MYOCARDIAL INFARCTION

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Break it down

MYOCARDIAL
myocardium
heart muscle

INFARCTION
infarct
die
When a coronary artery becomes completely obstructed, it can no longer carry blood to the heart muscle it supplies. The muscle then becomes ischemic for an extended period. The prolonged ischemia leads to death of the cells in the affected area—a myocardial infarction.
A little visual . . .

Otherwise known as

Myocardial Infarction Video

A heart attack
## Epidemiology

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<tbody>
<tr>
<td><strong>Total Population</strong></td>
<td>8.1 million (3.7%)</td>
<td>*565,000 New</td>
<td>151,004</td>
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<tr>
<td><strong>Total Males</strong></td>
<td>5.0 million (5.1%)</td>
<td>300,000 Recurrent</td>
<td>80,079</td>
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<td><strong>Total Females</strong></td>
<td>3.0 million (3.5%)</td>
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<td>70,925</td>
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Causes

Most Myocardial Infarctions are caused by a blood clot that blocks one of the coronary arteries. The coronary arteries bring blood and oxygen to the heart. If the blood flow is blocked, the heart starves for oxygen and heart cells die.

In atherosclerosis, plaque builds up in the walls of your coronary arteries. This plaque is made up of cholesterol and other cells. A MI can occur as a result of the following:

The slow buildup of plaque may almost block one of your coronary arteries. A MI occurs when oxygen-containing blood cannot flow through this blockage.

The plaque itself develops fissures or tears. Blood platelets stick to these tears and form a blood clot (thrombus). A MI can occur if this blood clot completely blocks the passage of oxygen-rich blood to the heart.
Risk Factors

- Family history of coronary artery disease
- Increasing age (over age 65)
- Male gender
- Too much fat in your diet
- Smoking
- High Blood Pressure
- High LDL ("bad") cholesterol and low HDL ("good") cholesterol
Clinical Aspects

Signs & Symptoms
Chest pain is the most common compliant
Dizziness
Confusion
Weakness
Sweating
Loss of consciousness
Clinical Aspects

Diagnostic Testing

Laboratory indicators of tissue damage

A blood analysis can be performed to determine whether tissue damage has occurred to the myocardium.

ECG

Electrocardiographic changes in leads overlying the area of infarction, consisting of ST-segment elevation (STEMI) and T-wave inversion.

Cardiac Imaging

Provides information regarding wall motion abnormalities and blood perfusion.

STEMI is an acronym meaning "ST segment elevation myocardial infarction. In a STEMI, the coronary artery is completely blocked off by the blood clot, and as a result virtually all the heart muscle being supplied by the affected artery starts to die.
Developed jointly by the American College of Cardiology (ACC) and AHA.

**Clinical Aspects**

**Tests and Evaluations**

**Status?**

**GXT**

- **High Risk Patient**: Cardiac Catheterization must be performed before any hospital release.

- **Lower Risk Patient**: Requires a submaximal exercise evaluation 4 to 7 days post Myocardial Infarction and before any hospital release.
Clinical complications of myocardial infarction will depend upon the size and location of the infarction, as well as pre-existing myocardial damage. Complications can include:

- Arrhythmias and conduction defects, with possible "sudden death"
- Extension of infarction, or re-infarction
- Congestive heart failure
- Cardiogenic shock
- Pericarditis
- Mural thrombosis
- Myocardial wall or papillary muscle rupture
- Ventricular aneurysm formation
Clinical Aspects

Complications

**Arrhythmias / Heart block:**
Almost any cardiac arrhythmia may occur in the setting of Myocardial Infarction.

Sometimes the dead area contains some of the specialized conduction tissues of the heart (e.g. AV node, bundle of HIS, bundle branches). When these areas are severely ischemic, they are no longer capable of conducting the electrical impulses.
Clinical Aspects

Complications

**Cardiogenic shock** is based upon an inadequate circulation of blood due to primary failure of the ventricles of the heart to function effectively.

