

VENTRICULAR TACHYCARDIA

Recognition, Rescue, and Recurrence Prevention

Audience: RN/APRN | **Contact hrs:** 1.5 | **Pharmacology hrs:** 0.5

Expected revision: October 5, 2028

Pharmacology hours are included in the total contact hours.

The rhythm is only the beginning

A patient with ventricular tachycardia may be talking, hypotensive, or pulseless. The tracing alone cannot tell the nurse which emergency pathway to activate. The first decision is therefore clinical: does this patient have a pulse, and is the tachycardia compromising perfusion? A second decision follows immediately: is the ventricular rhythm monomorphic or polymorphic? These distinctions determine whether a synchronized shock, an unsynchronized shock, or a monitored diagnostic and medication strategy is appropriate. (American Heart Association [AHA], 2025a, 2025b, 2025c, 2025d)

Surviving the episode is not the end of treatment. A healed infarct can leave a reentrant circuit; a medication interaction can prolong repolarization; a repaired congenital defect can create an anatomical isthmus. An implantable cardioverter-defibrillator can terminate an arrhythmia without removing its cause. Effective nursing care connects emergency rescue to investigation, medication safety, recurrence prevention, and an understandable plan for life after discharge. (Cronin et al., 2019; Lenarczyk et al., 2024)

Purpose, audience, and learning outcomes

This activity prepares registered nurses and advanced practice registered nurses to recognize ventricular tachycardia, support safe acute treatment, and coordinate longer-term care. Adult pathways predominate; pediatric and congenital-heart-disease differences are identified explicitly. U.S. guidance and product labeling are distinguished from internationally applicable principles. RNs assess, administer authorized treatment, monitor, educate, and escalate. Diagnosis, prescribing, device programming, and procedural decisions require appropriately authorized clinicians. The five cases are fictional teaching examples, not patient records.

After completing the activity, learners should be able to:

1. relate reentry, automaticity, and triggered activity to

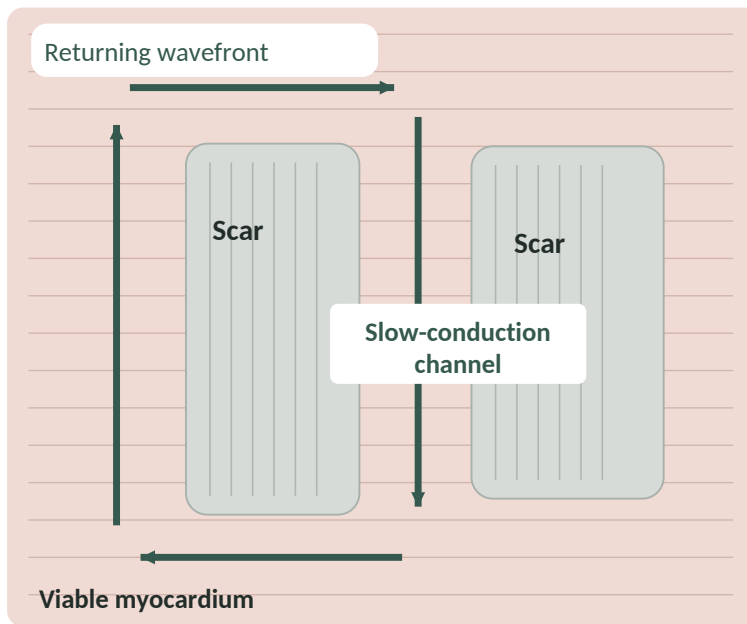
clinical presentations; **2.** differentiate wide-complex tachycardia using patient assessment and electrocardiographic context; **3.** prioritize pulse-, perfusion-, and morphology-specific rescue; **4.** identify antiarrhythmic dosing, interaction, and monitoring hazards; **5.** explain complementary roles of implantable defibrillators, catheter ablation, and substrate-directed treatment; and **6.** adapt surveillance, escalation, and education to pediatric, congenital, and other higher-risk populations.

