CLINICAL GUIDELINES
FOR THE MANAGEMENT
OF
CORONARY HEART DISEASE

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1.   INTRODUCTION

Of the cardiovascular diseases, acute myocardial infarction is the most common cause of death. The incidence of acute myocardial infarction is estimated at around 4,000/year in Mauritius, with a total mortality rate of 30% - 40% and a 25%-35% mortality rate before reaching the hospital. The underlying cause in most cases is atherosclerotic coronary artery disease.

These clinical guidelines on the management of coronary artery disease have been developed in this context in line with the decentralization and re-organization of the Ministry of Health's NCD programme.

They aim at improving the quality of health care through:

1.   recommendations for active primary prevention of coronary artery disease and its risk factors.

2.   the improvement and standardization of the management of patients suffering from coronary artery disease.

The recommendations made are, to a large extent, evidence-based.

The development of the guidelines is the combined effort of cardiologists, a consultant in Internal Medicine and community physicians in association with the Mauritius Institute of Health. It has sought consensus between the policy makers at the Ministry of Health, cardiologists at hospital level and community physicians at primary care level after having taken cognizance of local realities, limitations and scope for further development.

2.   DEFINITION OF CORONARY ARTERY DISEASE

Coronary artery disease results from an imbalance between myocardial oxygen supply and demand and is most commonly caused by the inability of atherosclerotic coronary arteries to perfuse the heart due to partial or total occlusion of the coronary arteries. Chronic angina pectoris is, by definition, stable, i.e., the severity and/or frequency of chest pain is not increasing or occurring at rest. Unstable angina, myocardial infarction and sudden ischaemic death are also manifestations of chronic ischaemic heart disease, presenting as acute coronary syndromes.

3. PATHOPHYSIOLOGY

Myocardial ischaemia occurs when myocardial oxygen delivery cannot meet metabolic myocardial demands. This discrepancy is termed supply-demand mismatch. Although this term is overly simplistic, myocardial oxygen delivery is mostly determined by the oxygen-carrying capacity of blood and coronary flow.
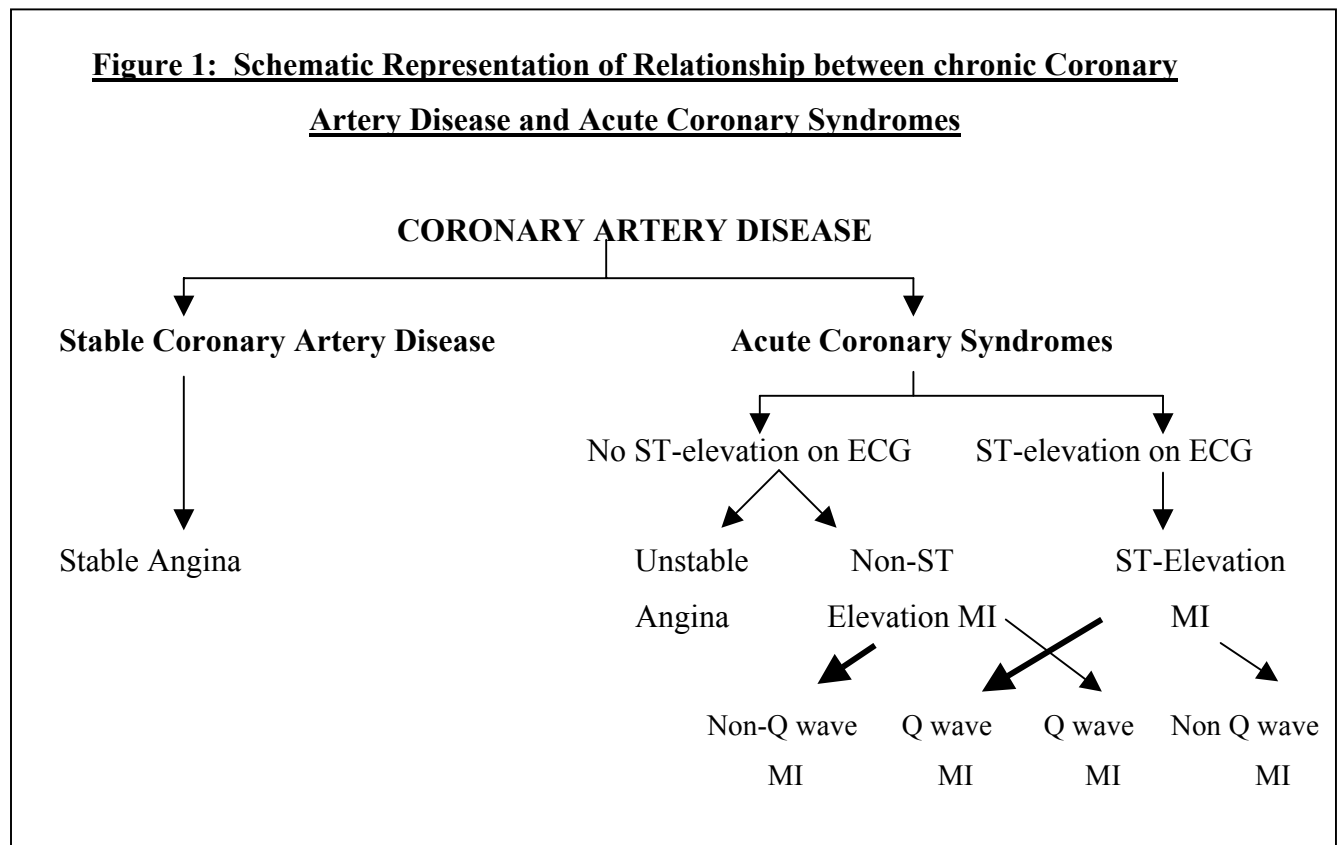
In normal coronary arteries, coronary blood flow can increase three to fivefold in response to exercise. This increase, termed coronary flow reserve, occurs mostly through decreased resistance in coronary microcirculation. Significant atherosclerotic plaquing in epicardial coronary arteries (> 75% cross-sectional area) results in a drop in blood pressure across the stenotic lesion. Coronary arterioles dilate to compensate for the reduced distal perfusion pressure, maintaining normal resting coronary blood flow. Consequently, at rest, most patients with significant coronary artery stenosis obstructions have no ischaemia and therefore no angina. During exercise, however, the capacity of coronary arterioles to dilate further is limited, and the myocardial oxygen demand soon outstrips the supply, resulting in ischaemia, followed usually by angina.

Acute coronary syndromes share a common pathophysiology: acute rupture (fissuration or ulceration) of a lipid-rich intracoronary atheromatous plaque, with subsequent mobilisation of a sequence of inflammatory and thrombotic cascades culminating in the formation of thrombus.

The possible triggers of acute plaque rupture are: sheer stress, inflammation and increased neurohormonal tone. Certain plaques are more susceptible to acute rupture than others – for example those which are eccentric, with a lipid-rich vulnerable core, a high content of active inflammatory cells, and a thin fibrous cap. Rupture of the plaque (ulceration/fissuration), with exposure of the highly thrombogenic contents to the bloodstream, initiates a cascade of cytokine release, inflammatory cell activation and platelet activation, with resulting platelet

aggregation and formation of occluding thrombus. As a result, the patient may present with one or more of the following:

1. If the vessel lumen is **completely occluded** or if inadequate collateralisation is present, **transmural myocardial injury and subsequent infarction** occurs, with ST elevation (or new left bundle branch block) most commonly seen on the electrocardiogram (ECG).

2. If following plaque rupture, however, **incomplete occlusion** of the lumen occurs or sufficient collateral flow exists, **ischaemia** or **nontransmural injury** results, **with** or **without** injury.

3. If ischaemia occurs **without** injury (i.e, with negative serial enzyme measurements), the clinical condition is termed **unstable angina** (UA). If cell injury is demonstrated by serial measurements of cardiac enzymes, **non-ST elevation MI** is diagnosed. (See Figure 1 below).

Figure 1: Schematic Representation of Relationship between chronic Coronary Artery Disease and Acute Coronary Syndromes

4. GRADING OF ANGINA PECTORIS
(According to the Canadian Cardiovascular Society Classification System)

Class I: Ordinary physical activity (e.g. walking, climbing stairs) does not cause angina.

Class II: Slight limitation of ordinary activity
Angina occurs on walking or climbing stairs rapidly, walking uphill, walking or climbing stairs after meals or in cold, in wind, under emotional stress, or during the few hours after awakening. Angina occurs on walking more than two blocks on the level and climbing more than one flight of ordinary stairs at a normal pace and in normal condition.

Class III: Marked limitation of ordinary physical activity. Angina occurs on walking one to two blocks on the level and climbing one flight of stairs in normal conditions and at a normal pace.

Class IV: Inability to carry out any physical activity without discomfort – anginal symptoms may be present at rest.

5.   CHRONIC STABLE ANGINA

5.1   CLINICAL PRESENTATION

Character: usually described more often as a discomfort, pressure, heaviness, or squeezing sensation and less often as a pain. Sometimes described as a burning or sharp sensation.

Location: substernal area, precordium or epigastrium with radiation to the left arm, jaw or neck, less commonly felt only in radiation area and not in the chest.

Precipitation: often provoked by exertion, cold weather, eating, smoking and strong emotions. Relieved by rest, removal of provoking factors, or sublingual nitrates.

Duration: few minutes usually, rarely more than 4-5 minutes.

History: description of the chest discomfort and its relationship to activity. Past history of HBP, DM, hyperlipidaemia, or smoking or a family history of premature IHD in lst degree relatives (age younger than 55 yrs).

5.2 CLINICAL EXAMINATION AND INVESTIGATIONS

5.2.1 Clinical Examination

Often normal.

To look for confirmatory information, i.e., hypertension, peripheral artery disease, xanthelasma, tendinous xanthomata, tobacco-stained fingers or teeth. Episodic ischaemia alters left ventricular compliance, thus a transient S4, S3, may be heard during an acute ischaemic episode.

5.2.2 Investigations

(i)   Basic screening :
   - Fasting blood glucose
   - Serum lipids including high density lipoproteins (HDL) and triglycerides
   - Full blood count
   - Blood urea and electrolytes
   - Serum urates

(ii)   ECG:

   - Often normal in the absence of myocardial infarction or left ventricular hypertrophy.

   - An ECG during an episode of angina may show transient ST depression, T wave inversion or ventricular arrythmias (VPB; isolated or in runs, monomorph or polymorph). An ambulatory ECG (Holter) may demonstrate ischaemic episodes with or without symptoms 'Silent Ischaemia'.

(iii)   Echocardiogram:

- May demonstrate wall motion abnormalities suggestive of ischaemia or of infarction.

(iv)   Exercise Stress Testing (EST):

- Is a sensitive and informative examination, particularly useful in the detection and quantification of chronic ischaemic heart disease in patients who are at increased risk.

Indications:

- Differential diagnosis of chest pain, i.e., evaluation of patients with symptoms suggestive of IHD.
- Assessment of the threshold of angina in patient with known IHD.
- Evaluation of therapy for angina
- Evaluation of the asymptomatic patient over 40 yrs who has multiple risk factors for IHD.

Contraindications:

- Recent acute MI (4-6 weeks), except for submaximal effort.
- Unstable angina
- Rapid ventricular or atrial arrhythmia
- Advanced atrio-ventricular (A-V) block
- Decompensated, uncontrolled congestive heart failure
- Acute noncardiac illness
- Severe aortic stenosis, hypertrophic obstructive cardiomyopathy (HOCM)
- BP more than 170/100 before onset of exercise

(v)   Coronary angiography:

- Not necessarily needed in all patients with chronic stable angina to confirm the diagnosis.