When the patient has severe CAD and have significant amounts of destroyed myocardium, the amount of viable myocardium is insufficient to supply the body’s needs. The patient’s blood pressure falls, the organ systems are inadequate and function poorly, metabolism is impaired, and the patient lapses into cardiogenic shock.

The leading cause of death in patients with myocardial infarction.
Clinical Aspects

Muscle rupture

When the infarction is 3 to 5 days old, the necrosis and inflammation are most extensive, and the myocardium is the softest, so that transmural infarctions may be complicated by rupture. A papillary muscle may rupture as well to produce sudden valvular insufficiency. Rupture through the septum results in a left-to-right shunt and right heart failure.
Treatment

Acute vs Chronic

- Focuses primarily on pain relief and reperfusion to salvage myocardium that would otherwise infarct.

- Focuses primarily on revascularization procedures, medical management, and risk factor reduction.

Primary Prevention
Secondary Prevention
Treatment

Primary Prevention

Risk Factor Modification

Regular moderate exercise or increased daily physical activity

Cardioprotective Mechanisms

*Moderate
*20 bpm
Treatment

Secondary Prevention

Long-term medical therapy

Beta-blockers, angiotensin-converting enzyme inhibitors, aspirin, anticoagulation therapy and lipid management.
Myocardial Infarctions can alter an individual’s cardiorespiratory and hemodynamic responses to both submaximal and maximal exercise.

Once one has suffered from a previous MI they often have a reduced level of cardiorespiratory fitness. The reduced oxygen transport capacity is primarily due to diminished cardiac output rather than a reduction in oxygen.

In others, cardiac output may be limited by the restriction in the rise of the heart rate.

Potential life threatening exercise induced ventricular arrhythmias occur more frequently in those who have had a previous myocardial infarction.
Vitamin supplementation has not been shown to promote added cardioprotective benefits.

Diuretics do not alter aerobic capacity.

Beta-blockers decrease submaximal and maximal heart rate and sometimes exercise capacity.

Vasodilators do not generally affect the heart rate response to exercise.

Calcium channel blockers do not generally impair functional capacity or exercise trainability and may increase exercise tolerance in individuals with angina.
Exercise Testing

Recommendations

LOW LEVEL EXERCISE TESTING

Assesses functional status

Provides diagnostic, prognostic and therapeutic guidance.

Testing can improve client confidence by providing reassurance that exercise can be done in a safe manner.

INTENSITY LEVEL SHOULD BE CONSIDERABLY LOWER THAN ANTICIPATED PEAK CAPACITY.
Before Exercise

Open communication
Activities should take place under a supervised program

• Ask about chest discomfort and faintness (if present, check with physician before proceeding).
• Measure blood pressure and heart rate (if >160/90 or <90/60 or if heart rate >110 or <60, check with physician).
• Check orthostatic blood pressure before beginning standing range-of-motion exercise or walking. If BP falls >20 or if fall associated with faintness, notify physician.
Exercise Rx

During Exercise

• Ask patient to report symptoms, particularly chest discomfort, dyspnea, or faintness. If symptoms occur, discontinue exercise until checking with physician.

• Ask for ratings of perceived exertion.

After Exercise

• Ask patient about symptoms.

• Measure heart rate, blood pressure, rating of perceived exertion. If symptoms occur, BP fall >20 or HR rises <20 over resting rate, check with physician.
Exercise Rx

Cardiovascular Training

At least 3 days a week for a minimum of 30 minutes.

Eventually increase to 6 to 7 days a week and increase duration to 40 to 60 minutes for selected patients.

Resistance Training

Weight machines, dumbbells, elastic bands and calisthenics can be adapted for most patients.

Resistance level should initially be set comfortably for 10 – 15 repetitions. (40% – 60% 1RM)

Progress training load slowly and within the patient’s capability.

Keep training sessions 15 to 45 minutes long.
A myocardial infarction is one of the main causes of hospitalization and death worldwide. Regular exercise has been shown to mediate the process and protect against MIs as well as mortality from all causes. Exercise training is an important process for those who have had a myocardial infarction.

