Heart insufficiency as a disease of old age constitutes a serious clinical picture as its complications often result in death. Cardiogenic pulmonary edema, as a consequence of heart insufficiency, as well as non-cardiogenic pulmonary edema, are diseases indicating immediate measures as they are accompanied by rapid respiratory insufficiency. This article will provide you with everything you need to know about the epidemiology, etiology, classifications, diagnostics and therapy for cardiac insufficiency and pulmonary edema.
Definition

Cardiac insufficiency refers to the inability of the heart to supply the body with normal cardiac volume under normal end-diastolic pressure conditions. The World Health Organization (WHO) defines cardiac insufficiency according to the degree of reduced physical capacity due to ventricular dysfunction.

Epidemiology

In cases of cardiac insufficiency, there is a clear prevalence associated with old age. While the percentage is only at 1% in patients over 50, it increases to 8% in patients over the age of 80. The men/women ratio is 1.5:1.

Etiology

There are several causes of cardiac insufficiency. The most common cause is hypertension in more than 50% of the cases which may result in coronary heart disease, myocardial infarction, and cardiac insufficiency. Other more uncommon causes are atrial fibrillation, cardiomyopathy, valvular heart diseases, high-output heart failure or pericardial diseases.

Systolic and Diastolic Dysfunction

Systolic dysfunction

- Left ventricular (LV) systolic dysfunction = pump is weak

Diastolic dysfunction

- LV diastolic dysfunction = pump is too stiff
- Impaired relaxation of the left ventricle
- Results in impaired LV filling with resultant pulmonary edema and clinical CHF
- At the bedside, difficult to differentiate the 2
- Diastolic dysfunction is becoming increasingly understood

Classifications of Cardiac Insufficiency

There are different models of classifying cardiac insufficiency according to various aspects. A very broad classification is the division into **compensated and decompensated cardiac insufficiency** or the classification into **acute or chronic cardiac insufficiency**.

**NYHA Classification**

A well-known model is the NYHA classification (NYHA: **New York Heart Association**), which divides cardiac insufficiency into 4 classes according to their clinical severity:

- Class 1: no symptoms and normal physical capacity
- Class 2: symptoms appear only during increased physical activity
- Class 3: symptoms already appear during light physical activity
- Class 4: symptoms already appear at rest
AHA Classification

According to the American Heart Association (AHA), cardiac insufficiency can be categorized into 4 stages as well:

- Stage 1: The patient is symptom-free and does not show any signs of structural heart disease, but there are risk factors for the development of cardiac insufficiency.
- Stage 2: The patient does not display any symptoms of cardiac insufficiency, but he has structural heart disease.
- Stage 3: Structural heart disease, in combination with cardiac insufficiency symptoms, is present.
- Stage 4: Terminal cardiac insufficiency is present.

Classification According to Cardiac Output

According to this classification, one can differentiate between low-output heart failure (heart failure with reduced ejection fraction) and high-output heart failure (also HF-PEF for heart failure with preserved ejection fraction). The 1st form constitutes forward heart failure with insufficient cardiac output, whereas, with high-output failure, cardiac output is increased due to a peripheral lack of oxygen supply.

Classification According to Affected Area

Depending on which chambers of the heart are affected, cardiac insufficiency may be referred to left ventricular heart failure, right ventricular heart failure or bilateral ventricular heart failure (congestive heart failure). Isolated right ventricular heart failure, in comparison, is rare and frequently occurs as a result of left ventricular heart failure causing a backflow of blood into the right chamber of the heart which results in right ventricular heart failure.

Pathophysiology of Cardiac Insufficiency

A particular problem with cardiac insufficiency is the fact that insufficient cardiac output, along with insufficient blood supply of the body organs, may lead to a number of compensatory mechanisms.

Among these compensatory mechanisms is the activation of the sympathetic nervous system, along with the release of catecholamines, as well as the activation of the renin-angiotensin-aldosterone-system (RAAS) and antiuretic hormone (ADH) production. The release of natriuretic peptides, as well as cardiac remodeling and cardiac hypertrophy, are among these compensatory mechanisms as well.