Why ventricular tachycardia starts—and persists

Ventricular tachycardia (VT) consists of at least three consecutive ventricular beats at a rate above 100 beats/min. Nonsustained VT terminates spontaneously within 30 seconds; sustained VT lasts at least 30 seconds or requires earlier termination because of hemodynamic compromise. Monomorphic VT has a relatively consistent QRS configuration; polymorphic VT changes configuration from beat to beat. Ventricular fibrillation (VF) lacks organized ventricular activation. These labels describe different patterns, not a reliable progression from harmless to dangerous. A short episode accompanied by syncope may be more concerning than a longer, tolerated episode in another setting. (Cronin et al., 2019)

Reentry requires a pathway that permits a wavefront to return to tissue that has recovered excitability. After myocardial infarction, surviving bundles of myocytes can conduct slowly through and around fibrous scar. A premature beat, unidirectional block, and sufficient conduction delay can establish a repeating circuit. The dense scar is not itself conducting normally; the viable channels within its border are important. Figure 1 separates this persistent substrate from the temporary trigger that initiates a particular episode. (Cronin et al., 2019)

Healing changes more than the visible scar. Fibrosis separates cells, gap-junction organization changes, and conduction becomes nonuniform. Altered ion currents

SCAR PROVIDES THE PATHWAY; A TRIGGER STARTS THE EPISODE**REENTRY**

A wavefront returns to recovered tissue through a viable circuit.

AUTOMATICITY

Ventricular or Purkinje tissue initiates impulses without a loop.

TRIGGERED ACTIVITY

Afterdepolarizations follow a prior action potential; QT and calcium context matter.

A healed scar can remain arrhythmogenic after the coronary obstruction has been treated.

Figure 1. A persistent substrate is different from its initiating trigger. The myocardial view is a conceptual tissue diagram, not a patient image.

Original educational visualization based on Cronin et al. (2019) and Gorenec et al. (2024).

and regional sympathetic innervation influence refractoriness and triggering. A patent coronary artery does not reverse every consequence of an old infarct. Conversely, new ischemia may provoke polymorphic VT or VF without an established scar circuit. The distinction explains why coronary treatment can be essential during an ischemic emergency yet insufficient as the only strategy for recurrent scar-mediated monomorphic VT. (Cronin et al., 2019; Gorenec et al., 2024)

Abnormal automaticity means that ventricular or Purkinje tissue generates impulses without requiring a circulating wavefront. Ischemia, metabolic disturbance, or catecholamine excess may contribute. Triggered activity depends on afterdepolarizations following a preceding action potential. Early afterdepolarizations arise before repolarization is complete and are important in long-QT-associated torsades de pointes. Delayed afterdepolarizations arise after repolarization and are associated with intracellular calcium overload, including catecholamine-sensitive outflow-tract VT, catecholaminergic polymorphic VT, and digoxin toxicity. A bedside rhythm strip does not by itself prove

a cellular mechanism. (Cronin et al., 2019; Lenarczyk et al., 2024)

Idiopathic VT is diagnosed only after an appropriate evaluation has not identified structural or inherited disease. A normal initial echocardiogram is reassuring but does not exclude every cardiomyopathy or scar. Some outflow-tract arrhythmias respond to adrenergic suppression; selected fascicular VT responds to verapamil. Those are specialist-established diagnoses, not permission to give an atrioventricular nodal blocker to an undifferentiated wide-complex tachycardia. Treatment should follow the level of diagnostic certainty actually available. (Cronin et al., 2019; AHA, 2025a)

Assess the patient, preserve the diagnostic evidence

At the bedside, assess responsiveness, breathing, a definite pulse when appropriate, blood pressure, skin perfusion, mental status, chest discomfort, and signs of acute heart failure. Activate the emergency response immediately for collapse or deterioration. A displayed

heart rate is not evidence of effective circulation. Pulse oximetry and an arterial waveform can support the assessment, but motion, low perfusion, damping, or disconnected equipment can mislead. Reassess the patient whenever the rhythm or clinical condition changes. (AHA, 2025a, 2025b)

