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SIMULATION APPROACH FOR EDUCATION AND TRAINING IN EMERGENCY

Cardiovascular emergencies

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Summary

Cardiovascular diseases are a common motive for patient presentation in an acute setting in emergency departments, as well as for admission to intensive care units due to aggravations of stable, chronic diseases. Conversely, non-cardiac diseases can have consequences on the cardio-circulatory system, leading to severe hemodynamic compromise and mimicking in terms of clinical signs, symptoms, or laboratory tests, the effects of primary cardiac diseases. At the same time, cardiovascular emergencies can have atypical presentations, which, in the absence of a high suspicion index, can lead to missing essential diagnoses. A systematic approach to cardiovascular symptoms is paramount for a prompt differential diagnosis, while guaranteeing patient safety.

Hypotension and shock

1. Introduction

Shock is a state of acute circulatory failure associated with inadequate utilization of oxygen by the tissues. Shock states are initiated by inadequate tissue perfusion, leading to tissue hypoxia, due to an imbalance between global transport of oxygen to tissues (DO_2) and global oxygen consumption (VO_2). It is frequently, but not invariably, associated with arterial hypotension, and, if left uncorrected, can lead to a severe, irreversible dysfunction of vital organs (multi-organ failure – MOF) and death.

2. Pathophysiology

The inadequate utilization of oxygen is, conventionally, classified as either due to an absolute decrease in oxygen delivery (DO_2), or an imbalance between an increased oxygen consumption (VO_2) and a normal, or even increased DO_2.
Essential pathophysiologic formulas with short glossary (table 1):

Table 1 - haemodynamic formulas for circulatory shock

DO2= CO x CaO2
CaO2 = (Hb x 1.38 x SaO2) + (0.0031 x PaO2)
VO2 = CO x (CaO2 - CvO2)
VO2 = CO x Hb x (SaO2 – SvO2)
SvO2 = SaO2 – VO2 / CO x Hb.
MAP = CO x SVR
CO= SV x HR
CO - cardiac output, CaO2 - the oxygen content of arterial blood (normal values 800-1,000 ml O2/min), CvO2 - the oxygen content of the mixed venous blood (normal values 200-250 ml O2/min), Hb - the value of haemoglobin in g per dl, PaO2 - the partial pressure of oxygen in arterial blood (normal values 16-20 ml O2/100 ml blood), SvO2 - the oxygen saturation of haemoglobin in the mixed venous blood, CaO2-CvO2 is called arterio-venous difference: Δ(a-v)O2, normal 4-5 ml O2/100 ml blood, MAP – mean arterial pressure, SVR – systemic vascular resistance, SV – stroke volume, HR, heart rate. *** Mixed venous blood is considered to be pulmonary artery blood only and can only be measured if the patient has a pulmonary arterial catheter (PAC).**

These pathophysiologic formulas can explain almost all types of shock and the diseases which cause them. It is essential to recognize that cardiac output is not the only determinant of tissue perfusion, as DO_2 is also dependant on Hb, and SaO2. At the same time, the determinants of cardiac output include heart rate and stroke volume, which can be altered by preload, contractility, and afterload.
The complex interaction between these determinants of tissue perfusion is different in various clinical circumstances.

3. Types of shock and aetiology

There are 4 classically defined types of shock, and those are **hypovolemic shock, cardiogenic shock, obstructive shock, and distributive shock.**
The first three types have in common that they are all types of **hypodynamic shock,** which means that there is a low CO and a high SVR. In all these cases, there is a low SvO_2 and a high Δ(a-v)O2.
The fourth is a **hyperdynamic shock**, of which the prototype is septic shock. In this case, there is an increased CO and a decreased SVR, while DO_2 is inadequate, although frequently much increased as compared to basal needs.
Hypovolemic shock appears due to a direct loss of circulating blood volume (internal and/or external), which leads to an acute loss in preload and stroke volume, low cardiac output, and low tissue perfusion. Macro-haemodynamically, it is characterised by normo- or hypotension (in later cases), tachycardia, low cardiac filling pressures due low preload and a compensatory increased afterload (high SVR). It can be haemorrhagic and non-haemorrhagic. The most common causes are listed in table 2.

Table 2 - common causes of hypovolemic shock

Hypovolemic shock	**Haemorrhagic**	- **Trauma – external or internal bleeding** - **Gastro-intestinal bleeding (oesophageal varices, gastro-duodenal ulcers, fistulas)** - **Intra-operative bleeding** - **Ruptured ectopic pregnancy** - **Peri-partum bleeding** - **Bleeding diathesis** - **Retroperitoneal bleeding (spontaneous, ruptured aortic aneurysm)** - **Iatrogenic bleeding**
	Non-haemorrhagic	- Gastro-intestinal losses – vomiting, diarrhoea - Third space accumulation of fluid – peritonitis, pancreatitis, ascites due to liver cirrhosis, bowel obstruction - Renal losses – diabetic ketoacidosis, adrenal failure, diabetes insipidus, iatrogenic induce diuresis - Skin losses – burns

Cardiogenic shock, classically, is characterized by tissue hypoperfusion due to a low stroke volume and cardiac output caused by an intrinsic deficit of the cardiac pump. It can occur due to diseases of the cardiac muscle, when it is called *cardiomyopathic*, due to heart rhythm disturbances (*arrhythmogenic*) or due to defects of the heart not related to cardiac contractility (*mechanical*). The most common causes of cardiogenic shock are listed in table 3.

Table 3 - common causes of cardiogenic shock

Cardiogenic shock	**Cardiomyopathic**	- **Acute coronary syndrome (myocardial infarction) with acute heart failure** - **Acute decompensation of chronic heart failure** - **Post cardiac compressions for resuscitation** - **Myocarditis (viral, autoimmune, etc.)** - **Drug-induced or toxic** - **Post-cardiotomy (cardiac surgery)** - **Myocardial contusion** - **Intrinsic myocardial depression (late septic shock, prolonged severe hypoxemia, severe acidosis)**

	Arrhythmogenic	- Tachyarrhythmia
		  o Atrial tachycardias – fibrillation, flutter
		  o Ventricular tachycardias
		- Bradyarrhythmia – second- or third-degree heart blocks

	Mechanical	- Severe valve stenosis - Severe valve insufficiency (acute – ruptured papillary in MI) - Dynamic stenosis in the left ventricle - Acute ventricular septal wall defect

A more recent classification of cardiogenic shock shows that it is a polymorphic syndrome, with several phenotypes, not always fitting to the classical hypodynamic paradigm.
These can be:

- Classical cardiogenic shock - as described, low CO, high SVR, high filling pressures;
- Euvolemic cardiogenic shock - low CO, high SVR, normal filling pressures;
- Vasodilatory cardiogenic shock – low CO, low or normal SVR, high filling pressures (this is common in mixed shocks or cardiogenic shock with severe systemic inflammation).

