AMI – Acute myocardial infarction

Patient X was admitted to the emergency ward with severe chest pain for the last 2 hours. He described the pain as if a big hand was squeezing his chest, the pain radiated down his left arm and back. He was short of breath and was sweating heavily, he complained of nausea.

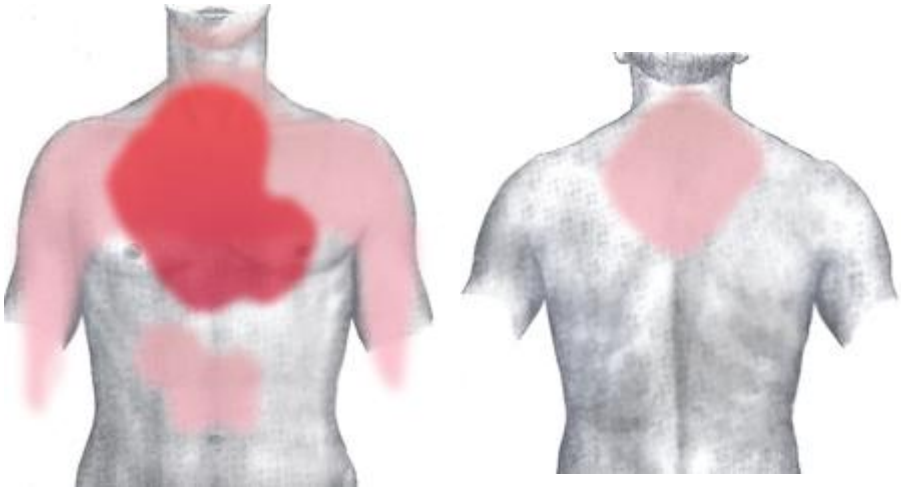
His physical examination and history:
59Male
Moderately overweight
History of hypertension and type two diabetes
His vital signs:
Blood pressure: Low
Heart rate: fast weak pulse

The doctor suspected acute myocardial infarction and orders an ECG and heart markers.

Signs and symptoms of AMI

The onset of symptoms in myocardial infarction (MI) is usually gradual and seldom instantaneous. Chest pain is the most common symptom of acute myocardial infarction and is often described as a sensation of tightness, pressure, or squeezing.

Chest pain due to ischemia (a lack of blood and hence oxygen supply) of the heart muscle is termed angina pectoris. Pain radiates most often to the left arm, but may also radiate to the lower jaw, neck, right arm, back, and epigastrium, where it may mimic heartburn



Rough diagram of pain zones in myocardial infarction; dark red: most typical area, light red: other possible areas; view of the chest