- It allows the localization and quantification of obstructive lesions, evaluation of left ventricular (LV) function and assessment of valvular or myocardial disease.

Indications:

- Angina refractory to medical management
- Angina or MI in patients less than 45 yrs of age
- Unstable angina (after medical stabilization)
- Patients with persistent angina and/or low level EST abnormalities after MI.
- Marked ST changes at low-level exercise or persisting several minutes after cessation of EST.
- Patients with life-treating arrhythmia associated with IHD.
- Suspected Prinzmetal's (variant) angina (coronary vasospasm)

5.3   DIFFERENTIAL DIAGNOSIS

- Oesophageal reflux
- Chronic gastritis
- Musculoskeletal pain
- costochondritis
- Chronic pancreatitis
- Chronic dissecting aorta
- Pneumothorax
- Pericarditis

5.4   MANAGEMENT

The treatment of chronic ischaemic heart disease has two major goals:

(i)   to prevent myocardial infarction (MI) and death, thereby improving life expectancy.

(ii)   to reduce symptoms of angina and the occurrence of ischaemia, which should improve quality of life.

Medical therapy with aggressive cardiovascular risk factor modification is the cornerstone of therapy for chronic ischaemic heart disease. This holds true for patients being treated either medically or with coronary revascularization. The treatment strategies for chronic stable angina is separated into two important divisions:

(1)   anti-anginal therapy and

(2)   education and risk factor modification.

5.4.1   <u>Anti-anginal therapy</u>

Conditions that exacerbate and provoke angina should be considered; these include medications such as vasodilators, excessive thyroid replacement therapy, and vasoconstrictors. Medical problems such as profound anaemia, uncontrolled hypertension, hyperthyroidism, and hypoxemia should also be considered. Primary cardiac disorders such as tachy-and brady-arrhythmias, valvular heart disease (especially aortic stenosis), and hypertrophic cardiomyopathy may also exacerbate angina pectoris and should be excluded. Careful attention to history, thorough physical examination, and selection of appropriate laboratory and other diagnostic studies can help identify these clinical conditions.

β-*blockers*

β-blockers decrease heart rate, contractility and arterial pressure, resulting in decreased myocardial oxygen demand. In patients with chronic stable exertional angina, β-blockers decrease heart rate and blood pressure during exercise, and the onset of angina (ischaemic threshold) is delayed or avoided. Various types of β-blockers are available for the treatment of hypertension and angina (Table 1). All β-blockers appear to be equally effective in reducing angina pectoris. In the treatment of stable angina, it is conventional to adjust the dose of these drugs to reduce the resting heart rate to 50-60 beats per minute.

Table 1: Properties of β-blockers in clinical use

Drugs	Selectivity	Partial agonist activity	Usual dose for angina
Propranolol	None	No	20-80 mg bid
Metoprolol	β1	No	50-200 mg bid
Atenolol	β1	No	50-200 mg bid
Nadolol	None	No	40-80 mg qd
Timolol	None	No	10 mg bid
Acebutolol	β1	Yes	200-600 mg bid
Betaxolol	β1	No	10-20 mg qd
Bisoprolol	β1	No	10 mg qd
Esmolol (intravenous)	β1	No	50-300 mg/kg/min
Labetalol*	None	No	200-600 mg bid
Carvedilol*	None	No	12.5-50 mg bid

* combined α- and β-blocker.

bid- twice a day ; qd – every day; tid – three times a day

The absolute cardiac contraindications for the use of β-blockers are severe bradycardia, pre-existing high degree atrioventricular block, sick sinus syndrome, and severe decompensated left ventricular failure (β-blockers have now been shown to reduce total mortality in patients with compensated heart failure). β-blockers should also be avoided in patients with pure vasospastic angina (Prinzmetal angina) because these agents may induce coronary vasospasm from unopposed α-receptor activity. Asthma and bronchospastic disease, severe depression, and severe peripheral vascular disease are relative contraindications. Most diabetic patients tolerate β-blockers, although these agents should be used cautiously in patients who require insulin. In the absence of contraindications, β-blockers are preferred as initial therapy. The evidence for this approach is strongest in patients with a history of prior MI, for which this class of drugs has been shown to reduce mortality.

Calcium antagonists

Calcium antagonists effectively treat hypertension and angina pectoris. These agents are commonly divided into the dihydropyridine and nondihydropyridine classes (Table 2). Calcium antagonists decrease coronary vascular resistance and increase coronary blood flow. All of these agents cause dilatation of the epicardial coronary vessels and the microcirculation arteriolar resistance vessels. Dilatation of the epicardial coronary arteries is the principal mechanism that allows calcium antagonists to relieve vasospastic angina. Calcium antagonists also concurrently decrease myocardial oxygen demand, primarily by reduction of systemic vascular resistance and reduction in arterial pressure. In addition, certain calcium antagonists (verapamil and diltiazem) reduce myocardial oxygen demand by decreasing heart rate and contractility.

Table 2: Properties of calcium antagonists in clinical use

Drugs	Usual dose	Duration of action	Side effects
Dihydropyridines			
Nifedipine	Immediate release: 30-90 mg daily orally Slow release: 30-180 mg orally	Short	Hypotension, dizziness, flushing, nausea, constipation, edema
Amlodipine	5-10 mg qd	Long	Headache, edema
Felodipine	5-10 mg qd	Long	Headache, edema
Isradipine	2.5-10 mg bid	Medium	Headache, fatigue
Nicardipine	20-40 mg tid	Short	Headache, dizziness, flushing, edema
Non-dihydropyridines			
Bepridil	200-400 mg qd	Long	Arrhythmias, dizziness, nausea
Diltiazem	Immediate release: 30-80 mg 4 times daily	Short	Hypotension, dizziness, flushing, bradycardia, edema
	Slow release: 120-320 mg qd	Long	
Verapamil	Immediate release: 80-160 mg tid Slow release: 120-480 mg qd	Short Long	Hypotension, myocardial depression, heart failure, edema, bradycardia

bid – twice a day; qd – every day; tid - three times a day

Short-acting dihydropyridine calcium antagonists have the potential to enhance the risk of adverse cardiac events and should be avoided. Long-acting calcium antagonists of the dihydro-and nondihydropyridine class relieve angina and are appropriate initial therapy in patients with contraindications to β-blockers. They can also be substituted for β-blockers in patients who develop unacceptable side effects or can be used in combination with β-blockers when initial β-blockers therapy is unsuccessful.

Nitrates

Nitrates dilate epicardial coronary arteries and arterioles and reduce cardiac preload. They also relieve coronary spasm and dynamic stenoses, especially at epicardial sites. Their use is associated with reflex tachycardia, an effect that may increase myocardial oxygen demand. This response may be blunted by the concomitant administration of β-blockers or calcium antagonists, such as diltiazem or verapamil, which slow conduction.

Nitrates are available in multiple preparations exhibiting wide duration in clinical effect (Table 3). Short-acting nitrates, which are most often administered as sublingual tablets or buccal mucosal spray, are commonly used to treat acute episodes of angina. Unless contraindications exist, all patients with chronic stable angina should be given a short-acting nitrate and should be properly instucted in its use. The efficacy and action of long-acting nitrates depend on the dosing, bioavailability, and half-life of the various preparations. The most important aspect of long-term nitrate therapy is to ensure an adequate nitrate-free interval (typically night time), which will prevent nitrate tolerance.

Table 3: Properties of nitrates in clinical use

Compound	Route	Dose	Duration of effect
Nitroglycerin	Sublingual tablets	0.3-0.6 mg up to 1.5 mg	Short 2-3 minutes
	Spray	0.4 mg as needed	Short 2-3 minutes
	Ointment	2% 6 X 6 in., 15 X 15 cm 7.5-40 mg	Effect up to 7 h
	Transdermal	0.2—0.8 mg/h every 12 h	8-12 h during intermittent therapy
	Oral sustained release	2.5-13 mg	4-8 h
	Buccal	1-3 mg 3 times daily	3-5 h
	Intravenous	5-200 μg/min.	Tolerance in 7-8 h
Isosorbide dinitrate	Sublingual	2.5-15 mg	Up to 60 min.
	Oral	5-80 mg, 2-3 times daily	Up to 8h
	Spray	1.25 mg on tongue as needed	2-3 min.
	Chewable	5 mg	2-2.5 h
	Oral slow release	40 mg 1-2 times daily	Up to 8 h
	Intravenous	1.25-5.0 mg/h	Tolerance in 7-8 h
	Ointment	100 mg/24 h	Not effective
Isosorbide-5-mononitrate	Oral	20 mg twice daily	12-24 h
		60-240 mg once daily	

In patients with exertional stable angina, nitrates improve exercise tolerance, increase the time to onset of angina, and decrease ST-segment depression during treadmill exercise testing. Combined with β-blockers or calcium antagonists, nitrates produce greater anti-anginal and anti-ischaemic effects in patients with stable angina.

Revascularization

CABG versus medical therapy

Evidence from randomized clinical trials has demonstrated that coronary artery bypass grafts (CABG) improves mortality when compared to medical therapy for patients with significant left main disease, three vessel disease with left ventricular ejection fraction (LVEF) of less than 50%, or multi-vessel disease with involvement of the proximal left anterior descending artery (LAD). Although these trials were performed in an era prior to the availability of percutaneous coronary intervention (PCI) and the routine use of aspirin, β-blockers, and lipid lowering therapy, these surgical indications remain currently valid.

CABG versus PCI

PCI may be considered in patients with multi-vessel CAD (including the proximal LAD), who have anatomy suitable for catheter-based technique, do not have diabetes, and have normal left ventricular function. CABG or PCI may be considered in appropriate patients with single or multi-vessel CAD who have not been treated successfully by medical therapy and can undergo revascularization with acceptable risk.

PCI versus medical therapy

Many trials illustrate that low-risk patients with chronic stable angina pectoris and normal left ventricular function can be treated safely with medical therapy. Because of the risk of procedure-related complications, clinicians must carefully balance this excess hazard with the need for greater symptomatic improvement when selecting patients for PCI.

Antiplatelet agents

Aspirin (75-150 mg) exerts its antithrombotic effect by inhibiting cyclo-oxygenase and synthesis of platelet thromboxane A_2. It is effective in preventing first heart attacks, improving mortality in acute coronary syndromes, and reducing adverse cardiovascular events in patients with stable angina pectoris. Aspirin should therefore be considered as first line therapy in all patients with chronic ischaemic heart disease. In patients with absolute aspirin contraindications alternative antiplatelet regimes such as dipyridamole (Persantine), ticlopidine (Ticlid), glycoprotein 11b/111a receptor antagonists may be considered, although the clinical data supporting their use in angina pectoris is less established.

Lipid lowering therapy

Of the classes of antihyperlipidemic drugs, the hydroxy-methylglutanyl coenzyme A reductase inhibitors or **statins** are the most potent agents available in lowering total and low density lipoprotein (LDL) and atherogenic cholesterol and are the only lipid-lowering agents shown to improve overall mortality in clinical trials. Furthermore, statins also exhibit anti-inflammatory properties, which may positively modify the artherosclerotic process.