Wide-complex tachycardia includes VT, supraventricular tachycardia with bundle-branch aberrancy, pre-excited rhythms, ventricular pacing, and metabolic or toxic conduction disturbances. In an adult, a QRS duration of at least 0.12 second is wide. Prior myocardial infarction or structural disease increases concern for VT, but preserved blood pressure, young age, or a familiar bundle-branch pattern does not establish a supraventricular origin. When the diagnosis is uncertain, use a VT-safe approach while obtaining expert interpretation. (AHA, 2025a; Cronin et al., 2019)

Atrioventricular dissociation, capture or fusion beats, and certain QRS morphology or axis patterns can support VT. Their absence does not rule it out, and sophisticated morphology rules should never delay rescue. Obtain a 12-lead ECG during the arrhythmia when feasible, compare it with a baseline tracing, and retain the onset and termination strips. Document the actual rhythm, symptoms, treatment sequence, and response rather than recording only the monitor alarm label. An antiarrhythmic may slow VT enough to fall below an alarm or device detection zone. (Cronin et al., 2019; Lenarczyk et al., 2024)

Artifact deserves a rapid clinical check, not reflex dismissal. A patient who is comfortable with an unchanged palpable pulse while a single lead appears chaotic may have movement or electrode artifact. Inspect other leads and connections while continuing direct observation. In contrast, an unresponsive patient with absent normal breathing and no definite pulse must not wait while someone replaces electrodes or prints a tracing. The important skill is matching signals to physiology without adding a preventable delay.

CASE 1

A reassuring blood pressure is not a diagnosis

A 67-year-old with a prior anterior infarction reports palpitations. The monitor shows a regular wide-complex rhythm at 168 beats/min; blood pressure is 118/72 mm Hg, and the patient is alert. A colleague suggests diltiazem because the patient looks comfortable. The nurse requests a VT-safe

evaluation, places defibrillation pads, obtains a 12-lead ECG without interrupting monitoring, and alerts the treating clinician. Tolerance describes the current circulation; it does not establish supraventricular tachycardia. Diltiazem or verapamil can be harmful in undifferentiated wide-complex tachycardia. (AHA, 2025a)

History should identify syncope, exertional or emotional triggers, fever, recent illness, ischemic symptoms, heart-failure deterioration, prior ablation, implanted devices, congenital repairs, and unexplained family deaths. Reconcile prescriptions, recent dose changes, over-the-counter medicines, supplements, stimulants, and substance exposure. Ask specifically about missed beta-blockers, newly added QT-prolonging medicines, diuretics, vomiting, diarrhea, and reduced intake. Retrieve the device card and previous ECG rather than relying only on recollection. (Lenarczyk et al., 2024)

Investigation after initial stabilization generally includes electrolytes, renal function, a 12-lead ECG in sinus rhythm, and echocardiography. Troponin and coronary assessment are guided by the presentation; a rise after prolonged tachycardia or shocks is not by itself proof of an acute coronary occlusion. Cardiac magnetic resonance can identify scar or inflammation and clarify an apparently normal structural assessment. Selected patients need ambulatory monitoring, exercise testing, electrophysiologic study, or genetic evaluation. The nurse helps ensure that an apparently successful conversion does not end the diagnostic process prematurely. (Cronin et al., 2019; Lenarczyk et al., 2024)

The first treatment branch: pulse, perfusion, morphology

Figure 2 is an orientation map, not a replacement for the full resuscitation protocol. Begin by separating pulseless VT from VT with a pulse. Next, identify serious clinical compromise attributable to the tachycardia: hypotension, altered mental status, shock, ischemic chest discomfort, or acute heart failure. Finally, determine whether the rhythm is monomorphic or polymorphic. Rate contributes to the assessment, but there is no safe rule that VT below 150 beats/min cannot cause instability. (AHA, 2025a, 2025c)

