# TACHYCARDIA

## Significance, Safety, and Continuing Care

**Audience:** RN/APRN | **Contact hrs:** 1.5 | **Pharmacology hrs:** 0.0

**Expected revision:** October 5, 2028

Pharmacology hours are included in the total contact hours.

### The rate is a finding—not the diagnosis

A fast pulse may be the body's attempt to maintain circulation, the first sign of an acute illness, or the rhythm disturbance that is causing deterioration. Those possibilities require different responses. A patient who is bleeding may depend on a faster sinus rate to support cardiac output; another with the same rate may have ventricular tachycardia (VT) and need urgent rhythm treatment. The practical question is not simply whether the rate exceeds a number. It is whether the rate fits the situation, what rhythm is present, and whether perfusion is threatened (American Heart Association [AHA], 2025a; Brugada et al., 2020).

In adults, tachycardia conventionally means a heart rate above 100 beats/min at rest. That definition identifies a finding, not its danger or treatment. Children require age-appropriate assessment. A rate below 150 beats/min does not exclude clinically important ventricular tachycardia, and a rate above 150 does not establish its mechanism. Symptoms, blood pressure, mental status, ventricular function, onset pattern, and rhythm evidence must be considered together (AHA, 2025a, 2025c; Zeppenfeld et al., 2022).

#### Purpose, audience, and learning outcomes

This activity prepares registered nurses and advanced practice registered nurses to connect tachycardia mechanisms with assessment, escalation, medication safety, and continuing care. Adult emergency examples are identified explicitly. Pediatric, pregnancy, congenital-heart-disease, and inherited-arrhythmia considerations are included, but this article does not replace population-specific protocols. RNs assess, monitor, administer authorized treatment, educate, and escalate within local policy. Diagnostic, prescribing, procedural, and device-programming decisions require appropriately authorized clinicians.

After completing this activity, learners should be able to: distinguish compensatory sinus tachycardia from primary tachyarrhythmia; explain automaticity, triggered activity, reentry, and remodeling; prioritize assessment using perfusion, regularity, and QRS width; select appropriate medication-safety and escalation checks; explain the complementary roles of mapping, ablation, and implantable defibrillators; and develop follow-up priorities for arrhythmia-induced cardiomyopathy and recurrent symptoms.

Five fictional cases illustrate the reasoning behind a next action. They are not standing orders. The strongest answer in practice often starts with a short, explicit statement of uncertainty: “This is a new wide-complex tachycardia with worsening perfusion; the mechanism is not yet confirmed.” Naming both the finding and the risk is more useful than attaching a confident rhythm label to incomplete information.

### Why tachycardia starts—and persists

#### Automaticity and the coupled pacemaker system

The sinoatrial node normally initiates each heartbeat through spontaneous diastolic depolarization. Membrane ion currents and intracellular calcium cycling interact rather than operate as independent clocks. Hyperpolarization-activated cyclic-nucleotide-gated channels, predominantly HCN4 in the sinoatrial node, contribute the mixed sodium/potassium “funny” current. Calcium release from the sarcoplasmic reticulum, calcium reuptake, and sodium–calcium exchange also influence the approach to the next action potential. Autonomic signaling adjusts this coupled system, allowing an appropriate increase during activity and a decrease during recovery (Maltsev & Lakatta, 2009; Sheldon et al., 2015).

A useful clinical distinction follows: acceleration of the normal pacemaker is not the same as a new circuit. Sinus tachycardia commonly accelerates and decelerates with the provoking condition. An automatic atrial focus may also warm up and cool down, whereas an atrioventricular reentrant rhythm often switches on abruptly. Onset behavior offers a clue, but does not establish a diagnosis without the electrocardiogram (ECG) and clinical context. Figure 1 links the major

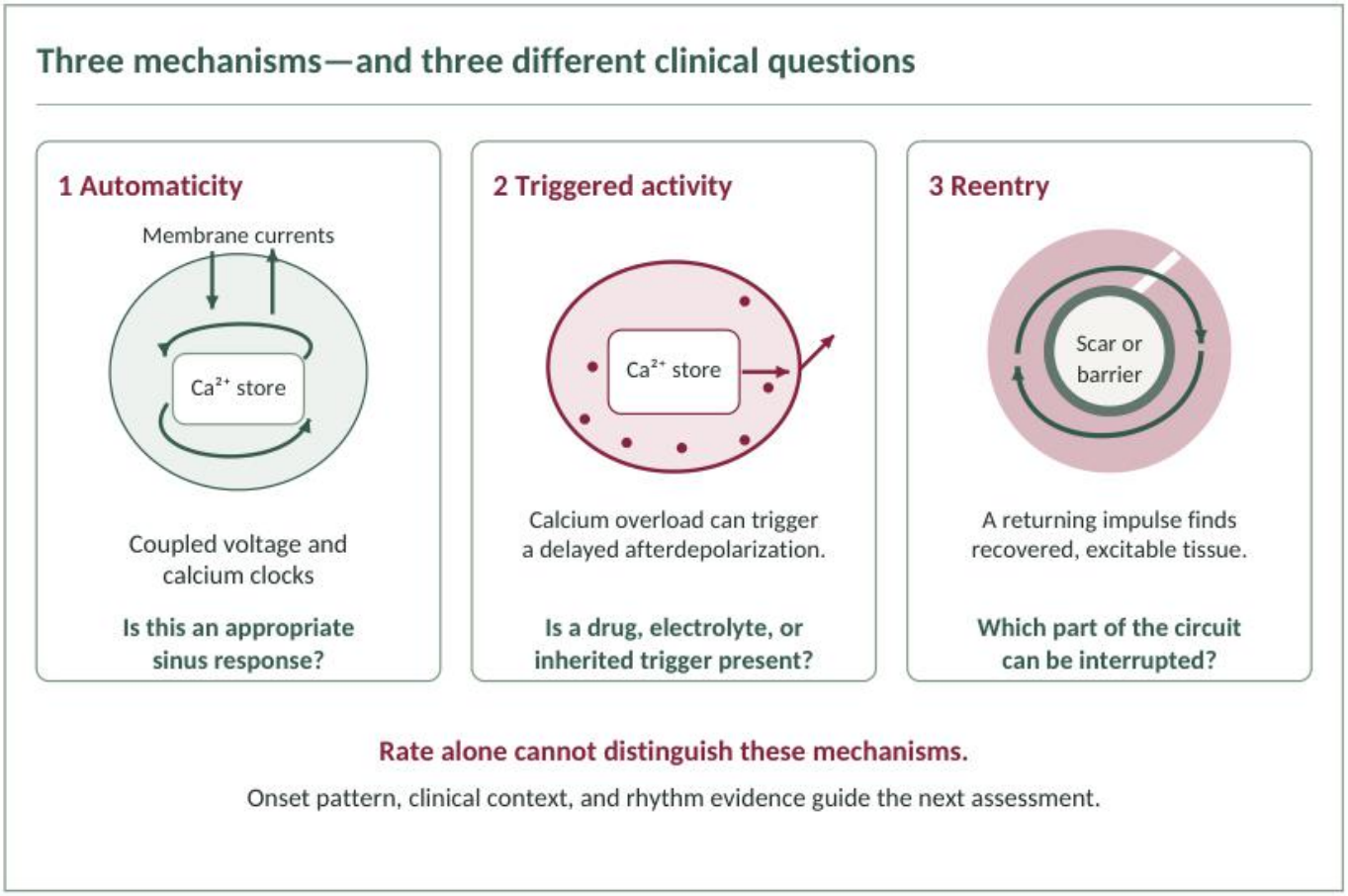


Figure 1. Automaticity, triggered activity, and reentry require different clinical reasoning. The calcium-overload panel illustrates delayed afterdepolarization; early afterdepolarizations occur before repolarization ends. Original cellular and circuit schematics, not diagnostic recordings. Sources: Maltsev and Lakatta (2009), Brugada et al. (2020), and Zeppenfeld et al. (2022).

mechanisms to their practical meaning (Brugada et al., 2020).