Therefore, statins should be considered in all patients with established CAD and LDL cholesterol $\geq$ 130 mg/dL, targeting the on-therapy LDL cholesterol to less than 100 mg/dL. In general, modification of diet and exercise are less effective than statins in achieving the target levels of LDL cholesterol; thus, lipid lowering pharmacotherapy is usually required in patients with angina and should be considered as first-line therapy in disease modification.

A high density lipoprotein (HDL), antiatherogenic cholesterol level of less than 35 mg/dL is an independent risk factor for adverse cardiovascular events. Hygienic measures such as weight loss, exercise, alcohol consumption, diabetes

control, and smoking cessation may provide modest increases in HDL cholesterol. Recently, a randomized trial comparing the fibrate gemfibrozil to placebo demonstrated reduced cardiovascular mortality in patients with CAD and an isolated HDL cholesterol of less than 35 mg/dL.

5.4.2 <u>Education and Risk Factor Reduction</u>

Identification and treatment of coronary disease risk factors in patients with angina is necessary for optimal secondary prevention of chronic ischaemic heart disease. Established and modifiable coronary disease risk factors, such as cigarette smoking, hypertension, diabetes mellitus, and hyperlipidemia, are common in patients with chronic ischaemic heart disease. These risk factors are readily amenable to modification, and their treatment can affect clinical outcome favourably.

Studies show that the risk of death from CAD is inversely proportional to education. It is therefore important for the physician to assess the patient's baseline understanding of his or her condition and to appropriately educate the patient and his or her family on the pathology and pathophysiology of the disease, prognosis of condition, and treatment and risk factor reduction options.

Obesity

Obesity is a common condition associated with an increased risk for coronary artery disease and mortality. Obesity is also associated with and contributes to other coronary risk factors, including high blood pressure, glucose intolerance, low levels of HDL cholesterol, and elevated triglycerides. Thus, much of the increased CAD risk associated with obesity is mediated by these factors. Weight reduction in obese patients with coronary disease can likely reduce the risk of future events because weight reduction will reduce other modifiable risk factors and reduce the increased myocardial oxygen demand imposed by obesity. Weight

reduction is therefore recommended in all obese patients with ischaemic heart disease. Strategies to improve weight reduction include:

a. lowering total caloric intake,
b. eating foods low in cholesterol and saturated fat and high in fibre and unsaturated fat, and
c. exercise training.

Exercise

Exercise training improves blood pressure, glucose control, lipid profile, exercise endurance, and objective measures of ischaemia. In addition, exercise training may lead to changes in weight and a sense of well-being. For these reasons, a regular exercise program should be part of a multifactorial intervention program in patients with stable chronic ischaemic heart disease.

5.5 FOLLOW-UP

Patients who are successfully treated for chronic ischaemic heart disease should have follow-up evaluation every 4 to 12 months. A more precise interval cannot be recommended because many factors influence the length of follow-up period. Five questions must be answered regularly during the follow-up evaluation:

(1) Has the patient decreased the level of physical activity?
(2) Have the patient's anginal symptoms increased in frequency and become more severe since the last visit?
(3) How well is the patient tolerating therapy?
(4) How successful has the patient been in reducing modifiable risk factors and improving knowledge about ischaemic heart disease?
(5) Has the patient developed any new comorbid illnesses or has the severity or treatment of known comorbid illnesses worsened the patient's angina?

Patients who cannot reliably identify and report changes in their status or who need more support with their treatment or risk factor reduction should be seen more frequently.

5.6   INDICATIONS FOR REFERRAL TO CARDIOLOGIST

In general, most patients with chronic stable angina initially present to their primary care physician. The preliminary diagnosis, risk stratification, medical management, risk factor modification, and patient education are performed under direction of the primary care physician. Patients with worsening anginal symptoms (Canadian Cardiovascular Society Angina Class III or IV) or presenting with any acute coronary syndrome should be referred to a cardiologist. Furthermore, patients with an increased risk for adverse cardiac events, left ventricular ejection fraction (LVEF) $\leq$ 35%, high-risk exercise treadmill score, multiple large ischaemic zones on stress imaging, successfully resuscitated from sudden cardiac death, sustained ventricular tachycardia, significant angina pectoris symptoms despite medical treatment should be considered for coronary angiography and evaluated by a cardiology specialist. Those patients who are co-managed by their primary care physician and cardiologist may alternate follow-up evaluations, provided that communication among physicians is excellent and all appropriate issues are addressed at each visit.

5.7 KEY POINTS

- Angina pectoris is the most common initial manifestation of chronic ischaemic heart disease.

- A mismatch between coronary blood flow and myocardial oxygen demand results in myocardial ischaemia.

- Medical treatment of patients with chronic ischaemic heart disease should include: aspirin and anti-anginals, β-blocker and blood pressure control, cholesterol lowering and cigarette cessation, dietary modification and diabetes assessment, and exercise and education.

- Coronary angiography should be considered in patients with chronic angina and any of the following:
  1. Left ventricular ejection fraction less than 35%
  2. High risk exercise treadmill score
  3. Multiple large ischaemic zones on stress imaging (especially if anterior distribution),
  4. Successful resuscitation from sudden cardiac death,
  5. Sustained ventricular tachycardia, and
  6. Significant angina pectoris despite medical treatment.

- Coronary artery bypass graft (CABG) should be considered in patients with chronic angina and any of the following:
  1. Significant left main disease,
  2. Three vessel coronary artery disease (CAD) and depressed left ventricular functions, and
  3. Multi-vessel CAD that includes the proximal segment of the left anterior descending (LAD).

- Percutaneous coronary intervention (PCI) may be considered in patients who have multi-vessel CAD (including the proximal LAD), have anatomy suitable for catheter-based technique, do not have diabetes, and have normal left ventricular function.

- Coronary revascularization with either PCI or CABG may be considered in appropriate patients with single or multi-vessel CAD who have failed medical management of chronic angina pectoris.

6.   ACUTE CORONARY SYNDROMES

Acute coronary syndromes share a common pathophysiology - rupture of a vulnerable plaque with subsequent activation of dynamic inflammatory, thrombotic and ischaemic cascades.

Acute coronary syndromes **(ACS)** may be divided into two broad categories:

1.   non-ST elevation ACS: **unstable angina (UA)**

                                        non-ST elevation MI and

2.   **ST elevation myocardial infarction (MI).**

Acute non ST elevation coronary syndrome without injury (ie, with negative serial enzyme measurements) is termed **unstable angina (UA)**.

Acute non ST elevation coronary syndrome with cell injury, demonstrated by serial measurements of cardiac enzymes is termed **non-ST elevation MI.**

6.2   UNSTABLE ANGINA AND NON ST ELEVATION ACUTE MYOCARDIAL INFARCTION

6.1.1   Clinical presentation

Non ST elevation acute coronary syndrome (unstable angina and non-ST elevation myocardial infarction) is defined as a change in the status of a patient's angina. It includes the following presentations: (a) **new-onset angina**, (b) **angina of increasing severity, frequency, duration and lesser or slower response to rest and/or sublingual nitrates,** (c) **angina occuring at rest for the first time** and (d) **post infarction angina**.

The term unstable angina implies a more serious clinical situation than chronic stable angina since it may be an immediate precursor of myocardial infarction.

6.1.2 <u>History, Examination and Investigations</u>

History

A carefully obtained history is **essential** in establishing the diagnosis.

A non-ST elevation ACS should be suspected in any patient who presents with new or worsening chest discomfort that may be consistent with angina. History should focus on: risk factors for coronary artery disease, pre-existing diagnosis of CAD and/or stress-testing, and the nature of the chest discomfort.

Important elements include location, quality, radiation, duration, associated symptoms, and factors that trigger, worsen, or relieve the pain.

The diagnosis is easier in patients with preexisting stable effort angina, acquiring new features during some recent time-usually 1 to 4 weeks.

In patients without prior (more than 4 weeks) history of angina (de novo angina) history may be less easy though.

Some patients may not present with chest pain, but rather with arm or jaw pain, epigastric discomfort, dyspnoea, or nausea.

Questions should also be directed toward establishing potential non-ischaemic sources of pain which may mimic an acute coronary syndrome .

Physical Examination

Physical examination during acute coronary syndromes may be relatively unremarkable. The patient may appear anxious or diaphoretic (cold sweats).

Tachycardia or bradycardia, as well as extremes of blood pressure, may be present, but vital signs are often normal.

Heart failure or valvular regurgitation are unusual acute consequences of non-ST elevation ACS, but may be concurrently present in patients with underlying cardiac disease.

Signs of peripheral vascular disease, such as carotid bruits or diminished extremity pulses, should increase the suspicion of atherosclerotic coronary disease.

Physical examination is perhaps most useful to **diagnose non-ischaemic causes of pain, such as costochondritis, pneumonia, obstructive pulmonary disease, pericarditis, aortic dissection, upper gastrointestinal disease etc.**

Laboratory Testing

Complete blood count, electrolytes, renal function tests, and chest x-ray are useful to diagnose other causes of symptoms and to evaluate for concurrent medical conditions, such as renal insufficiency. Clotting times should be obtained to exclude bleeding diatheses prior to potential initiation of antithrombotic therapies.
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The cornerstones of diagnostic testing, however, are ECG analysis and measurements of serum cardiac markers.

ECG analysis

A baseline ECG, preferably during the chest pain, is essential in any risk stratification for the purpose of management.

Non ST elevation acute coronary syndromes are subdivided into

1. those without ECG changes, i.e a normal ECG (about 40% of patients)
2. those whose ECG shows ischemic changes (biphasic or inverted T waves, ST-depression, and/or transient ST elevation – 60%)

The following ECG abnormalities are predictive of an incrementally higher risk: transient T wave inversion, transient ST elevation, ST depression, and transient both ST elevation and depression.

Transient or dynamic ECG changes are suggestive of ischaemia, and therefore the need for serial ECGs in the first few hours after presentation especially when pain persists, when pain recurs after initial improvement, or when pain subsides and initial ECG abnormalities disappear..

Development of persistent ST elevation or left bundle branch block should prompt urgent evaluation for thrombolytics or percutaneous intervention.

Serum cardiac markers

Various serum markers have demonstrated utility as markers of myocardial injury (infarction), including **serum creatine kinase (CK)-MB and troponin I or T**.

One main advantage of using both markers is their different time course: while either may detect early injury, CK-MB values typically return to normal by 1 to 2 days, while troponin levels usually remain elevated for 5 to 14 days.When both

markers are measured in the first 24 hours after presentation, situations arise in which one serum marker is normal while the other is mildly elevated.

Serum myoglobin is sometimes useful as an initial screen in doubtful cases because of its higher early sensitivity (2 hours),

Initial Risk Stratification

One important goal of the initial evaluation is identification of high risk patients, those with an increased risk of death or MI in the first 30 days.

Various clinical features are predictive of this higher risk.(Table 4).

Table 4: Features predictive of increased risk in non-ST elevation ACS

History
Older age
Diabetes mellitus
Known coronary artery disease
Postinfarction angina
Physical examination
Signs of depressed left ventricular function
Hypotension
ECG changes
ST depression (and/or transient elevation)
Laboratory: elevated CK-MB or troponin
Clinical course
Need for intravenous nitroglycerin
Recurrent or refractory ischaemia
Gallbladder, liver, gastric, or pancreatic disease

Identification of patients with these features should lead to strong consideration for institution of more intensive antithrombotic therapy

6.1.3   Differential Diagnosis

The diagnosis of non-ST elevation ACS should be suspected in any patient who presents with new or worsening ischaemic chest pain consistent with angina, with ECG abnormalities suggestive of ischaemia but without ST elevation.

