

Myocardial Infarction (Heart Attack) — STEMI vs. NSTEMI

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Myocardial infarction is one of the most common causes of death in industrialized countries and requires immediate intervention, according to the principle “Time is Muscle”. 40 % of all patients die before their first post-infarction day, and 50 % die in the first four weeks. Early detection and swift action are of crucial significance, especially in the case of ST-segment elevation myocardial infarction.



Definition

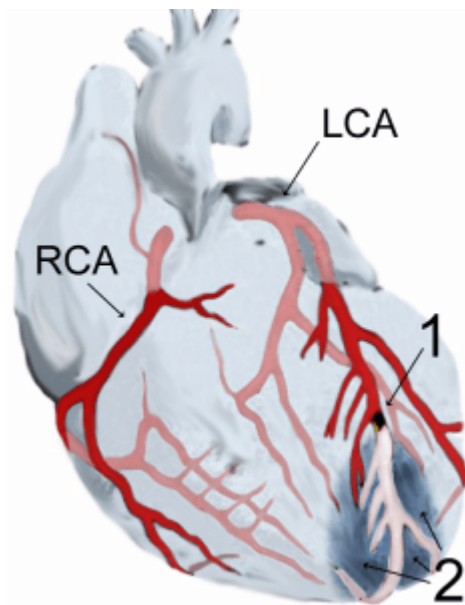


Image: Myocardial Infarction. By: J. Heuser.
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The term myocardial infarction refers to ischemia of the myocardial tissue due to a complete obstruction or drastic constriction of the **coronary artery**. This is usually accompanied by an increase in cardiac enzymes, typical ECG changes, and pain symptoms, or a thrombus or wall motion abnormality that is detected by means of medical imaging.

Epidemiology

Spread of myocardial infarction

Myocardial infarction is one of the most common causes of death in industrialized countries. Several million people suffer from myocardial infarction each year.

Regional differences can be observed, with Germany recording a significantly higher number of infarctions per year (300 infarctions in 100,000 inhabitants) compared to Mediterranean countries (100–200 infarctions in 100,000 inhabitants). Sweden, Belgium, and Japan have an even lower infarction rate of fewer than 100 infarctions in 100,000 inhabitants.

Etiology

Causes of myocardial infarction

The main cause of myocardial infarction is the development of atherosclerosis in the **coronary arteries**; hence, the name **coronary heart disease**. Coronary embolism is a rarer cause.