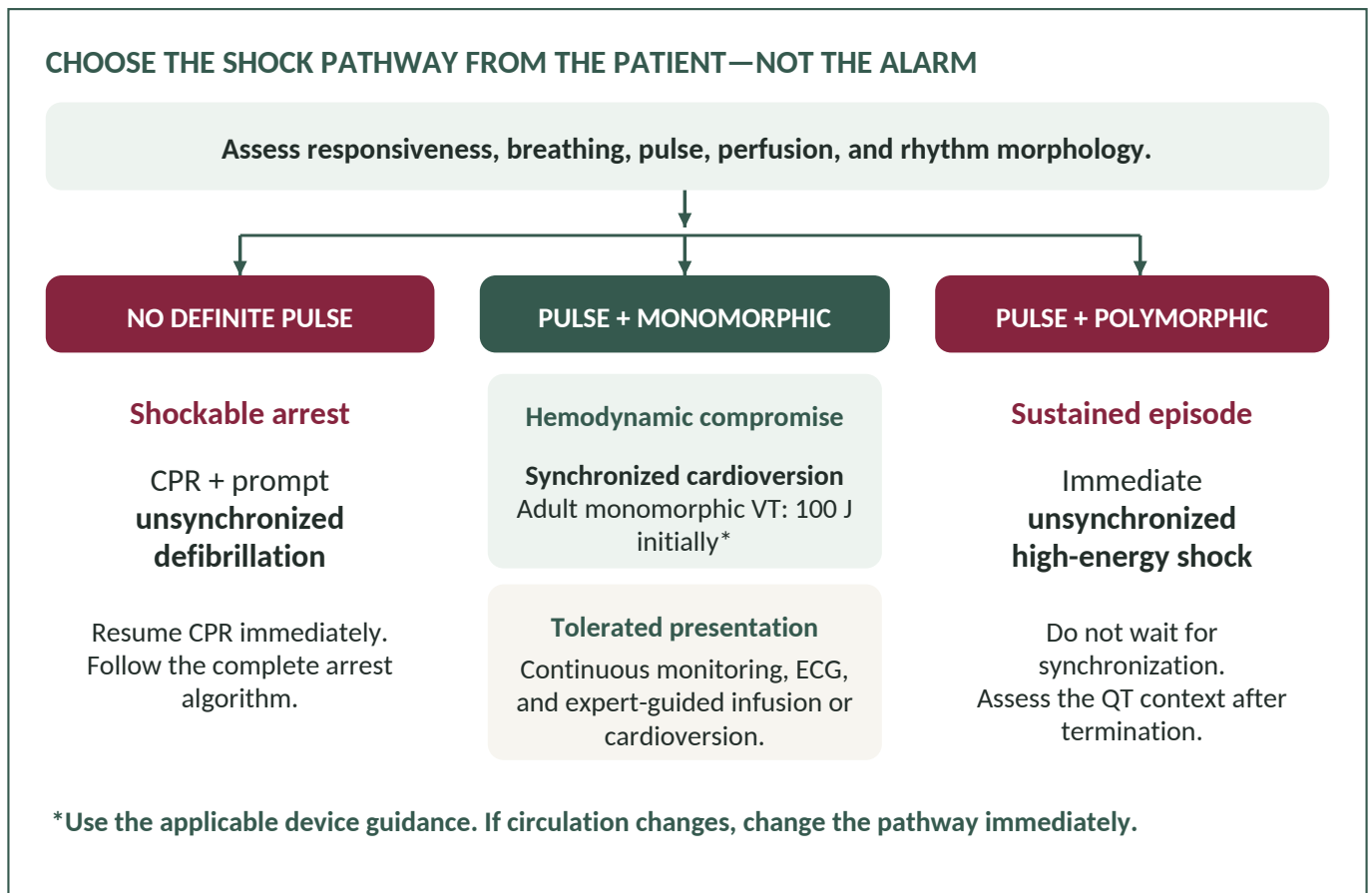


Figure 2. Pulse, perfusion, and morphology guide rescue. This orientation map is not the complete adult or pediatric resuscitation algorithm. Original decision map based on AHA (2025a–2025d).