### Triggered activity and reentry

Triggered activity depends on a preceding action potential. Early afterdepolarizations occur before repolarization has finished; delayed afterdepolarizations occur after repolarization and are associated with abnormal intracellular calcium handling. These mechanisms help explain why prolonged repolarization, catecholamine excess, and selected toxic or inherited states can produce dangerous rhythms. Correcting an electrolyte or medication trigger is therefore a treatment of the mechanism, not merely a laboratory housekeeping task (Zeppenfeld et al., 2022).

Reentry requires an impulse to return to tissue that has regained excitability. Differences in conduction and refractoriness can allow a premature impulse to block in one direction, travel slowly through another route, and reenter recovered tissue. In atrioventricular nodal reentrant tachycardia (AVNRT), functionally different nodal pathways support the circuit. In atrioventricular reentrant tachycardia (AVRT), an accessory connection joins the atria and ventricles outside the usual conduction route. In scar-related ventricular tachycardia, surviving muscle channels within or around scar can support repeated circulation of activation (Brugada et al., 2020; Cronin et al., 2020).

Fast sodium current supports rapid conduction in working myocardium and the His–Purkinje system, whereas nodal conduction relies strongly on calcium-dependent processes. Gap junctions couple neighboring cells. Fibrosis, ischemia, altered channel function, and nonuniform coupling can slow or fragment conduction. This is why the same rate has different implications in an otherwise healthy heart and in a ventricle containing an infarct scar. It also explains why a drug that slows conduction can either interrupt an arrhythmia or facilitate another one (Brugada et al., 2020; Zeppenfeld et al., 2022).

### Electrical, structural, and autonomic remodeling

Atrial fibrillation (AF) illustrates a self-reinforcing process. Rapid activation can alter calcium handling and refractoriness; stretch and fibrosis can create heterogeneous conduction; autonomic influences can

change triggers and substrate. The classic experimental observation that atrial fibrillation promotes its own persistence supports the distinction between stopping an episode and addressing the conditions that sustain recurrence. Obesity, hypertension, sleep-disordered breathing, alcohol exposure, and heart failure deserve attention alongside rhythm-directed treatment (Wijffels et al., 1995; Joglar et al., 2024).

Molecular findings involving ryanodine receptors, connexins, microRNAs, and profibrotic signaling are biologically informative. They do not make gene therapy, microRNA treatment, or autonomic denervation routine therapy for a patient with tachycardia. Similarly, a suspected channelopathy requires phenotype-directed specialist evaluation and genetic counseling—not an assumption that one gene explains every episode. Exertional syncope, unexplained sudden death in relatives, or characteristic baseline ECG abnormalities should lower the threshold for referral (Zeppenfeld et al., 2022).

## Find the driver before suppressing the response

The differential begins with context. Exercise, pain, fever, emotional stress, and pregnancy can increase sinus rate. Pathologic drivers include hypoxemia, infection, volume depletion, hemorrhage, anemia, pulmonary embolism, myocardial ischemia, heart failure, thyroid excess, hypoglycemia, and drug effects or withdrawal. Several may coexist. A postoperative patient can be in pain and also be bleeding; labeling the rate "pain-related" does not exclude the second problem (Brugada et al., 2020; De Backer et al., 2025).

Compare the rate with the patient's baseline and its trajectory. Ask when symptoms began, whether onset was abrupt or gradual, whether episodes end suddenly, and what activity or exposure preceded them. Review new prescriptions, missed doses, inhaled beta agonists, decongestants, stimulants, energy products, recreational substances, and recent changes in diuretics or thyroid replacement. The relevant history is what was actually taken, not simply what remains on the medication list (Brugada et al., 2020; Sheldon et al., 2015).

Assessment should link observations to plausible mechanisms. Temperature, oxygen saturation, respiratory effort, volume status, pain, bleeding, glucose, and perfusion help direct further testing. A complete blood count, electrolytes, magnesium, renal function, and thyroid studies may be appropriate, depending on presentation. Troponin testing and investigation for pulmonary embolism should answer a clinical question rather than follow automatically from every elevated rate. A tachycardia-related troponin increase alone does not establish a coronary occlusion (Brugada et al., 2020; Zeppenfeld et al., 2022).

**CASE 1 · A faster pulse supporting a failing circulation**

A 68-year-old patient with vomiting and fever has a sinus rhythm at 132 beats/min, blood pressure 88/54 mmHg, delayed capillary refill, and new confusion. The rate varies slightly with movement; a P wave precedes each QRS. A colleague asks whether an intravenous rate-slowing drug would improve the blood pressure.

The immediate problem is shock with an apparent compensatory sinus response, not proven primary tachyarrhythmia. Activate the appropriate deterioration pathway, support oxygenation when needed, assess infection and fluid losses, obtain authorized investigations, and implement ordered resuscitation with repeated assessment. A negative chronotropic drug can remove compensation without treating the cause.

The handoff should state the pressure, mental-status change, rhythm, suspected losses, and response to each intervention. Improvement is judged by perfusion and the underlying illness, not by achieving a cosmetically normal monitor number. Persistent or changing tachycardia after initial treatment requires reassessment rather than repeated attribution to the original explanation (AHA, 2025a; Brugada et al., 2020).

## Persistent sinus tachycardia is a diagnosis to investigate

Inappropriate sinus tachycardia is considered only after secondary causes are excluded; the Heart Rhythm Society (HRS) definition includes resting sinus rate above 100 beats/min with a mean 24-hour rate above 90 and distressing symptoms. Postural tachycardia syndrome is different: symptoms occur with standing, with an excessive upright rate rise and without orthostatic hypotension. Adult and adolescent thresholds differ. A single standing pulse or an anxious appearance cannot establish either diagnosis. Document posture, timing, symptoms, blood pressure, medications,

and reproducibility, then arrange appropriate evaluation (Sheldon et al., 2015).

## A structured rhythm assessment

### Start with the patient, then the tracing

Confirm a pulse and check that the monitor represents the patient rather than motion, poor electrode contact, or electrical interference. Compare a palpable pulse or reliable plethysmographic waveform with the displayed rhythm while maintaining observation. Artifact and true arrhythmia can coexist; an apparently well patient still needs a prompt assessment rather than dismissal of the alarm. Conversely, an unresponsive person without a definite pulse requires the cardiac-arrest pathway, not a prolonged search for a perfect ECG (AHA, 2025a).

Obtain a 12-lead ECG during symptoms whenever feasible without delaying urgent treatment. Preserve the onset strip, treatment response, and post-conversion ECG. Compare with a prior baseline for bundle-branch block, pre-excitation, Q waves, and QT findings. Ambulatory monitoring is useful when episodes escape capture, but the device should match event frequency. A short recording that misses infrequent symptoms is not proof that no arrhythmia occurred. Patient-triggered recordings help correlate symptoms with rhythm; consumer algorithms still require clinical confirmation (Brugada et al., 2020; Joglar et al., 2024).

### Use regularity and QRS duration to organize uncertainty

In the adult framework, a narrow QRS is less than 120 ms and a wide QRS is 120 ms or longer. These categories describe ventricular activation, not the location of every rhythm's origin. A supraventricular rhythm may be wide because of bundle-branch aberration or pre-excitation. A stable blood pressure does not prove a wide-complex rhythm is supraventricular. When uncertain, a sustained wide-complex tachycardia should be managed as potentially ventricular while expert assessment proceeds (AHA, 2025a; Brugada et al., 2020).

Regular narrow-complex supraventricular tachycardia (SVT) includes sinus tachycardia, atrial tachycardia, atrial flutter with fixed conduction, AVNRT, and orthodromic AVRT. A ventricular rate near 150 beats/min can be atrial flutter with 2:1 conduction, but that number is a clue rather than a diagnosis. Search for atrial activity and its relationship to the QRS. Atrioventricular (AV) nodal slowing may reveal flutter waves without terminating the atrial rhythm; persistent atrial activity during transient block is diagnostically different from termination of an AV-node-dependent circuit (Brugada et al., 2020).

An irregular narrow-complex rhythm may reflect atrial fibrillation, flutter or atrial tachycardia with variable conduction, or multifocal atrial tachycardia. Visible atrial activity, P-wave variation, and the isoelectric baseline help an experienced interpreter distinguish them. An irregular wide-complex rhythm demands particular caution because the possibilities include atrial fibrillation with aberrancy, pre-excited atrial fibrillation, and polymorphic ventricular tachycardia. These alternatives are not safely treated by a blanket “SVT medication” order (Brugada et al., 2020; Joglar et al., 2024).

