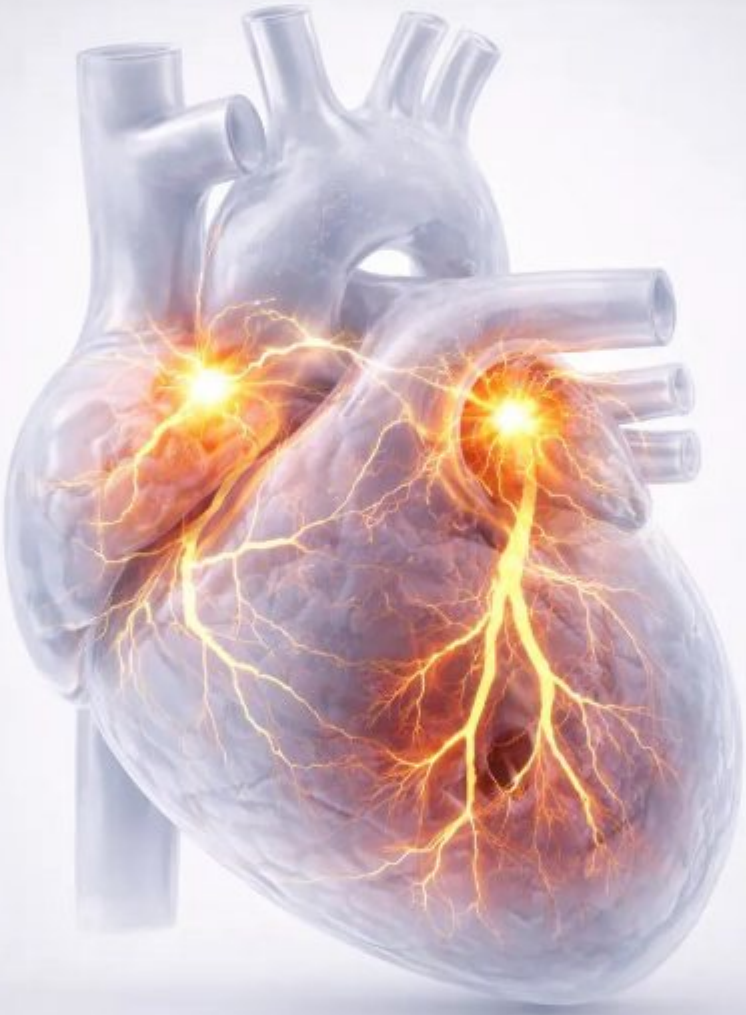
ARRHYTHMIA

National Standard Treatment Guideline



Ministry of Health
Republic of Maldives

ADB

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Japan Fund for Prosperous and Resilient Asia and the Pacific

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World Health Organization
Maldives

National Standard Treatment Guidelines

- Acid Peptic Disease
- Acute Anxiety
- Acute Pancreatitis
- Acute Psychosis
- Acute kidney Injury
- Arrhythmia
- Chronic Liver Disease
- Chronic Pancreatitis
- Chronic kidney disease
- Congenital Heart Diseases
- Dementia
- Depression
- Diabetes Mellitus Type 1
- Diabetes Mellitus Type 2
- Gestational Diabetes
- Epilepsy
- Heart Failure
- Hyponatremia
- Hypernatremia
- Hypokalemia
- Hyperkalemia
- Interstitial Lung Disease
- Liver Failure
- Obesity
- Obstructive Sleep Apnoea
- Osteoarthritis
- Ovarian Cancer
- Pneumonia
- Stroke
- Upper Gastrointestinal bleed
- Unstable Angina

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GUIDELINES DEVELOPMENT METHODOLOGY

The development of the Maldives Standard Treatment Guidelines (STGs) followed a structured, evidence-informed, and consensus-driven methodology adapted from internationally accepted guideline-development standards and the Delhi Society for Promotion of Rational Use of Drugs (DSPRUD) model. The process combined systematic evidence retrieval, critical appraisal, contextual adaptation, and multidisciplinary expert review to ensure feasibility, clinical relevance, and national ownership.

1. Determining Scope and Priority Conditions

Priority clinical conditions were identified through consultation with national programme managers, specialty clinicians, and health-system stakeholders. Selection criteria included: (i) major causes of morbidity and mortality, (ii) observed variation in clinical practice or prescribing patterns, (iii) potential to improve patient outcomes, and (iv) the feasibility of implementation across health-facility levels in Maldives. The final list of diseases reflected national epidemiology, service-delivery capacity, and essential-medicine availability.

2. Identification of Existing Evidence and Source Guidelines

A targeted search strategy was used to identify high-quality existing clinical guidelines. Searches were conducted across international guideline repositories (e.g., WHO, NICE, SIGN and other intergovernmental bodies, international and national guideline repositories, specialty societies and professional associations.

3. Quality Appraisal of Source Guidelines

Retrieved guidelines were screened for transparency of development, methodological rigour, clarity of recommendations, applicability to health-system reality, editorial independence. Guidelines were included if they met the Institute of Medicine (IOM) definition of a clinical guideline and addressed treatment or management of priority conditions. Guidelines that did not meet minimum quality standards, review articles, diagnostic criteria, or technical standards were excluded.

4. Adoption, Adaptation, and Contextualization

The guideline-development team employed an adopt–adapt–contextualize model:

- **Adoption**: High-quality recommendations that aligned with Maldivian health-system realities were retained without modification.
- **Adaptation**: Recommendations were modified when local considerations such as diagnostic capacity, medicine availability, workforce skills, referral pathways, or cost constraints affected feasibility.

- **Contextualization**: Where evidence was absent or inconclusive, conditional recommendations were formulated based on expert consensus, with explicit consideration of pragmatism, safety, and local workflows.
  Medicines were selected in alignment with the Maldives National Essential Medicines List (NEML), based on suitability, efficacy, safety, and availability.

5. Expert Consensus and Multidisciplinary Input

Draft recommendations were initially prepared by experts from the DSPRUD, India, providing a strong methodological foundation for the process. Building on this, a collaborative and participatory process brought together clinicians from internal medicine, paediatrics, obstetrics-gynaecology, surgery, emergency medicine, endocrinology, cardiology, general practitioners, and public health representing different levels of healthcare. Consensus was achieved through moderated discussions, iterative revisions, and resolution of divergent views. For topics lacking strong evidence, recommendations were derived from expert clinical judgment grounded in extensive practice experience.

6. Drafting, Peer Review, and Validation

Each guideline section was organized in a standard format including key clinical features, essential investigations, non-pharmacological management, pharmacological therapy (with step-up/step-down options where relevant), referral criteria, paediatric considerations, and follow-up requirements. Drafts were peer-reviewed by senior clinicians and national experts. Reviewer comments were systematically integrated to strengthen clarity, accuracy, and applicability.

7. Addressing Conflicts of Interest

All contributors declared the absence of conflicts of interest. Individuals with potential or perceived conflicts were excluded from authorship or decision-making roles.

8. Updating and Future Revisions

The STGs were conceptualized as a living document. Future updates will incorporate new scientific evidence, changes in essential-medicine availability, national programme priorities, and user feedback from clinicians. Periodic review cycles will ensure the continued relevance and reliability of recommendations.

9. Distinctive Features of the Guidelines

Developed through a collaborative process involving a large group of multidisciplinary experts from different levels of healthcare, the guidelines incorporate the following distinctive features:

- **Diagnostic Assumption and Confirmation**: While assuming that an initial diagnosis has been established by the healthcare provider, the guidelines provide essential information for confirming diagnoses. This includes a comprehensive overview of major signs and symptoms, descriptions of confirmatory tests, and clear guidance on practices that are prohibited, discouraged, or unreliable—promoting evidence-based medicine supported by relevant references.
- **Comprehensive Treatment Approach**: The guidelines offer a systematic, up-to-date framework for managing medical conditions across the continuum of care. They begin at the primary care level and extend to secondary and tertiary care, incorporating protocols for treatment response assessment and referral criteria as integral components.
- **Diverse Treatment Modalities**: Recommendations encompass both non-pharmacological and pharmacological interventions and surgical intervention where applicable, providing flexibility for individualized treatment plans. Cautionary notes are included where necessary to ensure safe and effective use of therapies.
- **Assessment and Referral Criteria**: Clear criteria and goals for evaluating patient response to treatment are provided, along with guidance on when referral to higher levels of care is warranted ensuring continuity and comprehensiveness in patient management.

ACKNOWLEDGEMENTS

The Government of the Republic of Maldives is committed to ensuring universal access to quality health services for all citizens. The Constitution of Maldives mandates the progressive realization of rights, including the right to good standards of health care for the population. In line with this national commitment, standardized quality health services are regarded as the foundation of a strong and equitable healthcare system.

This important work would not have been possible without the cooperation and support of many individuals and institutions. We express our sincere appreciation to the Honourable Minister of Health, Abdullah Nazim Ibrahim, for his leadership, commitment, and continuous guidance throughout the development process. We are grateful to WHO and ADB for their significant contribution, support, and technical assistance.

Our heartfelt gratitude is extended to the technical lead and editor, Dr. Sangeeta Sharma, Professor, Neuropsychopharmacology, IHBAS and President, Delhi Society for Promotion of Rational Use of Drugs (DSPRUD), and her team. We express our deepest appreciation to the Maldivian and DSPRUD experts and contributors who played a pivotal role in this process. Their technical expertise and dedication to adapt the standards to the Maldivian context have been instrumental in the development and finalization of these guidelines. The time, experience, generous sharing of knowledge and insights contributed by all parties have not only enriched the work but also have been invaluable in making these standards practical, locally acceptable, and aligned with the needs of the resident population.

It is important to acknowledge the immense efforts, involvement, timely coordination, collaboration, and dedication of the Quality Assurance and Regulation Division team who made it possible for these Clinical Treatment Guidelines to come into existence.

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ARRHYTHMIA

QUICK REFERENCE GUIDE

Arrhythmias affect about 1–1.5% of adults, with prevalence rising sharply with age. Atrial fibrillation and bradyarrhythmia's are more common in older adults, while paroxysmal supraventricular tachycardia (SVT) tends to occur in younger individuals; ventricular arrhythmias can appear at any age when structural heart disease is present. Morbidity includes increased risk of stroke, heart failure, and sudden cardiac death & untreated malignant ventricular arrhythmias have a mortality exceeding 90%, and atrial fibrillation raises stroke risk fivefold, making timely diagnosis and management critical for favorable outcomes.

Arrhythmia is defined as any cardiac rhythm other than normal sinus rhythm due to abnormal impulse formation or conduction.

- By rate: Bradyarrhythmia (<60 bpm); Tachyarrhythmia (>100 bpm).
- By origin: Supraventricular tachyarrhythmia (SVT): atrial fibrillation (AF), atrial flutter, atrioventricular nodal re-entry tachycardia (AVNRT), atrioventricular re-entry tachycardia (AVRT/Wolff-Parkinson-White [WPW]), atrial tachycardia. Ventricular: ventricular tachycardia (VT), ventricular fibrillation (VF), torsades de pointes (TdP).
- Conduction/brady disorders: Sinus node dysfunction (sick sinus syndrome), atrioventricular (AV) block (first-, second- [Mobitz I/II], third-degree).

Causes, risk factors & triggers

- Cardiac: Ischemic heart disease, cardiomyopathy, myocarditis, valvular disease, congenital pathways (WPW).
- Systemic: Thyroid disorders, electrolyte imbalance (potassium/ magnesium/calcium), hypoxia, infection/sepsis, drug toxicity (digoxin, QT-prolonging agents).
- Triggers: Alcohol binges, high caffeine/ energy drinks, stimulant drugs, stress, fever (Brugada), surgery, poor sleep, dehydration.

