Differential diagnosis of chest pain

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# Table of contents

1. Objective ........................................................................................................................................ 4
2. Terminology ...................................................................................................................................... 4
3. Necessary devices .............................................................................................................................. 4
4. Personal conditions ............................................................................................................................ 5
5. Methods ........................................................................................................................................... 5
6. Dangers, consequences ....................................................................................................................... 5
7. Drugs ............................................................................................................................................... 5
8. Documentation ................................................................................................................................... 5
9. Ethical and legal aspects .................................................................................................................... 6
10. Tasks of the instructor and the students ......................................................................................... 6
   10.1. Tasks of the instructor ............................................................................................................. 6
   10.2. Tasks of the students ............................................................................................................. 6
11. General features and epidemiology of chest pain ........................................................................ 6
12. Causes and pathophysiology of chest pain ..................................................................................... 7
   12.1. Musculoskeletal pain ............................................................................................................. 7
       12.1.1. Rib cartilage pain ........................................................................................................... 8
       12.1.2. Pain associated to rheumatic diseases ......................................................................... 8
       12.1.3. Chest pain associated to non-rheumatic diseases ....................................................... 9
13. Cardiovascular diseases and chest pain ........................................................................................ 9
   13.1. Ischemic heart disease-angina pectoris ................................................................................. 9
   13.2. Stable angina pectoris .......................................................................................................... 9
   13.3. Unstable angina pectoris ...................................................................................................... 12
   13.4. Substrates of chest pain not related to coronary artery disease ........................................... 13
   13.5. Sympathetic activation and chest pain ................................................................................. 20
14. Gastroenterological diseases and chest pain .................................................................................. 21
   14.1. Diseases of the esophagus ................................................................................................... 21
   14.2. Other gastroenterological origins of chest pain ................................................................... 24
15. Pulmonary diseases and chest pain .............................................................................................. 25
15.1. Chest pain caused by pulmonary vessels.........................................................25
16. Psychogenic substrates of chest pain.................................................................28
17. Diagnosis of chest pain.......................................................................................29
  17.1. Radiological and laboratory diagnosis .........................................................29
18. References ...........................................................................................................31
19. Self-controlling questions....................................................................................32
20. Case reports .........................................................................................................33
21. Tests.....................................................................................................................38
  21.2. Multiple choice questions...............................................................................39
  21.3. Single choice questions ..................................................................................40
  21.4. Relational analysis.........................................................................................41
1. Objective

The aim of this chapter is to show the possible pathophysiological backgrounds and mechanisms responsible for the occurrence of chest pain. Furthermore, this chapter aims to describe the clinical symptoms, diagnostic, and therapeutic possibilities that can help to elucidate the exact underlying mechanisms that may play a role in the genesis of this clinical symptom.

2. Terminology

- **Angina pectoris** chest pain due to myocardial ischemia
- **Arrhythmia** Rhythm disturbance of the heart
- **Coronary X syndrome** angina pectoris due to a microvascular disease of the myocardium
- **Dissection** pathological splitting or longitudinal rupture of the arterial wall
- **Musculoskeletal pain** Pain due to muscular or skeletal origin
- **Paresthesia** a skin sensation, such as burning, pickling
- **Pericarditis** inflammation of the pericardium
- **Pneumothorax** a collection of air in the chest or pleural space that causes part or all of a lung to collapse
- **Reflux** regurgitation
- **Syndrome** a group of symptoms

3. Necessary devices

- arterial cannula
- infusion set
- physiological saline solution (500 ml)
- pulsoxymeter
- blood-pressure meter
- stethoscope
- ECG monitor
• skin disinfectant
• sterile cotton wool

4. **Personal conditions**

A minimum of two students and one instructor must take part in the practical.

5. **Methods**

Students will join into groups of five. Each group has to solve one clinical case. The setting will imitate the Intensive Care Unit where a patient with chest pain arrives. The students begin to examine the patient and according to the further development of the clinical case and questions given by the instructor, they together work out an examination and treatment plan.

6. **Dangers, consequences**

Whenever intravenous injections are given, there is a risk of damage to the skin and vessel due to improper technique. In order to decrease this consequence, the activity will be done under strict observation of the leader.

7. **Drugs**

• Nitromint sublingual tablet
• Tensiomin 25 mg tablet
• Controloc 40 mg tablet and injection
• Algopyrin tablet and injection
• Aspirin tablet

8. **Documentation**
The attendance in the practical has to be proven by the instructor’s signature.

9. Ethical and legal aspects

It is obligatory to keep all ethical and legal rules of the medical profession during the practicals, which is supervised by the instructor.

10. Tasks of the instructor and the students

10.1. Tasks of the instructor

The instructor and the students discuss the important topics like the characteristic clinical features of chest pain. It is important for the students to be familiar with the theoretical knowledge so that they can improve their manual skills during the practical.

10.2. Tasks of the students

It is obligatory for the students to arrive at the practical on time. It is advisable for the students to be familiar with the theoretical knowledge so that they can do the manual tasks during the practicals. They should be able to perform and know the diagnosis and differential diagnosis of chest pain.

11. General features and epidemiology of chest pain

Chest pain is one of the most frequent clinical symptoms occurring in the emergency units. In most of the cases it has a benign background. Nevertheless, sometimes it can be the first sign of a life-threatening disease. 36-49 % of emergency patients have benign, musculoskeletal chest pain. On the contrary in 15-18 % cardiac, while in 8-19 % gastroenterological underlying disease can be proved. Furthermore 8-19 % of patients has pulmonary and 8-11 % has psychiatric background.