Obstructive shock is caused by a mechanical (intra-cardiac or extra-cardiac) obstruction to cardiac filling, which leads to a low SV, CO, and tissue perfusion. Intracardiac pressures are high. It can also occur due to an obstruction in the pulmonary vasculature, in which case it is associated with acute cor pulmonale and severe right heart failure. A list of causes is available in table 4.

Table 4 - causes of obstructive shock

Obstructive shock	**Mechanical (extra-cardiac and intra-cardiac)**	- **Pericardial tamponade (acute aortic dissection, uremic pericarditis, infectious pericarditis etc.)** - **Constrictive pericarditis** - **Restrictive cardiomyopathy** - **Tension pneumothorax** - **Haemothorax (can also lead to severe hypovolemia)** - **Severe dynamic hyperinflation** - **Abdominal compartment syndrome**
	Pulmonary vascular	- Massive pulmonary embolus (PE) - Severe pulmonary hypertension - Massive venous air embolus

Distributive shock is characterized by a severe peripheral vasodilation, leading to very low SVR, a high CO, but low tissue perfusion of vital organs due to regional maldistribution of flow. The prototype of distributive shock is septic shock. In septic shock, there is a high DO_2, but a much higher VO_2, leading to an acute imbalance. It is also associated with normal or low cardiac filling pressures, high or normal SvO_2, low arterio-venous oxygen gradient and altered oxygen extraction at a tissue level. However, septic shock is not the only kind of distributive shock, and this pathophysiological phenotype can be encountered in other clinical settings, with variations specific to aetiology. Other, non-septic causes of distributive shock can be read in table 5.

Table 5 - common causes of distributive shock

Distributive shock	**Septic**	- **Bacterial (gram-negative or gram-positive microorganisms)** - **Fungal** - **Viral** - **Parasitic** - **Mycobacterial**

	Non-septic	- Non-septic inflammation
		  ○ Burns
		  ○ Trauma
		  ○ Pancreatitis
		  ○ Post-myocardial infarction (see above cardiogenic shock phenotypes)
		  ○ Post cardiac arrest syndrome
		- Anaphylactic shock
		- Neurogenic shock (spinal trauma, high spinal anaesthesia, traumatic brain injury)
		- Toxic (iatrogenic or non-iatrogenic – vasodilatory drugs)
		- Peri-cardiopulmonary by-pass

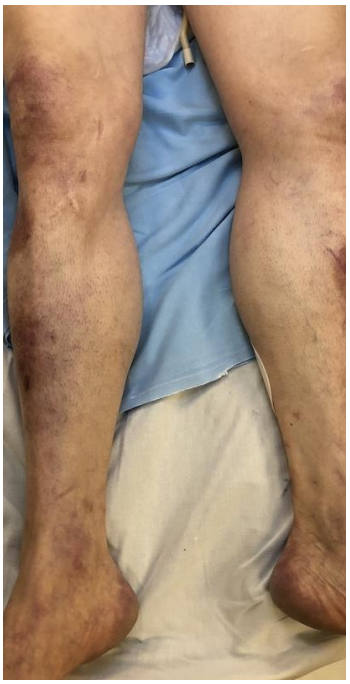
4. Clinical features of shock

Regardless of aetiology, patients with shock have in common low tissue perfusion, manifesting at all organic levels. Classically, there are three "organic windows" easily accessible to the clinician to assess tissue perfusion: the skin, the kidney, and the brain.

Non-specific clinical manifestations that almost invariably appear in shock patients are:

- Arterial hypotension – it is defined as a systolic blood pressure (SBP) of less than 90 mmHg or MAP of less than 65 mmHg, or a drop of more than 40 mmHg comparing to the baseline. It is not mandatory in the diagnosis of shock, but most patients, regardless of the type of shock, are also hypotensive, albeit hypotension is not the first clinical manifestation in the clinical history. Nonetheless, patients can be hypotensive in various clinical scenarios, without shock.
- Tachycardia – it is ubiquitous in most types of shock (exception bradyarrhythmia), as it is a compensatory mechanism in the presence of low tissue perfusion and/or low cardiac output (see CO formula above). It can be masked by chronic negative chronotropic medication.
- Skin perfusion alterations – in most cases of hypodynamic shock patients have cool, clammy skin, due to centralization of circulation; this is also associated with discolorations (mottling, which can be graded 1 to 5 and is associated with severity of shock – see figure 1 for mottled skin), increased capillary refill time (more than 3 seconds).

Figure 1 - grade 5 skin mottling in a cardiogenic shock patient



However, a warm, red-coloured skin, does not exclude shock, as this is a common presentation of septic shock and other types of distributive shock.

Also, mottling and temperature alterations are not pathognomonic of shock. It can also be present in other causes of regional hypoperfusion, such as acute or chronic limb ischemia.

- Mental status alterations – a manifestation of brain hypoperfusion which can often be the first clinical manifestation in an otherwise stable patient. It can be present as mild modifications in mood and unexplained anxiety, but can progress to overt agitation, delirium, stupor, and coma.
- Oliguria – autoregulation maintains unchanged renal blood flow as MAP varies between 60 and 160 mmHg. A drop below 60, as often encountered in shock, translates to oliguria, but this is not the only mechanisms of kidney injury and oliguria – others include centralization of circulation to vital organs, direct injury, and severe hypovolemia.
- Hyperlactatemia – lactic metabolic acidosis (defined as lactate greater than 2 mmol/L with pH lower than 7.35) is an essential element in the diagnosis of shock and is mandatory to

diagnose septic shock in the most recent recommendations of the intensive care societies (Sepsis-3). Hyperlactatemia is a reliable marker of tissue hypoperfusion and anaerobic metabolism and has been used to diagnose and monitor response to treatment in shock patients, due to its correlation with mortality.

- Aetiology-specific features – as seen above, there is a pleiotropism of shock causes and manifestations. A targeted medical history can help direct diagnosis and start early adequate treatment of these patients.

