

## CHAPTER 1: CARDIAC PATHOPHYSIOLOGY

### I. OBJECTIVES

Upon completion of this chapter, the student will be able to:

- Understanding cardiovascular pathological mechanisms such as heart failure, arrhythmias, heart conditions, and congenital heart disease
- Analyze how the normal functioning of the heart and cardiovascular system deteriorates
- Understanding how compensatory mechanisms can transform into a pathology
- Develop the different strategies for diagnostic and therapeutic management

### II. GENERAL INFORMATION

#### II.1 CARDIAC ANATOMY AND CIRCULATION

The circulatory system is a **closed circuit** where the heart acts as the central pump, moving blood through a continuous network of arteries, veins, and capillaries. Located in the **middle mediastinum**, the heart is divided into two functional sides:

- **The right heart:** Handles **deoxygenated blood**. It receives blood from the systemic circulation (via the venae cavae and coronary sinus) and pumps it to the lungs.
- **The left heart:** Handles **oxygenated blood**. It receives blood from the lungs and pumps it to the rest of the body via the aorta.

#### 1. Internal chambers and valves

Each side consists of an **atrium** (receiving chamber) and a **ventricle** (pumping chamber), separated by specific valves to ensure one-way flow:

- **Left side:** Mitral valve (2 cusps) and Aortic valve (3 cusps).
- **Right side:** Tricuspid valve (3 cusps) and Pulmonary valve (3 cusps).

#### 2. Blood supply and innervation

- **Coronary circulation:** The heart muscle is nourished by the right and left coronary arteries and drained by coronary veins.
- **Electrical conduction system:** The heartbeat is initiated at the **Sinoatrial (SA) node** (RA), travels to the **Atrioventricular (AV) node**, and continues through the **Bundle of His** (right and left branches) to trigger ventricular contraction.
- **Autonomic control:** The heart is regulated by the **sympathetic nervous system** (T1–T5) for acceleration and the **parasympathetic nervous system** (Vagus nerve) for deceleration.

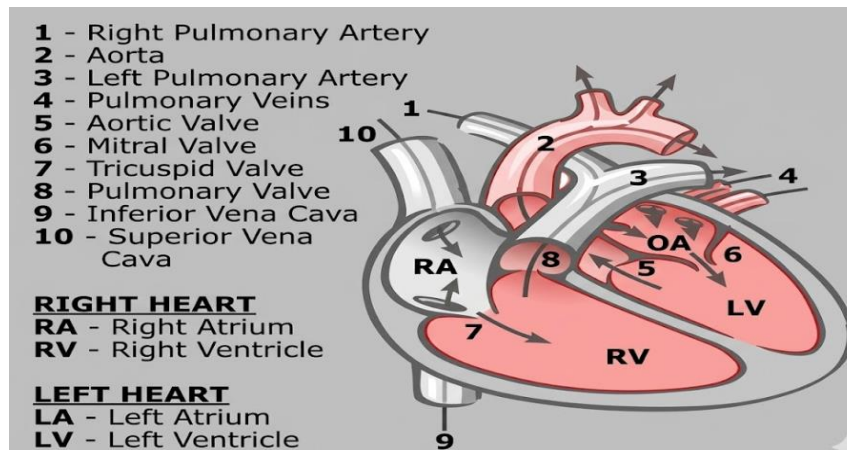


Figure 1: Anatomy of a healthy heart and its main components

The right atrium (RA) is connected to the venous system, the right ventricle (RV) to the pulmonary system. The left atrium (LA) receives oxygenated blood from the lungs. Finally, the left ventricle (LV) pumps blood throughout the body.

## II.2 CARDIAC PHYSIOLOGY AND THE BLOOD FLOW CYCLE

The cardiovascular system is an adaptive network designed to meet the body's metabolic demands through a continuous, two-sided cardiac cycle.

