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DEPARTMENT OF GENERAL PRACTICE – FAMILY MEDICINE
AND INTERNAL DISEASES

ESSENTIAL INTERVENTIONS OF THE FAMILY PHYSICIAN IN CARDIOLOGICAL EMERGENCY CARE

STUDY GUIDE
for 6th studying-year students, international faculty
speciality «Medicine»

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This study guide for students' practical classes and individual work was designed in accordance with the educational discipline «General practice-family medicine», speciality «Medicine». It provides an algorithm for the activities of a family physician in cases of acute coronary syndrome, cardiac arrhythmias, hypertensive crises, and heart failure. The study book was aimed at improving theory acquiring by 6th studying-year students of the international faculty over their preparation for practical classes.

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Preface

Cardiovascular diseases remain the leading cause of mortality worldwide, and their burden is particularly significant in Ukraine, where cardiovascular mortality exceeds that of many developed countries. Premature cardiovascular death, often occurring 10–12 years earlier than in high- income nations, is largely attributable to acute myocardial infarction, stroke, cardiac arrhythmias, and heart failure. This pattern reflects both reduced life expectancy and earlier onset of cardiovascular pathology within the population.

Given the central role of primary care in early detection, risk reduction, long- term management, and emergency response, strengthening the competencies of family physicians is a critical public health priority. Timely recognition of life- threatening conditions and adherence to evidence- based management pathways are essential for improving outcomes and reducing preventable mortality.

This teaching manual, developed in accordance with the curriculum of the discipline General Practice – Family Medicine, provides structured principles and clinical algorithms for the management of acute coronary syndrome, hypertensive crises, cardiac rhythm disturbances, and acute heart failure in the primary care setting. The resource addresses a recognised gap in educational materials that fully correspond to the core components of the national training programme in family medicine.

The study guide is intended for use by sixth- year medical students, interns, and practising physicians in general practice/family medicine, as well as clinicians of other specialties who require a concise, evidence- based reference for cardiological emergencies.

The source of the picture: https://www.todaysrdh.com/ten-curious-signs-of-cardiovascular-disease/

ESSENTIAL INTERVENTIONS OF THE FAMILY PHYSICIAN IN CARDIOLOGICAL EMERGENCY CARE

I. Relevance of the Topic

Cardiovascular diseases remain the leading global cause of mortality, accounting for more than half of all deaths and a substantial proportion of long- term disability. According to WHO estimates, cardiovascular conditions claim approximately 17.5 million lives annually, with projections rising to 23.5 million by 2030.

In Ukraine, the burden is even more pronounced, with cardiovascular mortality significantly exceeding that of high- income countries and occurring at a younger age. Early onset of disease, reduced life expectancy, and high rates of acute myocardial infarction, stroke, arrhythmias, and heart failure highlight the urgent need for effective prevention, timely diagnosis, and evidence- based treatment.

Primary care plays a decisive role in reducing complications and mortality. Rapid recognition of life- threatening conditions and competent pre- hospital emergency management by family physicians are essential for preventing irreversible organ damage and improving clinical outcomes.

II. Learning Objectives

To develop the ability to recognise and diagnose acute coronary syndrome, cardiac rhythm disturbances, hypertensive crises, and heart failure, and to apply evidence- based diagnostic and therapeutic algorithms, including the provision of emergency care in these conditions.

III. Core Learning Objectives

Theoretical Competencies

- Understanding models of primary care delivery and intersectoral collaboration in emergency settings.
- Identifying cardiovascular and cerebrovascular conditions requiring urgent intervention.
- Knowing the principles of pre- hospital management of acute coronary syndrome, arrhythmias, hypertensive crises, and heart failure.
- Understanding indications, contraindications, and dosing regimens for essential emergency medications.

Practical Competencies

- Rapid assessment of patient status and provision of emergency care in primary care settings for acute coronary syndrome, arrhythmias, hypertensive crises, and heart failure.

IV. Materials for Self- Study

Students should independently review:

- The role of family physicians in emergency care in urban and rural settings.

- The importance of timely intervention in reducing mortality, disability, and healthcare expenditure.

- Types of hypertensive crises and principles of emergency management.

- Arrhythmias requiring urgent care and their treatment strategies.

- Pre- hospital emergency management of acute coronary syndrome.

V. Methodological Guidance for Practical Classes

Practical sessions begin with baseline knowledge assessment, followed by topic- focused discussion. Under instructor supervision, students analyse outpatient records, discharge summaries, and clinical scenarios using a syndromic diagnostic approach.

Attention is given to laboratory and instrumental diagnostics, interpretation of findings, and evidence- based treatment strategies for acute coronary syndrome, arrhythmias, hypertensive crises, and heart failure.

Students also develop prevention and rehabilitation plans. The instructor provides clarification and addresses questions arising during the session.

I. Topic Overview

ACUTE CORONARY SYNDROME

Acute coronary syndrome (ACS) refers to a cluster of symptoms and clinical signs that raise suspicion of acute myocardial infarction (MI) or unstable angina (UA). The term ACS is applied at the first medical contact as a provisional diagnosis.

Identifying patients with ACS among all presentations of cardiac disease is diagnostically challenging, particularly when symptoms are atypical or when electrocardiographic (ECG) changes are absent. Despite advances in contemporary therapy, mortality, the incidence of MI, and the rate of rehospitalisation in this patient group remain high. Pathological, imaging, and biochemical studies have demonstrated that, in most cases, ACS is caused by rupture or erosion of an atherosclerotic plaque, followed by thrombus formation of varying extent and distal embolisation, ultimately resulting in impaired myocardial perfusion.

Chest pain is the principal symptom of acute coronary syndrome (ACS); however, patient classification is based primarily on electrocardiographic (ECG) findings. Two major categories are distinguished:

1. Patients with acute chest pain and persistent ST- segment elevation (>20 minutes)

This presentation corresponds to ACS with persistent ST- segment elevation and typically reflects acute, complete coronary occlusion.

Most of these patients subsequently develop ST- segment elevation myocardial infarction (STEMI), often a Q- wave MI.

The therapeutic objective is to achieve rapid, complete, and sustained reperfusion, either through primary percutaneous coronary intervention (PCI) or fibrinolytic therapy.

2. Patients with acute chest pain but without ST- segment elevation

In these patients, ECG findings may include persistent or transient ST- segment depression, T- wave inversion, flattening, pseudonormalisation, or no abnormalities at all.

The initial management strategy focuses on reducing ischaemia and symptoms, continuous ECG monitoring, and repeated measurement of myocardial necrosis markers.

After troponin assessment, the working diagnosis of non- ST- segment elevation ACS (NSTE- ACS) is refined to either unstable angina or non- ST- segment elevation myocardial infarction (NSTEMI).

Epidemiology

International registry data, including contributions from centres in Ukraine, indicate that non- ST- segment elevation ACS (NSTE- ACS) occurs more frequently than ST- segment elevation ACS (STE- ACS), with an annual incidence of approximately 3 cases per 1,000 inhabitants.

Hospital mortality is higher in patients with ST- segment elevation myocardial infarction (STEMI) compared with those without ST elevation (7% vs. 3–5%, respectively). However, by six months, cardiovascular mortality becomes similar in both groups, reaching 12% and 13%, respectively.

Long- term follow- up has shown that, after four years, mortality among patients with NSTE- ACS is twice as high as in those with STE- ACS. This difference is attributed to the characteristics of the NSTE- ACS population, who are typically older and have a higher prevalence of comorbidities such as diabetes and renal impairment.

Therefore, the management strategy for NSTE- ACS must address not only the acute phase of the condition but also long- term risk reduction.

Pathophysiology

The underlying cause of acute coronary syndrome (ACS) is acute thrombosis triggered by rupture or erosion of an atherosclerotic plaque, leading to a critical reduction in coronary blood flow. Plaque destabilisation and structural disruption are considered the central pathophysiological mechanisms.

In rare cases, ACS may arise from non- atherosclerotic causes, including arteritis, trauma, coronary dissection, thromboembolism, congenital anomalies, cocaine use, or complications related to cardiac catheterisation.

Clinical Presentation

The clinical presentation of acute coronary syndrome (ACS) encompasses a wide range of symptoms. Traditionally, several key manifestations are recognised:

- Prolonged anginal pain at rest (>20 minutes)
- New- onset angina of Canadian Cardiovascular Society (CCS) class II or III
- Recent destabilisation of previously stable angina, consistent with CCS class III (crescendo angina)
- Post- infarction angina

Prolonged chest pain occurs in approximately 80% of patients, whereas new- onset or progressive angina is observed in the remaining 20%.

Typical symptoms include pressure, tightness, or heaviness behind the sternum, often radiating to the left arm, neck, or jaw. These sensations may be intermittent (usually lasting several minutes) or persistent. They may be accompanied by sweating, nausea, abdominal discomfort, dyspnoea, or syncope.

Atypical presentations are also common, such as epigastric pain, dyspepsia, sharp chest pain, pleuritic- type pain, or worsening breathlessness. These are more frequently seen in patients over 75 years, women, and individuals with diabetes, chronic kidney disease, or dementia.

Absence of chest pain increases the risk of diagnostic delay and mismanagement.

Diagnosis is particularly challenging when the ECG is normal or minimally abnormal, or when abnormalities are attributable to intraventricular conduction defects or left ventricular hypertrophy (LVH).

Certain symptom- related features may facilitate recognition of ischaemic heart disease (IHD) and guide management. Symptoms that worsen with exertion and improve with rest or nitrates support an ischaemic origin.

It is also essential to consider clinical conditions that may precipitate or exacerbate ACS, such as anaemia, infection, inflammation, fever, metabolic or endocrine disturbances (including thyroid dysfunction).

When symptoms are present, several factors increase the likelihood of IHD

and, consequently, ACS - particularly non- ST- segment elevation ACS (NSTE- ACS). These include older age, male sex, family history, and evidence of peripheral or carotid atherosclerosis.

Risk factors such as diabetes, renal impairment, and a history of IHD (including MI, PCI, or CABG) further increase the probability of NSTE- ACS.

Diagnostics

Physical Examination

Physical examination findings are often unremarkable. The primary purpose of the physical assessment is to exclude non- cardiac causes of chest pain and non- ischaemic cardiac conditions, such as pulmonary embolism, aortic dissection, pericarditis, or valvular disease, as well as potential extracardiac causes, including acute pulmonary disorders (pneumothorax, pneumonia, pleuritis).

Accordingly, differences in blood pressure between limbs, an irregular pulse, cardiac murmurs, a pericardial friction rub, or chest wall tenderness may indicate diagnoses other than ACS.

Other signs - such as pallor, excessive sweating, or tremor - may suggest underlying conditions such as anaemia or thyrotoxicosis.

Electrocardiography

A 12- lead electrocardiogram (ECG) is the diagnostic method of choice for patients with suspected non- ST- segment elevation ACS (NSTE- ACS).

ECG recording should be performed within 10 minutes of first medical contact.

Characteristic ECG abnormalities in NSTE- ACS include:

- ST- segment depression
- Transient ST- segment elevation
- T- wave inversion or flattening

The ECG should be compared with previous tracings, which is particularly important in patients with comorbid cardiac conditions such as left ventricular hypertrophy (LVH) or a history of myocardial infarction.

Repeat ECG recordings are required at 3 hours, 6–9 hours, and 24 hours after symptom onset, and immediately if chest pain or other symptoms recur.

A completely normal ECG does not exclude NSTE- ACS.

Ischaemia in the territory of the left circumflex artery or isolated ischaemia of the right coronary artery may be missed on a standard 12- lead ECG but can be detected in leads V7–V9 and V3R–V4R, respectively.

Transient conduction blocks may occur during episodes of ischaemia.

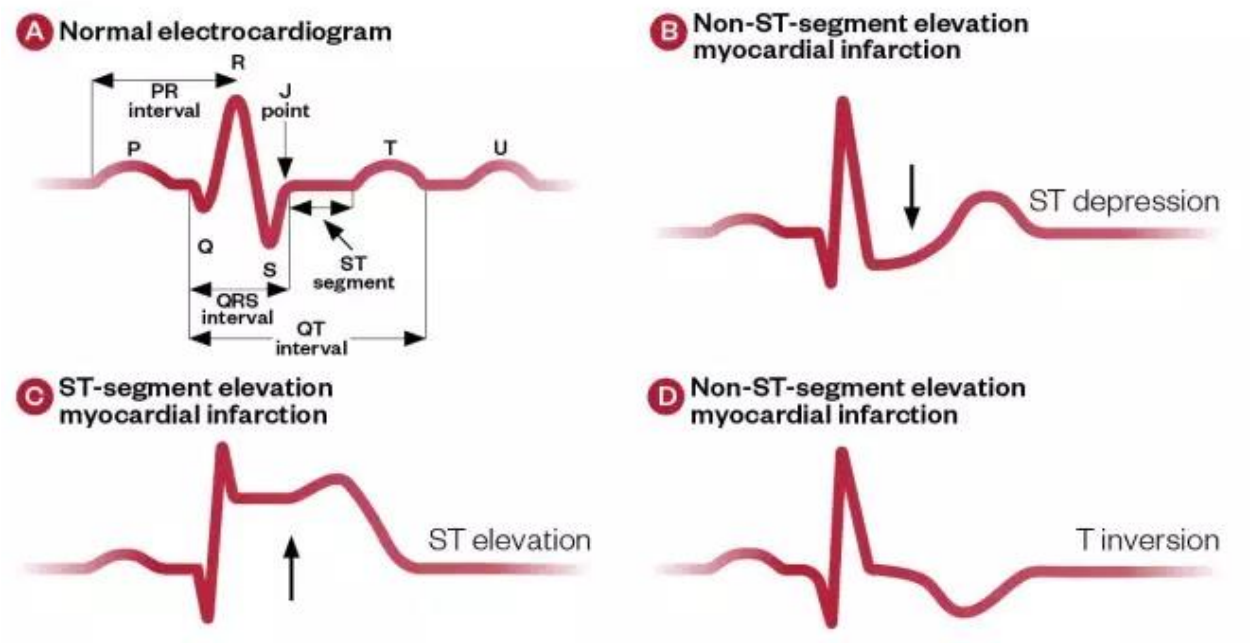

Figure 1. Typical electrocardiogram changes associated with acute coronary syndrome

Image source:: https://pharmaceutical-journal.com/article/ld/acute-coronary-syndrome-risk-factors-diagnosis-and-treatment

ECG- Based Diagnosis in ST- Segment Elevation ACS (STE- ACS)

In ST- segment elevation ACS (STE- ACS), the ECG is rarely normal, even in the early stages.

ST- segment elevation at the J- point in acute myocardial infarction is typically present in at least two contiguous leads and is defined as:

- ≥0.25 mV in men under 40 years,

- ≥0.20 mV in men aged 40 years or older,
- ≥0.15 mV in women in leads V2–V3, and/or
- ≥0.10 mV in other leads, provided that left ventricular hypertrophy or left bundle branch block (LBBB) is absent.

For patients with inferior myocardial infarction, recording right- sided precordial leads (V3R and V4R) is recommended to detect concomitant right ventricular infarction.

Similarly, ST- segment depression in leads V1–V3 suggests posterior myocardial ischaemia, particularly when the terminal T wave is positive (an ST- elevation equivalent). This can be confirmed by ST- segment elevation ≥0.1 mV in leads V7–V9.

Situations in Which ECG Interpretation Becomes More Challenging

Left or Right Bundle Branch Block

Diagnosis of acute myocardial infarction is difficult in the presence of LBBB, although marked ST- segment deviations may still indicate acute coronary occlusion.

Concordant ST- segment elevation (in leads with a positive QRS complex) strongly suggests acute MI with coronary occlusion.

Most patients with LBBB, however, do not have acute coronary occlusion and do not require immediate reperfusion.

Previous ECGs help determine whether the block is new; a new or presumably new LBBB with symptoms of active ischaemia warrants immediate reperfusion therapy, preferably urgent coronary angiography with primary PCI, or intravenous thrombolysis if PCI is unavailable.

A positive troponin test obtained 1–2 hours after symptom onset may help determine whether urgent angiography is necessary.

Patients with MI and right bundle branch block (RBBB) also have a poor prognosis, although RBBB usually does not prevent interpretation of ST- segment elevation.

Treatment should begin when ischaemic symptoms occur in the presence of

RBBB, regardless of whether the block is new.

Pacemaker Rhythm

A paced rhythm may obscure ST- segment changes and lead to urgent angiography for diagnostic clarification.

Reprogramming the pacemaker to allow better ECG assessment may be useful in selected patients.

Patients Without Diagnostic ECG Changes

Some patients with acute coronary occlusion may initially present without ST- segment elevation, especially if assessed very early after symptom onset.

In such cases, clinicians should look for hyperacute T waves, which may precede ST elevation.

Repeat ECGs and continuous ST- segment monitoring are essential.

Isolated Posterior Myocardial Infarction

Acute MI in the posterior (inferobasal) wall, often supplied by the left circumflex artery, typically presents with isolated ST- segment depression ≥0.05 mV in V1–V3.

This condition should be managed as STEMI.

Additional leads V7–V9 (≥0.05 mV, or ≥0.1 mV in men under 40 years) are recommended to detect posterior ST elevation.

Left Main Coronary Artery Obstruction

ST- segment depression in eight or more leads, combined with ST- segment elevation in aVR and/or V1, suggests left main coronary artery obstruction or severe multivessel disease, particularly in haemodynamically unstable patients.

Limitations of Standard Resting ECG

A standard resting ECG does not always reflect the dynamic nature of coronary thrombosis and myocardial ischaemia.

Nearly two- thirds of ischaemic episodes during unstable phases are clinically silent and may not be detected on routine ECG.

If the ECG does not match the clinical picture, it should be repeated and

compared with previous tracings.

Additional leads V7, V8, V9 and Slapak–Portillo leads may assist in diagnosing selected cases.

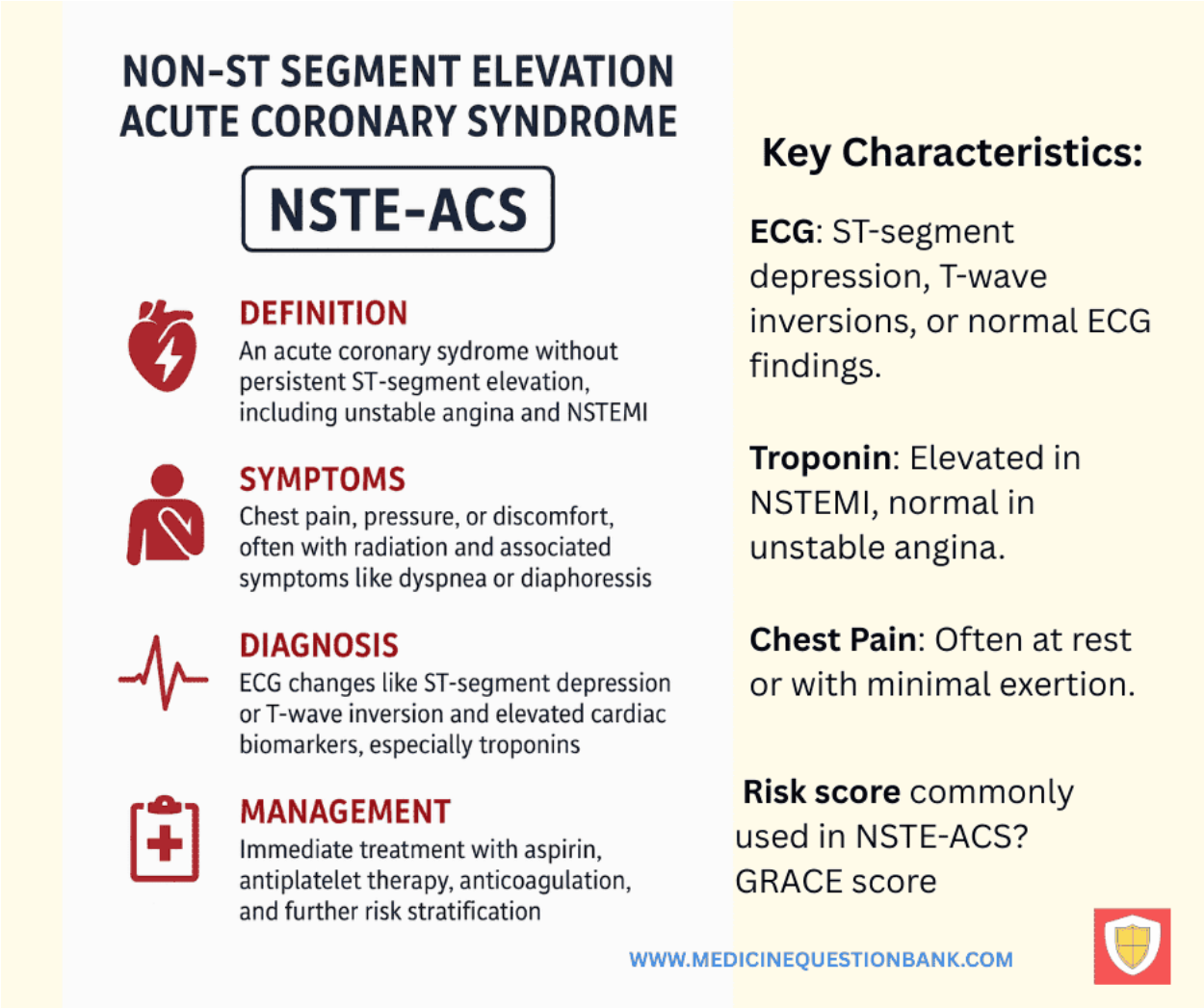

Figure 2. Non- ST- Segment Elevation Acute Coronary Syndrome (NSTE- ACS)

Image source: https://medicinequestionbank.com/non-st-segment-elevation-acute-coronary-syndrome/

A characteristic sequence of ECG changes occurs in patients depending on the timing of ACS onset (Fig. 3).

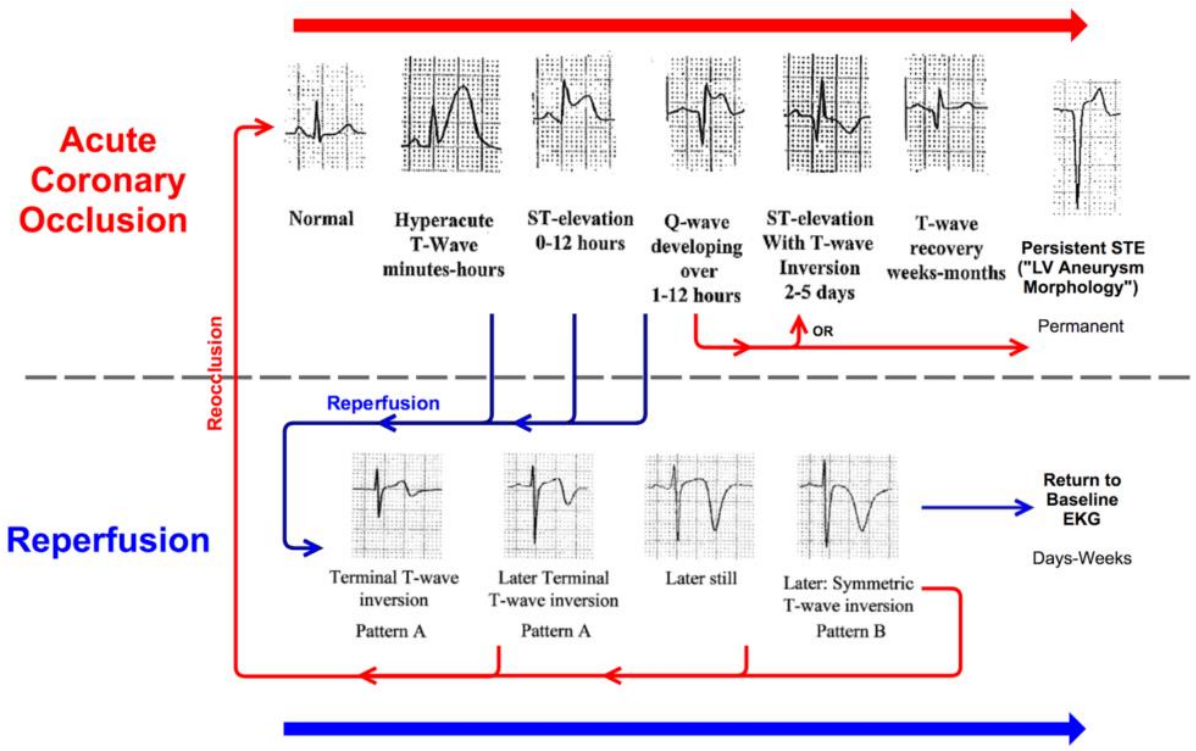

Figure 3. ECG Evolution in Acute Myocardial Infarction - LITFL (Life in the Fast Lane)

Image source: https://litfl.com/omi-replacing-the-stemi-misnomer/

Evolution of ECG Changes in Acute Myocardial Infarction

In the first hours after symptom onset, the Pardee sign (injury phase) appears.

Within 1–3 days, the ECG shows a pathological Q wave, a reduction in R- wave amplitude, and the development of a QS- type complex (acute phase).

Over the following 1–3 weeks, there is a gradual return of the ST segment toward the isoelectric line and the appearance of a negative T wave (subacute phase).

During the scarring phase, the pathological Q wave persists, the ST segment is at the isoelectric line, and the T wave may be negative, flattened, or positive.

A persistent ST- segment elevation that remains beyond the completion of the scarring phase (approximately 4 weeks) is considered a sign of left ventricular aneurysm formation.

The localisation of myocardial infarction is determined according to the leads in which typical ECG changes are recorded (Table 1).

Table 1

Myocardial Infarction Localisation Based on ECG Changes [1]

Myocardial Infarction Localisation	Leads
Anterior	I, II, AVL
Septal	V_1, V_2
Apical	V_3, V_4
Lateral	V_5, V_6
Anteroseptal	I, AVL, V_1, V_2, V_3,
Antero- septal- apical	I, AVL, V_1, V_2, V_3 V_4
Inferior (diaphragmatic)	II, III, AVF
Inferolateral	II, III, AVF, V_5, V_6
Posterior (basal)	Additional leads S1–S4 (Slopak–Portillo leads), D, A, I (Nehb leads), or V7–V9.

Q- Wave and Non- Q- Wave Myocardial Infarction; Depth of Myocardial Injury

Depending on the presence of a fully developed pathological Q wave or a QS complex, myocardial infarction is classified as Q- wave MI or non- Q- wave MI.

The presence of a QS complex indicates that necrosis has penetrated all three layers of the myocardium - a transmural infarction.

- A Q wave alone corresponds to a large- focal infarction;
- a negative T wave alone indicates an intramural infarction;
- ST- segment elevation reflects a subepicardial infarction;
- and ST- segment depression reflects a subendocardial infarction.

Thus, ECG analysis allows not only confirmation of myocardial infarction but also determination of its localisation, depth of myocardial involvement, and temporal phase, which, in combination with clinical and laboratory data, helps predict the clinical course and establish appropriate rehabilitation timelines.

Biomarkers

Cardiac troponins play a central role in diagnosis and risk assessment.

Troponins have greater specificity and sensitivity than traditional cardiac enzymes such as creatine kinase, its MB isoenzyme, and myoglobin.

Elevated cardiac troponin levels indicate myocardial cell injury, which in non- ST- segment elevation ACS may result from distal embolisation of thrombotic or atheromatous material.