Evaluation for diagnosis

- Clinical features: Palpitations, dyspnea, chest discomfort, presyncope/syncope, fatigue; red flags = hypotension, shock, ongoing chest pain, hypoxia, altered consciousness.
- Physical examination: Vitals (heart rate, blood pressure, respiratory rate, temperature, oxygen saturation), pulse regularity, signs of heart failure (elevated jugular venous pressure, edema), murmurs, thyroid signs, focused neurologic check.

  Investigations:
- Electrocardiogram (ECG): 12-lead during symptoms (gold standard); baseline ECG between episodes. Telemetry/rhythm strip.

- Ambulatory monitoring: Holter (24-48 h), event/loop recorder, implantable loop recorder for infrequent syncope.
- Laboratory tests: Complete blood count, electrolytes, renal/liver function, thyroid panel, troponin if ischemia, inflammatory markers if myocarditis, toxicology as indicated.
- Imaging: Transthoracic echocardiography (TTE) ± transesophageal echocardiography (TEE) for thrombus/valves; cardiac magnetic resonance (CMR) if myocarditis/scar is suspected.
- Confirmation: Symptom-rhythm correlation; exclude artifacts (lead misplacement/motion).

Classification / severity assessment

- Regular vs irregular; narrow vs wide QRS; stable vs unstable.
- Unstable (emergency): Systolic blood pressure <90 mmHg, shock, acute pulmonary edema, ischemic chest pain, syncope - requires immediate synchronized cardioversion for unstable tachyarrhythmia; defibrillation for VF/ pulseless VT; atropine/pacing for unstable bradyarrhythmia.
- Potentially unstable: Sustained VT (with pulse), AF with rapid ventricular response >150 bpm, high-grade AV block.
- Stable: No hemodynamic compromise; proceed with algorithmic care.

Differential diagnosis

Sinus tachycardia (physiologic), anxiety/ panic, hyperthyroidism, sepsis, pulmonary embolism, drug effects (β-agonists, decongestants), seizures, hypoglycemia, artifact.

Management goals & principles

- Stabilize airway, breathing, circulation; restore/control rhythm or rate; maintain cardiac output; prevent thromboembolism.
- Match therapy to rhythm, hemodynamics, and substrate; correct reversibles; avoid pro-arrhythmia; use stepwise escalation; standardize referral.

Approach to management (stepwise)

1. Assess stability if unstable = shock/ cardioversion/defibrillation/pacing per rhythm.
2. Identify rhythm on ECG (regular/ irregular, narrow/wide).
3. Treat per rhythm (see drug table).
4. Search/treat causes (electrolytes, ischemia, hypoxia, drugs, thyroid).
5. Decide on referral level (primary / secondary /tertiary) and monitoring.

Non-pharmacological interventions

- SVT: Modified Valsalva; carotid sinus massage only if trained and no carotid disease.
- Bradyarrhythmia: Transcutaneous pacing where available; temporary transvenous pacing at higher level.
- VT/VF: Early defibrillation; high-quality cardiopulmonary resuscitation (CPR).
- System measures (low-resource): Pulse/ECG opportunistic screening in ≥65 y or high-risk; tele-ECG; essential drug/defibrillator checklists; electrolyte repletion protocols; transport/referral network.

Pharmacological therapy

Scenario	Drug & dose (route)	Duration/notes	Key cautions
Paroxysmal SVT (stable) after vagal failure	Adenosine 6 mg IV rapid bolus + 20 mL flush - if no effect in 1-2 min administer 12 mg; may repeat 12 mg once	Single-episode termination	Avoid in asthma/severe bronchospasm, heart transplant (use smaller dose), 2°/3° AV block without pacemaker; transient asystole/flush common
SVT persistent	Metoprolol 2.5-5 mg IV over 2 min, repeat to max 15 mg or Verapamil 5-10 mg IV over 2 min	Consider oral maintenance or ablation referral	Avoid non-dihydropyridine calcium channel blockers (verapamil/diltiazem) in WPW with AF, hypotension, left ventricular dysfunction
Atrial fibrillation/ flutter (<48 h, stable)	Rate control: Metoprolol IV as above followed by oral 25-100 mg twice daily or Diltiazem 0.25 mg/kg IV then titrate to 5-15 mg/h infusion	Rhythm control if eligible (electrical or pharmacologic)	Watch for hypotension/ bradycardia; avoid AV nodal blockers in WPW
Atrial fibrillation/ flutter (≥48 h or unknown)	Anticoagulation: Apixaban 5 mg orally twice daily (dose-adjust per criteria) or Warfarin (international normalized ratio [INR] 2-3)	Rate control ongoing; rhythm control after anticoagulation or TEE	Balance stroke vs bleeding (use CHA_2DS_2-VASc and HAS-BLED scores)
Rhythm control option	Amiodarone 150 mg IV over 10 min followed by 1 mg/min for 6 h then 0.5 mg/min	Convert/maintain; consider oral loading 200 mg three times daily (1 week) then taper	QT prolongation; thyroid/liver/ lung toxicity; drug interactions
Monomorphic VT (stable, with pulse)	Amiodarone as above or Procainamide 20-50 mg/min IV until suppression/hypotension/QRS increase >50% or total 15 mg/kg	Continuous monitoring	Procainamide: avoid in prolonged QT or heart failure
VF / Pulseless VT	Defibrillation + Epinephrine 1 mg IV/IO every 3-5 min; Amiodarone 300 mg IV bolus; 150 mg if recurrent	Follow ACLS cycles	Minimize CPR pauses; treat causes (Hs & Ts)
Torsades de pointes (polymorphic VT with long QT)	Magnesium sulfate 2 g IV over 1-2 min (unstable) or 10-15 min (perfusing); may repeat	Correct potassium/ calcium; stop QT-prolonging drugs; pace if pause-dependent	Monitor blood pressure and QT interval

Symptomatic sinus bradycardia	Atropine 0.5 mg IV every 3-5 min (max 3 mg); if ineffective Dopamine 2-10 mcg/kg/min IV or Epinephrine 2-10 mcg/min IV	Prepare for pacing if no response	Atropine may be ineffective in high-grade block; caution in suspected ischemia

Assessment of response, review, follow-up

- Immediate: Symptom relief; hemodynamic stability; ECG shows conversion or adequate rate control; no malignant rhythms; no major adverse effects.
- Early (24-48 h): Continuous monitoring for recurrence/QT; maintain potassium 4.0-5.0 mmol/L and magnesium ≥0.8 mmol/L; check drug interactions/renal-hepatic function.
- Weeks: Symptom diary; ECG/Holter; drug tolerance (bradycardia, hypotension, QT).
- Adjust treatment / step-up: Persistent instability, recurrent symptomatic episodes on optimal therapy, new high-grade AV block, refractory VT/VF then escalate, consider ablation/device.
- Step-down: Stable rhythm/rate on minimal therapy, no recent events, comorbidities controlled, space follow-ups, continue education and trigger control.

Referral (tiered pathway)

- Primary to Secondary (urgent): Unstable rhythm, ventricular arrhythmia, high-grade AV block, AF with ischemia, suspected acute coronary syndrome.
- Primary to Secondary (planned): Stable arrhythmia needing Holter/echo; suspected genetic syndrome; symptomatic bradycardia for pacing evaluation.
- Secondary to Tertiary: Complex arrhythmia for EPS/ablation; device therapy (pacemaker, ICD, CRT); refractory or recurrent VT/VF; advanced heart failure care; genetic evaluation.

Complications

Hemodynamic collapse (syncope, shock), thromboembolism (stroke/systemic), tachycardia-induced cardiomyopathy, heart failure decompensation, sudden cardiac death, treatment-related harms (torsades, organ toxicity, device/procedural complications), psychological burden and reduced quality of life.

Patient education & instructions

- Explain the arrhythmia type and risks (stroke, heart failure, sudden death).
- Recognize warning signs (palpitations, dizziness, chest pain, fainting) and when to seek urgent care.
- Medicines: Take exactly as prescribed; know side effects; never stop/change doses without advice.
- Anticoagulation in AF: Adhere strictly; keep INR checks if on warfarin; watch for bleeding.

- Lifestyle/Triggers: Heart-healthy diet, hydration, regular moderate exercise as cleared; avoid smoking, binge alcohol, high caffeine, stimulants; manage stress and sleep.
- Home monitoring: Learn pulse check; keep symptom/trigger diary; use devices if advised.
- Over-the-counter/herbal products: Discuss with clinician before use.
- Emergency readiness: Keep medication/ allergy list and contacts; caregivers aware of cardiopulmonary resuscitation (CPR) basics and nearest facility; carry updated records when traveling.
- Devices (pacemaker/ICD): Attend checks; follow activity guidance; avoid strong magnets or devices that interfere.

INTRODUCTION

Arrhythmias are abnormal heart rhythms caused by disordered impulse formation or conduction; they range from harmless to life-threatening. Early recognition and treatment prevent stroke, heart failure, syncope, and sudden death. Cardiovascular disease causes about 17.9 million deaths each year, and in Southeast Asia arrhythmias often accompany ischemic heart disease, hypertension, rheumatic disease, and metabolic disorders. Atrial fibrillation (AF) affects roughly 1-1.5% of adults and rises with age; Maldives hospitals report more AF, paroxysmal supraventricular tachycardia (PSVT), and bradyarrhythmia's linked to coronary artery disease, obesity, and diabetes. Patterns vary by age: AF and bradyarrhythmia's in older adults, PSVT in younger people, and ventricular arrhythmias at any age with underlying heart disease. Risks are substantial as untreated malignant ventricular arrhythmias can be fatal in over 90%, AF raises stroke risk fivefold, and high-grade atrioventricular block can cause sudden death. Timely, accurate management cuts these risks and avoids both over- and undertreatment. Standardized, evidence-based algorithms help teams triage and escalate care consistently, especially in resource-limited settings. Structured protocols allow frontline workers to triage and manage arrhythmias efficiently, escalate care when necessary, and minimize practice variation.

The Maldives' National Multisectoral Action Plan for non-communication diseases (NCDs) 2023-2031 supports earlier detection, integrated care, and risk-factor control through stronger primary care and community engagement to advance universal health coverage.

SCOPE OF THE GUIDELINES

These guidelines cover the recognition, diagnosis, and management of the most common tachyarrhythmias and bradyarrhythmias encountered in clinical practice. The primary focus is on medical evaluation and stabilization, with surgical and electrophysiological interventions such as catheter ablation, pacemaker, and implantable cardioverter-defibrillator (ICD) implantation mentioned only in brief to maintain clinical relevance without overcomplicating decision-making at non-specialist levels. The document is designed for use across primary and secondary healthcare settings, particularly in environments where cardiology subspecialty services may not be immediately accessible. It aims to bridge the gap in care during the critical period before referral to tertiary facilities, ensuring timely, standardized, and effective management in resource-variable settings.

The guidelines are intended for a broad range of healthcare professionals involved in the acute and ongoing care of patients with arrhythmias. These include primary care doctors, emergency physicians, internists, nurses, paramedics, and general practitioners. By providing a clear, stepwise approach, the guidelines help these professionals identify,

stabilize, and initiate evidence-based management for arrhythmia patients, ensuring that interventions are appropriate to the clinical situation and available resources.

These guidelines focus on recognizing, diagnosing, and initially managing common tachyarrhythmias and bradyarrhythmia's. The emphasis is on medical evaluation and stabilization; advanced procedures such as ablation, pacemakers, and ICDs are mentioned only for context. The guidelines are intended for a broad range of healthcare professionals involved in the acute and ongoing care of patients with arrhythmias including teams at primary and secondary care where cardiology subspecialists may not be immediately available, including primary care doctors, emergency physicians, internists, nurses, paramedics, and general practitioners. A clear stepwise approach supports rapid identification, ECG recording, stabilization, evidence-based treatment, and timely referral.

Roles by level: primary care recognizes and stabilizes; secondary care confirms the rhythm, starts indicated drugs, monitors response, and prepares transfer; tertiary centres perform electrophysiology studies, ablation, and device implantation and manage refractory or high-risk cases.