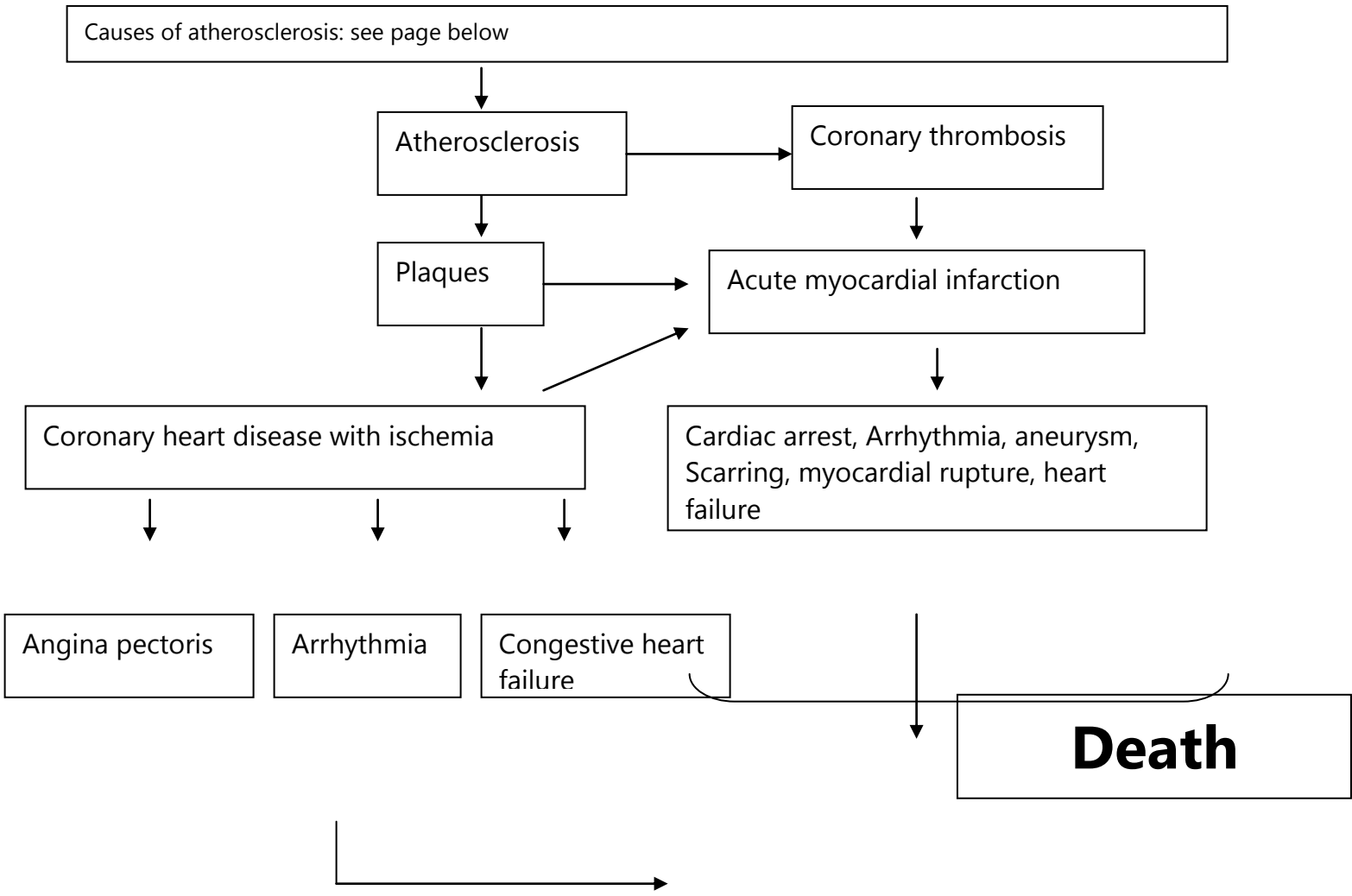
http://upload.wikimedia.org/wikipedia/commons/thumb/2/24/AMI_pain_front.png/220px-AMI_pain_front.png

Shortness of breath (Dyspnea) occurs when the damage to the heart limits the output of the left ventricle, causing left ventricular failure and consequent pulmonary edema. Other symptoms include diaphoresis (an excessive form of sweating), weakness, light-headedness, nausea, vomiting, and palpitations

Women and older patients more frequently report atypical symptoms. The most common symptoms of MI in women include dyspnea (shortness of breath), weakness, and fatigue. Fatigue, sleep disturbances, and dyspnea have been reported as frequently occurring symptoms that may manifest as long as one month before the actual clinically manifested ischemic event. In women, chest pain may be less predictive of coronary ischemia than in men.

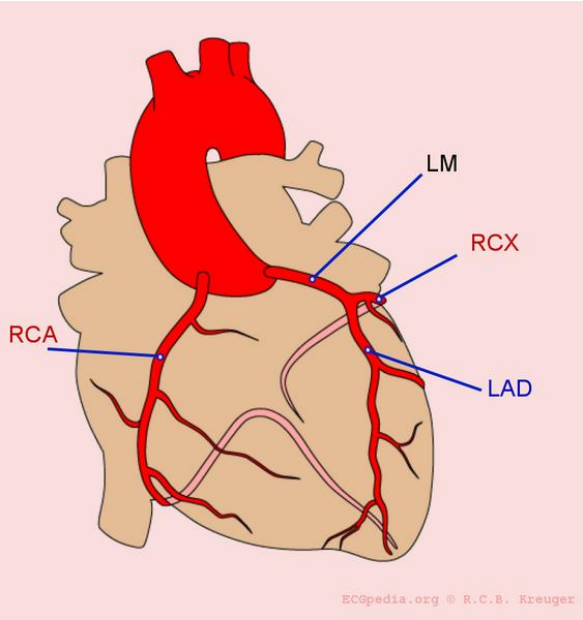
Approximately one-fourth of all myocardial infarctions are silent, without chest pain or other symptoms.

What is AMI:
Acute myocardial infarction is world wide the leading cause of heart failure and death for both men and women.



Heart failure has been defined as a failure of the cardiac output to meet the needs of the tissues.

Coronary heart disease (CHD) or atherosclerotic heart disease is a major cause of AMI and is due to the accumulation of plaques within the walls of the coronary arteries that supply the myocardium with oxygen and nutrients. Patients of AMI could have had CHD for years without any symptoms.