Table 5: Non-ischaemic causes of chest discomfort

Costochondritis	Muscle strain
Gastroesophageal reflux	Oesophageal rupture
Aortic dissection	Pericarditis
Congestive heart failure	Arrhythmias
Pulmonary embolism	Pneumothorax
Pneumonia	Pleurisy
Obstructive always disease	Anxiety

However, as noted previously, in many cases the initial ECG may be mildly abnormal or even normal. In these cases, the diagnosis rests on the clinical history of chest discomfort of **a new or more severe nature, occurring more frequently, at lower levels of activity, or at rest**. It is this group of patients, wherein typical ischaemic ECG changes are absent, that presents perhaps the greatest diagnostic and management challenge to primary care providers, emergency room personnel, and cardiologists alike.

In general the clinician should ask himself the following question: **Given the clinical history of new or changing chest discomfort, combined with the**

physical examination, ECG, and laboratory findings, do I believe that there may have been acute plaque rupture with thrombus formation?

If the answer is no, then, although hospitalization and observation of the patient might still be warranted, interventions such as treatment with heparin, intravenous nitrates , glycoprotein receptor inhibitors, and cardiac catheterization may not be indicated.

6.1.4 <u>Management</u>

Patients with low risk unstable angina may be managed as outpatients with early follow-up evaluations.

Patients with high risk unstable angina (see table 4 above) require hospitalisation with imposition of bed rest and sedation in an ECG-monitored environment.

Initial medical management of High Risk Unstable Angina and non Q-wave MI

Hospitalisation*, preferably in a coronary care setting*

Bed rest (24 – 48 hrs)
Sedation as needed

Correction of precipitating conditions such as HBP, arrhythmias, anaemia, hypoxaemia
Goals of treatment:
    Relieve ischaemic symptoms with antianginal drugs at therapeutic dosage
    Inhibit thrombosis

Drug treatment

A. Intravenous nitroglycerin (GTN, ISDN)- for 24 hrs. If asymptomatic after 24 hrs - oral long acting nitrates

B. Beta blockers (if not contraindicated)

C. Calcium antagonists (verapamil, diltiazem) if beta-blockers contraindicated
N.B. Nifedipine only if associated with β-blockers

D. Narcotic analgesics for pain refractory to antianginal drugs

E. Heparin- bolus of 70-80 units/kg + infusion of 15-18 units/kg/hour.
KPTT should be checked every 6 hrs until therapeutic level of 1.5 – 2.0 (50-70 sec)

Low molecular weight heparin (enoxaparin) – 1 mg/kg bd for 48 hours (or more if angioplasty contemplated)

F. Aspirin 150-300 mg od
Ticlopidine if aspirin contra-indicated - 250 mg bd
Glycoprotein 11b- 111a receptor inhibitors (Reopro, Integrelin)

G. Lipid lowering therapy

Unless contraindicated, all patients with suspected ACS should be given aspirin (150–300 mg orally).

If pain is present, nitroglycerin tablets should be given sublingually, with subsequent further sublingual, transdermal, or intravenous doses as necessary.

If pain is not completely relieved with nitrates, intravenous opiates such as morphine sulphate should be considered.

If oesophageal or gastric pain is suspected, an oral antacid can be given as well. Heart rate, blood pressure, and rhythm should be monitored closely.

If there is significant hypertension (e.g., blood pressure > 170/100 mmHg), tachycardia, persistent chest pain, or ischaemic ECG changes, intravenous beta-blockers should be administered in the absence of contraindications.

Oral beta-blockade (eg., atenolol 25–50 mg once daily) should be given to all patients without contraindications, even when vital signs are normal.

If the clinical suspicion of ACS is quite low, then the above treatment combined with observation in a monitored setting may be adequate.

For patients at higher risk, and for all patients with persistent pain, recurrent pain, or ECG changes, further antithrombotic therapy is indicated, consisting of heparin with or without a glycoprotein receptor inhibitor .

Antithrombotic and Anticoagulant Therapy

The mainstays of antithrombotic and anticoagulant therapy have been **aspirin** and unfractionated **heparin**. Two newer classes of agents, however, show substantial benefit in the treatment of non-ST elevation ACS. These are (a) low molecular weight heparins and (b) glycoprotein IIb/IIIa receptor inhibitor

Low molecular weight heparins

Treatment with the low molecular weight enoxaparin (Lovenox) reduces significantly the combined endpoint of death, myocardial infarction, and recurrent angina or urgent revascularization, compared with unfractionated heparin.

Dosing is 1 mg/kg subcutaneously twice daily, started on admission and typically continued until in-hospital risk stratification, revascularization, or hospital discharge.

Practical advantages of this therapy are simplified dosing and administration, absence of coagulation profile monitoring, and potential cost savings.

Glycoprotein IIb/IIIa receptor antagonists

Platelet glycoprotein (GP) IIb/IIIa receptors represent the final common pathway for platelet aggregation, via fibrin binding and cross-linking of the GP IIb/IIIa receptor. Antibody fragment (abciximab) and peptide (eptifibatide, tirofiban) antagonists to the GP IIb/IIIa receptor have been developed, which demonstrate powerful in vitro and in vivo inhibition of platelet aggregation.

Use of GP IIb/IIIa antagonists, therefore, should be considered in addition to aspirin and heparin in any individual suspected of having a non-ST elevation ACS, especially if high-risk features are present, such as elevated serum enzymes, persistent chest pain, or ST depression . In this setting, lower doses of intravenous heparin are indicated, with goal of prothrombin time 1.5 to 2 times control. Contraindications due to bleeding risk are similar to those for thrombolytics. **The cost of those drugs is still, however, very prohibitive.**

In-hospital risk stratification

If chest pain persists or recurs on the medical and antithrombotic therapies described, in association with high clinical suspicion or objective evidence of ischemia such as positive serum markers or ECG changes, then cardiac catheterization is typically indicated.

However, while this may occur in a significant proportion of patients, many patients with non-ST elevation ACS can be made pain-free with nitrates, β-blockers, opiates, and antithrombotic therapy.

Additionally, others may have recurrent chest pain, but with low clinical suspicion and no objective evidence of ischemia. In these groups of patients, in-hospital risk stratification is indicated after an initial period of medical stabilization (typically 18–48 hours).

Revascularization

If cardiac catheterization is performed, either due to refractory ischaemia or as part of risk-stratification as described above, then revascularization should be considered if suitable coronary anatomy is present.

The choice of percutaneous v/s surgical revascularization depends on both the coronary anatomy and individual patient characteristics and preferences.

All patients should receive maximal preventive therapy.

6.1.5   <u>Follow-up and Secondary Prevention</u>

Many interventions have shown utility in the secondary prevention of ACS, including control of cardiac risk factors such as smoking, hypertension, hyperlipidemia, and diabetes, regular use of aspirin, use of beta-blockers and angiotensin-converting-enzyme inhibitors in selected patients, and adherence to cardioprotective diets containing n-3-omega fatty acids, monounsaturated fats, and antioxidants .

All of these interventions act by reducing the inflammatory and thrombotic milieu of the atherosclerotic vessel.

6.1.6 Indications for referral to Cardiologist

Patients with worsening anginal symptoms or presenting with any acute coronary syndrome should be referred to a cardiologist.

Those patients who are co-managed by their primary care physicians and cardiologist may alternate follow-up evaluations, provided that communication among physicians is excellent and all appropriate issues are addressed at each visit.

6.2 ST elevation acute myocardial infarction

6.2.1 <u>Clinical presentation</u>

ST elevation myocardial infarction occurs when myocardial oxygen supply is abruptly stopped by prolonged and totally occlusive coronary thrombosis leading to necrosis of the myocardial tissue.

Acute myocardial infarction is a medical emergency in which the mortality is greatest within the first 2 hours after the onset of symptoms. Mortality from acute myocardial infarction can be significantly reduced by rapid transport to a coronary care setting, institution of prompt thrombolytic or mechanical (angioplasty) reperfusion, treatment of ventricular arrhythmias and haemodynamic complications.

<u>**History**</u>

Diagnosis

The diagnosis of acute myocardial infarction requires the presence of at least 2 of the following criteria (1) **clinical symptoms suggestive of an acute MI (2)ECG findings consistent with ischaemia or necrosis and (3) elevated biochemical markers**.

The decision to institute thrombolytic therapy usually is based on clinical findings and ECG, because laboratory confirmation of the clinical diagnosis of acute MI requires several hours.

History

The cardinal characteristics of chest pain of acute myocardial infarction are:

a. **localisation and to a lesser extent - radiation**

b. **duration of continuous ischaemic pain**

c. **response to oral nitrates**

Prodromal symptoms of unstable angina such as de novo angina pectoris during or after exercise or even at rest with increasing frequency are often reported (unstable angina) if properly queried.

The pain of myocardial infarction is usually described as a pressure, compression, constriction, squeezing, boring, or burning in the chest, characteristically radiating to the left arm, but also sometimes involving the neck, jaw, epigastrium, right arm, or back, lasting continuously for over 30 minutes, not relieved by rest or nitroglycerin administration, and often accompanied by anxiety, apprehension, restlessness, hypotension, nausea, paleness and sweating.

Almost 25% of patients with acute myocardial infarctions may be completely asymptomatic at the onset of coronary occlusion ("silent infarcts").

In the elderly or patients with diabetes, chest pain might be less severe and other symptoms such as syncope, faintness, or dyspnea may be present.

The acute symptoms of MI are frequently associated with stress such as anger or upsetting life events, or occasionally excessive physical exertion .

6.2.2 Examination and Investigations

1. Physical Examination

The rapid, risk-stratification orientated physical examination should be directed towards identification of:

1. haemodynamic instability, and
2. pulmonary congestion

New systolic murmurs are particularly important, suggesting the presence of ischaemic mitral regurgitation (due to papillary muscle dysfunction or more dramatically due to papillary muscle rupture) or an acquired ventricular septal defect (due to rupture of the infarcted area of the septum)

There is no diagnostic physical sign of acute MI. The clinical appearance is variable (from the healthy-looking athlete carrying his own suitcase to the somnolent patient in cardiogenic shock). However, to detect complications of acute MI, physical examination—in particular, auscultation of the lungs and the heart—should be done repeatedly every day in all patients.

2. Electrocardiographic Findings

The electrocardiogram serves as the hallmark for diagnosis of acute MI because characteristic ST, T, and Q-wave changes are detectable in most of the patients.

In the early stage of acute coronary occlusion (phase 1 or hyperacute phase) giant positive T waves appear with taller than normal R waves .

In phase 2 (acute phase, more commonly seen at hospital admission), ST-segment elevation is evident with a decrease in amplitude of the R wave, followed by

pathologic Q-wave development that lasts 0.04 seconds or more and reaches 25% of the amplitude of the R wave (pathologic Q- wave, phase 3, when cell necrosis occurs over hours to days).

Over time, the ST segment returns to normal with T wave inversion (terminal negative T wave); the R wave may be lost as a result of transmural necrosis and may be replaced by a QS wave (phase 4, chronic phase). The Q wave or QS wave persists, whereas the T wave may return to positive.

ST-wave elevation may persist in cases of left ventricular dyskinesis or aneurysm. The electrocardiographic leads not representing the infarcted area may show inverse, reciprocal ST-segment alterations.

A new left bundle branch block may be present and, if it persists, usually indicates large anterior wall infarction.