The data clearly show the importance of getting the diagnostic and differential diagnostic information on this issue. In this chapter the detailed causes of chest pain are shown in order to
improve the diagnostic capability of students, doctors, and nurses who take part in the everyday clinical work.

Figure 1. Chest pain may be generated by various clinical conditions. In most of the cases benign musculoskeletal origin can be determined. Not so frequently life threatening cardiovascular origin can be explored.

12. Causes and pathophysiology of chest pain

12.1. Musculoskeletal pain

At least 36% of patients arriving the emergency unit has benign musculoskeletal pain. Interestingly, females more commonly present with this type of pain compared to male patients. Certain movements, body positions or breathing can provoke the clinical symptom. Usually, the pain is continuously present and sometimes it may even last for weeks. Occasionally, it cannot be localized and in most of cases it can be characterized as sharp pain. At other times the localization (punctum maximum) can be clearly defined (e.g. rib, sternum etc.) and the pain may be provoked by the compression of this well-defined anatomic area.
The differential diagnosis of musculoskeletal pain can be divided into three groups: 1. Isolated musculoskeletal pain syndrome (e.g. rib, bone, sternum etc.), 2. Pain based upon rheumatic disorder, 3. Non-rheumatic disease.

12.1.1. Rib cartilage pain

In an isolated musculoskeletal pain syndrome, usually rib cartilage pain can be identified (due to costochondral inflammation). The pain is generally diffuse and can be provoked by the compression of multiple points of the chest wall. In a majority of cases, the upper ribs are involved and no dermal lesions can be detected.

In the case of Tietze-syndrome the pain can be well localized. In most of the cases costosternal, sternoclavicular and the 2nd and 3rd costochondral joints are affected. The painful area is bloated.

Chest pain may appear after heart surgery. In these cases, the mechanical stress of the chest wall and the sutures directly can play a role in the genesis of clinical symptoms.

In the case of costovertebral joint syndrome the pain is often localized to the posterior chest wall sometimes mimicking acute pulmonary embolism.

Protrusion and herniation of the thoracic intervertebral discs may also lead to chest pain, which is mainly localized to the posterior segment of the chest wall. Rarely, this pain can radiate towards both sides of the chest and the abdomen.

12.1.2. Pain associated to rheumatic diseases

This type of pain often occurs in rheumatoid arthritis, spondylitis ankylopetica psoriasis and fibromyalgia. Rarely, it may appear in the case of systemic lupus erythematosus (SLE), SAPHO syndrome (synovitis, acne, pustulosis, hyperostosis, and osteitis) and recurrent polychondritis.
12.1.3. Chest pain associated to non-rheumatic diseases

This type of pain may appear in the case of traumatic lesions of the ribs or in paraneoplastic fractures. In these cases, coughing, sneezing, and local compression often provoke the pain. Similar pain may occur in the case of septic arthritis, osteomyelitis, and sickle cell anemia. Even the disease of the dermal nerves can provoke such pain. Herpes zoster infection is a typical example for this type of disease, where diagnosis can easily be achieved by finding the typical dermal lesions (exanthemas respecting dermatomes) and paresthesia. Post-irradiation pain may also provoke similar complaints.

13. Cardiovascular diseases and chest pain

13.1. Ischemic heart disease-angina pectoris

Decreased oxygen supplementation and increased oxygen need of the myocardium can lead to angina pectoris. Coronary atherosclerosis and secondary obstruction, furthermore coronary spasms, anemia, hypertension, ventricular hypertrophy and left ventricular strain are thought to be the most important underlying factors. Angina is characterized as a compressing retrosternal chest pain, that may radiate to the left arm, shoulder, towards the left scapula, epigastrium, neck and may be accompanied by dyspnea, palpitation, paleness, sweating, numbness of the IV-V fingers of the left hand, and mortal fear. These symptoms can be terminated by rapidly acting nitroglycerin within 10 minutes. According to the severity of the symptoms two types of angina can be separated: unstable and stable angina.

13.2. Stable angina pectoris

Stable angina pectoris is the most common syndrome of ischemic heart disease. Stable angina can be diagnosed if the pain is provoked by the same activity, it has similar intensity and is rapidly terminated after rest. According to the latest data, nearly 40,000 patients live with stable angina in Hungary. By 2020, the diagnosis and management of ischemic heart disease will result in increased strain on the health system worldwide.

Stable angina has a good prognosis. In this particular population the incidence of acute myocardial infarction is calculated to be within 0.5-2.6 %., furthermore mortality rate is between
0.9-1.4 percent. The clinical appearance of this disease can be variable, so patients must each be taken into with individual consideration. The presence of extracardiac factors that may contribute to similar symptoms along with the severity of the disease must be determined during the initial evaluation. Therapy is based upon the anamnestic data, physical examination, laboratory results, and the result of a special cardiological evaluation. The Canadian Grading System is used to classify stable angina pectoris according to its severity.

<table>
<thead>
<tr>
<th>Grade I</th>
</tr>
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<tbody>
<tr>
<td>Grade I stable angina develops upon strenuous, rapid, and/or prolonged exertion during work or recreation but is not induced by ordinary physical activity, such as walking and climbing stairs.</td>
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</table>

<table>
<thead>
<tr>
<th>Grade II</th>
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<tr>
<td>Grade II stable angina is characterized by a slight limitation of ordinary activity and is induced by the following:</td>
</tr>
<tr>
<td>- Walking or climbing stairs rapidly</td>
</tr>
<tr>
<td>- Walking uphill</td>
</tr>
<tr>
<td>- Walking or stair-climbing after meals</td>
</tr>
<tr>
<td>- Walking more than two level blocks or climbing more than one flight of ordinary stairs at a normal pace and in normal conditions</td>
</tr>
<tr>
<td>- Emotional stress</td>
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<tr>
<td>- During the few hours after waking</td>
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<table>
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<tr>
<th>Grade III</th>
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<tbody>
<tr>
<td>Grade II stable angina is characterized by marked limitation of ordinary physical activity. It is induced by walking one or two level blocks and climbing one flight of stairs in normal conditions and at a normal pace.</td>
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</tbody>
</table>
Grade IV

Grade IV stable angina is characterized by an inability to carry on any physical activity without discomfort. Anginal syndrome may be present at rest.