### Clues to ventricular origin—and their limits

Atrioventricular dissociation, capture beats, and fusion beats can strongly support ventricular tachycardia when correctly identified. Their absence does not exclude it. Ventricular tachycardia can conduct backward to the atria and produce a 1:1 atrioventricular relationship. Morphology algorithms and a prior infarction increase diagnostic confidence, but no single bedside shortcut should delay treatment of deteriorating perfusion. Explain the uncertainty explicitly when requesting help (Zeppenfeld et al., 2022; Brugada et al., 2020).

Nonsustained ventricular tachycardia comprises at least three consecutive ventricular beats, usually faster than 100 beats/min, terminating within 30 seconds. Sustained VT lasts at least 30 seconds or requires earlier termination because of hemodynamic compromise. Duration is a classification, not reassurance: a short run associated with syncope, ischemia, structural disease, or an inherited syndrome can be important. Monomorphic and polymorphic describe the beat-to-beat QRS appearance and direct different emergency considerations (Zeppenfeld et al., 2022).

## Stability determines the next urgent action

The immediate assessment asks whether the tachyarrhythmia is producing hypotension, altered mental status, shock, ischemic chest discomfort, or acute heart failure. Establish monitoring, vascular access, blood-pressure surveillance, and oxygenation support as indicated. Clinical stability can change during a conversation or infusion. Assign someone to reassessment while others obtain the ECG, prepare

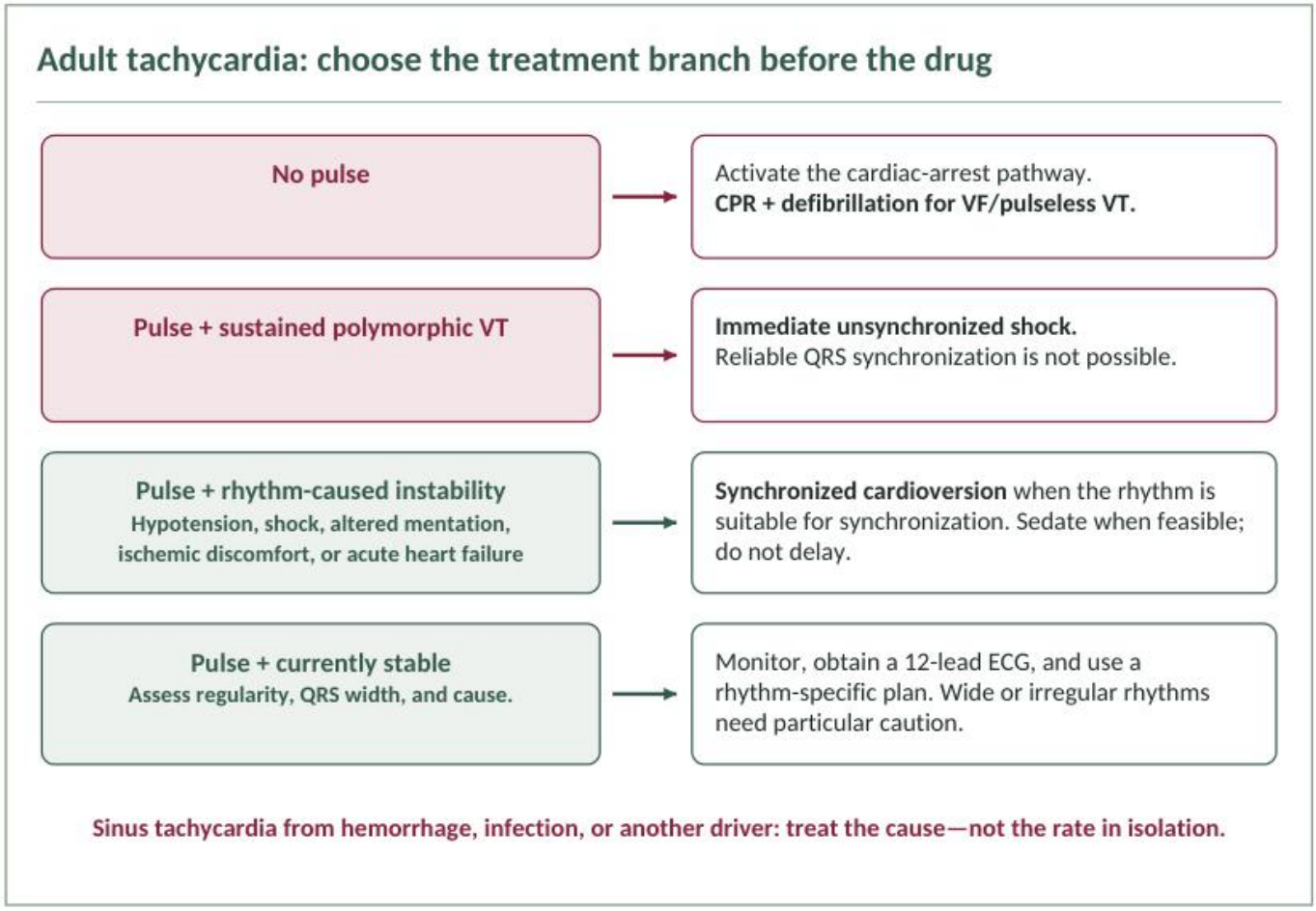


Figure 2. These adult decision lanes separate cardiac arrest, polymorphic VT, rhythm-caused instability, and currently tolerated tachycardia. Reassess continuously and follow authorized local protocols. Original clinical decision graphic. Sources: AHA (2025a, 2025b).

equipment, and communicate with the responding clinician. Figure 2 emphasizes the treatment branches that must not be confused (AHA, 2025a, 2025b).

For an unstable tachyarrhythmia with a pulse that is suitable for synchronization, prepare prompt synchronized cardioversion. Verify synchronization markers on each QRS and reassess synchronization before subsequent shocks; some devices reset after a shock. Use the manufacturer's recommended energy or the relevant current resuscitation guidance. Provide sedation when feasible, but do not delay lifesaving treatment for a lengthy sedation process. Rhythm-caused instability is the key: cardioversion does not correct sinus tachycardia driven by hemorrhage or sepsis (AHA, 2025a, 2025b).

Sustained polymorphic VT is different because reliable synchronization is generally not possible: deliver an immediate unsynchronized shock. Pulseless VT and ventricular fibrillation (VF) require defibrillation with high-quality cardiopulmonary resuscitation (CPR) and the cardiac-arrest sequence. An organized-looking fast rhythm without a pulse is not managed as a stable patient who merely needs a slower rate. Equipment preparation must therefore include the ability to move promptly between cardioversion and defibrillation pathways (AHA, 2025a).

### CASE 2 · "Still talking" does not mean stable

A 74-year-old with a previous myocardial infarction develops a regular wide-complex rhythm at 174 beats/min. Initially alert, the patient now has chest pressure, a blood pressure of 76/44 mmHg, cool skin, and increasing drowsiness. A pulse remains present.

This is an unstable wide-complex tachycardia, highly concerning for VT. Call the emergency response, apply defibrillator pads, prepare synchronized cardioversion, and support airway and circulation. Do not spend additional minutes trying sequential AV nodal blockers to prove a supraventricular origin. Obtain a rhythm strip if it does not delay treatment.

Before the shock, verify QRS synchronization and

the agreed energy setting. If the rhythm becomes polymorphic or the pulse is lost, the treatment branch changes. After conversion, reassess perfusion, obtain a 12-lead ECG, investigate ischemia and reversible triggers, and document the intervention and response. Termination of the rhythm is the beginning of the etiologic evaluation, not its completion (AHA, 2025a, 2025b; Zeppenfeld et al., 2022).