Pulseless VT: treat as a shockable cardiac arrest

Start high-quality cardiopulmonary resuscitation and deliver prompt unsynchronized defibrillation. Resume compressions immediately after the shock and follow repeated two-minute resuscitation cycles with brief rhythm checks. For adults, use the manufacturer-recommended biphasic energy, commonly 120–200 J initially; when unknown, use the maximum available setting. Subsequent shocks should be at least equivalent, with escalation considered. Monophasic defibrillation uses 360 J. Airway tasks and vascular access must not displace effective compressions or timely shocks. (AHA, 2025b)

For persistent shockable arrest, the AHA algorithm adds epinephrine 1 mg IV/IO every 3–5 minutes after initial defibrillation attempts and amiodarone or lidocaine for refractory VF/pulseless VT. Amiodarone is 300 mg initially and 150 mg for the second dose; lidocaine is 1–1.5 mg/kg initially and 0.5–0.75 mg/kg for the second dose. These are arrest doses, not the infusion used for a perfusing patient. Search concurrently for reversible causes, including hypoxia, potassium disturbance,

toxins, tamponade, and coronary or pulmonary thrombosis. (AHA, 2025b)

Unstable monomorphic VT with a pulse

Prepare immediate synchronized cardioversion. The 2025 AHA electrical cardioversion algorithm lists 100 J for monomorphic VT; use the applicable device-specific recommendations and escalate if unsuccessful. Check that synchronization markers consistently identify QRS complexes rather than T waves or artifact. Sedation is appropriate whenever feasible, with airway preparation, but must not postpone a needed shock. Recheck synchronization before each subsequent attempt because some devices reset after a shock. (AHA, 2025c, 2025d)

When synchronization cannot be achieved promptly and the patient is critically deteriorating, do not allow a technical delay to prevent lifesaving therapy; follow the emergency pathway for unsynchronized shocks. If the pulse is lost, switch immediately to the shockable-arrest sequence. A successful electrical conversion is followed by reassessment of circulation, consciousness, oxygenation, blood pressure, and the precipitating

cause. Persistent shock after conversion requires treatment of the underlying hemodynamic problem, not an assumption that sinus rhythm has restored adequate cardiac output. (AHA, 2025b, 2025d)

CASE 2

The pathway changes when the patient changes

A 74-year-old has monomorphic VT at 176 beats/min, new confusion, diaphoresis, and blood pressure 76/44 mm Hg. The team prepares synchronized cardioversion, verifies QRS markers, and does not wait for a prolonged medication infusion. Before the shock, the patient becomes unresponsive and loses a definite pulse. The nurse announces the change, begins compressions, and the team uses unsynchronized defibrillation. Continuing to wait for synchronized delivery would now be the wrong pathway. The rhythm name has not changed, but the circulation has. (AHA, 2025b, 2025d)

Tolerated monomorphic wide-complex tachycardia

A stable presentation creates time for a monitored evaluation, not unmonitored observation. Maintain continuous ECG and frequent blood-pressure assessment, obtain IV access, keep a defibrillator immediately available, and seek expert advice. Appropriate antiarrhythmic infusion or synchronized cardioversion may be selected according to the rhythm, ventricular function, QT interval, contraindications, and local expertise. If the patient deteriorates, abandon the slow diagnostic or infusion pathway and prioritize electrical treatment. (AHA, 2025a, 2025c)

Adenosine is a limited diagnostic or therapeutic option only for a stable, regular, monomorphic wide-complex rhythm when the cause is uncertain. The adult regimen is 6 mg as a rapid IV push with flush, followed by 12 mg if required. Its effect does not always distinguish VT from supraventricular tachycardia because some idiopathic VT is adenosine-sensitive. Do not use it for unstable, irregularly irregular, or polymorphic wide-complex rhythms. Verapamil and diltiazem should not be used for undifferentiated wide-complex tachycardia. (AHA, 2025a, 2025c)

Polymorphic VT: the QT context changes prevention

Sustained polymorphic VT requires immediate unsynchronized high-energy shock because reliable

synchronization is not possible. This applies even when a pulse is initially detectable. After termination, inspect the preceding ECG and the QT interval in sinus rhythm, identify pauses and bradycardia, and evaluate ischemia. Do not assume that every polymorphic episode is torsades de pointes. Figure 3 illustrates why the distinction becomes crucial when preventing recurrence. (AHA, 2025a, 2025d)

