBs,ACEIs, reperfusion therapy,statins	Higher risk of bleeding on antiplatelet therapy
Congestive heartfailure	Dietary salt restriction, ACEIs, ARBs, carvediolol and bisoprolol (limited evidence)	Use aldosterone antagonists with caution, specialist supervision for GFR below 30ml/min/1.73m^2
Stroke	Low dose antiplatelet therapy, statins, intravenous thrombolysis	Avoid intravenous tissue plasminogen activator for patients undergoing dialysis (due to use of heparin in these patients)
Atrial fibrillation	Warfarin for those at high risk for stroke (those with CHF, previous stroke, LVH, hypertension)	Risk stratification excluded ESRD patients, hence best to avoid warfarin in these patients
Peripheral artery disease	Smoking cessation, aspirin,revascularization or amputation	Higher risk of bleeding on antiplatelet therapy
Sudden cardiac death		Avoid digoxin
Hypertension	ACEI, ARB as a compelling indication, targets BP below 130/80mmHG	A limited rise in creatinine of up to 35% on ACEIs and ARBs should not preclude therapy unless hyperkalemia develops

Hypertensive disorders in pregnancy

Classification: ***preecclampsia*** (HTN onset after 20 weeks with proteinuria or end-organ damage); **eclampsia** (preeclampsia with generalized tonic-clonic convulsions); **chronic hypertension** (hypertension which antedates pregnsncy); and **gestational hypertension** (hypertension in pregnancy with no proteinuria)

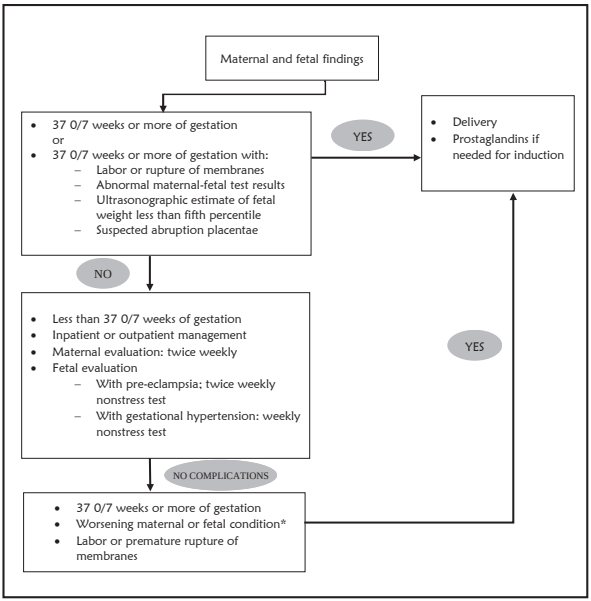
Preeclampsia

Risk factors: extremes of age <18yrs or >40 years), blak race, famly history, primiparity, pregnancy interval >10 years, diabetes, multiple gestation, preeclampsia history in previous pregnancy

Table 53: Diagnostic criteria of preeclampsia

Parameter	Criteria
BP	SBP≥140mmHg and/or DBP≥90mmHg on two occasions>4 hours apart If SBP≥160mmHg or DBP≥110mmHg, cofirm diagnosis within a few minutes
and	
Proteinuria	≥300mg/24hrs Or Protein/creatinine ratio ≥0.3 Dipstick reading of 1+
Or in the absence of proteinuria, new-onset HTN with new onset of any of the following	
Thrombocytopeania	Platelets <100/mL
Renal insufffeciency	Serum creatinine>1.1mg/dL or doubling of the serum creatinine in absence of other renal disease
Impaired liver function	Elevated blood concentration of liver transaminases (doubling)
Pulmonary edema	
Cerebral or visual symptoms	

The figures below outline the treatment for uncomplicated and complicated preeclampsia