### A stable patient still needs a plan

In suitable stable, regular narrow-complex tachycardia, an authorized vagal maneuver is reasonable. The modified Valsalva studied in REVERT combines a standardized strain with immediate repositioning supine and passive leg elevation; it improved conversion compared with the standard maneuver in that trial. Use a locally taught technique and assess whether positioning and straining are appropriate. Do not teach unsupervised carotid massage as a universal home remedy. Failed maneuvers should lead to the next planned step, not prolonged delay (Appelboam et al., 2015; Brugada et al., 2020).

A stable wide-complex rhythm warrants continuous monitoring and expert-directed antiarrhythmic treatment or cardioversion as appropriate. Keep a defibrillator immediately available. Stability is not permission to combine several drugs without reassessment: additive effects on pressure, conduction, and repolarization may turn a tolerated episode into an emergency. The treatment record should identify each actual administration time, dose, infusion rate, and subsequent change in the patient (AHA, 2025a; Ortiz et al., 2017).

## Medication safety: match the drug to the rhythm

### Adenosine: short action, specific role

Adenosine transiently blocks AV nodal conduction and can terminate AV-node-dependent reentry. It does not directly cure atrial fibrillation or typical atrial flutter. In monitored adults with appropriate regular rhythms, the AHA regimen is a 6 mg rapid intravenous push followed immediately by a saline flush, then 12 mg if needed. The rapid administration and a patent, appropriately located intravenous line matter because the drug is cleared very quickly. Table 1 separates this bolus technique from antiarrhythmic infusions (AHA, 2025a, 2025b).

Record continuously through administration and explain that brief flushing, chest discomfort, breathlessness, or a pause may occur. Resuscitation capability must be available. Check significant bronchospastic disease, conduction disease, and interacting drugs; asthma is a major contraindication in current AHA guidance. Dipyridamole can potentiate the response, while methylxanthines such as theophylline can blunt it. A transplanted heart or central venous administration may require a substantially reduced dose under the applicable protocol rather than automatic use of the usual adult bolus (AHA, 2025a; Brugada et al., 2020).

Adenosine can be considered in an undifferentiated wide-complex tachycardia only when the patient is stable and the rhythm is regular and monomorphic. It must not be given for an unstable, irregularly irregular, or polymorphic wide-complex rhythm. Conversion after adenosine does not replace a full evaluation: selected ventricular rhythms can also respond. The medication response is evidence to interpret, not a substitute for the rest of the ECG and history (AHA, 2025a).

### The pre-excited atrial fibrillation safety gate

In pre-excited atrial fibrillation, rapid conduction over an accessory pathway can reach the ventricles without the normal AV nodal filtering effect. AV nodal blockers—including adenosine, beta blockers, diltiazem, verapamil, digoxin, and amiodarone—are contraindicated because they can precipitate deterioration or ventricular fibrillation. A stable patient may receive specialist-directed intravenous procainamide or ibutilide, or electrical cardioversion; instability requires immediate cardioversion. This is not the same situation as regular orthodromic AVRT, in which the AV node is part of the circuit (Joglar et al., 2024).

**CASE 3 · The irregular wide-complex warning**

A 29-year-old with documented ventricular pre-excitation presents with palpitations. The ECG shows an irregular wide-complex rhythm with changing QRS appearance and very rapid ventricular activation. Blood pressure is currently 112/70 mmHg. An order for intravenous diltiazem is entered as “AF rate control.”

The known pre-excitation and irregular wide-complex rhythm make that order unsafe to administer without immediate clarification. State

## Table 1. Selected adult regimens for monitored tachycardia with a pulse

| Agent and role | Adult dose / administration | Safety and stop checks |
|---|---|---|
| **Adenosine**<br>Appropriate regular AV-node-dependent tachycardia; selected stable, regular monomorphic wide-complex rhythms. | **6 mg rapid IV push**, immediately followed by saline flush. Give **12 mg** if needed under the authorized protocol. | Continuous ECG and resuscitation readiness. Do not use in asthma or pre-excited AF, or for unstable, irregular, or polymorphic wide-complex rhythms. |
| **Procainamide**<br>Stable wide-QRS tachycardia, with expert-directed selection. | **20–50 mg/min IV** until suppression or a stop criterion. Maximum **17 mg/kg**. Maintenance: **1–4 mg/min**. | Stop for hypotension or QRS widening **>50%** from baseline; stop when maximum dose reached. Avoid prolonged QT or congestive heart failure. |
| **Amiodarone**<br>Selected stable ventricular tachycardia; not the cardiac-arrest bolus regimen. | **150 mg IV over 10 minutes**; repeat as needed if VT recurs. Then **1 mg/min for the first 6 hours**. | Monitor blood pressure, rhythm, and conduction. Do not use as routine treatment for pre-excited AF or long-QT torsades. |

These are selected adult regimens, not a complete protocol or pediatric orders. Confirm the rhythm, contraindications, local formulation, and prescribed endpoints. Sources: AHA (2025a, 2025b) and Joglar et al. (2024).

the concern directly, maintain continuous monitoring, apply pads, and obtain urgent expert review for pre-excited AF. Relative hemodynamic tolerance does not make AV nodal blockade safe. Prepare the authorized cardioversion or pathway-active drug strategy, and reassess continually.

The error would be to transfer an ordinary AF rate-control habit to a different conduction mechanism. The useful nursing action is a specific safety stop linked to evidence, followed by closed-loop confirmation of the replacement plan—not simply documenting that the original order was questioned (Joglar et al., 2024; AHA, 2025a).

### Rate-control drugs and ventricular function

For atrial fibrillation without pre-excitation, beta blockers or nondihydropyridine calcium-channel blockers may control ventricular rate in appropriate patients. Selection depends on blood pressure, ventricular function, bronchospasm, conduction disease, and acute illness. Intravenous diltiazem or verapamil should not be used in moderate or severe left-ventricular (LV) systolic dysfunction. Digoxin may be useful in selected patients but requires attention to renal clearance, interacting drugs, and potassium; it is not a reflex treatment for every fast pulse. A falling rate accompanied by worsening blood pressure is not a successful response (Joglar et al., 2024; AHA, 2025a).

Before and during an infusion, verify the indication, concentration, pump units, vascular access, prescribed endpoints, and stop criteria. A dose in mg/min cannot be entered as mL/h without a concentration-based conversion. Reassess pressure, symptoms, QRS duration, and QT where relevant rather than relying only on the heart-rate display. Make the difference between ordered, prepared, and administered treatment explicit in the record and handoff. When renal function changes, review the regimen again rather than carrying forward yesterday's assessment.

### Antiarrhythmic benefit comes with proarrhythmic risk

Procainamide can terminate stable monomorphic wide-complex tachycardia, but hypotension, QRS widening, and QT prolongation limit use. Avoid it when the QT is prolonged or congestive heart failure makes the AHA regimen unsuitable. Amiodarone can also cause infusion-related hypotension and bradycardia. The small PROCAMIO trial favored procainamide over amiodarone for termination and fewer major adverse events in tolerated wide-QRS tachycardia; it did not establish a treatment for unstable patients or make either

drug universally preferable (AHA, 2025b; Ortiz et al., 2017).

For longer-term treatment, selection must account for the underlying heart. Flecainide and propafenone are not appropriate substitutes in patients with prior myocardial infarction or significant structural disease. Sotalol and dofetilide require QT, electrolyte, renal-function, and initiation-protocol checks. Amiodarone has important thyroid, hepatic, pulmonary, ocular, and interaction burdens despite its useful rhythm effects. A medication that has reduced palpitations still requires scheduled surveillance, a reconciled medication list, and instructions about symptoms that should prompt review (Joglar et al., 2024; Zeppenfeld et al., 2022).

### Torsades is not every polymorphic rhythm

Torsades de pointes is polymorphic VT occurring with prolonged repolarization. Sustained episodes require immediate unsynchronized shock. Recurrent episodes call for magnesium, correction of potassium and other reversible factors, and withdrawal of QT-prolonging contributors under an emergency protocol. Pause-dependent acquired torsades may require expert-directed pacing or other rate support. By contrast, polymorphic VT with a normal QT often raises concern for ischemia; routine magnesium is not the recommended substitute for treating that cause. Do not automatically add a QT-prolonging antiarrhythmic to long-QT torsades (AHA, 2025a; Zeppenfeld et al., 2022).