### 1. The right heart: deoxygenated (pulmonary) circuit

- **Reception:** The **Right Atrium** receives deoxygenated blood (high  $\text{CO}_2$ ) from the systemic circulation via the venae cavae and coronary sinuses.
- **Transfer:** Blood passes through the **tricuspid valve** into the **Right Ventricle**.
- **Delivery:** The ventricle pumps blood through the **pulmonary valve** into the pulmonary artery, leading to the lungs.
- **Gas Exchange:** In the pulmonary alveoli, blood releases  $\text{CO}_2$  and absorbs  $\text{O}_2$

### 2. The left heart: oxygenated (systemic) circuit

- **Reception:** Oxygen-rich blood returns to the **Left Atrium** via the pulmonary veins.
- **Transfer:** It is pumped through the **mitral valve** into the **Left Ventricle**.
- **Ejection:** **Myocardial contraction** forces blood through the **aortic valve** into the **aorta**.
- **Distribution:** The blood travels to the tissues via capillaries, where oxygen is delivered, before returning to the right atrium to restart the cycle.

## II.3 CARDIAC HISTOLOGY

The heart pump is a muscle, the myocardium. Its inner, or endocavitary surface, is lined with an endothelium called the endocardium. The outer surface is the epicardium, which constitutes the visceral layer of the pericardial sac (pericardium).

The functional unit of the heart is the cardiomyocyte. This striated cardiac muscle cell is separated from its neighbors by specialized intercellular junctions (gap junctions) that ensure simultaneous contraction. These cells are supported by specialized nodal tissue, which is responsible for initiating the heart's electrical activity. Additionally, Purkinje cells are differentiated cardiomyocytes specialized in transmitting nerve impulses throughout the ventricular conduction system (such as the bundle of His).

### III. HEART DISORDERS

#### III.1 Myocardial Disorders

##### III.1.1 Ischemic heart disease

This is a leading cause of death in developed countries. It involves the necrosis (death) of cardiomyocytes, which can lead to heart failure. The pathophysiology is based on coronary occlusion caused by atherosclerotic plaques (fatty deposits). A thrombus forms through the interaction of macrophages and coagulation cascades on these deposits. Key risk factors include high cholesterol, physical inactivity, obesity, hypertension, and diabetes.

##### III.1.2 Cardiomyopathies

These include myocardial degenerations (dystrophies) often caused by nutritional deficiencies (protein, glucose, potassium, vitamins), hypoxia, or intoxication. To a lesser extent, inflammation (**myocarditis**) plays a role, typically as a subacute or chronic reaction to primary degeneration. These conditions reduce cardiac functional capacity, leading to failure and rhythm disorders.

#### III.2 Disorders of the pericardium

Pericarditis is the inflammation of the pericardial layers. In veterinary medicine, it often accompanies diseases such as distemper, leptospirosis, or **Traumatic Reticuloperitonitis (TRP/RBI)** in cattle. This may lead to fluid effusion (containing leukocytes or fibrin), causing friction rubs or "splashing" sounds. While viral forms are often benign, bacterial pericarditis is serious and can lead to **cardiac tamponade** (compression of heart chambers causing circulatory failure). Diagnostic signs include muffled heart sounds, enlarged cardiac dullness, and **low voltage** on an ECG. Detection is confirmed via X-ray and ultrasound.

### III.3 Endocardial and valvular disorders

#### III.3.1 Endocarditis

Typically a bacterial infection (e.g., *Streptococci* or *Staphylococci*) of the endocardium. It is a grave condition that can cause valvular insufficiency or stenosis. Management requires immediate intravenous antibiotics and, if necessary, surgical valve replacement.

**III.3.2 Valvular Disorders:** Often a consequence of chronic bacterial endocarditis leading to fibrosis.

- **Stenosis:** The valve narrows, obstructing forward flow (e.g., blood has difficulty exiting the LV in aortic stenosis).
- **Insufficiency (Regurgitation):** The valve fails to close, allowing blood to flow backward.
- **Auscultation:** Turbulent blood flow creates **murmurs**. Stenosis typically creates "crescendo-decrescendo" sounds, while insufficiency often results in harsh, constant systolic murmurs.