Classification

Distinction based on the type of myocardial infarction

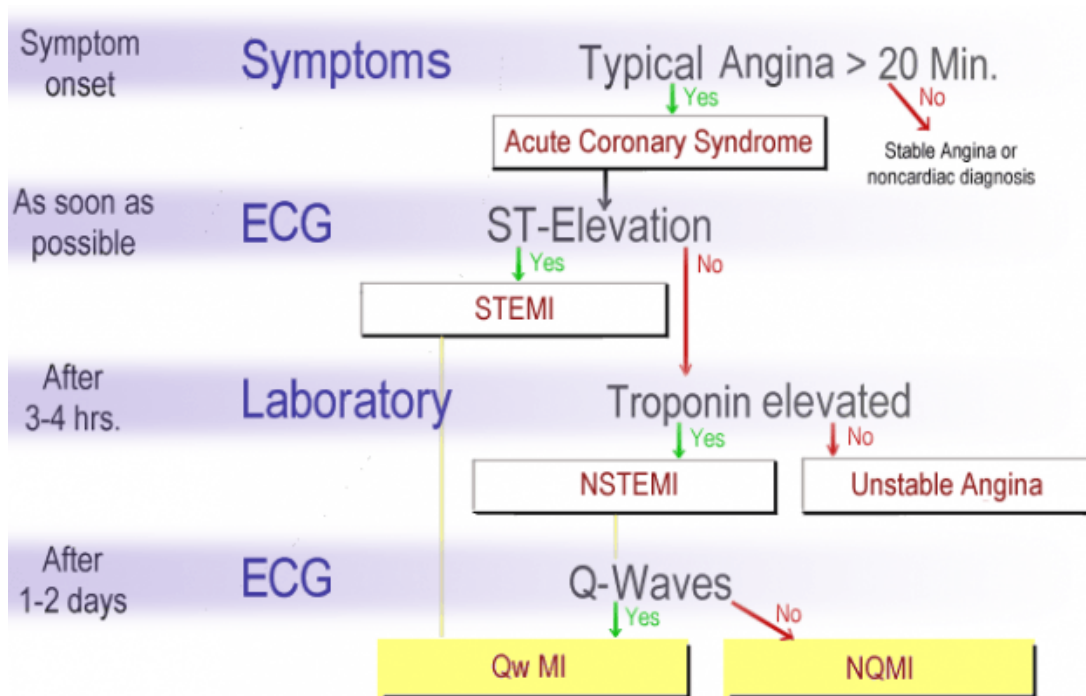
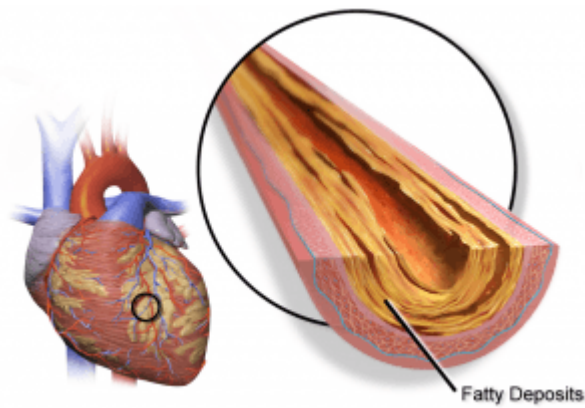


Image: Abbreviations used for ACS (Acute coronary syndrome). By: J. Heuser. License: [CC BY-SA 3.0](https://creativecommons.org/licenses/by-sa/3.0/)

The general distinction is based on ECG changes: STEMI (ST-segment-elevation myocardial infarction) is distinguished from NSTEMI (Non-ST-segment-elevation myocardial infarction). Furthermore, the European Society of Cardiology differentiates between the following types:

- **Type 1:** The infarction is spontaneous and can be attributed to a primary coronary incident.
- **Type 2:** The infarction is related to ischemia, the cause of the ischemia being, for example, coronary embolism or anemia.
- **Type 3:** The symptoms preceding cardiac death or an autopsy point to myocardial ischemia.
- **Type 4a:** The myocardial infarction occurs as part of a PCI.
- **Type 4b:** The myocardial infarction is caused by stent thrombosis.
- **Type 5:** The myocardial infarction develops in connection with a CABG.

Pathophysiology



[Image](#): Coronary Artery Disease. By: Bruce Blaus. License: [CC BY 3.0](#)

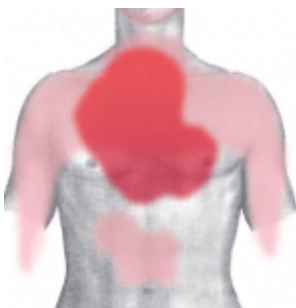
As a result of atherosclerosis, plaques start to form in the **coronary arteries**, which are at first still stable. Over time, an unstable (vulnerable) plaque forms. The rupturing of this plaque leads to a thrombotic obstruction of one of the **coronary arteries**, **hypoxia** of the tissue fed by the **coronary artery**, and ischemia, followed by the formation of necroses.

The rupture can be triggered by e.g. physical or psychological stress, drastic blood pressure fluctuations or in the course of circadian rhythms that are accompanied by changes in coagulation activity (stronger coagulation early in the morning).

Symptoms

Clinical signs of myocardial infarction

The typical symptoms include severe retrosternal pain that typically radiates to the left arm (Note: right arm pain is also possible), neck, and upper jaw and does not improve after nitroglycerin administration. These symptoms are observed in only about 40% of patients.