### A new U.S. option for selected adult PSVT

Etripamil nasal spray (Cardamyst) is now U.S.-approved for conversion of acute symptomatic paroxysmal supraventricular tachycardia (PSVT) to sinus rhythm in adults. The initial dose is 70 mg: one spray in each nostril from one device. Persistent symptoms after 10 minutes allow a second 70 mg dose; the maximum is 140 mg in 24 hours. Administer while seated. Seek clinical or emergency help if symptoms have not improved within 20 minutes after the second dose; emergency warning signs warrant help immediately (Milestone Pharmaceuticals USA, 2025).

Contraindications include New York Heart Association (NYHA) class II–IV heart failure, Wolff–Parkinson–White (WPW) or Lown–Ganong–Levine syndrome or manifest pre-excitation, sick sinus syndrome without a pacemaker, Mobitz II or higher AV block, and hypersensitivity. Dizziness, syncope, and local nasal effects require counseling. This is a prescribed plan for selected patients with established PSVT—not treatment for undiagnosed palpitations, AF, VT, or unstable episodes. The U.S. label is not a worldwide authorization, and symptom recognition and escalation teaching remain essential (Milestone Pharmaceuticals USA, 2025).

## Beyond conversion: recurrent SVT and atrial fibrillation

Recurrent episodes affect more than a monitor reading. Patients may avoid exercise, driving, travel, or work because they fear sudden symptoms. An apparently anxious patient may have an intermittent arrhythmia that has not yet been captured. Ask what the episodes prevent the person from doing, correlate symptoms with rhythm, and avoid using psychological explanations to close the diagnostic process prematurely. Conversely, a documented arrhythmia and anxiety may both need attention (Brugada et al., 2020).

For symptomatic AVNRT, AVRT, and many focal atrial tachycardias, catheter ablation is an important initial option after discussion of benefits, procedural risks, and patient preferences. Chronic antiarrhythmic therapy is not a mandatory first step for every patient. Ablation strategy and risk differ by mechanism and anatomy, including proximity to the normal conduction system. Referral should communicate the documented rhythm, prior treatment response, structural disease, and the patient's goals—not merely the label “SVT.” (Brugada et al., 2020).

Atrial fibrillation requires parallel attention to comorbidities, stroke prevention, symptom reduction, and reassessment. Rhythm control may involve medication, cardioversion, or ablation; rate control does not remove embolic risk. Anticoagulation decisions follow the applicable validated risk framework, valve disease, bleeding considerations, and shared decision-making. A normal pulse after cardioversion or ablation is not, by itself, a reason to stop anticoagulation. Newly detected AF during acute illness also warrants follow-up because the trigger does not guarantee permanent resolution (Joglar et al., 2024; Van Gelder et al., 2024).

For elective cardioversion, document episode duration and actual anticoagulant adherence. Under the U.S. guideline, AF lasting at least 48 hours generally requires three weeks of uninterrupted therapeutic anticoagulation

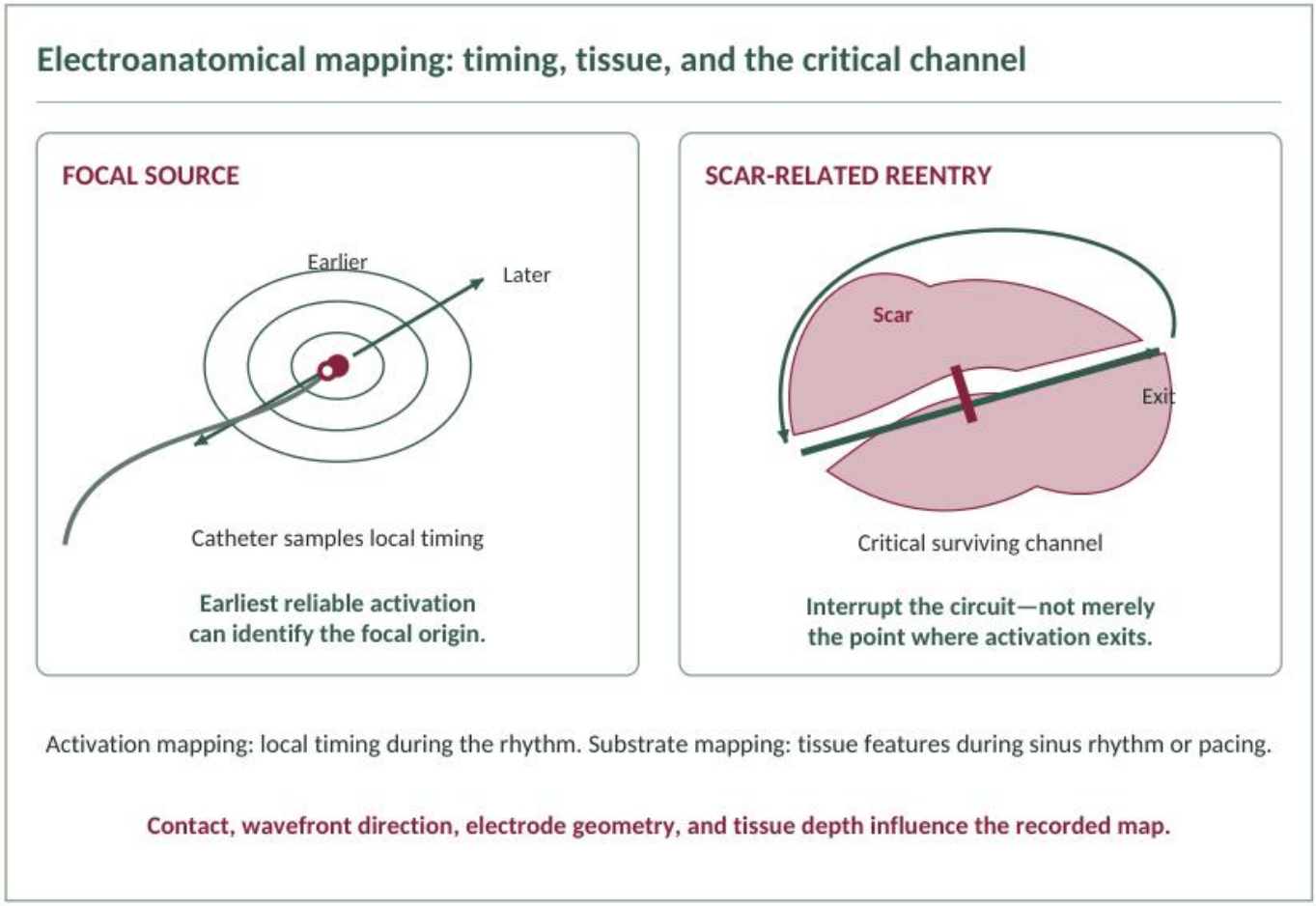


Figure 3. A focal rhythm spreads outward from an origin; scar-related VT may circulate through surviving myocardium. The burgundy cross-channel mark illustrates an ablation target, not a patient-specific lesion plan. Original conceptual maps. Sources: Cronin et al. (2020) and Zeppenfeld et al. (2022).

or imaging to exclude intracardiac thrombus before elective cardioversion, followed by at least four weeks of uninterrupted anticoagulation. The ESC's 2024 approach uses a more conservative safety threshold when AF exceeds 24 hours. Shorter duration is not automatically risk-free. Follow the jurisdictional protocol; emergency cardioversion for life-threatening instability must not be delayed for an elective schedule (Joglar et al., 2024; Van Gelder et al., 2024).

## Mapping and ablation: finding the mechanism

### Different maps answer different questions

Electroanatomical mapping joins catheter location with electrical information. An activation map displays when local tissue depolarizes relative to a selected reference. For a focal rhythm, the earliest reliable local activation may identify the origin. For reentry, the aim is to understand how activation traverses the circuit and identify a critical channel that can be interrupted. Figure 3 contrasts a focal source with scar-related reentry and shows why an exit point is not necessarily the best ablation target (Cronin et al., 2020).

Entrainment uses pacing during an ongoing tachycardia to test whether a site participates in the circuit. Responses such as concealed fusion and a post-pacing interval close to the tachycardia cycle length may support circuit participation, but interpretation requires attention to capture, pacing-induced changes, conduction delay, and the relationship of the recorded electrogram to the tissue being stimulated. These are specialist measurements, not a checklist that can be applied confidently from a surface strip alone (Cronin et al., 2020).