The diagnosis of acute infarction in patients with acute chest pain who present with left bundle branch block is highly probable when there is ST-segment elevation of 1 mm or more concordant with the QRS complex, ST-segment depression of 1 mm or more in lead V1, V2, or V3, and ST-segment elevation of 5 mm or more discordant to the QRS complex. When possible, the current ECG tracing should be compared with previous records.

Determination of myocardial infarct localization according to ECG leads is listed in Table 5.

Table 5: Location of myocardial infarction according to electrocardiographic leads and involved coronary artery

ST-segment elevation in	Ventricular location	Coronary artery involved
V1 through V3	Anteroseptal	Proximal LAD, septal perforators
V2 through V4	Anteroapical	LAD, diagonal branches
I, aVL, V6	Lateral	LAD, diagonal branch, or circumflex
I, aVL	High lateral	1st diagonal branch or circumflex
I, aVL, V3 through V6	Anterolateral	Mid LAD or circumflex
I, aVL, V1 through V6	Extensive anterior	Proximal LAD
II, III, aVF	Inferior	RCA or circumflex, distal LAD
V1 through V2 (ST depression)	Posterior	Posterior descending of RCA, circumflex
II, III, aVF, V5 through V6	Inferolateral	RCA or circumflex
V1, V3R, V4R	Right ventricular	RCA

(LAD—left anterior descending RCA—right coronary artery.)

3. **Cardiac enzymes**

After myocardial injury and cell death, cellular enzymes are released into the bloodstream. The typical time course of enzyme alterations that occur can help to determine the phase of MI.

The most commonly used tests are creatinine kinase (CK), CK-MB, and the very specific troponins (troponin I or T).

For clinical use, it is recommended that cardiac troponin T be measured to distinguish between cardiac and other sources of chest pain. In case of elevated troponin, follow-up measurements of CK are sufficient to determine the course of

myocardial infarction. If chest pain is recurrent, intermittent analyses of CK-MB or troponin T are recommended for detection of reinfarction.

4. **Imaging Techniques**

Global ventricular function and mechanical and hemodynamic complications such as papillary muscle rupture, septal   rupture, pericardial effusion, mitral regurgitation, intraventricular thrombi, and their follow-up all can be assessed accurately by echocardiography.

6.2.3 **Differential diagnosis**

Although in the majority of cases the diagnosis of ST elevation acute MI is straightforward, a rapid but well thought differential diagnosis is essential. It is mandatory that those diseases in which thrombolysis is not only of no use but may even be contraindicated be excluded.

These are: **pericarditis, early repolarisation on ECG**, acute aortic dissection, pneumothorax, ventricular aneurysms, Prinzmetal's angina, left ventricular strain pattern, left bundle branch block, hyperkalaemia, cerebro-vascular accident etc.

6.2.4 **Management**

Acute Management Strategies include:
1. Thrombolysis
2. Platelet antiaggregants
3. Anticoagulants
4. Maintenance of oxygen balance

Because acute MI always represents a life-threatening event, the patient with suspected acute myocardial infarction should be hospitalized immediately to minimize delay in appropriate therapy.

Therapy for acute MI consists of the following six steps:1) relief of pain, 2) relief of anxiety and restlessness, and sedation, 3) reperfusion, 4) anticoagulation, 5)therapy for complications, 6) secondary prevention.

For steps 1 and 2, repeated administration of intravenous morphine (2 to 10 mg or more), is the treatment of choice, which can be combined with antiemetics to reduce side effects.

Initial Management in ALL patients

(thrombolysis or not)

- **General measures**: IV line, blood samples for CE, electrolytes, FBC, lipid profile
- Monitoring for cardiac rhythm, vital signs.
- If no ST elevation – repeat ECG in 1 hour (or earlier) if ongoing chest discomfort
- Pain: adequate analgesia- Morphia IV 2-4 mg to be repeated in 10 min interval until pain is under control (or side effects +)
- Nausea/vomiting – Primperan IV
- Hypotension – adequate volume expansion
- GTN s/l every 5 min (if no C/I), then infusion
- Oxygen 2-4 l/min
- Aspirin per os or Aspegic IV
- Heparin - IV bolus 5000 u/kg, then 1000u/h or enoxaparin 1 mg/kg bd sk
- Beta blockers- Atenolol 5-10 mg IV (if no C/I- bradycardia, hypotension, LVF, HB, COPD, severe peripheral vascular diseases)

Once the patient arrives at the hospital, initial evaluation of the patient's history, coronary risk factors, physical signs, 12-lead ECG tracing, possible contraindications for reperfusion therapy, and steps for treatment should be initiated within 20 to 30 minutes ("door-to-needle time"). The presence of a special thrombolysis nurse might reduce the delay in instituting reperfusion therapy for patients with acute MI.

In the emergency ward the patient should immediately receive aspirin in chewable form, oxygen by nasal prongs or masks (blood gases may be checked, especially in patients with chronic pulmonary diseases), and sublingual nitrates (unless systolic arterial pressure is less than 90 mmHg or heart rate is less than 50 or greater than 100 beats per minute).

The patient should be monitored and have bed rest for 12 to 24 hours as life-threatening arrhythmias, reinfarction, mechanical complications, and death occur most frequently within the first 24 hours after acute MI.

Reperfusion Therapy

The main goal in the treatment of patients with acute MI is to reduce the extent of irreversible myocardial tissue damage and necrosis.

Fibrinolytic therapy should be initiated as early as possible, preferably within the first 3 hours after onset of persistent chest pain, but some benefit may still be derived if administered up to even 12 hours (if pain persists with ST elevation).

Eligibility for thrombolytic therapy

- Chest pain of >30 min and < 12 hrs
- ST segment elevation of > 1.0 mV in two continuous ECG leads indicative of AMI

- New LBBB on ECG

Because fibrinolytic agents increase the risk of hemorrhage, including intracerebral hemorrhage, several cautions and contraindications must be considered before treatment can be initiated. These are:

A. Absolute contraindications:
- Active internal bleeding
- Suspected aortic dissection
- Known intracranial neoplasm
- History of any haemorragic CVA ever
- Or other cerebrovascular event during the past 1 year

B. Relative contraindications:
- Severe hypertension on presentation (BP > 180/110 mm.Hg.)
- History of chronic severe hypertension
- History of prior CVA or other intracranial pathology
- Recent trauma (< 2 weeks) or major surgery (< 3 weeks)
- Prolonged or traumatic CPR (> 10 min)
- Non-compressible vascular punctures
- Recent internal bleeding (< 2-45 weeks)
- Known bleeding diathesis or current INR > 2-3
- For streptokinase- prior exposure or known allergy

Primary Percutaneous Coronary Intervention

Primary percutaneous coronary intervention (PCI) can serve as an alternative to thrombolysis only if performed in a timely fashion by individuals skilled in the procedure and supported by experienced personnel in high-volume centers . Indications include patients with contraindications to thrombolysis, patients at risk for bleeding, and patients with cardiogenic shock

Adjunctive Therapy

Platelet antiaggregants.

Early adjunctive therapy with thrombolysis consists of aspirin, which quickly prevents thromboxane A2 production in platelets and prostacyclin in endothelial cells. Aspirin should be administered at the time of admission to the emergency ward at a dose of 150 to 300 mg, preferably in a chewable form because of its faster absorption, followed by a daily administration indefinitely thereafter.

In case of true aspirin allergy, dipyridamole or ticlopidine may be used.

Heparin (which forms a complex with antithrombin III, thus inactivating thrombin) is recommended in patients with large or anterior MI, known left ventricular thrombus, or previous embolic events. In patients not treated with thrombolytic therapy or with nonselected thrombolytics (streptokinase, anistreplase, urokinase), and in patients without increased risk for systemic emboli, subcutaneous administration of 7500 U (or 1 mg/kg of enoxaparin) twice daily may be administered . Prolonged intravenous heparin therapy has not been shown to decrease the rate of reocclusion.

β-**Adrenergic blockers** are known to reduce myocardial oxygen consumption, as well as acute and long-term mortality and morbidity, and should be administered intravenously as early as possible (on day 1 within 12 hours) followed by oral therapy if contraindications are excluded. Furthermore, β-blockers may reduce infarct size and the incidence of ventricular fibrillation and reinfarction.

ACE inhibitors should be initiated on day 1 within hours of hospitalization in patients with evolving acute MI with ST-segment elevation or left bundle branch block and continued indefinitely in case of impaired left ventricular function (the lower the ejection fraction, the greater the benefit).

Intravenous nitroglycerin is recommended during the first 12-24 hours and beyond this time frame in patients with recurrent angina, large infarction, and pulmonary congestion.

Calcium antagonists, predominantly short-acting nifedipine, may be harmful and should be avoided in patients with acute MI. If β-blockers are ineffective, verapamil or diltiazem might be used in patients with ischaemia or tachycardia in the absence of left ventricular dysfunction or AV block .

Magnesium administration is recommended only to correct documented magnesium deficits, especially in patients receiving diuretics, in the case of torsades de pointes with a prolonged QT interval, and in high-risk patients not receiving reperfusion therapy.

Intensive insulin-glucose administration (24h) during acute MI reduces long-term mortality in patients with diabetes and might be considered in this cohort .

Complications of Acute Myocardial Infarction
Arrhythmias
The incidence of arrhythmias may be as high as 100% in patients with acute MI, and manifests predominantly as premature ventricular beats.

The most serious and life-threatening arrhythmias are ventricular fibrillation and sustained polymorphic ventricular tachycardia requiring early electrical defibrillation (200 J), which is repeated if unsuccessful (up to 360 J). Monomorphic ventricular tachycardia not associated with angina or acute heart failure may be treated with the antiarrhythmic agents lidocaine (1 to 1.5 mg/kg as a bolus, followed by 2 to 4 mg/min intravenously), procainamide, amiodarone, or synchronized electrical cardioversion.

For bradyarrhythmia and heart block, either intravenous atropine or temporary transvenous (or, in some cases, transcutaneous) pacing is recommended. Indications for pacing are symptomatic bradycardia (less than 50 beats per minute) with symptoms of hypotension not responding to atropine; asystole; bilateral bundle branch block; newly developed or indeterminate bifascicular block with first-degree AV block; second-degree symptomatic or Mobitz type II AV block, advanced (complete) AV-block; and incessant ventricular tachycardia, which requires atrial or ventricular overdrive pacing.

Atrial fibrillation is often transient and may be associated with heart failure, atrial ischemia, or pericarditis. It should be treated by either electric cardioversion in patients with hemodynamic instability or ischemia; or by rapid digitalization, β-blockade in patients without contraindications, diltiazem or verapamil, and heparin to avoid embolic events.

Hemodynamic and Mechanical Complications

Congestive heart failure may result from systolic contractile dysfunction due to large necrotic areas or to postischaemic contractile wall motion abnormalities (stunned myocardium). Mechanical complications that worsen cardiac function include myocardial infarct expansion and aneurysm formation, ruptured ventricle, ventricular septal defect, papillary muscle dysfunction and rupture. The primary symptom is dyspnea, caused by pulmonary congestion and tachycardia. Therapy for congestive heart failure should be initiated with diuretics (furosemide, 20 mg intravenously, which may be repeated), nitrates, and oxygen, followed by positive inotropic agents (dobutamine, 2 to 20 μg/kg/min; dopamine 2 to 10 μg/kg/min intravenously).

In case of severe ventricular dysfunction or cardiogenic shock, a Swan-Ganz pulmonary artery catheter for measurements of cardiac output, pulmonary artery capillary wedge pressure pulmonary and systemic resistances may be placed.