[Image](#): Pain in acute myocardial infarction (front). By: J. Heuser. License: [CC BY-SA 3.0](#)



[Image](#): Pain in acute myocardial infarction (rear). By: J. Heuser. License: [CC BY-SA 3.0](#)

Painless (silent) infarctions especially affect diabetic patients due to neuropathy or old age. These painless infarctions are present in about 20% of all cases.

However, infarction pain can also be atypical, such as upper abdominal pain, feeling of faintness combined with fear and sweating, dyspnea, nausea, or vomiting. These atypical

pain symptoms are frequently found in women, diabetics, or older people.

Diagnostics

History and physical examination for myocardial infarction

In the course of the disease, the typical retrosternal pain can be evaluated. Physical examination can reveal e.g. signs of cardiac decompensation or changes in blood pressure. Cardiac risk factors can be ascertained in the process as well or be unveiled during the physical examination.

Noticeable noises, especially in connection with complications, may be detected in the course of the auscultation. Pericardial rub can be heard, which suggests underlying pericarditis. Systolic murmur occurs during **ventricular septal perforation** or **mitral valve insufficiency**. **Pulmonary vascular** congestion or **lung edema** can cause bilateral basal rales.

Laboratory diagnostics for myocardial infarction

The classification of the heart enzymes provides information on the presence and extent of myocardial damage. The determination of Troponin I or T (depending on the laboratory) is considered the gold standard. Troponin, as a cardiac marker, is specific to the myocardial tissue.

Troponin increases shortly after the infarction. About 70% of patients show an increase 3 hours after the incident, and that number rises to 90% 6 hours after the incident. This timeline stresses the necessity of measuring cardiac enzymes again at a later time even if the initial result was negative.

Reinfarction after myocardial infarction cannot be diagnosed by means of troponin-recording since the troponin concentration only returns to normal after about 1-2 weeks. In contrast, myoglobin is unspecific and increases also when damage to skeletal muscles occurs but goes back to normal after 1 day at the latest. CK-MB is specific to the infarction, but it only starts to rise 4 hours after the occurrence of ischemia. A CK-MB concentration of between 6-20% of the total CK indicates myocardial damage.

LDH can serve as a long-term marker of myocardial damage. It starts to increase only after 6-12 hours, but reaches its maximum after 2-4 days and stays at an increased level up to 2 weeks following the infarction. GOT, which is actually a marker for liver cells, also exists in the heart muscle and can also be used for diagnostics. In that case, GPT, which is specific to the liver, has to be determined as well in order to distinguish heart disease from liver cell damage.