DEFINITIONS

An arrhythmia is any cardiac rhythm other than normal sinus rhythm, due to abnormalities in impulse formation or conduction. (ESC)

Arrhythmias are broadly categorized based on heart rate, site of origin, and regularity of the cardiac rhythm.

- By rate: bradyarrhythmias (< ~60 bpm at rest) and tachyarrhythmias (> ~100 bpm at rest).
- By site of origin:
  - **Supraventricular** (above/below AV node but not ventricles): atrial fibrillation, atrial flutter, focal/multifocal atrial tachycardia, AV nodal re-entrant tachycardia (AVNRT), AV re-entrant tachycardia via accessory pathway (AVRT/WPW), inappropriate sinus tachycardia. (ESCARDIO)
  - **Ventricular:** premature ventricular complexes (PVCs), non-sustained VT, sustained monomorphic VT, polymorphic VT (including torsades de pointes), ventricular fibrillation; "electrical storm" is ≥3 separate sustained VAs in 24 h.

- **Conduction/brady disorders:** sinus node dysfunction (sinus bradycardia, pauses/ arrest, tachy-brady), AV block (first-degree, Mobitz I/II, high-grade/complete), bundle-branch/intraventricular conduction disease.

Within these groups, distinct types have characteristic mechanisms, ECG patterns, and clinical implications.

1. Tachyarrhythmias (Rhythms with a HR greater than 100 beats per minute)

1.1 Supraventricular Tachyarrhythmias (SVTs)

Originate above the ventricles (atria or AV node). QRS complexes are usually narrow (<120 ms) unless there is pre-existing bundle branch block or rate-related aberrancy.

- **Atrial Fibrillation (AF):** A supraventricular arrhythmia characterized by disorganized atrial electrical activity leading to ineffective atrial contraction. The ventricular response is irregularly irregular. AF increases the risk of thromboembolism, especially stroke.
- **Atrial Flutter:** A macro-reentrant atrial rhythm producing saw-tooth flutter waves on ECG, usually with a fixed conduction ratio (e.g., 2:1). The rhythm is usually regular unless variable conduction is present.
- **Paroxysmal Supraventricular Tachycardia (PSVT)**: A sudden-onset, regular, narrow-complex tachycardia, most often due to AV nodal reentrant tachycardia (AVNRT) or atrioventricular reentrant tachycardia (AVRT via an accessory pathway). Episodes terminate abruptly and are often triggered by premature beats.

1.2 Ventricular Tachyarrhythmias

Originate in the ventricles, producing wide QRS complexes (≥120 ms) and often associated with hemodynamic instability.

- **Ventricular Tachycardia (VT)**: A regular wide-complex tachycardia originating from a re-entrant circuit within diseased ventricular myocardium, often post-myocardial infarction or in cardiomyopathy. Sustained VT (>30 seconds) is potentially life-threatening.
- **Ventricular Fibrillation (VF):** Chaotic, disorganized ventricular electrical activity resulting in the absence of effective cardiac output. This is a cardiac arrest rhythm requiring immediate defibrillation.
- **Torsades de Pointes**: A form of polymorphic VT associated with a prolonged QT interval. The QRS complexes appear to twist around the baseline. Often precipitated by electrolyte disturbances, certain medications, or congenital long QT syndrome.

2. Bradyarrhythmias

Rhythms with a heart rate of less than 60 beats per minute, caused by failure of impulse generation or conduction.

2.1 Sinus Bradycardia

A sinus rhythm with a rate below 60 bpm, originating from the sinoatrial (SA) node. It may be physiological (e.g., in well-trained athletes) or pathologic (e.g., due to hypothyroidism, increased vagal tone, or drug effects such as beta-blockers and digoxin).

2.2 Sick Sinus Syndrome (Sinus Node Dysfunction)

A collection of SA node disorders leading to inappropriate bradycardia, sinus pauses, or alternating bradycardia and tachyarrhythmia (tachy-brady syndrome). Common in elderly patients due to SA node fibrosis.

2.3 Atrioventricular (AV) Block

Impaired conduction from atria to ventricles, classified into:

- **First-Degree AV Block:** Prolonged PR interval (>200 ms) with all atrial impulses conducted. Usually benign.
- **Second-Degree AV Block:**
  - *Mobitz Type I (Wenckebach):* Progressive PR prolongation until a ventricular beat is dropped.
  - *Mobitz Type II:* Constant PR interval with intermittent non-conducted P waves; often progresses to complete heart block and requires pacing.
- **Third-Degree (Complete) AV Block:** No atrial impulses are conducted to the ventricles; ventricular escape rhythm maintains a slower, independent rhythm.

Other Rhythm Disturbances

While not always classified as sustained tachy- or bradyarrhythmias, these ectopic beats and irregular rhythms can be markers of underlying pathology:

- **Premature Atrial Contractions (PACs):** Early atrial depolarizations resulting in abnormal P-wave morphology; usually benign but may precede AF.

- **Premature Ventricular Contractions (PVCs):** Early ventricular depolarizations producing wide, bizarre QRS complexes; frequent or polymorphic PVCs may indicate structural heart disease.
- **Multifocal Atrial Tachycardia (MAT):** Irregular atrial rhythm with at least three distinct P-wave morphologies, often in severe pulmonary disease.

CAUSES, RISK FACTORS & TRIGGERS

Arrhythmias may arise from structural, functional, metabolic, or environmental factors that alter the heart's normal electrical conduction.

Component	Factor	How it causes arrhythmia	Tools / Rhythms
Cardiac	Ischemic heart disease	Scar/ischemia disrupts conduction pathways	VT, VF, AF, PVCs
	Myocarditis	Inflammation injures myocytes and the conduction system	AF, AT, VT, AV block
	Cardiomyopathy (dilated/ hypertrophic/restrictive)	Structural remodeling and fibrosis	AF, VT/VF
	Valvular disease (MS/MR/AS)	Pressure/volume overload leading to atrial enlargement or LV strain	AF; PVCs/VT in severe disease
	Congenital defects (e.g., WPW, congenital AV block)	Accessory pathways or conduction system maldevelopment	AVRT (WPW), AV block, SVT
Systemic	Thyroid disease	Hyperthyroid -increases adrenergic tone; hypothyroid slows conduction	AF/SVT (hyper), brady/AV block (hypo)
	Electrolyte imbalance (K^+, Mg^{2+}, Ca^{2+}, Na^+)	Alters membrane potentials and repolarization	HypoK/HypoMg - PVCs, VT/ TdP; HyperK -brady/asystole
	Hypoxia (lung disease, OSA, high altitude)	Myocardial irritability and autonomic shifts	AF, PVCs/VT
	Infections (sepsis, endocarditis)	Cytokines, fever, ± myocardial involvement	AF, VT; brady in severe sepsis
	Drug toxicity (digoxin, antiarrhythmics, QT-prolonging, β-agonists)	Proarrhythmia, triggered activity, altered conduction	TdP, AT with block (digoxin), SVT/VT
Triggers	Caffeine (high intake)	Increased sympathetic tone	PACs/PVCs, SVT
	Alcohol (binge; "holiday heart")	Acute atrial irritability; chronic remodeling	AF
	Stimulants (cocaine, amphetamines, some decongestants)	Marked catecholamine surge, ischemia	SVT, VT/VF

	Stress (emotional/physical)	Catecholamine surge	Sinus tachy, AF/SVT; VT in susceptible patients
	Fever (e.g., Brugada syndrome)	Unmasks sodium-channel defects	VT/VF
	Surgery/anesthesia	Electrolyte shifts, inflammation, autonomic changes	AF, PVCs/VT, perioperative brady

Abbreviations: WPW Wolff-Parkinson-White, MS/MR/AS mitral stenosis/regurgitation/ aortic stenosis, TdP torsades de pointes, PAC/PVC premature atrial/ventricular complexes, OSA obstructive sleep apnea.

EVALUATION FOR DIAGNOSIS

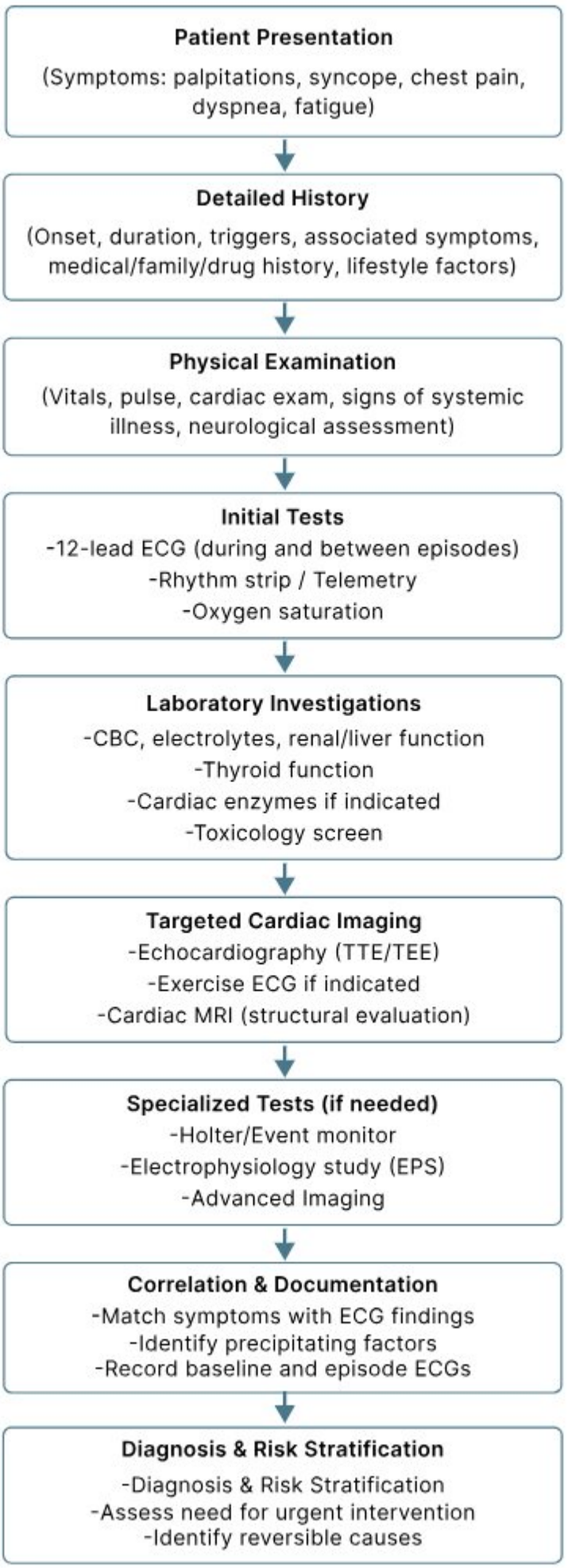
A structured evaluation is essential for accurately identifying the type of arrhythmia, determining its clinical significance, identifying reversible causes, and guiding management.

