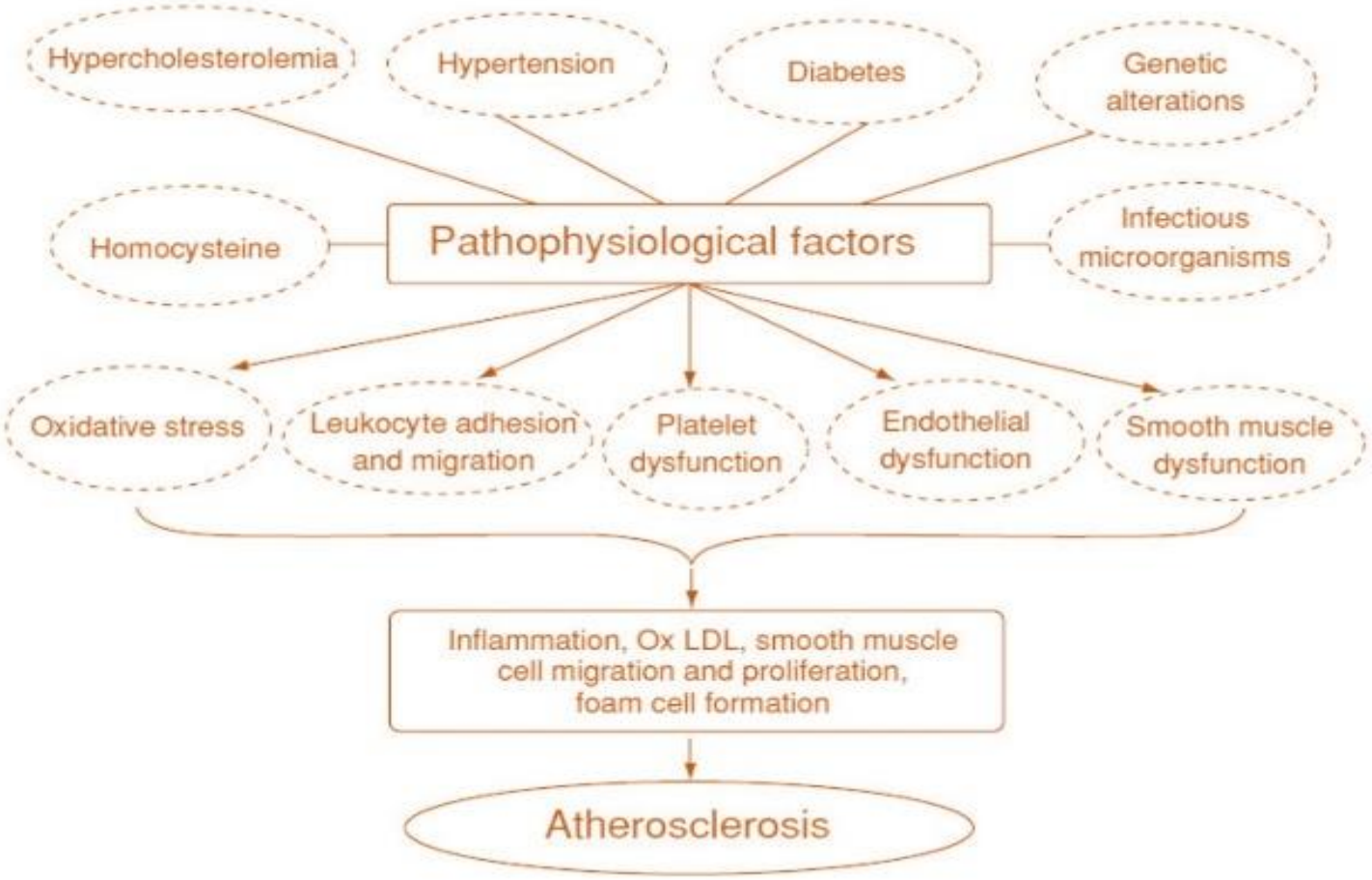

In the name of GOD

Secondary Prevention After ACS

Aims

- Reduced Late Mortality
- Reduced Re-MI
- Increased Functional Capacity
- Increased Life Expectancy


Hypercholesterolemia
Hypertension
Diabetes
Genetic alterations
Homocysteine
Pathophysiological factors
Infectious microorganisms
Oxidative stress
Leukocyte adhesion and migration
Platelet dysfunction
Endothelial dysfunction
Smooth muscle dysfunction
Inflammation, Ox LDL, smooth muscle cell migration and proliferation, foam cell formation
Atherosclerosis