The coronary arteries supply the myocardium with oxygen and nutrients. The mayor coronary arteries are the left coronary artery (the **Left Main** or LM) divides itself in the **left anterior descending** artery (LAD) and the **ramus circumflexus** (RCX). The **right coronary artery** (RCA) connects to the **ramus descendens posterior** (RDP).

http://nl.ecgpedia/6.coronary_anatomy.png

The plaques consist of an unstable collection of lipids (cholesterol and fatty acids) and white blood cells (especially macrophages) in the wall of an artery.

How does arteriosclerosis form?
Important risk factors are previous cardiovascular disease, older age, tobacco smoking, high blood levels of certain lipids (triglycerides, low-density lipoprotein) and low levels of high density lipoprotein (HDL), diabetes, high blood pressure, obesity, chronic kidney disease, excessive alcohol consumption, the abuse of certain drugs (such as cocaine and methamphetamine), and chronic high stress levels.
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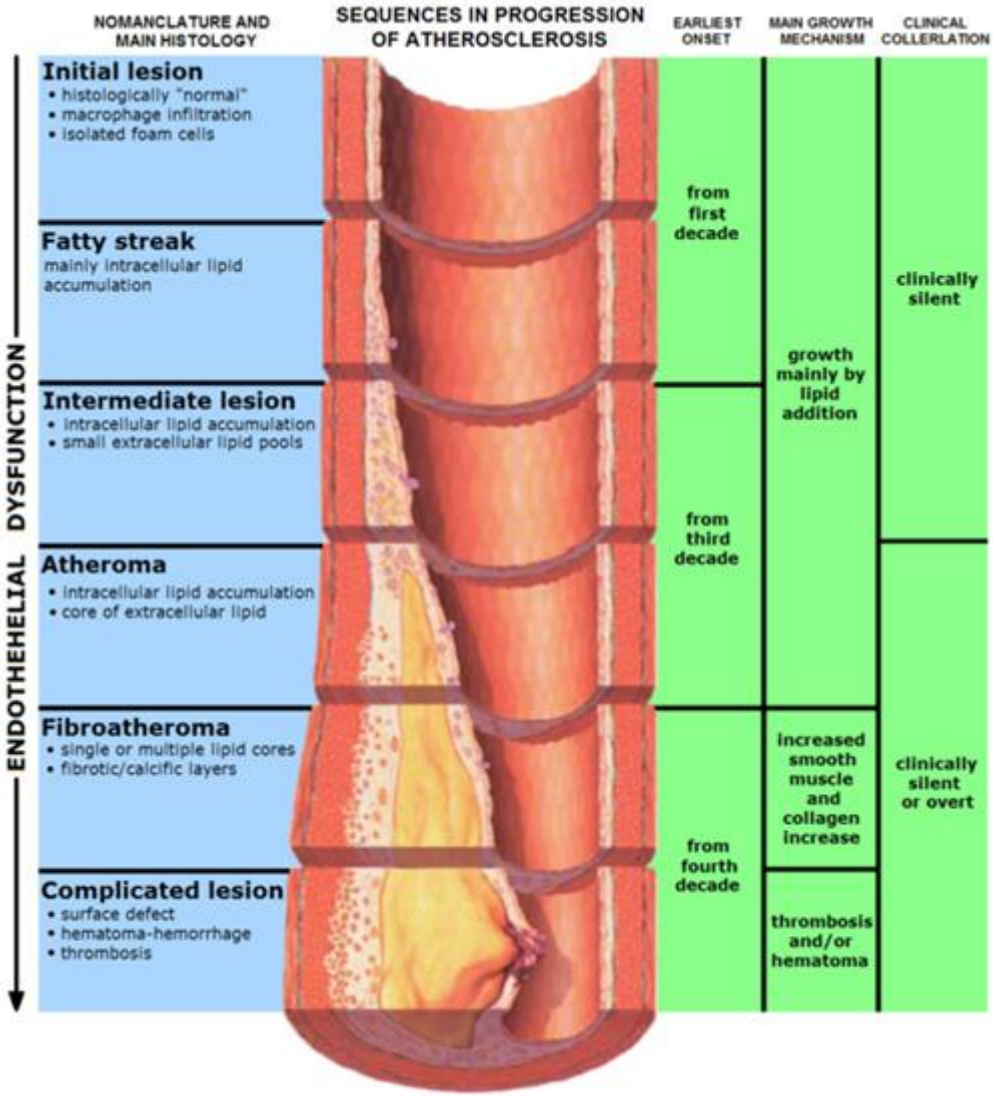
teamrich.files.wordpress.com/2008/02/atherosc...coppied 12/3/12



www.virtualsciencefair.org/2009/hsuj9j2/ather...