A substrate map seeks potentially arrhythmogenic tissue during sinus rhythm or pacing when sustained VT cannot be safely tolerated. Low-amplitude, late, or fractionated electrograms can identify diseased myocardium and surviving conduction channels. Voltage is affected by catheter contact, electrode size and spacing, activation direction, tissue depth, and anatomy. A low-voltage region is not automatically inert scar, and a single numerical threshold does not fully describe its ability to support reentry (Cronin et al., 2020).

Pace mapping compares the paced QRS with the clinical arrhythmia. A close match may localize an exit region, but can occur across an area larger than the true target. High-density mapping improves sampling, while cardiac magnetic resonance or computed tomography can help define scar and anatomy. None replaces interpretation of the mechanism. Epicardial or intramural disease may explain failure of a purely endocardial approach; a repeat procedure is not automatically evidence that the original rhythm diagnosis was wrong (Zeppenfeld et al., 2022; Cronin et al., 2020).

### Hemodynamic tolerance shapes the procedure

Not every VT can be maintained long enough for detailed activation and entrainment mapping. In unstable rhythms, substrate-based strategies and short, targeted periods of induction may be preferable. Selected high-risk procedures may use circulatory support, but support itself introduces vascular, bleeding, and other risks and is not a routine guarantee of better outcomes. The team balances the value of additional mapping against deterioration, anesthesia effects, fluid load, and the patient's reserve (Cronin et al., 2020; Zeppenfeld et al., 2022).

Procedural success is multidimensional. Noninducibility of relevant VT and elimination of targeted abnormal signals can be useful endpoints, but they do not prove that every future arrhythmia has been abolished. Different studies and centers use different endpoints, which complicates comparison of success rates. A patient may still need an ICD, heart-failure therapy, medication, and surveillance after technically successful ablation (Cronin et al., 2020).

### Newer evidence supports earlier discussion—not universal promises

VANISH2 randomized 416 patients with prior myocardial infarction, clinically significant VT, and an ICD to an initial ablation strategy or antiarrhythmic drug treatment. Over a median 4.3 years, the composite endpoint occurred in 50.7% versus 60.6%, respectively, with a hazard ratio of 0.75. The endpoint included death and clinically important arrhythmia events; the result is not proof of a standalone mortality benefit. Procedure-related and drug-related adverse effects both matter. The trial supports timely discussion of ablation in comparable patients, not extrapolation to every nonsustained VT or every nonischemic cardiomyopathy (Sapp et al., 2025).

### Nursing surveillance around ablation

Before ablation, confirm the medication and anticoagulation plan, renal function, allergies, vascular-access considerations, baseline neurologic findings, and device plan. Afterward, reassess access-site bleeding, distal perfusion, chest symptoms, rhythm, and hemodynamics. New hypotension, dyspnea, neurologic change, or a painful enlarging groin swelling requires prompt escalation. A stable access dressing does not exclude internal bleeding or pericardial complications (Cronin et al., 2020).

Post-ablation antithrombotic treatment must reflect the procedure, lesion burden, embolic risk, bleeding risk, and any independent AF indication. Aspirin is an antiplatelet drug, not an anticoagulant. Do not treat aspirin and anticoagulation as interchangeable or assume that every ventricular ablation requires the same regimen. Confirm the ordered agent, the reason it is needed, the actual start time, and the planned reassessment date before discharge (Cronin et al., 2020).

## ICDs: protection, detection, and appropriate response

An implantable cardioverter-defibrillator (ICD) can terminate dangerous ventricular arrhythmias in selected patients, but it does not prevent every arrhythmia or correct every underlying disease. Secondary-prevention decisions follow assessment of a survived arrest or sustained ventricular arrhythmia, reversible causes, and competing risks. Primary prevention integrates ventricular function, disease substrate, response to medical treatment, timing after acute events, expected meaningful survival, and patient preferences. A fast heart rate alone is not an ICD indication (Zeppenfeld et al., 2022).

### Detection is a programmed decision, not a diagnosis by rate alone

Device detection combines a programmed rate zone with duration or interval-count requirements and, where available, rhythm discriminators. Onset, interval stability, electrogram morphology, and atrial–ventricular relationships help distinguish ventricular rhythms from supraventricular rhythms or oversensing. These tools have limitations. Fast AF can enter a ventricular detection zone, and a slower clinical VT can fall below the programmed treatment threshold. A device that has not shocked does not prove that no important arrhythmia occurred (Stiles et al., 2020).

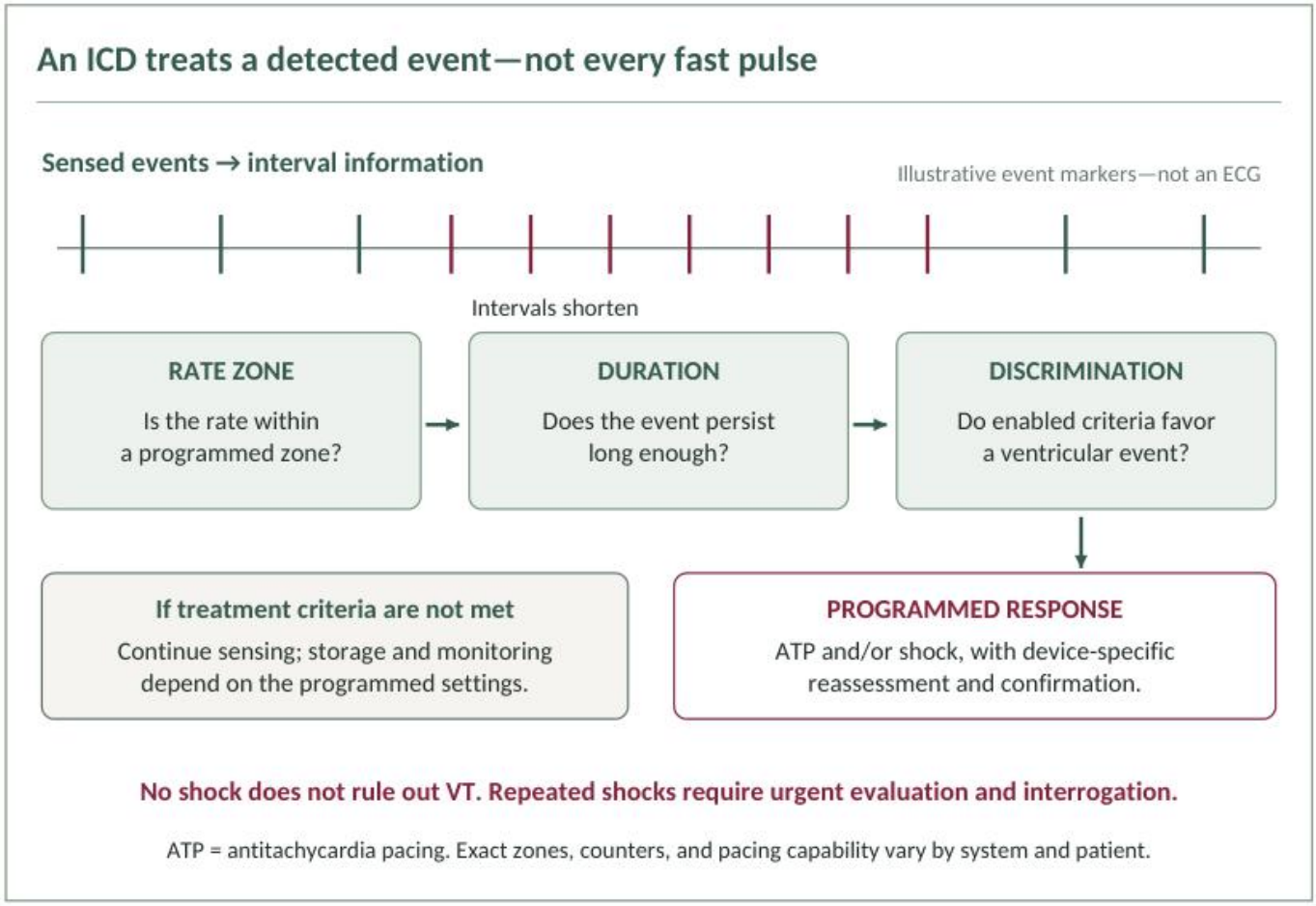


Figure 4. Rate, duration, and rhythm discrimination influence ICD detection and treatment. This conceptual sequence does not specify settings or reproduce a manufacturer algorithm. Device capabilities and the clinical history must guide interpretation. Sources: Moss et al. (2012) and Stiles et al. (2020).