Accordingly, the presence of troponins may reflect active thrombus formation.

An elevated troponin level indicates the presence of myocardial infarction [4].

In patients with MI, the initial rise in troponin levels occurs within up to 4 hours after symptom onset.

Troponin levels may remain elevated for up to 2 weeks, due to proteolysis of the contractile apparatus.

In NSTE- ACS, minimal troponin elevation typically appears within 48–72 hours.

There is no significant difference between troponin T and troponin .

In addition to troponins, patients with ACS may show elevations of other biochemical markers of cardiomyocyte necrosis:

- hyperenzymaemia - increased levels of aspartate aminotransferase (AST), lactate dehydrogenase (total and LDH- 1 isoenzyme), creatine phosphokinase (CPK and MB- CPK);
- and increased levels of proteins such as myoglobin and myosin.

Each marker has a characteristic temporal pattern of concentration changes, determined by the gradual elimination of marker proteins from the necrotic zone (Table 2).

Table 2

Dynamics of Laboratory Changes in Patients with Myocardial Infarction Depending on the Stage of Disease [1]

Marker	Onset of concentration rise	Peak concentration	Return to normal
Leukocytosis	3–4 hours	Day 2–4	Day 3–7
ESR elevation	Day 2–3	Day 8–12	Week 3–4
AST	6–8 hours	24–36 hours	Day 5–6

Total LDH	8–10 hours	Day 2–3	Day 10–12
LDH-1	8–10 hours	Day 2–3	Day 10–12
Myoglobin	2 hours	6–10 hours	24–32 hours
Total CPK	3–4 hours	24–36 hours	Day 3–6
CK-MB	4 hours	24 hours	72 hours
Troponin I	3–4 hours	24–36 hours	Day 5–14
Troponin T	4 hours	24–48 hours	Day 5–21

Actions of General Practitioners / Family Physicians in the Provision of Emergency Care

Pre- hospital medical care must be provided to patients with acute coronary syndrome (ACS) as rapidly as possible from the onset of symptoms.

Emergency or urgent care at the pre- hospital stage is delivered by:

- Emergency medical service (EMS) teams of Emergency Medical Care Centres, disaster medicine units, and emergency departments of multidisciplinary hospitals within the emergency care system.
- Urgent care teams for adult and paediatric populations.
- General practitioners / family physicians and physicians of other specialties (when such a patient is identified at home or during a clinic visit).

When a patient (or relatives/witnesses) contacts the outpatient clinic with suspected ACS, or when a call is received from a patient (or relatives) reporting symptoms suggestive of acute myocardial infarction (AMI), the family physician must ensure urgent hospitalisation by an EMS team, after performing risk stratification based on primary and secondary risk factors and the GRACE score.

Required Actions of the Physician

1. Immediate response:

Urgent home visit must be performed without delay.

2. Rapid history taking:

2.1. History of present illness

- Determine the exact time of chest pain onset and its duration.
- Assess the nature, location, and radiation of pain.

- Clarify whether nitroglycerin was used and its effect.
- Identify provoking factors — physical or emotional stress.
- Determine whether previous episodes of pain or dyspnoea occurred during walking, whether they forced the patient to stop, their duration, and whether they were relieved by nitroglycerin.
- Assess similarity to previous angina episodes.
- Determine whether pain has intensified or become more frequent, whether exercise tolerance has decreased, and whether nitrate requirement has increased.

2.2. Past medical history

- Identify daily medications.
- Clarify medications taken after symptom onset.
- Assess cardiovascular risk factors: hypertension, smoking, diabetes, hypercholesterolaemia.
- Identify comorbidities: arrhythmias, cerebrovascular disease, cancer, peptic ulcer disease, haematological disorders, history of bleeding, COPD, etc.
- Collect allergy history, including drug allergies.

3. Physical examination

3.1. Assessment of general condition and vital functions:

Consciousness, breathing, circulation - according to the ABCDE algorithm.

3.2. Immediate correction of life- threatening abnormalities:

Support of airway, breathing, and circulation as indicated.

3.3. Visual assessment: Skin colour, moisture, jugular venous distension.

4. Cardiovascular and respiratory assessment

- Pulse - rate and characteristics.
- Respiratory rate.
- Blood pressure - measure on both arms.
- Cardiac percussion - assess for cardiac enlargement.
- Cardiac palpation - apical impulse and its location.

- Auscultation of heart and vessels - tones, murmurs, presence of S3 or S4.
- Lung auscultation - presence of crackles.

Note: Many patients with AMI may have no abnormal findings on physical examination.

5. Instrumental assessment in the outpatient setting

5.1. ECG.

Record a 12- lead ECG or transmit ECG signals to a telemetric consultation centre for urgent interpretation.

If ECG signs of AMI are present → call EMS immediately.

If early in symptom onset and ECG is normal, repeat ECG every 20–30 min.

6. Therapeutic strategy

6.1. Mandatory Measures

1. Nitroglycerin should be administered sublingually in tablet form (0.5–1.0 mg) or as an aerosol (1–2 doses or 0.4–0.8 mg) if chest pain is present at the time of contact.

If necessary, and if blood pressure is normal, the dose may be repeated every 5–10 minutes.

In cases of severe pain, 2.0 ml of a 1% nitroglycerin solution may be diluted in 200 ml of 0.9% sodium chloride or 5% glucose solution (ex tempore; concentration 100 mg/ml) and administered intravenously by infusion, with continuous monitoring of blood pressure and heart rate.

With an automatic infusion pump: initial rate 10–20 μg/min.

Without a pump: initial rate 2–4 drops/min, gradually increasing to a maximum of 30 drops/min (≈3 ml/min).

The infusion must be stopped if systolic BP falls below 90 mmHg or if mean arterial pressure decreases by 10–25% from baseline.

Further BP reduction worsens coronary perfusion, enlarges the infarct zone, and may cause severe headache.

2. Acetylsalicylic acid (ASA)

If the patient has not taken ASA independently, they should chew 160–325 mg immediately.

For medical teams, intravenous ASA 1.0 g may be used.

3. Clopidogrel (if ASA is contraindicated)

If ASA cannot be used, clopidogrel 300 mg orally is an alternative.

A combination of ASA + clopidogrel is effective when ASA is not contraindicated.

4. Early dual antiplatelet therapy (DAPT)

Early initiation of DAPT is indicated for all patients (in the absence of contraindications).

For patients at moderate or high risk of ischaemic complications (elevated cardiac troponins, convincing ECG signs of ischaemia, and typical symptoms):

On top of ASA, ticagrelor 180 mg loading dose is recommended (if available and not contraindicated).

If ticagrelor is unavailable or contraindicated, clopidogrel 300 mg loading dose is used.

5. DAPT for patients transported for PCI

All patients being transported for percutaneous coronary intervention (PCI) should receive DAPT as early as possible.

For patients transferred to a specialised centre capable of coronary angiography:

Preferred: ASA + ticagrelor.

If ticagrelor is unavailable or contraindicated: clopidogrel 300–600 mg loading dose.

6. Beta- blockers

Beta- blockers should be initiated as early as possible in all ACS patients without contraindications.

7. Opioid analgesics

Morphine is preferred.

Administer 2–5 mg IV in fractional doses every 5–15 minutes until pain and dyspnoea resolve or adverse effects occur (hypotension, respiratory depression, nausea, vomiting).

Further management depends on ECG findings.

8. Management of ST- segment elevation ACS (STEMI / Q- wave MI)

If STEMI is diagnosed, care must follow the Unified Clinical Protocol for emergency, primary, secondary (specialised), and tertiary (highly specialised) medical care and rehabilitation:

"Acute Coronary Syndrome with ST- Segment Elevation", approved by the Ministry of Health of Ukraine (Order No. 455, 02.07.2014).

9. Management of non- ST- segment elevation ACS (NSTE- ACS)

If NSTE- ACS is diagnosed, care must follow the Unified Clinical Protocol:

"Acute Coronary Syndrome without ST- Segment Elevation", approved by the Ministry of Health of Ukraine (Order No. 164, 03.03.2016).

Prognosis

Unstable angina: The prognosis in unstable angina depends on the extent and location of coronary artery involvement.

Proximal coronary stenosis is associated with an unfavourable prognosis.

Distal lesions generally allow for a more favourable prognostic assessment.

The functional capacity of the left ventricle is also of major importance: left ventricular dysfunction significantly worsens prognosis .

Myocardial infarction: In myocardial infarction, the ST- segment elevation variant carries a less favourable prognosis than non- ST- segment elevation MI.

The extent of myocardial damage is a key determinant: the larger the infarct area, the more severe the patient's condition.

Other factors influencing prognosis include:

- age,
- harmful habits (e.g., smoking),
- body weight,
- comorbidities.

In an unfavourable course, acute coronary syndrome may be complicated by cardiogenic shock or scarring of the affected area, with the risk of recurrent MI.

Prevention

Screening for early detection of dyslipidaemia, arterial hypertension, diabetes mellitus, and ischaemic heart disease should include assessment of pre- test probability and stress testing, with imaging when necessary (stress ECG, stress echocardiography, MRI, etc.).

In patients identified as having high ischaemic risk, timely referral to specialised centres for diagnostic coronary angiography is recommended.

HYPERTENSIVE CRISIS

A hypertensive crisis is an acute, marked elevation of arterial blood pressure from a normal or previously elevated level, accompanied by the appearance or worsening of symptoms related to target- organ involvement or the autonomic nervous system, secondary to hypertension.

Diagnostic criteria of hypertensive crisis:

- Sudden onset.
- Significant elevation of arterial blood pressure.
- Appearance or worsening of target- organ symptoms.

Depending on the presence of target- organ damage and the urgency of blood pressure reduction, hypertensive crises are classified into:

- Complicated hypertensive crises - characterised by acute or progressive target- organ damage, pose an immediate threat to life, and require urgent BP reduction (within minutes to 1–2 hours).
- Uncomplicated hypertensive crises - without acute or progressive target- organ damage, pose a potential threat, and require rapid BP reduction (within several hours).

Epidemiology

Arterial hypertension (AH) is one of the most prevalent diseases worldwide, affecting approximately one- third of the adult population.

The introduction of modern antihypertensive therapy has significantly reduced the incidence of hypertensive emergencies (to <1% of all hypertensive patients).

Nevertheless, complications of hypertension remain among the leading causes of EMS calls and hospitalisations in cardiology .

Hypertensive crises represent the most dangerous manifestation of hypertension (25–40%).

Although recent epidemiological data are limited, it is estimated that 1–5% of hypertensive patients experience a hypertensive crisis at least once in their lifetime.

Hypertensive crises occur more frequently in:

- older adults,
- men,
- individuals of African descent.

In most cases, the cause is inadequate antihypertensive therapy or poor adherence.

Predictors of inadequate BP control include:

- older age,
- fewer antihypertensive medications,
- decompensated diabetes mellitus,
- hyperlipidaemia,
- heart failure,
- nephropathy.

Complicated Hypertensive Crisis

A complicated hypertensive crisis is a life- threatening condition.

It occurs almost twice as often in men as in women.

Without treatment, mortality reaches 70–80% within 1 year.

Patients who survive a complicated hypertensive crisis often die within the next three years from:

- renal failure (evidence level A),
- stroke (evidence level B).

This risk increases with:

- older age,
- elevated serum creatinine (evidence level A),
- longer duration of hypertension (evidence level B).

Adequate blood pressure control can reduce mortality to 1% .

Pathophysiology

The factors leading to a rapid and significant rise in arterial blood pressure in patients with hypertensive crisis (HC) are not fully understood.

In some cases, HC may develop de novo, although most patients have a history of primary or secondary hypertension .

Hypertensive crises may complicate the following diseases and conditions:

- Hypertensive disease
- Renal disorders:
  - parenchymal kidney diseases
  - chronic pyelonephritis
  - primary glomerulonephritis
  - vasculoglomerular diseases
  - tubulointerstitial nephritis
- Renal vascular diseases:
  - renal artery stenosis
  - fibromuscular dysplasia
  - atherosclerotic lesions
- Toxic effects of medications and illicit drugs
- Pregnancy
- Endocrine disorders
- Diseases of the central nervous system

Initial mechanisms

The initial step in the development of HC is believed to be a sharp increase in systemic vascular resistance, caused by the release of humoral vasoconstrictors from the vascular wall, combined with impaired autoregulation of blood pressure.

As a result, a rapid rise in intravascular pressure leads to:

- endothelial damage
- increased vascular permeability
- platelet activation
- activation of the coagulation cascade
- fibrin deposition

These processes promote progressive endothelial dysfunction, development of fibrinoid necrosis of arterioles, and further release of vasoconstrictors.

If this vicious cycle is not interrupted, tissue ischaemia develops, target organs become damaged, and autoregulatory mechanisms deteriorate further

Role of the renin–angiotensin–aldosterone system (RAAS)

Dysfunction of the RAAS, sodium and fluid retention, and acute impairment of the baroreflex play key roles in the development and progression of HC.

Under normal conditions, RAAS maintains and regulates blood pressure.

Excess renin synthesis stimulates the production of large amounts of angiotensin II, a potent vasoconstrictor, leading to increased vascular resistance and elevated BP.

A high renin level (>0.65 $ng \cdot ml^{-1} \cdot h^{-1}$) is characteristic of the R- type (renin- dependent) hypertension and certain forms of HC.

From a pathophysiological perspective, such patients may benefit from:

- β- blockers
- ACE inhibitors
- revascularisation / denervation / removal of an ischaemic kidney

V- type hypertension

In contrast, patients with V- type hypertension primarily exhibit sodium and fluid retention, with normal or slightly reduced renin levels.

These patients respond best to:

- diuretics
- aldosterone antagonists
- calcium channel blockers
- α- blockers

Baroreflex dysfunction

The arterial baroreflex helps maintain stable blood pressure and prevents major fluctuations.

Acute impairment of baroreflex function is mainly associated with:

- surgical interventions damaging the glossopharyngeal or vagus nerves
- carotid endarterectomy
- acute stroke (brainstem dysfunction)

- genetic disorders of chromaffin tissue
- pheochromocytoma
- radiation therapy to the head and neck
- tumours of the larynx or pharynx.

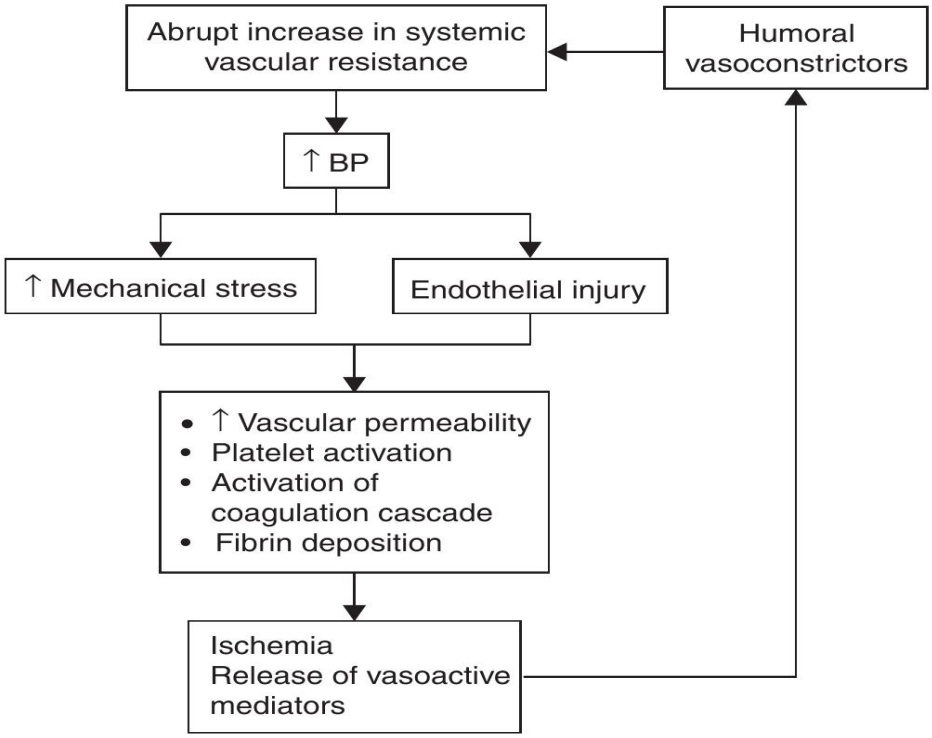

Figure 4. Pathogenesis of Hypertensive Crises

Image source: https://www.academia.edu/figures/13101839/figure-1-pathophysiology-of-hypertensive-crisis-acute-severe

A rise in blood pressure due to acute baroreflex failure may be extremely severe (systolic BP around 300 mmHg) and may sometimes be accompanied by marked bradycardia or atrioventricular conduction blocks.

For this group of patients, central sympatholytics (clonidine, urapidil) are recommended to reduce blood pressure, and in cases of heart block, temporary cardiac pacing may be required.

Disturbances in the autoregulation of cerebral, coronary, and/or renal blood flow play a major role in the onset and progression of hypertensive crises.

In individuals with normal blood pressure, cerebral autoregulatory mechanisms become overwhelmed when systolic BP exceeds 170–180 mmHg,

leading to:

- cerebral oedema with increased intracranial pressure (ICP)
- headache
- increased seizure activity
- encephalopathy
- retinopathy.

Impairment of local renal blood- flow regulation results from overload of feedback mechanisms and efferent arterioles of the glomeruli under conditions of markedly elevated BP.

Consequences include:

- proteinuria
- reduced urine output
- in severe cases, development of renal failure.

Classification of Hypertensive Crises (According to the Unified Clinical Protocol of Primary, Emergency and Secondary (Specialised) Medical Care. Arterial Hypertension, 2012)

Depending on the presence or absence of target- organ damage and the urgency of BP reduction, hypertensive crises are classified into:

- Complicated hypertensive crises - with acute or progressive target- organ damage, posing a direct threat to life and requiring immediate BP reduction within one hour.
- Uncomplicated hypertensive crises - without acute or progressive target- organ damage, posing a potential threat and requiring rapid BP reduction within several hours.

Complicated Hypertensive Crises:

1. Myocardial infarction
2. Stroke
3. Acute aortic dissection
4. Acute left ventricular failure
5. Unstable angina

6. Arrhythmias (paroxysmal tachycardia, atrial fibrillation with rapid ventricular response, high- grade ventricular ectopy)
7. Transient ischaemic attack
8. Eclampsia
9. Acute hypertensive encephalopathy
10. Haemorrhage

Uncomplicated Hypertensive Crises:

1. Cerebral uncomplicated crisis
2. Cardiac uncomplicated crisis
3. Systolic BP elevation to 240 mmHg or diastolic BP to 140 mmHg
4. Marked postoperative BP elevation

Complicated Hypertensive Crises

The clinical course is characterised by signs of acute or progressive target- organ damage.

This damage may be organic (myocardial infarction, stroke, acute aortic dissection), or functional (unstable angina, acute left ventricular failure, etc.).

Such crises are always accompanied by the appearance or worsening of target- organ symptoms.

They are life- threatening and require blood pressure reduction within minutes to one hour.

Management must be carried out in an intensive care unit, using parenteral antihypertensive therapy.

This category also includes cases of marked blood pressure elevation in which the threat to life arises not from target- organ damage, but from haemorrhage, most commonly in the postoperative period.

Uncomplicated Hypertensive Crises

Uncomplicated hypertensive crises are characterised by the absence of clinical signs of acute or progressive target- organ damage.

However, they still pose a potential threat to life, as delayed treatment may lead to complications and death.

These crises are typically accompanied by the appearance or worsening of symptoms, such as target- organ–related symptoms:

- intense headache
- chest pain
- extrasystoles
- Autonomic nervous system symptoms
- autonomic dysfunction
- tremor
- frequent urination.

Depending on the organ generating the symptoms, uncomplicated crises are classified as:

- cerebral uncomplicated crises
- cardiac uncomplicated crises.

Hypothalamic paroxysms (formerly “diencephalic–autonomic crises”) are considered a manifestation of cerebral crisis.

A systolic BP increase to 240 mmHg or diastolic BP to 140 mmHg should also be regarded as a hypertensive crisis, regardless of the presence of symptoms, as such levels are dangerous for any patient.

A marked rise in BP in the early postoperative period is also considered threatening due to the risk of haemorrhage.

All these clinical presentations require blood pressure reduction within several hours. Hospitalisation is not mandatory.

Treatment is carried out using oral or intramuscular antihypertensive medications.

Diagnosis

The most important task at the initial diagnostic stage is to distinguish between complicated and uncomplicated hypertensive crises, that is, to identify signs of possible target- organ damage.

A thorough history and physical examination often allow clarification of the nature of hypertension, the degree of BP elevation, the presence of target- organ involvement, and help determine the plan for further evaluation and treatment.

History Taking

When collecting the history of the present illness, attention should be paid to: duration of hypertension, the patient's usual ("baseline") blood pressure levels, possible previous target- organ damage, comorbidities, details of current antihypertensive therapy and treatment adherence, use of medications (sympathomimetics, NSAIDs, herbal preparations) or illicit drugs (cocaine, amphetamine, ephedrine) that may provoke a hypertensive crisis.

A hypertensive crisis may also be triggered by abrupt withdrawal of β- blockers or central sympatholytics (rebound hypertension).

Symptoms

Symptoms in hypertensive crisis can be divided into general and local.

General symptoms: flushing or pallor of the skin and face, sweating, dizziness, palpitations, dry mouth, tinnitus, fear, agitation, tremor.

Local symptoms (depending on the affected organ): anginal pain (unstable angina, myocardial infarction, aortic dissection); dyspnoea (acute pulmonary oedema); back pain (aortic dissection), headache, impaired consciousness, focal neurological deficits; nausea, vomiting, visual disturbances (intracranial or subarachnoid haemorrhage, hypertensive encephalopathy); bleeding (postoperative).

Physical Examination

Blood pressure must be measured on both arms.

A significant difference (>20 mmHg) may indicate aortic dissection.

Measuring BP in different positions (standing and supine) helps detect occult dehydration, which is common in hypertensive crises due to compensatory natriuresis.

It is essential to use a properly sized cuff, as a cuff that is too small may artificially elevate BP readings in overweight patients .

Cardiovascular Examination

Assessment should include evaluation of new murmurs and additional heart sounds.

A diastolic murmur over the aorta may indicate aortic dissection.

A gallop rhythm, accentuated second heart sound over the pulmonary artery, or increased systolic murmur of mitral regurgitation suggests progressive heart failure.

Other signs of worsening heart failure include jugular venous distension, hepatomegaly, and peripheral oedema.

Acute left ventricular failure presents with crackles in the lower lung fields or throughout the lungs.

During a hypertensive crisis, paroxysmal tachycardia or atrial fibrillation may occur, significantly worsening the clinical situation.

Assessment for Secondary Hypertension

The presence of atherosclerotic disease in any vascular territory, especially in smokers, should raise suspicion of renovascular hypertension.

A systolic/diastolic bruit over the abdominal aorta or renal arteries supports this diagnosis.

Neurological and Ophthalmological Examination

Every patient must undergo a careful neurological and ophthalmological assessment.

Focal neurological deficits indicate possible ischaemic or haemorrhagic stroke.

Global neurological symptoms and impaired consciousness may suggest hypertensive encephalopathy.

This diagnosis is made only after excluding other causes of CNS dysfunction (ischaemic/haemorrhagic stroke, subarachnoid haemorrhage).

All patients should undergo fundoscopic examination.

Retinopathy of Grade III (flame- shaped haemorrhages, hard and soft exudates, cotton- wool spots), or Grade IV (optic disc oedema and degenerative retinal changes) according to the Keith–Wagener–Barker classification is a sign of complicated hypertensive crisis .

Laboratory Diagnostics

Laboratory diagnostics provide additional information regarding target- organ damage.

A complete blood count with microscopic evaluation of blood cells is a mandatory baseline test.

The presence of schistocytes indicates microangiopathic haemolytic anaemia.

Assessment of serum urea, creatinine, and electrolytes assists in diagnosing renal involvement.

Urinalysis (dipstick) is essential for detecting haematuria and/or proteinuria, and microscopic urine examination helps differentiate dysmorphic from non- dysmorphic erythrocytes.

A reduction in circulating blood volume and secondary hyperaldosteronism may lead to hypokalaemia and metabolic alkalosis.

Acute hepatic ischaemia during a hypertensive crisis may cause elevated liver transaminases (AST, ALT) .

Additional laboratory tests include toxicology screening, endocrine studies, and pregnancy testing, which may help identify the underlying cause of the crisis.

In suspected secondary hypertension, measurement of renin, aldosterone, or catecholamines is recommended .

Other Diagnostic Methods

An electrocardiogram (ECG) is mandatory for all patients with hypertensive crisis.

It helps detect signs of acute ischaemia or myocardial infarction, as well as left ventricular hypertrophy, which reflects chronic hypertension.

Important diagnostic studies also include:

- Chest X- ray - to assess cardiac, aortic, and pulmonary artery contours, and detect pulmonary congestion.

- Echocardiography - to evaluate chamber size, aortic dimensions, and intracardiac haemodynamics.

The need for additional laboratory and instrumental tests depends on which target organs are affected or suspected based on history and physical examination.

Mandatory and additional investigations for patients with hypertensive crises are summarised in Table 3.

Table 3

Mandatory and Additional Investigations in Patients with Hypertensive Crises [1]

Investigation	Purpose	Category
Blood pressure measurement (both arms; standing & supine)	Detect aortic dissection; identify dehydration; confirm severity of BP elevation	Mandatory
Complete blood count with schistocyte evaluation	Detect microangiopathic haemolytic anaemia	Mandatory
Serum urea, creatinine, electrolytes	Assess renal function; detect hypokalaemia/metabolic alkalosis	Mandatory
Urinalysis (dipstick) for haematuria/proteinuria	Screen for renal involvement	Mandatory
Microscopic urine examination	Differentiate dysmorphic vs non-dysmorphic erythrocytes	Mandatory
Electrocardiogram (ECG)	Detect acute ischaemia, MI, LV hypertrophy	Mandatory
Chest X-ray	Assess cardiac/aortic contours; detect pulmonary congestion	Mandatory
Echocardiography	Evaluate chamber size, aortic dimensions, haemodynamics	Mandatory (if available)
Toxicology screening	Identify drug-induced hypertensive crisis	Additional
Endocrine tests (renin, aldosterone, catecholamines)	Evaluate suspected secondary hypertension	Additional

Investigation	Purpose	Category
Pregnancy test	Identify pregnancy-related hypertensive disorders	Additional
CT/MRI of brain	Evaluate suspected stroke or hypertensive encephalopathy	Additional (based on symptoms)
CT angiography of aorta	Confirm or exclude aortic dissection	Additional (urgent if suspected)
Renal Doppler ultrasound	Assess renovascular hypertension	Additional
Fundoscopy	Detect grade III–IV hypertensive retinopathy	Mandatory for complicated crisis

Management of Hypertensive Crises

(According to the Unified Clinical Protocol of Primary, Emergency and Secondary (Specialised) Medical Care. Arterial Hypertension, Order of the Ministry of Health of Ukraine No. 384, 24.05.2012)

Algorithm for the General Practitioner in the Management of Uncomplicated Hypertensive Crisis

Primary objective: to prevent the development of complications.