The problem with these compensatory mechanisms is that, while helpful at first, they will lead to a significant deterioration of cardiac insufficiency if chronically activated. The critical heart weight is, for instance, at 500 g. If it weighs more than that, the oxygen supply of the heart becomes critical. Furthermore, cardiac insufficiency frequently leads to a loss in contractility, despite pathological myocyte growth.

Clinical Signs of Cardiac Insufficiency
The clinical signs of cardiac insufficiency are variable, depending on the severity of the insufficiency. Among them is dyspnea on exertion or even at rest at a more advanced stage, asthma (cardiac asthma) and orthopnea, symmetric edema, especially on the ankles, on the tibia and on top of the foot. There is also nocturia due to nocturnal voiding of edema.

**Pulmonary edema**, dry cough, **diminished vital capacity, and fatigue** can be signs of cardiac insufficiency as well. **Cerebral symptoms** such as confusion and fear are caused by diminished blood flow to the brain.

Dyspnea and pulmonary edema are more likely caused by acute LV heart failure, whereas acute right ventricular (RV) heart failure manifests itself in gastrointestinal disorders such as liver pain caused by fluid backing up into the liver which results in tender hepatomegaly.

**Pleural effusion**, engorged kidneys with proteinuria and **changes in body weight** due to water retention, can also be signs of cardiac insufficiency.
Diagnostics of Cardiac Insufficiency

When diagnosing cardiac insufficiency, gathering the patient’s medical history is important, particularly focusing on symptoms and existing risk factors such as, for instance, smoking, high blood pressure or diabetes mellitus. The physical examination can yield different findings:
- Engorged neck veins
- Cyanosis
- Displaced apex beat
- Cardiomegaly
- Tachycardia and arrhythmia
- Pulmonary rattling sounds and audible signs of asthma
- **Hepatomegaly with the rounded edge of the liver, the liver is sensitive to pressure and jaundice**
- Edema
- Muscular atrophy

Diagnostic markers of cardiac insufficiency are brain natriuretic peptide (BNP) and N-terminal pro-brain natriuretic peptide (NT-proBNP) in particular, which are released by cardiomyocytes during physical exertion. Other lab tests are non-specific and usually done in order to determine comorbidities or possible causes or to rule out differential diagnoses.

Other laboratory tests include **blood glucose, electrolytes, cardiac markers** for myocardial damages such as creatine kinase (CK), creatine kinase-muscle/brain (CK-MB) and troponin, **liver, and kidney function tests** (glutamic-oxaloacetic transaminase (GOT), glutamic-pyruvic transaminase (GPT), gamma-glutamyl transferase (γ-GT), bilirubin, urea), **cholesterol, triglycerides**, and **thyroid function tests** (thyroid-stimulating hormone (TSH), free thyroxine (FT4)).

Aside from laboratory findings, **ECG** and **echocardiography** are suitable for diagnosing cardiac insufficiency. Typical changes in **ECG** are cardiac arrhythmia or signs of atrial or ventricular hypertrophy. Cardiac echocardiography is particularly suitable for diagnosing cardiac dysfunctions: It will show ventricular hypertrophy, valvular heart disorders or changes in cardiac wall motion and ventricular dilatations.
In addition, tests such as chest X-rays, coronary angiography, computed tomography (CT) and magnetic resonance tomography (MRT), long-term and stress electrocardiogram (ECG), as well as pulmonary function tests, are suitable to recognize an enlarged heart, calcifications or pulmonary congestion, and to rule out pulmonary diseases.

**Differential Diagnosis**

Bilateral pitting edema due to cardiac diseases should be differentiated from other causes of bilateral lower limbs edema, which include:

1. Kidney diseases
2. Liver diseases
3. Hypoproteinemia

Differential diagnosis with regard to cardiac insufficiency relates to edemas, 1st and foremost, which must be separated from cardiac edema. Among them are kidney and liver diseases, and hypoproteinemia, which is responsible for soft tissue edema; in the clinical picture, they represent non-symmetric edema.