*BP Targets: Systolic 140 -160 mmHg, Diastolic 90 -100 mmHg

Figure 25: Management of pre-eclampia without severe features

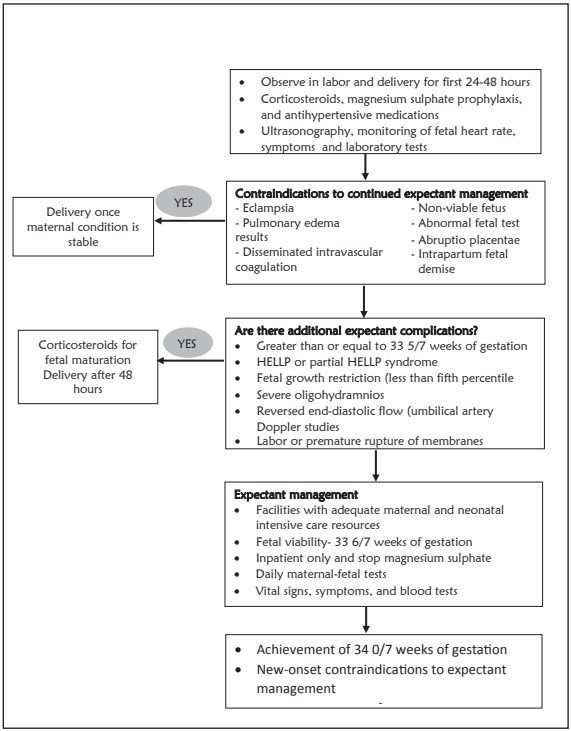


Figure 26: Management of pre-eclampia with severe features and HELLP syndrome

Table 54: Oral anti-hypertensives in pregnancy

Drug	Dosage
Labetalol	200-2,400 mg/d orally in two to three divided doses
Nifedipine	30-120 mg/d orally of a slow- release preparation
Methyldopa	0.5-3 g/d orally in 2-3 divided doses
Thiazide diuretics	Depends on agent. Second-line agents

ACEI and ARBs are associated with fetal anomalies. Contraindicated in pregnancy and preconception period

Table 55: Antihypertensives for urgent BP control

Drug	Dosage
Labetalol	10-20 mg IV then 20-80 mg every 20-30 min to a maximum dose of 300 mg or constant infusion 1-2 mg/min IV
Hydralazine	5 mg IV or Im than 5-10 mg IV every 20- 40 min or Constant infusion 0.5-10mg/h
Methyldopa	0.5-3 g/d orally in two to three divided doses
Nifedipine	10-20 mg orally repeat in 30 minutes if needed; than 10-20 mg every 2-6 hours

Management of eclampsia

1. Magnesium sulphate

Intravenous (Zuspan or Sibai)	Intramuscular (Pritchard)
✓ Loading dose of 4 g should be given IV over 5 minutes Followed by: ✓ Maintenance dose (infusion) of 1-2 g/hour ✓ Give until 24 hours after the last convulsion or delivery, whichever comes $_1$st	✓ IM MgSo4 20 % solution, 4 g by deep IM injection over a period of 5 minutes, Followed by : ✓ Two deep IM injections of 5 MgSO4 50 % solution into each buttock (Total dose of 10 g) ✓ Maintenance dose is 5 g MgSO4 50 %, given by deep IM injection, every 4 hours. ✓ Alternate the buttocks in which the injection is administered ✓ Give until 24 hours after the last convulsion or delivery, whichever comes 1st

2. **Phenytoin:** 10 mg/kg loading dose infused IV no faster than 50 mg/min, followed by maintenance dose started 2 hrs later at 5 mg/kg

3. **Diazepam:** Give Diazepam rectally when IV access is not possible. The loading dose is 20mg in 10ml syringe or a catheter may be used. If convulsions are not controlled within 10 minutes administer an additional 10mg per hour

Cardiac Diseases in pregnancy

a. Peri-partum cardiomyopathy and Heart Failure in Pregnancy
PPCM is an idiopathic cardiomyopathy, where patients present with symptoms of heart failure due to left ventricular dysfunction. It is a diagnosis of exclusion when no other cause of heart failure is found Usually presents in late pregnancy or uerperium
Presentation
- Pregnant or in peuperium
- Shortness of breath especially on lying flat or at night
- Nocturnal cough
- Lower limb and/or facial oedema

Diagnosis: ECG and Echocardiogram. Chest Xray(cardiomegaly, and rule out other chest pathology)

Management
Acute HF: management in pregnancy same as that of Acute HF due to other causes. Oxygen, IV diuretics, IV nitrate(if BP>110mmgHg); inotropic agents (in patients with features of low output states/hypoperfusion)
Chronic HF: After delivery, mange HF as per the cuurent guidelines. During pregnancy, the following restrictions apply:
- Anticoagulation for patients with intracardiac thrombus or systemic embolism
- Therapeutic anticoagulation with LMWH or VKA according to stage of pregnancy for patients with atrial fibrillation
- ACEI or ARB contraindicated due to renal and fetal toxicity
- Beta-1 selective blockers preferred because beta-2 blockers can have anti-tocolytic action

b. Ischemic heart disease
Chest pain characterized as a crushing pain radiating to the left arm. In pregnancy this pain may also be experienced as epigastric pain. Diagnosis: Perform an ECG and for all pregnant women with chest pain.Serum cardiac troponin should be measured