1. Blood pressure monitoring: Continuous or repeated BP measurements are required to assess the dynamics of the crisis.

2. Reassure the patient and explain the treatment strategy: Sedative medications may be used if necessary.

3. Clinical assessment: Evaluate the patient's clinical condition, including:

- risk of life- threatening complications
- possible cause of BP elevation
- timing of the last dose of scheduled antihypertensive therapy

4. Administration of antihypertensive medications

Medications are given sublingually or orally (see Table 4).

The physician selects the drug (or combination) based on:

- age

- BP level
- heart rate
- presence of autonomic symptoms
- comorbidities
- protocol recommendations
- patient’s previous experience with antihypertensive therapy
- Rate of BP reduction
- BP reduction in uncomplicated crisis should occur over several hours to 24 hours.

Mean arterial pressure should be reduced by no more than 25% during the first hour.

To avoid excessive BP reduction, treatment should begin with low doses, repeating them after 1–2 hours if needed.

Special caution

In elderly patients with marked atherosclerosis, rapid BP reduction may cause myocardial ischaemia.

Short- acting dihydropyridine calcium channel blockers (e.g., nifedipine) may provoke angina and coronary steal phenomenon.

If nifedipine is necessary, it should be used at ½–⅓ of the standard dose, preferably combined with propranolol (if no contraindications).

5. Coordination of scheduled antihypertensive therapy

Agree with the patient on the dose and timing of the next scheduled antihypertensive medication to prevent rebound BP elevation.

Adjust long- term therapy if necessary.

6. Additional diagnostic tests

If needed, perform ECG, Fundoscopy, other laboratory or instrumental tests to identify potentially dangerous target- organ damage or the cause of the crisis.

7. Hospitalisation criteria

If BP and clinical symptoms cannot be adequately monitored, the patient must be hospitalised.

Common Problems in the Management of Hypertensive Crisis:

- delayed diagnosis
- inadequate assessment of symptoms
- late initiation of therapy
- failure to prevent target- organ damage
- incorrect choice of medication
- insufficient BP and symptom monitoring
- excessively rapid BP reduction leading to impaired organ perfusion.

Table 4

Medications for the Treatment of Uncomplicated Hypertensive Crises [1]

Medication	Pharmacological class	Administration	Adverse effects
Captopril	ACE inhibitor	Dosage: 25 mg PO or SL, if necessary, the dose may be repeated after 90–120 min (max 100 mg). Onset of action: 15–30 min; peak effect: 30–90 min; duration: 4–6 hours	Useful in patients with suspected RAAS activation; avoid in pregnancy and bilateral renal artery stenosis.
Nifedipine (tablets/drops) (+propranolol 20 mg to prevent reflex tachycardia)	Dihydropyridine calcium channel blocker	Dosage: 5–10 mg sublingually / 5 drops Onset of action: 15–30 min; duration: 6 hours	Use with caution; risk of rapid BP drop and coronary steal; consider reduced dose. Headache, tachycardia, facial flushing.
Clonidine	Central α_2 agonist	Dosage: 0,075-0,3 mg PO Onset of action: 30-60 min	Reduces sympathetic outflow; may cause sedation or dry mouth; avoid abrupt withdrawal. Contraindicated in atrioventricular block
Propranolol	Non-selective β-blocker	Dosage: 20-40 mg PO Onset of action: 15-30 min, duration – 6 hours	May be combined with nifedipine to prevent reflex tachycardia; avoid in asthma/COPD.
Furosemide	Loop diuretic	Dosage: 40 mg Onset of action: 30-60 min	Consider in volume overload; risk of dehydration and electrolyte imbalance. Orthostatic hypotension, weakness
Torasemife	Loop diuretic	Dosage: 10-20 mg Onset of action: 30-60 min	Orthostatic hypotension, weakness

Bendazol (1% solution) (or dibazol)	vasodilator	Dosage: 3-5 ml IV / 4-8 ml IM Onset of action: 10-30 min	More effective when combined with other antihypertensive agents
Moxonidine	Imidazoline receptor agonist	Dosage: 0,2-0,4 mg PO Onset of action: 30-60 min	Favourable in patients with metabolic syndrome; avoid in severe heart failure. Dry mouth, drowsiness
Urapidil	Peripheral α_1 -blocker + central 5-HT_1 A agonist	Dosage: Initial 12.5–25 mg IV bolus over 1–2 min. Maintenance: 5–15 mg/hour IV infusion, titrated to BP (target 10–20% reduction). Onset of action: 5-20 min, duration – 2-4 hours	Effective in crises with autonomic hyperactivity; minimal reflex tachycardia.

Differentiated therapeutic approach in patients with complicated hypertensive c is given in the table 5.

Table 5

Differentiated Therapeutic Approach in Patients with Complicated Hypertensive Crises [1]

Clinical condition	Therapeutic objective	Preferred treatment approach
Hypertensive emergency with acute pulmonary oedema	Reduce preload and afterload; improve oxygenation	Intravenous vasodilators (e.g., nitroglycerin), loop diuretics; avoid β-blockers in acute LV failure
Acute coronary syndrome	Reduce myocardial oxygen demand; control BP and pain	Intravenous nitrates, β-blockers (if no contraindications), analgesia; avoid rapid BP drops
Acute aortic dissection	Rapid BP and shear-stress reduction	β-blocker first-line; add vasodilator if needed; target SBP <120 mmHg and HR <60 bpm
Acute ischaemic stroke	Maintain cerebral perfusion; avoid excessive BP reduction	BP lowering only if SBP >220 mmHg (or >185/110 mmHg before thrombolysis); gradual reduction
Intracerebral haemorrhage	Reduce risk of haematoma expansion	Controlled BP reduction to SBP 140–160 mmHg depending on guidelines
Hypertensive encephalopathy	Reduce cerebral oedema and BP	Intravenous antihypertensives with controlled reduction (≤25% MAP in first hour)

Clinical condition	Therapeutic objective	Preferred treatment approach
Eclampsia / severe pre-eclampsia	Prevent seizures; stabilise BP	Magnesium sulphate; labetalol or hydralazine; avoid ACE inhibitors
Acute renal failure / hypertensive nephropathy	Preserve renal perfusion; avoid nephrotoxicity	Intravenous vasodilators; avoid aggressive BP reduction; monitor renal function
Sympathetic crisis (pheochromocytoma, drug-induced)	Control catecholamine surge	α-blockade first; β-blockers only after α-blockade; benzodiazepines if stimulant-induced

Comorbid background / Target organ	Initiation of therapy	Therapeutic goal	Preferred agents	Not recommended
Hypertensive encephalopathy	Initial BP >140/90 mmHg	Reduce MAP by 25% within 8 hours	Labetalol, esmolol	Nitroprusside, hydralazine
Acute ischaemic stroke — with thrombolysis	SBP >185 or DBP >110 mmHg	Reduce and maintain SBP <180 and DBP <105 for 24 hours	Labetalol, urapidil	Nitroprusside
Acute ischaemic stroke — without thrombolysis	SBP >220 or DBP >120 mmHg	Reduce MAP by 10–15% in 2–3 hours, 15–25% within 24 hours	Labetalol, urapidil	Nitroprusside
Intracerebral haemorrhage	SBP >180 or MAP >130 mmHg	If ICP <25: SBP <160 and MAP <110 within 24 h. If ICP >25: SBP <180, MAP <130, CPP >60–80. SBP reduction to 140 mmHg is considered safe.	Labetalol, urapidil, esmolol	Nitroprusside, hydralazine
Subarachnoid haemorrhage	SBP >160 mmHg	Before surgery: maintain SBP <140 mmHg. After surgery: maintain SBP <200 mmHg. Nimodipine for all patients (vasospasm prevention).	Labetalol, urapidil, esmolol; nimodipine	Nitroprusside, hydralazine

Comorbid background / Target organ	Initiation of therapy	Therapeutic goal	Preferred agents	Not recommended
Acute coronary syndrome	SBP >160 or DBP >100 mmHg	Reduce MAP by 20–30%	β-blockers, nitroglycerin	Nitroprusside, enalaprilat
Acute left ventricular failure	Initial BP >140/90 mmHg	Reduce MAP by 20–30%	First-line: nitroglycerin / nitroprusside + loop diuretic. Alternatives: enalapril, urapidil	Esmolol, metoprolol, labetalol
Aortic dissection	SBP >120 mmHg	SBP 100–120 mmHg, MAP <80 mmHg; HR <60 bpm desirable	Esmolol / labetalol / metoprolol (first-line) or diltiazem / verapamil (if β-blockers contraindicated) + nitroprusside, enalapril, urapidil (second-line if β-blockers insufficient)	Vasodilators before β-blockade
Intra- and postoperative hypertension	SBP or MAP >20% above preoperative BP	Reduce DBP by 10–15% or to 110 mmHg within 30–60 min; overall MAP reduction ≤25%; BP lowering with moderate IV fluid support	Urapidil, labetalol, esmolol	–
Cardiac surgery — postoperative hypertension	SBP >140/90 or MAP >105 mmHg	Maintain SBP <140 and DBP <90 mmHg	Urapidil, nitroglycerin, labetalol, esmolol, nitroprusside	β-blockers not recommended in heart failure
Eclampsia	Seizures with BP ≥140/90 in pregnancy, labour, or postpartum	Stop seizures; secure airway	Magnesium sulphate	ACE inhibitors
Sympathetic crisis (pheochromocytoma / cocaine, amphetamine intoxication / clonidine withdrawal)	Initial BP >140/90 mmHg	Reduce MAP by 20–30%	α-blocker (urapidil). Alternatives: nitroglycerin / nitroprusside, verapamil	β-blockers without prior α-blockade

Note: SBP – systolic blood pressure; DBP – diastolic blood pressure; MAP – mean arterial pressure; TLT – thrombolytic therapy; ICP – intracranial pressure; CM – cerebral matter (brain); CPP – cerebral perfusion pressure = MAP – ICP.

"If urgent hospitalisation is required, an immediate call to the Emergency Medical Service (EMS) is recommended, with EMS arrival at the scene in accordance with the national standard for emergency medical care: within 10 minutes in urban areas and within 20 minutes in rural areas, taking into account population density, road conditions, and traffic intensity (according to the Order of the Ministry of Health of Ukraine No. 370 of 01.06.2009 'On the unified system of emergency medical care').

Transportation of a critically ill patient is recommended with prior notification of the receiving healthcare facility. This approach has been proven to reduce mortality and improve treatment outcomes. The list of required actions for the family physician during hospitalisation is presented below.

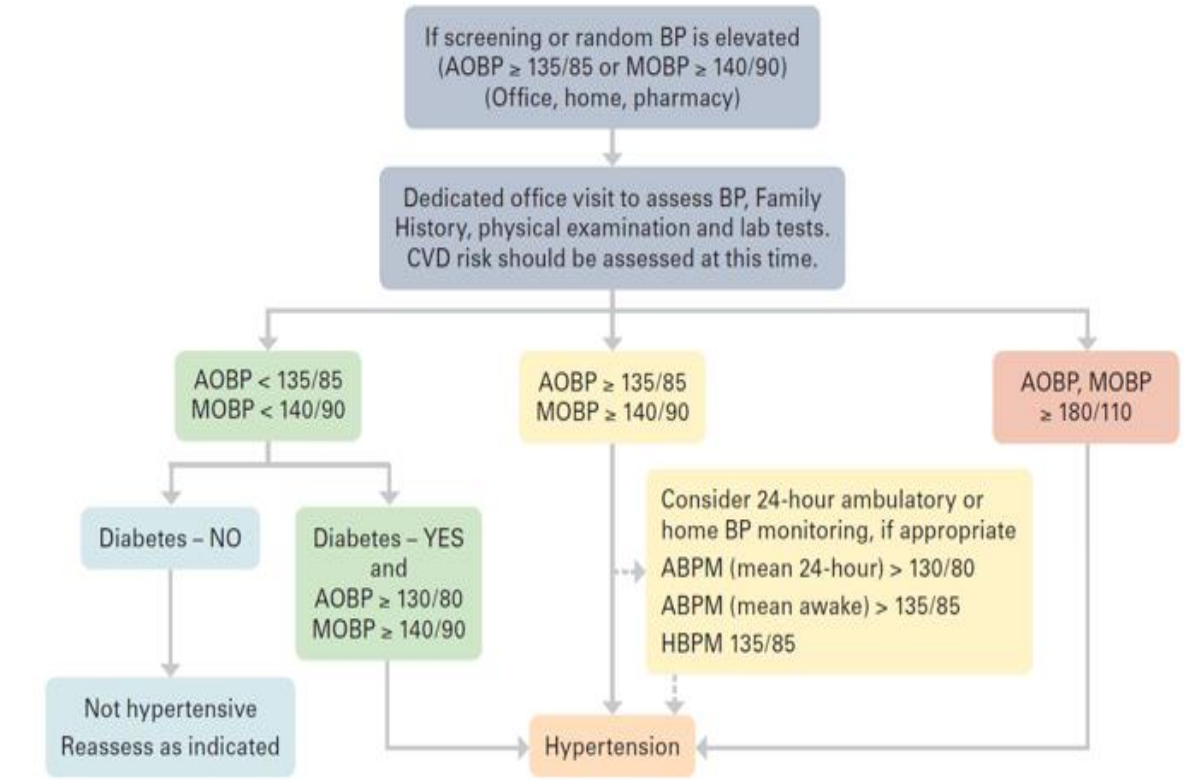

Figure 5. Diagnosis f Hypertension algorithm

Image source: https://www2.gov.bc.ca/gov/content/health/practitioner-professional-resources/bc-guidelines/hypertension

ESH 2024: Updated Clinical Guidelines, New Hypertension Classification, and Simplified Cardiovascular Risk Stratification

The 2024 European Society of Hypertension (ESH) Clinical Practice Guidelines provide a concise, evidence-based update to support the diagnosis, risk assessment, and management of arterial hypertension. These recommendations emphasize simplified clinical decision-making, individualized treatment, and improved implementation in routine practice. The guideline is endorsed by the International Society of Hypertension (ISH), the European Federation of Internal Medicine (EFIM), and the European Renal Association (ERA).

Blood Pressure Measurement and Diagnosis

Accurate blood pressure (BP) measurement remains the cornerstone of hypertension diagnosis.

Recommendations:

- Use validated automated BP devices.
- Confirm office BP with:
    - Home Blood Pressure Monitoring (HBPM), or
    - Ambulatory Blood Pressure Monitoring (ABPM) whenever possible.
- Identify:
    - White-coat hypertension
    - Masked hypertension
    - Nocturnal hypertension
- Screen adults regularly, especially individuals ≥40 years or those with cardiovascular risk factors.

Cardiovascular Risk Assessment

Treatment decisions should be based on **overall cardiovascular (CV) risk**, rather than blood pressure values alone.

Risk assessment includes:

- Age
- Sex
- Smoking status

- Diabetes mellitus
- Dyslipidemia
- Chronic kidney disease (CKD)
- Obesity
- Family history of premature cardiovascular disease
- Hypertension-mediated organ damage (HMOD)
- Established cardiovascular disease (CVD)

Patients are stratified into:

- Low risk
- Moderate risk
- High risk
- Very high risk

Lifestyle Modifications

Lifestyle intervention is recommended for **all patients**, regardless of BP level.

Key recommendations include:

- Salt intake <5 g/day
- Mediterranean or DASH diet
- Weight reduction
- Regular aerobic and resistance exercise
- Smoking cessation
- Limiting alcohol intake
- Adequate sleep and stress management

Initiation of Pharmacological Therapy

Drug treatment should be started according to BP level and cardiovascular risk.

Initial therapy

For most patients:

- **Start with a two-drug combination**, preferably in a **single-pill combination (SPC).**

Preferred combinations:

- ACE inhibitor or ARB + Calcium Channel Blocker (CCB)

- ACE inhibitor or ARB + Thiazide/thiazide-like diuretic

Monotherapy may be considered in:

- Frail elderly patients
- Grade 1 hypertension with low cardiovascular risk
- Patients with symptomatic hypotension

Blood Pressure Targets

General population

- **Target BP:** <130/80 mmHg if tolerated.

Older adults

- Individualize treatment according to frailty and tolerability.

High-risk patients

More intensive BP control is recommended in patients with:

- Diabetes
- Chronic kidney disease
- Coronary artery disease
- Previous stroke
- Heart failure

Resistant Hypertension

Evaluate for:

- Poor adherence
- White-coat effect
- Secondary hypertension
- Inadequate drug regimen

Recommended fourth-line therapy:

- **Spironolactone** (when appropriate)

Alternative options:

- Beta-blockers
- Alpha-blockers
- Other diuretics
- Device-based therapies may be considered in selected patients.

Improving Treatment Adherence

The guideline emphasizes:

- Use of **single-pill combinations**
- Patient education
- Home BP monitoring
- Telemedicine and digital health tools
- Regular follow-up
- Simplified treatment algorithms

ESH 2024 Blood Pressure Classification

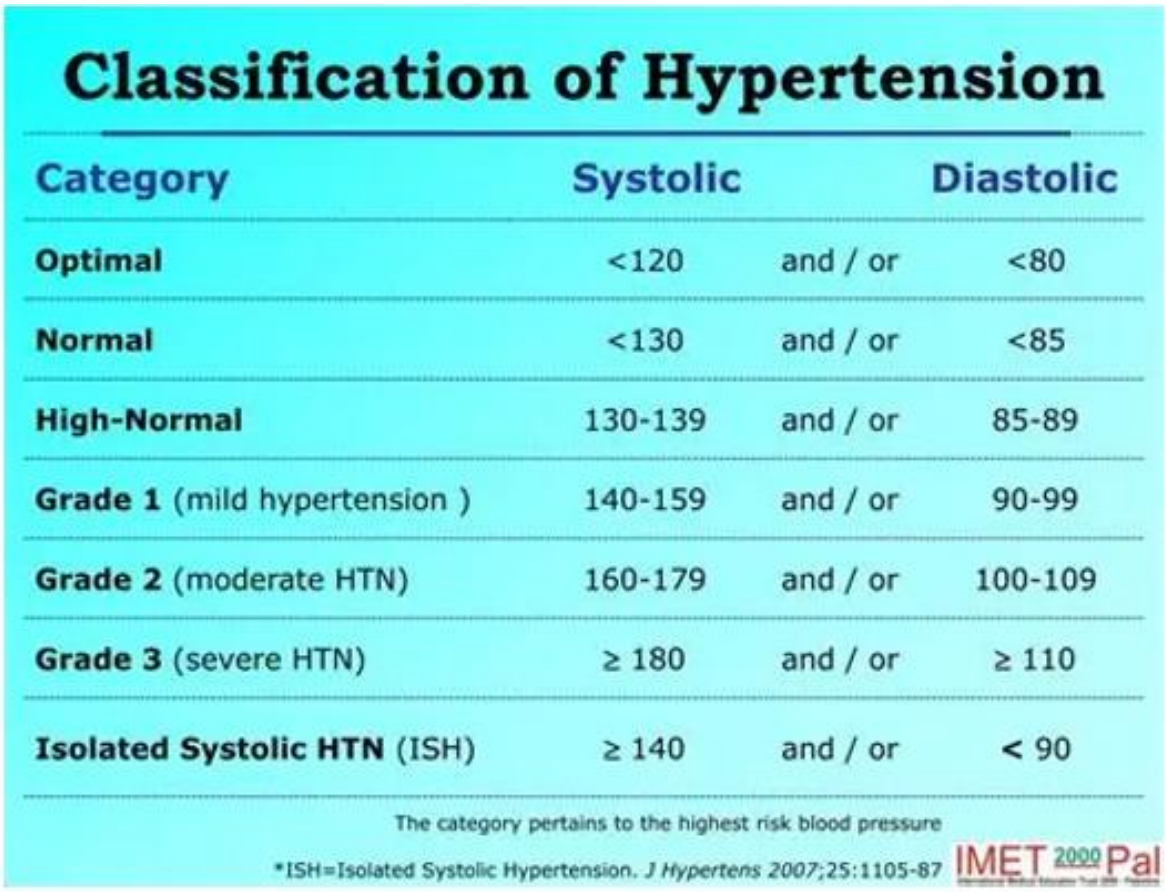

Figure 6. Blood Pressure Classification (ISH / International Standards)

Image source:

https://ts1.mm.bing.net/th?id=OIP.rz5B3Z1CCfWnxQjLC4sH3wHaFj&pid=15.1&o=7&rm=3

Simplified Cardiovascular Risk Stratification

Background

The 2024 European Society of Hypertension (ESH) Clinical Practice Guidelines emphasize that hypertension management should be based on total cardiovascular (CV) risk, rather than blood pressure (BP) values alone. Compared with the comprehensive 2023 guideline, the 2024 update presents a simplified, practice-oriented approach (the "MASTERplan") that facilitates rapid risk assessment and treatment decisions in routine clinical care.

Principles of Cardiovascular Risk Stratification

The ESH 2024 recommendations advocate integrating BP measurements with the patient's overall cardiovascular profile. Risk assessment should include:

- Age and sex
- Smoking status
- Diabetes mellitus
- Dyslipidaemia
- Chronic kidney disease (CKD)
- Obesity
- Family history of premature cardiovascular disease
- Hypertension-mediated organ damage (HMOD)
- Established cardiovascular disease (CVD)

The primary goal is to identify patients who would benefit from earlier and more intensive BP-lowering therapy.

Four Cardiovascular Risk Categories

Patients are classified into four risk categories:

Risk Category	Clinical Characteristics
Low Risk	No major cardiovascular risk factors or organ damage
Moderate Risk	One or more risk factors without established CVD or significant organ damage
High Risk	Multiple risk factors, diabetes, CKD, or hypertension-mediated organ damage

Very High Risk	Established cardiovascular disease, advanced CKD, or multiple high-risk conditions

Increasing risk category corresponds to a higher probability of future ardiovascular events and supports more aggressive prevention strategies.

Hypertension-Mediated Organ Damage (HMOD)

Detection of HMOD is central to risk stratification because it may reclassify patients into a higher risk category.

Common manifestations include:

- Left ventricular hypertrophy
- Increased carotid intima-media thickness or carotid plaque
- Microalbuminuria or proteinuria
- Reduced estimated glomerular filtration rate (eGFR)
- Hypertensive retinopathy
- Increased arterial stiffness

Assessment of HMOD is recommended during the initial evaluation whenever feasible.

Use of SCORE2 and SCORE2-OP

ESH recommends estimating 10-year cardiovascular risk using validated European prediction models:

- SCORE2 for adults aged 40–69 years
- SCORE2-OP for adults aged 70 years and older

These models estimate the risk of both fatal and non-fatal cardiovascular events and support treatment decisions in primary prevention.

Patients Automatically Considered High or Very High Risk

Formal risk calculation is generally unnecessary in patients with:

- Established cardiovascular disease
- Previous myocardial infarction
- Stroke or transient ischemic attack
- Heart failure

- Peripheral arterial disease
- Diabetes with target-organ damage
- Moderate-to-severe chronic kidney disease
- Significant hypertension-mediated organ damage

These individuals should receive intensive risk factor modification regardless of calculated SCORE2 risk.

Practical ESH 2024 Risk Assessment Algorithm

Step 1. Measure and confirm blood pressure (office BP, HBPM, or ABPM).

Step 2. Assess traditional cardiovascular risk factors:

- Age
- Sex
- Smoking
- Diabetes
- Lipid profile
- Obesity

Step 3. Screen for:

- Hypertension-mediated organ damage
- Chronic kidney disease
- Established cardiovascular disease

Step 4. Estimate cardiovascular risk using:

- SCORE2 (40–69 years)
- SCORE2-OP (≥70 years), if applicable.

Step 5. Classify the patient into:

- Low risk
- Moderate risk
- High risk
- Very high risk

Step 6. Individualize treatment intensity according to both BP level and total cardiovascular risk.

CARDIAC ARRHYTHMIAS

Cardiac arrhythmias are disturbances of the normal heart rate, rhythm, site of impulse origin, or impulse conduction, including abnormalities in the relationship and/or sequence of atrial and ventricular activation.

Classification of Cardiac Rhythm and Conduction Disorders

ICD-10 Codes for Cardiac Rhythm and Conduction Disorders

I. Disorders of Impulse Formation

I49.8 — Sinus Tachycardia (>90 beats per minute)

Sinus Bradycardia (<60 beats per minute)

Sinus Arrhythmia

I45.5 — Sinus Node Arrest (Sinoatrial Arrest)

I49.8 — Escape Beats and Escape Rhythms

- Atrial escape rhythms: slow; accelerated
- AV-junctional escape rhythms: slow; accelerated
- Ventricular escape rhythms: slow; accelerated

I45.8 — AV Dissociation

I49.8 — Migration of Supraventricular Pacemaker

Extrasystoles (“Premature Depolarizations” — ICD-10 Term)

I49.1 — Atrial Premature Beats

I49.2 — Atrioventricular (AV) Premature Beats

I49.3 — Ventricular Premature Beats

- single (≤30 ectopic beats/hour)
- frequent (≥30 ectopic beats/hour)
- allorhythmia (bi-, tri-, quadrigeminy)
- polymorphic
- paired
- early (R-on-T phenomenon)

Tachycardias

I47.1 — Supraventricular Tachycardias

- Reciprocal tachycardias: chronic; paroxysmal

- Focal (ectopic) tachycardias: chronic; paroxysmal

Supraventricular Tachycardias (Detailed)

- sinoatrial
- atrial
- AV-nodal
- typical
- atypical (with accessory pathways)
- orthodromic
- antidromic

Ventricular Tachycardias

I47.2 - Non-sustained Ventricular Tachycardia (3 ventricular beats up to 30 seconds)

- monomorphic
- polymorphic

I47.2 - Sustained Ventricular Tachycardia (>30 seconds)

- monomorphic
- polymorphic

I47.0 - Repetitive Ventricular Tachycardia

- monomorphic
- polymorphic

Atrial Fibrillation and Flutter

I48.0 — Atrial Fibrillation and Flutter

Paroxysmal (self-terminating within 7 days)

- bradycardic ventricular response (<60 bpm)
- tachycardic ventricular response (>110 bpm)

Persistent: bradycardic; tachycardic

Long-standing persistent

Permanent AF: bradycardic; tachycardic

I49.0 — Ventricular Fibrillation and Flutter

II. Disorders of Impulse Conduction

I45.5 - Sinoatrial Block

Atrioventricular (AV) Block

- I44.0 - First-degree AV block
  - I44.1 - Second-degree AV block: Mobitz I; Mobitz II
- I44.2-— Third-degree (Complete) AV block

Intraventricular Conduction Blocks

Unifascicular Blocks

- I45.0 - Right Bundle Branch Block (RBBB)
- I44.4 - Left Anterior Fascicular Block (LAFB)
- I44.5 - Left Posterior Fascicular Block (LPFB): permanent or intermittent

I45.2 - Bifascicular Blocks

- complete LBBB
- RBBB + LAFB
- RBBB + LPFB

I45.2 - Trifascicular Block

III. Combined Disorders of Impulse Formation and Conduction

I49.4 – Parasystole: atrial; AV-junctional; ventricular

IV. Diseases, Syndromes, and Phenomena

I49.8 - Idiopathic Arrhythmias

Pre-excitation Syndromes:

- Wolff–Parkinson–White (WPW)
- Short PR interval (Lown–Ganong–Levine)

I49.8 - Early Ventricular Repolarization Syndrome

I49.8 - Long QT Syndrome: congenital; acquired

I49.5 - Sick Sinus Syndrome

I46.9 - Morgagni–Adams–Stokes Syndrome

I49.8 - Arrhythmogenic Right Ventricular Dysplasia

I49.8 - Brugada Syndrome

I49.0 - Frederick's Syndrome

Sudden Cardiac Death (Arrhythmic)

I46.1 - Sudden Cardiac Death with Resuscitation

- ventricular fibrillation
- asystole
- electromechanical dissociation

I46.9 - Sudden Cardiac Death (Irreversible)

- ventricular fibrillation
- asystole
- electromechanical dissociation
- cardiac arrest occurring >1 hour after symptom onset

The management strategy for patients with cardiac rhythm disorders (CRDs) has undergone profound changes over the past 10–15 years. The principal focus of patient evaluation has shifted towards the assessment of the mechanisms underlying arrhythmia onset and the possibilities for radical elimination of cardiac arrhythmias. Catheter- based interventions and device implantation (pacemakers, implantable cardioverter- defibrillators) have become the leading modalities in the treatment of CRDs. Conversely, the importance of long- term antiarrhythmic drug therapy has markedly diminished due to an unfavourable balance between expected benefit and potential therapeutic risk.