<table>
<thead>
<tr>
<th>Occurs when the heart must work harder, usually during physical exertion</th>
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<tbody>
<tr>
<td>Doesn't come as a surprise, and episodes of pain tend to be alike</td>
</tr>
<tr>
<td>Usually lasts a short time (5 minutes or less)</td>
</tr>
<tr>
<td>Is relieved by rest or medicine</td>
</tr>
<tr>
<td>May feel like gas or indigestion</td>
</tr>
<tr>
<td>May feel like chest pain that spreads to the arms, back, or other areas</td>
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</table>

Table 2. Symptoms of stable angina pectoris

On one hand, the management of stable angina pectoris is based on effective oxygenation, on the other hand drug therapy has to be initiated (aspirin, statin, nitrate, beta receptor blocker and calcium channel blocker). Two types of nitrates are commonly used during the everyday clinical practice. The rapidly acting nitroglycerin (Nitromint sublingual tablet) aims to terminate the pain as soon as possible. Therapy with slowly acting (extended release) nitrates (e.g. isosorbid mononitrate) is applied to prevent symptoms. The negative chronotropic and inotropic effects of beta blockers reduce cardiac index, resulting in the decrease of myocardial oxygen need. Therefore the pressor response to catecholamines and vascular resistance will also be decreased. Third generation beta blockers also has advantageous side effect profile, mainly from the viewpoint of metabolism. Besides beta blocking effect, carvedilol has an alpha adrenoceptor blocking activity, moreover treatment with nebivolol can result in nitrogen monoxide release and an antioxidant effect. Thus, these are considered to be the most appropriate therapeutic choices.
Trimetazidin optimizes the energetic status quo of the myocardial cell. It results in a reduction in fatty acid oxidation, an increase in glucose oxidation, a decrease in the intracellular calcium load, as well as an antioxidant effect. In patients with persistent complaints despite the combination therapy with beta blockers and calcium channel blockers, the introduction of ivabradin can be an effective decision. It modulates the sinus node’s pacemaker activity (If current blocker), resulting in the lowering of sinus frequency and a concomitant decrease in myocardial oxygen need.

### 13.3. Unstable angina pectoris

In the case of a non-occlusive coronary artery disease (due to a coronary artery thrombus) or in the presence of well-functioning collateral vascular system, unstable angina pectoris or non-Q myocardial infarction with the elevation of cardiac necroenzymes may appear. Various clinical symptoms can appear. The ischemic pain can last for more than 20 minutes, importantly it may be definitive and may be accompanied by dyspnea, nausea, vomitus, palpitation, and presyncope. A patient suffering from unstable angina must immediately be brought to a coronary unit and should be continuously monitored. Therapy should include oxygen, aspirin, nitrate (intravenous), beta receptor blocker, and anticoagulant (low molecular weight heparin). In the case of persistent complaints, symptoms of congestive heart failure, and elevation in cardiac necroenzymes, coronary angiography and revascularization should be performed immediately. In this latter case the combination of aspirin and clopidogrel is indicated.

| Often occurs while you may be resting, sleeping, or with little physical exertion |
| Comes as a surprise |
| May last longer than stable angina |
| Rest or medicine usually do not help relieve it |
13.4. Substrates of chest pain not related to coronary artery disease

Besides the above mentioned pathomechanisms other underlying factors may also lead to chest pain (e.g. long lasting coronary spasm, certain arrhythmias – atrial fibrillation, atrial flutter, paroxysmal supraventricular tachycardia, ventricular tachycardia etc.-, hypotension and hypertension, valvulopathies – mitral prolapse, aortic stenosis.

![Figure 2. Rapid ventricular frequency due to an atrial flutter may lead to hyperkinetic circulatory insufficiency and coronary perfusion defect that can result in angina pectoris syndrome.](image)

Chest pain provoked by coronary artery spasm is called Prinzmetal angina. It can occasionally be initiated by hyperventilation or physical activity, but frequently it appears in dawn. Its real danger can be the occurrence of ventricular tachycardia. As a sign of subepicardial ischemia secondary to coronary spasm, ST segment elevation is likely to appear on the surface electrocardiogram. To prevent vascular spasm, management with beta blockers or calcium blockers has to be initiated.
In the case of cardiac X syndrome, no significant stenosis is present in the main epicardial arteries, nevertheless in 20-50 % of cases microcirculatory insufficiency occurs. It frequently appears in females during premenopausal period. Chest pain is mostly atypical, during stress test ST segment depression is likely to occur and may be provoked by panic reaction.

Sometimes certain valvulopathies can lead to the occurrence of chest pain. Atypical chest pain, dyspnea, and syncope commonly appear in the case of aortic stenosis. Physical examination, electrocardiography and echocardiography can help to achieve the correct diagnosis.

In the case of mitral stenosis, the mostly atypical chest pain is caused by the elevated left atrial pressure load. Furthermore atrial arrhythmias (atrial flutter and fibrillation) may result in the clinical signs of chest pain. Chest pain may even be induced by pulmonary stenosis –which is a rare and mostly inherited disease. Furthermore, pulmonary stenosis can occur as part of a carcinoid syndrome mainly in adults.