5. Initial approach to the patient with hypotension and shock

In the presence of undifferentiated severe hypotension, the patient should be suspected of shock and approached using an ABCDE algorithm first:

- Assess airway, breathing, circulation and conscious state
- Secure airway and start CPR if appropriate
- Institute monitoring (ECG, SpO2, ETCO2 as required)
- Ask for help
- Obtain IV access and start fluid resuscitation
- Obtain 12 lead ECG, acid-base balance test (ABG), with Hb monitoring and full blood chemistry
- Perform point-of-care ultrasound using a guided algorithm – FOCUS, POCUS, RUSH, eFAST, as per personal training and clinical circumstance.
- Use echo results to advance on clinical management and diagnosis

6. Differential diagnosis

The patient presenting with clinical features of shock should be evaluated, depending on history, for the most common causes of shock, warranting urgent life-saving interventions:

- Anaphylactic shock – hypotension with stridor, oral and facial oedema, history of allergies or exposure to allergens
- Cardiogenic shock due to myocardial infarction – hypotension with chest pain, respiratory failure, typical ECG modifications
- Cardiogenic shock due to acute valve insufficiency - hypotension with chest pain, respiratory failure, typical auscultation, or echocardiographic findings
- Acute aortic dissection – severe chest pain, either anterior or back pain, can present with tamponade or MI
- Pulmonary embolism
- Tension pneumothorax
- Life-threatening arrhythmias
- Septic shock - hypotension with warm, erythematous skin and suspicion of infection
- Obstructive shock due to pericardial tamponade – hypotension with dyspnoea, tachycardia, congestive jugular veins, muffled heart sounds at auscultation, pulsus paradoxus, suggestive POC echocardiography
- Severe haemorrhage, either traumatic or non-traumatic
- Adrenal crisis

7. Treatment

The objective of treatment of patients with shock, regardless of aetiology, is to restore tissue perfusion, while, simultaneously, addressing the cause of shock.

To restore tissue perfusion, it is most often required to increase cardiac output by restoring blood volume, increasing peripheral vascular resistance and blood pressure, increasing stroke volume by increasing myocardial contractility, and optimizing preload and afterload through advanced hemodynamic monitoring.

Volume expanders which are commonly used in the resuscitation of shock patients are classified into 2 main categories: crystalloids and colloids.

Commonly used crystalloids are:

- Normal saline – NaCl 0.9%
- Lactated Ringer solution
- PlasmaLyte
- Sterofundin ISO

Colloid solutions

- Starches – most starch-based solutions have been disproved in the treatment of shock and are not currently recommended, due to adverse effects
- Gelatins
- Human albumin

Vasoactive medication used in the treatment of shock, depending on cause and haemodynamic objective are listed in table 6.

Table 6 - Vasoactive medication used in shock

Class	Molecule	Action	Effects
Catecholamines	Norepinephrine	α-1, β-1 agonist	increase SVR, CO
	Epinephrine	α-1, β-1 agonist	increase SVR, CO, HR
	Dobutamine	β-1, β-2 agonist	increase CO, decrease SVR, increase HR
	Dopamine	can act as α-1, β-1 and dopaminergic agonist at different concentrations	increase CO, SVR, increase HR
Non-adrenergic drugs	Milrinone	Phosphodiesterase 3 inhibitor	increase CO, decrease SVR, increase HR
	Levosimendan	Calcium sensitiser K channel opener	increase CO, decrease SVR, neutral or increase HR

Vasopressin and analogues	Vasopressin	ADH receptor	Increase SVR, neutral HR, neutral or decrease CO
	Angiotensin	Vasopressin receptor	Increase SVR, increase HR, neutral or decrease CO

Septic shock

- ABCDE approach
- Give oxygen if SpO2 <94%
- Cultures and broad-spectrum antibiotics in the first hour
- Volume resuscitation with crystalloids, preferably balanced, 30 ml/kg in the first 3 hours
- Invasive monitoring using arterial line, central line, and dynamic measures of fluid responsiveness
- Monitor hourly urine output and lactate
- ICU admission is mandatory
- If persistent hypotension after fluid resuscitation, initiate vasopressor
    - Norepinephrine should be preferred over other agents
    - Second vasopressors if refractory hypotension – vasopressin, epinephrine
    - Dobutamine can be considered if cardiac dysfunction is present

Cardiogenic shock

- ABCDE approach
- Give supplemental oxygen if SpO2 <94%
- Consider non-invasive ventilation
- Obtain iv access and have CPR cart nearby
- Continuous monitoring of ECG, BP, SpO2
- Consider early intubation if severe respiratory failure, severe shock, or alteration of mentation
- 12-lead ECG and look for signs of ischaemia
- Blood work including ABG, electrolytes, full blood count and cardiac enzymes (high sensitivity troponin preferred)
- Focused echocardiography to look for causes and establish a hemodynamic phenotype
- Review the differential diagnosis
- If ischaemia present, give aspirin 325 mg
- Start norepinephrine for MAP >65 mmHg
- Start dobutamine if after normal MAP, there is significant cardiac dysfunction
- **If appropriate, ask for early reperfusion therapy or interventional cardiology for definitive diagnosis**
- **Consider mechanical circulatory support**

Anaphylactic shock

- ABCDE approach
- Consider early intubation, as oedema can lead to difficult airway; be prepared for cricothyrotomy
- Give oxygen
- IV access and volume resuscitation with 20 ml/kg crystalloid
- Give epinephrine
- If symptoms do not recede, start epinephrine infusion
- Insert arterial line
- If no response, consider alternative agents
- Start adjunctive therapy when the patient is stabilized

Epinephrine can be given intravenously, intramuscularly or in nebulization, depending on resources and indication.
IM bolus should be of 0.3-.5 mg (1:1000 concentration), IV bolus 5-20 mcg (1:100000 concentration) and continuous iv infusion can be administered at a rate of 1-20 mcg/min.
If poor response to standard therapy, the following can be attempted:

- Epinephrine 100 mcg iv bolus
- Norepinephrine infusion 0.1 mcg/kg/min
- Vasopressin 0.01-0.04 units/min or 2 U bolus
- Glucagon 1 mg iv over 5 min
- Methylene blue 1.5-2 mg/kg iv bolus and continuous infusion if refractory

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Hypertensive emergencies

1. Introduction and definition

Patients with blood pressure (BP) values of more than 180/120 mmHg and signs of acute, ongoing, end-organ damage are said to have "hypertensive emergencies". Acute injuries can occur to the heart, brain, and the microvasculature and are associated with high morbidity and mortality, if not managed appropriately.

However, these values cannot be held as absolute for diagnosing hypertensive emergencies, as in a previously normotensive patient, a sudden rise to lower BPs, such as 160/100, can cause severe injury. This can occur in patients with eclampsia, pheochromocytoma, or drug-induced acute hypertension.