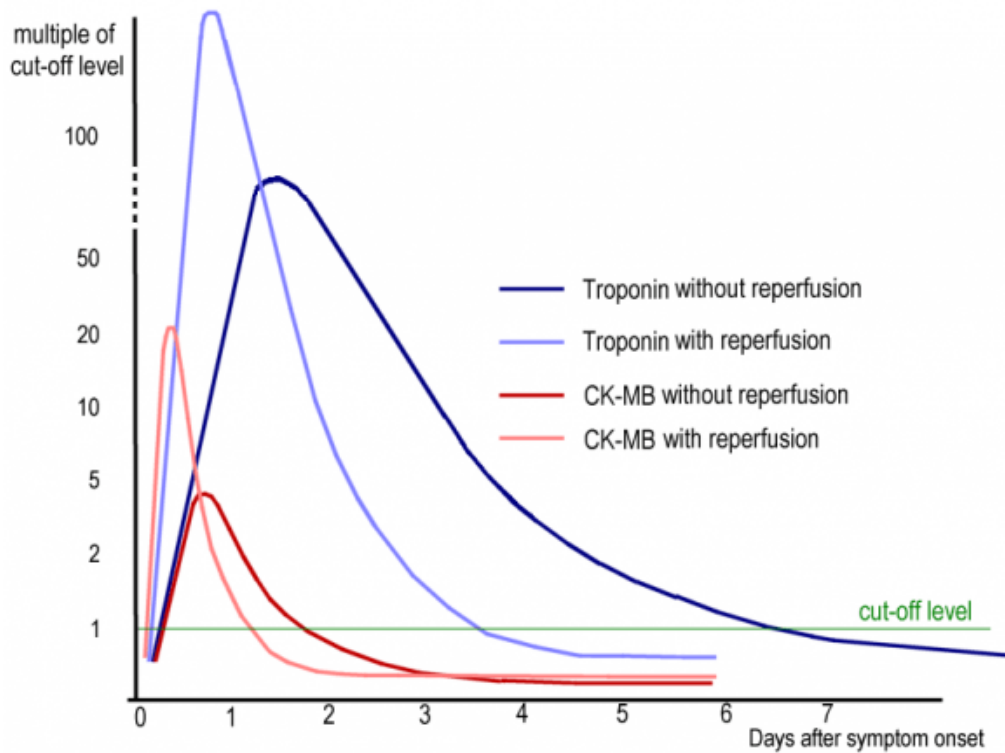


Image: Typical changes in CK-MB and cardiac troponin in acute myocardial infarction. By: J. Heuser. License: [CC BY-SA 3.0](https://creativecommons.org/licenses/by-sa/3.0/)

The following table provides an overview of the various parameters:

Parameter	Begin of increase	Maximum	Normal levels
Troponin	3-8 hours	12 hours	1-2 weeks
CK-MB	3-12 hours	24 hours	2-3 days
Myoglobin	1-4 hours	4 hours	1 day
LDH	6-12 hours	2-4 days	1-2 weeks
GOT	4 hours	48 hours	3-6 days

ECG diagnostics for myocardial infarction

In the case of NSTEMI, the ST-segment may be temporarily depressed and may be accompanied by a negative pre-terminal T-wave. The ECG changes in NSTEMI are often atypical, which means that an infarction can only be ruled out through repeated heart enzyme counts.

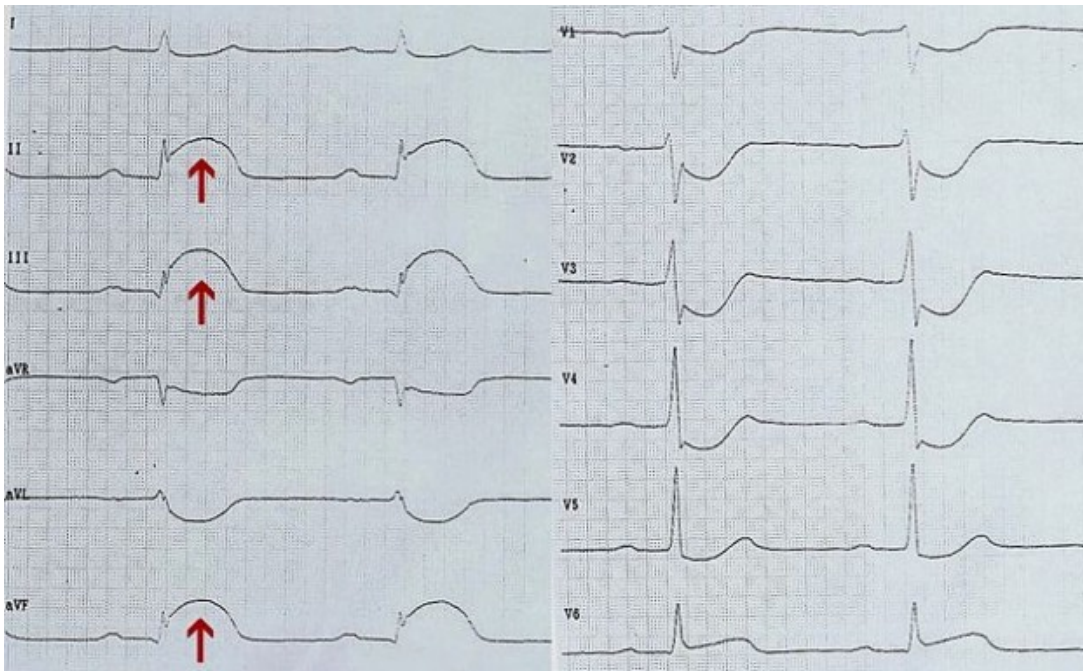


Image: Diagram showing a representation of myocardial infarction on an ECG. By: J. Heuser. License: [CC BY-SA 3.0](https://creativecommons.org/licenses/by-sa/3.0/)