Component	Key information	Why it matters	Tools / notes
Clinical history	Onset/duration: sudden vs gradual; paroxysmal vs persistent vs permanent. Symptoms: palpitations, dizziness, syncope/near-syncope, chest pain, dyspnea, fatigue, exercise intolerance. Triggers: exertion, rest, stress, caffeine, alcohol, drugs, fever, sleep. Associated features: sweating, nausea, focal neuro deficits. PMH: IHD/structural disease, HTN, thyroid issues, prior arrhythmias. Family history: SCD, Long QT, Brugada. Medications: antiarrhythmics, β-blockers, CCBs, digoxin, QT-prolonging drugs, stimulants. Lifestyle: alcohol, caffeine, recreational drugs.	Narrows rhythm type, flags red-flags (e.g., syncope with exertion), and surfaces reversible causes.	Check symptom diary if available.
Physical exam	Vitals: HR, BP, RR, Temp, SpO_2. Pulse: rate, regularity, strength, both arms. Cardiac: murmurs, extra sounds, gallops; signs of HF (S3, JVP, edema). Systemic: goiter/ tremor, pallor, clubbing, cyanosis, fever, infection signs. Neuro screen for stroke/TIA.	Defines stability, suggests substrate (valvular disease, HF, thyrotoxicosis), detects complications (embolic events).	Check during episode and between episodes if possible.
Bedside & continuous monitoring	12-lead ECG: during symptoms (diagnostic); between episodes (baseline blocks, pre-excitation). Rhythm strip/telemetry for inpatients. Holter (24-48 h) for intermittent symptoms. Event/loop recorder for infrequent episodes (patient-activated/auto).	Captures the rhythm, correlates symptoms with ECG, reveals conduction disease.	Ensure time-stamped symptom buttons/notes for correlation.
Laboratory tests	CBC; electrolytes (K^+, Mg^{2+}, Ca^{2+}, Na^+); renal/liver function; thyroid panel; troponin/CK-MB if ischemia suspected; CRP/ESR if myocarditis/systemic inflammation; toxicology screen if indicated.	Identifies reversible drivers, informs drug dosing and safety.	Correct abnormalities before labeling arrhythmia "idiopathic."

Imaging & specialized tests	Echo (TTE/TEE): chambers, valves, LV function, thrombus. Exercise ECG/TMT: exercise-induced arrhythmias/ischemia. Cardiac MRI: myocarditis, infiltrative disease, scar. EPS: invasive mapping when mechanism unclear or ablation considered.	Defines structure/ substrate, refines risk, guides therapy (anticoagulation, ablation, devices).	Choose tests based on history/ ECG and local access.
Correlation & documentation	Match symptoms to ECG/rhythm events; keep baseline ECG and episode ECG; note activity/ trigger at onset; record treatments and response.	Ensures accurate diagnosis, avoids over/ undertreatment, supports referral decisions.	Standardized template improves continuity across levels of care.

Key Points in Diagnostic Approach

- Arrhythmia diagnosis should never rely solely on history; ECG confirmation is essential.
- Continuous or event monitoring is critical when arrhythmias are transient.
- Always assess for reversible causes concurrently with rhythm evaluation.
- Early identification of high-risk arrhythmias (e.g., VT, VF, high-grade AV block) can be life-saving.


Patient Presentation
(Symptoms: palpitations, syncope, chest pain, dyspnea, fatigue)
Detailed History
(Onset, duration, triggers, associated symptoms, medical/family/drug history, lifestyle factors)
Physical Examination
(Vitals, pulse, cardiac exam, signs of systemic illness, neurological assessment)
Initial Tests
-12-lead ECG (during and between episodes)
-Rhythm strip / Telemetry
-Oxygen saturation
Laboratory Investigations
-CBC, electrolytes, renal/liver function
-Thyroid function
-Cardiac enzymes if indicated
-Toxicology screen
Targeted Cardiac Imaging
-Echocardiography (TTE/TEE)
-Exercise ECG if indicated
-Cardiac MRI (structural evaluation)
Specialized Tests (if needed)
-Holter/Event monitor
-Electrophysiology study (EPS)
-Advanced Imaging
Correlation & Documentation
-Match symptoms with ECG findings
-Identify precipitating factors
-Record baseline and episode ECGs
Diagnosis & Risk Stratification
-Diagnosis & Risk Stratification
-Assess need for urgent intervention
-Identify reversible causes

CONFIRMATION OF DIAGNOSIS

A diagnosis is confirmed when:

1. A documented ECG or rhythm strip demonstrates arrhythmia during a symptomatic or relevant episode.
2. Pattern analysis matches known arrhythmia morphology (e.g., saw-tooth waves in atrial flutter, wide-complex rhythm in VT).
3. Clinical context supports the diagnosis, and alternative explanations (artifact, non-cardiac causes) have been excluded.

Step	Action / Tool	Diagnostic value	Indication	Pitfalls / notes
Confirm with ECG (gold standard)	12-lead ECG during symptoms	Identifies rhythm origin (SVT vs VT), regularity, QRS width, AV relation; shows blocks, pre-excitation	Any symptomatic episode if possible	If not captured, you can't label mechanism confidently
Baseline ECG	12-lead ECG between episodes	Finds conduction defects, prior MI, pre-excitation, repolarization changes	After initial visit or post-event	Compare with event ECGs for change over time
Capture intermittent events	Holter (24-48 h)	Continuous recording for frequent, brief arrhythmias; symptom-rhythm correlation	Daily symptoms/ palpitations	Misses rare events
	Event/loop recorder (external)	Patient-activated/auto-triggered capture of less frequent episodes	Weekly to monthly symptoms	Needs patient training to activate/log symptoms
	Implantable loop recorder (ILR)	Long-term rhythm capture for unexplained syncope or very infrequent events	Events < monthly or high-risk syncope	Invasive; requires follow-up for downloads
Correlate with symptoms	Time-stamped logs + BP/ SpO_2	Confirms that symptoms align with ECG change; gauges hemodynamic impact	All patients with recorded events	Asymptomatic ectopy may be incidental - avoid overtreatment
Exclude artifacts	Check leads; repeat tracing	Rules out motion/poor contact/electrical noise mimicking arrhythmia	Any suspicious tracing	Verify limb/chest lead placement; look for baseline wander
Structural assessment	Echocardiography (TTE/TEE)	Chamber size, LV/RV function, valves, thrombus (TEE)	New AF, HF signs, murmur, suspected structural disease	Guides anticoagulation and rhythm strategy

Exercise link	Exercise ECG (TMT)	Unmasks exercise-induced arrhythmias or ischemia	Symptoms on exertion, CPVT suspicion	Stop if ischemia or significant arrhythmia appears
Mechanism mapping	Electrophysiology study (EPS)	Defines pathway/focus; guides ablation (SVT, WPW, some VT)	Recurrent symptomatic SVT/ VT, unclear syncope	Invasive; plan ablation when appropriate
Address reversibles	Labs: K^+, Mg^{2+}, Ca^{2+}, Na^+; TSH/T4; renal/liver; troponin/ CK-MB; CRP/ESR; tox screen	Finds correctable drivers (electrolytes, thyroid), ischemia, inflammation, drug effects	At workup or if change in status	Correct abnormalities alongside rhythm care

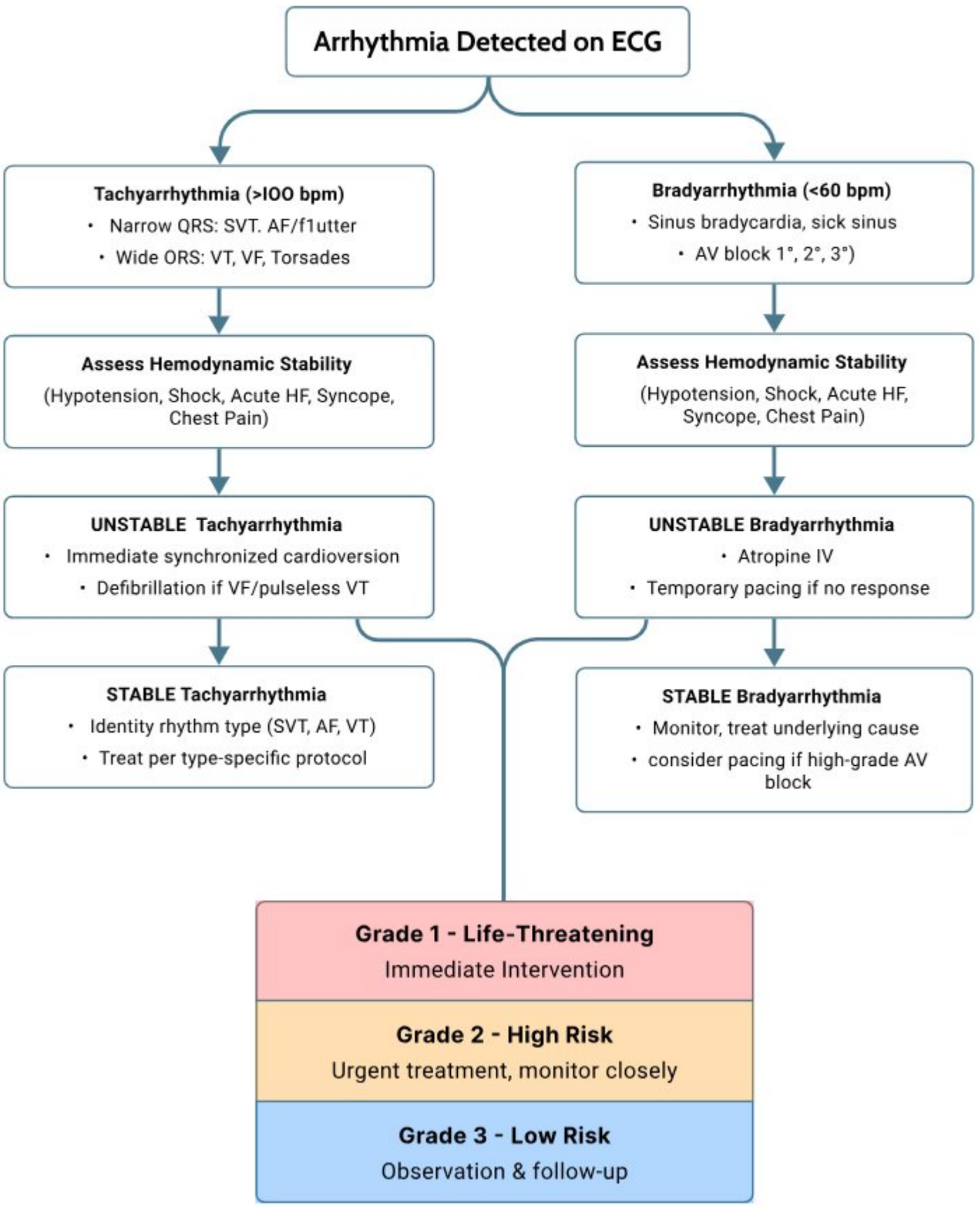
CLASSIFICATION & SEVERITY

Arrhythmias can be classified according to heart rate, site of origin, and regularity of rhythm. Severity is determined by the rhythm type, its hemodynamic impact, and the potential for life-threatening complications. This dual approach helps guide urgency of intervention and resource allocation in clinical settings.