The radicalisation of approaches to the treatment of CRDs and the widespread introduction of new technologies have led to a substantial evolution in diagnostic methods, particularly prolonged ECG monitoring, arrhythmia mapping, and electrophysiological studies. Nevertheless, the principles of primary diagnosis of CRDs in clinical practice have not changed significantly. Moreover, a distinct

differentiation of levels of care for patients with CRDs has emerged. At present, the boundaries of competence for general practitioners (GPs), cardiologists/internists, and specialists in clinical cardiac electrophysiology (cardiac subspecialists) in the management of patients with CRDs can be clearly defined.

General Clinical Assessment

The general practitioner is usually the first specialist to whom a patient with a CRD presents. The GP's primary tasks are to attempt to establish the type of arrhythmia, to assess its clinical and prognostic significance, and to determine the need for specialised care. The main components of the diagnostic process that fall within the GP's competence are outlined below .

Arrhythmia - Focused History

Arrhythmia- focused history taking involves identifying conditions that precipitate or exacerbate arrhythmias. Arrhythmias may be triggered by physical exertion, psychological or emotional stress, body position, food intake, caffeine, alcohol, smoking, the use of medications containing sympathomimetics, and recreational substances such as cocaine. Some arrhythmias occur at specific times of day.

Underlying conditions include thyrotoxicosis, mitral valve prolapse, pericarditis, and hypokalaemia. Insufficient attention to recognising these factors may lead to misinterpretation of CRDs and inappropriate management strategies. The development of certain CRDs is based on hereditary disorders .

Interaction of Structural and Functional Factors

Arrhythmias are provoked by the interaction between structural cardiac abnormalities and functional disturbances. For example:

Ventricular tachycardia typically does not occur in the absence of myocardial pathology.

The number of ventricular premature beats correlates with the severity of left ventricular systolic dysfunction.

The likelihood of the development and stabilisation of atrial fibrillation (AF) depends on left atrial size.

Supraventricular tachycardias are most observed in younger patients without significant structural myocardial abnormalities, whereas ventricular tachycardias

occur predominantly in patients with myocardial disease of various origins. AF is typical in patients with heart failure, mitral stenosis, thyrotoxicosis, and in older individuals.

A graphical representation of sinoatrial block is provided in Figure 7.

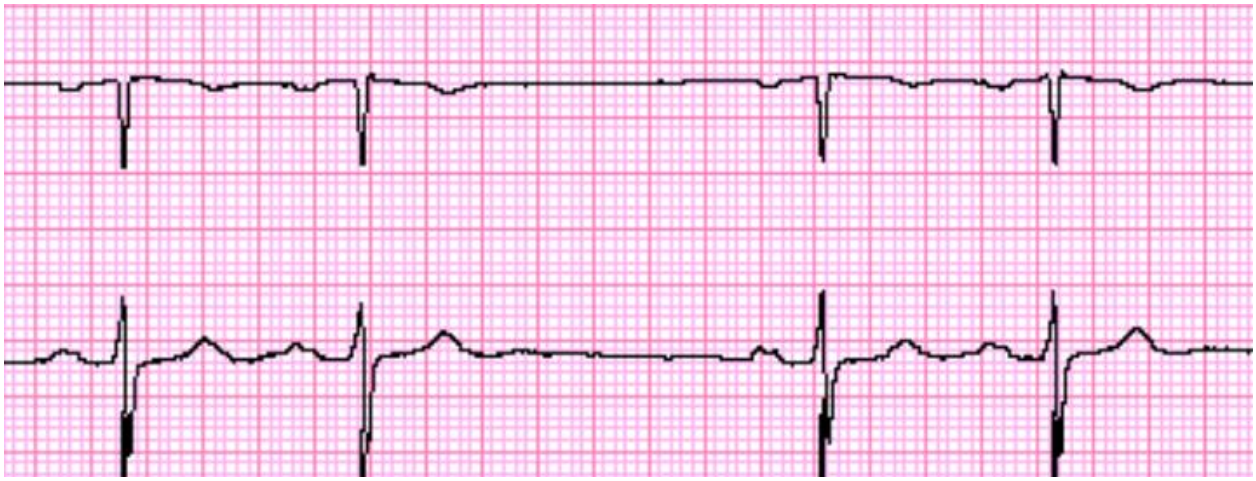

Figure 7. Sinoatrial block

Image source: https://ecgmadesimple.ca/glossary/sino-atrial-block/

The questioning of patients is aimed at differentiating between "haemodynamic" and "non- haemodynamic" symptoms of arrhythmia. The occurrence, during an arrhythmic episode, of syncope, dizziness, angina, or heart failure indicates a high likelihood of haemodynamic compromise caused by the cardiac rhythm disorder and signifies an unequivocal need for treatment and prevention. In such cases, the patient should be referred urgently to a cardiologist, preferably to a specialised arrhythmology centre where electrophysiological studies, catheter- based interventions, and pacemaker implantation are performed.

By contrast, "irregular beats", palpitations, anxiety, and fear related to the arrhythmia primarily impair the patient's quality of life. In such situations, the therapeutic priority is often the relief of clinically significant symptoms, rather than the elimination of the arrhythmia itself (most commonly extrasystoles), which in many cases does not affect long- term prognosis. Arrhythmias are frequently entirely asymptomatic and discovered incidentally. In asymptomatic or minimally

symptomatic (even frequent) extrasystoles, the risk of antiarrhythmic therapy may exceed the benefit, and therefore antiarrhythmic treatment is generally not indicated.

In asymptomatic atrial fibrillation, the key issue is determining the appropriateness of antithrombotic therapy to prevent thromboembolic complications. In addition, ventricular rate control must be ensured. In the vast majority of cases, there is no need to refer such patients to a subspecialist.

During the interview, it is often possible to obtain a preliminary impression of the nature of the rhythm disorder. Observant patients may describe changes in heart rhythm by tapping their hand on the chest or on a table. This can greatly assist in distinguishing between extrasystoles and atrial fibrillation, tachycardia and bradycardia, and regular versus irregular palpitations. For example, the sudden appearance of rapid pulsations in the neck suggests the presence of atrioventricular nodal re- entry tachycardia (AVNRT). A gradual onset and termination of a tachycardia episode may indicate sinus tachycardia, accelerated ectopic rhythms, or arrhythmias due to abnormal automaticity.

Physical Examination

Physical examination is primarily aimed at identifying arterial hypertension, myocardial dysfunction, heart failure, or valvular disease, all of which significantly influence the management of patients with cardiac rhythm disorders. Cardiac auscultation and pulse palpation often allow a preliminary assessment of the arrhythmia type.

In patients with tachycardia of unknown origin and wide QRS complexes, additional waves in the jugular venous pulse, caused by atrial contraction against a closed tricuspid valve, indicate AV dissociation associated with paroxysmal ventricular tachycardia.

Carotid sinus massage may provide diagnostically important information by provoking a reflex increase in vagal tone and reduction in sympathetic activity. Abrupt termination of a tachycardia episode suggests a high likelihood of supraventricular re- entry tachycardia. In such cases, the patient should be referred to a cardiologist to assess indications for catheter ablation.

Assessment of the Relationship with Physical or Emotional Stress

An important aspect of evaluating patients with rhythm disorders is determining their relationship with physical or emotional stress. An increase in extrasystoles or the appearance of tachyarrhythmias during exertion suggests a possible association with myocardial ischaemia, requiring further investigation.

Conversely, “benign” arrhythmias typically disappear during exertion and reappear at rest. This distinction assists in differentiating “adrenergic” from “vagal” arrhythmias, particularly paroxysmal atrial fibrillation.

Vagal paroxysms of AF typically occur in middle- aged men, often after heavy meals or occasionally alcohol. They peak in the evening or at night, are not accompanied by marked tachycardia, and may coexist with atrial flutter. They tend to appear, cease, and recur over many years without progressing to permanent AF.

Adrenergic paroxysms of AF are more characteristic of patients with structural heart disease, especially coronary artery disease, often in older age. They occur against a background of tachycardia, during physical or emotional stress, and are usually accompanied by pronounced tachycardia.

Laboratory and Additional Evaluation

A mandatory component of the general clinical assessment of patients with rhythm disorders includes measurement of serum potassium and sodium, and biochemical markers reflecting liver and kidney function. At the first detection of a tachyarrhythmia (particularly atrial fibrillation), thyroid gland palpation and, if necessary, thyroid- stimulating hormone (TSH) testing should be performed.

Electrocardiographic Diagnosis. In most cases, the ECG method allows reliable identification of the type of arrhythmia. Whenever possible, a 12- lead ECG should be recorded during the arrhythmic episode, as P waves, flutter waves, or components of the QRS complex may be isoelectric in certain leads. Longer- term rhythm recording should be performed in the leads in which P waves are easily distinguishable, typically lead V1 or lead II .

During the initial assessment of patients with suspected tachyarrhythmias, attention should be paid to the presence of signs of ventricular pre- excitation. If such signs are detected, specialised electrophysiological evaluation is required to verify the diagnosis. A similar diagnostic approach is followed in cases of syncopal episodes associated with rhythm disorders. In other situations, the primary diagnostic task is simply to document an episode of tachyarrhythmia. For this purpose, the general practitioner may routinely use the simple and accessible method of fragmentary ECG monitoring .

Fragmentary (Event) Monitoring. This method is primarily used to document rhythm or conduction disturbances that occur relatively infrequently, for example weekly or monthly. In such cases, “event monitoring” is employed. In the outpatient setting, the patient continuously carries a small, simple device that allows them to record a single- channel ECG whenever symptoms occur. The ECG is then transmitted by telephone or email to the physician for analysis.

In “loop” monitoring, the recorder remains attached to the chest for several weeks. The device continuously stores only a short segment of ECG (e.g., the last minute), and activation of the device preserves this segment for subsequent analysis. This enables the physician to evaluate the ECG not only after the onset but also immediately before the occurrence of the arrhythmia. Loop recorders are most appropriate for infrequent, relatively mild but persistent symptoms likely caused by rhythm disorders. Unlike 24- hour Holter monitoring, this method is fully accessible for use by the general practitioner.

Possible Relationships Between Arrhythmias and Clinical Symptoms

Symptoms occur simultaneously with documented arrhythmias, which are the likely cause.

Subjective symptoms occur without any arrhythmia being recorded.

Documented arrhythmias occur without clinical symptoms.

Neither arrhythmias nor symptoms occur during the monitoring period.

The first and second scenarios have high diagnostic value, as they allow a clear determination of whether symptoms are related to the arrhythmia and may guide clinical decisions or specialised investigations.

In the third scenario, it is difficult to determine whether the arrhythmia is related to previously reported symptoms. It is possible, for example, that the symptoms were caused by a similar arrhythmia of greater severity or duration.

The fourth scenario indicates insufficient monitoring duration. In the third and fourth scenarios, electrophysiological study may sometimes be indicated.

Interpretation of ECG Findings

When interpreting 12- lead ECGs and fragmentary monitoring, it must be remembered that some rhythm and conduction disturbances may occur in practically healthy individuals . In particular, no specific treatment is usually required for:

- isolated, monomorphic supraventricular or ventricular extrasystoles
- migration of the atrial pacemaker
- sinus bradycardia (especially in young individuals and athletes)
- first- degree AV block
- second- degree AV block, Mobitz type I
- second- degree sinoatrial block, type I, with pauses up to 2 seconds between ventricular beats.

Conversely, the detection of certain arrhythmias or conduction disturbances precludes considering the patient healthy, even in the complete absence of symptoms. These include:

- ventricular tachycardia
- supraventricular tachycardias
- atrial fibrillation or atrial flutter
- pauses longer than 2.5–3 seconds
- second- degree AV block, Mobitz type II
- third- degree AV block
- second- degree sinoatrial block, type II
- sinus node arrest
- active or passive atrial, junctional, or idioventricular rhythms.

When these arrhythmias or conduction disturbances are identified, patients require further evaluation, appropriate treatment, or clinical follow- up.

SUPRAVENTRICULAR PAROXYSMAL TACHYCARDIA

Supraventricular paroxysmal tachycardia (SVPT) is a paroxysmal increase in heart rate exceeding 160 beats per minute, with the source of automaticity located in the atria or the atrioventricular (AV) junction. AV- nodal re- entry tachycardia (AVNRT) occurs predominantly in women of young and middle age without structural heart disease. The paroxysm is poorly tolerated by patients. During an episode, the patient typically experiences palpitations, and may also report weakness, a sensation of discomfort or chest pain, dyspnoea, or fear. Prolonged episodes in the presence of organic heart disease are accompanied by clinical and haemodynamic manifestations (hypotension, angina, acute left ventricular failure) .

A graphical representation of sinoatrial block is shown in Figure 8.

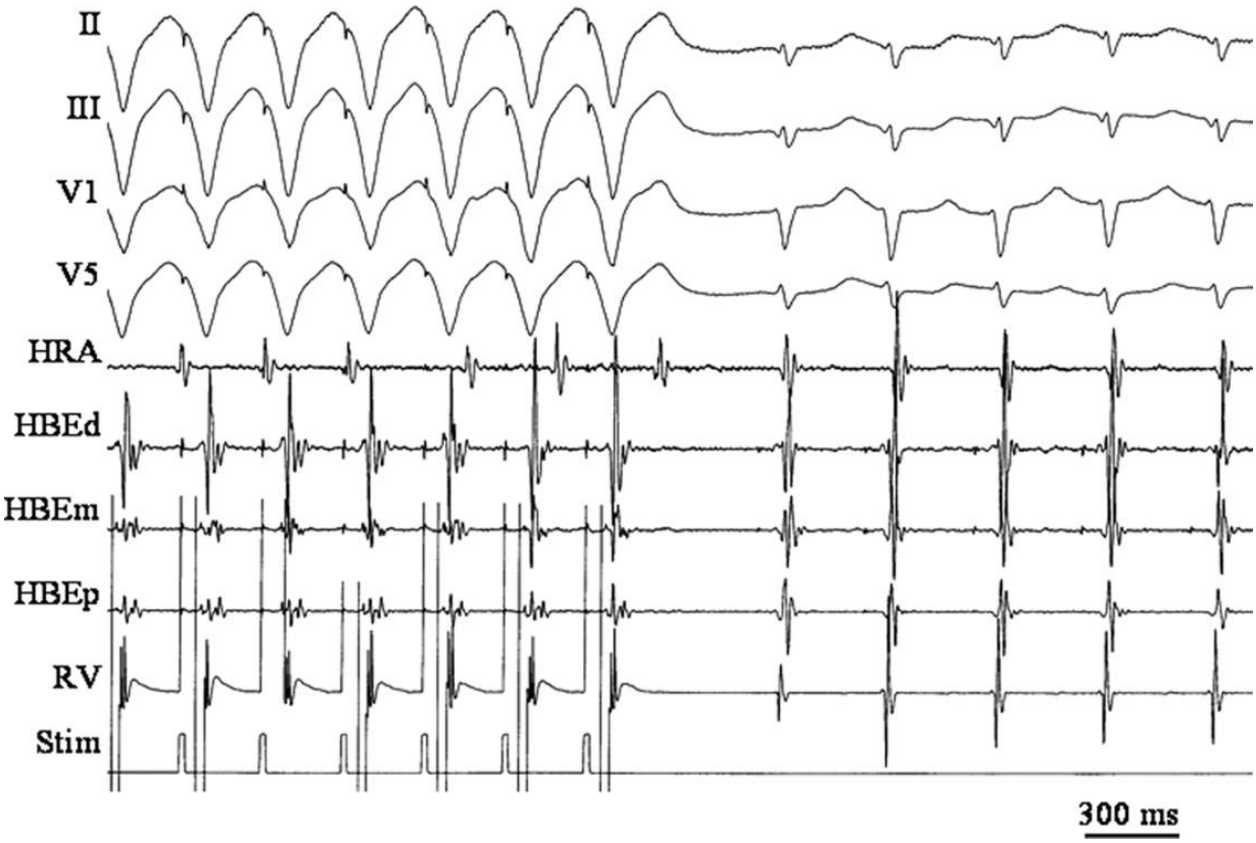

Figure 8. Typical paroxysm of supraventricular paroxysmal tachycardia

Image source: https://www.heartrhythmjournal.com/article/S1547-5271(06)02229-6/abstract

Supraventricular paroxysmal tachycardia (SVPT), according to its mechanism of onset, is classified into the following types:

- Paroxysmal AV- nodal tachycardia (macro–re- entry) – the arrhythmia begins and terminates abruptly. The paroxysm is triggered by atrial extrasystoles. The heart rate may vary from 150 to 225 beats per minute. The P wave is usually superimposed on the QRS complex or recorded immediately after it (RP < 0.09 s). This tachycardia is most common in individuals over 20 years of age.
- Atrial tachycardia (automatic or intra- atrial re- entry) – characterised by a gradual increase in heart rate (the "warm- up" phenomenon), and termination of the tachycardia is preceded by a gradual slowing of the atrial rhythm (the "cool- down" phenomenon).

The atrial rate is 100–200 beats per minute.

P waves are always located before the QRS complex and differ in morphology from the normal sinus P wave; PP intervals are regular.

The non- sustained form occurs more frequently in patients without organic myocardial disease, whereas in myocardial infarction and cor pulmonale, a sustained form of atrial tachycardia is observed.

- Multifocal (polytopic) atrial tachycardia – an irregular rhythm with a gradual onset, arising from multiple atrial ectopic foci. The heart rate (>100 beats per minute) is only slightly higher than the baseline rhythm.

Characteristic features include non- sinus P waves of different morphologies and variable PP, PQ, and RR intervals. Aberrant ventricular conduction is often present.

Multifocal atrial tachycardia is frequently misinterpreted as atrial fibrillation.

Reducing the heart rate is difficult because all impulses are conducted to the ventricles.

It occurs in older patients, in COPD, acute cor pulmonale, heart failure, after surgery, in sepsis, pulmonary oedema, diabetes mellitus, and with theophylline toxicity.

ECG Characteristics

- RR intervals < 20–25 mm
- QRS complexes ≤ 0.1 s, resembling those of sinus rhythm
- occasionally, a modified P wave is detected before each QRS complex

A graphical representation of atrial tachycardia is shown in Figure 9.

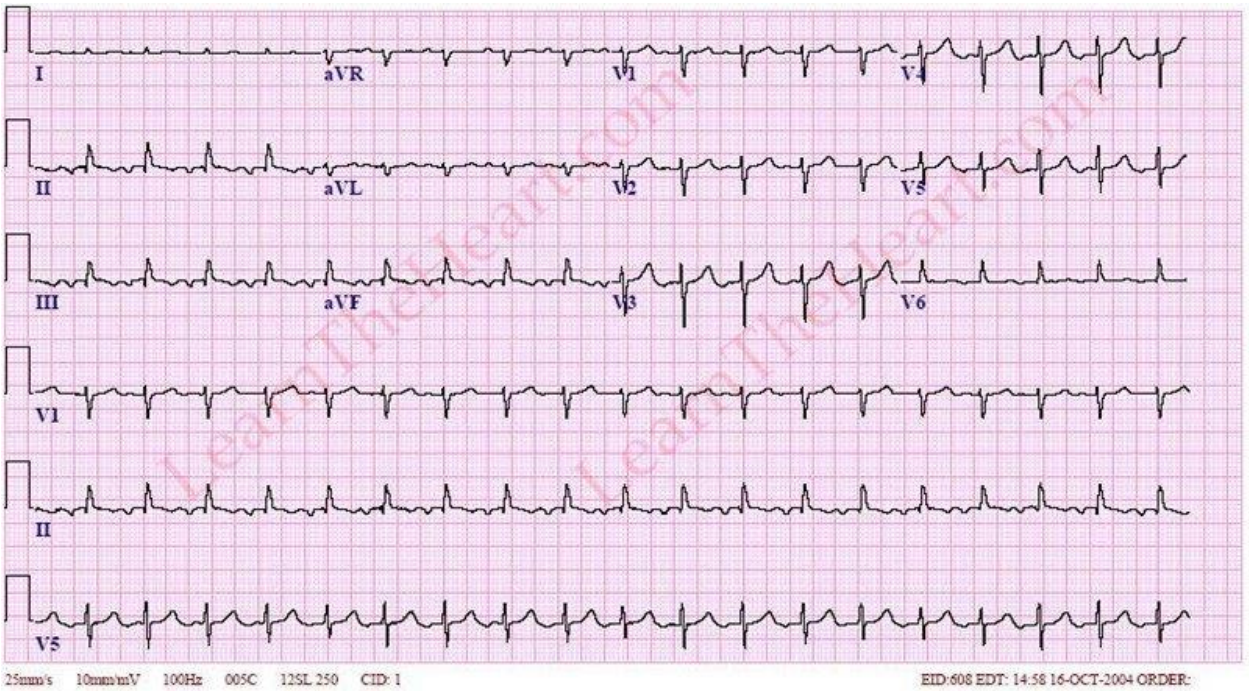

Figure 9. Atrial tachycardia

Image source: https://www.healio.com/cardiology/learn-the-heart/ecg-review/ecg-archive/atrial-tachycardia-ecg-example-1

Emergency Care

1. Vagal manoeuvres:

- carotid sinus massage
- Valsalva manoeuvre **(**breath-holding and straining at the height of inspiration)
- immersion of the face in cold water **or** swallowing cold water

2. If vagal manoeuvres are ineffective, antiarrhythmic medicinal agents are administered:

- adenosine triphosphate (ATP) 1% 1–2 ml intravenously as a rapid bolus, **or** adenosine 0.3% 2 ml. If ineffective after 2 minutes, adenosine is repeated at a dose of 4 ml. The effect appears within 3–5 minutes**.**
- verapamil (phenoptin**)** 0.25% 2–4 ml intravenously diluted in 10–20 ml of 0.9% sodium chloride over 3–5 minutes under blood pressure monitoring. If necessary, verapamil may be repeated after 10 minutes at a dose of 2–4 ml.

3. If the above measures fail, after 20 minutes one of the following antiarrhythmic agents is initiated:

- amiodarone **5**% 6 ml diluted in 200 ml of 5% glucose intravenously by infusion over 60 minutes. If sinus rhythm is not restored, the dose is continued at 15 mg/kg intravenously over the next 23 hours**.**
- procainamide (novocainamide) 10% 5–10 ml diluted in 10 ml of 0.9% sodium chloride or 10 ml of 5% glucose, administered intravenously slowly over 10–20 minutes.
- propafenone 0.35% 20 ml intravenously as a bolus at 1–2 mg/kg over 10 minutes.
- metoprolol (betaloc) intravenously at an initial dose of 5 ml at a rate of 1 mg/min, with the possibility of repeated administration at 5-minute intervals until a therapeutic effect is achieved. The total dose must not exceed 10–15 ml**.**

4. If pharmacological therapy is ineffective or if serious haemodynamic disturbances occur, electrical cardioversion is performed.

5. Patients with frequent recurrences of arrhythmia or with haemodynamic compromise during paroxysms should undergo radiofrequency catheter ablation **.**

ATRIAL FIBRILLATION

Atrial fibrillation (AF) is a form of supraventricular tachyarrhythmia characterised by uncoordinated atrial electrical activity, resulting in impaired atrial contractile function.

On the electrocardiogram (ECG), the characteristic features of AF are the absence of P waves (sometimes replaced by rapid oscillations or f waves) and typically irregular RR intervals.

The ventricular rate in AF depends on the electrophysiological properties of the AV node, the level of sympathetic and parasympathetic activity, and the effects of pharmacological agents.

Regular RR intervals may occur in the presence of AV block or concomitant ventricular tachycardia.

In patients with artificial pacemakers, temporary deactivation of the device may be required to detect atrial fibrillatory activity.

In cases of persistent tachycardia with an irregular rhythm and wide QRS complexes, the most likely diagnoses are AF with conduction over an accessory pathway or AF with concomitant bundle branch block.

A very high ventricular rate (>200 beats per minute) suggests the presence of an accessory conduction pathway.

Epidemiology

AF remains one of the most common cardiac rhythm disorders.

Paroxysmal or persistent AF affects 2.3 million people in the United States and 4.5 million in the European Union.

In the USA and EU, the hospitalisation rate for AF has increased by 66% over the past 20 years.

The cost of AF management in EU countries approaches €13.5 billion (US$15.7 billion).

Data on AF- related healthcare expenditure in the USA have not been published.

Aetiology

AF, particularly when it presents at a young age, may be a genetically determined disorder, independent of concomitant cardiovascular disease.

Risk factors for AF include:

- Arterial hypertension, especially with left ventricular hypertrophy
- Coronary artery disease, including myocardial infarction
  - schaemic mitral regurgitation (due to papillary muscle ischaemia) may markedly increase left atrial pressure and precipitate AF
- Valvular heart disease of rheumatic and non- rheumatic origin (predominantly conditions associated with elevated left atrial pressure):

a) mitral valve prolapse with mitral regurgitation

b) aortic regurgitation

c) aortic stenosis

d) mitral stenosis

Cardiomyopathies (dilated, restrictive, hypertrophic)

Congenital heart disease, particularly atrial septal defect in adults

Acute pulmonary embolism (PE). In approximately 10% of patients, atrial fibrillation (AF) appears as the principal manifestation, most likely associated with hypoxaemia occurring during PE.

Acute myocarditis and acute pericarditis, particularly the constrictive form, may also precipitate AF .

Advanced age. Senile cardiac changes are associated with AF to an even greater extent than organic cardiac pathology (Framingham Study data).

Postoperative period following cardiac surgery. AF may result from changes in circulating blood volume, withdrawal of antihypertensive therapy, and increased sympathetic tone induced by pain stimuli.

Other rhythm disorders. AF may develop as a transformation of atrial flutter, atrial paroxysmal tachycardia, junctional paroxysmal tachycardia, and others.

Focal atrial tachycardia may lead to the development of so- called focal AF.

Extracardiac pathology. Pulmonary diseases:

- acute – pneumonia, pleuropneumonia, thoracic surgery

- chronic – COPD (particularly with excessive use of bronchodilators), obstructive sleep apnoea syndrome.

Alcohol consumption. The “holiday heart syndrome” refers to the occurrence of acute AF on the day following heavy alcohol intake (commonly at weekends).

Chronic alcohol misuse may lead to persistent AF.