2. Pathophysiology

The autoregulation of blood flow is essential for maintaining adequate oxygenation of vital organs, and this is performed via local and general reflexes between set BP values.

Cerebral autoregulation, for example, occurs between a MAP of 50 and 150 mmHg, in the healthy adult. Between these values, blood flow is maintained at a constant level. Below 50 mmHg, a small drop in pressure leads to a significant reduction in flow, and to organ hypoperfusion. Conversely, on the high end of the curve, small changes in MAP cause important augmentations of flow, leading to cerebral oedema and other severe complications.

The autoregulatory curve is not identical in all patients. In chronic hypertensive patients, it shifts to the right, thus allowing a higher tolerance to high BPs before developing cerebral oedema. However, this shift also exposes patients to a risk of hypoperfusion at BP values that would otherwise seem normal. Caution should be exercised when treating these patients to avoid such complications.

3. Aetiology

The most common cause of acute severe hypertension is nonadherence to chronic anti-hypertensive medication, causing acute rebound. Other clinically relevant causes of hypertensive emergencies are listed in table…

Hypertensive emergencies
Nonadherence to treatment (acute on chronic)
Drug intoxications • **Illicit drugs – cocaine, amphetamines** • **Non-illicit drugs – NSAIDs, corticosteroids**
Head injury
Acute stroke • **Ischaemic** • **Haemorrhagic**
Acute heart failure
Acute glomerulonephritis
Preeclampsia
Pheochromocytoma
Scleroderma crisis
Thyrotoxicosis

In hospitalised patients

- **Urinary retention**
- **Pain**
- **Excessive fluid infusions**

4. Clinical presentation and management

Patients with high BP, even at extreme values, who do not have target-organ damage, can be asymptomatic or present with non-specific symptoms, such as headache, dyspnoea, dizziness, light-headedness.

When evaluating a patient with high BP, active efforts should be undertaken to identify proof of acute target-organ damage, including the brain, heart, large vessels, kidneys, and microvasculature.

The history and physical examination should search for one or more of the following as both potential causes and/or markers of severity:

- Head injury
- Neurologic symptoms
  - Generalized – agitation, delirium, seizures, visual disturbances
  - Focal – potentially caused by a stroke
- Nausea and vomiting - could point to high intracranial pressure (ICP) due to cerebral oedema
- Fundoscopic examination – retinopathy can be associated with encephalopathy
  - Fresh flame haemorrhages
  - Exudates
  - Papilledema
- Chest pain or discomfort – could point to myocardial ischaemia or aortic dissection
- Acute back pain – aortic dissection
- Dyspnoea
- Pregnancy – could develop preeclampsia or eclampsia
- Recent discontinuation of antihypertensive drugs (e.g., clonidine)
- Use of drugs such as cocaine, amphetamines, phencyclidine

If after clinical examination, a hypertensive emergency has been diagnosed or is suspected, the following steps should be performed:

- Obtain IV access
- Institute continuous monitoring
- Request additional tests:
  - 12-lead ECG
  - Chest X-ray
  - Full blood count, blood chemistry, including cardiac enzymes and electrolytes
  - Urinalysis, including pregnancy test, if appropriate
  - Point-of-care ultrasound
- Transfer to ICU and insert arterial line

- After an exhaustive review of potential causes, start IV treatment immediately with antihypertensive drugs
    - Individualize treatment according to underlying disease
- If neurological symptoms are present, request immediate head CT
- If an aortic dissection is suspected, request emergency contrast-enhanced CT or transoesophageal echocardiography, with adequate pain control and sedation

5. Treatment

Parenteral antihypertensive medication is mandatory in hypertensive emergencies, as it is fast-acting and can be more easily titrated to achieve the desired clinical effect, but also to avoid "over-shooting", and thus potentially causing hypoperfusion or target-organs.
A summary of available medication to be administered in acute severe hypertension is available in table 7.

Table 7 - Medication used for treating severe hypertension

Drug	Dose	Onset	Duration	Caution
Nicardipine	start at 5 mg/h, increase by 2.5 mg/h every 15 min until goal BP, then decrease to 3 mg/h	5-15 min	30-40 min	reflex tachycardia
Clevidipine	start at 2 mg/h, increase every 2 min with 2 mg/h until goal BP, max usual dose 16 mg/h	2-4 min	5-15 min	Nausea, A Fib, lipid formulation (potential allergens, max 1000 ml/24 h)
Urapidil	12.5-25 mg bolus, 5-40 mg/h afterwards	3-5 min	4-6 h	Aortic coarctation, A-V shunts
Labetalol	initial bolus 20 mg iv, 20 mg boluses every 10 min until goal BP (max dose 300 mg)	5-10 min	2-4 h	High degree AV block, heart failure, asthma, can cause bronchoconstriction and foetal bradycardia
Esmolol	250-500 mcg/kg loading dose over 1 min, 25-50	1 min	10-30 min	Heart failure, bradycardia, infusion-site pain, can cause bronchospasm

	mcg/kg/min infusion			
Hydralazine	5 mg iv over 1-2 min, 5 mg every 20 min until goal BP, max dose 30 mg	15 min	1-4 h	Can cause sudden hypotension, tachycardia, aggravation of angina
Nitrolycerin	start at 5 mcg/min, up-titrate until BP goal, maximal dose 200 mcg/min	<1 min	5-10 min	Increased intracranial pressure, headache, reflex tachycardia, tachyphylaxis, do not associate with phosphodiesterase inhibitors
Nitroprusside		<1 min	1-5 min	Cyanide intoxication, Increased intracranial pressure, reflex tachycardia
Phentolamine	5 mg iv bolus every 10 min until goal BP	1-2 min	10-30 min	Coronary artery disease

As a "rule of thumb", it is advised to avoid decreasing BP too quickly or too much, as ischemic lesions can occur due to the shift of autoregulation curves to the right. BP targets in most hypertensive emergencies are as follows:

- A reduction of MAP by 10-20% in the first hour
- A further reduction of MAP by 5-15% in the following 23 hours

There are, however, particularities related to the individual clinical setting, which will be summarized further.