Venous outflow obstruction doesn’t cause bilateral lower limb edema and it’s better to be removed from the differential diagnosis.

**Treatment of Cardiac Insufficiency**

With regard to cardiac insufficiency, one has to differentiate between acute and chronic cardiac insufficiency, as well as between causal and symptomatic therapy.

**Chronic cardiac insufficiency therapy**

The causal approach considers the disease at hand, i.e. the treatment of arterial hypertension, myocarditis, cardiomyopathy or cardiac arrhythmia. Furthermore, revascularization in cases of coronary heart disease, as well as decreasing the risk factors, is part of cardiac insufficiency therapy. Surgical intervention, for instance in cases of a heart valve defect, is also part of the causal approach.

Symptomatic therapy can be divided into conservative and medical approaches. Among the conservative measures are reducing risk factors and a therapeutic sports program in cases of stable cardiac insufficiency, as well as physical and emotional support in cases of decompensated cardiac insufficiency, respectively. As far as nutrition
is concerned, a low-salt, light diet rich in potassium, is recommended.

The goal is to avoid hypokalemia and hyponatremia and possibly limit fluid supply if there is a tendency to develop edema. Furthermore, medications that may deteriorate the condition of the cardiac insufficiency, such as non-steroidal-anti-rheumatic drugs (NSAR), glucocorticoids or calcium channel blockers, must be eliminated.

Medical treatment conforms to the severity of cardiac insufficiency as defined by the NYHA classification. Angiotensin-converting enzyme (ACE) inhibitors or angiotensin receptor antagonists (in case of intolerance to ACE inhibitors which may cause a dry cough) are indicated as improving the prognosis in all classes. Starting with class 2, beta-blockers and diuretics as well as aldosterone antagonists and ivabradine, are indicated. Cardiac glycosides (digitalis) will be used starting with class 3.

Furthermore, cardiac resynchronization therapy to optimize cardiac contractions, implanting a cardioverter defibrillator (ICD) or a combination of both may be sensible. The last option remaining is heart transplantation.

**Acute cardiac insufficiency therapy**

Acute cardiac insufficiency therapy can also be divided into causal and symptomatic treatment. Causal therapy consists of, for instance, the treatment of a hypertensive crisis, recanalization in cases of heart attack, surgical cardiac interventions in cases of acute insufficiency or congenital defects, as well as the treatment of bradycardia or tachycardia.

Symptomatic therapy for acute left ventricular heart failure constitutes a sitting position, sedation, and oxygen, as well as preload reducing measures such as administering nitroglycerin and short-acting diuretics such as furosemide. The administration of positive inotropic beta-receptor agonists, such as dobutamine, may be sensible as well. Furthermore, the goal is to control and optimize respiratory rate (RR), central venous pressure (CVP), and carbon monoxide (CO), even with the aid of medical devices, if necessary.

**Complications of Cardiac Insufficiency**

Complications from cardiac insufficiency are to be taken seriously as they frequently result in death. In cases of cardiogenic shock, for instance, which constitutes acute forward heart failure with reduced organ perfusion, the mortality rate is above 50%.

Another complication is pulmonary edema, which may cause hypoxemia. Cardiac arrhythmias, thromboembolism, as well as chronic cardiac insufficiency turning into acute decompensated cardiac insufficiency, are other complications to be expected.

**Prevention**

Preventative measures that are suitable for cardiac insufficiency are those geared toward the most common causes of cardiac insufficiency. The goal is to avoid arterial hypertension, to control one’s body weight with healthy and balanced nutrition, as well as exercise. Abstinence from nicotine, as well as limited alcohol consumption, are preventative measures as well.
Pulmonary Edema

Definition

Pulmonary edema constitutes the **accumulation of fluid in the lungs.**

Etiology

In cases of pulmonary edema, a distinction has to be made between cardiogenic and non-cardiogenic pulmonary edema, whereby the 1st type does not involve lung disease but occurs much more frequently in the clinical routine.