Higher rate thresholds and longer detection intervals reduced unnecessary therapies in selected primary-prevention patients in MADIT-RIT. That finding supports evidence-based individualized programming, not copying one threshold to every device recipient. A patient with documented slow VT may need a different plan. The device type, manufacturer, indication, previous rhythms, medications, and pacing needs all influence programming. Figure 4 illustrates the logic without prescribing universal numerical settings (Moss et al., 2012; Stiles et al., 2020).

### Antitachycardia pacing and shock confirmation

Antitachycardia pacing delivers a short sequence of paced impulses intended to interrupt selected organized ventricular tachycardias. It can end an episode without a painful shock, but may fail or accelerate an arrhythmia. Whether pacing occurs before or during charging, how a rhythm is rechecked, and when a shock is withheld are device- and programming-dependent. Terms such as committed shock, redetection, and episode termination must be interpreted using the actual device report, not a universal count copied from another manufacturer's algorithm (Stiles et al., 2020).

Not all ICD systems have the same pacing capabilities. A subcutaneous ICD does not provide conventional ongoing bradycardia pacing or transvenous-style antitachycardia pacing. Other extravascular and transvenous systems have different features. Record the exact device type and obtain the programmed settings when clinically relevant. Never assume that a generic "ICD present" label tells the responding team which therapies the patient can receive (Zeppenfeld et al., 2022; Stiles et al., 2020).

**CASE 4 · Repeated shocks require more than reassurance**

A 66-year-old with ischemic cardiomyopathy reports three painful ICD shocks over two hours. The patient is alert but frightened; blood pressure is 106/68 mmHg. The emergency monitor currently shows sinus rhythm. A recent diuretic increase and reduced oral intake are noted.

Repeated shocks require urgent evaluation even when the current tracing is reassuring. Obtain continuous monitoring, pads, electrolytes, renal studies, medication history, and expert device interrogation. Determine whether therapies were appropriate for VT/VF, related to a supraventricular rhythm, or caused by oversensing or lead problems. Treat identified reversible contributors while the electrophysiology team plans rhythm suppression, programming changes, or ablation.

Do not place a magnet reflexively to stop distressing shocks. It can suspend tachyarrhythmia therapy, with device-specific behavior, and should be used only under an authorized monitored plan with immediate external defibrillation available. Acknowledge the patient's fear and explain each step. Psychological support and a clear future shock plan belong alongside the electrical evaluation (Zeppenfeld et al., 2022; Stiles et al., 2020).

After any shock or device alert, the follow-up plan should specify whom to call, which symptoms require emergency services, and how remote monitoring is reviewed. Recurrent shocks, syncope, persistent chest discomfort, or severe dyspnea require urgent emergency assessment. Decisions about future shock therapies near the end of life should be discussed in advance with the patient and care team; deactivation is an individualized goals-of-care decision, not an automatic consequence of age (Zeppenfeld et al., 2022).

## When the rhythm weakens the ventricle

Persistent or frequently recurrent tachyarrhythmia can cause or aggravate ventricular dysfunction. The broader term arrhythmia-induced cardiomyopathy includes the adverse effects of irregular activation and frequent ectopy, not simply a sustained high rate. AF, atrial flutter, incessant atrial tachycardia, AVRT, and ventricular arrhythmias can contribute. Frequent premature ventricular complexes may impair function through abnormal activation and mechanical inefficiency even when the average heart rate is not markedly elevated (Huizar et al., 2019; Zeppenfeld et al., 2022).

The mechanisms include reduced filling time, increased energetic demand, disturbed calcium handling, neurohumoral activation, and adverse remodeling. A normal-rate snapshot does not exclude a high arrhythmia burden over the preceding days. Symptoms may be gradual fatigue, exercise intolerance, or congestion rather than dramatic palpitations. Conversely, structural heart disease may be the primary cause of the arrhythmia. The relationship can be bidirectional; clinicians often need treatment response and longitudinal data to determine how much dysfunction is reversible (Huizar et al., 2019).

Suspect an arrhythmia contribution when ventricular dysfunction is otherwise unexplained, when symptoms track a persistent rhythm change, or when a previously stable patient develops a substantial ectopic burden. Quantify rhythm burden using appropriate monitoring and assess ventricular structure and function. Review ischemic, valvular, toxic, inflammatory, genetic, and other causes rather than declaring the diagnosis solely because tachycardia is present. Figure 5 shows why rhythm and ventricular reassessment must proceed together (Huizar et al., 2019; Zeppenfeld et al., 2022).

### CASE 5 · Improvement needs to be measured

A 58-year-old reports three months of worsening exercise tolerance. AF with an average ventricular rate of 124 beats/min is documented on ambulatory monitoring, and echocardiography shows a left-ventricular ejection fraction (LVEF) of 30%. There is no prior echocardiogram. The team suspects an arrhythmia-related contribution while investigating other causes.

The plan combines an individualized rhythm strategy, stroke-prevention assessment, and guideline-directed treatment of ventricular dysfunction. Nursing follow-up tracks symptoms, weight, blood pressure, adherence, adverse effects, and whether the patient can obtain prescribed treatment. Repeat rhythm monitoring and echocardiography are scheduled rather than left to an unspecific “follow up as needed.”

Later improvement after effective rhythm control supports the diagnosis, but does not prove that the underlying substrate has disappeared. Recurrent arrhythmia can again impair function. The patient should not independently stop heart-failure or