6. **Therapeutic approaches in specific clinical scenarios**
   - *Diffuse microvascular injury (malignant hypertension)*
     - Decrease BP by 20-25% during first h and to 160/100 by 2-6 h
     - Use labetalol, nicardipine, nitroprusside
   - *Hypertensive encephalopathy*
     - Decrease BP by 20% during first h and to 160/100 by 2-6 h (no more than 25% at 24 h)
     - Use labetalol, nicardipine, clevidipine, nitroprusside, urapidil, avoid hydralazine
   - *Acute intracerebral haemorrhage*

    - If systolic BP is 150-220 mmHg, decrease to 140-150 mmHg within 1 h
    - If large haematoma and evidence of high ICP, SBP target should be 140-180 mmHg
    - Use labetalol, nicardipine, clevidipine, nitroprusside, urapidil, avoid hydralazine
- *Acute ischaemic stroke*
    - If thrombolysis, target SBP to <185/110 mmHg before administration and during first 24 h
    - If no thrombolysis and no target organ dysfunction, no intervention is indicated in the first 48-72 h at BP <220/120 mmHg
    - At SBP ≥220/120 mmHg or target-organ dysfunction, decrease SBP by 15% within 1 h.
    - Use labetalol, nicardipine, clevidipine, nitroprusside, urapidil, avoid hydralazine
- *Acute coronary syndromes*
    - Target SBP <140 mmHg at 1 h, keep diastolic BP >60 mmHg
    - Use nitroglycerin, labetalol, esmolol (caution for severe LV failure), avoid hydralazine; consider ACE inhibitors
- *Acute heart failure*
    - Target SBP <140 mmHg at 1 h
    - Use nitroglycerin, nitroprusside, loop diuretics, consider ACE inhibitors, caution with beta-blockers
- *Aortic dissection*
    - Target SBP <120 mmHg and heart rate <60/min at 20 min
    - Use esmolol, nicardipine, nitroprusside, nitroglycerin; beta-blockers and vasodilators should be associated.
    - Use opioids for pain control
- *Pre-eclampsia*
    - Do not treat unless SBP >160/110 mmHg, target SBP 140-160 mmHg, DBP 90-110 mmHg
    - Do not reduce MAP more than 25% within 2 h
    - If severe hypertension, use magnesium sulfate 4-6 g iv bolus and 1-2 g every hour for seizure prophylaxis
    - Use nicardipine, labetalol or hydralazine, avoid ACE inhibitors, nitroglycerin (unless pulmonary oedema).
- *Cocaine/amphetamine toxicity*
    - Treat agitation with benzodiazepines
    - Give antihypertensives if evidence of target organ damage
    - Use phentolamine, nitroglycerin if coronary ischemia, avoid beta-blockers

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Arrhythmias

1. **Introduction and definitions**

Cardiac arrhythmias represent a group of conditions in which the electrical activity of the heart is perturbed. The electrical anomalies result in either a slowing (bradycardia), acceleration (tachycardia), and/or an alteration of the regularity of the heart rhythm.
The aetiology of such alterations can be complex, and any modifications in heart rhythm should give rise to suspicions regarding a potentially more severe underlying diseases (ischaemic or non-ischaemic).
The object of this section is not an exhaustive approach to cardiac arrhythmias, from a cardiologist physician's perspective, but rather a systematic, concise pathway towards diagnosing and treating arrhythmias which pose an immediate risk to patients and thus represent medical emergencies.

2. Classification

The most straightforward classification is into tachy- and bradycardias. Classically, a patient is considered tachycardic at heart rates (HR) superior to 100 beats per minute (BPM), and bradycardic at HR inferior to 50 BPM.
Furthermore, tables 8 and 9 show anatomical classification of arrhythmias, based on the origin of the electrical disturbance in the heart.

Table 8 - classification of tachyarrhythmias

Supraventricular (SVT)	**Atrial extrasystoles (premature beats)**
	Sinus tachycardia
	Atrial focal tachycardia
	Atrial flutter
	Atrial fibrillation (AF)
AV junction	AV nodal re-entrant tachycardia
	Junctional tachycardia
	AV re-entrant tachycardia
Ventricular	Ventricular extrasystoles (premature beats)
	Ventricular tachycardia (VT) Sustained Non-sustained
	Ventricular fibrillation (VF)

Table 9 - classification of bradyarrhythmias

Sinus node dysfunction	**Sinus bradycardia**
	Sinus pause
	Sinoatrial blocks
	Chronotropic incompetence
AV conduction blocks	Grade I AV block
	Grade II AV block type 1 – Wenckebach type 2
	Complete AV block (grade III)

Some arrhythmias, although not to be taken lightly, do not warrant immediate medical treatment and can be investigated in an outpatient setting. Such examples are atrial or ventricular

extrasystoles, or asymptomatic sinus tachycardia or bradycardia. However, symptomatic bradycardia, AF with rapid rates, sustained VT, are medical emergencies and should be treated as such. At extremes, patients can develop malignant arrhythmias and require cardio-pulmonary resuscitation, according to adult life support protocols.

3. Diagnosis

3.1. Clinical signs and symptoms

Patients may present with mild or moderate symptoms, such as palpitations, dizziness, light-headedness, or anxiety. More severely symptomatic patients present can develop syncope or dyspnoea, which are markers of significant hemodynamic alterations.

The most severe form of clinical presentation of arrhythmias is sudden cardiac death, which is most often caused by VT or VF. It is associated with structural heart disease, such as ischaemic heart disease, acquired or congenital cardiomyopathies, channelopathies (long QT syndrome, Brugada syndrome, catecholaminergic ventricular tachycardia).

In bradyarrhythmia patients, there is a decrease in cardiac output in all patients, due to low HR, but this can be aggravated by pre-existing structural heart conditions making heart unable to compensate by increasing stroke volume, such as valvular regurgitations, or cardiomyopathies. Clinical manifestations can range from dizziness, tiredness, syncope, to overt markers of tissue hypoperfusion and signs of cardiogenic shock (see above).

3.2. 12-lead electrocardiogram

The ECG remains essential for the differential diagnosis of arrhythmias in the emergency setting. A single-lead rhythm strip (easily obtainable from 3-lead monitors) is often inadequate, and for adequate diagnosis the 12-lead ECG is mandatory.

Based on the 12-lead ECG, tachycardias can be classified into wide complex (QRS duration >120 milliseconds – fig 2) and narrow complex (QRS ≥120 milliseconds). Based on this classification, important inference on the origin of the disturbance can be made, as narrow QRS arrhythmias are always SVTs, and are often benign, not requiring aggressive, immediate treatment, except for severely symptomatic patients at extreme heart rates. However, wide QRS tachycardias can be VTs or an SVT with conduction disturbances (branch blocks) or pre-excitation. Wide complex tachycardias are almost always a medical emergency and should be treated in a department with continuous monitoring and/or able to manage emergency patients.