6.2.5 Follow-up and Rehabilitation

1.   **Follow-up**

In all patients following myocardial infarction without contraindications, long-term treatment with aspirin and β-adrenergic blockers is recommended .

Recurrent angina should be treated with intravenous nitroglycerin, aspirin, heparin, analgesics, and β-blockers.

ACE inhibitors should be a part of secondary prevention in patients with left ventricular dysfunction.

Calcium antagonists are not routinely recommended after infarction but may be selectively prescribed in patients with specific indications such as hypertension or angina in the presence of preserved left ventricular function.

Carvedilol has been shown to attenuate ventricular remodeling in patients with left ventricular dysfunction after MI, but its long-term effects need to be investigated further.

Table 6: Long-term therapy and secondary prevention after myocardial infarction Treatment

Drug therapy	Comments
Aspirin	In all patients without contraindications
β-Blockers	In all patients without contraindications
ACE inhibitors	In patients with left ventricular dysfunction
Dietary therapy	Low saturated fats and cholesterol
Weight-reduction and exercise in overweight patients	
Lipid-lowering drugs	If LDL cholesterol > 100 mg/dL, HDL cholesterol < 35 mg/dL, triglycerides > 400 mg/dL

2. Rehabilitation after MI

Physical activity should be progressively advanced in stable patients. Before discharge from the hospital patients should receive verbal and if possible written instructions concerning physical activities, medications, diet, and secondary preventive measures.

Rehabilitation should concern all facets of patient's activities: physical, psychological, emotional, socioeconomic, life-style changes, risk factors management, smoking cessation, education, return to work (if feasible), self-control, knowledge of limitations etc.

6.2.6 Post-infarction Evaluation and Secondary Prevention

Coronary angiography and subsequent revascularization, either by PCI or bypass grafting, ultimately should be performed in patients with recurrent angina or evidence of large areas of reversible ischemia on stress testing, and might be considered selectively in all survivors of acute MI.

The prognosis after acute myocardial infarction is related to **four** main factors:

- Extent of left ventricular dysfunction, including degree of left ventricular dilation
- Presence of residual ischemia
- Degree of electrical instability of the myocardium
- Progression of coronary atherosclerosis

A risk stratification should be evaluated in each patient. Noninvasive evaluation of low-risk patients includes submaximal or symptom-limited stress ECG at 4 to 6 days or 10 to 14 days, respectively. For prognostic assessment, after mobilization, ie after 1 week, standard exercise testing should be performed to assess potential exercise-induced angina or left ventricular dysfunction and functional capacity. Stress ECG should be repeated early after hospital discharge and after 4 to 6 weeks.

Patients should be educated about risk factor modification, ie, cessation of smoking, necessity of physical exercise, and effective lifestyle intervention. Diet recommendations consist of low saturated fat and cholesterol for all patients following acute MI.

6.2.7   Key Points

- Acute coronary syndromes share a common pathophysiology—rupture of a vulnerable plaque with subsequent activation of dynamic inflammatory and thrombotic cascades.

- Initial evaluation, using history, physical examination, electrocardiography, and measurement of serum cardiac markers, should be directed toward risk-stratification of the patient as low, intermediate, or high.

- Unless contraindicated, initial treatment should include aspirin, nitrates, beta blockade, and unfractionated or low molecular weight heparin, with the addition of more intensive antiplatelet therapies for high-risk patients.

- After medical stabilisation, strategies of either early cardiac catheterization or initial stress testing are appropriate for further risk stratification.

- All patients should be discharged on preventive therapies, which are effective by reducing the inflammatory and thrombotic milieu of the atherosclerotic vessel.

- Myocardial infarction is defined as cardiomyocyte necrosis due to lack of oxygen supply in relationship to oxygen demand. The underlying cause in most cases is rupture of an intracoronary atherosclerotic plaque with subsequent platelet aggregation and thrombotic occlusion.

- Q wave myocardial infarction is defined as myocardial cell necrosis with development and maintenance of Q waves on the electrocardiogram.

- The key symptom occurring with acute coronary artery occlusion is severe retrosternal chest pain, but symptoms may vary.

- Early hospitalization and intensive monitoring in an intensive care or coronary care unit is required.

- Worldwide mortality of myocardial infarction is 30% to 40%; one third die within the first hours.

- Treatment of choice is early recanalization by either thrombolytic or interventional therapy, including stent implantation, treatment of complications, and secondary prevention.

- The main prospective goal is public education, early identification of myocardial ischemia, and urgent reperfusion therapy.

- The future trend for acute management is the search for the optimal reperfusion regimen with the smallest risk.

- For chronic management, the trend is toward inhibition of ventricular remodeling and long-term improvement of ventricular function.

GUIDELINES ON PRIMARY PREVENTION

OF

CORONARY ARTERY DISEASE IN MAURITIUS

AT

PRIMARY HEALTH CARE LEVEL

Table of Contents

1.1   INTRODUCTION AND RATIONALE

Worldwide, the mortality from cardiovascular disease is projected to rise from 13.2 million in 1985 to 24.5 million, an 85% increase in three decades. This indication has led the World Health Organisation to term coronary heart disease as the 'World's public health enemy number 1'. By 2015, the increase in cardiovascular deaths will be more than double in developing nations, jumping from 72 million to 167 million, or by 132 percent increase in thirty years, while mortality in industrialized nations will rise by 28 percent. This means that coronary artery disease will become the leading killer in both developing and developed country.

Coronary artery disease has also been identified as the primary non-communicable health problem in Mauritius, accounting for nearly one-third deaths in Mauritius. It has a profound and adverse impact on household, families and society and is a major contributor to morbidity, invalidity and mortality.

Pharmacological approaches, intensive care units, coronary angioplasty and bypass surgery can reduce modality from coronary heart disease by one third. However, two third of the decline is credited to measures such a healthy diets, exercise and smoking cessation, optimum control of blood pressure and a normo-glycemic level of glucose. Premature death of Mauritians can be avoided and quality of life improved if a comprehensive risk factors intervention program for CAD can be implemented at the primary health care level, widely perceived to be the backbone of a rational health service system.

1.2   DEFINITION OF PRIMARY PREVENTION

Primary prevention refers to guidance given to persons with no known cardiovascular disease. The first goal of prevention is to prevent the development

of risk factors. The primary care physicians should regularly check for smoking, physical inactivity, elevated lipid levels, elevated blood sugar, high blood pressure and eating habits and other environmental hygiene factors.

1.3 OBJECTIVES OF CORONARY ARTERY DISEASE PREVENTION

The objectives of a coronary artery disease prevention program at the first level of contact of a health system are to:

a. Remain free from coronary heart diseases and enjoy an improved quality of life.

b. Reduce the risk of coronary heart disease events and thereby reduce premature disability, mortality and extend overall survival.

c. Decrease the need for interventional procedures such as angioplasty, bypass grafting and thereby decreasing hospital costs.

d. Reduce the incidence of subsequent myocardial infarctions

1.9 PRINCIPLES OF SCREENING FOR CORONARY ARTERY DISEASE RISK FACTORS:

A coronary artery disease prevention program must meet a number of criteria before it can be used in the general population. These criteria include:

1. Coronary heart disease is a serious public health problem.
2. Techniques for risk factor detection are easy, precise, cost-effective and acceptable to the community.
3. There is a defined screening strategy and a defined intervention and follow-up policy.
4. Trained staff and facilities for screening and intervention are available.
5. Screening and intervention results in a reduction in clinical events, including morbidity and mortality.
6. Screening has no adverse effects.
7. Cost of screening and intervention is justified in relation to the outcome.

All these 7 criteria are met by CAD in Mauritius, qualifying a screening programme.

1.5 SITUATIONAL ANALYSIS OF RISK FACTORS FOR CAD

NATIONAL RISK FACTORS OF CORONARY ARTERY DISEASE SURVEYS (87/92/98)

Table one: Prevalence of selected risk factors for CAD in adults aged 25 and above

Risk Factors	87	92	98
1. Obesity %			
Male	24.8	33.4	36.1*
Female	35.7	47.7	44.2*
2. Smoking			
Male	57.9	47.3	42
Female	7.0	4.8	3.3
3. Abusive Alcohol intake			
Male	17.0	22.2	23.2*
Female	1.3	2.7	7.0*
4. Physical Activity			
Male			
Female	17.0	22.2	23.2
	1.3	2.7	7.0
5. Lipids			
a. Male			
Total cholesterol > 5.2 mmol	56.4	33.8	49.7*
HDL cholesterol < 1.0 mmol	22.7	18.4	57.0*
Triglycerides > 2.0 mmol	28.2	25.1	41.1*
b. Female			
Total cholesterol > 5.2 mmol	52.9	31.7	37.6*
HDL cholesterol < 1.0 mmol	13.9	9.2	47.3*
Triglycerides > 2.0 mmol	13.7	11.2	22.2*

N.B: *   problem areas where primary prevention measures need to be reinforced

DIABETES MELLITUS AND IMPAIRED GLUCOSE TOLERANCE PREVALENCE NATIONAL SURVEYS 87/92/98 IN ADULTS AGED 25 AND ABOVE

	87	92	98
MEN DM Prevalence % IGT Prevalence %	14.2 15.6	16.3 14.9	20.8 11.8
WOMEN DM Prevalence % IGT Prevalence %	14.5 22.5	17.4 19.3	20.6 20.2

PREVALENCE OF HYPERTENSION IN ADULTS AGED 25 AND ABOVE

	87	92	98
MEN	31.7	26.5	30.0
WOMEN	30.2	26.2	29.6

1.6   DEFINITION OF RISK FACTOR

The term risk factor describes those "characteristics found in healthy individuals to be independently related to the subsequent occurrence of coronary heart disease and, where modifiable, to be reversible.' For example, cholesterol is an independent risk factor for coronary heart disease.   Epidemiological and evidence-based studies have shown that cholesterol lowering by one percent is associated with a decrease in the risk of coronary heart disease of about 2%. There is overwhelming scientific evidence that lifestyle modification and risk factor reduction can retard the development of coronary heart disease both before and after the occurrence of a clinical event. Previously undue emphasis was placed on individually high risk factors rather than the overall level of risk based on a combination of risk factors. A multifactorial assessment of coronary heart disease acknowledges 3 important facts:

1. Coronary heart disease has a multifactorial aetiology
2. Risk factors have a multiplicative effect.
3. As physicians, we are dealing with the whole person (holistic view), not isolated risk factors.

LIFESTYLE AND CHARACTERISTICS ASSOCIATED WITH INCREASED RISK OF FUTURE CHD EVENTS

Lifestyles	Modifiable characteristics	Non-modificable characteristics
Diet high in saturated fat, cholesterol and calories	Elevated blood pressure	Age
Tobacco smoking	Elevated plasma Total cholesterol LDL cholesterol Low HDL cholesterol Elevated plasma TG	Sex Post menopause
Excess alcohol consumption Sedentary life Sustained psycho-emotional stress	Diabetes Obesity Thrombogenic factor ?	Family history of CHD at early age Male < 55 yrs Females < 65 yrs

1.7   PRIORITIZATION FOR CORONARY HEART DISEASE PREVENTION

Primary prevention activities for coronary heart disease should be downsized and prioritized under the following targeted groups:

a. Patients with established coronary heart disease should be given first priority for prevention as they are at high risk of fatal and non-fatal complications of their disease.

b. Next priority should be given to individuals in the general population who are at high risk of developing coronary heart disease.

c. Third priority for prevention is close relatives of patients with very early onset of coronary heart disease particularly the first-degree blood relatives of patients.

d. Increasing age, ethnicity, socially economically deprived communities, geographical regions are other variables to be considered for primary prevention.