Class	Criteria	Examples	Action / Urgency
Heart rate			
Tachyarrhythmias - Supraventricular (SVT)	HR >100 bpm; origin above ventricles	Atrial fibrillation (AF), atrial flutter, paroxysmal SVT (AVNRT/AVRT)	Rate/rhythm control; anticoagulation for AF as indicated
Tachyarrhythmias - Ventricular	HR >100 bpm; origin in ventricles	Ventricular tachycardia (VT), ventricular fibrillation (VF), torsades de pointes (TdP)	Emergency if VT/VF/TdP; cardioversion/defibrillation, magnesium for TdP
Bradyarrhythmias - Sinus node	HR <60 bpm (context-dependent)	Sinus bradycardia, sinus arrest, sick-sinus (tachy-brady)	Treat only if symptomatic/ unstable; consider atropine, pacing
Bradyarrhythmias - AV block	Delayed/failed AV conduction	First-degree, Mobitz I (Wenckebach), Mobitz II, third-degree (complete)	High-grade/complete block: urgent pacing
Regularity			
Regular	Predictable R-R intervals	Sinus rhythm, PSVT, monomorphic VT	Diagnose mechanism; treat per type
Irregular	Variable R-R intervals	AF, multifocal atrial tachycardia, polymorphic VT	Address cause; stabilize if hypotensive/ischemic

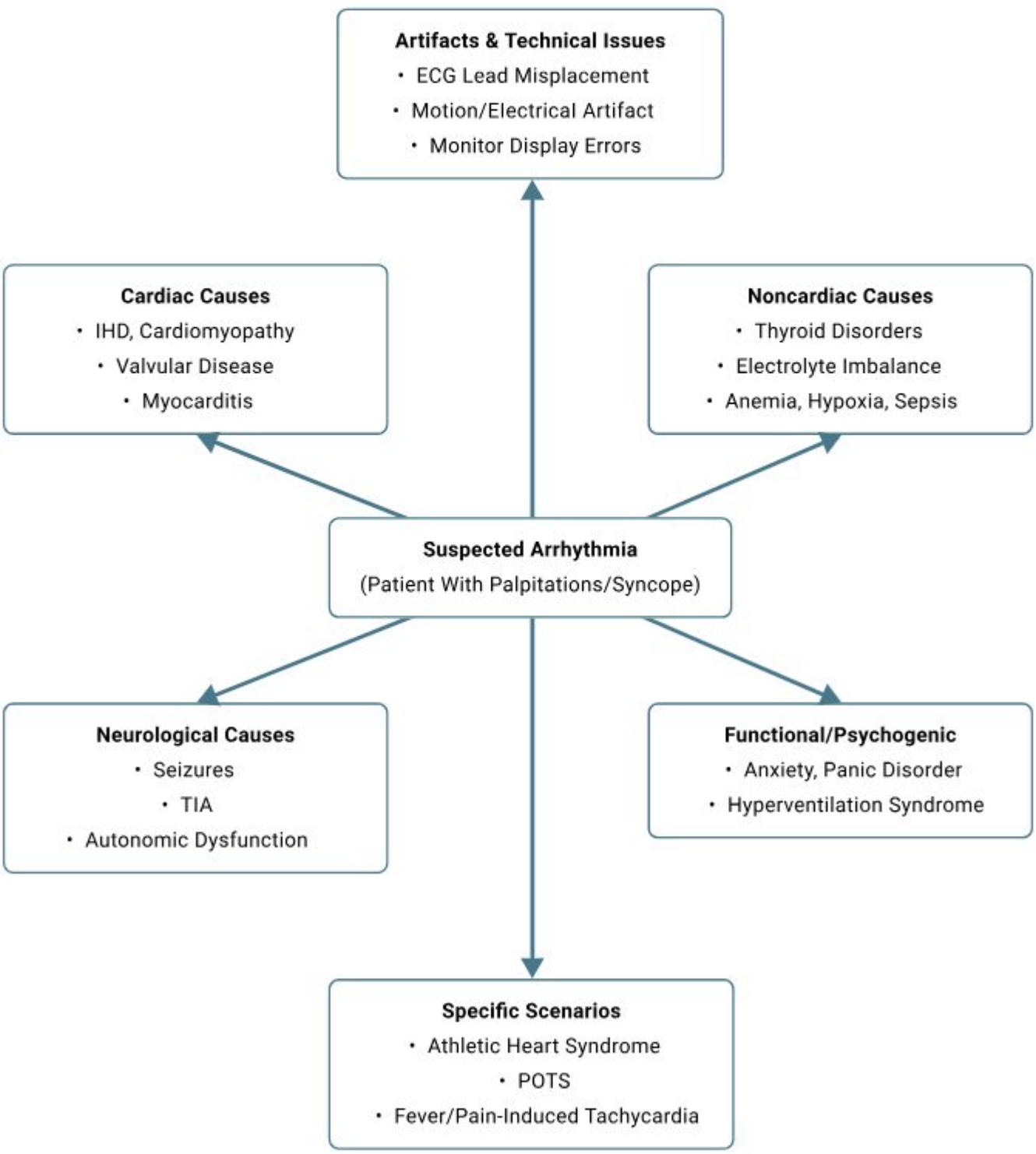
Class	Criteria	Examples	Action / Urgency
Duration			
Paroxysmal	Abrupt start/stop; usually <7 days	PSVT, paroxysmal AF	Document with ECG/monitor; consider ablation if recurrent
Persistent	>7 days; needs intervention to terminate	Persistent AF	Consider cardioversion and maintenance strategy
Permanent	Rhythm accepted; no longer pursuing sinus restoration	Permanent AF	Rate control, stroke prevention, risk-factor control
Severity			
Unstable (Emergency)	Any rhythm causing: SBP <90 mmHg, shock, acute pulmonary edema, ongoing ischemic chest pain, syncope	Any tachy- or bradyarrhythmia with these features	Immediate: synchronized cardioversion for unstable tachy; defibrillation for VF/pulseless VT; atropine/pacing for unstable brady
Potentially unstable	No current instability but high risk of deterioration	Sustained VT (stable), AF with RVR >150 bpm, high-grade AV block (asymptomatic)	Urgent: monitored setting, targeted therapy, prepare for escalation
Stable	No hemodynamic compromise; mild/none symptoms	Controlled AF, asymptomatic athlete's sinus brady, occasional PVCs/PACs without structural disease	Routine: observation, evaluation, risk-factor control, follow-up

DIAGNOSTIC FLOWCHART



DIFFERENTIAL DIAGNOSIS

Consider other conditions that can mimic or precipitate rhythm disturbances. Some non-cardiac and cardiac disorders present with similar symptoms such as palpitations, dizziness, syncope, or chest discomfort without an underlying primary arrhythmia.

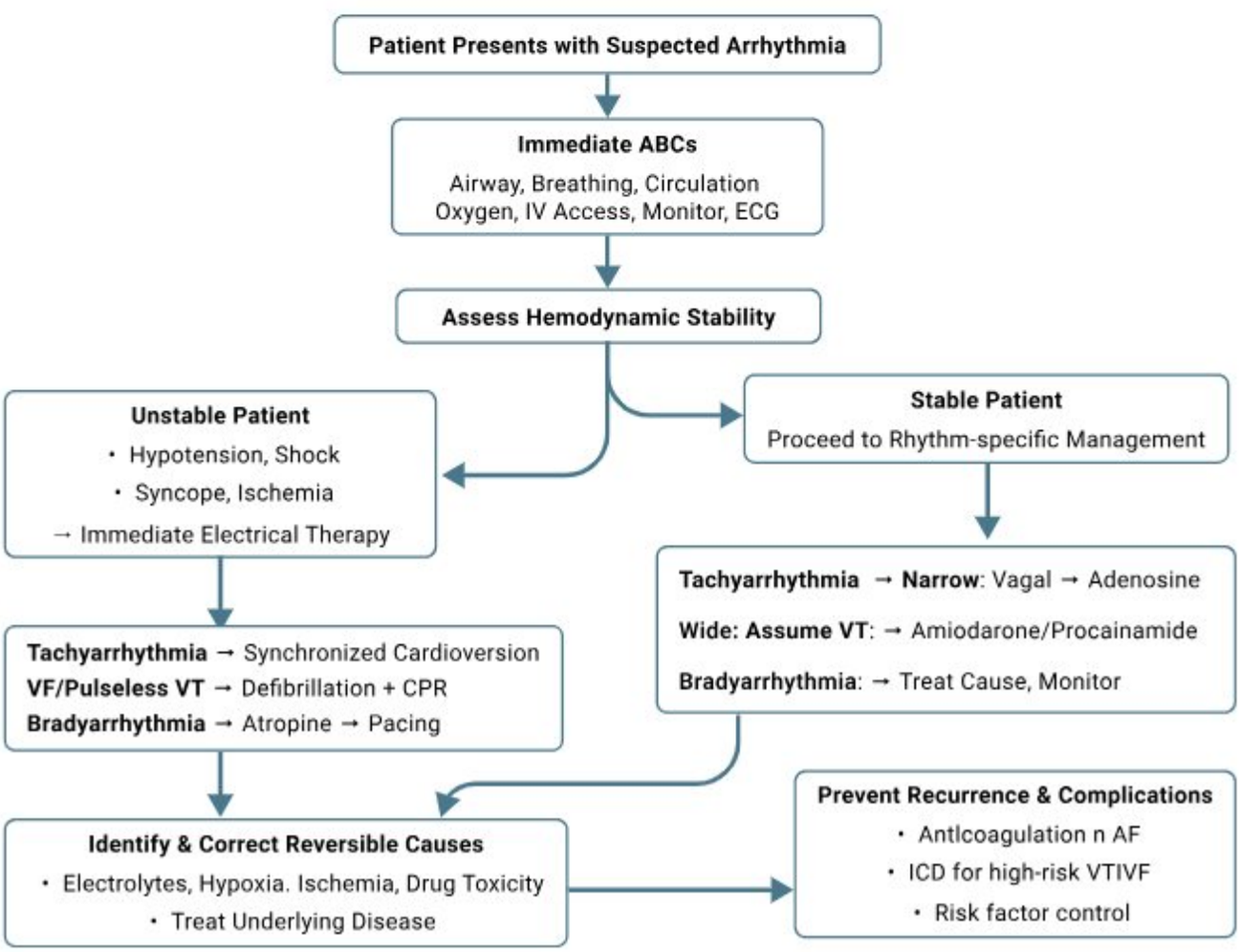


MANAGEMENT GOALS

Prevent death and disability by stabilizing the patient, relieving symptoms, restoring or controlling rhythm/rate to maintain cardiac output, and preventing thromboembolism. In AF, use anticoagulation when indicated to avoid stroke and avoid it when low risk to prevent bleeding. In ventricular tachyarrhythmias, prompt cardioversion/defibrillation saves lives.

Management Principles

- Recognize instability fast and act immediately.
- Match evidence-based therapy to rhythm type, hemodynamics, and comorbidities.
- Avoid harm: don't delay defibrillation in shockable arrest; don't use antiarrhythmics for benign ectopy.
- Escalate stepwise: vagal maneuvers and rate control/antiarrhythmics/ ablation/devices when indicated.
- Coordinate across care levels: stabilize early in primary/secondary care and prepare timely tertiary referral.



APPROACH TO MANAGEMENT OF PATIENTS PRESENTING WITH NO PULSE

Step-by-step approach for a patient with no pulse (adult) (AHA)

1. **Recognize arrest fast**
   - Unresponsive, no normal breathing, no definite pulse in ≤10 s - start CPR and activate code/EMS, bring defibrillator.
2. **Start high-quality CPR**
   - Compress at 100-120/min, depth 5-6 cm, full recoil, minimize pauses; switch compressor every 2 min. Give oxygen; use bag-mask with 30:2 until an advanced airway is in place, then 10 breaths/min.
3. **Attach monitor/defibrillator + IV/IO access**
   - Do not delay compressions for access.
4. **Rhythm check (≤10 s) - follow the correct branch**

   A. Shockable (VF/pVT)
   - Immediate shock (biphasic per device; if unknown, 200 J, escalate). Resume CPR immediately for 2 min; charge during compressions.
   - Give epinephrine 1 mg IV/IO every 3-5 min (start after the 2nd rhythm check).
   - After the 3rd shock, give amiodarone 300 mg IV bolus; may repeat 150 mg for refractory VF/pVT (lidocaine is an alternative). Consider magnesium 1-2 g for torsades.

   B. Non-shockable (PEA/asystole)
   - Resume CPR immediately for 2 min. Give epinephrine 1 mg IV/IO as soon as feasible, then every 3-5 min. Do **NOT** shock unless the rhythm becomes shockable.
5. **Manage airway and monitor effectiveness**
   - Use waveform capnography if available; avoid hyperventilation; continue uninterrupted compressions during airway placement if possible.

6. **Search and treat reversible causes (Hs & Ts) during CPR cycles**
   - Hypoxia, hypovolemia, hydrogen ion (acidosis), hypo/hyperkalemia (and other metabolic), hypothermia; tension pneumothorax, tamponade, toxins, thrombosis (PE/MI). Use bedside ultrasound if it doesn't interrupt CPR.
7. **Every 2 minutes**
   - Quick rhythm/pulse check (≤10 s), rotate compressors, deliver shock if indicated, give meds on schedule, and continue cycles. (cpr.heart.org)
8. **After ROSC - immediate post-arrest care**
   - Oxygenate (avoid hyperoxia), support BP (e.g., target MAP ≥65), obtain 12-lead ECG and treat the cause (e.g., coronary occlusion).
   - Temperature control: maintain a constant 32-37.5°C and continue for at least 24 h; prevent fever in comatose patients.
9. **If no ROSC**
   - Reassess rhythm, reversible causes, CPR quality, and consider appropriateness of continued efforts. Document events, timing, drugs, shocks.