Figure 10. Atrial fibrillation

Image source: https://www.aclsmedicaltraining.com/atrial-fibrillation

Thyroid pathology, primarily hyperthyroidism, including drug- induced hyperthyroidism, is a recognised cause of atrial fibrillation (AF).

Imbalance of the sympathetic and parasympathetic divisions of the autonomic nervous system may also precipitate AF: vagal AF, adrenergic AF, hypersympathicotonia in certain infectious diseases.

Electrolyte disturbances, particularly hypokalaemia, are important contributing factors. In a proportion of cases, the underlying cause cannot be identified (idiopathic AF).

Pathogenesis

The focal theory of AF onset proposes that focal triggers, located predominantly in the region of the pulmonary vein ostia, play a key role. This is supported by the observation that ablation in these areas effectively suppresses the arrhythmia.

The multiple- wavelet theory suggests that AF may be maintained by the chaotic circulation of numerous independent small wavelets of excitation throughout the atrial myocardium.

Classification

The following forms should be distinguished:

- First- diagnosed episode of AF, irrespective of whether it was symptomatic or self- terminating. It should be noted that the duration of an AF episode may be uncertain, and previous episodes may have gone unnoticed.
- Persistent AF – lasting more than 7 days, including episodes terminated by pharmacological or electrical cardioversion after 7 days.
- Long- standing persistent AF – an AF episode lasting more than 1 year, provided that a rhythm- control strategy has been chosen.
- Permanent AF – a form in which the joint decision of the physician and patient is not to pursue rhythm restoration. If this decision is later revised, the AF should be reclassified as long- standing persistent AF .

EHRA Symptom Classification

The EHRA (European Heart Rhythm Association) symptom classes for AF are presented in Table 8.

Table 8

EHRA Symptom Classes for Atrial Fibrillation [1]

Clas	Symptoms	Description
1	no	No symptoms
2a	mild	Mild symptoms; normal daily activity not affected
2b	moderate	Mild symptoms; normal daily activity not affected
3	severe	Severe symptoms; normal daily activity affected
4	disabling	Disabling symptoms; normal daily activity discontinued

Persistent AF may represent either the first manifestation of the arrhythmia or the logical progression of recurrent paroxysmal AF.

This category also includes cases of long- standing AF (e.g., lasting more than 1 year) in which cardioversion has been ineffective or not performed.

This form typically progresses to permanent AF.

The above terminology applies to AF episodes lasting >30 seconds, not associated with a reversible cause.

Clinical Classification of AF

- Secondary AF associated with structural heart disease – in patients with left ventricular systolic/diastolic dysfunction, long- standing hypertension with LV hypertrophy, and/or other structural cardiac abnormalities.
- Focal AF – in patients with recurrent runs of atrial tachycardia and short episodes of paroxysmal AF. Symptoms are often pronounced.

  Typical in younger patients with atrial ectopic activity that transforms into AF.

  Associated with local triggers in the pulmonary veins.
- Polygenic AF – in carriers of genetic polymorphisms associated with AF development. Characteristically presents at a young age.
- Monogenic AF – in patients with genetically determined cardiomyopathies, including channelopathies.
- Postoperative AF.
- AF in patients with mitral stenosis or prosthetic heart valves.
- AF in athletes – usually paroxysmal.

  Related to increased vagal tone and left atrial enlargement.

Associated Rhythm Disorders

AF may occur in isolation or in combination with other arrhythmias, most commonly atrial flutter or atrial tachycardias.

Atrial flutter may develop during treatment with antiarrhythmic drugs used to prevent AF recurrence.

Atrial flutter is a more organised rhythm disturbance than AF and is characterised on ECG by “saw- tooth” F waves, reflecting atrial activation at regular intervals.

F waves are most prominent in leads II, III, and aVF, and there is no isoelectric baseline between them.

Without treatment, the atrial rate usually ranges from 240 to 320 beats per minute, with downward- directed F waves in leads II, III, and aVF, and upward- directed F waves in lead V1.

The excitation wave in the right atrium may circulate in the opposite direction, producing positive F waves in leads II, III, and aVF, and negative F waves in V1.

Atrial flutter is usually associated with AV block, most commonly 2:1, resulting in a ventricular rate of 120–160 beats per minute, typically around 150 bpm.

Several types of atrial flutter are recognised, although no universally accepted classification exists.

Flutter may convert to AF, and AF may promote the development of flutter.

Both rhythms may appear simultaneously on ECG, reflecting dynamic changes in atrial activation.

Other Atrial Tachycardias

AV re- entry tachycardias and AV nodal re- entry tachycardias may also precipitate AF.

In other atrial tachycardias, P waves are clearly visible and separated by an isoelectric baseline in one or more ECG leads.

The morphology of the P wave may help identify the origin of the tachycardia.

A recently described unique form of atrial tachycardia, most commonly originating in the pulmonary veins, is characterised by a ventricular rate >250 beats per minute, often progressing to AF.

Electrophysiological studies with intracardiac mapping can assist in differentiating between various types of atrial arrhythmias and identifying their underlying mechanisms.

ESC 2024 CLINICAL GUIDELINES

The 2024 European Society of Cardiology (ESC) Guidelines for the Management of Atrial Fibrillation represent a major update and introduce a new patient-centered framework called AF-CARE. They were published in the *European Heart Journal* on 30 August 2024.

Key changes in the 2024 ESC AF Guidelines

1. AF-CARE replaces the ABC pathway

The new management pathway is:

- C – Comorbidity and risk factor management
  - Weight reduction
  - Blood pressure control
  - Diabetes management
  - Treatment of obstructive sleep apnea
  - Exercise
  - Alcohol reduction
  - Smoking cessation
- A – Avoid stroke and thromboembolism
  - Assess stroke risk.
  - Oral anticoagulation is recommended based on individual risk rather than AF type.
  - Direct oral anticoagulants (DOACs) remain first-line for most patients.
- R – Reduce symptoms
  - Choose rate or rhythm control according to symptoms, patient preference, and clinical characteristics.
  - Earlier rhythm-control strategies are encouraged when appropriate.
- E – Evaluation and dynamic reassessment
  - AF management should be reviewed regularly because stroke risk, symptoms, and comorbidities evolve over time.

2. Stroke prevention

Major recommendations include:

- Use validated stroke-risk assessment.
- Oral anticoagulation is recommended when stroke risk justifies treatment.
- DOACs are preferred over warfarin except in patients with:
    - mechanical prosthetic valves
    - moderate-to-severe rheumatic mitral stenosis
- Bleeding-risk scores should be used to identify modifiable bleeding risks, not to withhold anticoagulation by themselves.

3. Rhythm control

The guideline places greater emphasis on:

- Early rhythm control for suitable patients.
- Catheter ablation as an effective treatment for symptomatic AF and, in selected patients, as first-line therapy.
- Rhythm control may improve symptoms and reduce cardiovascular events in appropriate patients.

4. Lifestyle modification

Lifestyle intervention is considered a core part of AF treatment rather than an adjunct:

- Weight loss
- Regular physical activity
- Blood pressure optimization
- Alcohol reduction
- Sleep apnea treatment
- Smoking cessation

These interventions reduce AF burden and improve treatment outcomes.

5. Shared decision-making

The guidelines strongly recommend:

- Patient education
- Shared decision-making

- Multidisciplinary care involving cardiologists, primary care clinicians, nurses, and other healthcare professionals.

Diagnosis and Detection of AF

According to the 2024 ESC Guidelines for the Management of Atrial Fibrillation, the diagnosis and detection of AF have been updated to emphasize ECG confirmation, appropriate use of wearable technologies, and comprehensive evaluation after diagnosis.

1. Diagnostic criteria for atrial fibrillation

Class I Recommendation

Clinical AF requires ECG documentation before initiating risk stratification and AF management.

Acceptable ECG documentation includes:

- Standard 12-lead ECG
- Single-lead ECG recording
- Multiple-lead ECG recording

For recordings from ambulatory devices, an ECG tracing showing AF for ≥30 seconds is generally accepted as diagnostic, although the guideline acknowledges that the exact duration threshold is based mainly on expert consensus rather than robust evidence.

Important update

The ESC specifically states that:

- Photoplethysmography (PPG)-based devices (e.g., many smartwatches and fitness trackers) do not establish the diagnosis of AF.
- If AF is suspected by a smartwatch or other screening device, confirmation with an ECG recording interpreted by a clinician experienced in ECG rhythm analysis is required before diagnosing AF or starting treatment.

2. Detection of atrial fibrillation

The guideline recognizes several methods for AF detection:

ECG-based methods

- 12-lead ECG

- Hand-held single-lead ECG
- Patch ECG monitors
- Holter monitoring
- Event recorders
- Implantable loop recorders
- Cardiac implantable electronic devices (pacemakers/ICDs)

Non-ECG methods

- Smartwatches
- Fitness bands
- Smartphone camera applications
- Blood pressure monitors with AF detection algorithms

These devices are useful **for screening**, but abnormal findings require ECG confirmation.

3. Diagnostic evaluation after newly diagnosed AF

The ESC recommends a comprehensive assessment in every patient with newly diagnosed AF.

Essential evaluation

- Detailed medical history
  - AF pattern
  - Family history
  - Comorbidities
  - Stroke and bleeding risk factors
- Symptom assessment
- 12-lead ECG
- Laboratory investigations:
  - Complete blood count
  - Renal function
  - Liver function
  - Electrolytes
  - Glucose/HbA1c

- Thyroid function

- Transthoracic echocardiography (when it will guide management decisions)
- Assessment of patient-reported outcomes when appropriate.

4. Additional investigations in selected patients

Depending on the clinical scenario, further investigations may include:

- Ambulatory ECG monitoring to assess AF burden or ventricular rate
- Exercise ECG
- Transesophageal echocardiography (e.g., before cardioversion or to assess left atrial thrombus)
- Coronary CT angiography or ischemia testing
- Cardiac MRI
- Brain imaging or cognitive assessment.

5. AF screening recommendations

The 2024 ESC guideline supports opportunistic and targeted screening in populations at increased risk, especially older adults and those with cardiovascular risk factors.

Key principles include:

- ECG-based devices are preferred for screening.
- Consumer wearable devices may help identify possible AF.
- Any positive screening result must be confirmed with an ECG before treatment decisions are made.

Assessment of Thromboembolic Risk and Prevention of Thromboembolic Complications

The 2024 ESC Guidelines for the Management of Atrial Fibrillation introduce important updates to the assessment of thromboembolic risk and prevention of stroke/systemic embolism. The major changes include the adoption of the $CHA_2\ DS_2$ -VA score (removing female sex as a risk factor), stronger emphasis on appropriate anticoagulation, and minimizing bleeding risk without unnecessarily withholding anticoagulant therapy.

1. Assessment of thromboembolic risk

CHA_2 DS_2 -VA score (New ESC recommendation)

The ESC recommends using the **CHA_2 DS_2 -VA** score rather than CHA_2 DS_2 -VASc for routine stroke risk assessment.

Risk Factor	Points
Congestive heart failure/LV dysfunction	1
Hypertension	1
Age ≥75 years	2
Diabetes mellitus	1
Prior stroke/TIA/systemic embolism	2
Vascular disease (MI, PAD, aortic plaque)	1
Age 65–74 years	1

Female sex has been removed as a scoring component because it is considered a risk modifier rather than an independent stroke risk factor.

Oral anticoagulation recommendations:

CHA_2 DS_2 -VA Score	ESC Recommendation
0	No routine oral anticoagulation
1	Oral anticoagulation **should be considered** (Class IIa)
≥2	Oral anticoagulation **is recommended** (Class I)

Anticoagulation decisions should be individualized through shared decision-making after balancing thromboembolic and bleeding risks.

2. Choice of anticoagulant

Class I recommendations

- Direct oral anticoagulants (DOACs) are preferred over vitamin K antagonists (VKAs) for most patients with AF.
- VKAs remain indicated for patients with:
    - Mechanical prosthetic heart valves.
    - Moderate-to-severe rheumatic mitral stenosis.

Appropriate DOAC dosing

The guideline emphasizes:

- Use the approved evidence-based dose according to renal function, age, body weight, and interacting drugs.
- Do not reduce the DOAC dose off-label, as inappropriate dose reduction may increase stroke risk without meaningfully reducing bleeding.

3. Bleeding risk assessment

The ESC recommends assessing bleeding risk before and during anticoagulation, primarily to identify and correct modifiable risk factors.

Examples include:

- Uncontrolled hypertension
- Excess alcohol intake
- Concomitant NSAIDs
- Unnecessary antiplatelet therapy
- Poor INR control (for VKA users)
- Renal dysfunction
- Anemia

Bleeding risk scores should not be used alone to deny anticoagulation when stroke prevention is indicated.

4. Antiplatelet therapy

The guideline recommends:

- Do not use antiplatelet therapy alone (aspirin or aspirin plus clopidogrel) for stroke prevention in AF.
- Avoid combining oral anticoagulants with antiplatelet agents unless there is a clear indication (e.g., recent acute coronary syndrome or coronary stenting), and keep combination therapy as short as clinically appropriate.

5. Left atrial appendage occlusion (LAAO)

LAAO may be considered in selected patients:

- Those with AF who have a contraindication to long-term oral anticoagulation.
- Decisions should be individualized after multidisciplinary evaluation.

It is not recommended as a routine alternative to anticoagulation in patients who can safely receive oral anticoagulants.

6. Dynamic reassessment

Stroke risk should be reassessed regularly because it changes over time with:

- Increasing age
- New hypertension
- Diabetes
- Heart failure
- Vascular disease
- Previous thromboembolic events

Patients with initially low risk may later become candidates for anticoagulation.

Ventricular Rate Control

Experts emphasise the multidimensional importance of ventricular rate control, which is:

- the foundation of initial therapy in all patients with AF
- first- line therapy in asymptomatic patients or those with minimal symptoms
- mandatory when attempts to maintain sinus rhythm are ineffective, or when the risks of rhythm- control therapy outweigh the benefits

The established principle remains that the initial therapeutic target is to achieve a ventricular rate <110 bpm (“lenient control”).

Other guidelines (e.g., Canadian) recommend a lower threshold (<100 bpm), which may be justified when correctly interpreting the RACE II trial, the basis for this lower target.

According to the current ESC guidelines, intensification of therapy (to achieve a lower target rate) should be applied when symptoms persist, when left ventricular dysfunction is present, and - explicitly emphasised - when near- 100% biventricular pacing cannot be achieved in patients with cardiac resynchronisation therapy (CRT).

Choice of Rate- Control Medication

The choice of medication should depend on the left ventricular ejection fraction (LVEF), as previously recommended.

However, recommendations for patients with LVEF ≥40% have changed: digoxin is no longer recommended in this group.

This appears somewhat inconsistent with the findings of the RATE- AF trial, presented at ESC 2020 (on the same day as the AF guidelines).

In that study, elderly patients with permanent AF and heart failure but preserved LVEF had outcomes with digoxin that were not inferior, and in some respects superior, to those receiving bisoprolol.

It is also noteworthy - although not mentioned in the guidelines - that in patients treated with a β- blocker (unlike those receiving verapamil or diltiazem), dose escalation does not produce a proportionally greater reduction in ventricular rate.

Long- Term Rate Control

The guidelines reaffirm the important principle that antiarrhythmic drugs (Class I or III) should not be used for long- term rate control in patients with permanent AF (Class III/A). This also applies to amiodarone.

Unfortunately, in everyday practice, deviation from this principle remains common.

Restoration and maintenance of sinus rhythm constitute another therapeutic strategy for patients with atrial fibrillation (AF).

The principle has been retained that candidates for this strategy are patients with symptoms secondary to the arrhythmia.

An algorithm has been developed to facilitate decision- making.

The guidelines acknowledge that in some patients symptoms may be ambiguous (possibly unrelated to AF), or the patient may consider themselves asymptomatic, perhaps incorrectly.

In such cases, it may be reasonable to perform only a single cardioversion.

If it is ultimately concluded that the patient is indeed symptomatic (and that symptoms are AF- related), multiple factors should be evaluated when considering

a rhythm- control strategy. These include: younger age, first AF episode or short AF history, tachycardia- induced cardiomyopathy, normal or mildly enlarged left atrial volume, absence of significant comorbidities or only minimal structural heart disease, difficulty achieving adequate rate control, AF triggered by an acute illness, and—critically—patient preference.

Simultaneously with publication of the guidelines, results of the important randomised controlled trial EAST- AFNET 4 were presented at ESC 2020.

The study demonstrated that early initiation of rhythm- control therapy (using antiarrhythmic drugs or ablation), compared with conventional management (primarily rate control), was associated with a lower risk of cardiovascular events.

Acute Restoration of Sinus Rhythm

Acute restoration of sinus rhythm may be achieved by pharmacological or electrical cardioversion.

Based on the findings of the RACE 7 study, delayed cardioversion - up to 48 hours - is acceptable due to the high likelihood of spontaneous conversion in paroxysmal AF.

For pharmacological cardioversion, the same antiarrhythmic agents as previously recommended remain in use.

Despite numerous national publications supporting the efficacy and safety of antazoline, this drug has not been included in the guidelines.

The option of patient- initiated pharmacological cardioversion ("pill- in- the- pocket") has been retained (Class IIa/B).

Long- Term Use of Antiarrhythmic Drugs

Expert opinion regarding long- term antiarrhythmic therapy for maintaining sinus rhythm remains unchanged.

The following well- established principles are reiterated:

- The efficacy of antiarrhythmic drugs in AF is moderate.
- Antiarrhythmic drugs do not eliminate AF recurrences; they only reduce their frequency.
- Proarrhythmia and extracardiac adverse effects are common.

- Therapeutic decisions should prioritise safety profile over expected efficacy.

More detailed rules for selecting a specific drug remain similar to previous guidelines and consider three clinical scenarios:

- No structural heart disease or minimal structural abnormalities
- Coronary artery disease, significant valvular disease, or heart failure with preserved LVEF (this is a new category)
- Heart failure

The list of antiarrhythmic drugs is limited and includes propafenone, sotalol, and amiodarone. A substantial downgrading of the recommendation class for sotalol (from Class I to Class IIb) has been introduced.

This is explained by the risk of proarrhythmic effects associated with sotalol.

To the best of current knowledge, no new publications have emerged recently that would further strengthen concerns regarding this risk.

When prescribing sotalol, it must be remembered that the drug is almost entirely renally excreted in unchanged form.

Therefore, dose reduction or complete avoidance is required in patients with impaired renal function.

Unfortunately, the guideline authors did not avoid an inconsistency: in one section they state that the drug is contraindicated when CrCl <30 ml/min, while elsewhere in the same table they list the contraindication as CrCl <50 ml/min.

Although amiodarone is not a new drug, many controversies regarding its dosing remain.

In the ESC guidelines under discussion, experts again recommend intensive loading (although less aggressive than in the 2016 guidelines):

600 mg daily for 4 weeks, followed by a reduction to 200 mg daily.

However, the current Summary of Product Characteristics provided by the manufacturer recommends 600 mg daily for only one week.

Unfortunately, no new antiarrhythmic drug for AF management has become available.

It is therefore noteworthy that the role of β- blockers in rhythm- control strategy has been further reduced - essentially ignored.

Previously, their use was recommended in patients with adrenergic AF and after the first AF episode.

It is worth recalling that randomised trials in post- cardioversion patients demonstrated that long- acting metoprolol was more effective than placebo in maintaining sinus rhythm (51% vs 40%, 46% vs 26%).[6 – 7]

Indications for Catheter Ablation

Indications for transcatheter AF ablation have changed, though not dramatically expanded.

The main indications (Class I recommendations) continue to apply to symptomatic AF patients in whom at least one Class I or III antiarrhythmic drug has been ineffective or poorly tolerated.

In the current guidelines, this recommendation applies not only to paroxysmal AF, but also to persistent AF, particularly in patients without major risk factors for post- ablation recurrence.

These risk factors include:

- markedly enlarged left atrial volume
- advanced age
- long- standing AF
- renal dysfunction
- other cardiovascular risk factors.

Ablation is also recommended (Class I, with stronger evidence than in 2016) in patients with a high likelihood of left ventricular dysfunction due to tachyarrhythmia.

Ablation may be considered as first- line therapy:

- in paroxysmal AF (same as in previous guidelines, Class IIa/B)
- in persistent AF, although with a weaker recommendation (Class IIb/C), and only in the absence of the above- listed recurrence risk factors.

Two new Class IIa/B indications have been added:

- when β- blockers used for symptom control in paroxysmal or persistent AF are ineffective or not tolerated;
- in selected patients with heart failure with reduced LVEF, to improve survival and reduce heart- failure hospitalisations.

The previously introduced indication for ablation in patients with bradycardia dependent on AF or symptomatic pauses after AF termination has been retained (Class IIa/C), to avoid pacemaker implantation.

Surgical Ablation

The guidelines were developed jointly with the European Association for Cardio- Thoracic Surgery (EACTS).

Therefore, considerable attention is given to surgical AF ablation.

Patient selection should be performed by a multidisciplinary team consisting of:

- a cardiologist experienced in arrhythmia management
- an electrophysiologist
- a cardiac surgeon performing AF ablation procedures.

Surgical ablation should be considered in symptomatic patients with paroxysmal or persistent AF refractory to antiarrhythmic drugs, primarily after failed catheter ablation or when the likelihood of catheter ablation success is low (Class IIa/B).

The procedure should be minimally invasive.

Management of Comorbidities and Risk Factors

All patients with atrial fibrillation (AF) should undergo systematic identification of risk factors and comorbidities, followed by appropriate management of these conditions (Class I/B).

As a priority, experts recommend adequate blood pressure control in patients with AF and arterial hypertension to reduce the risk of AF recurrence, stroke, and bleeding (Class I/B).

Recommendations for modifying other risk factors - such as obesity, alcohol misuse, low physical activity, and obstructive sleep apnoea - carry a lower recommendation class (IIa/B or IIa/C).

Special Situations

The current ESC guidelines describe numerous special clinical situations associated with AF and outline management strategies for each. These include:

- heart failure
- valvular heart disease
- chronic kidney disease
- peripheral vascular disease
- endocrine disorders
- gastrointestinal diseases, among others.

Due to the extensive list of conditions, the limited structure of the document, and often insufficient available evidence, the information provided in this section is sometimes not comprehensive.

Based on clinical trials evaluating anticoagulant therapy (dabigatran in RE- DUAL PCI, rivaroxaban in PIONEER- AF, apixaban in AUGUSTUS, edoxaban in ENTRUST- AF PCI) and antiplatelet therapy in patients with AF and various forms of coronary artery disease, clear recommendations have been formulated.

If a patient has low stent- thrombosis risk, concerns regarding bleeding risk, and has undergone uncomplicated PCI, early discontinuation of aspirin (≤1 week) is recommended, with continuation of an oral anticoagulant plus a P2Y12 inhibitor (primarily clopidogrel):

- up to 12 months in acute coronary syndrome
- up to 6 months in chronic coronary syndrome (Class I/B).

Triple therapy for >1 week but ≤1 month should be considered when the risk of stent thrombosis outweighs bleeding risk (Class IIa/C).

These recommendations are largely consistent with the ESC 2019 Chronic Coronary Syndromes Guidelines and the 2020 NSTE- ACS Guidelines.

ATRIAL FLUTTER

According to the 2024 European Society of Cardiology (ESC) Guidelines for the Management of Atrial Fibrillation, atrial flutter (AFL) is defined as:

A macro-reentrant atrial tachycardia characterized by regular atrial activation, most commonly involving the cavotricuspid isthmus (typical atrial flutter), with an atrial rate usually of 240–340 beats/min.

This arrhythmia occurs in the setting of organic myocardial disease (rheumatic heart disease, mitral valve defects, myocarditis, coronary artery disease, acute myocardial infarction).

At the onset of rhythm disturbance, palpitations become more frequent, and patients may experience marked weakness, dizziness, presyncope, or syncope, accompanied by a sharp drop in blood pressure.

These symptoms may collectively lead to loss of consciousness.

ECG Findings

- Absence of an isoelectric baseline
- Presence of atrial F waves at 220–350 beats per minute, with a characteristic saw- tooth pattern in leads V1–V2, II, III, and aVF, with equal F–F intervals
- Normal, unchanged QRS complexes, each preceded by multiple atrial waves (e.g., 2:1, 3:1, 4:1 conduction)

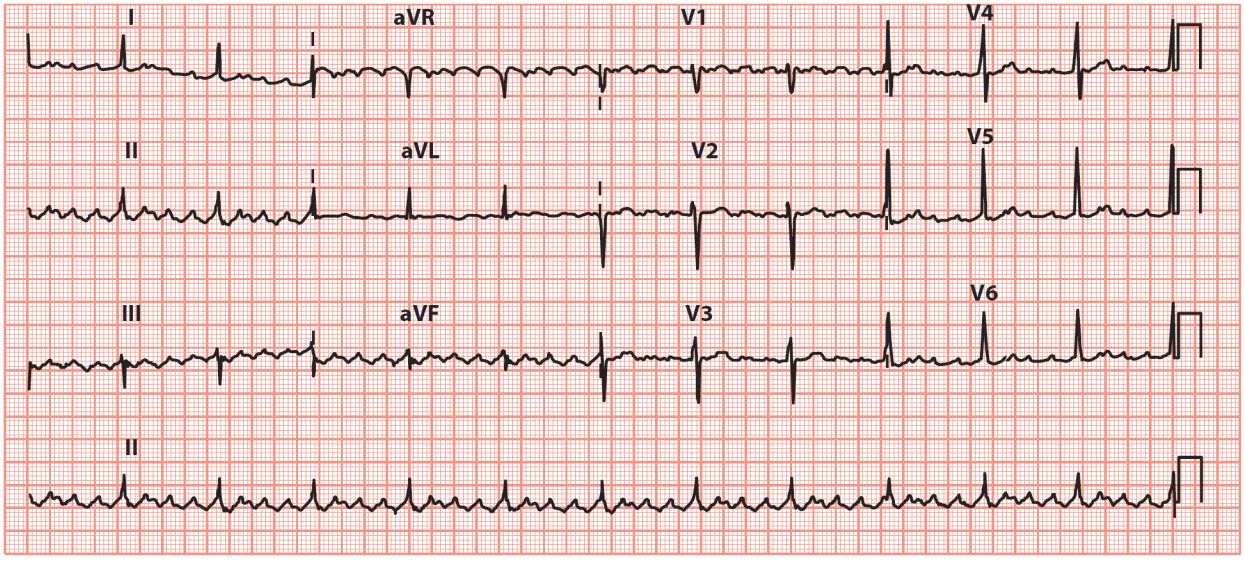

Figure 11. Atrial flutter with 4-1 AV Block

Image source: https://manualofmedicine.com/ecgs/atrial-flutter-ecg-interpretation/

Aetiopathogenesis

Organic heart disease (rheumatic valvular defects, coronary artery disease, arterial hypertension, sick sinus syndrome, previous cardiac surgery, or myocarditis), hyperthyroidism, and chronic lung disease are common underlying conditions.

Atrial flutter (AFL) is often a consequence of an acute pathological process, such as acute myocardial infarction, pneumonia, or surgical intervention.

Depending on the electrophysiological mechanism, AFL is classified as:

- typical (the most common form)
- typical counter- clockwise
- atypical.

Determining the type is essential for transcatheter ablation planning.

Clinical Presentation and Typical Course

The arrhythmia may be paroxysmal or persistent, most often recurrent, and may progress to permanent atrial fibrillation (AF) over time.