In the case of STEMI, the ECG shows typical changes, such as the monophasic ST-segment elevation, which transitions directly into the T-wave, with pathologically enlarged Q-spikes. Other ECG changes are typical depending on the stage of the infarction:

- **Initial stage:** Up to 6 hours after the infarction, typically characterized by pointed elevation of the T-wave, which is called anoxic T-wave
- **Acute infarction:** Few hours to days after the infarction, characterized by the typical monophasic ST-elevation
- **Intermediate stage:** The ST-elevation and R-spike decrease and so-called poor R-wave progression occurs. Moreover, the enlargement of the Q-spike is noted, which, as a Pardee-Q, signals the end of the myocardium, and pointed negativity of the T-wave develops.
- **Final stage:** More than 6 months after the infarction, the changes in the QRS complex remain, while the ST-segment and the T-wave return to normal.

Localization of infarction

The ECG lead(s) in which changes are detected provide information on which coronary artery is likely obstructed and where the infarction is located. In exams, students are often asked to explain how anterior myocardial infarction leads to ECG changes in leads V1-V6, aVL, and I. This frequently affects the proximal part of the **anterior interventricular artery**. Changes in leads V1 and V2, however, point to a **posterior myocardial infarction**, where the **posterior descending artery** is being affected frequently.

Medical imaging for myocardial infarction

Echocardiography as another diagnostic tool helps to detect wall motion abnormalities, determine the remaining function, and exclude complications, such as **cardiac valvular defects**. Cardiac catheterization is considered the gold standard and should be performed as quickly as possible in the case of STEMI. MRI cardioangiography serves as another means of detecting wall motion abnormalities and determining the size of

infarction scars.

Pathology

The pathology of myocardial infarction can be traced to the insufficient perfusion of cardiac tissue. At a perfusion rate of less than 25% of the normal blood flow, the **tissue** is damaged irreversibly, and after 6–12 hours, damages in the shape of yellowish focuses become visible with light microscopy.

Complete myocytolysis takes place, the cell nuclei starts to pale, and the cross striations get blurred. Granulation tissue with a hemorrhagic edge and numerous peripheral leukocytes develop a few days later. After only 2 weeks, **whitish scar tissue** develops.

Differential Diagnoses

Symptoms similar to those of myocardial infarction

The typical pain symptoms can also point to unstable **angina pectoris**, with the exception that there is no increase in troponin. Particularly, when pain spreads to the **upper abdomen**, the symptoms can be mistaken for an acute abdomen. Another differential diagnosis to be considered is **Takotsubo cardiomyopathy**, which is often caused by emotional stress.

ECG changes can also be caused by other diseases. ST-elevation can also occur in **pericarditis** or **left ventricular hypertrophy**, while low Q-spikes can be noted in lung embolisms, Wolff-Parkinson-White syndrome, or **hypertrophic cardiomyopathies**.

Furthermore, all other causes of thoracic pain have to be distinguished from myocardial infarction, including lung embolism, aortic dissection, tension pneumothorax, and spontaneous rupture of the esophagus (**Boerhaave-syndrome**). Myocarditis and pericarditis, as well as musculoskeletal thoracic pain, are also differentials of myocardial infarction and are possible exam answers on the subject.