In the case of pericarditis, extended ST segment elevation together with chest pain may appear. During the auscultation of the heart, the sounds may be dull and pericardial murmurs.
may be audible. The dullness of the heart is enlarged during the percussion as well as signs of pretamponade may be recognized. In the case of acute pericarditis, chest pain suddenly appears and is characterized as sharp and related to inhalation. Nevertheless, it can be dull too, and may soften after sitting up.

Figure 4. In the case of pericarditis pleural effusion may also appear, which can be detected by means of echocardiography

In the case of myocarditis chest pain is related to the concomitant pericarditis

In the case of aortic dissection, a pathological splitting or longitudinal rupture of the arterial wall happens. As a consequence of the splitting arterial wall, blood appears between the layers. Connective tissue diseases (e.g. Marfan’s syndrome), hypertension, and cocaine use may play a role in its genesis.
Figure 5. In the case of **aortic dissection** a pathological splitting or longitudinal rupture of the arterial wall happens. As a consequence blood appears between the layers.

With regard to the expansion and localization of aortic dissection two clinical classification systems are used. These are DeBakey and Stanford/Daily classifications. DeBakey type I dissection starts at the ascending aorta and spreads to the aortic arch, sometimes extending to the descending aorta, too. DeBakey type II dissection is only located at the ascending aorta. DeBakey type III dissection starts at the descending aorta and may spread proximal or distal. With regard to the Stanford/Daily classification, we make a difference between type A and B. Stanford A involves the dissection located to the ascending aorta, while in the case of type B dissection the ascending aorta is not affected.
Aortic dissection generally results in a strong, rending chest pain that radiates to the back. On rare occasions, there is a lack of definite symptoms with the patient only noticing chest pain and an itching sensation on the neck. Two-third of aortic dissections starts acutely, while one-third of all cases is chronic, namely lasting for more than 2 weeks. If a dissection is suspected, an X-ray is obligatory as it may prove an aortic irregularity with a widened mediastinum. It must be remembered that a patient with congestive heart failure and new onset acute aortic regurgitation may present with a similar clinical picture. If the dissection results in spinal cord ischemia or has carotid artery involvement, neurological symptoms are likely to appear (altered mental status, parapalsy, stroke etc.). Aortic rupture may result in sudden cardiac death or syncope. Moreover, intrapleural bleeding, limb ischemia and myocardial ischemia secondary to coronary artery dissection can occur. Mesenterial arterial dissection can lead to severe bowel ischemia and
abdominal angina, while renal artery dissection may cause progressive worsening of kidney function and severe back pain. Compression of the cervical sympathetic ganglion due to dissection can result in Horner’s syndrome, while compression of the left recurring laryngeal nerve can led to the paralysis of the vocal cord.

Figure 7. Miosis, ptosis and anhydrases on the affected side are the clinical characteristics of Horner’s syndrome. Miosis on the ill side is shown.

Transesophageal echocardiography and CT angiography are the cornerstones of achieving the exact diagnosis.
Figure 8. By means of transesophageal echocardiography both the ascending aorta and the aortic arch can be visualized. In the presence of an intimal flap the diagnosis of aortic dissection can be confirmed.

In acute aortic dissection, surgical intervention is obligatory. Furthermore, in the case of chronic dissection with the occlusion of an important artery, or completed/threatening arterial rupture, or 5 cm widening of the aorta and secondary aortic regurgitation acute surgery is also indicated. In the postoperative period or in chronic dissection not requiring surgical intervention, it is important to lower blood pressure, and this is where beta blockers have a primary role. The Bentall procedure with aortic arch replacement is the recommended surgical solution.
13.5. **Sympathetic activation and chest pain**

Catecholamines (e.g. epinephrine and norepinephrine) cause sympathetic nerve tone increase, tachycardia and vasoconstriction that can result in chest pain which is based upon an increased myocardial oxygen need and transient coronary perfusion defect followed by tissue hypoxia. The sudden release of catecholamines may happen in various diseases. Sometimes cocaine and amphetamine intoxication appear in the emergency care. These materials have vasoconstrictive effect that can lead to hypertension, tachycardia resulting in increased myocardial oxygen need and altered coronary perfusion. As a result myocardial ischemia occurs leading to angina pectoris syndrome. Moreover certain endocrinological diseases (e.g. pheochromocytoma) can cause significant catecholamine release resulting in sudden elevation in blood pressure, tachycardia, headache, sweating, tremor, paleness and flush. Rarely angina pectoris can be present, too.

**Takotsubo-cardiomyopathy** is a rare, reversible, but severe disease resulting in transient ventricular dysfunction caused by stress and sympathetic overdrive conditions. Angina pectoris
may be present without definitive coronary artery disease. During echocardiography apical wall motion abnormality can appear which does not respect the usual territories of coronary perfusion. During the attack the danger of ventricular arrhythmias is increased. Surface electrocardiography can show ST segment elevation, T wave inversion and the prolongation of QT interval.

![Electrocardiogram](image)

Figure 10. Electrocardiogram of a patient suffering from Takotsubo-cardiomyopathy. During the attacks ST segment elevation is observed.

14. **Gastroenterological diseases and chest pain**

14.1. **Diseases of the esophagus**

Gastroesophageal reflux (GERD) is one of the most important gastroenterological disorder that may result in chest pain. This pain is described as a compressing one and can be very similar to angina pectoris. It may appear as a burning pain and commonly occurs after a meal, which radiates towards the neck, back and arms and can last for hours. These symptoms may be abolished by the administration of antacids. Reflux disease can only be diagnosed after the exclusion of a cardiac origin. The lowering of gastric acid production with H2 blockers and proton pump inhibitors are the cornerstones of the management. Furthermore, prokinetics (e.g. metoclopramide), diet, and sleeping with lifted head can be advised and beneficial.
Figure 11. In the case of gastroesophageal reflux gastric acid regurgitates from the stomach towards the esophagus. This may cause the irritation of the esophageal mucus membrane resulting in characteristic symptoms. The insufficiency of the lower esophageal sphincter is the underlying cause of this phenomenon. Laryngitis, pharyngitis, even laryngeal and glottis spasm can be the severe consequences of the regurgitating acid.