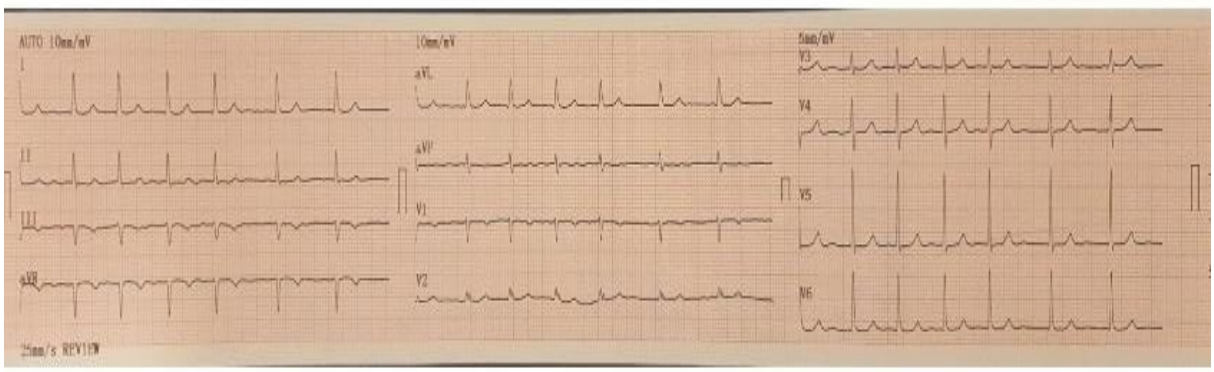


Figure 2 - 12-lead ECG showing AF

3.3 Other investigations

If the patient is unstable and requires immediate intervention, the clinical picture and the 12-lead ECG are enough to make treatment decisions. In stable patients, further investigations can be ordered, and patients should be referred for expert advice by a cardiologist.

4. Treatment

Treatment of tachycardias can have a double objective: rate control and/or conversion to sinus rhythm, depending on type of arrhythmia and hemodynamic impact.
This can be obtained either pharmacologically, vagal manoeuvres, or by electrical cardioversion.

4.1 Medication

Commonly used medication in arrhythmia treatment is listed in table 10, presenting a summarized version of the updated (2018) Vaughan Williams classification of cardiac antiarrhythmic drugs.

Table 10 - Updated Vaughan Williams classification of antiarrhythmic drugs

Class	Action	Drugs	Mechanism of action	Uses
0	HCN channel blockers	Ivabradine	Reduction in sinoatrial node automatism	Stable angina, chronic heart failure
I	Na+ channel blockers			
Ia	Intermediate dissociation	Quinidine, disopyramide, procainamide	Reduction in ectopic automatism and accessory pathway conduction Increase in refractory period	SVT, AF, VT, VF
Ib	Rapid dissociation	lidocaine	Reduction in ectopic ventricular automatism Reduced re-entrant tendency	VT, VF, particularly after MI
Ic	Slow dissociation	Flecainide, Propafenone	Reduction in ectopic automatism Reduced re-entrant tendency Slowed conduction and reduced excitability	SVT (atrial tachycardia, atrial flutter, AF) VT resistant to other treatments in the absence of structural heart disease
Id	Late current	Ranolazine	Decrease in action potential recovery time	Stable angina, VT
II	Autonomic inhibitors and activators			

IIa	beta blockers	Nonselective: carvedilol, propranolol Selective: atenolol, bisoprolol, metoprolol, esmolol, landiolol	Reduction in sinoatrial node and AV node automatism Reduction in ectopic automatism Reduced sinoatrial node (SAN) re-entry reduced AV node re-entry	Sinus tachycardia, SVT (AF, atrial flutter) Rate control in AF and VT Ventricular extrasystoles
IIb	beta agonists	Nonselective - isoproterenol	Increased escape ventricular automatism	Complete AV block before pacemaker implantation Drug-related bradycardia
IIc	Muscarinic M2 receptor inhibitors	Atropine, hyoscine, scopolamine	Increase in SAN automatism Increase in AV node conduction	Symptomatic sinus bradycardia AV node or conduction block
IId	Muscarinic M2 receptor activators	Carbachol, pilocarpine, digoxin	Reduction in SAN automatism Reduced SAN re-entry Reduced AV node conduction	Sinus tachycardia SVT
IIe	Adenosine A1 receptor activators	Adenosine	Reduction of SAN automatism Reduction of AV node conduction	Acute termination of AV node tachycardia Differentiation of sinus from atrial tachycardia
III	K+ channel blockers and openers			
IIIa	voltage dependent K+ channel openers	Amiodarone, dronedarone, dofetilide, ibutilide, sotalol, vernalakant	Increase in action potential recovery time, refractory period Amiodarone slows sinus rhythm Vernalakant has atrium specific actions	VT in patients without structural heart disease, tachyarrhythmias with WPW syndrome AF VF Vernalakant for immediate conversion of AF

IIIb	metabolically dependent K+ channel openers	Nicorandil	Increase in action potential recovery time Decreased re-entry tendency	Stable angina
IV	Ca++ handling modulators			
IVa	surface membrane Ca++ channel blockers	Verapamil, diltiazem	Reduction in AV node conduction Reduction in after depolarisation-induced triggered activity	SV arrhythmias VT without structural heart disease Rate control in AF
IVb	intracellular Ca++ channel blockers	Flecainide, propafenone		
V	Mechanosensitive channel blockers	In clinical trials		
VI	Gap junction channel blockers	In clinical trials		
VII	Upstream target modulators	ACE inhibitors Angiotensin receptor blockers Statins Omega-3 fatty acids	Reduction of structural and electrophysiological remodelling in various settings	

4.2 Cardioversion

Cardioversion is the conversion of an electrical disturbance of the heart by administering either a drug, or a direct electrical current, or both. Successful cardioversions restore normal sinus rhythm, reduce heart rate, and restore cardiac output, thus alleviating symptoms. Drugs used to convert or to facilitate electrical cardioversions are mentioned in table…

Electrical cardioversion is done using either external defibrillators or internal defibrillators, in patients with previously implanted such devices. External direct current administration terminates arrhythmias by delivering a shock, which causes depolarisation pf the excitable tissue, thus making it unavailable (due to the physiological refractory period) for propagation or sustainment of the arrhythmia. When the shock is synchronised with the QRS complex, the procedure is called cardioversion, while non-synchronised delivery is called defibrillation.