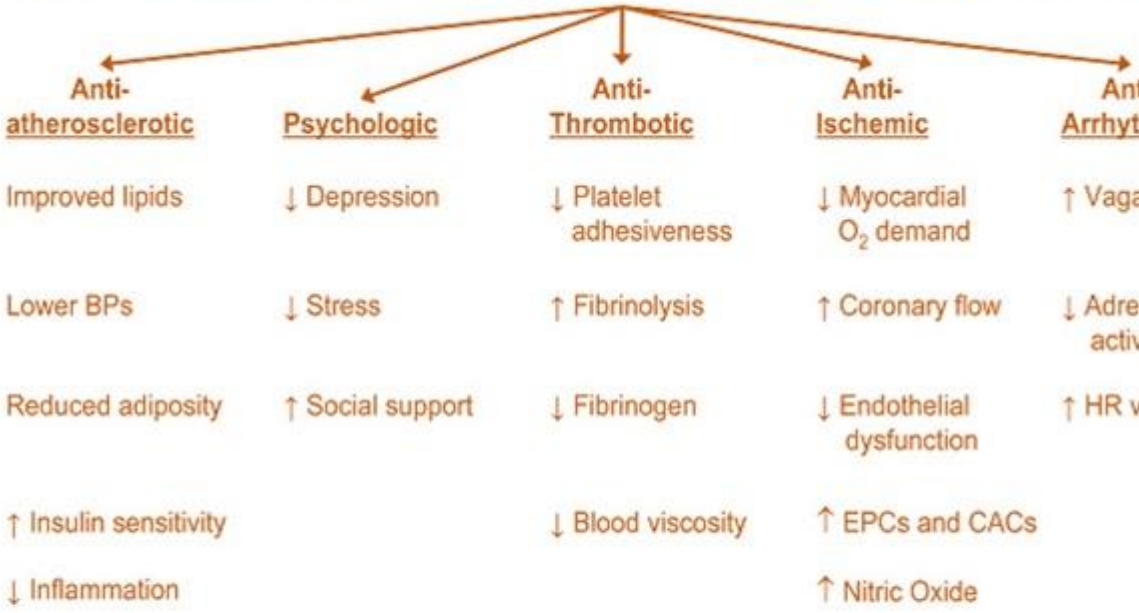
Secondary Prevention After ACS

- Cardiac Rehabilitation
- Life Style Modification
- Depression
- Modification of Lipid Profile
- Antiplatelet Agent
- ACEI drugs
- Beta –Blocking Agent
- Antocoagulants
- Nitrates
- Ca channel Blockers
- HRT
- NSAIDs

Cardiac Rehabilitation

Contemporary exercise-based cardiac rehabilitation after STEMI is aimed at increasing functional capacity, reducing disability, improving quality of life, modifying coronary risk factors, and reducing morbidity and mortality rates.[192-194] The key components of cardiac rehabilitation include patient assessment; ongoing medical surveillance; nutritional counseling; management of hypertension, lipids, and diabetes mellitus; cessation of smoking; psychosocial counseling; physical activity counseling; exercise training; and pharmacologic treatment, as appropriate.[195] When compared with usual care, cardiac rehabilitation is associated with lower total and cardiac mortality, but despite these outcomes, cardiac rehabilitation services remain vastly underused.[1]

Potential Cardioprotective Effects of Regular Physical Activity



Legend

Figure 1. Mechanisms by which moderate to vigorous exercise training may reduce the risk of nonfatal and fatal cardiovascular events. The cardioprotective vascular conditioning effect may include enhanced nitric oxide vasodilator function, improved vascular reactivity, altered vascular structure, or combinations thereof. BP indicates blood pressure; EPCs, endothelial progenitor cells; CACs, cultured/circulating angiogenic cells; and HR, heart rate.