1.8   SCIENTIFIC BASIS FOR RISK FACTOR MODIFICATION

Unifactorial randomized control trials of diet; blood pressure and cholesterol lowering have shown reductions in cardiovascular morbidity and mortality. However, the WHO collaborating group study has confirmed that the multi factorial intervention is more cost effective in modifying the risk factors of CAD.

A. DIET

Diet is a determinant of CAD. Its effect on the development of atherosclerosis is mediated through the influence of biological risk factors e.g. LDL, HDL, BP, obesity, diabetes and hypertension.

A healthy diet is characterized as:

1. Low in saturated fatty acids
2. High in unsaturated fatty acids
3. Rich in complex carbohydrates

The traditional Mediterranean diet (low saturated fat + high unsaturated fat especially monounsaturated) and Japanese diet (low saturated fat + high complex carbohydrates) are associated with the best life expectancy in the world. The physician's training does not encompass aspects of dietary counseling. Therefore, assistance from a dietician or a specially trained nurse for dietary interviews is of considerable value.

B. OBESITY

Obesity can be assessed by three measures mainly:
   a. Body mass index
   b. Waist hip ratio
   c. Waist circumference

a. Body mass index

Body weight is expressed in terms of BMl. It is calculated as weight in kg / height (in m^2) squared and is recommended by the WHO expert committee for assessing degree of obesity as shown in the table below.

ASSESSMENT OF OBESITY USING BODY MASS INDEX

Body mass index (kg/m^2)	WHO classification	Popular description
< 18.5	Underweight	Thin
18.5 – 24.9	Normal weight	Normal weight
25.0 – 29.9	Grade 1 overweight	Overweight
30.0 – 39.9	Grade 2 overweight	Obese
> 40.0	Grade 3 overweight	Morbidity obese

b. Waist hip ratio

The ratio of hip to waist circumference is also widely used in epidemiology as an index of central obesity. Central obesity with the accumulation of fat to the trunk and abdominal cavity is associated with high prevalence of lipid abnormalities, hypertension and abnormal glucose tolerance and insulin resistance leading to increased coronary artery disease. The normal value of waist hip ratio should not be greater than 1.

c. Waist circumference

There is also good evidence from population based studies that waist circumference is also a useful index of obesity. Measurement of waist circumference (one measurement and no calculation with the person standing), midway between the lowest rib and the iliac crest, is also recommended for clinical assessment of the degree of obesity and follow up during weight reduction.

ACTION LEVELS FOR WAIST CIRCUMFERENCE IN MEN & WOMEN

	Non-obese	Action level (Alerting zone)	Action Level 2 (Professional advice needed)
Men	< 94 cm	94 – 101 cm	> 102 cm
Women	< 80 cm	80 – 87 cm	> 88 cm

C.   SMOKING

The health hazards of smoking are well documented.   Smoking is responsible for 50% of all avoidable deaths and one half of these are due to cardiovascular death. The risk of future cardiovascular death is particularly high if smoking starts before the age of 15. Passive smoking also increases the risk of cardiovascular disease. According to WHO estimates, annual premature adult mortality among males in developed countries due to smoking is 800,000 per year. Of this total, 300,000, (40%) occur due to cardiovascular disease.

SMOKING PATTERN ACROSS DEVELOPED & DEVELOPING COUNTRIES

	Global	Developed countries	Developing countries
Men	48%	41%	50%
Women	12%	21%	8%

D.   ALCOHOL

Alcohol consumption shows a J-shaped OR U-shaped relationship to the risk of all cause mortality. Moderate use of alcohol (10 – 30 g of ethanol per day for men and 10 – 20 g for women) is considered safe. Safe drinking levels increase

plasma HDL cholesterol and this may explain the protective effect of alcohol. However, heavy drinking particularly binge drinking, increases the risk of sudden arrhythmic death. According to the Mauritius non-communicable disease survey 1998 (preliminary report), alcohol consumption like smoking does not vary significantly across age groups. However, the proportion of heavy drinkers is highest in men in their forties. Hence, this group should be targeted to prevent the harmful effects of alcohol related problems.

E.   PLASMA LIPIDS

Plasma lipids can be measured by using:

1.   Friedwald's formula
2.   Ratio of total cholesterol to HDL cholesterol

1.   FRIEDWALD'S FORMULA

Total cholesterol, triglycerides and HDL cholesterol are measured in our laboratory from a venous sample taken in the non-fasting state. With these three measurements, the part of cholesterol carried in LDL can be calculated according to Friedwald's formula.

a.   In mmol per litre (normal value LDL - C < 3.0 mmol per litre)

LDL cholesterol = total cholesterol – HDL cholesterol – (0.45 x triglyceride level)

b.   In mg per deciliter (normal LDL – C < 115 mg per deciliter)

LDL cholesterol = total cholesterol – HDL cholesterol – (0.2 x triglyceride level)

Friedwald's calculation is a cheaper and more reliable estimation of LDL cholesterol than commercially available direct measurement of LDL-cholesterol based on immuno separation.

2. RATIO OF TOTAL CHOLESTEROL TO HDL-CHOLESTEROL

Measurement of the above ratio is also a good indicator of risk. A total cholesterol/HDL ratio > 5, indicates coronary heart disease risk.

HDL cholesterol is cardio-protective whereas LDL cholesterol causes atherosclerosis. High triglyceride levels (> 2.0 mmol) have also been identified as a risk factor for coronary heart disease. A large body of evidence has demonstrated that cholesterol lowering reduces the risk of coronary heart disease events. In general, a reduction of LDL cholesterol of 10% would lead to a reduction in the risk of coronary heart disease of about 20% and this has turned out to be the case in clinical trials of both primary and secondary prevention.

There are four classes of lipid lowering drugs in current use (statins, fibrates, resins, niacin) and one or more drugs of each class have been shown to reduce CHD morbidity and mortality. Evidence for efficacy and safety in primary prevention is strongest for the statins.

F. PHYSICAL ACTIVITY

Regular aerobic physical activities have favourable effects on body weight, blood pressure, plasma lipids, glucose tolerance and insulin sensitivity. Physical activity recommendations have to define the intensity, duration and frequency of exercise. The intensity of exercise for healthy individuals is best defined in terms of target heart rate during peak exercise as shown below.

SUITABLE TARGET HEART RATE DURING EXERCISE, ACCORDING TO AGE [60-75% of maximum heart rate (220-age)]

Age (years)	Heart rate per minute
20 – 29	115 – 145
30 – 39	110 – 140
40 – 49	105 – 130
50 – 59	100 –125
60 – 69	95 – 115

The target heart rate is easily achieved by exercises involving the use of large muscle groups.   Brisk walking, jogging, cycling, swimming, volleyball, aerobic dancing and rope jumping are examples of such activities. Below is provided an indication of the number of calories spent during various types of exercise:

Brisk Walking	3.6 calories/minute
Jogging	5.0 calories/minute
Running	5.0 calories/minute
Swimming	6.0 calories/minute
Cycling	4.5 calories/minute
Tennis	7.0 calories/minute

The duration of physical activity should preferably be 30 – 40 minutes, including 5 – 10 minutes warm up phase before the 20 – 30 minutes phase and 5 – 10 minutes cool down phase at its end and as frequent as 4-5 times weekly.

G.   BLOOD PRESSURE

Many epidemiological studies have demonstrated the importance of elevated blood pressure as a risk for coronary heart disease in both men and women. The decision to start drug therapy depends not only on the grading of blood pressure level but also on the overall cardiovascular risk which calls for a proper history, physical examination and laboratory examinations to identify the:

1.   Presence of cardiovascular disease
2.   Coexistence of risk factors
3.   Incidence of target organ damage

Blood pressure should be lowered slowly and carefully because myocardial necrosis, coronary atherosclerosis and cardiac hypertrophy may render coronary autoregulation less effective in preserving organ perfusion when blood pressure is reduced too rapidly.
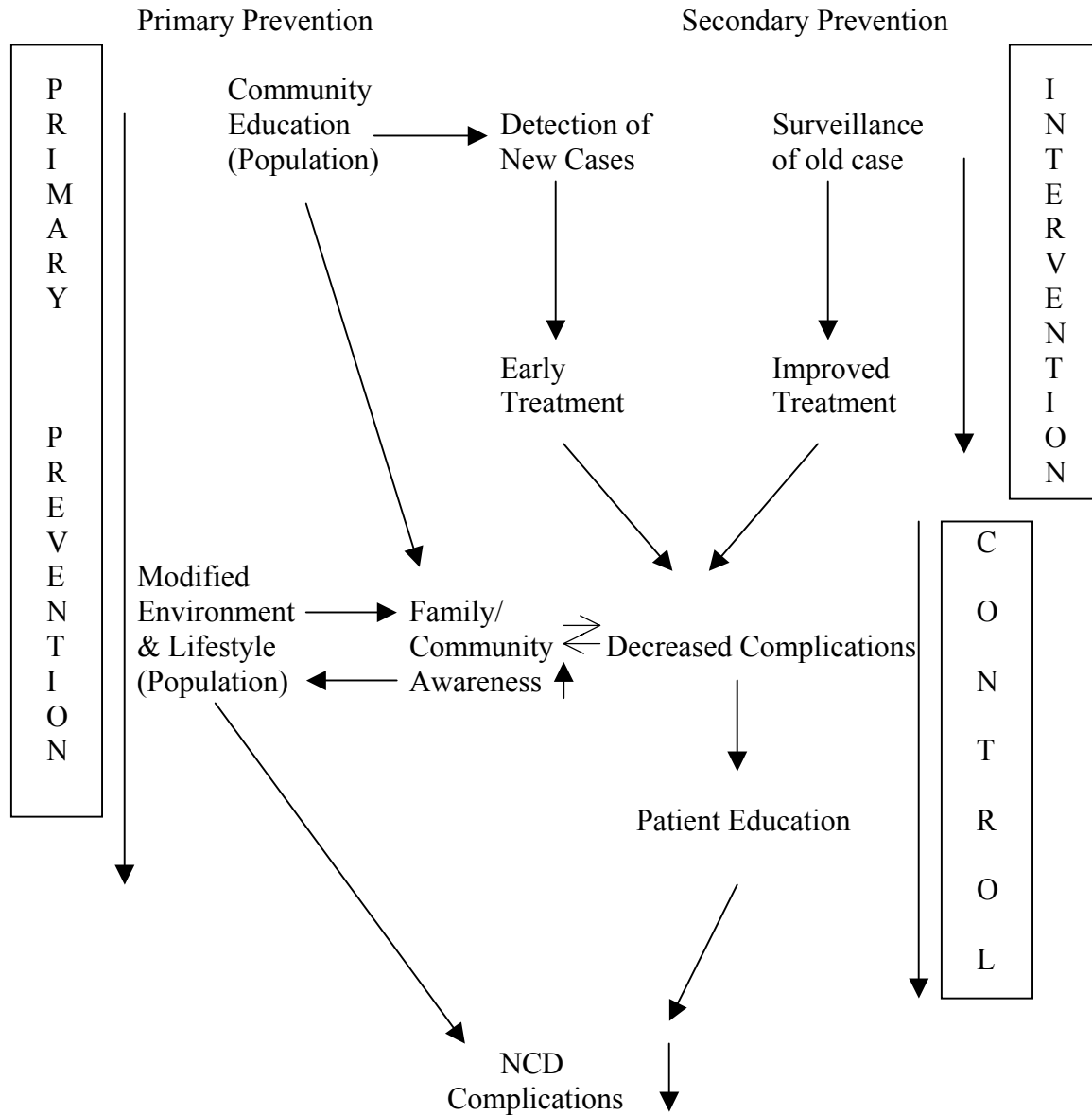
(Refer to hypertension guidelines)

H.   DIABETES

Type 1 and type 2 diabetes are associated with a marked increase of CHD. The excess cardiovascular risk associated with type 1 diabetes becomes evident after the age of 30. Type 2 diabetes is associated with more profound abnormalities in cardiovascular risk factors than type 1 diabetes. Even IGT is associated with a cardiovascular risk pattern characteristic of type 2 diabetes namely high triglyceride level, low HDL value, increased value of hypertension and hyperinsulinemia. The Diabetes Complication and Control Trial (DCCT) showed that glucose control is important for the prevention of long term complications.