Therapy for Myocardial Infarction

Oxygen (4–8 L/min per nasal O₂ tube) can be administered from the initial treatment until the diagnosis is confirmed. Additionally, it is recommended to elevate the upper body for relief (30°) in the case of dyspnea or heart insufficiency. Administering **nitroglycerin** (1 capsule or 2 puffs = 0.8 mg, 1–5 mg intravenously through a perfusor (Note: nitroglycerin is contraindicated if systolic blood pressure is 90 mmHg). The simultaneous administration of PDE-5 inhibitors and **morphine** (3–5 mg intravenously every 5–10 minutes) can be administered when severe pain occurs.

Furthermore, all patients receive **aspirin** (initial dose 150–300 mg) as well as **heparin** (UFH 70 IE/kg, KG or LMWH) and a **P2Y12 inhibitor** such as **clopidogrel** (initial dose 300 mg). Intramuscular injections should be avoided since they lead to a non-specific increase of CK-levels. The administration of beta-blockers and ACE-inhibitors and AT1 receptor antagonists is also indicated unless there is a contraindication.

Cardiac catheterization has to be used for **diagnosis** of NSTEMI, and can be performed between 2 and 72 hours after the incident, depending on the risk factors of the respective patient. In the case of STEMI, the obstructed coronary vessel has to be revascularized as quickly as possible by means of **PCI**. Primary PCI is the therapy of choice and should be

performed within 90 minutes.

Alternatively, a conservative therapy of **fibrinolytics** and **thrombolytics** for reperfusion can be administered. The substances used are **streptokinase, alteplase, reteplase, and tenecteplase**, all of which should accompany heparin therapy. Alternatively, **fondaparinux** or **enoxaparin** may be administered. It is crucial to ensure that no contraindications such as hemorrhages exist and that the STEMI is recent, in which case an acute PTCA is not possible.

Note: Fibrinolytic treatment should not be administered in NSTEMI.

Subsequently, more therapy is necessary to avoid **coronary rethrombosis**. Dual platelet inhibition is typically employed in this process, and in the case of aspirin, it is combined with **ticagrelor, prasugrel, or clopidogrel**. Platelet aggregation inhibition using ASA has to be continued throughout life, whereas, inhibition by means of P2Y12 inhibitors is only used for 12 months following the infarction.

Moreover, all patients should receive cholesterol-lowering agents, such as statins. The intake of beta-blockers decreases the reinfarction risk as well as the risk of **arrhythmia-related** and sudden cardiac death.

Complications of Myocardial Infarction

The complications associated with myocardial infarction are extensive, and their severity depends on the degree of myocardial damage.

One can distinguish between early and late complications, the transition between those phases lying at about 48 hours following the infarction. Early complications include **cardiac arrhythmias** that develop when an area of the **myocardium** is under-supplied. Ventricular fibrillation, which accounts for 80% of deaths due to myocardial infarction, frequently occurs within the first few four hours following the infarction. Premature ventricular contractions or atrial fibrillation can also occur.

Depending on what area is damaged, right or left heart failure and cardiogenic shock may occur, or damage in the **papillary muscle** may lead to an insufficiency of the **atrioventricular valves**. After ventricular fibrillation, congestive heart failure due to cardiac insufficiency is the second most frequent cause of death in patients of myocardial infarction.

Long-term complications include **myocardial aneurysms** that can rupture. The myocardium may also rupture. Arterial embolisms, arrhythmias, and insufficiencies also count as long-term complications. As a result of the infarction, pericarditis can occur up to 6 weeks later.

Prevention of Myocardial Infarction

Since 95% of the cases of acute coronary syndrome are manifestations of coronary heart disease, the disease can be prevented. Thus, risk factors, especially the main risk factors, which are high cholesterol, arterial hypertension, diabetes mellitus, and nicotine abuse must first be addressed. Patients should be advised to live a healthy lifestyle by paying attention to nutrition and exercise. However, these approaches can be supported with drugs.

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Figure 14.30. Mortality rate for Acute Myocardial Infarction (heart attack), 2004 and 2014 (online only). (n.d.). doi:10.1787/888933534727

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