AFL episodes are frequently accompanied by tachyarrhythmia, are often resistant to antiarrhythmic drugs, and significantly impair quality of life.

AFL is observed in approximately one- third of patients with AF.

During treatment with Class Ic antiarrhythmic drugs or amiodarone, AF may often transform into AFL.

If an AFL episode is associated with an acute illness, it usually does not recur once the illness resolves.

Symptoms and objective findings largely depend on the underlying disease and its severity: palpitations (most common), dyspnoea, weakness or chest discomfort, occasionally asymptomatic, rapid, regular ventricular rate ≈150 bpm, jugular venous pulsation at ≈300 bpm, carotid sinus massage typically slows the ventricular rate only during compression.

During physical exertion, or in patients with highly efficient AV conduction, 1:1 AV conduction may occur (instead of the usual 2:1), leading to hypotension and syncope.

Emergency Care

Transoesophageal atrial pacing – a highly effective method for treating typical atrial flutter.

To reduce ventricular rate:

- digoxin 0.025% 1–2 ml twice daily with potassium–magnesium supplementation
- verapamil (phenoptin) 0.25% 2–4 ml IV bolus diluted in 10–20 ml 0.9% NaCl
- metoprolol IV, initial dose 5 ml at 1 mg/min.

If antiarrhythmic drugs are ineffective, electrical cardioversion is used to terminate the AFL episode.

Long- Term Management

The principles of drug selection are similar to those for AF; however, the effectiveness of antiarrhythmic drugs is lower than that of ablation (typically <40%).

For typical AFL, indications for transcatheter ablation are broad; it may be offered even after the first well- tolerated episode.

For atypical AFL, an initial attempt with antiarrhythmic drug therapy is recommended.

PAROXYSMAL VENTRICULAR TACHYCARDIA

Paroxysmal ventricular tachycardia (VT) is characterised by sudden episodes of palpitations with a ventricular rate of 140–220 beats per minute and a regular ventricular rhythm.

It occurs predominantly in the setting of severe diffuse myocardial disease.

VT paroxysms are accompanied by marked haemodynamic disturbances and are frequently life- threatening.

Ventricular tachycardia may be sustained or non- sustained.

Non- sustained VT usually terminates rapidly and is often asymptomatic, making it detectable only through prolonged ECG monitoring.

However, some patients experience typical arrhythmic symptoms such as palpitations, chest pain, dizziness, or syncope.

Sustained VT presents, in addition to the usual symptoms, with features characteristic of this arrhythmia:

- beat- to- beat fluctuations in systolic blood pressure
- reduced jugular venous pulsation frequency compared with the pulse.

The ventricular rate in sustained VT is typically 100–220 bpm.

Rates exceeding this range indicate ventricular flutter.

Haemodynamic compromise may include:

- tachycardia- induced hypotension
- sweating
- varying degrees of arterial hypotension
- altered consciousness (stupor, agitation, loss of consciousness).

Clinical deterioration may progress to cardiogenic shock or sudden circulatory arrest.

ECG Findings

- QRS complex >0.12 s, deformed, with discordant ST- segment and T- wave orientation, predominantly either positive or negative
- Sudden onset of a rapid, regular ventricular rhythm at 140–220 bpm
- In lead V1, typical ventricular complexes of the R, QR, or RR type
- In lead V6, ventricular complexes of the QR or QS type.

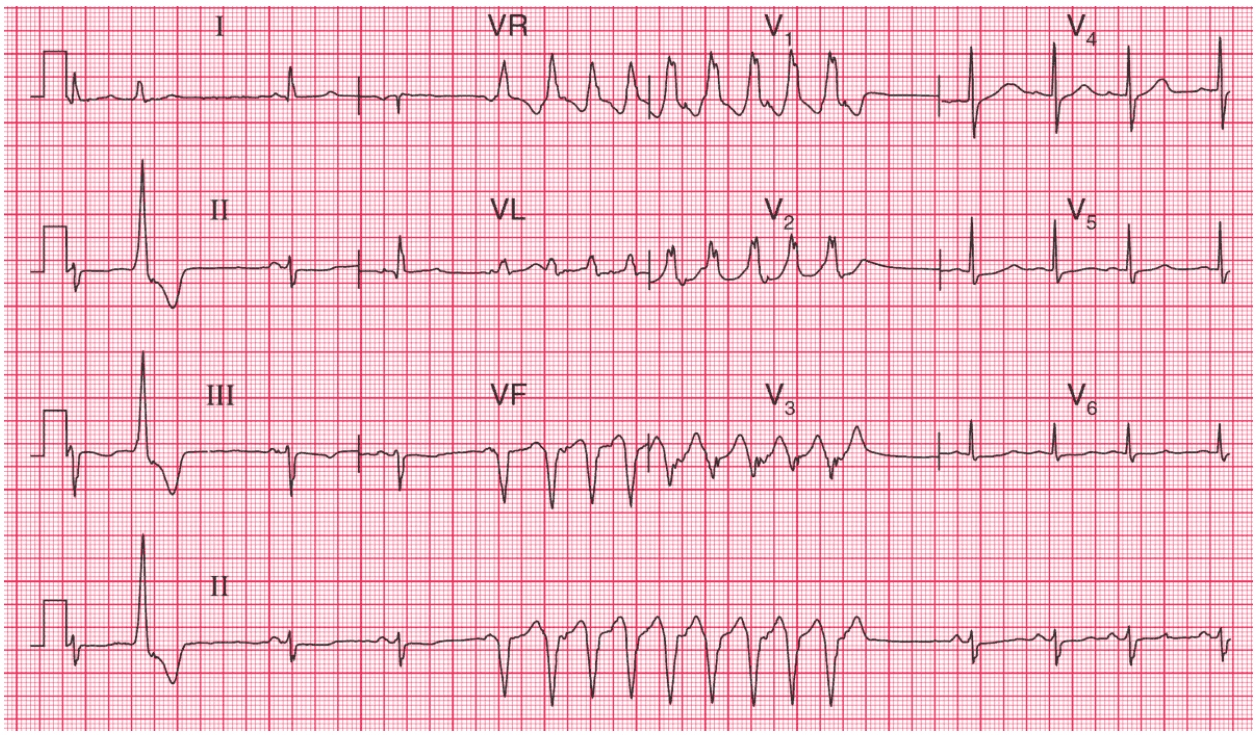

Figure 12. Sinus Rhythm with Paroxysmal Ventricular Tachycardia

Image source: https://manualofmedicine.com/ecgs/ecg-case-99-sinus-rhythm-with-paroxysmal-ventricular-tachycardia/

Emergency Care

Electrical cardioversion is performed to terminate the paroxysm.

Prior to the procedure, the patient is placed into medication- induced sleep (propofol 1% Fresenius 40 mg IV administered slowly as a bolus, followed by 20 mg every 2 minutes until adequate depth of anaesthesia is achieved).

Pharmacological therapy:

- Amiodarone 5% 6 ml IV infusion diluted in 200 ml of 5% glucose over 60 minutes
- Lidocaine 2% 4 ml IV bolus at a dose of 1 mg/kg over 3–4 minutes;
- if ineffective, a repeat dose of 0.5–0.75 mg/kg may be administered every 5–10 minutes after 10–15 minutes
- Propafenone 2 mg/kg IV bolus over 10–20 minutes
- Procainamide 10% 5–10 ml diluted in 10 ml of 5% glucose or 0.9% NaCl, administered IV over 10–20 minutes, up to a total dose of 1–1.5 g under blood pressure and ECG monitoring.

To enhance the effect of antiarrhythmic drugs, potassium–magnesium preparations (Panangin) or magnesium sulphate may be administered

Role of the General Practitioner

A considerable number of essential aspects of diagnosing and managing ventricular arrhythmias fall within the competence of the general practitioner (GP). These include:

General clinical assessment

Identification of probable aetiological factors and triggers

Evaluation of the relationship between arrhythmia and physical or emotional stress

Assessment of clinical and prognostic significance

The electrocardiographic method, including intermittent ECG monitoring, remains the cornerstone of initial diagnosis of ventricular arrhythmias.

The GP makes key decisions regarding the strategy and tactics of AF management, the most common tachyarrhythmia.

Their responsibilities include:

- Assessment of thromboembolic risk
- Monitoring the effectiveness and safety of antithrombotic and antiarrhythmic therapy in outpatient settings
- Determining the need for hospitalisation

There is a clear need to improve the training level of general practitioners in the diagnosis and management of ventricular arrhythmias.

ACUTE HEART FAILURE

Acute heart failure (AHF) is defined as the rapid onset or worsening of symptoms and signs characteristic of impaired cardiac function.

It is a life- threatening condition requiring urgent intervention and typically leads to emergency hospitalisation.

In most cases, AHF develops as a result of deterioration in patients with previously diagnosed heart failure (HF).

Cardiac dysfunction may be predominantly systolic (HF with reduced ejection fraction – HFrEF) or diastolic (HF with preserved ejection fraction – HFpEF), and may be triggered by arrhythmias, or by changes in preload and afterload.

AHF may also represent the first manifestation of HF (de novo AHF) in a patient without prior cardiac dysfunction.

In patients with pre- existing HF, a precipitating factor or trigger is often identified (e.g., arrhythmia, discontinuation of diuretics, volume overload, severe hypertension).

Speed of Symptom Progression

The rate of symptom progression varies widely:

- over several days or weeks (e.g., worsening dyspnoea or oedema)
- within hours or even minutes (e.g., in acute myocardial infarction)

Patients may present with a spectrum of conditions ranging from life- threatening pulmonary oedema or cardiogenic shock to states characterised primarily by worsening peripheral oedema.

Aetiopathogenesis

AHF may develop de novo (in individuals without prior cardiac dysfunction) or as acute decompensation of chronic HF.

The increasing proportion of elderly individuals in most populations, together with improved survival after acute myocardial infarction (AMI), has led to a substantial rise in the number of patients with chronic HF and in hospitalisations due to HF decompensation.

Ischaemic heart disease is the underlying cause of AHF in 60–70% of cases, particularly in older patients.

In younger individuals, AHF more commonly results from dilated cardiomyopathy, arrhythmias, congenital or acquired valvular defects, or myocarditis.

Each year in the United States, AHF is the primary discharge diagnosis in approximately 1 million hospitalisations and a secondary diagnosis in an additional 2 million.

The rate of early re-hospitalisation is high: ≈20% within 30 days, ≈50% within 6 months. The average length of hospital stay is 10 days.

Importantly, over the past two decades, 30- day mortality in AHF has remained unchanged at >10%.

Hospitalisation for AHF should therefore be regarded as a serious emergency, given: in- hospital mortality: ~8%, 6- month mortality: 25–30%. Mortality is particularly high in AMI complicated by HF, with 12- month mortality reaching 30%.

In pulmonary oedema, in- hospital mortality is 12%, and 1- year mortality is 40%.

These data are supported by large registries such as the ADHERE Registry, EuroHeart Survey Programme, and others.

Causes and Underlying Conditions Leading to Acute Heart Failure (AHF)

- Decompensation of chronic heart failure (e.g., in cardiomyopathy)
- Acute coronary syndrome:
  - myocardial infarction / unstable angina with severe ischaemia and ischaemic myocardial dysfunction
  - mechanical complications of MI
  - right ventricular infarction
- Hypertensive crisis

- Acute arrhythmia (ventricular tachycardia, atrial fibrillation or flutter, other supraventricular tachycardias, bradyarrhythmias of various aetiologies)
- Valvular regurgitation (endocarditis, chordae tendineae rupture, worsening of pre- existing regurgitation)
- Severe aortic stenosis
- Severe acute myocarditis
- Cardiac tamponade
- Aortic aneurysm rupture
- Peripartum cardiomyopathy
- Non- cardiovascular comorbidities:
  - reduced adherence to pharmacotherapy
  - volume overload
  - infections, especially pneumonia and septicaemia
  - severe stroke
  - postoperative state
  - renal dysfunction
  - bronchial asthma
  - dependence on psychotropic substances
  - alcohol dependence
  - phaeochromocytoma
- High- output states:
  - septicaemia
  - thyrotoxic crisis
  - anaemia
  - shunt syndromes
- Effects of medications and other chemical substances (poisoning with beta- blockers, cardiac glycosides, etc.)

Classification

In a patient with AHF, one of the following clinical states may be identified:

(I) Acute decompensated heart failure

De novo or decompensation of chronic HF, with typical symptoms and signs of AHF of moderate severity, not meeting criteria for cardiogenic shock, pulmonary oedema, or hypertensive emergency.

(II) Hypertensive AHF

Symptoms and signs of HF accompanied by markedly elevated blood pressure with relatively preserved left ventricular function.

Chest X- ray often shows no signs of alveolar pulmonary oedema.

(III) Pulmonary oedema

Confirmed on chest radiography, accompanied by severe respiratory distress, orthopnoea, and pulmonary crackles.

Pre- treatment oxygen saturation is usually <90%.

(IV) Cardiogenic shock

Defined as inadequate perfusion of vital organs and tissues due to reduced cardiac pump function after correction of preload.

Although haemodynamic definitions vary, cardiogenic shock typically presents with:

- systolic BP <90 mmHg or mean arterial pressure reduced by >30 mmHg
- urine output <0.5 ml/(kg·h)
- heart rate >60 bpm

This occurs regardless of the presence or absence of congestion.

Cardiogenic shock represents the extreme manifestation of low- output syndrome.

(V) Right ventricular failure

Characterised by low cardiac output due to right ventricular pump failure (myocardial injury or increased afterload - e.g., pulmonary embolism, bronchiolitis), with:

- elevated jugular venous pressure
- hepatomegaly
- hypotension

Other Classifications of the Acute Heart Failure (AHF) Syndrome

Additional classifications of AHF are used in coronary care units and intensive care units.

The Killip classification is based on clinical symptoms and chest radiography findings. It is validated for heart failure following acute myocardial infarction (AMI).

The “clinical severity” classification is validated for cardiomyopathies and is based solely on clinical signs. It is most commonly applied in decompensated chronic heart failure (CHF).

Killip Classification

- Killip Class I – No heart failure. No clinical signs of decompensation.
- Killip Class II – Heart failure. Diagnostic criteria include pulmonary crackles, gallop rhythm, pulmonary hypertension. Pulmonary congestion with moist crackles in the lower lung fields (below the scapular angle), unilateral or bilateral.
- Killip Class III – Severe heart failure. Pulmonary oedema with crackles over the entire lung surface.
- Killip Class IV – Cardiogenic shock. Hypotension (SBP < 90 mmHg), signs of peripheral vasoconstriction (oliguria, cyanosis, profuse sweating).

Clinical Severity Classification

This classification is based on the assessment of peripheral perfusion and pulmonary auscultation (congestion).

Patients are divided into:

Class I (Group A) – “warm and dry”

Class II (Group B) – “warm and wet”

Class III (Group L) – “cold and dry”

Class IV (Group C) – “cold and wet”

Its prognostic value has been demonstrated in patients with cardiomyopathy, and it is used to assess patients with decompensated CHF both in hospital and outpatient settings.

Clinical Syndrome of Acute Heart Failure

AHF is a clinical syndrome typically characterised by:

- reduced cardiac output
- tissue hypoperfusion
- elevated pulmonary capillary wedge pressure (PCWP) in left- sided failure
- systemic and pulmonary congestion.

Cardiac dysfunction may result from:

- systolic or diastolic myocardial dysfunction
- acute valvular dysfunction
- pericardial tamponade
- arrhythmias or conduction disturbances
- changes in preload or afterload.

Extracardiac Pathology Contributing to AHF

Multiple extracardiac conditions may precipitate AHF by altering cardiac loading conditions:

a) Increased afterload

– systemic or pulmonary hypertension

– massive pulmonary embolism

b) Increased preload

– expanded blood volume

– reduced fluid excretion in renal or endocrine disorders

c) High- output states

– infections

– thyrotoxicosis

– anaemia

– Paget's disease

Heart failure may also worsen pre- existing dysfunction of other organs and systems. Severe HF can itself induce multiorgan failure, a potentially fatal condition.

Clinical Variants of the Acute Heart Failure (AHF) Syndrome

AHF with congestion "below" the level of the lesion (left- or right- sided)

AHF with congestion below the level of the lesion may be:

- mild, presenting only with reduced exercise tolerance
- severe, with resting tissue hypoperfusion, manifested by:
  - weakness
  - confusion
  - dizziness
  - pallor with peripheral cyanosis
  - cold, clammy skin
  - low arterial blood pressure
  - thready pulse
  - oliguria / anuria

This clinical picture corresponds fully to cardiogenic shock .

This syndrome may be caused by various conditions:

a) acute coronary syndrome

b) acute myocarditis with signs of recent viral infection

c) acute valvular dysfunction, including chronic valvular disease, postoperative valve complications, infectious processes with possible bacterial endocarditis, chest trauma

d) pulmonary embolism

e) pericardial tamponade

Physical examination may provide important diagnostic clues, such as:

- jugular venous distension and pulsus paradoxus (pericardial tamponade)
- muffled heart sounds due to systolic dysfunction
- auscultatory abnormalities characteristic of valvular disease.

Emergency management

Emergency care in AHF with congestion below the lesion aims to increase cardiac output and improve tissue oxygenation.

This may be achieved through: vasodilators, fluid therapy to optimise preload, short- term inotropic support, mechanical circulatory support, such as intra- aortic balloon counterpulsation.

AHF with congestion “above” the level of the lesion (left- sided)

Left- sided heart failure with congestion above the lesion results from left ventricular dysfunction of varying severity:

- mild – slight exertional dyspnoea
- severe – pulmonary oedema with:
  - cough (sometimes with frothy sputum)
  - pallor or cyanosis
  - cold, clammy skin
  - normal or elevated arterial blood pressure

Moist crackles are heard throughout both lungs.

Chest radiography shows pulmonary congestion.

This type of AHF may develop in the following conditions

Myocardial dysfunction due to long- standing disease; myocardial ischaemia or infarction; dysfunction of the mitral or aortic valve; thrombotic obstruction of the atrioventricular orifice; malfunction of a prosthetic valve (thrombosis); arrhythmias and conduction disturbances; tumours of the left heart chambers.

Extracardiac causes include arterial hypertension, high- output states (anaemia, thyrotoxicosis), and neurogenic disorders (brain tumours or trauma).

During clinical cardiovascular examination, it is essential to assess:

- the apical impulse
- auscultatory findings
- presence of murmurs
- moist crackles in the lungs
- prolonged expiration (cardiac asthma).

Management of left- sided AHF with congestion above the lesion

Treatment includes:

- vasodilators
- diuretics
- bronchodilators
- opioid analgesics, if required.

Respiratory support is crucial. It may involve assisted ventilation with continuous positive airway pressure (CPAP), and in some cases tracheal intubation with mechanical ventilation.

AHF with congestion above the lesion (right- sided)

Acute right ventricular failure results from disorders affecting the lungs and right heart, including:

- exacerbations of pulmonary disease with pulmonary hypertension
- acute severe illnesses (e.g., massive pneumonia or thromboembolic events)
- right ventricular myocardial infarction
- tricuspid valve dysfunction (traumatic or infectious)
- acute or subacute pericarditis.

Advanced stages of left- sided heart disease frequently progress to right ventricular failure.

Non- cardiac and non- pulmonary causes include:

- nephritis
- nephrotic syndrome
- end- stage liver disease
- tumours secreting vasoactive peptides.

Typical symptoms: weakness, oedema of the lower limbs, discomfort in the right upper abdomen (hepatic congestion), dyspnoea (pleural effusion), abdominal distension (ascites).

Fully developed right- sided failure may present with anasarca, hepatic dysfunction, and oliguria.

Diagnosis

Diagnosis of acute right ventricular failure is based on: history, physical examination, additional investigations, including:

– ECG

– arterial blood gas analysis

– plasma D- dimer

– chest radiography

– Doppler echocardiography

– angiography

– computed tomography

Management. Treatment includes:

- diuretics, including spironolactone and eplerenone
- short courses of low- dose dopamine (“diuretic dose”)
- antibiotics (for pulmonary infection or endocarditis)
- calcium- channel blockers, nitric oxide, or prostaglandin in primary pulmonary hypertension
- anticoagulants, thrombolytics, interventional or surgical thrombectomy in pulmonary embolism.

Pathophysiology

The vicious circle of acute cardiac failure refers to the self- perpetuating deterioration in cardiac output, perfusion, and organ function that accelerates the progression of AHF. The final consequence of the AHF syndrome

The ultimate consequence of the acute heart failure (AHF) syndrome is the critical inability of the myocardium to maintain a cardiac output sufficient to ensure adequate peripheral circulation.

For a favourable clinical outcome in AHF, myocardial dysfunction must be reversible.

This is particularly important in ischaemic AHF, myocardial stunning, or hibernation, because appropriate treatment can lead to recovery of myocardial function.

Myocardial Stunning

Myocardial stunning refers to persistent myocardial dysfunction (reduced contractility) following an episode of acute ischaemia, even after full restoration of coronary blood flow.

The severity and duration of stunning depend on the intensity and duration of the preceding ischaemic episode.

Myocardial Hibernation

Myocardial hibernation refers to reduced myocardial function caused by markedly decreased coronary blood flow due to a haemodynamically significant stenosis, while myocardial cells remain viable.

When blood flow and oxygen delivery improve, contractility in the affected region recovers.

Hibernation and stunning may coexist. Hibernation resolves gradually with improved perfusion and oxygenation, whereas stunned myocardium retains inotropic reserve and responds to inotropic stimulation.

These mechanisms depend on the duration of myocardial ischaemia, and rapid restoration of perfusion and oxygenation leads to reversal of these changes.

Diagnosis

Diagnosis of AHF is based on:

- characteristic symptoms and clinical signs
- ECG abnormalities
- chest radiography
- biomarkers
- Doppler echocardiography.

AHF should be classified according to the criteria described above into:

- systolic and/or diastolic dysfunction
- left- or right- sided failure
- congestion above or below the level of the lesion.

Diagnosis and treatment of AHF are usually performed simultaneously, especially in patients with severe symptoms who require immediate therapy.

During initial assessment and treatment, vital signs must be closely monitored, and some patients are best managed in intensive care or cardiac intensive care units.

Although the immediate goal of treatment is symptom relief and haemodynamic stabilisation, long- term management - including post- discharge care - is essential to prevent recurrences and improve prognosis.

Pre- discharge and post- discharge care should follow the recommendations outlined in this document.

Initial Patient Assessment (Table 9)

During the initial evaluation, the following questions must be addressed:

Does the patient have heart failure, or is there an alternative explanation for their symptoms or signs (e.g., chronic lung disease, anaemia, renal failure, pulmonary embolism)?

– Differential diagnosis of dyspnoea

If heart failure is present, is there a trigger, and does it require urgent treatment or correction (e.g., arrhythmia, acute coronary syndrome)?

– HF triggers requiring urgent management.

Is the patient's condition life- threatening due to hypoxaemia or hypotension leading to inadequate perfusion of vital organs (heart, kidneys, brain)?

– Assessment of organ hypoperfusion

Table 9

Initial Assessment of a Patient With Suspected Acute Heart Failure [1]

Step / Clinical Finding	Examples	Immediate Actions
1. Initial simultaneous assessment	• History and physical examination • Chest X-ray • Echocardiography • Biochemical blood tests • ECG • Oxygen saturation • Complete blood count • Natriuretic peptides (BNP/NT-proBNP)	Perform all investigations in parallel to confirm or exclude AHF
2. Inadequate ventilation / systemic oxygenation	Severe pulmonary oedema, respiratory failure	• Oxygen therapy • NIV (CPAP/BiPAP) • ETT and mechanical ventilation if needed
3. Life-threatening tachyarrhythmia or bradycardia	Ventricular tachycardia, ventricular fibrillation, rapid AF, severe bradycardia	• Electrical cardioversion / defibrillation • Temporary pacing if required
4. Low blood pressure / shock	Cardiogenic shock, severe hypotension	• IV fluids (if hypovolaemia suspected) • Inotropes / vasopressors • IABP or other mechanical circulatory support
5. Acute coronary syndrome (ACS)	STEMI, NSTEMI with ischaemia	• Antiplatelet therapy • Anticoagulation • Urgent coronary reperfusion (PCI or thrombolysis)

Step / Clinical Finding	Examples	Immediate Actions
6. Mechanical cause / severe structural disease	Acute valvular regurgitation, papillary muscle rupture, ventricular septal rupture, tamponade	• Urgent echocardiography • Pericardiocentesis (if tamponade) • Emergency cardiac surgery

NUP — natriuretic peptide; NIV — non- invasive ventilation; ; TT — endotracheal tube; IABP — intra- aortic balloon pump

Diagnostic Algorithm for Acute Heart Failure (AHF):

- Suspicion of AHF: assessment of symptoms and clinical signs.
- ECG / brain natriuretic peptide (BNP or NT- proBNP) / chest radiography.
- Assessment of cardiac function using echocardiography or other imaging modalities; determination of the type of cardiac involvement.
- Selection of further investigations (coronary angiography, haemodynamic monitoring, pulmonary artery catheterisation).

Assessment of Left Ventricular Function in AHF:

- Evaluation of ventricular function - determination of left ventricular ejection fraction (LVEF).
- Left ventricular systolic dysfunction.
- Transient systolic dysfunction.
- Diastolic dysfunction.

Clinical Assessment

Right ventricular filling in decompensated HF is usually assessed by measuring central venous pressure (CVP) in the subclavian or jugular veins, or in the right atrium.

If the internal jugular veins are difficult to use (e.g., due to venous valves), external jugular catheterisation may be performed.

Interpretation of elevated CVP in AHF requires caution, as it may be reflexive in response to reduced venous compliance or due to increased right ventricular stiffness, even when filling is inadequate.

Left- sided filling pressure can be assessed indirectly by auscultation.

The presence of moist crackles in the lungs usually indicates elevated pressure.

Congestion, its severity, and the level of pleural effusion should be identified or confirmed by chest radiography.

Electrocardiogram

In AHF, the ECG is rarely normal.

Determination of rhythm and signs of cardiac overload may help identify the aetiology of AHF.

If acute coronary syndrome is suspected, ECG recording is mandatory.

ECG may reveal:

- signs of increased load on the left or right ventricle
- atrial enlargement
- features of pericarditis
- ventricular hypertrophy

Arrhythmias may also be detected during continuous ECG monitoring.

Table 10

Laboratory Evaluation Scheme for Patients With AHF [1]

Laboratory Test	Clinical Purpose
Complete blood count (CBC)	Detect anaemia, infection, leukocytosis
Electrolytes (Na^+ , K^+ , Cl^-)	Identify abnormalities affecting arrhythmias and perfusion
Renal function (creatinine, urea)	Assess renal impairment; evaluate cardiorenal syndrome
Liver function tests	Detect hepatic congestion or dysfunction
BNP / NT-proBNP	Confirm or exclude AHF
Cardiac troponins	Identify myocardial injury / ACS
CRP / procalcitonin	Detect infection or systemic inflammation
D-dimer	Evaluate for pulmonary embolism
Arterial blood gases (ABG)	Assess hypoxaemia, hypercapnia, acidosis
Thyroid function tests	Exclude thyrotoxicosis as a trigger

Chest Radiography and Other Imaging Modalities

Chest radiography and other imaging techniques should be performed as early as possible in all patients with acute heart failure (AHF) to assess:

- the heart (size and contours of the cardiac silhouette)
- the presence and severity of pulmonary congestion.