Management
Acute:
- 100% Oxygen by mask
- Analgesia: Opioids preferred e.g. Morphine
- Immediate referral to a Level 6 facility for coronary angiography and possible percutaneous coronary intervention (PCI).
- Thrombolytic therapy may cause sub-placental hemorrhage, therefore should only be used as a life saving measure when PCI is unavailable

Medical therapy: ACEi and ARBs, are contraindicated during pregnancy . β-Blockers and low dose acetylsalicylic acid are considered to be safe. Other management as in non-pregnant state: Lifestyle modification (stop alcohol, smoking, weight control) BP control and glucose control

Venous Thrombo-Embolism in pregnancy

Clinical presentation of DVT
Patients may present with unilateral or bilateral lower limb swelling. DVT is left sided in 85% of cases, due to compression of the left iliac vein by the right iliac artery and the gravid uterus. Iliac vein thrombosis may manifest with isolated pain in the buttock, groin, flank, or abdomen
The clinical symptoms and signs of pulmonary embolism during pregnancy are the similar to the non-pregnant state (dyspnoea, chest pain, tachycardia, haemoptysis, and collapse). Subjective clinical assessment of pulmonary embolism may be difficult, because dyspnoea and tachycardia are not uncommon in normal pregnancy.

Diagnosis: See VTE section above

Treament of VTE

a. Low Molecular Weight Heparin (LMWH)

Drug of choice for the treatment of VTE in pregnancy and puerperium
Dosage: Enoxaparin 1mg/kg body weight twice daily Dalteparin 100 IU/kg body weight twice daily

b. Unfractionated Heparin (UFH)

It does not cross the placenta, however it is associated with more thrombocytopenia and osteoporosis.
It is indicated in the acute treatment of massive PE or in renal failure when urgent reversal of anticoagulation by protamine may be needed.

Dosage

In patients with acute PE with haemodynamic compromise, IV administration of UFH is recommended (loading dose of 80 U/kg, followed by a continuous IV infusion of 18 U/kg/h).

Monitoring

Do aPTT 4–6 h after the loading dose, 6 h after any dose change, and then at least daily when in the therapeutic range. Target aPTT ratio is usually 1.5– 2.5 times the average laboratory control value. The dose is then titrated to achieve a therapeutic aPTT, defined as the aPTT that corresponds to an anti-Xa level of 0.3–0.7 IU/mL. When haemodynamics are improved and the patient is stabilized, UFH can be switched to LMWH in therapeutic doses and maintained during pregnancy.

LMWH should be switched to IV UFH at least 36 h before the induction of labour or caesarean delivery. UFH should be discontinued 4–6 h before anticipated delivery, and restarted 6 h after delivery if there are no bleeding complications.
Both UFH and LWMH are safe during breastfeeding.

c. Warfarin

Warfarin may be used where LMWH is not feasible from the second trimester. It should be stopped at 37 0/7 weeks, and the patient started on LMWH in anticipation of delivery.

- Dose is titrated to achieve a therapeutic INR of 2-3
- Warfarin does not enter breast milk and is therefore safe for use while breastfeeding

Post-partum management

- In patients with VTE, pre-partum heparin treatment should be restarted 6h after a vaginal birth and 12 h after a caesarean delivery, if no significant bleeding has occurred.
- Women should be offered the option to continue with heparin or warfarin, with the attendant risks, costs and monitoring protocols explained
- If a woman opts for warfarin, it should be started on the second day post partum to overlap with heparin treatment for 3-5 days, until a therapeutic INR of 2-3 is achieved.
- Anticoagulation should be continued until at least 6 weeks post partum, or until 3 months of treatment have been completed. Before stopping treatment,the on-going risk of VTE should be assessed.

We define athletes broadly and include participants at all levels of sport, including:
- Competitive athletes
- Scholastic (e.g. high school)
- Club
- Collegiate
- Professional
- Recreational athletes
- Persons who exercise for fitness and health

The main conditions of focus are athlete's heart, hypertension in athletes, heart failure in athletes and sudden cardiac death.