After decades of progression the plaques limit the blood flow to the myocardium or heart muscle causing near-complete or complete occlusion (blockage) of a coronary artery. The plaques may also rupture causing a thrombus blocking the blood flow. The resulting ischemia (restriction in blood supply) and ensuing oxygen shortage can cause angina (pain) or in severe blockage damage or death (*infarction*) of heart muscle tissue and cells (*myocardium*).

A distinction should be made between myocardial ischemia and myocardial infarction. Ischemia means that the amount of blood supplied to the tissue is inadequate to supply the needs of the tissue. When the myocardium becomes ischemic, it does not function optimally. When a large area of the myocardium becomes ischemic, there can be impairment in the relaxation and contraction of the myocardium. If the blood flow to the tissue is improved, myocardial ischemia can be reversed. Infarction means that the tissue has undergone irreversible damage due to death of a part of the myocardium.

http://upload.wikimedia.org/wikipedia/commons/thumb/9/92/Heart_ant_wall_infarction.jpg/220px-Heart_ant_wall_infarction.jpg

Angina manifests as pain in the chest that result from reduced blood supply to the heart (ischemia) due to atherosclerosis.

The typical pain of angina is in the chest but it can often radiate to the left arm, shoulder or jaw. If you have angina you will have noticed that the pain is related to exertion and is relieved by rest. An angina attack is also associated with shortness of breath and sweating. If you are a woman you may experience angina slightly differently. Women appear to have more pain in their shoulder and middle back area, and more throat, neck, and jaw pain than men.

If your angina symptoms rapidly worsen and occur at rest this may precede myocardial infarction.

Angina is classified in one of two types: 1) stable angina or 2) unstable angina.

Stable angina

Stable angina is the most common type of angina and occur during exertion (for example, walking up a flight of steps causes' chest pain) and commonly last less than five minutes. They are relieved by rest or medication, such as nitroglycerin under the tongue.

Unstable angina

Unstable angina is less common and more serious. The symptoms are more severe and less predictable. Moreover, the pains are more frequent, last longer, occur at rest, and are not relieved by nitroglycerin under the tongue (or the patient needs to use more nitroglycerin than usual). Unstable angina is often a precursor to a heart attack.

What causes AMI?

High risk groups for cardiovascular disease will also have high risk for AMI:

Genetic	
Age & Sex	Males - Older than 44 years
	Females - Older than 54 years or premature menopause without estrogen replacement therapy
Genetic	Family history of premature left ventricular heart failure.
Race	Blacks are more vulnerable to atherosclerosis than whites.
Lifestyle	
Major	Cigarette smoking
	Hypertension
	Diabetes Mellitus or impaired glucose tolerance
	HDL cholesterol <0.9 mmol/L
	Hyperlipidemia, which includes hypercholesterolemia and hypertriglyceridemia.
	Increased lipoprotein (a)
Minor	Sedentary life style
	Psychological stress
	Personality type

Diagnosis of AMI:

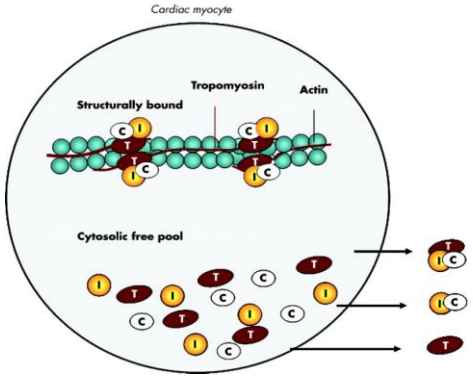
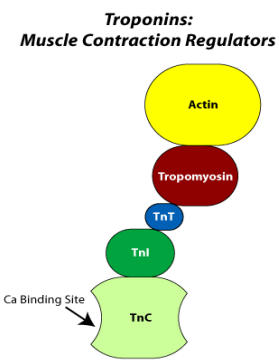
WHO criteria have been used to diagnose MI; a patient is diagnosed with myocardial infarction if two (probable) or three (definite) of the following criteria are satisfied:

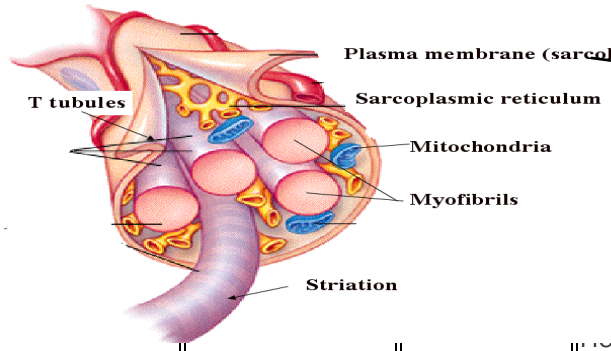
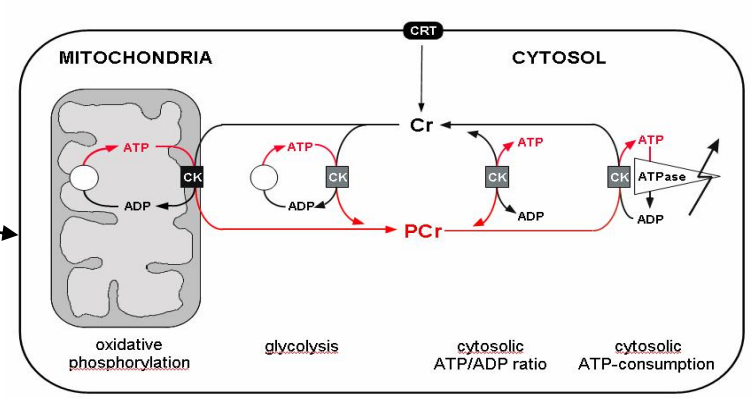
1. Clinical history of ischaemic type chest pain lasting for more than 20 minutes
2. Changes in serial ECG tracings
3. Rise and fall of serum cardiac biomarkers such as creatine kinase-MB fraction and troponin

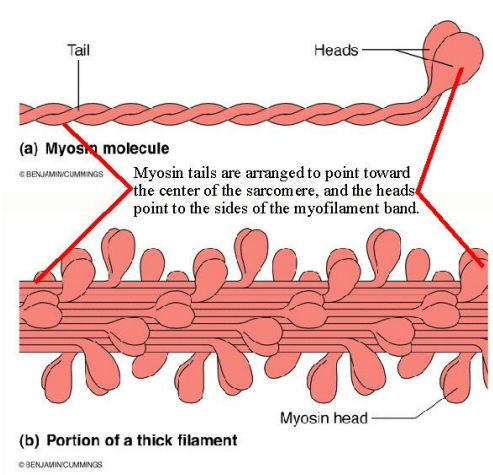
The WHO criteria were refined in 2000 to give more prominence to cardiac biomarkers. According to the new guidelines, a cardiac troponin rise accompanied by typical symptoms, pathological Q waves, ST elevation or depression, or coronary intervention is diagnostic of MI.

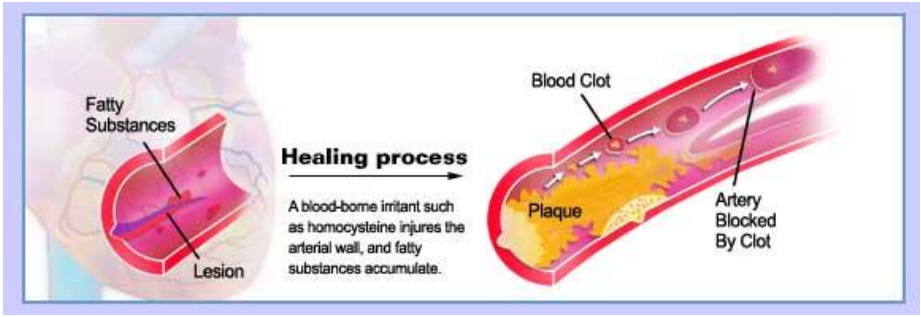
Two troponin measurements taken at least 6 hours apart is the minimum number of measurements that should be performed but more frequent measurements may be required depending on the clinical circumstances.