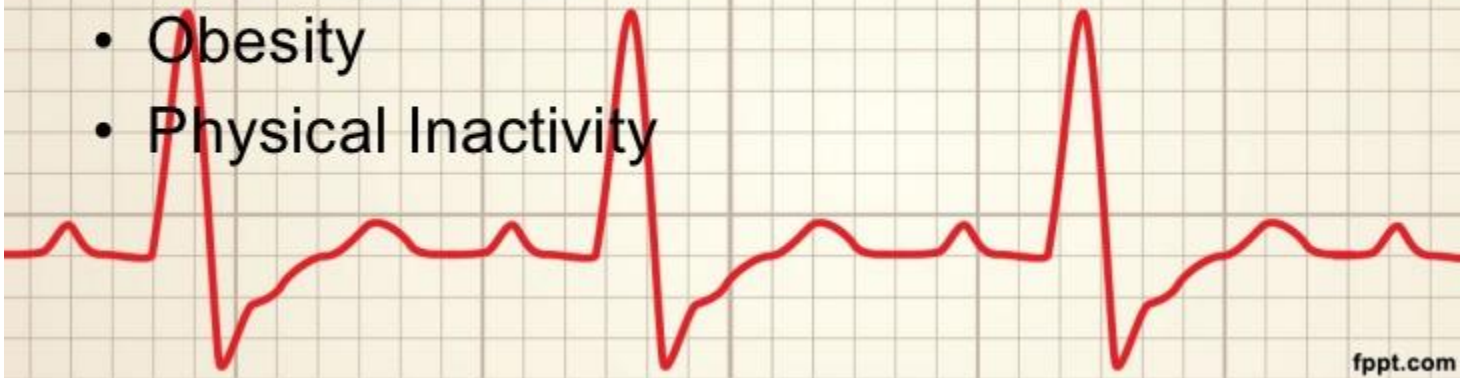
Lifestyle Modification

Efforts to improve survival and quality of life after MI that relate to lifestyle modification of known risk factors are considered in **Chapter 42**. Of these, cessation of smoking and control of hypertension are probably the most important. Use of hospital-based smoking cessation programs and referral to cardiac rehabilitation programs have led to successful smoking cessation.[196]

Risk Factors

Modifiable

- Smoking
- Diabetes Control
- Hypertension
- Hyperlipidemia
- Obesity
- Physical Inactivity



Modification of Lipid Profile (See Chapters 42 and 45)

A target low-density lipoprotein cholesterol level of less than 100 mg/dL with an optimal target of less than 70 mg/dL has been recommended in patients with clinically evident CAD.[199] High-intensity statin therapy should be initiated or continued in all patients with STEMI and no contraindications to its use.[1] Obtaining a lipid profile on admission is reasonable in all patients admitted with acute infarction. Total cholesterol levels may fall 24 to 48 hours after infarction.

Antiplatelet Agents (See also Chapter 82)

On the basis of compelling data from the Antiplatelet Trialists' Collaboration of a 22% reduction in the risk for recurrent infarction, stroke, or vascular death in high-risk vascular patients receiving prolonged antiplatelet therapy, in the absence of true aspirin allergy all patients with STEMI should receive 75 to 325 mg of aspirin daily indefinitely, with 81 mg being the preferred maintenance dose.[1,154] Additional benefits of long-term aspirin therapy that can accrue in patients with STEMI include an increased likelihood of patency of the infarct artery and smaller infarcts if MI recurs. Patients with true aspirin allergy can be treated with clopidogrel (75 mg once daily) on the basis of experience in patients with unstable angina/non–ST-segment elevation MI. In the absence of contraindications, all patients after STEMI should receive a platelet inhibitor in addition to aspirin for 12 months according to one of the following regimens: clopidogrel (75 mg/day) in patients with STEMI treated with or without PCI, prasugrel (10 mg/day) in patients treated with PCI, or ticagrelor (90 mg twice daily) in patients to be treated with PCI.[1] In patients treated with PCI, prasugrel and ticagrelor have been found to be superior to clopidogrel and are recommended as preferred in some professional guidelines.[4] However, in some practice environments, economic or formulary barriers may render access to prasugrel or ticagrelor difficult for some patients. Given the critical importance of dual antiplatelet therapy in patients who have received drug-eluting stents, access to a $P2Y_{12}$ inhibitor must be ensured. The twice-daily dosing regimen for ticagrelor should be considered for patients with concern regarding adherence to this regimen. The optimum duration of treatment with dual antiplatelet therapy remains uncertain. Nonetheless, its benefit has continued after 30 days, and for now, a $P2Y_{12}$ inhibitor along with aspirin should be administered to most patients for at least 1 year after STEMI, with aspirin treatment being maintained indefinitely.[1]

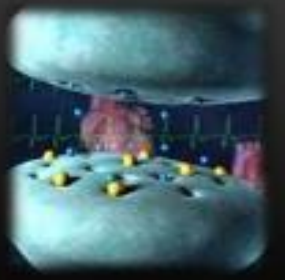
Anticoagulants

- Warfarin (INR=2-3)
- Rivaroxaban(2.5 mg –twice/daily)