Electrical cardioversion procedure – how to prepare

- Continuous monitoring, including continuous ECG, BP, pulse, SpO2, ETCO2
- Intravenous line in place
- Equipment in place for airway management – oxygen, suction, intubation materials
- Standard medication for advanced life support

Electrical cardioversion procedure – how to deliver shock

- Placement of paddles/pads – while some physicians prefer the anterior-posterior placement of pads for AF, the most common positioning is in the anterior/lateral

position. In this situation, one pad/paddle is placed under the right clavicle, above the nipple, and the other (the left one) is placed laterally, below the nipple. Most commercially available pads have drawings of correct positioning (fig 3).

- Energy selection – it should be adequate to accomplish the objective on the first attempt. However, high-energy shocks can cause myocardial damage, so prudence should be exercised. Choice should be made by considering the type of arrhythmia, the type of defibrillator and the particularities of the patient.
- When compared to monophasic defibrillators, biphasic defibrillators require less energy.
- Obese patients, or patients with high muscle mass may require shocks with higher energy, due to higher impedance (thicker chest wall).

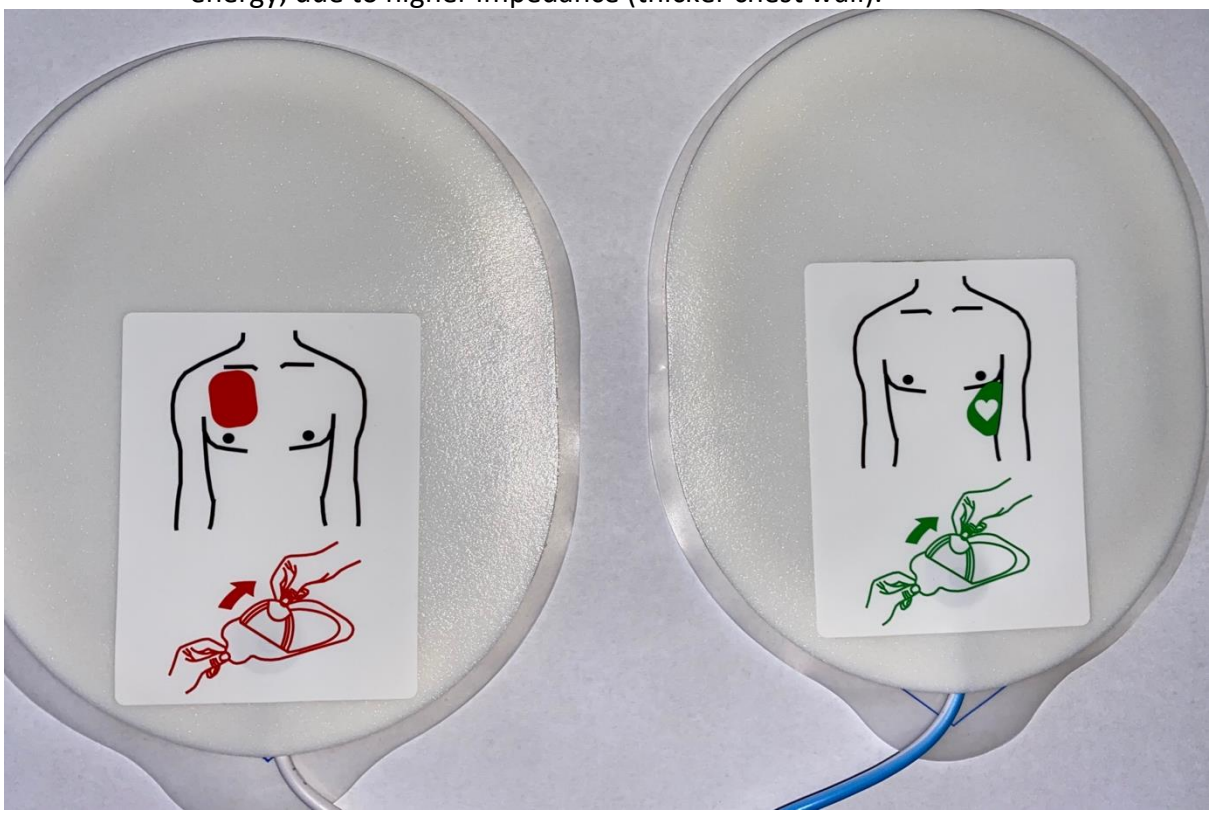


Figure 3 - defibrillator pads

- A common approach is the following
  - For AF, 120-200 J
  - For atrial flutter – 50-150 J
  - For pulsatile VT – 120 – 200 J
  - For VF and pulseless VT – 200 – 360J
- When performing electrical cardioversion, the patient should be sedated by physicians with abilities to control the airway, if needed; sedation technique can vary and is at the discretion of the attending physician.

5. Algorithms for management of acute adult arrhythmias

5.1 Tachycardia

The 2021 European Resuscitation Guidelines recommend managing the patient with an acute tachyarrhythmia following a stepwise approach:

- Assess with ABCDE approach
- Give oxygen if SpO2 <94%
- Obtain IV access
- Monitor ECG, BP, SpO2 record 12-lead ECG, use ETCO2 monitoring if administering sedation
- Identify and treat reversible causes

At this point, if the clinical examination shows signs of life-threatening features (any of shock, syncope, myocardial ischaemia, or severe heart failure), *synchronized electric shock* should not be delayed, under sedation or anaesthesia. If, after 3 attempts, cardioversion has not been successful, amiodarone 300 mg iv over 10-20 min, or procainamide 10-15 mg/kg over 20 min should be administered, after which synchronized shock should be repeated.

If neither of the signs of severity are present, the patient is considered stable, and expert consultation should be obtained. Further management should consider if on the ECG there is a narrow QRS (<0.12 s) or not.