1. Athletes Heart

Athlete's heart is a constellation of structural and functional changes that occur in the heart of people who train for > 1 h most days. The changes are asymptomatic; signs include bradycardia, a systolic murmur, and extra heart sounds. ECG abnormalities are common. Diagnosis is clinical or by echocardiography.

Symptoms and Signs

There are usually no symptoms

Signs vary but may include:
- Bradycardia
- LV impulse that is laterally displaced, enlarged, and increased in amplitude
- Systolic ejection (flow) murmur at the left lower sternal border
- A 3rd heart sound (S3) due to early, rapid diastolic ventricular filling
- A 4th heart sound (S4), heard best during resting bradycardia because diastolic filling time is increased
- Hyperdynamic carotid pulses

These signs reflect structural cardiac changes that are adaptive for prolonged intense exercise. No treatment is necessary other than reassurance. Athlete's heart is significant because it must be distinguished from serious cardiac disorders.

2. Hypertension in Athletes

Hypertension is a common cardiovascular condition affecting athletes. However, the management of hyper- tension in athletes can differ from standard approaches, primarily due to the potential side effects of some medications that may impair training and performance.
The fundamental parameters defining hypertension in both adults and children do not differ in athletes.

Table 56: Recommendations for management of hypertension in athletes

Stage of Hypertension	Recommendations
Prehypertension or Stage 1 hypertension without signs of end organ damage	• No restrictions on their participation in sport • Athletes with prehypertension should have their BP rechecked annually • Athletes diagnosed with Stage 1 hypertension should be treated and have their BP monitored according to standard guidelines.
Stage 1 hypertension (BP between 140/90 and159/99) accompanied by signs of end organ damage	• **Should not** participate in sport until their BP is well-controlled
Stage 2 hypertension (BP>160/100)	• **Should not** participate in sport until their BP is well-controlled

All athletes with a new diagnosis of hypertension or prehypertension should receive an appropri- ate clinical, laboratory and other workup as would be performed for any patient.

Special considerations for child athletes

Renal ultrasound is recommended for child athletes with established hypertension. In addition, an echocardiogram is recommended for child athletes with diabetes or renal disease associated with a BP between the 90th and 94th percentiles. An echocardiogram is part of the initial evaluation of all children with a BP in the 95th percentile or higher. When indicated, these imaging studies are performed in addition to the thorough history and physical examination, including a retinal examination, performed in any child with unexplained hypertension.

Medical Therapy Considerations

The ideal medication should control BP without compromising exercise capacity. In addition, the medication selected should be permissible under the rules of the governing body for their sport (i.e., not a banned substance).

- The general principle of initiating treatment with monotherapy still holds, but the choice of medications requires caution.
- Diuretics are relatively contraindicated, as they can impair athletic performance due to volume depletion, and they are prohibited by many of the governing bodies in sport because they may be used to prevent detection of performance enhancing drugs.
- Beta blockers decrease heart rate and thus can reduce exercise tolerance; they too are prohibited in some sports.
- Angiotensin-converting enzyme (ACE) inhibitors, angiotensin II receptor blockers (ARBs), and long-acting calcium channel blockers are recommended. Starting doses for athletes are the same as for members of the general population, as is the approach to increasing the dose as needed based on patient response.
- Follow-up on a biweekly basis is performed to monitor BP, treatment compliance, general adverse effects, and effects on sport performance.

Persistent hypertension — Athletes with hypertension that has persisted for a longer period (6 to 12 months) or that has not responded to lifestyle modifications and pharmacotherapy should be assessed with an echocardiogram(2,3). Concentric LV hypertrophy with diminished LV volume is a form of end-organ damage and requires modification of activity (strenuous athletic activity is restricted) and implementation of appropriate medical therapy.

Heart Failure in Athletes

Heart failure in athletes may be secondary to various conditions as shown in table below:

Table 57: Causes of heart failure in athletes

Causes	Clinical consideration	Diagnostic tools
Myocarditis	• Mainly athletes below 35 years • Viral or rarely parasitic origin • Sometimes subclinical; usually preceded /precipitated by a respiratory tract infection. • Athletes with a history of acute myocarditis should be regularly screened for symptoms of HF, and diastolic global and regional LV dysfunction on echocardiography.	It is advisable that athletes who have suffered a recent respiratory tract infection undergo medical check-up (detailed physical examination, resting ECG - negative T-waves, pathological Q-waves, intraventricular conduction disturbances can indicate acute myocarditis even in athletes with normal echocardiogram) before they resume intensive physical training. CMR can help differentiate between inflammatory and ischemic LV dysfunction
Arterial hypertension	High static and high dynamic sports can contribute to hypertensive diastolic and/or systolic HF	Echocardiography, CMR
Drug abuse or doping	Cocaine or amphetamine addiction, anabolic abuse	Echocardiography, CMR
Iatrogenic myocardial damage	Chemotherapy (anthracyclines), radiotherapy (chest irradiation) – dose-dependent. Ventricular function testing necessary prior to and during chemotherapy. Responds well to ACE-Inhibitor treatment.	Echocardiography, CMR
Inherited dilated cardiomyopathy	Genetically conditioned (e.g., deletion in dystrophin gene)	Echocardiography, CMR
Ischemic cardiomyopathy	Post-myocardial infarction contractile dysfunction with lowered EF (below 50%)	Echocardiography, CMR
Hypertrophic cardiomyopathy	Diastolic and/or systolic HF	Echocardiography, CMR, genetic tests
Left ventricle non-compaction (LVNC)	Genetically conditioned, differential diagnosis with exercise-induced hypertrabeculations in athletes	Echocardiography, CMR – the most reliable diagnostic tool
Acquired or congenitalvalve defects	Mitral or aortic insufficiency, aortic stenosis, VSD, ASD, Ebstein anomaly	Echocardiography, CMR

Athletes suffering or recovering from any of these causes should be on close follow up and screening for Heart Failure.

Symptoms of heart failure in athletes

Ambitious athletes are inclined to dissimulate symptoms or blame them on non-cardiac causes. The most challenging group is elderly athletes who often attribute their exertional dyspnea or fatigue to ageing.

The following symptoms may indicate heart failure in athletes:

- Shortness of breath, unexpected drop in performance, dyspnea on exertion
- Persistent fatigue and muscle pains of uncertain etiology, refractory to anti-in flammatory medications, physiotherapy, reduction in training intensity or exercise cessation
- Mental disorder – especially when combined with fatigue
- Pre-syncope or dizziness
- Persistent cough
- Nocturia or oliguria
- Lower extremities swelling
- Anginal pains
- Recent history of respiratory tract infection and drop in performance
- Persistent heartbeat irregularities

Diagnosis and appropriate management of chronic Heart Failure (HF) in this group does not differ from the general population.

4. Sudden Cardiac Death (SCD) in Athletes

There is overwhelming evidence that exercise can trigger ventricular arrhythmias and cardiac arrest in individuals with preexisting heart conditions, even in well-conditioned young athletes. The vast majority of these sudden deaths are caused by previously unidentified and asymptomatic underlying cardiovascular conditions.

Pathophysiology of SCD

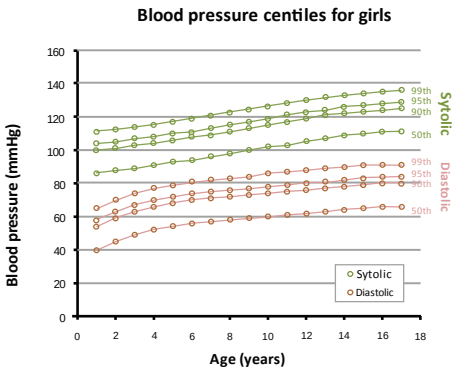
The cardiovascular conditions triggering SCD result in a common final pathway of SCD leading to ventricular tachyarrhythmias. Below is a list of common causes of SCD.

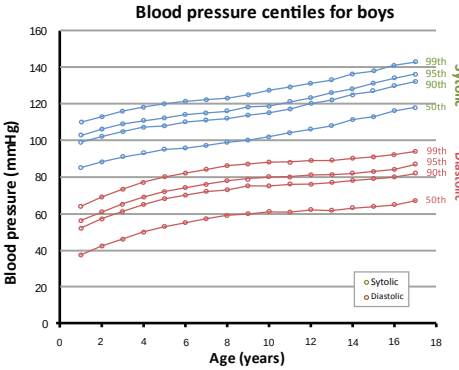
- **Hypertrophic Cardiomyopathy**
- **Arrhythmogenic right ventricular cardiomyopathy (ARVC)**
- **Congenital coronary artery anomalies**
- **Arrhythmias**

Assessment for an underlying cardiac condition as part of the regular health assessment is vital for prevention of SCD among athletes.

Annex 1

Blood Pressure Centiles for Boys and Girls:





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