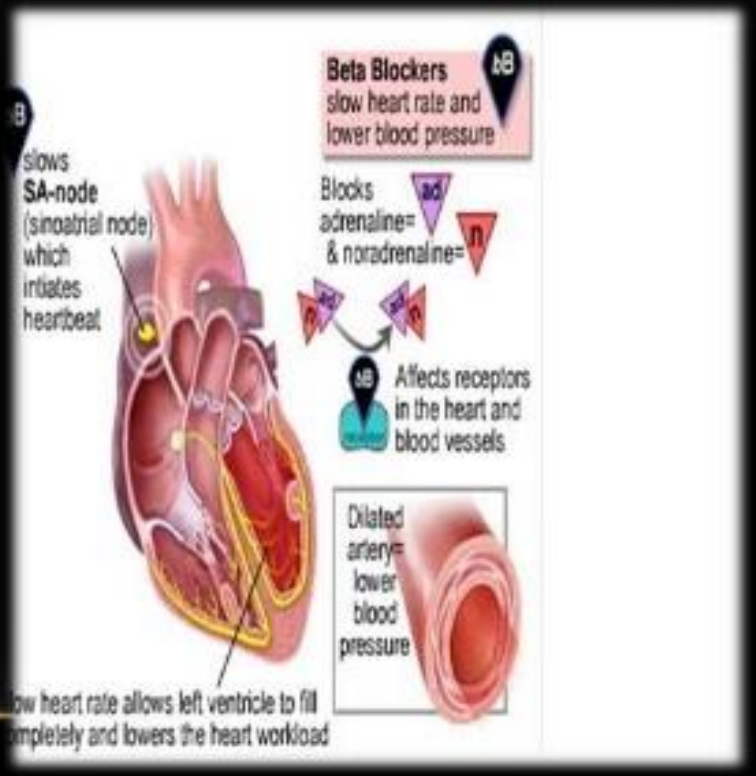
Inhibition of the Renin-Angiotensin-Aldosterone System

See Inhibition of the Renin-Angiotensin-Aldosterone System in the section Pharmacologic Therapy. To prevent late remodeling of the left ventricle and to decrease the likelihood of recurrent ischemic events, we advocate indefinite therapy with an ACE inhibitor in patients with heart failure, a moderate decrease in global ejection fraction, or a large regional wall motion abnormality, even in the presence of a normal global ejection fraction. Other candidates for long-term management with ACE inhibitors or ARBs are discussed in **Chapter 54**.

β- BLOCKERS



- β receptor antagonist
- Cardioprotective effect by
  - Antihypertensive effect – inhibiting vascular Adr receptors, ↓ Renin Angiotensin production
  - ↓ Myocardial demand & Cardiac contractility
  - ↓ HR -> ↑ diastolic coronary perfusion
  - Antiarrythymic property → Improve left ventricular structure and function



Beta-Adrenergic Blocking Agents

Meta-analyses of trials from the prethrombolytic era involving more than 24,000 patients who received beta blockers in the convalescent phase of STEMI have shown a 23% reduction in long-term mortality. In most patients who have beta blockade initiated during the convalescent phase of STEMI, the reduction in long-term mortality is probably caused by the combination of an antiarrhythmic effect (prevention of sudden death) and prevention of reinfarction.

Given the well-documented benefits of therapy with a beta blocker, it is disturbing that this form of treatment continues to be underused, especially in high-risk groups such as older adults. Patients with a relative contraindication to beta blockers (e.g., bradyarrhythmias) should undergo a monitored trial of therapy in the hospital. The dosage should be sufficient to blunt the heart rate response to stress or exercise. Much of the impact of beta blockers in preventing mortality occurs in the first weeks; consequently, treatment should commence as soon as possible. Programs that provide physician feedback to improve adherence to guidelines should be used.

Nitrates

Although these agents are suitable for the management of specific conditions after STEMI (such as recurrent angina) or as part of a treatment regimen for congestive heart failure, little evidence indicates that they reduce mortality over the long term when prescribed on a routine basis to all patients with infarction.

Calcium Channel Antagonists

At present we do not recommend the routine use of calcium antagonists for secondary prevention of infarction. A possible exception is a patient who cannot tolerate a beta blocker because of adverse effects on bronchospastic lung disease but who has well-preserved left ventricular function; such patients may be candidates for a rate-slowing calcium antagonist such as diltiazem or verapamil.

Antioxidants

(See Chapter 46)

Dietary supplementation with omega-3 polyunsaturated fatty acids has been associated with a reduction in death from coronary heart disease and nonfatal reinfarction in patients within 3 months of MI. Contemporary randomized studies, however, have shown no convincing benefit in the context of guidelines-based medical therapy.[204,205] Presently available data therefore do not support the use of antioxidant therapy for secondary prevention after STEMI.

Hormone Therapy (See also Chapters 42 and 77)

The decision to prescribe hormone therapy is often a complex one that involves the desire to suppress postmenopausal symptoms versus the risk for breast and endometrial cancer and vascular events. At present we recommend that hormone therapy with estrogen plus progestin not be started after STEMI and be discontinued in post-menopausal women after STEMI.

Nonsteroidal Anti-Inflammatory Drugs

Evidence has emerged that COX-2–selective drugs and NSAIDs that have varying COX-1/COX-2 inhibitory ratios promote a prothrombotic state and that their use is associated with an increased risk for atherothrombotic events.[206,207]