In the case of eosinophil esophagitis, symptoms of reflux disease are likely to appear, however antacids are not able to terminate the complaints. Burning chest pain similar to angina pectoris are the most common symptoms. Sometimes dysphagia can appear as well. The diagnosis is based on histological evaluation.

In the case of esophageal hypersensitivity, the pain threshold of these particular patients are lower compared to the average population. The pain can be provoked by intra-esophageal balloon dilation which easily proves the esophageal origin of the complaints.

Motility disorders of the esophagus (mainly esophageal spasm) can also cause chest pain. The rare and benign lesion of the esophagus, achalasia is based on the relaxation inability of the
esophageal muscles. Endoscopic dilation of the affected area and administration of calcium channel blockers and beta blockers are the most important therapeutic possibilities. Botulinum toxin injected directly to the esophageal muscle may result in temporary improvement, thus it is thought to be an alternative way of treatment. In the case of a refractory disease, surgical intervention may be indicated.

Figure 12. In the case of achalasia the muscles of the cardia are not able to relax, thus consequent intra-esophageal pressure overload occurs, leading to the irritation of the esophageal mucus membrane and chest pain.

Foreign body, rupture and perforation of the esophagus may also cause chest pain.

Certain antibiotics (e.g. tetracycline, doxycycline, clindamycin), non-steroid anti-inflammatory drugs (e.g. aspirin), furthermore bisphosphonates, potassium-chloride, and chinidin may irritate the esophageal mucus membrane resulting in chest pain.

Sometimes the infective diseases of the esophagus (e.g. fungal infections, cytomegalovirus) can result in the appearance of chest pain. These are likely to occur in patients with altered immune reactivity (as it can appear in acquired immune deficiency syndrome).
14.2. Other gastroenterological origins of chest pain

In the case of biliary inflammation, bilious attack, peptic ulcer, kidney stone disease, appendicitis, infarction of the spleen and subphrenic abscess the pain often radiates towards the chest, making the diagnostic activity more complicated.

In the case of pancreatitis, intensive epigastric pain may appear that radiates toward the hypochondria, the chest, and the back. It is often accompanied by fever, nausea, vomitus, diarrhea, and anorexia. In severe cases, hypovolemia, circulatory insufficiency, and even shock can occur. Rarely, hemorrhagic lesion in the lumbar area (Grey-Turner’s sign), periumbilical hemorrhage (Cullen’s sign), and periumbilical ecchymosis (Grunewald’s sign) can appear due to the toxic damage of the vessels. The sternum’s xiphoid process may sore when touched (Kamenchik’s sign). The costovertebral joint of rib XII may also be sensitive when compressed (Mayo-Robson’s sign). Furthermore, determination of serum amylase concentration can help to get the exact diagnosis (in the case of triple elevation specificity nearly reaches 100 %). In the case of alcohol induced pancreatitis, lipase is known to be more elevated compared to amylase, but peak concentration is reached later. Moreover, elevated CRP, leukocytosis, neutrophilia, and radiological abnormalities can also help in clearing the diagnosis. Fleischer’s atelectasis may appear on X-ray. In order to determine the severity of pancreatitis Ranson’s criteria are widely used.

Severe diet, volume intake, pain killers, antibiotics, naso-jejunal tube and parenteral feeding are the basic therapeutic tools. In the case of pancreas necrosis, surgical intervention may be reasonable.

At admission or at diagnosis

- age>55
- Leukocytosis $>16\text{G}/\text{L}$
- Hyperglycemia $>11\text{ mmol}/\text{L}$
- Se LDH $>400\text{ U}/\text{L}$
- Se AST $>250\text{ U}/\text{L}$

**During the first 48 hours**

- HTC drop $>10\%$
- volume loss $>4000\text{ ml}$
- Hypocalcaemia $<1.9\text{ mmol}/\text{l}$
- Hypoxemia (PO$_2 < 60\text{ mmHg}$)
- Urea nitrogen increase $>1.8\text{ mmol}/\text{L}$ despite intravenous volume intake
- Hypalbuminemia $<32\text{ g}/\text{L}$

Table 4. Ranson’s score is a useful diagnostic tool in establishing the severity of pancreatitis. Three or more criteria proves a severe disease.

15. **Pulmonary diseases and chest pain**

Chest pain due to pulmonary diseases is usually caused by pulmonary vessels, the parenchymal lesions of the lungs, abnormalities of the airways and the pleura.

15.1. **Chest pain caused by pulmonary vessels**

In the case of acute pulmonary embolism, various clinical signs may appear. According to the data obtained from the Prospective Investigation of Pulmonary Embolism Diagnosis (PIOPED) study, dyspnea occurs in 73 % of all cases. Furthermore 66 % of these subjects complain about pleural pain, while in 37 % coughing, and in 13 % hemoptysis can be elucidated.
Eighty-four percent of patients have X-ray abnormalities (higher diaphragm level – 40 %, atelectasis, wide central pulmonary artery with peripheral amputation, pleural effusion-30 %, pyramid shape infiltrate, right ventricular enlargement). Furthermore in more than 50 % of the cases non-specific electrocardiographic abnormalities may appear (S1Q3, P-pulmonale, complete or incomplete right bundle branch block).