Community Measures - Strengthen public CPR training, AED access in public places, and rapid EMS activation to improve survival.

APPROACH TO MANAGEMENT OF A PATIENT PRESENTING WITH TACHYARRHYTHMIA

1. Immediate Assessment

- Airway, Breathing, Circulation (ABC) - stabilize life-threatening issues first.
- Oxygen if hypoxic.
- IV access and cardiac monitoring.
- Obtain 12-lead ECG as early as possible.
- Check vital signs: BP, HR, SpO_2, temperature.

2. Determine Hemodynamic Stability

Signs of instability:

- Hypotension (SBP <90 mmHg)
- Shock (altered mental status, cold/clammy skin)
- Acute heart failure (pulmonary edema)
- Ongoing chest pain/ischemia
- Syncope or near-syncope

3. Identify QRS Width and Rhythm Regularity

- Narrow complex (<120 ms): Usually supraventricular origin.
- Wide complex (≥120 ms): Ventricular origin until proven otherwise.
- Regular vs Irregular rhythm - guides differential and drug choice.

4. Immediate Management Based on Stability

A. Unstable Patient

1. Immediate cardioversion (synchronized if QRS and T waves are clearly identified, otherwise defibrillation - as in VF)
    a. Narrow complex: 50-100 J biphasic, escalate as needed.
    b. Wide complex: 100 J biphasic, escalate as needed.
2. If regular, narrow-complex and available consider adenosine only if SVT is suspected and patient not in AF/flutter.
3. Treat the underlying cause in parallel.

B. Stable Patient

1. **Regular Narrow Complex (SVT)**
    1. Vagal maneuvers (Modified Valsalva, carotid sinus massage - if no carotid bruit).
    2. If ineffective: Adenosine 6 mg rapid IV push - flush and repeat with 12 mg if needed.
    3. Beta-blocker or non-DHP calcium channel blocker (diltiazem/verapamil) if persistent.

2. **Irregular Narrow Complex (Most often AF/flutter)**

    i. **AF with onset <48 hours:** Consider cardioversion after stroke risk assessment; anticoagulation if indicated.

    ii. **AF with onset >48 hours or unknown:** Rate control (beta-blocker, diltiazem, digoxin) + anticoagulation; defer cardioversion (till at least 4 weeks of anticoagulation) unless unstable.

3. **Regular Wide Complex**

    i. Assume Ventricular Tachycardia until proven otherwise.

    ii. Amiodarone 150 mg IV over 10 min then infusion or procainamide (20 to 50 mg/min until arrhythmia terminates).

    iii. If experts are available and diagnosis of SVT with aberrancy confirmed, can use adenosine.

4. **Irregular Wide Complex**

    i. Possible AF with pre-excitation (WPW) or polymorphic VT.

    ii. Avoid AV nodal blockers (beta-blockers, CCBs, digoxin) in pre-excitation.

    iii. Treat as VT: Amiodarone or procainamide; if torsades, administer IV magnesium.

5. **Concurrent Measures**

- Treat precipitating factors:

    a. Electrolyte correction (K^+, Mg^{2+})

    b. Oxygenation and ventilation

    c. Manage ischemia, sepsis, thyrotoxicosis

- Monitor for conversion complications (hypotension, embolic events).

6. **Post-Stabilization**

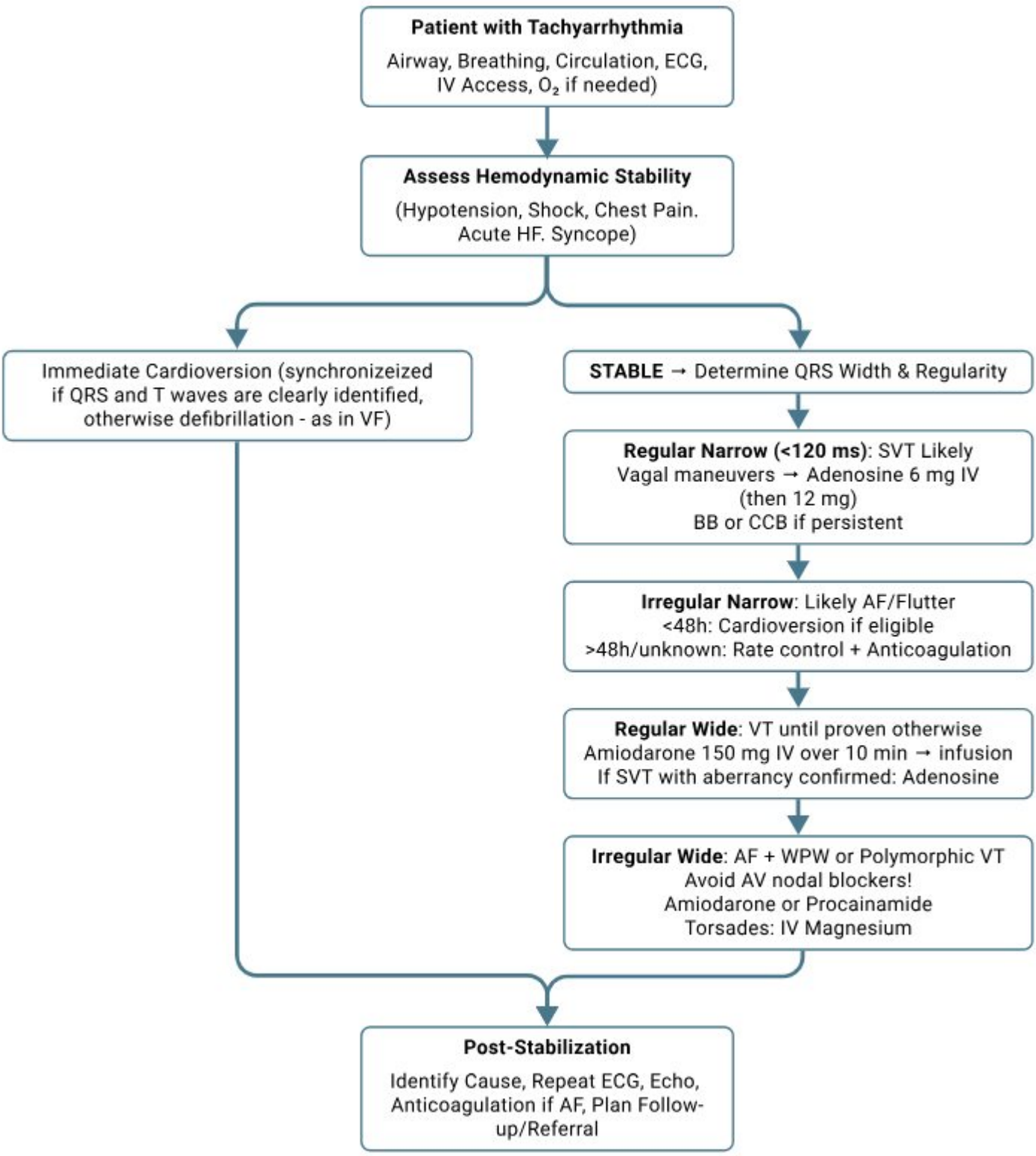
- Treat precipitating factors:

    i. Repeat ECG to document rhythm after conversion.

    ii. Echocardiography to assess for structural heart disease.

    iii. Anticoagulation decision for AF/flutter based on CHA_2DS_2-VASc score.

    iv. Arrange cardiology follow-up or referral for recurrent or high-risk arrhythmias.


Patient with Tachyarrhythmia
Airway, Breathing, Circulation, ECG, IV Access, O_2 if needed)
Assess Hemodynamic Stability
(Hypotension, Shock, Chest Pain. Acute HF. Syncope)
Immediate Cardioversion (synchronizeized if QRS and T waves are clearly identified, otherwise defibrillation - as in VF)
STABLE → Determine QRS Width & Regularity
Regular Narrow (<120 ms): SVT Likely
Vagal maneuvers → Adenosine 6 mg IV (then 12 mg)
BB or CCB if persistent
Irregular Narrow: Likely AF/Flutter
<48h: Cardioversion if eligible
>48h/unknown: Rate control + Anticoagulation
Regular Wide: VT until proven otherwise
Amiodarone 150 mg IV over 10 min → infusion
If SVT with aberrancy confirmed: Adenosine
Irregular Wide: AF + WPW or Polymorphic VT
Avoid AV nodal blockers!
Amiodarone or Procainamide
Torsades: IV Magnesium
Post-Stabilization
Identify Cause, Repeat ECG, Echo, Anticoagulation if AF, Plan Follow-up/Referral

PHARMACOLOGIC THERAPY

Tachyarrhythmias

Clinical features	ECG findings	First step	Next step / alternatives	Next step / alternatives	Key cautions
A. SVT / PSVT					
Sudden palpitations, chest discomfort, anxiety, lightheadedness; often abrupt onset/ offset; usually stable BP	Regular narrow-complex tachycardia 150-250 bpm; absent/ retrograde P waves; short RP (typical AVNRT)	Vagal maneuvers	Valsalva; carotid sinus massage	-	Avoid carotid massage in carotid stenosis or elderly
Persistent palpitations after vagal attempts	Same as above; adenosine may unmask flutter or AF	If vagal fails	Adenosine 6 mg IV rapid bolus, flush 20 mL; if no effect in 1-2 min administer 12 mg IV; may repeat 12 mg once	-	Avoid in asthma/ severe bronchospasm; transient asystole possible; contraindicated in 2°/3° AV block without pacemaker; single-episode use only
Ongoing regular narrow tachy after adenosine	Regular narrow-complex tachy; consider re-entry vs focal AT	If adenosine ineffective	Metoprolol 2.5-5 mg IV over 2 min, repeat to max 15 mg	Verapamil 5-10 mg IV over 2 min	Avoid non-DHP CCBs in WPW with AF, hypotension, or LV dysfunction
B. AF / Atrial flutter (stable, onset <48 h)					
Palpitations, dyspnea, fatigue; may have mild hypotension if RVR	AF: irregularly irregular R-R, no P waves, fibrillatory baseline. Flutter: saw-tooth F waves, often 2:1 block - ~150 bpm regular	Rate control	Metoprolol 2.5-5 mg IV q5 min to max 15 mg followed by PO 25-100 mg BID	Diltiazem 0.25 mg/kg IV over 2 min followed by infusion 5-15 mg/h	-
Symptomatic candidate for rhythm control	AF/flutter pattern as above	Rhythm control (eligible)	Amiodarone 150 mg IV over 10 min followed by 1 mg/min for 6 h then reduce to 0.5 mg/min	Electrical cardioversion per local protocol	-
B. AF / Atrial flutter (stable, >48 h or unknown)					
Similar symptoms; often less abrupt	AF/flutter as above; duration informs thromboembolic risk	Rate control + anticoagulation before rhythm control	Anticoagulation e.g., Apixaban 5 mg BID; or Warfarin with INR 2-3	Rhythm control after anticoagulation or TEE rule-out of thrombus	Avoid AV-nodal blockers in WPW with AF