For patients with a *narrow QRS*:

- If the rhythm is *regular,* the following algorithm should be attempted
  - Vagal manoeuvres; if unsuccessful
  - Adenosine 6 mg iv; if unsuccessful
  - Adenosine 12 mg iv, if unsuccessful
  - Adenosine 18 mg iv; if unsuccessful
  - Verapamil, diltiazem, or a beta-blocker can be considered; if unsuccessful
  - Synchronized shock up to 3 attempts can be considered.
- If the rhythm is *irregular*, the following algorithm should be attempted
  - AF is the most likely diagnosis
  - Control rate with beta-blocker or diltiazem
  - If there is evidence of heart failure, consider digoxin or amiodarone
  - If duration >48 h, anticoagulated

For patients with a *wide QRS*:

- If the rhythm is *regular,* the following algorithm should be attempted
  - Vagal manoeuvres; if unsuccessful
  - Adenosine 6 mg iv; if unsuccessful
  - Adenosine 12 mg iv; if unsuccessful
  - Adenosine 18 mg iv; if unsuccessful
  - Give procainamide 10-15 mg/g iv over 20 min, or amiodarone 300 mg iv over 10-60 min; if unsuccessful
  - Synchronized shock up to 3 attempts can be considered.
- If the rhythm is *irregular*, the following algorithm should be attempted
  - If AF with bundle branch block – treat as narrow complex
  - If Torsade de pointes – give 2 g Magnesium iv over 10 min

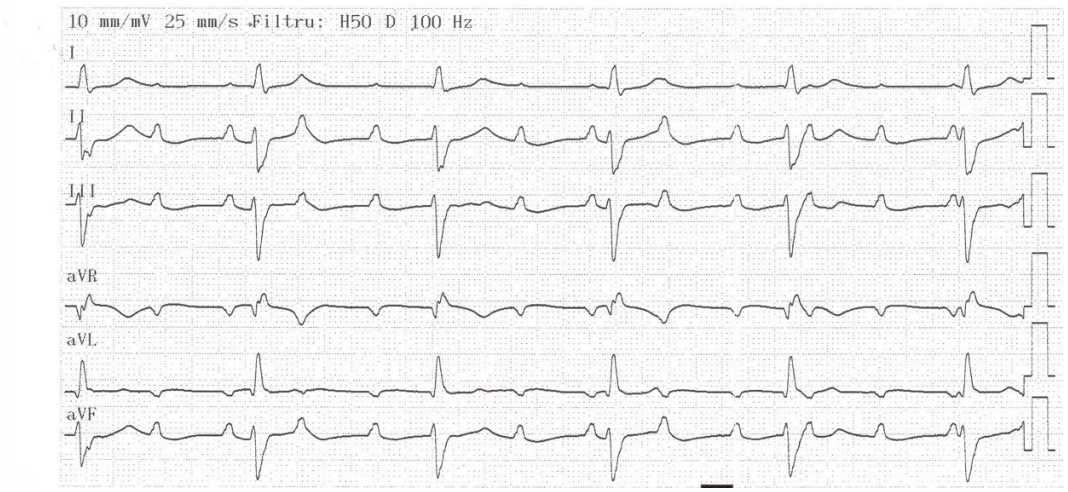
5.2 Bradycardia

For patients presenting with symptomatic bradycardia, the same stepwise protocol is recommended by the 2021 ERC guideline.

- Assess with ABCDE approach
- Give oxygen if SpO2 <94%
- Obtain IV access
- Monitor ECG, BP, SpO2 record 12-lead ECG, use ETCO2 monitoring if administering sedation
- Identify and treat reversible causes (electrolyte abnormalities, hypovolemia)

At this point, if the clinical examination shows signs of life-threatening features (any of shock, syncope, myocardial ischaemia, or severe heart failure), the following algorithm should be followed:

- Give atropine 0,5 mg iv; if no improvement
- Consider interim measures:
    - Atropine 0,5 mg iv boluses up to maximum 3 mg
    - Isoprenaline 5 mcg/min iv infusion
    - Adrenaline 2-10 mcg/min iv infusion
    - Alternative drugs
        - Aminophylline
        - Dopamine
        - Glucagon (if intoxication with beta-blocker or calcium channel blocker)
        - Glycopyrrolate
    - Transcutaneous pacing
- If available, transvenous temporary pacing should be performed (fig 4, ECG of gr III AV block before and after transvenous pacing). If not, the patient should immediately transfer to a facility with such capabilities, under transcutaneous pacing.



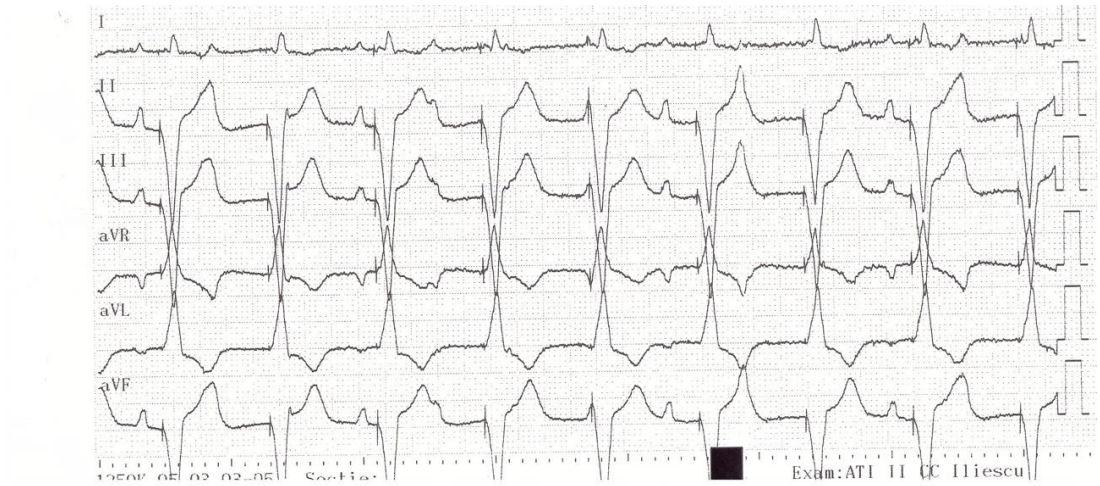


Figure 4 - ECG showing complete heart block and transvenous pacing

If the patient is in asystole, the ECG should be carefully inspected for P waves. If P waves are present, emergency pacing should be attempted. If P waves are not present, pacing attempt is superfluous and ineffective.

If after the initial examination, there are no life-threatening features, and there is no risk of asystole (recent asystole, Mobitz II AV block, complete heart block with wide QRS, or ventricular pause >3s), the patient should be monitored continuously, and expert help should be searched.

If there is risk of asystole, the algorithm is identical as for patients with life-threatening features:

- Give atropine 0,5 mg iv; if no improvement
- Consider interim measures:
    - Atropine 0,5 mg iv boluses up to maximum 3 mg
    - Isoprenaline 5 mcg/min iv infusion
    - Adrenaline 2-10 mcg/min iv infusion
    - Alternative drugs
        - Aminophylline
        - Dopamine
        - Glucagon (if intoxication with beta-blocker or calcium channel blocker)
        - Glycopyrrolate
    - Transcutaneous pacing
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6. Bibliography

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