Figure 13. In the case of pulmonary embolism the sensitivity of electrocardiography was found to be low (approximately 23 %). P pulmonale is shown on this electrocardiogram. It appeared due to right atrial and ventricular pressure overload secondary to pulmonary embolism.

In the case of acute pulmonary embolism with hemodynamic consequences, thrombolysis is indicated. With regard to pulmonary microembolism without hemodynamic deterioration, the introduction of anticoagulant therapy is recommended. If thrombolysis is contraindicated in the case of a massive embolism, acute surgical embolectomy may be considered.

In the case of pulmonary hypertension and cor pulmonale, numerous clinical symptoms can occur. These are generated by increased right ventricular afterload and secondary right ventricular dysfunction. Patients commonly report about fatigue and chest pain, rarely, syncope may even occur. The chance of typical angina pectoris secondary to physical activity is increased in patients with valvulopathies (especially in the case of mitral valve involvement). This phenomenon may be induced by the increased stretch of the pulmonary artery and right ventricular ischemia.

Sometimes the parenchymal lesions of the lungs can lead to chest pain. Pulmonary infarction, neoplasia, sarcoidosis, chronic obstructive pulmonary disease (asthma, emphysema,
chronic bronchitis) are the most important in this field. In 30\% of pneumonia cases chest pain appears, where the initiation of antibiotic treatment is substantial.

Chest pain due to a pleural disease is described as stabbing in quality, related to breathing, and may be pronounced during inhalation. This type of pain is often generated by the pleura itself, and can be seen in viral infections mainly in youngsters. To reduce pain, the use of non-steroid anti-inflammatory drugs (paracetamol, diclofenac) are recommended.

Pneumothorax is the appearance of air in the intrapleural space, which is likely to appear when the visceral pleura is damaged (e.g. emphysema, tuberculosis, abscess, trauma, iatrogenic etc.). Generally, chest pain is heavy and can be accompanied by dyspnea, cyanosis and tachycardia. Symptoms are highly depending on the duration of onset and the amount of intrapleural air. Tension (ventil) pneumothorax can be extremely dangerous, where the airflow during inspiration is free, but outflow during expiration is not possible. Therefore, intrapleural pressure is continuously increasing, resulting in mediastinal dislocation, pulmonary compression, i.e. life threatening consequences. Consequently, breathing insufficiency occurs, with no breathing sound on the affected side. Furthermore, tactile fremitus and bronchophonia are decreased, while percussion becomes tympanic above the affected area. In this dangerous situation, immediate decompression is indicated. In the case of open pneumothorax, both the heart and the mediastinum show fluctuating movement that may worsen their function. After the clinical diagnosis of pneumothorax, a suction drain should be implanted into the chest.
Figure 14. Pneumothorax has different types: open and ventil pneumothorax. In the case of open pneumothorax air can go in and out freely. On the contrary, in the case of ventil (tension) pneumothorax the airflow is free during inspiration, but blocked during expiration resulting in mediastinal dislocation.

16. **Psychogenic substrates of chest pain**

Chest pain can be the part of panic syndrome. Importantly, psychogenic origin can be stated only after the exclusion of organic diseases.

Munchausen-syndrome is a type of neurosis, where patients complain about clinical symptoms in order to draw the attention of the healthcare professionals. More than 50 % of these patients arrive the emergency with chest pain as well as dyspnea and back pain may appear.

These patients require regular psychiatric follow up.
17. Diagnosis of chest pain

17.1. Radiological and laboratory diagnosis

Chest X-ray can clarify the position and the size of the heart, also information may be collected about pleural and pulmonary diseases.

Surface electrocardiography can provide information about the electrical and structural status of the heart. Nevertheless, its unique importance is based on the data related to the diagnosis or exclusion of myocardial ischemia and infarction (with the sensitivity to the maximum of 50 %).

<table>
<thead>
<tr>
<th>Information provided by electrocardiography</th>
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<tbody>
<tr>
<td>• Pulse generation abnormalities</td>
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<tr>
<td>• Pulse conduction disorders</td>
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<tr>
<td>• Depolarization</td>
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<tr>
<td>• Repolarization</td>
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<tr>
<td>• Rhythm/arrhythmia</td>
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<tr>
<td>• Ischemia</td>
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<td>• Ventricular hypertrophy</td>
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<td>• left and right heart pressure or volume overload</td>
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<tr>
<td>• Pulmonary embolism</td>
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<tr>
<td>• Electrolyte imbalance</td>
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<tr>
<td>• Myocarditis/pericarditis</td>
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<td>• Ion channel diseases</td>
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<td>• Congenital heart diseases</td>
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Table 5. Information provided by electrocardiography

Laboratory examination has a basic role in the diagnosis of acute myocardial infarction based upon the measurement of cardiac necroenzymes. Troponin, creatine-kinase (CK), and its isoenzyme CK-MB are the most important parameters measured in the everyday emergency practice. Transaminases and lactate dehydrogenase are not specific enough. Although myoglobin...
is a rapidly elevating necrosis marker, it has proven to be aspecific, thus its emergency routine use in the diagnosis of acute coronary syndrome is still not recommended.

Echocardiography can provide data on certain cardiac wall motion abnormalities of a patient with chest pain (hypokinesis, akinesis, and dyskinesis). Echocardiography’s sensitivity reaches 90% in the diagnosis of acute myocardial infarction, however its specificity is limited due to the indefinite onset of these wall motion disturbances. Moreover, echocardiography is an important diagnostic tool in the case of a suspected aortic dissection.

Figure 15. Transesophageal echocardiography is an important tool in the emergency diagnosis of aortic dissection. An intimal flap may be detected in the aorta which separates the real lumen from the pseudo lumen.