(Refer to Diabetes Guidelines)

MODEL FOR PRIMARY AND SECONDARY PREVENTION PROGRAM FOR NCD

Primary Prevention   Secondary Prevention

P R I M A R Y P R E V E N T I O N	

Community Education (Population) → Detection of New Cases

Surveillance of old case

I N T E R V E N T I O N

Early Treatment   Improved Treatment

Modified Environment & Lifestyle (Population) → Family/ Community Awareness ⇌ Decreased Complications

C O N T R O L

Patient Education

NCD Complications

GUIDE TO COMPREHENSIVE RISK FACTOR REDUCTION FOR PATIENTS WITH CARDIOVASCULAR DISEASE

NO.	RISK INTERVENTION	RECOMMENDATION
1.	Smoking goal : complete cessation	Strongly encourage patient and family to stop smoking. Provide counseling, nicotine replacement, group cessation program
2.	Physical activity Minimum goal 30 mins 3-4 X/week	Encourage minimum 30-60 mins, moderate intensity exercise 3-4 times/week (walking, jogging, cycling etc) maximum benefit 5-6 hours per week. Advise medically supervised program for moderate to high risk patient
3.	Weight management	Intensive diet and appropriate physical activity intervention. Emphasize need for weight loss in patients with HBP, increased TG & glucose levels
4.	Blood pressure control Goal equal or less than 140/90 mmHg	Initiate lifestyle modification (weight control, physical activities, alcohol moderation, moderate sodium restriction) Add antihypertensive drug individualized to patient requirements if BP is more than 140/90, consistently raised for three months or if initial BP more than 160/100 mmHg
5.	Diabetes management Euglycemic control	Appropriate OHA to achieve near normal FPG. Treatment of other risks (e.g. exercise, management, BP and cholesterol management)
6.	Oestrogen	Consider oestrogen replacement therapy in all post memopausal women especially those with multiple CHD risk factors. Individualise treatment
7.	Antiplatelets agents/anticoagulants	Start ASA 75 to 300 mg if no contra indication. Manage warfarin to international normalized ratio 2 to 3.5 for post MI not able to take aspirin

NO.	RISK INTERVENTION	RECOMMENDATION			
8.	Lipid management Primary goal LDL < 100 mg/ dl Secondary goal HDL > 35 mg/dl Tg < 200 mg/dl	Diet < 30 % fat, < 7% saturated fat, < 200 mg/day cholesterol Assess fasting lipid profile. Post MI patient – 4 to 6 weeks to stabilize. Add drug therapy according to following guide :			

	LDL < 100 mg/dl No drug therapy Follow up every 5 years	LDL 100-130 mg/dl Consider therapy to diet as follows	LDL > 130 mg/dl Add drug treatment to diet as follows	HDL < 35 mg/dl Emphasize weight management, physical activity, quit smoking, increased fruit consumption

Suggested drug therapy

Tg < 200 mg/dl	Tg 200-400 mg/dl	Tg > 400 mg/dl
Statin	Statin	Combined treatment fibrate niacin + statin
Niacin	Niacin	

If LDL goal not achieved, consider combination therapy

CONCLUSION:

There is no ideal response or universal blue print to reverse the epidemic trends of CAD. However, some common principles for a successful program on primary prevention of CAD should comprise a combination of the following features:

1. Political will and leadership.

2. A strategic plan - stakeholders (rnulti-sectorial and multi-level).

    *   Situational analysis of past and present.
    *   Functional information support system.
    *   Goals, targets and implementation.
    *   Protocols and guidelines.
    *   Resources/downsizing and prioritization of services.
    *   Technical support from international agencies.
    *   Monitoring and evaluation.
    *   Re-programming.

3. Community involvement

4. Access to secondary and tertiary care

Thirty years ago, Eastern Finland was described as the world capital of cardiovascular mortality. The North Karelia Health Promotion project based on the adoption of healthy lifestyle had the following desirable outcomes:

1. Coronary mortality for males 35 - 54 dropped by 51% (720 reduced to 350 per 100,000).

2. 11% reduction in average blood cholesterol level.

3.   Male smoking prevalence decreased by half to one third.

4.   Cancer mortality declined by 45 %.

5.   Finland now ranks 6th for males and 14th for females in death from CAD.

Can Mauritius meet the challenge of reversing the trend in cardiovascular disease? The answer is positive because many risk factors are associated with a broad spectrum of non- communicable diseases which have been created by, and can be controlled by Mauritians themselves.

REFERENCES

1.   Is primary care essential? Barbara Starfleld. The Lancet. Vol 344. October 22 1994.

2.   Prevention of coronary heart disease in clinical practice. Task Force Report. Prepared by David Wood et al. European Heart Journal (1998) 19, 1434 – 1503.

3.   Guide to primary prevention of cardiovascular disease. A statement for Health Care professional. From the Task Force on Risk Reduction. PREPARED BY Scot M et al.

4.   How to implement physical activity in primary and secondary prevention. American Heart Association. Gerald F Fletcher. AHA Medical/ Scientific Statement.

5.   Mauritius Non Communicable Disease Surveys 87/92.

6.   Mauritius Non Communicable Disease Surveys 98. A preliminary report.

7.   National Health Statistic 1999.

8.   1999 World Health Organisation - International Society of hypertension. Journal of hypertension 1999, 17: 151 – 183.

9.   Epidemic of Cardiovascular Disease in post communist countries. Heart beat number 3, September 1995. Journal of International society and federation of cardiology.

10.   Asian medical Journal. Vol 14 number 9, September 1997 Counter measures for smoking cessation.

11. Control of cardiovascular disease in developing countries – research, development and institutional strengthening. Chrostopher P Housen et al. Bulletin of World Health Federation. Number 1. March 1999.

12. Physical Activity for healthy adults as a primary prevention intervention in the clinical setting. Sally S Haris et al. JAMA, June 23/30 - 1999 vol 261 number 24.

13. Physical exercise in the management of hypertension. Kikua Arakawa - Asian Medical Journal. Vol 43. number 3- 107- 111, March 2000.

Behavioural change

Procjaska and Diclemente have proposed a 'stages of change' model [387-388] which argues that everyone is not equally ready to change their behaviour at a given point in time even if they have all been invited to have screening and risk factor modification. They maintain that it is important to assess the individual's behaviour thoughts, attitudes and beliefs concerning their perceived ability to change, their behaviour over the past 6 months as well as the environmental context in which they will attempt to change, and maintain the lifestyle change.

Five stages are proposed: pre-contemplation, contemplation, preparation action and maintenance. In the pre-contemplation stage, individuals do not intend to change their high-risk behaviour in the foreseeable future i.e over the next 6 months. Individuals can be in this stage because:

(a) they are unaware of the long-term consequences of their behaviour

(b) they are discouraged about their ability to change and do not want to think about it

(c) they are defensive due to social pressure to change. Pre-contemplation is a very stable stage. As a group they evaluate the pros of their risk behaviour as greater than the cons.

Contemplation is the stage where people seriously intend to change their behaviour within the next 6 months. Despite their intentions, however, they typically stay in this stage for long periods of time, e.g. 2 years or more. They talk about change, but keep putting it off. Those who substitute thinking for acting are called chronic contemplators. Contemplators evaluate the pros and cons of their behaviour as about equal; hence there is considerable ambivalence about changing.

People in these first two stages should not enter behaviour change programme, eg. Smoking cessation, dietary or exercise programme. Most current smokers are in these stages. Instead they should be provided with information, motivation and advice that will help them move to the next stage, in which behavioural change is seriously considered.

In the preparation stage, individuals intend to take action in the near future, typically within the next month. They have a plan of action, and have usually some modest behavioural change, such as reducing the number of cigarettes smoked per day or slightly modifying diet. In this stage there are both behavioural and intentional criteria; the cons are evaluated as greater than the pros. In this stage, cues to action should be provided, such as the demonstration of association between lifestyle and symptoms, illness in other family members, social pressures and so on.

Overt behavioural change within the past six months characterises the action stage, which is highly unstable and where the greatest risk for relapse may occur. The criterion for achieving this stage is to have actually changed behaviour, eg. Stopped smoking, as opposed to reducing smoking or switching brand to lower tar, etc., During this period most of the processes of change come into play and the intrapersonal (eg. Perceived self-efficacy), interpersonal (e.g. social support) and environmental (eg. Unavoidable exposure to smoking environment) factors associated with these processes are principal determinants of whether the newly quit smoker proceeds to the next stage   (maintenance) or regresses to the earliest stage. Obviously this is the critical period where all the interventions strategies must focus on the above mentioned processes. A wide variety of instructive materials are usually available from national Heart Associations and Foundations including tailored self-instruction programmes, support groups and counselling strategies.

Maintenance is considered from six months after an individual has changed behaviour until about five years of continuous maintenance has elapsed. (N.B. : Most smoking cessation programmes consider six months to one-year continuous abstinence as "success"). For example, smokers who successfully move from action to maintenance demonstrate a gradual decrease in overall temptation and lower use of cognitive/experiential processes, and a heightened confidence and greater use of behavioural techniques such as stimulus control, counter-conditioning, helping relationship and contingency management. Mapping out templates of change associated with successful recovery could allow comparisons to be made across smoking and alcohol problems to highlight the general principles underlying successful addictive behaviour change.

In summary, behaviour change programmes achieve much higher success rates when they assess the state or readiness of the individual to commit to the change process. The 'stages of change' model reviewed here is becoming increasing employed by health care professionals who are concerned with treating and/or referring patients for appropriate assistance, as it proves it's value in providing services to those who are ready to benefit from them.

Coronary Risk Chart (not intended for clinically established CHD patients), has several functions:

- absolute risk of developing CHD over next 10 years can be read off the chart without any calculations.

Lifetime risk can be read by following the tables upwards (increased risk with increase in age).

Relative risk can readily be estimated by comprising the risk in one cell with any other in the same age group.

- One can predict the effect of changing from one risk category to another. For example, the reduction in risk associated with cessation of smoking, reduction of BP or cholesterol level can readily be shown to an individual.

- a Coronary Risk Chart developed by each country is recommended. May be constructed from results of prospective cohort studies.

RECOMMENDATIONS

1. All Area Health Centres and Community Health Centres should have an ECG apparatus

2. All Nurses should be trained to do ECG

3. Doctors should be re-trained in interpreting ECG's

4. Morphia should be made available at all Area Health Centres and Community Health Centres