## VI. HEART FAILURE

### VI.1 Definition

Heart failure (HF) is a clinical syndrome resulting from the heart's inability to maintain sufficient cardiac output to meet the metabolic and functional needs of the body's organs. This failure in pump function leads to decreased tissue perfusion and can manifest through clinical signs (abnormal heart sounds), anatomical changes (cardiac dilation), or hemodynamic abnormalities.

### VI.2 Etiology

HF is caused by abnormalities in **preload, afterload, or contractility (inotropy)**.

- **Primary decrease in contractility:** Caused by intrinsic heart conditions (myocarditis, ischemia, cardiomyopathies) or extracardiac factors (toxins like alcohol/lead, endocrine diseases like diabetes, or nutritional deficiencies).
- **Secondary decrease (Hemodynamic Overload):**
  - **Pressure Overload (↑ Afterload):** Hypertension or valvular stenosis.
  - **Volume Overload (↑ Preload):** Valvular insufficiency (regurgitation).
- **Decreased Filling:** Restrictive cardiomyopathy, pericardial disease (tamponade), or extreme tachycardia which shortens diastole.

### VI.3 Classification

- **Functional: Systolic dysfunction** (failure to eject) vs. **Diastolic dysfunction** (failure to fill).
- **Anatomical: Left-sided** (pulmonary congestion), **Right-sided** (systemic congestion), or **Global** (both).
- **Evolution: Acute** (sudden onset, e.g., MI or pulmonary embolism) vs. **Chronic** (stable but progressive, often post-infarction).

### VI.4 Clinical manifestations

- **Left Heart Failure:** Accumulation of blood in the pulmonary circuit leads to **dyspnea**, pulmonary edema, and **crackles (rattles)** upon auscultation.
- **Right Heart Failure:** Systemic stagnation leads to **jugular venous distension**, **hepatomegaly** (cardiac liver), and peripheral **edema** (swollen limbs).
- **Global Failure:** Potentiation of both sets of symptoms, including ascites and severe renal impairment.

### VI.5 Compensatory mechanisms

 The body uses neuro-hormonal pathways to maintain output:

1. **Sympathetic Nervous System:** Baroreceptors detect low BP, triggering **tachycardia** and vasoconstriction.
2. **RAAS (Renin-Angiotensin-Aldosterone System):** Renal hypoperfusion triggers renin release, leading to **Angiotensin II** (vasoconstriction) and **Aldosterone** (water/salt retention).
3. **Natriuretic Peptides (ANP/BNP):** Secreted by distended chambers to promote diuresis and vasodilation (the body's "counter-mechanism").
4. **Remodeling:** Concentric hypertrophy (from pressure) or eccentric hypertrophy/dilation (from volume).

### VI.6 Diagnostic and treatment

- **Gold standard diagnosis: Thoracic Echocardiography** and **BNP assays**.
- **Treatment goals:** Reduce workload and fluid overload.
  - **Diuretics:** Reduce volume (edema).
  - **ACE Inhibitors:** Block RAAS (reduce afterload).
  - **Beta-blockers:** Counteract chronic sympathetic stress (Note: avoid in acute phases).
  - **Inotropes:** Boost contractility (only when necessary).

## V. HEART RHYTHM DISORDERS

The study of heart rhythm involves identifying **R-waves**. This rhythm is characterized by two properties: the frequency of R-waves-expressed in **beats per minute (bpm)**-and their regularity. In the absence of pathology, the human heart rhythm is regular, with a frequency between **60 and 100 bpm** during the day and **40 to 80 bpm** at night. Deviations from these ranges may indicate a rhythm disturbance, though some irregularities, such as those caused by stress or exertion, are not necessarily pathological.