Radiography is also used to:

- confirm the diagnosis

- monitor improvement or, conversely, refractoriness to therapy
- assist in the differential diagnosis between left- sided HF and pulmonary infection, as well as other conditions.

Chest computed tomography (CT), with or without contrast angiography, is required to detect pulmonary pathology and diagnose pulmonary embolism.

If aortic dissection is suspected, CT or transoesophageal echocardiography is mandatory.

Laboratory Investigations

Laboratory tests required in AHF are listed in Table 9.

Arterial blood gas analysis (ABG) allows assessment of:

- oxygenation (pO_2)
- ventilatory function (pCO_2)
- acid–base status (pH) and its disturbances

Therefore, ABG is recommended for all patients with severe HF.

Non- invasive methods - pulse oximetry and end- tidal CO_2 measurement - may substitute ABG, but not in states associated with low cardiac output or vasoconstrictor shock.

Natriuretic Peptides

Measurement of BNP or NT- proBNP in plasma (released from ventricular myocardium in response to wall stretch and volume overload) is used to confirm or exclude chronic HF in patients hospitalised with dyspnoea.

Threshold values: NT- proBNP ≥ 300 pg/mL, BNP ≥ 100 pg/mL.

However, elderly patients were under- represented in the studies establishing these cut- offs.

In acute pulmonary oedema, BNP levels may be normal at the time of admission.

BNP has a high negative predictive value for excluding HF.

Various clinical conditions may influence BNP concentration, including renal failure and septicaemia.

If elevated levels are detected, further diagnostic testing is required.

If AHF is confirmed, elevated BNP or NT- proBNP provides important prognostic information.

Echocardiography

Echocardiography is essential for identifying structural and functional abnormalities resulting from AHF and for evaluating patients with acute coronary syndrome.

Doppler echocardiography should be used to assess:

- systolic and diastolic function of both ventricles
- valvular structure and function
- pericardial pathology
- mechanical complications of myocardial infarction
- localisation of myocardial injury.

Cardiac output is determined using Doppler parameters in the aorta and pulmonary artery.

Echocardiography also allows estimation of pulmonary artery pressure (via tricuspid regurgitation) and monitoring of left ventricular preload.

Other Diagnostic Methods

In the presence of coronary artery disease (e.g., unstable angina or myocardial infarction), coronary angiography is essential to determine the need for revascularisation, which significantly improves prognosis.

Treatment of Acute Heart Failure

Immediate goals include: reduction of clinical symptoms and stabilisation of haemodynamics.

Improvement in haemodynamic parameters requires simultaneous relief of dyspnoea and/or weakness, which favourably influences long- term prognosis.

It is essential to avoid any potential myocardial injury.

Other important therapeutic objectives include:

- reduction of body weight
- increased diuresis (critical in congestive and oliguric forms of AHF)
- improved oxygenation

- improved hepatic and renal function
- normalisation of serum electrolytes.

Plasma BNP concentration may reflect haemodynamic improvement, and reducing BNP levels is desirable.

Clinically relevant outcomes include:

- duration of IV vasoactive therapy
- length of hospital stay
- time to rehospitalisation.

Efforts should be directed toward reducing in- hospital and long- term mortality.

Organisation of Acute Heart Failure (AHF) Management

Optimal outcomes in patients with AHF are achieved when care is provided by specially trained staff in specialised units, such as cardiac intensive care units or coronary care units.

Patients with AHF should be managed by an experienced cardiologist.

Continuous access to advanced diagnostic modalities is essential, including:

- Echocardiography
- Coronary angiography
- Invasive haemodynamic monitoring.

General Considerations in the Management of AHF

Infections

Patients with severe AHF frequently develop infectious complications, particularly:

- respiratory tract infections
- urinary tract infections
- septicaemia
- nosocomial infections, often caused by Gram- positive organisms.

Elevated CRP and clinical deterioration may be the only signs of infection; fever is often absent.

Strict infection control and maintenance of skin and mucosal integrity are essential.

When indicated, appropriate antibiotic therapy must be initiated.

Diabetes Mellitus

AHF is associated with worsening metabolic control, and hyperglycaemia is common.

Oral hypoglycaemic agents should be discontinued, and glycaemic control maintained using short- acting insulin, adjusted according to blood glucose levels.

Normoglycaemia improves survival in critically ill patients with diabetes.

Catabolism

Negative energy balance is a major problem in AHF due to reduced caloric intake and impaired intestinal absorption.

Optimal caloric and amino acid intake must be ensured.

Serum albumin concentration and nitrogen balance help monitor metabolic status.

Renal Dysfunction

AHF may cause or worsen existing renal impairment.

Renal function must be monitored closely.

Preservation of renal function should be considered a therapeutic goal in these patients [20].

Rationale for Oxygen Therapy in AHF

Oxygen may be administered to treat hypoxaemia (SpO_2 < 90%), which is associated with increased short- term mortality.

Oxygen should not be routinely used in patients without hypoxaemia, as it may cause vasoconstriction and reduce cardiac output.

Maintaining SpO_2 within the normal range (95–98%) is essential to ensure adequate tissue oxygen delivery and prevent organ dysfunction and multiorgan failure.

Initial steps include:

- ensuring airway patency

- administering oxygen with increasing FiO_2 as needed.

Endotracheal intubation is required if these measures fail to achieve adequate oxygenation.

High- concentration oxygen is clearly necessary in patients with HF and hypoxaemia, but may be harmful in those without hypoxaemia and should therefore be avoided [20].

Non- Invasive Respiratory Support (Without Endotracheal Intubation)

Two main techniques are used:

- Continuous positive airway pressure (CPAP) during spontaneous breathing
- Non- invasive positive pressure ventilation (NIPPV).

NIPPV allows mechanical ventilation without endotracheal intubation.

There is consensus that one of these techniques should be attempted before intubation and invasive mechanical ventilation.

Use of non- invasive ventilation has significantly reduced the need for intubation and invasive ventilation in AHF.

Rationale

The use of continuous positive airway pressure (CPAP) can restore lung function by increasing functional residual capacity.

It reduces lung stiffness, the transdiaphragmatic pressure gradient, and diaphragmatic activity.

Together, these effects decrease the work of breathing and reduce the overall metabolic demands of the body.

Usefulness of CPAP and NIPPV in Left- Sided Heart Failure

Respiratory support using CPAP in patients with cardiogenic pulmonary oedema improves oxygenation, reduces symptoms of AHF, and decreases the need for endotracheal intubation .

Respiratory Support in AHF With Endotracheal Intubation

Invasive respiratory support (mechanical ventilation with endotracheal intubation) should not be used solely to correct hypoxaemia, as this can often be achieved with non- invasive ventilation or even oxygen therapy.

Invasive ventilation is indicated when AHF leads to respiratory muscle weakness, which may present as:

- reduced respiratory rate
- hypercapnia
- impaired consciousness.

Mechanical ventilation should be used only when acute respiratory failure does not improve with vasodilators, oxygen therapy, and/or non- invasive ventilation (CPAP or NIPPV).

Pharmacotherapy

Use of Opiates in Acute Heart Failure

Opiates such as morphine may be beneficial in some patients with acute pulmonary oedema because they:

- reduce anxiety
- decrease agitation associated with dyspnoea
- cause venous dilation and mild arterial dilation
- reduce pulmonary artery pressure
- lower heart rate.

However, opiates may also:

- cause nausea
- depress the respiratory centre
- increase the likelihood of requiring invasive ventilation.

Anticoagulant Therapy

Anticoagulants are widely used in the treatment of acute coronary syndrome, regardless of the presence of HF.

This also applies to atrial fibrillation.

Evidence supporting the use of unfractionated heparin or low- molecular- weight heparins (LMWH) as part of initial AHF therapy is limited.

A large placebo- controlled trial using 40 mg enoxaparin subcutaneously in acutely hospitalised patients (including many with HF) showed:

- reduced incidence of venous thrombosis
- no effect on clinical symptoms
- LMWHs are contraindicated when creatinine clearance is < 30 mL/min.

Vasodilators in the Treatment of AHF

Although vasodilators such as nitroglycerin reduce preload and afterload and increase stroke volume, there is no strong evidence that they improve dyspnoea or other clinical outcomes.

Vasodilators are likely most useful in patients with hypertension, and should be used with caution in patients with SBP < 110 mmHg.

Excessive blood pressure reduction is associated with high mortality in AHF.

Vasodilators should be used cautiously in patients with significant mitral or aortic stenosis .

Vasodilators are recommended in AHF patients with:

- hypoperfusion
- normal blood pressure
- pulmonary congestion
- reduced urine output

In such cases, vasodilators improve peripheral circulation and reduce preload.

Nitrates

Nitrates reduce pulmonary congestion without worsening stroke volume or increasing myocardial oxygen demand - an important advantage in acute coronary syndrome.

- At low doses, they dilate venous vessels.
- At higher doses, they also dilate arteries, including coronary arteries.

Adequate dosing maintains a balance between arterial and venous dilation, reducing preload and afterload without impairing tissue perfusion.

Initial nitrate therapy may be given orally, but intravenous administration is also well tolerated, including in patients with MI.

Titration to the maximum haemodynamically tolerated dose, combined with low- dose furosemide, is more effective than high- dose diuretics alone.

Available forms

- Oral and inhaled nitroglycerin (spray 400 μg: 2 puffs every 5–10 min)
- Sublingual isosorbide dinitrate (1 or 3 mg), with BP monitoring
- Intravenous nitrates (e.g., nitroglycerin 20-200 μg/min; isosorbide dinitrate 1-10 mg/h)

IV nitrates should be used only when necessary, with careful BP monitoring to avoid hypotension.

Special caution is required in aortic stenosis; nitrates should be used only when clearly indicated.

Calcium Channel Blockers

Diltiazem, verapamil, and dihydropyridines are contraindicated in acute heart failure (AHF).

Angiotensin- Converting Enzyme Inhibitors (ACEIs) in Acute Heart Failure.

Indications: In patients with reduced ejection fraction who are not yet receiving an ACE inhibitor, treatment should be initiated as early as possible, provided that blood pressure and renal function allow.

The dose should be increased to the maximum tolerated level before hospital discharge, and further titration should follow chronic HF guidelines after discharge.

Effects and Mechanisms of Action

The haemodynamic effects of ACEIs result from:

- reduced formation of angiotensin II
- increased levels of bradykinin.

These changes lead to:

- decreased systemic vascular resistance
- enhanced natriuresis.

Short- term therapy reduces plasma angiotensin II and aldosterone, while increasing angiotensin I and renin activity.

Practical Use

- Avoid intravenous ACEIs.
- Start with a low dose, increasing gradually after stabilisation, with monitoring of blood pressure and renal function.
- Risk of intolerance increases with NSAIDs and bilateral renal artery stenosis.

Diuretics

Indications. Most patients with dyspnoea due to pulmonary oedema experience rapid symptom relief after IV diuretics, due to:

- early venodilation
- subsequent fluid removal.

Optimal dosing and mode of administration (bolus vs continuous infusion) show no clear superiority.

A recent small RCT compared 12- hour bolus vs continuous infusion and low dose (equal to prior oral dose) vs high dose (2.5× oral dose).

There was no difference in primary outcomes (global symptom assessment and change in serum creatinine).

High- dose therapy improved several secondary outcomes (including dyspnoea) but caused greater transient renal impairment.

Effects and Mechanisms

Diuretics increase urine output by enhancing excretion of water, sodium, other ions. This leads to:

- reduced plasma and intravascular volume
- decreased total body water and sodium
- reduced filling pressures of both ventricles
- relief of congestion and pulmonary oedema.

IV loop diuretics also have vasodilatory effects, producing early (5–30 min) reductions in:

- right atrial pressure
- pulmonary artery wedge pressure
- pulmonary vascular resistance.

High- dose bolus furosemide (>1 mg/kg) may cause reflex vasoconstriction.

In acute coronary syndrome, diuretics should be used in low doses, preferably IV.

Practical Use:

- IV loop diuretics (furosemide, torasemide) are the treatment of choice in AHF.
- A loading dose followed by continuous infusion is more effective than bolus alone.
- Thiazides, spironolactone, and eplerenone (in MI and post- MI patients) may be combined with loop diuretics.
- Combining loop diuretics with dobutamine, dopamine, or nitrates may enhance effectiveness.
- Dose should be titrated to clinical response.
- Dose reduction is needed once fluid retention improves.
- Monitor serum K^+ and Na^+, and renal function every 1–2 days; correct K^+ and Mg^{2+} losses as needed.

Diuretic Resistance

Causes of Diuretic Refractoriness:

- Reduced intravascular volume
- Neurohormonal activation
- Post- diuretic sodium reabsorption after volume depletion
- Hypertrophy of the distal nephron
- Reduced tubular secretion (renal failure, NSAIDs)
- Reduced renal perfusion (low cardiac output)
- Impaired intestinal absorption of oral diuretics
- Non- adherence to medication or diet (high sodium intake)

Management of Diuretic Resistance:

- Normalise sodium and fluid intake and monitor serum electrolytes
- Correct hypovolaemia if present
- Increase diuretic dose and/or frequency
- Use intravenous infusion (more effective than oral or bolus administration).

Combination Therapy:

- Furosemide + hydrochlorothiazide
- Furosemide + spironolactone
- Diuretic + dopamine or dobutamine
- Reduce ACE inhibitor dose or use very low doses
- If ineffective, consider ultrafiltration or haemodialysis

Secondary Effects and Drug Interactions.

These include:

- neurohormonal activation, especially of the renin–angiotensin–aldosterone system and sympathetic nervous system
- hypokalaemia
- hypomagnesaemia
- hypochloraemic alkalosis

These disturbances may lead to:

- severe arrhythmias
- nephrotoxicity
- worsening renal dysfunction.

Excessive diuresis may reduce:

- venous pressure
- pulmonary capillary wedge pressure
- diastolic filling.

This can decrease stroke volume and cardiac output, especially in patients with:

- severe HF
- predominantly diastolic dysfunction
- ischaemic right ventricular dysfunction.

Beta- Blockers

Indications and Rationale

In patients with reduced ejection fraction who are not yet receiving a beta- blocker, therapy should be initiated as soon as stabilisation occurs, provided blood pressure and heart rate allow.

The dose should be increased before discharge, with further titration to the maximum tolerated dose thereafter.

Evidence shows that beta- blocker therapy can be continued safely in many patients during an episode of decompensation and can be initiated before discharge once stabilisation is achieved.

Practical Use:

- In patients with AHF and pulmonary crackles (not only basal), beta- blockers should be used with caution.
- If needed, in patients without pulmonary crackles but with ischaemia or tachycardia, intravenous administration may be considered.
- In patients with acute myocardial infarction whose condition stabilises rapidly despite AHF, beta- blockers should be started as early as possible.
- In patients with AHF on the background of chronic HF, beta- blockers should be initiated immediately after stabilisation (usually within 3–4 days).

Mineralocorticoid Receptor Antagonists (MRAs)

In patients with reduced ejection fraction who are not yet receiving a mineralocorticoid receptor antagonist, therapy should be initiated as early as possible, provided that renal function and serum potassium allow.

Because the doses used for HF treatment have minimal effects on blood pressure, even patients with arterial hypotension may begin MRA therapy during hospitalisation.

The dose should be increased before discharge and subsequently titrated to the maximum tolerated dose.

Inotropic Agents. Clinical Indications.

Inotropic agents are recommended in the presence of peripheral hypoperfusion (arterial hypotension, worsening renal function), regardless of

pulmonary congestion or pulmonary oedema, when these are refractory to optimal doses of diuretics and vasodilators.

Agents such as dobutamine are generally reserved for patients with severely reduced cardiac output, where perfusion of vital organs is threatened.

These patients almost always present with hypotension or clinical signs of shock.

Inotropes may cause:

- sinus tachycardia
- myocardial ischaemia
- arrhythmias.

There is pharmacological and clinical rationale for using levosimendan in patients already receiving beta- blockers.

Dopamine

- At low doses (< 2 μg/kg/min IV), dopamine acts on dopaminergic receptors, reducing peripheral resistance.
- Vasodilation occurs in the kidneys, abdominal organs, coronary and cerebral vessels, improving:
  - renal blood flow
  - glomerular filtration rate
  - urine output
  - sodium excretion
  - response to diuretics
- At > 2 μg/kg/min, dopamine stimulates β- adrenergic receptors, increasing myocardial contractility and cardiac output.
- At > 5 μg/kg/min, dopamine stimulates α- adrenergic receptors, increasing peripheral vascular resistance—potentially useful in hypotension but harmful in AHF.

Dobutamine

Dobutamine is a positive inotropic agent acting mainly via β1- adrenergic and α1- adrenergic stimulation, producing:

- dose- dependent positive inotropic and chronotropic effects
- reflex sympathetic activation
- increased vascular resistance.

At low doses, dobutamine induces moderate arterial vasodilation, increasing stroke volume by reducing afterload.

Pulmonary artery pressure and wedge pressure typically decrease.

Increased diuresis during dobutamine infusion is due to improved renal blood flow secondary to increased cardiac output.

Practical Use

- In AHF, dopamine may be used as an inotrope (> 2 μg/kg/min IV) when hypotension is present.
- Low- dose dopamine (< 2–3 μg/kg/min) may improve renal perfusion and enhance diuresis in patients with chronic HF, hypotension, and oliguria.

Prolonged dobutamine infusion (> 24–48 h) is associated with:

- tolerance
- reduced haemodynamic effect
- risk of rebound hypotension, congestion, and renal dysfunction upon withdrawal.

These complications can be avoided by gradual tapering (reducing dose by 2 μg/kg/min daily) and optimising oral vasodilator therapy (e.g., ACE inhibitors).

Levosimendan

Levosimendan is a newer inotropic agent with two main mechanisms:

- Increases calcium sensitivity of myocardial contractile proteins → positive inotropy without impairing diastolic relaxation
- Opens ATP- dependent potassium channels in vascular smooth muscle → peripheral vasodilation

Levosimendan:

- increases cardiac output, stroke volume, and LVEF
- reduces systolic and diastolic BP
- decreases pulmonary capillary wedge pressure

- lowers right atrial pressure
- reduces systemic and pulmonary vascular resistance

It may also have mild phosphodiesterase- inhibitory activity.

Levosimendan is recommended in HF patients with low cardiac output due to systolic dysfunction without significant hypotension.

It is administered as a prolonged IV infusion (6–24 h), with haemodynamic effects lasting > 48 h after infusion.

High doses may cause tachycardia and hypotension, limiting use when SBP < 85 mmHg.

A key advantage is that levosimendan maintains its haemodynamic benefits even when used concurrently with beta- blockers.

It may be used:

- in combination with dobutamine
- in repeated cycles every 7–14 days.

Vasopressor Therapy in Cardiogenic Shock

When, despite inotropic support and fluid administration, adequate perfusion is not achieved, vasopressor therapy must be initiated.

Vasopressors are also used during resuscitation and to maintain adequate perfusion in cases of life- threatening hypotension.

Adrenaline (Epinephrine)

Adrenaline is a catecholamine with high affinity for β1- , β2- and α- adrenergic receptors.

- Used as a continuous infusion at 0.05–0.5 μg/kg/min
- Indicated when dobutamine fails to produce a clinical effect and blood pressure remains low
- Requires direct blood pressure monitoring and haemodynamic assessment, preferably with pulmonary artery catheterisation

Noradrenaline (Norepinephrine)

Noradrenaline is a catecholamine with predominant α- adrenergic activity, used to increase systemic vascular resistance.

- Causes less reflex tachycardia than adrenaline
- Recommended doses are similar to adrenaline
- Particularly useful in low blood pressure with low systemic vascular resistance, e.g., septic shock
- Typical dose: 0.2–1 μg/kg/min
- Often combined with dobutamine to optimise haemodynamics

Cardiac Glycosides

Cardiac glycosides inhibit myocardial Na^+/K^+ - ATPase, increasing Ca^{2+}/Na^+ exchange, producing a positive inotropic effect.

In AHF, glycosides:

- slightly increase cardiac output
- reduce filling pressures

However:

- After myocardial infarction, glycosides prolong elevated creatine phosphokinase levels
- In atrial fibrillation with AHF, digoxin may provoke life- threatening arrhythmias

Therefore, inotropic support with cardiac glycosides is not recommended in AHF, especially in acute coronary syndrome.

In patients with reduced EF and atrial fibrillation, digoxin may be used for ventricular rate control, particularly when beta- blocker titration is not possible.

Surgical Treatment. Indications:

- Coronary artery disease with multivessel involvement causing severe myocardial ischaemia
- Acute mechanical complications of myocardial infarction
- Acute mitral or aortic regurgitation due to endocarditis, trauma, or aortic dissection (aortic valve)
- Selected complications of PCI

Key Points from the 2016 ESC Guidelines for Acute and Chronic Heart Failure

The most common causes of AHF include:

- Acute myocardial dysfunction (ischaemic, inflammatory, or toxic)
- Acute valvular regurgitation
- Pericardial tamponade

Decompensation of chronic HF may occur without triggers, but more often arises due to:

- infection
- uncontrolled hypertension
- arrhythmias
- non- adherence to diet or medication

These are summarised in Table 11:

Table 11

Provoking Factors of AHF [1]

Precipitating Factors of Acute Heart Failure (AHF)**
Acute coronary syndrome (ACS)
Tachyarrhythmias (e.g., atrial fibrillation, ventricular tachycardia)
Sudden increase in arterial blood pressure
Infections (e.g., pneumonia, infective endocarditis, sepsis)
Non- adherence to salt/fluid restriction or medication regimen
Bradyarrhythmias
Alcohol or drug abuse

Key Points in the Initial Evaluation of Patients With Acute Heart Failure (AHF)

When AHF is suspected, three elements are essential:

- Measurement of natriuretic peptides, recommended for all patients with acute- onset dyspnoea and suspected AHF

- Early initiation of appropriate therapy.

Use of the new combined diagnostic and therapeutic algorithm

Table 12

Causes of Elevated Natriuretic Peptide Levels [1]

Cardiac Causes	***Non-Cardiac Causes***
• Heart failure • Acute coronary syndrome • Pulmonary embolism • Myocarditis • Left ventricular hypertrophy (LVH) • Hypertrophic or restrictive cardiomyopathy • Valvular heart disease • Congenital heart disease • Atrial and ventricular tachyarrhythmias • Cardiac contusion (blunt cardiac trauma) • Cardioversion • Cardiac surgery • Pulmonary hypertension	• Advanced age • Ischaemic stroke • Subarachnoid haemorrhage • Renal failure • Hepatic failure (especially cirrhosis with ascites) • Paraneoplastic syndromes • COPD • Severe infections (including pneumonia and sepsis) • Major burns • Anaemia • Severe metabolic and endocrine disorders (e.g., diabetic ketoacidosis, thyrotoxicosis)

An essential next step in the management of acute heart failure (AHF) is the early initiation of therapy.

Once AHF is confirmed - regardless of whether the acute precipitating cause has already been identified or whether diagnostic evaluation is still ongoing—treatment must begin immediately, ideally within 60–120 minutes of the patient's first contact with a healthcare professional.

New Combined Diagnostic and Therapeutic Algorithm for AHF

The new combined algorithm is based on the presence or absence of congestion and the presence or absence of hypoperfusion.

In Table13:

The horizontal axis represents the presence or absence of congestion

The vertical axis represents the presence or absence of hypoperfusion

This classification divides all patients with acute HF into four clinical profiles, which help guide therapeutic strategy:

1. Warm–Dry (no congestion, no hypoperfusion)

Warm–dry profile represents compensated or mildly decompensated HF

Usually stable haemodynamics

Often requires optimisation of chronic HF therapy

2. Cold–Dry (no congestion, hypoperfusion present)

Cold–dry profile indicates low cardiac output without fluid overload

Often associated with volume depletion or excessive diuresis

Requires careful fluid assessment and perfusion support

3. Warm–Wet (congestion present, no hypoperfusion)

Warm–wet profile is the most common presentation of AHF

Characterised by pulmonary or systemic congestion with preserved perfusion

Requires diuretics and vasodilators

4. Cold–Wet (congestion present, hypoperfusion present)

Cold–wet profile is the most severe form

Indicates both fluid overload and inadequate tissue perfusion

Requires urgent, aggressive therapy (inotropes, vasopressors, diuretics, mechanical support).

Table 13

Combined Diagnostic Algorithm for Acute Heart Failure [1]

	Congestion (–)	Congestion (+): • Pulmonary congestion • Orthopnoea / paroxysmal nocturnal dyspnoea • Peripheral (bilateral) oedema • Jugular venous distension • Congestive hepatomegaly • Intestinal congestion • Hepatojugular reflux
no hypoperfusion (-)	**Warm–Dry** (no congestion, no hypoperfusion)	**Warm–Wet** (congestion present, no hypoperfusion)
Hypoperfusion (+) • Cold, clammy extremities • Oliguria • Confusion • Dizziness • Low pulse pressure	**Cold–Dry** (no congestion, hypoperfusion present)	**Cold–Wet** (congestion + hypoperfusion)

The treatment algorithm is based on the same four- profile classification of patients shown in Table 14.

The recommended algorithm, which facilitates the selection of an appropriate therapeutic strategy in patients with heart failure and specifies the medication classes required for each profile, is presented in Table 14.

Table 14

Combined Treatment Algorithm for Acute Heart Failure [1]

Adequate Peripheral Perfusion?	Clinical Profile	Recommended Therapy
Yes	Warm–Wet (congestion + adequate perfusion)	• Vasodilators • Diuretics • Consider ultrafiltration if diuretics ineffective
Yes	Warm–Dry (no congestion, adequate perfusion)	• Oral therapy • Patient is compensated
No → SBP < 90 mmHg	Cold–Wet (congestion + hypoperfusion, hypotensive)	• Inotropes • Vasopressors if inadequate response • Diuretics *after* perfusion improves • Consider mechanical circulatory support if refractory
No → SBP ≥ 90 mmHg	Cold–Wet (congestion + hypoperfusion, normo-/hypertensive)	• Vasodilators • Diuretics • Inotropes if ineffective
No perfusion + no congestion	Cold–Dry (hypoperfusion + hypovolaemia)	• Fluid resuscitation • Inotropes if hypoperfusion persists

Prevention

Regular follow- up with a specialist at least twice a year is essential for patients with chronic cardiovascular diseases, along with timely medical consultation and strict adherence to treatment recommendations.

The most effective prevention of cardiovascular diseases is the reduction of modifiable risk factors:

- Smoking cessation and avoidance of excessive alcohol consumption (for men, no more than 30 g of pure alcohol per day)
- Avoidance of psycho- emotional stress

- Maintaining optimal body weight (BMI = weight in kg / height in meters²; normal range: 20–25)

  Regular physical activity: Daily dynamic aerobic exercise such as brisk walking, running, swimming, skiing, cycling.

  Each session: 25–40 minutes (5- minute warm- up, 15–30 minutes main phase, 5- minute cool- down).