Ultrasound, CT and MR provide effective diagnostic tools to define the underlying cause of musculoskeletal chest pain. Computer tomography has significant role in the diagnosis of acute pulmonary embolism. Lately, a novel diagnostic tool, coronary CT angiography has been introduced in order to evaluate the coronary status. It has a negative predictive value of 100%, but in the case of a positive result further cardiological examinations are needed. Moreover, CT is widely used in the diagnosis of abdominal diseases too.
18. References

- Mark H Beers, Robert Berkow (Eds) MSD Orvosi Kézikönyv, Melanía Kiadó Kft, Budapest, 1999, pp 1662-1668
- Tomcsányi János (Szerk) Klinikai Kardiológia. Medintel Könyvkiadó, Budapest, 1997, pp 14-17, 122-123
19. **Self-controlling questions**

1. **What is the definition of angina pectoris?**
   - Chest pain caused by ischemia due to decreased oxygen supply and increased oxygen need of the myocardium.

2. **What are the most common clinical features of angina pectoris?**
   - Compressing chest pain
   - Retrosternal localization
   - Tingling of the left arm
   - Pain radiates to the neck, to the left shoulder and to the left arm
   - Pain may be accompanied by palpitation feeling, dyspnea and sweating

3. **What are the main types of angina pectoris?**
   - stable
   - unstable

4. **Can angina pectoris be classified according to its severity?**
   - Yes, it is classified according to the Canadian Grading System.

5. **Can chest pain be caused by extracardiac diseases?**
   - Yes commonly musculoskeletal, gastroenterological, pulmonary and abdominal pain occurs.

6. **Which are the main characteristics of musculoskeletal pain?**
It can be provoked by certain movements, body positions and breathing. It may continuously persist, sometimes it cannot be localized and mostly characterized as sharp pain. On certain occasions the punctum maximum can be well determined (e.g. rib, sternum etc.). The pain can be provoked by compression of the affected area.

7. What are the characteristics of pain caused by gastroesophageal reflux?

Chest pain is similar to angina pectoris because it has a compressing feature. On the contrary, rarely it can be burning and substernal and may occur after having a meal in the supine position. Pain can radiate to the back, arms, neck and may last for hours. Most importantly, symptoms can be rapidly diminished by the administration of antacids.

20. Case reports

Case 1.

A middle-aged male patient arrives at the emergency due to retrosternal, compressing chest pain which is accompanied by the tingling of the left arm and rest dyspnea. These symptoms were provoked by nervous break-down and blood pressure of 220/110 mmHg. Blood pressure at the emergency is still measured to be 190/110 mmHg.

Question: What is the suspected diagnosis?

Answer: Angina pectoris due to hypertension

Question: What examinations would you perform to clear the patient’s condition?

Answer: Physical examination, ECG, arterial blood gas, laboratory tests (cardiac necroenzymes) continuous pulsoxymetry.

Question: At the beginning of the emergency management what would be your therapeutic consideration?
Answer: The administration of oxygen, nitrate, beta-blocker and due to hypertension captopril.

Question: How would you exclude acute myocardial infarction?

Answer: With the help of ECG and laboratory examinations. Echocardiography may also help, as it has a high negative predictive value.

Question: What could be the pathomechanisms of chest pain?

Answer: The elevation of blood pressure resulting in an increased left ventricular afterload, provoking myocardial ischemia and worsening coronary perfusion.

Case 2.

A 65 year-old male patient is transported to the emergency center by the ambulance. He has been smoking for 20 years, and has been diagnosed with type 2 diabetes mellitus 10 years ago. A heavy compressing chest pain with dyspnea, tingling of the left arm, paleness, sweating have appeared 2 hours ago. Dyspnea is continuously increasing, bluish discoloration of the lips can be recognized.

Question: What is the most feasible diagnosis?

Answer: Acute left ventricular failure as part of acute coronary syndrome.

Question: How would you clarify the diagnosis?

Answer: By means of ECG and laboratory tests.

Question: You find a significant elevation in CK and troponin. Furthermore, with regard to electrocardiogram 3- mm ST segment elevations are recognized in leads V2-6. What is the possible diagnosis and what would you do?

Answer: The diagnosis is likely to be acute myocardial infarction (anterior STEMI). Primary coronary intervention (PCI) is the most important therapeutic goal. Furthermore, in the primary
care the administration of 5000 IE sodium-heparin, oxygen, morphine, nitrate, aspirin, and clopidogrel and due to pulmonary congestion diuretics is recommended.

Case 3.

A 50 years old male patient has been drinking alcohol regularly. She has been suffering from epigastric and chest pain for 2 days. These complaints were getting stronger in the last 3 hours, that’s why she decided to visit the emergency. During the first evaluation epigastric pain can be provoked with palpation. Bowel motility is found to be decreased and fever is recognized.

Question: What is the possible source of these symptoms?

Answer: Pancreatitis due to alcohol.

Question: How would you clarify the diagnosis?

Answer: With laboratory tests (amylase, lipase, leukocyte, CRP), and with abdominal ultrasound.

Question: How can acute myocardial infarction be excluded?

Answer: With the determination of cardiac necroenzymes and with electrocardiography. Abdominal pain aggravated by palpation, anamnestic data and fever do not suggest the presence of acute myocardial infarction.

Case 4.

A 70 year-old patient suffering from pulmonary emphysema is transported to the emergency by the ambulance due to a right sided, sudden and sharp chest pain. During the first evaluation tympanic sound can be heard above the affected area with percussion, nor can breathing sound be audible.
Question: What may cause these symptoms?

Answer: Spontaneous pneumothorax is likely to be the underlying disease.