Laboratory diagnosis:

Marker	Normal values	Sensitivity and specificity	levels	Description
Troponin T and I	TNI = 0-0.04 ng/ml TNT = 0-0.03 ng/ml	Most sensitive + specific test for myocardial damage.	Increased within 3 – 12 hours Peak at12-24 hours return to normal in 5-14 days	  heart.bmj.com /content/92/7/ 987/F1.large.jp gn opened 2/3/11 The troponin complex consists of three subunits—troponin C, troponin I, and troponin T—and is located on the myofibrillar thin (actin) filament of striated (skeletal and cardiac) muscle (fig 1). The cardiac isoforms troponin T and I are only expressed in cardiac muscle Troponins are contractile proteins that are released during MI from myocytes. Its subsequent release is prolonged with degradation of actin and myosin filaments. Its is increased in acute infarction, heart failure, haemodynamic compromise e.g. shock or sepsis, pulmonary embolism, severe hypertension and myocarditis
Creatine Kinase	M	It is relatively	Increased	CK resides in the cytosol (intracellular fluid) and facilitates high energy phosphates into

(CK-MB) test (mass) Creatine Kinase (CK)	0 – 7.6ng/ml F 0 – 4.6 ng/ml M 39 – 308 U/l F 26 – 192 U/ll	specific when skeletal muscle damage is not present.	within 3 – 12 hours Peak with in12 hours return to normal in 48 – 72 hours	and out of mitochondria. It is distributed in a large number of tissues also in the skeletal muscle. The 3 CK isoenzymes are as follows: • CK with muscle subunits (CK-MM), which is found mainly in skeletal muscle • CK with brain subunits (CK-BB), which is found predominantly in the brain • CK-MB, which is found mainly in the heart It is increased in Myocardial infarction, myocarditis, muscular dystrophies, polymyositis, seizures, muscle trauma, exercise, Intramuscular injection, post-operative. Since it has a short duration, it cannot be used for late diagnosis of acute MI but can be used to suggest infarct extension if levels rise again.   How energy flows in the cell: Energy-rich ATP is produced in the mitochondria (left). This ATP transfers its phosphate group to creatine (Cr), which acts as a fast carrier to transport energy in the form of phospho-creatine (PCr) to the site where it is consumed (right). Creatine is channelled into the muscle cells by a special creatine transporter (CRT).www.ucl.ac.uk/~sjjgsca/MuscleCell.gif opened 2/3/11 archiv.ethlife.ethz.ch/.../herzenerschema-l.jpg
Myoglobin (Mb)	M 28 – 72 µg/l	low specificity for	2 hours	Myoglobin is a low-molecular –weight heme protein and the primary oxygen-carrying

	F 25 – 58 µg/l  	myocardial infarction		pigment of muscle tissue like that of the skeletal and heart. Because of its low molecular weight and its cytoplasmic location it appear early after heart and skeletal muscle injury. But It lacks specificity but has the advantage of responding very rapidly, rising and falling earlier than CK-MB or troponin. Myofibrils are contractile units within the muscle cells which consist of a regular array of protein **myofilaments**. Each myofilament runs longitudinally with respect to the muscle fiber. There are two types: the **thick bands** and the **thin bands**. Thick bands are made of multiple molecules of a protein called **myosin**. http://www.unm.edu/~jimmy/myosin.jp
Pro-brain natriuretic peptide (NT Pro BNP)	<125 pg/ml Values less then 300 pg/ml excludes Acute heart failure but levels > 150 pg/ml in females and >100 pg/ml may indicate symptomatic heart failure	Not specific for AMI but is used in treatment and monitoring of patients. Values > 450 pg/ml indicate Acute heart failure in patients under 50 years >900 pg/ml in patients 50-75 years		Natriuretic peptides(NP's) are secreted to regulate fluid volume, blood pressure and electrolyte balance. BNP is released my the myocardium in response to arterial/ ventricular stretch from volume overload, wall tension and myocardial stretching. Blood BNP's increase in Acute congestive heart failure, myocardial infarction, left ventricular hypertrophy, chronic renal failure, chronic obstructive pulmonary disease, atrial fibrillation, severe pneumonia and cushing's syndrome. BNP levels may be falsely decreased by hypothyroidism, treatment with diuretics, vasodilators or ACE inhibitors.
Homocystein	15 – 64 years	Increased levels		Homocysteine is an amino acid complex that is created in the liver, and is a

	<15 µmol/l >64 years <20 µmol/l	indicate risk of cardiac disease and artherioschlerosis		intermediary "by product" of normal amino acids methionine and cysteine. Homocysteine causes damage to the lining of arteries because It is an irritant and increased levels leads to injuries of the arterial vessels causing inflammation and plaque formation which may lead to blockages of blood flow to the heart. Increased values are caused by genetic factors, deficiency of Vit B 12 and folate, renal failure – harmful substance not effectively removed, diabetes and some medications. Other conditions associated with increased levels are Alzheimer's and Osteoporosis. http://www.mediniche.com/images/homocysteine2.jpg 

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