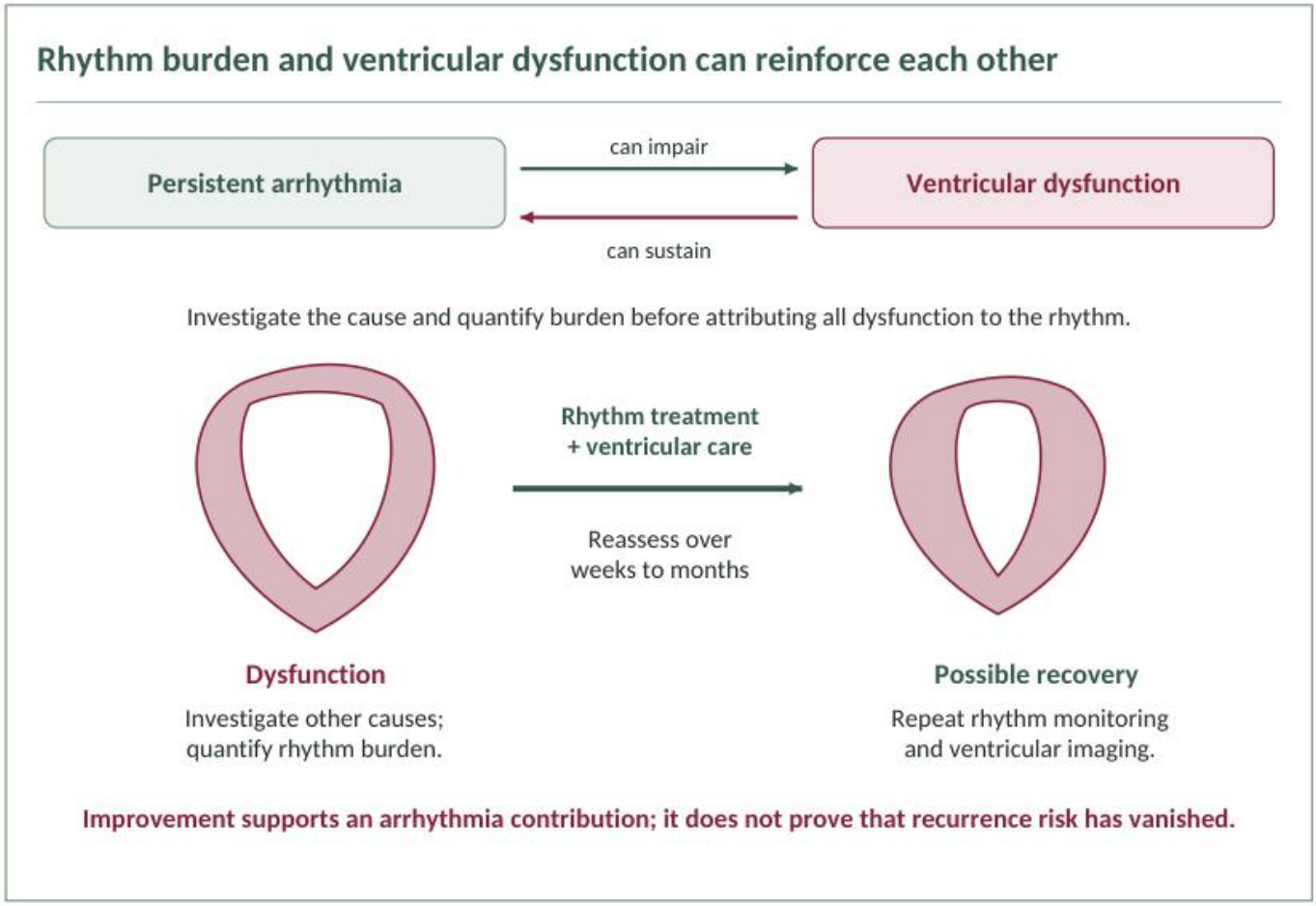


Figure 5. Arrhythmia and ventricular dysfunction may be bidirectionally related. These schematic ventricular sections illustrate a possible response, not measured anatomy or a guaranteed outcome. Continue surveillance after improvement. Sources: Huizar et al. (2019), Joglar et al. (2024), and Zeppenfeld et al. (2022).

> anticoagulant medication because palpitations resolve or one ejection-fraction measurement improves. Those decisions require the treating team's reassessment (Huizar et al., 2019; Joglar et al., 2024; Zeppenfeld et al., 2022).

Recovery can begin within weeks but may take several months; a rigid six-week deadline is inappropriate. Persistent dysfunction after rhythm treatment should prompt reassessment of control, recurrence, and alternative disease. Long-term surveillance remains important because recurrent arrhythmia can produce a rapid decline in a vulnerable ventricle. For nursing practice, the essential outcome is a completed reassessment plan with documented rhythm and function—not simply a successful conversion recorded during the initial admission (Huizar et al., 2019).

## Adapt the pathway to the patient

### Infants and children

The adult threshold of 100 beats/min is not a pediatric diagnostic rule. In infants, poor feeding, sweating, pallor, irritability, tachypnea, or lethargy may replace a verbal report of palpitations. Compare the rhythm with age, fever, distress, and perfusion. A very regular rhythm with abrupt onset and little rate variability raises concern for SVT, but rates alone cannot distinguish every case. Persistent unexplained tachycardia warrants assessment before shock or cardiomyopathy becomes obvious (AHA, 2025c).

Use the pediatric resuscitation pathway for weight-based drugs, cardioversion energy, and monitoring. In cardiopulmonary compromise, synchronized cardioversion begins at 0.5–1 J/kg and may increase to 2 J/kg if unsuccessful under current AHA guidance. Adenosine can be used in appropriate regular SVT, but its preparation and maximum doses differ from simply scaling an adult order. Avoid routine verapamil use in infants. Pediatric electrophysiology

input is especially important for incessant rhythms, recurrent symptoms, structural disease, or suspected inherited conditions (AHA, 2025c; Brugada et al., 2020).

### Pregnancy and the postpartum period

Pregnancy increases heart rate, but persistent or symptomatic tachycardia should not automatically be labeled normal. Assess anemia, infection, thyroid disease, pulmonary embolism, and ventricular dysfunction when suggested by the presentation. A multidisciplinary pregnancy heart team can guide medication selection and monitoring. Vagal maneuvers and intravenous adenosine are options for suitable stable regular SVT; electrical cardioversion should not be withheld when instability requires it. Maternal stabilization is the immediate priority, with fetal assessment incorporated as feasible (De Backer et al., 2025).

Long-term antiarrhythmic and anticoagulant decisions in pregnancy require drug-specific maternal and fetal assessment; ordinary nonpregnant AF prescriptions cannot simply be carried forward. New postpartum dyspnea, syncope, or persistent tachycardia deserves evaluation for cardiomyopathy and thromboembolism. Explain which symptoms require emergency help and ensure the follow-up plan extends beyond delivery. A medication's short duration of action or nasal route does not establish pregnancy safety (De Backer et al., 2025).

### Congenital disease, frailty, and inherited risk

Adults with repaired congenital heart disease may have atrial incisions, patches, residual hemodynamic lesions, and ventricular scars that support complex circuits. An atrial rhythm that would be tolerated in another patient can destabilize a Fontan or systemic-right-ventricle circulation. Seek congenital-heart-disease expertise and preserve previous operative and rhythm information. In older or frail adults, renal changes, interacting medications, falls, cognition, and competing illness shape treatment safety; age alone neither determines nor excludes benefit (Brugada et al., 2020; Zeppenfeld et al., 2022).

Inherited-arrhythmia evaluation is particularly important after exertional syncope, unexplained cardiac arrest, or a suggestive family history. A structurally normal echocardiogram does not exclude an electrical disease. Referral, family assessment, and counseling should follow the phenotype and specialist findings. Avoid indiscriminate genetic testing or assuming that an uncertain variant establishes a diagnosis. Document the circumstances of syncope and family events as carefully as the rate on the monitor (Zeppenfeld et al., 2022).

## A safer handoff, discharge, and reassessment

A useful handoff describes the rhythm evidence, onset and duration, symptoms, perfusion trend, relevant structural disease, and actual treatment response. Include administered drugs and times, infusion concentration and rate, electrolyte results and replacements, cardioversion mode and energy, and the status of expert consultation. For an ICD recipient, add the device type, therapies delivered, interrogation findings, and any temporary change to therapy settings. An unresolved safety concern should remain explicit rather than disappear into the phrase “patient stable.”

Discharge teaching should match the documented diagnosis. Explain the difference between an appropriate sinus response and a recurrent arrhythmia; provide a symptom and episode log when useful. Teach a prescribed home maneuver or rescue medicine by demonstration and teach-back, not merely a handout. Ask the patient to describe the dose, repeat-dose limit, situations in which not to use it, and the point at which to seek help. Ensure that the plan is achievable given cost, access, language, dexterity, and available support (Brugada et al., 2020; Milestone Pharmaceuticals USA, 2025).

Syncope, severe or persistent chest discomfort, severe breathlessness, new neurologic symptoms, or sustained symptoms with poor perfusion require emergency services rather than self-driving. After recurrent ICD shocks, do not wait for a routine clinic appointment. For less urgent recurrent symptoms, specify the contact route and monitoring plan. Driving, occupational restrictions, exercise advice, and return to competitive sport require individualized assessment and applicable local rules; one generic timeline is not appropriate for every rhythm or jurisdiction (Zeppenfeld et al., 2022; Brugada et al., 2020).

Arrange purposeful reassessment: confirm whether the rhythm has recurred, whether treatment is tolerated, whether anticoagulation remains appropriate, and whether ventricular function is recovering. Monitoring should answer these questions, not generate uninterpreted data. A successful educational conversation ends with the patient explaining what will happen next and the team documenting who is responsible for reviewing each result.

## Clinical synthesis

Tachycardia matters because of its cause, mechanism, hemodynamic effect, and cumulative burden. Stabilize the patient before refining the label. Treat a compensatory sinus response by addressing its driver; protect against medication errors when the rhythm is wide, irregular, or pre-excited. Use mapping, ablation, medication, and ICD therapy as complementary tools with different purposes. Finally, measure what happens after conversion: recurrence, ventricular recovery, treatment harm, and the patient's ability to carry out the plan are the outcomes that turn an acute intervention into safer continuing care.

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