Question: How would you clarify the diagnosis?

Answer: Chest X-ray should be performed.

Question: X-ray proves the diagnosis of pneumothorax on the right side. What would be the next step?

Answer: The implantation of a suction-drain should follow. Suction should be maintained for at least 3 days.

Question: What caused pneumothorax?

Answer: Pneumothorax was likely to be caused by emphysema and bulla rupture.

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**Case 5.**

A middle-aged male patient is carried to the emergency due to hypokalemia. The electrolyte imbalance was caused by diarrhea. During his observation suddenly a regular, wide QRS tachycardia occurs with the ventricular frequency of 180/min. It is accompanied by hypotension (RR 90/70 mmHg), cyanosis, increasing rest dyspnea and strong compressing chest pain.

Question: What can cause these symptoms?

Answer: It is possibly caused by ventricular tachycardia with hemodynamic deterioration (acute heart failure, coronary perfusion defect, myocardial ischemia).

Question: What therapy would you chose?

Answer: Acute electrical cardioversion should be chosen.
21. Tests

21.1. True or false questions

Please underline the letter T if the answer is true and letter F if it is false. More true answers are accepted!

1. **TF** Chest pain is induced only by myocardial ischemia
2. **TF** Angina pectoris is provoked by coronary artery dilation
3. **TF** In the case of unstable angina patient should be monitored in a coronary unit
4. **TF** In the case of stable angina the provoking factors are known

1. **TF** Tietze syndrome is caused by the inflammation of rib cartilage
2. **TF** Tietze syndrome should be treated with nitrates
3. **TF** In the case of gastroesophageal reflux aspirin therapy is indicated
4. **TF** Beta blockers cannot be used in the case of angina pectoris

1. **TF** The use of antacids is contraindicated in reflux disease.
2. **TF** A heavy, stubbing pain may occur in the case of aortic dissection.
3. **TF** Blood pressure lowering is contraindicated in the case of aortic dissection
4. **TF** Arrhythmia may not cause chest pain

1. **TF** Hypertension never leads to increased ventricular strain and chest pain
2. **TF** The determination of myoglobin is obligatory in a suspected acute myocardial infarction
3. **TF** Pulmonary hypertension is often associated to deep vein thrombosis.
4. **TF** The electrocardiographic signs of right ventricular strain are likely to appear in pulmonary embolism

1. **TF** Psychiatric disease cannot cause chest pain
2. **TF** In the case of Munchausen syndrome the use of oxygen is mandatory

3. **TF** Panic syndrome commonly leads to chest pain

4. **TF** Thrombolysis is contraindicated in pulmonary embolism.

### 21.2. Multiple choice questions

Please underline the correct answers! More answers may be true.

**Which diseases can cause angina pectoris?**

- Coronary artery disease
- Arrhythmias
- Hypertension
- Tietze syndrome

**What are the true statements with regard to pulmonary embolism?**

- Never causes chest pain
- Coughing and hemoptysis may be the first symptoms
- Thrombolysis is indicated in the case of hemodynamic deterioration
- Surgical embolectomy is never recommended

**What are the true statements with regard to reflux disease?**

- Never causes pharyngitis
- Chest pain may decrease after the administration of antacids
- Anticoagulant treatment is obligatory
- Lying with elevated head is recommended

**Which are the false statements?**
- Diuretics are used as first line in the management of angina pectoris
- Beta blockers are recommended in the therapy of angina pectoris
- In the case of reflux disease prokinetics are useful
- The diagnostic value of ECG is low with regard to acute myocardial infarction

What are the symptoms of acute aortic dissection?

- Heavy, stubbing chest pain
- Signs of circulatory abnormalities of the lower limbs
- Signs of cerebral perfusion defect
- Pharyngitis

21.3. Single choice questions

Please underline the correct answer!

1. Tetracycline may cause esophagus irritation
2. Chest pain is not likely to appear in hypertension
3. Arrhythmias induce chest pain for sure
4. Beta blockers are the basic drugs for the treatment of reflux disease

1. The administration of diuretics is obligatory in panic disorder
2. Munchausen syndrome is based on severe cardiac disease
3. Transesophageal echocardiography is an important diagnostic tool with regard to aortic dissection
4. Right ventricular failure is not likely to appear in pulmonary hypertension

1. In the case of pleuritis, symptoms are generated by the peritoneum
2. Acute pulmonary edema is caused by right ventricular failure
3. Pleural pain may be associated to breathing
4. Pulmonary embolism leads to the appearance of left bundle branch block
1. Stable angina means crescendo type chest pain
2. Unstable angina may appear as a surprise
3. Nitroglycerine increases blood pressure
4. Underlying factor cannot be determined in the case of stable angina

1. Psychogenic control of a patient with Munchausen syndrome is necessary
2. Pneumothorax never appears after big vein cannulation
3. Acute intervention is not indicated in tension pneumothorax
4. Chest pain never appears in the case of bronchial asthma

21.4. Relational analysis

A: Both statement and justification are true and the latter well explains the statement.
B: Both are true, but the justification do not explain the statement.
C: Statement is true, but justification is false.
D: Statement is false, but justification is true.
E: Both are false.

- In the case of reflux esophagitis acid regurgitates towards the esophagus, which causes chest pain. (A)
- Angina pectoris is caused by rib cartilage inflammation, so physiotherapy is the essential treatment (E)
- In the case of tension pneumothorax immediate detensioning is indicated, therefore acute open surgery has to be performed (C)
- Unstable angina is caused by coronary artery disease, thus patients should be monitored in a coronary unit (A)
- Pulmonary embolism is based on the occlusion of pulmonary vein, which always indicates immediate surgical intervention (E)