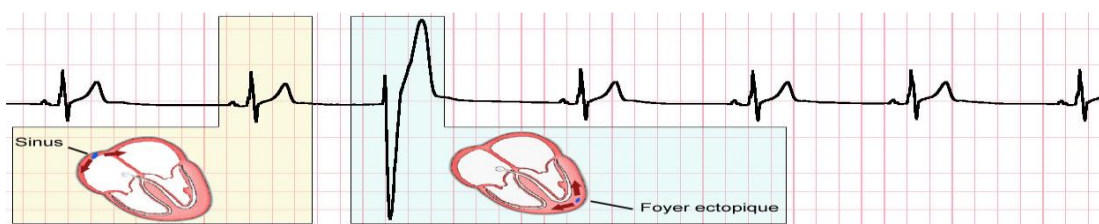
Electrocardiogram (ECG) analysis is based on examining successive heartbeats; while a single beat offers limited diagnostic value, variations in parameters across a recording are essential. Holter monitoring focuses on two key areas: heart rhythm (frequency/regularity) and wave morphology (amplitude, duration, and relative distance).

Arrhythmias result from impairments in the heart muscle's **excitability** or **conduction**. These may be functional (nervous influence) or organic (myocardial lesions) in origin.

### V.1 Extrasystoles

Extrasystoles are premature contractions occurring before the end of the normal resting period. They are triggered by ectopic stimulation in the sinoatrial (SA) node, atrioventricular (AV) node, or bundle of His.

- **Ventricular Extrasystole (VES):** These arise from a ventricular ectopic focus rather than the sinus cells. They are characterized by an absent P-wave and a widened QRS complex. They are typically followed by a compensatory pause (figure 2).



**Figure 2:** Ventricular extrasystole (VES)

### V.2 Fibrillation and Flutter

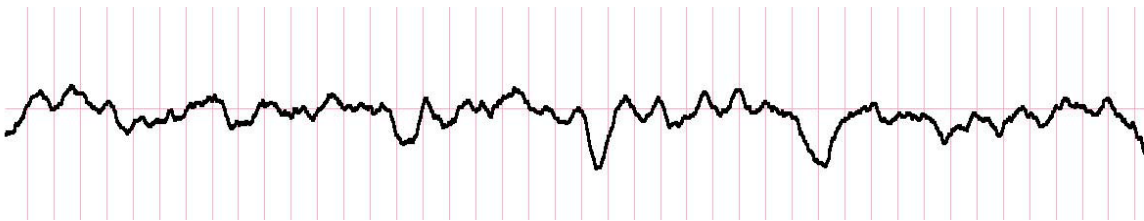
- **Atrial Flutter:** Characterized by rapid P-wave rates (up to 300 bpm or higher). The AV node often cannot conduct every impulse, leading to "blocks" (e.g., 2:1 or 3:1). A major risk is the formation of **emboli** due to turbulent atrial blood flow.

- **Atrial Fibrillation (AF):** The atria contract chaotically, producing "noise" on the ECG instead of distinct P-waves. While ventricles often fill normally, a common complication is **thrombus** formation, which can lead to a stroke (figure 3).



**Figure 3:** Atrial fibrillation

- **Ventricular Fibrillation:** A critical emergency where ventricles discharge desynchronized, causing a loss of effective pumping and oxygen supply. If not corrected via electrical cardioversion within three minutes, it leads to cardiac arrest (figure 4).



**Figure 4:** Ventricular fibrillation

### V.3 The Blocks

Blocks are conduction defects where electrical impulses are delayed or stopped.

- **Sinoatrial Block (SAB):** The impulse fails to reach the atrial muscle, resulting in absent P-waves and a slowed heart rate as the AV node takes over.
- **Atrioventricular Block (AVB):** Communication is interrupted between the AV node and the bundle of His.
  1. **First-degree:** Conduction is slow (prolonged PR interval), but every P-wave is followed by a QRS.
  2. **Second-degree:** Includes **Mobitz I (Wenckebach)**, featuring progressive PR lengthening until a beat drops, and **Mobitz II**, where beats drop regularly without PR prolongation.
  3. **Third-degree:** Complete dissociation between atrial and ventricular beats.

- **Bundle Branch Block:** Occurs when one branch of the bundle of His conducts less efficiently, causing non-simultaneous ventricular contraction (figure 5) and a widened R-wave (often with two peaks).



**Figure 5:** Branch block

*(Note here that the contraction of the two ventricles is therefore not perfectly simultaneous, which is observed by two successive peaks corresponding to the two ventricles. The total duration of the R wave is therefore longer here than during normal heartbeats).*