  Avoid exercise for 2 hours after meals; avoid eating for 20–30 minutes after exercise.
- Blood pressure control
- Balanced and heart- healthy diet:
- High- fiber foods (vegetables, fruits, greens), avoidance of fried, canned, overly hot or spicy foods
- Cholesterol monitoring

TEST QUESTIONS (MULTIPLE CHOICE)

1. Indicate the upper limit of normal diastolic blood pressure.
A. 60 mmHg
B. 69 mmHg
C. 79 mmHg
D. 89 mmHg
E. 99 mmHg

2. A 60-year-old woman with a 20-year history of hypertension suddenly develops an episode of breathlessness following emotional stress.
On examination: orthopnoea, pulse 120/min, BP 210/120 mmHg. The first heart sound at the apex is diminished; an additional diastolic sound is heard. Respiratory rate 32/min. Breath sounds over the lower lung fields are reduced; occasional soft fine crackles are present. Which complication has developed?
A. Acute left ventricular failure
B. Hysterical attack
C. Pulmonary artery branch thromboembolism
D. Pneumonia
E. Aortic dissection

3. A 46-year-old woman complains of sudden episodes of palpitations accompanied by pulsation in the neck and head, fear, and nausea. The palpitations last 15–20 minutes and resolve with breath-holding and straining.
Which cardiac rhythm disturbance is most likely?
A. Atrial flutter attack
B. Paroxysmal ventricular tachycardia
C. Paroxysmal supraventricular tachycardia
D. Atrial fibrillation attack
E. Extrasystolic arrhythmia attack

4. A 30-year-old woman reports episodes of headache, temporal pulsation, dizziness, palpitations, and muscle weakness, accompanied by pallor and marked elevation of blood pressure (270/160 mmHg). The attacks occur mainly at night and are not relieved by oral antihypertensive drugs.
Phaeochromocytoma is suspected.
Which drug is most effective in this situation?

A. Intravenous phentolamine
B. Intravenous benzogexonium
C. Intravenous clonidine
D. Intravenous dibazol
E. Intravenous furosemide

5. A 45-year-old man develops an episode of atrial fibrillation with an irregular ventricular rate of 150–160/min and a fall in blood pressure. Which treatment is most appropriate?
A. Intravenous verapamil
B. Intravenous procainamide
C. Cardiac glycosides
D. Electrical cardioversion
E. Intravenous lidocaine infusion

6. A 66-year-old patient with arterial hypertension (BP 190/110 mmHg) is found to have ischaemic heart disease, angina pectoris (Class II), and generalised atherosclerosis of the lower limb vessels. Blood glucose is 6.7 mmol/L, cholesterol 7 mmol/L, triglycerides 3 mmol/L.
Choose the most appropriate antihypertensive agent.
A. Enalapril
B. Hydrochlorothiazide
C. Atenolol
D. Reserpine
E. Methyldopa

7. A 67-year-old man complains of intermittent dizziness. On examination: muffled heart sounds, marked bradycardia. Pulse 36/min, regular. BP 150/70 mmHg.
ECG: ventricular rate 32/min; atrial and ventricular complexes occur independently of each other. What is the most likely cause of the dizziness?
A. Complete atrioventricular block
B. Sinus bradycardia
C. Sinus arrhythmia
D. Sinoatrial block
E. Incomplete atrioventricular block

8. A 65-year-old man feels irregular heartbeats. ECG shows premature, widened and deformed QRS complexes followed by a long compensatory pause. Heart rate 100/min. BP 100/60 mmHg.
Which rhythm disturbance is most likely?
A. Atrial fibrillation attack
B. Paroxysmal ventricular tachycardia
C. Paroxysmal supraventricular tachycardia

D. Supraventricular extrasystole
E. Ventricular extrasystole

9. In sinus bradycardia, the following may occur:
A. Prolongation of the PQ interval
B. Increase in T-wave amplitude
C. Widening of the P wave
D. Prolongation of the QT interval
E. All of the above

10. The possible outcome of newly-onset angina pectoris is:
A. Clinical recovery
B. Development of myocardial infarction
C. Progression to stable angina
D. Sudden death
E. All of the above

11. A characteristic sign of clinical death that has developed as a result of sinoatrial or atrioventricular block is:
A. Sudden onset
B. A relatively gradual onset following a Morgagni–Adams–Stokes attack
C. Accompanied by marked cyanosis
D. Develops after physical exertion
E. Involuntary urination or defecation

12. The following drugs are used to treat a hypertensive crisis combined with acute coronary syndrome, EXCEPT:
A. Nifedipine
B. Intravenous nitroglycerin
C. Sodium nitroprusside
D. Propranolol
E. Angiotensin-converting enzyme inhibitors

13. A patient develops a complex cardiac rhythm disturbance: atrial fibrillation and a paroxysm of ventricular tachycardia.
Name the universal antiarrhythmic drug most appropriate in this situation:
A. Panangin
B. Procainamide
C. Lidocaine
D. Verapamil
E. Digoxin

14. To what safe level should blood pressure be reduced at the first stage of treating a hypertensive crisis?
A. By 50%
B. By 40%
C. By 30–35%

D. By 20–25%
E. It does not matter

15. Which group of drugs is most appropriate for stabilising blood pressure in phaeochromocytoma?
A. ACE inhibitors
B. Calcium channel blockers
C. Alpha- and beta-adrenoceptor blockers
D. Angiotensin receptor blockers
E. Diuretics

16. The drug of choice for torsades de pointes ventricular tachycardia with stable haemodynamics is:
A. Magnesium sulphate
B. Calcium chloride
C. Class Ia, Ic and III antiarrhythmic drugs (except amiodarone)
D. Adrenaline
E. Verapamil

17. Which of the following is NOT included in the management plan when acute myocardial infarction is suspected?
A. Sublingual nitroglycerin (three times at 5-minute intervals); if ineffective – intravenous nitroglycerin infusion
B. Humidified oxygen via nasal cannulae
C. 160–325 mg aspirin orally
D. Intravenous morphine for pain relief
E. Intravenous lidocaine

18. Which of the following manoeuvres is NOT a non-pharmacological method for treating tachycardia with a “narrow” QRS complex?
A. Astvatsaturov’s nasolabial test
B. Valsalva manoeuvre – breath-holding with straining at peak inspiration for 5–10 seconds
C. Chermak–Hering test – carotid sinus massage
D. Inducing the gag reflex
E. Balloon inflation test

19. A 45-year-old man with persistent bronchial asthma (moderate severity) has been using 200 μg of beclomethasone daily and salmeterol. Peak expiratory flow was >80% of predicted. His condition improved and

he stopped taking his medication. An exacerbation developed.
On examination: the patient sits leaning forward; dyspnoea; loud wheezing; speaks in short phrases; agitated. Respiratory rate 25/min. Use of accessory muscles. Pulse 112/min, BP 120/80 mmHg. Heart sounds loud and regular. Percussion: hyperresonant ("box-like") tone. Auscultation: expiratory wheeze. Choose the appropriate management:**
A. Outpatient management: increase the dose of short-acting inhaled β_2 -agonists (2–4 puffs every 20 minutes during the first hour). Reassess after one hour
B. Immediate hospitalisation
C. Immediate intravenous β_2 -agonists
D. Immediate intravenous glucocorticosteroids
E. Intubation and mechanical ventilation

20. After an episode of burning retrosternal pain, the ECG shows: Q waves in leads II, III, aVF > R waves in II, III, aVF; ST-segment elevation; negative T waves in II, III, aVF. What is your diagnosis?
A. Large-focal (macro-focal) posterior–inferior myocardial infarction in the acute stage
B. ECG changes characteristic of acute coronary insufficiency
C. ECG changes characteristic of a small-focal posterior myocardial infarction
D. Transmural anterior myocardial infarction in the acute stage
E. ECG changes characteristic of acute pulmonary artery thromboembolism

Answers to the test questions:

1	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**
D	A	C	A	D	A	A	E	E	E
11	**12**	**13**	**14**	**15**	**16**	**17**	**18**	**9**	**20**
B	A	B	D	C	A	E	A	A	A

Appendix 1

A differentiated approach to the therapy of patients with complicated hypertensive crises [1]

Complication	Initiation of Therapy	Therapeutic Target	Drugs of Choice	Not Recommended Drugs
Acute hypertensive encephalopathy	at the presenting blood pressure level	reduction of systolic BP by 25% within 8 hours	labetalol, urapidil	sodium nitroprusside, hydralazine, nifedipine
Acute ischaemic stroke	If thrombolytic therapy (TLT) is planned: SBP >185 mmHg or DBP >110 mmHg	maintain SBP <180 mmHg and DBP <105 mmHg for 24 hours	labetalol, urapidil	sodium nitroprusside, hydralazine, nifedipine
	Without thrombolytic therapy: SBP >220 mmHg or DBP >120 mmHg	reduce SBP by 10–15% within 2–3 hours, by 15–25% within 24 hours	labetalol, urapidil	sodium nitroprusside, hydralazine, nifedipine
Haemorrhagic stroke	SBP >180 mmHg or MAP >130 mmHg	Normal intracranial pressure: SBP <160 mmHg and MAP <110 mmHg for 24 hours Raised intracranial pressure: SBP <180 mmHg, MAP <130 mmHg, cerebral perfusion pressure >60–80 mmHg. Reduction of SBP to 140 mmHg is considered safe	labetalol, urapidil	sodium nitroprusside, hydralazine, nifedipine
Subarachnoid haemorrhage	SBP >160 mmHg	Before surgery: reduce and maintain SBP <140 mmHg After surgery: maintain SBP <200 mmHg	labetalol, urapidil, nimodipine for all patients (to prevent cerebral vasospasm)	sodium nitroprusside, hydralazine, nifedipine
Acute coronary syndrome	SBP >160 mmHg or DBP >100 mmHg	reduction of systolic BP by 20–30%	β-adrenoceptor blockers, nitroglycerin; alternative - urapidil	sodium nitroprusside, enalaprilat
Acute left ventricular failure	at the presenting BP level	reduction of systolic BP by 20–30%	nitroglycerin / sodium nitroprusside + loop diuretic;	esmolol, metoprolol, labetalol

			alternatives - urapidil, enalaprilat	
Intra- and postoperative hypertension	SBP or MAP >20% above preoperative baseline	A decrease in DBP by 10–15% or to 110 mmHg within 30–60 min. Overall, the reduction in mean arterial pressure should not exceed 25%. BP lowering should be performed against a background of moderate infusion therapy.	urapidil, labetalol, esmolol	-
	Cardiac surgery: BP >140/90 mmHg or MAP >105 mmHg	maintain SBP <140 mmHg and DBP <90 mmHg	urapidil, labetalol, esmolol, sodium nitroprusside	β-blockers in patients with concomitant heart failure
Eclampsia / pre-eclampsia	SBP >160 mmHg or DBP >105 mmHg	SBP 140–160 mmHg and DBP 90–105 mmHg	magnesium sulphate, labetalol, urapidil, nifedipine	ACE inhibitors, sodium nitroprusside, diuretics
Hypersympathetic state (phaeochromocytoma, cocaine or amphetamine intoxication, clonidine withdrawal)	at the presenting BP level	reduction of systolic BP by 20–30%	urapidil; alternatives - nitroglycerin, sodium nitroprusside, verapamil	β-adrenoceptor blockers without prior α-blockade

*Notes.SBP – systolic blood pressure; DBP – diastolic blood pressure; MAP – mean arterial pressure; TLT – thrombolytic therapy; ICP – intracranial pressure.

.

Appendix 2

Parenteral therapy for complicated hypertensive crises [1]

Drug name		**Onset of action**	**Duration of action**	**Notes**
Sodium nitroprusside	intravenous infusion 0.25–10 μg/kg/min (50–100 mg in 250–500 ml of 5% glucose)	immediate	1–3 min	Suitable for urgent BP reduction in any hypertensive crisis. Must be administered only via a controlled infusion pump with continuous BP monitoring.
Nitroglycerin	intravenous infusion 50–100 μg/min	2–5 min	3–5 min	Particularly effective in acute heart failure and myocardial infarction.
Verapamil	IV bolus 5–10 mg; may be continued as IV infusion 3–25 mg/h	1–5 min	10–30 min	Contraindicated in heart failure and in patients receiving β-blockers
Enalaprilat	IV 1.25–5 mg	15–30 min	6 h	Effective in acute left ventricular failure
Nimodipine	IV infusion 15 μg/kg/h, then 30 μg/kg/h	10–20 min	2–4 h	Used in subarachnoid haemorrhage.
Antiadrenergic drugs				
Labetalol	IV bolus 20–80 mg at 2 mg/min, or IV infusion 50–100 mg	5–10 min	4–8 h	Effective in most hypertensive crises. Avoid in heart failure.
Propranolol	IV infusion 2–5 mg at 0.1 mg/min	10–20 min	2–4 min	Preferred in aortic dissection and acute coronary syndrome.
Esmolol	80 mg bolus, then infusion 250 μg/kg/min	1–2 min	10–20 min	Drug of choice in aortic dissection and postoperative hypertension
Clonidine	IV 0.5–1.0 ml or IM 0.5–2.0 ml of 0.01% solution	5–15 min	2–6 h	Undesirable in stroke

Phentolamine	IV or IM 5–15 mg (1–3 ml of 0.5% solution)	1–2 min	3–10 min	Preferred in phaeochromocytoma and clonidine-withdrawal syndrome.
Other drugs				
Furosemide	IV bolus 40–80 mg	5–30 min	6–8 h	Preferred in hypertensive crises with acute heart or renal failure
Torasemide	IV 10–100 mg	5–30 min	6–8 h	Preferred in hypertensive crises with acute heart or renal failure
Magnesium sulphate	IV bolus 5–20 ml of 25% solution	30–40 min	3–4 h	Used in seizures and eclampsia

Note: As of 01.03.2012, the medicinal product phentolamine is not registered in Ukraine.

Appendix 3

**to the Unified Clinical Protocol of Emergency, Primary, Secondary (Specialised) and Tertiary (Highly Specialised) Medical Care and Medical Rehabilitation "Acute Coronary Syndrome with ST- segment Elevation"

PATIENT ASSESSMENT – ACCORDING TO THE ABCDE ALGORITHM

1. A – Airway

1.1. Identify signs of airway obstruction:

Obstruction of the airway may lead to paradoxical breathing and the use of accessory respiratory muscles. Central cyanosis is a late sign of airway obstruction. In critically ill patients, impaired consciousness often causes airway compromise (tongue or soft palate falling back).

1.2. High- concentration oxygen:

Administer via a reservoir mask; ensure that the oxygen flow is adequate (>10 L/min).

2. B – Breathing

During the assessment of breathing, it is essential to identify and treat life- threatening conditions such as severe asthma attack, pulmonary oedema, tension pneumothorax, and haemothorax.

2.1. Identify symptoms that may indicate respiratory compromise:

Excessive sweating, central cyanosis, use of accessory muscles, or abdominal breathing.

2.2. Determine the respiratory rate:

Normal range is 12–20 breaths per minute.

2.3. Assess the pattern of breathing:

Evaluate the depth of breaths and check whether chest movements are symmetrical.

2.4. Observe for:

Distended neck veins (e.g., in severe asthma or tension pneumothorax), the presence and patency of a pleural drain, and other relevant signs.

2.5. Perform auscultation and percussion of the lungs.

2.6. Assess tracheal position:

Deviation may indicate tension pneumothorax, lung fibrosis, or fluid in the pleural cavity.

3. C – Circulation

3.1. Assess skin colour on exposed areas (hands):

Blue, pink, pale, or mottled.

3.2. Assess limb temperature:

Cold or warm.

3.3. Assess capillary refill time:

Normally ≤2 seconds. Prolonged refill may indicate reduced peripheral perfusion.

3.4. Assess venous filling:

Veins may be moderately filled or collapsed in hypovolaemia.

3.5. Determine heart rate:

Check peripheral and central pulses; assess presence, rate, quality, regularity, and symmetry.

3.6. Measure arterial blood pressure.

3.7. Auscultate heart sounds.

3.8. Look for other signs of reduced cardiac output:

Altered consciousness, oliguria (urine output <0.5 ml/kg/h).

4. D – Disability (Neurological status)

The most common causes of altered consciousness are severe hypoxia, hypercapnia, cerebral ischaemia, or the use of sedative or analgesic medications.

4.1. Assess pupils:

Diameter, symmetry, and reaction to light.

4.2. Rapidly assess level of consciousness using the AVPU scale:

Alert, responds to Voice, responds to Pain, Unresponsive.

The Glasgow Coma Scale may also be used.

4.3. Measure blood glucose to exclude hypoglycaemia:

If glucose is <3 mmol/L, administer 50 ml of 20% glucose intravenously.

5. E – Exposure (Additional information)

5.1. Obtain a detailed history from the patient, relatives, or friends.

5.2. Review the patient’s medical documentation:

Check vital signs and their trends and verify which medications have been prescribed and which are being taken.

Appendix 4

Medications for the treatment of uncomplicated hypertensive crises [1]

Drug	Dose and route of administration	Onset of action (min)	Side effects
Nifedipine	10–20 mg orally or sublingually	15–30 min	headache, tachycardia, flushing, angina pectoris
Captopril	12.5–50 mg orally or sublingually	15–45 min	hypotension in patients with renin-dependent hypertension
Prazosin	0.5–2 mg orally	30 min	orthostatic hypotension
Propranolol	20–80 mg orally	30–60 min	bradycardia, bronchoconstriction
Dibazol (Bendazol)	1% solution: 3.0–5.0 ml IV or 4.0–8.0 ml IM	10–30 min	more effective when combined with other antihypertensive agents
Piroxan	1% solution: 2.0–3.0 ml IM	15–30 min	orthostatic hypotension
Diazepam	0.5% solution: 1.0–2.0 ml IM	15–30 min	dizziness, drowsiness
Furosemide	40–120 mg orally or IM	5–30 min	orthostatic hypotension, weakness
Torasemide	10–100 mg orally or IM	5–30 min	orthostatic hypotension, weakness
Metoprolol	50–100 mg orally or 5–10 mg IV slowly	20–30 min or (IV) 3–5 min	bradycardia, bronchoconstriction
Clonidine	0.01% solution: 0.5–2.0 ml IM or 0.075–0.3 mg orally	30–60 min	Side effects: dry mouth, drowsiness Contraindicated in patients with AV block or bradycardia

Note:

We propose the recommendations developed by the Working Groups on Emergency Cardiology and Arterial Hypertension of the Ukrainian Association of Cardiologists, in cooperation with the Scientific and Practical Society of Neurologists, Psychiatrists and Narcologists of Ukraine (Arterial Hypertension, 2011, No. 3, pp. 64–95).

Appendix 5

Diagnostic Criteria for the Severity of Pre-eclampsia / Eclampsia [1]

Diagnosis	Clinical features	Provision of medical care
Gestational hypertension or mild pre-eclampsia	DBP 90–99 mmHg Proteinuria <0.3 g/day	Obstetrician-gynaecologist at the antenatal clinic
Moderate pre-eclampsia	DBP 100–109 mmHg Proteinuria 0.3–5.0 g/day Oedema of the face and hands	Occasional headache Hospitalisation to an obstetric inpatient unit
Severe pre-eclampsia	DBP ≥110 mmHg Proteinuria >5 g/day Generalised, pronounced oedema Severe headache Visual disturbances Pain in the epigastrium and/or right upper quadrant Hyperreflexia Oliguria (<500 ml/day) Thrombocytopenia	Urgent hospitalisation by an emergency medical team to the intensive care unit of an obstetric hospital providing tertiary medical care
Eclampsia	DBP ≥90 mmHg Proteinuria ≥0.3 g/day One or more convulsive seizures	Emergency medical care: seizure control, restoration of airway patency, hospitalisation to the intensive care unit of an obstetric hospital providing tertiary medical care, by an emergency medical team

Note

The presence of at least one criterion of a more severe form of pre- eclampsia is sufficient to establish the corresponding diagnosis.

Appendix 6

Management Strategy for Hypertensive Crises

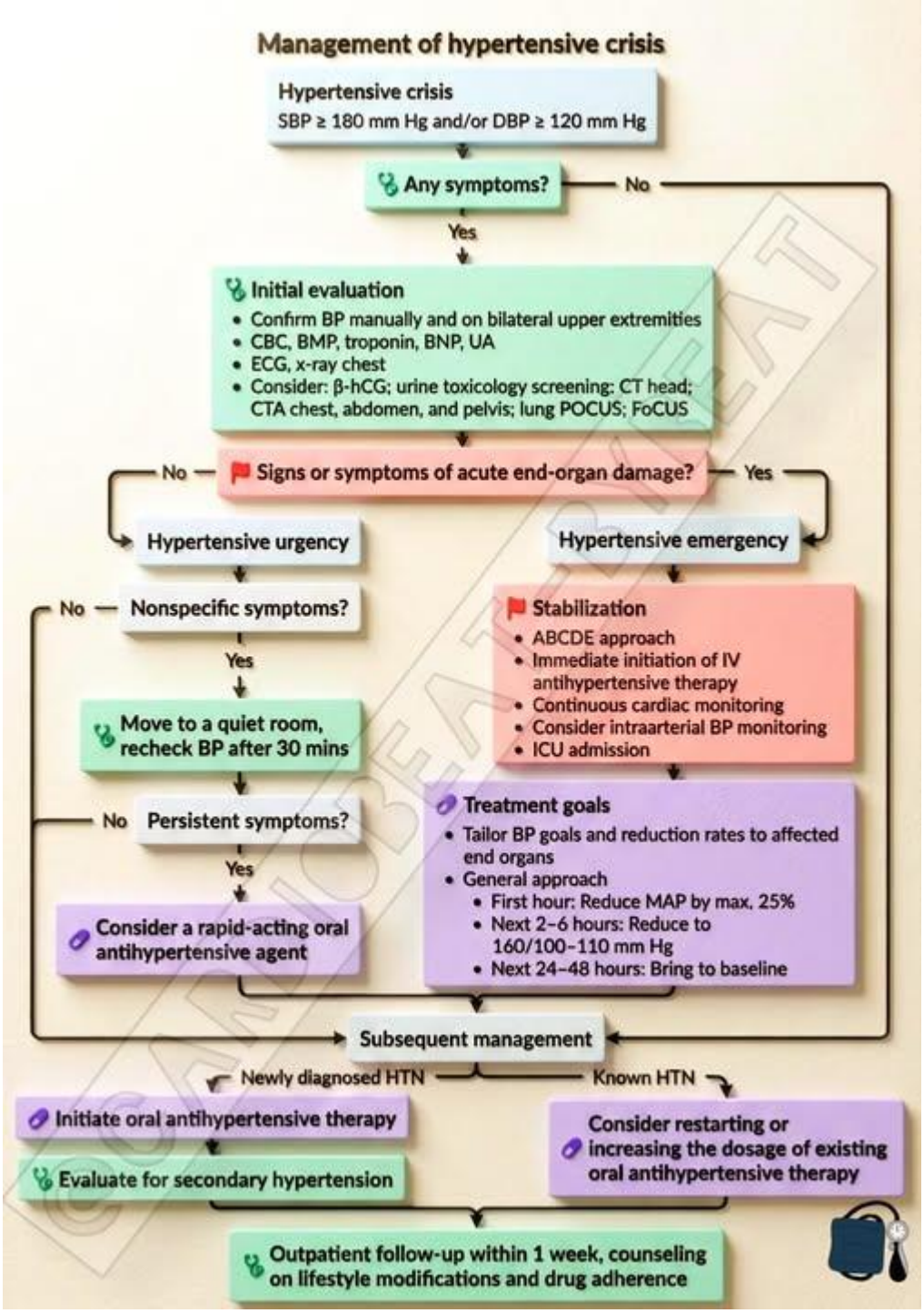

Image source: https://www.facebook.com/abdallah89othman/posts/-hypertensive-crisis-managementhypertension-hypertensivecrisis-emergencymedicine/1280958147382949/

Appendix 7

Management algorithm in patients with acute pulmonary edema

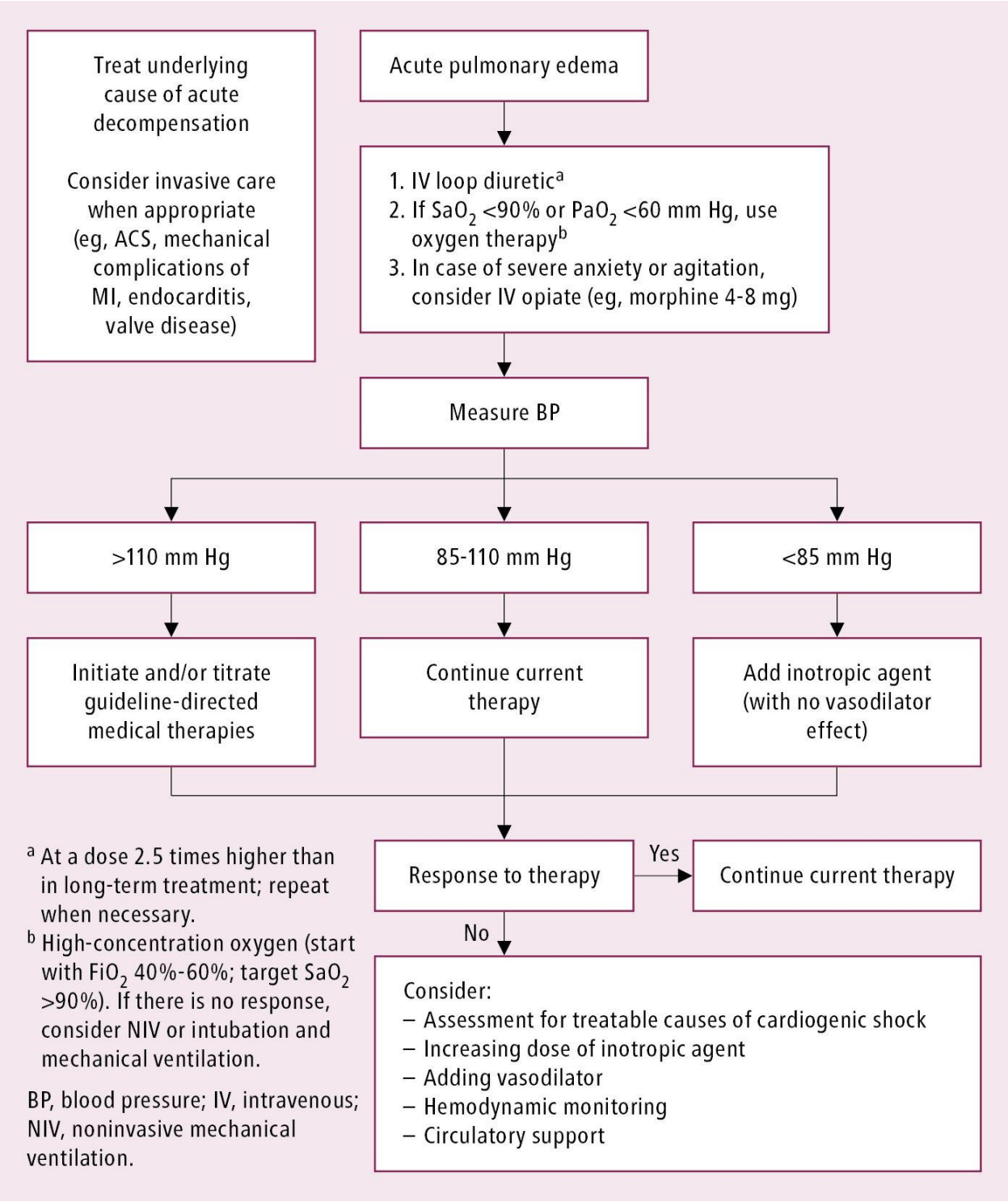

Image source: https://ebm.one/en/chapter-figure/management-algorithm-patients-acute-pulmonary-edema.-based-eur-j-heart-fail.

RECOMMENDED LITERATURE

Basic

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