Cardiogenic pulmonary edema, also hydrostatic pulmonary edema, is frequently caused by **acute left ventricular heart failure** as the heart is no longer capable of adequately pumping blood from the pulmonary circulation into the systemic circulation, thus causing blood to back up into the lungs. Other causes may be **aortic mitral valve stenosis**, **tachycardia** or **hypertensive heart diseases.**

Non-cardiogenic pulmonary edema may be caused by permeability, oncotic pressure or hypoxia. Pulmonary edema, due to permeability, may have **toxic causes** such as gases, or be caused by **medications**, i.e. azathioprine. Other causes include **infections** or **aspiration.** Pulmonary edemas caused by oncotic pressure have a renal origin, i.e. renal failure. Pulmonary edema caused by hypoxia is associated with high altitude (‘high-altitude pulmonary edema’).

**Classification**

Aside from the classification according to causes into **cardiogenic and non-cardiogenic pulmonary edema**, there is another classification according to where the edema is located. Hereby, the **alveolar pulmonary edema** has to be separated from the **interstitial pulmonary edema.** Interstitial pulmonary edema can rapidly progress into the alveolar form as the connective tissue frame of the lungs has little space for fluid accumulation.
Pathophysiology

The pathophysiology of pulmonary edema is based on an imbalance of fluid reabsorption and filtration. Increased pulmonary capillary pressure quickly leads to fluid build-up in the lungs and massively impairs gas exchange, which explains the respiratory failure: Lung compliance and vital capacity decrease, airway resistance and range in path length to gas exchange increase.

The pathophysiology of high-altitude pulmonary edema may be explained by a combination of a decrease in pulmonary oxygen contents, pulmonary vasoconstriction, as well as decreased alveolar pressure.

Symptoms and Clinical Signs of Pulmonary Edema

Depending on the stage of pulmonary edema, symptoms may include dyspnea, cough, thick mucus discharge, tachycardia, signs of cyanosis, as well as restlessness. While interstitial pulmonary edema is more characterized by tachypnea, dyspnea, orthopnea, as well as sharp breathing noises (cardiac asthma, ‘asthma cardiale’), in cases of alveolar pulmonary edema, fear, cyanosis, paleness, and extreme dyspnea, as well as discharge, may occur accompanied by moist rattling sounds that are audible with a stethoscope.

Progression and Special Forms of Pulmonary Edema

The progression of pulmonary edema can be divided into 4 stages:

- Stage 1: This is connective tissue edema – interstitial pulmonary edema
- Stage 2: Progression into alveolar pulmonary edema
- Stage 3: Increased fluid accumulation and formation of foam
- Stage 4: Asphyxia

Diagnosis of Pulmonary Edema

Aside from the medical history and the clinical picture, in cases of alveolar pulmonary edema, moist rattling sounds are noticeable that, in part, are already audible without
using a stethoscope. Furthermore, chest X-rays and echocardiography may also be helpful.

**Differential Diagnosis**

Differential diagnosis includes, aside from the all-important distinction between cardiogenic and non-cardiogenic pulmonary edema, pneumonia, which is accompanied by fever and frequently by unilateral findings, as well as asthma (bronchial asthma) which is characterized, however, by dry rattling sounds.

![Image: Pneumonia in the Thoracic X-rays. By Hellerhoff, License: CC BY-SA 3.0](image)

**Therapy of Pulmonary Edema**

Immediate measures include, at any rate, a sitting position with the legs dangling in order to improve pulmonary vascular pressure. Sedation, administration of oxygen, as well as aspiration, are among the immediate measures as well.

In cases of cardiogenic pulmonary edema, preload reduction is necessary and measures to treat the cause must be taken, i.e. treating acute LV heart failure or treating cardiac arrhythmia.

![Image: Safety Needle. By BinimGarten, License: CC BY-SA 3.0](image)

When treating toxic or allergic pulmonary edema, inhaled corticosteroids are
indicated. Mechanical positive-pressure ventilation may also be an option. Therapy for pulmonary edema, caused by renal failure with overhydration, consists of dialysis. In cases of high-altitude edema, it is necessary to administer oxygen and move the patient to a lower altitude.

References


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