C. Monomorphic VT (stable)					
Palpitations, chest discomfort, diaphoresis, presyncope; BP preserved	Regular wide-complex tachycardia >100 bpm; QRS ≥120 ms; AV dissociation, capture/ fusion beats, extreme axis, concordance	Antiarrhyth mic	Amiodarone 150 mg IV over 10 min; repeat if needed followed by 1 mg/min for 6 h then reduce to 0.5 mg/min; then PO 200 mg TID x1 week, taper	Procainamide 20-50 mg/min IV until suppressed, hypotension, QRS increased >50%, or total 17 mg/kg	Amiodarone: QT prolongation, thyroid/liver toxicity. Procainamide: avoid in prolonged QT or HF
D. VF / Pulseless VT					
Unresponsive, apneic or agonal, no pulse	VF: chaotic irregular waveform, no organized QRS. Pulseless VT: rapid wide complexes without pulse	Defibrillate + CPR	Immediate defibrillation; high-quality CPR	Epinephrine 1 mg IV/IO q3-5 min during CPR; Amiodarone 300 mg IV bolus then 150 mg if recurrent	Follow ACLS shock/med cycles; minimize pauses
E. Torsades de pointes					
Syncope, palpitations, intermittent dizziness; may deteriorate to VF	Polymorphic VT with QRS "twisting" around baseline; usually preceded by prolonged QT; often pause-dependent	Magnesium	Magnesium sulfate 2 g IV over 1-2 min (pulseless/ unstable) or over 10-15 min (perfusing); may repeat in 10-15 min	Correct K^+/ Ca^{2+}, stop QT-prolonging drugs; defibrillate if unstable	Monitor QT and BP; treat precipitating cause (electrolytes, drugs, brady)

Bradyarrhythmias

Clinical features	ECG findings	First step	Drug / action and dose	Next step / alternatives	Key cautions
A. Symptomatic sinus bradycardia					
Dizziness, fatigue, presyncope/ syncope, chest discomfort, dyspnea, hypotension, confusion	Sinus rhythm with HR <60 bpm; upright P before each QRS; possible sinus pauses	Atropine	0.5 mg IV every 3-5 min (max 3 mg)	If ineffective: Dopamine 2-10 mcg/kg/min IV infusion or Epinephrine 2-10 mcg/min IV infusion; prepare transcutaneous pacing	Atropine may worsen ischemia in acute MI; relative caution in narrow-angle glaucoma; often ineffective in transplanted hearts or high-grade blocks
B. AV block - First-degree or Mobitz I (Wenckebach)					
Often asymptomatic; may have lightheadedness or exertional fatigue	First-degree: PR >200 ms, all P conducted. Mobitz I: progressive PR lengthening then dropped QRS	Observation if stable	-	If symptomatic or hypotensive: treat as bradycardia (atropine/ dopamine/ epinephrin; consider pacing)	Avoid AV-nodal blockers (β-blockers, diltiazem, verapamil, digoxin) if causing/ worsening block

B. AV block - Mobitz II or complete (third-degree)					
Fatigue, presyncope/ syncope (Stokes-Adams), hypotension, exercise intolerance, heart failure signs	Mobitz II: constant PR with intermittent non-conducted P (dropped QRS). Complete heart block: AV dissociation; atrial and ventricular rates independent; junctional or ventricular escape rhythm (narrow or wide)	Atropine (may be ineffective)	0.5 mg IV every 3-5 min (max 3 mg)	Immediate temporary pacing if unstable or no response; consider dopamine/epi infusion while arranging pacing	High risk of asystole - do not delay pacing; avoid AV-nodal blockers
C. Sick sinus syndrome (sinus node dysfunction)					
Intermittent dizziness, fatigue, syncope; tachy-brady pattern; palpitations	Sinus brady, sinus pauses/ arrest, SA exit block; alternating AF/atrial flutter with long pauses after termination	Stabilize; remove triggers (stop negative chronotropes, correct electrolytes)	No routine acute drug therapy if stable	Temporary pacing for symptomatic brady while arranging permanent pacemaker	Avoid AV-nodal-blocking drugs unless necessary (may worsen pauses); evaluate for reversible causes (ischemia, drugs, hypothyroidism)

NON-PHARMACOLOGIC INTERVENTIONS

Non-pharmacologic measures play a vital role in long-term control of arrhythmias. They can be used alone in selected cases or alongside medications to improve outcomes, prevent recurrence, and reduce complications.

Long-term/Prevention

Intervention	Indications	Key action	Practical notes	Cautions
Permanent pacemaker	Symptomatic bradyarrhythmias: sick sinus syndrome, Mobitz II, complete heart block	Single/dual-chamber pacing to prevent brady-related syncope and HF	Program to patient's needs and comorbidities	Check for pacing-induced cardiomyopathy risk; avoid AV-nodal blockers if worsening block
Implantable cardioverter-defibrillator (ICD)	Survivors of SCA; patients at high risk of VT/VF	Detects and terminates malignant VT/VF via shock or ATP	Consider anti-tachy pacing programming to reduce shocks	Evaluate life expectancy, infection risk, and driving/ work implications
Catheter ablation	Recurrent SVT, AF, or VT not controlled or not tolerated on drugs	Eliminates arrhythmogenic focus/pathway	Often first-line for AVNRT/AVRT; consider AF ablation per risk/symptoms	Procedure risks: vascular injury, tamponade; AF ablation may need repeats
Lifestyle and trigger avoidance	Any patient with trigger-provoked arrhythmia or risk factors	Remove precipitants and optimize substrates	Limit caffeine/alcohol, avoid stimulants; stress management; treat HTN, thyroid disease, diabetes, OSA	Review medication list for QT-prolonging or AV-nodal-blocking drugs when inappropriate

ASSESSMENT OF RESPONSE

Phase	Clinical endpoints	ECG/monitoring endpoints	Labs/other checks	If off-target, do this
1) Immediate (acute phase)	Symptoms ease: palpitations, chest discomfort, dyspnea, presyncope resolve. Hemodynamics: SBP ≥90 mmHg, MAP ≥65 mmHg, warm perfusion, mental status normal, SpO_2 ≥94% (88-92% if COPD). Tolerates therapy (no severe hypotension, bronchospasm, sedation issues).	Rhythm corrected or controlled: conversion to sinus or rate control (AF goal HR <110 bpm at rest if stable). No malignant rhythms on monitor.	Point-of-care K^+/Mg^{2+}/Ca^{2+}, glucose. Consider troponin if ischemia suspected.	If unstable: shock (for VT/VF)/synchronized cardioversion (unstable tachy), pacing/atropine/ pressors (unstable brady). Correct electrolytes; stop offending/QT-prolonging drugs; adjust doses or switch agents.
2) Early post-intervention (first 24-48 h)	Stable vitals within targets; no recurrent chest pain, dyspnea, syncope.	Continuous telemetry: no recurrence or new arrhythmias; QTc <500 ms, QRS not progressively widening; AF/Flutter HR in target. Verify capture if paced.	Maintain K^+ 4.0-5.0 mmol/L, Mg^{2+} ≥0.8 mmol/L (≥2.0 mg/dL); Ca^{2+} normal. Drug levels if indicated (e.g., digoxin 0.5-0.9 ng/mL). Review renal/hepatic function and drug interactions.	Replete electrolytes; titrate/hold culprit drugs; reconsider rhythm vs rate strategy; start/ optimize anticoagulation when indicated; arrange EPS/ablation or device if recurrent events.
3) Medium-term follow-up (first weeks)	Symptom diary shows no or rare episodes; functional recovery (normal activity without limitation).	12-lead ECG and ambulatory monitor (Holter/patch) to assess rhythm control and silent arrhythmias; device interrogation if applicable.	Check for drug tolerance/adverse effects: bradycardia, hypotension, QT prolongation. If on amiodarone or similar, baseline/interval TSH, LFTs; if on DOAC/ warfarin, adherence/INR (2-3).	Adjust meds (dose/ formulation/class); address adherence; treat triggers (sleep apnea, thyroid, alcohol/caffeine). If recurrence or intolerance then consider ablation, pacemaker, or ICD per indication.
4) Long-term evaluation	No strokes/TIA, no HF exacerbations, no cardiac arrest; acceptable quality of life and exercise capacity.	Sustained rate/rhythm targets (e.g., AF burden low/ acceptable); appropriate ICD therapies (if present) without frequent shocks.	Ongoing risk-factor control (BP, diabetes, lipids, weight, OSA). Periodic labs per therapy (e.g., thyroid/liver for amiodarone). Anticoagulation on board when indicated.	Reassess strategy: de-escalate if stable, or escalate to non-pharmacologic options (ablation/ device). Re-stratify stroke/sudden death risk; update anticoagulation and device programming as needed.

FOLLOW-UP

Follow-up ensures ongoing rhythm stability, early detection of recurrence, and prevention of complications. The approach depends on the patient's clinical status and response to therapy.

Indications	Actions
Step-Up in Care	
Persistent hemodynamic instability; recurrent symptomatic arrhythmias despite optimal medical therapy; new high-grade AV block; refractory ventricular arrhythmias	■ Urgent referral to higher-level facility with electrophysiology expertise ■ Prepare for advanced interventions: catheter ablation, device implantation (pacemaker, ICD), or surgical evaluation ■ Stabilize during transfer: oxygen, anti-arrhythmic infusion, continuous monitoring
Step-Down in Care	
Stable sinus rhythm or well-controlled ventricular rate on minimal/no anti-arrhythmic therapy; no recent symptomatic episodes; comorbidities stable	■ Transition to periodic follow-up at primary/secondary care ■ Continue patient education: symptom recognition, adherence, trigger avoidance ■ Schedule regular ECG checks and review anticoagulation (if applicable)

PROGNOSIS

Condition	Context	Prognosis	Key predictors of outcome	What improves/ worsens it	Follow-up / secondary prevention
Benign ectopy (PACs/PVCs)	Occasional premature beats in a structurally normal heart	Excellent; often no treatment needed	Structural heart disease (absent = good), PVC burden (<10-15% = low risk), symptom burden	Worse: stimulants, anxiety; Better: trigger control, reassurance	Reassure; address triggers; no routine therapy if asymptomatic
SVTs (e.g., PSVT)	Re-entry tachycardias without structural disease	Very good; ablation often curative	Structural heart disease, episode frequency/ duration, response to adenosine/vagal maneuvers, WPW/accessory pathway features	Recurrent episodes ↓ QoL; ablation markedly improves outlook	Consider ablation if recurrent; teach vagal maneuvers

Atrial fibrillation (AF) with good control	Controlled rate/ rhythm; risks treated	Good if anticoagulated and rates controlled	CHA_2DS_2-VASc (stroke risk), HAS-BLED (bleeding risk), LA size, LV function, AF burden/duration, OSA/obesity	Worse: poor anticoagulation, uncontrolled rate, untreated OSA/HTN; Better: risk-factor control	Anticoagulate per CHA_2DS_2-VASc; control rate/rhythm; manage OSA/ HTN; periodic ECG/echo
AF untreated/poorly controlled	RVR; no anticoagulation	High risk of stroke and HF	Same as above + persistent tachycardia (tachy-cardiomyopathy), low adherence	Worse: no AC when indicated, persistent RVR; Better: start AC, rate/rhythm strategy	Start AC when indicated; control rate/ rhythm; tighten comorbidity control
Ventricular tachyarrhythmias / VF	Ischemic scar, cardiomyopathy, channelopathies	High immediate mortality without rapid defib; ongoing risk after survival	LVEF (low = worse), time to defibrillation, ischemia/scar burden, recurrent VT, syncope, genetics (LQTS/ Brugada/CPVT)	Worse: delayed shock, severe LV dysfunction; Better: revascularization, GDMT, ICD	Etiology workup; revascularize if needed; ICD for secondary prevention; optimize HF GDMT
Bradyarrhythmias (high-grade AV block, sick sinus)	Degenerative conduction disease, ischemia, drugs	Excellent after pacing; fatal if untreated	Level of block (infra-His worse), escape rhythm rate/QRS width, syncope, reversible causes	Worse: drug toxicity, long pauses; Better: prompt pacing, remove triggers	Remove reversibles; permanent pacemaker when indicated; device checks
Reversible-cause arrhythmias	Electrolytes, ischemia, drug toxicity, thyroid	Good once corrected	Speed/ completeness of correction, QTc normalization, absence of structural disease	Worse: ongoing triggers, recurrent electrolyte shifts; Better: durable correction	Maintain K^+/ Mg^{2+}; review meds/QT; treat thyroid/ ischemia; education
Long-term determinants (all arrhythmias)	Dependent on adherence and risk control	Strongly modifiable	Medication adherence, BP/ DM/lipid control, weight/OSA treatment, alcohol/caffeine, smoking, follow-up reliability	Worse: poor adherence, uncontrolled risks; Better: structured care plans, rehab, reminders	Education, lifestyle change, regular ECG/ anticoagulation review, consider ablation/devices when indicated

Referral

Referral decisions should be guided by the type of arrhythmia, hemodynamic status, available resources, and the potential need for advanced interventions. Early and appropriate referral improves survival and reduces complications.

Care level	Core role	Diagnostics	Immediate/ initial therapy	When to refer upward	Planned/early referrals	Step-down/ communication
Primary care	Recognize arrhythmia; triage stability	Focused history/ exam; 12-lead ECG; pulse oximetry	If unstable: oxygen, IV access, emergency drugs; defibrillate if VF/pulseless VT and device available; treat reversible causes (electrolytes, hypoxia, drug	Urgent: hemodynamic instability (hypotension, shock, altered consciousnes s); ventricular arrhythmia; high-grade AV block; unstable SVT; suspected ACS with arrhythmia	Planned: stable arrhythmia needing Holter/echo; suspected genetic syndromes; symptomatic bradycardia for pacing review	Send ECG and treatment given; arrange monitored transfer; give clear referral note and contact details
Secondary care	Definitive evaluation and initiation of therapy	Continu ous ECG/ telemetry; echocardi ography; cardiac biomarke rs; targeted labs; basic EP tests if available	Start antiarrhythmi cs; anticoagulat ion in AF when indicated; correct electrolytes; temporary pacing if required	Immediate: refractory VT/ VF; recurrent unstable arrhythmias despite treatment; complex arrhythmia needing ablation/ device; severe brady unresponsive to drugs/ temporary pacing	Early: AF/ flutter with high stroke risk needing specialized rhythm control; suspected structural heart disease needing advanced imaging/ intervention	Provide stabilization summary, drug list, and monitoring data to tertiary; outline goals for return/ step-down follow-up
Tertiary care	Advanced arrhythmia management	Advanced imaging; EP study/ mapping	Catheter ablation (SVT/ AF/VT); device therapy (pacemaker/ ICD/CRT); advanced HF care if arrhythmia-related; genetic evaluation for inherited syndromes	-	Receives stabilized patients from lower levels; may schedule staged procedures	Issue detailed plan for step-down care, anticoagulatio n/device checks, and triggers for re-referral; coordinate follow-up intervals

COMPLICATIONS

Arrhythmias can lead to a broad spectrum of complications, ranging from mild symptoms to life-threatening events. The nature and severity of complications depend on the type of arrhythmia, duration, underlying cardiac status, and timeliness of treatment.

Examples	Mechanism / why it happens	Higher-risk scenarios	Prevention / mitigation	Escalate if Red flags
Hemodynamic (Immediate)				
Syncope, presyncope	Transient cerebral hypoperfusion from very slow/ fast rhythms	High-grade AV block, VT, rapid AF/flutter, elderly, autonomic dysfunction	Treat rhythm promptly; fluids/pressors if needed; pacing for brady; cardioversion for unstable tachy	Recurrent syncope, injury with syncope, exertional syncope, new neurologic deficit
Hypotension, cardiogenic shock	Inadequate stroke volume/CO during sustained VT/VF or rapid AF in weak LV	LV dysfunction, acute MI, sepsis, anesthesia	Immediate ACLS; defibrillate VF/ pVT; synchronized cardioversion for unstable tachy; optimize preload/ afterload	SBP <90, cold/ clammy skin, altered mentation, rising lactate
Low cardiac output syndrome	Persistent tachy/brady impairs filling or rate-dependent output	Long-standing tachyarrhythmia, SSS, advanced HF	Rate/rhythm control; pacing if indicated; GDMT for HF	Worsening fatigue, oliguria, rising BNP
Thromboembolic (Intermediate)				
Ischemic stroke	Atrial stasis - thrombus in AF/ flutter	CHA_2DS_2-VASc high, prior stroke/TIA, LV dysfunction	Anticoagulation per risk; TEE before cardioversion if >48 h/unknown AF duration	Focal neuro signs, aphasia, acute vision loss
Systemic embolism	Emboli to limb/organ arteries	AF/flutter, LV thrombus, cardiomyopathy	Anticoagulati on; treat LV thrombus; manage AF burden	Acute limb pain/pallor, mesenteric ischemia signs

Examples	Mechanism / why it happens	Higher-risk scenarios	Action / Prevention / mitigation	Escalate if Red flags
HF & structural (Long term)				
Tachycardia-induced cardiomyopathy	Chronic RVR depresses LV function	Persistent AF/AT, inappropriate sinus tachy	Achieve durable rate/rhythm control; consider ablation	Declining EF, worsening NYHA class
HF decompensation	Brief rapid AF/VT tips fragile patients into HF	Pre-existing HF, renal dysfunction, infection	Early rate control/ diuresis; trigger management	Pulmonary edema, escalating diuretics, hypoxia
Atrial remodeling	Long AF - structural/ electrical change - AF persistence	Delayed rhythm control, obesity/OSA, HTN	Early rhythm strategy where appropriate; risk-factor control	AF becomes persistent despite therapy
Sudden cardiac death				
Ventricular fibrillation	Terminal rhythm in ischemia/structural disease	Prior MI/scar, low LVEF, channelopathies	Rapid defibrillation; ICD for secondary prevention; revascularize	Cardiac arrest, recurrent VT/VF shocks
Asystole	Advanced AV block or prolonged sinus arrest	Degenerative conduction disease, drug toxicity	Prompt pacing; remove causative drugs	Prolonged pauses, syncope with injury
Management-related				
Drug: TdP, brady, organ toxicity (e.g., amiodarone lung/liver/thyroid)	QT prolongation, AV-nodal blockade, cumulative toxicity	Class Ia/III agents, polypharmacy, renal/ hepatic impairment	Check QTc, K^+/ Mg^{2+}; med reconciliation; periodic LFT/TSH	QTc ≥500 ms, syncope on QT drugs, new dyspnea/cough on amiodarone
Device: lead fracture, infection, inappropriate ICD shocks	Hardware failure or oversensing; pocket/ lead infection	Diabetes, CKD, reinterventions	Asepsis; remote/ device checks; optimize programming (ATP)	Fever, pocket erythema, recurrent shocks
Procedural: vascular injury, tamponade, thromboembolism (ablation)	Catheter manipulation/ anticoagulation issues	Left-sided ablation, anticoagulation gaps	Periprocedural anticoagulation; ultrasound-guided access; echo if hypotensive	Sudden hypotension, pericardial effusion signs
Psychological / QoL				
Anxiety, depression; activity limitation	Fear of recurrence/ shocks; symptom unpredictability	Recurrent symptomatic episodes, ICD shocks	Education, reassurance, CBT referral, cardiac rehab, shared plans	Suicidal ideation, severe avoidance impacting ADLs

PREVENTION & HEALTH PROMOTION

Prevention of arrhythmias focuses on addressing modifiable risk factors, early detection of underlying conditions, and public awareness. A structured prevention approach integrates lifestyle modification, management of comorbidities, and population-level screening. Key strategies include controlling hypertension, diabetes, and dyslipidemia, promoting regular physical activity, maintaining a healthy weight, reducing dietary salt and unhealthy fats, and limiting alcohol, caffeine, and stimulant use. Smoking cessation and stress management are vital components.

Health promotion also involves strengthening primary care systems to provide routine blood pressure, glucose, and lipid checks, ensuring timely diagnosis and treatment of ischemic heart disease, thyroid disorders, and other systemic causes. Patient education on recognizing warning signs such as palpitations, dizziness, or syncope and seeking urgent care can reduce complications. Training healthcare workers in arrhythmia recognition and initial management is essential, especially in rural and island communities.

In the Maldives Action Plan Framework for Non-Communicable Diseases (NCDs), prevention of cardiovascular diseases, including arrhythmias, is addressed through integrated strategies:

1. Population-wide risk reduction - implementing policies to reduce trans fats, salt, and sugar in processed foods; promoting active lifestyles through community programs; and running mass media campaigns on heart health.
2. High-risk individual interventions - establishing screening protocols at atoll and island health centres for cardiovascular risk factors, ECG access for high-risk patients, and standardized referral pathways to secondary/tertiary care.
3. Health system strengthening - improving diagnostic infrastructure, ensuring availability of essential medicines for arrhythmia and NCD control, and maintaining emergency readiness for acute events.
4. Monitoring and evaluation - integrating arrhythmia-related outcomes into the national NCD surveillance system to track trends and guide interventions.

This combined approach linking individual-level prevention, community engagement, and system-level readiness ensures sustainable reduction in arrhythmia burden while aligning with Maldives' broader NCD targets for 2025 and beyond.

PATIENT EDUCATION

Patient education is central to the effective management of arrhythmias, reducing recurrence risk, and preventing complications. The objectives should empower patients and caregivers with the knowledge, skills, and motivation to participate actively in their care.

Topic	What to know	Do	Don't
Understand your arrhythmia	Know the type (tachy or brady), how it affects the heart, and risks (stroke, heart failure, sudden death).	Keep a simple summary of your diagnosis and latest ECG. Learn your target heart rate and goals.	Assume it's harmless or "just stress."
Warning signs	Palpitations, dizziness, chest discomfort, breathlessness, sudden fatigue, fainting.	Note start time, triggers, and duration of symptoms.	Ignore repeated or worsening episodes.
When to seek urgent care	Severe chest pain, fainting, severe shortness of breath, fast or very slow pulse that doesn't settle.	Call emergency services; keep numbers handy; tell caregivers your plan.	Drive yourself if unstable or delay calling for help.
Medicines	Drugs control rate/rhythm and prevent clots; side effects can occur.	Take exactly as prescribed; use reminders; report dizziness, severe fatigue, rash, wheeze, or bleeding.	Skip doses, double up after a miss, or stop on your own.
Anticoagulation (AF patients)	Lowers stroke risk; warfarin needs INR checks; DOACs need strict timing.	Take at the same time daily; keep INR appointments if on warfarin; watch for bleeding or black stools.	Take NSAIDs or herbal blood thinners without advice; miss INR checks.
Over-the-counter and herbal products	Some raise heart rate or prolong QT.	Check with your clinician or pharmacist before use.	Use decongestants, stimulants, or new supplements without review.
Lifestyle	Healthy weight, diet, sleep, and hydration reduce events.	Eat low salt, low trans/ saturated fat; limit sugar; drink water; sleep 7-8 h; manage stress.	Binge alcohol; high caffeine/ energy drinks; smoke; use stimulants.
Physical activity	Regular, moderate exercise is helpful for most.	Follow a plan cleared by your clinician; warm up and cool down.	Do extreme exertion without clearance or exercise when unwell/dehydrated.
Triggers	Alcohol binges, high caffeine, stress, poor sleep, some foods/ drugs can trigger episodes.	Keep a symptom/trigger diary; avoid personal triggers.	Assume triggers are the same for everyone or "test" them repeatedly.
Home monitoring	Pulse checks and home devices can guide care.	Learn to check pulse; record heart rate, rhythm alerts, and symptoms.	Panic over isolated extra beats if you feel well.

Caregiver readiness	Family can help in emergencies.	Teach basic CPR awareness and your emergency plan; share medication/allergy list.	Keep plans private or outdated.
Follow-up	Regular reviews prevent complications.	Attend all appointments; bring logs and medication list.	Skip visits because you feel fine.
Travel	Preparation reduces risk.	Carry updated records, meds in hand luggage, and emergency contacts.	Travel without supplies or information.
Devices (pacemaker/ICD)	Devices need checks and simple precautions.	Attend routine device checks; carry your device card; ask before MRI.	Keep strong magnets or security wands close to the device; miss follow-ups.

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