Torsades de pointes is polymorphic VT associated with prolonged ventricular repolarization. Acquired contributors include QT-prolonging medicines, drug interactions, bradycardia, and potassium or magnesium depletion. Congenital long-QT syndromes require a separate inherited-arrhythmia assessment. A reported QTc of 500 ms or greater is an important warning signal in the appropriate context, but automated QTc values require review, especially with wide QRS complexes, pacing, or changing heart rates. Compare measurements and the clinical trajectory rather than treating one machine-generated value as infallible. (Lenarczyk et al., 2024)

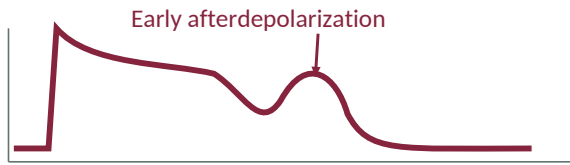
For recurrent long-QT-associated torsades, administer IV magnesium under the emergency protocol, remove offending medicines, and correct electrolyte abnormalities with serial monitoring. Magnesium may be useful even when the serum magnesium is not below the laboratory reference range. The prescribed dose, dilution, and administration speed should match the urgency, renal function, and local protocol. Do not apply a routine electrolyte-replacement infusion rate to every resuscitation setting or assume that faster replacement is safer. Additional QT-prolonging antiarrhythmics may aggravate the mechanism. (AHA, 2025a; Lenarczyk et al., 2024)

Recurrent pause-dependent acquired torsades may require temporary pacing or carefully selected chronotropic treatment directed by experts. Isoproterenol is not a generic therapy for all polymorphic VT, particularly when congenital long-QT disease, catecholaminergic disease, or active ischemia is possible. In polymorphic VT with a normal preceding QT, urgently consider myocardial ischemia; selected lidocaine or amiodarone therapy may be used to suppress recurrence while the cause is treated. Routine magnesium is not recommended for normal-QT polymorphic VT simply because the tracing changes shape. (AHA, 2025a; Lenarczyk et al., 2024)

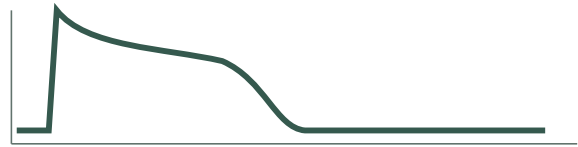
AFTER RESCUE, THE REPOLARIZATION CONTEXT GUIDES PREVENTION

Sustained polymorphic VT: immediate unsynchronized high-energy shock.

PROLONGED QT / TORSADES



NORMAL PRECEDING QT



Schematic cellular potentials—not ECGs or measured QT intervals.

- 1 Remove implicated QT-prolonging medicines; review interactions.
- 2 Give IV magnesium and correct electrolytes under the protocol.
- 3 Address bradycardia and pauses; expert-directed pacing may be needed.

- 1 Urgently evaluate myocardial ischemia and other reversible triggers.
- 2 Selected lidocaine or amiodarone may suppress recurrence.
- 3 Do not give magnesium routinely solely because VT is polymorphic.

Figure 3. Long-QT-associated torsades and normal-QT polymorphic VT need different recurrence-prevention strategies after electrical rescue. Curves are illustrative cellular models, not diagnostic waveforms.

Original visualization based on AHA (2025a) and Lenarczyk et al. (2024).

CASE 3

A medication problem becomes an electrical emergency

A 72-year-old taking sotalol has three days of diarrhea and worsening renal function. Heart rate is 48 beats/min, potassium 2.9 mmol/L, magnesium 1.4 mg/dL, and the reviewed QTc is 560 ms. A newly prescribed antibiotic also prolongs QT. Recurrent self-terminating polymorphic bursts follow long pauses. The nurse urgently escalates the combined bradycardia, electrolyte, renal, and medication findings; the team withholds implicated drugs under the treatment plan, provides magnesium and monitored electrolyte correction, and prepares for defibrillation if an episode persists. Adding amiodarone by habit would ignore the long-QT mechanism. (Lenarczyk et al., 2024; Proficient Rx LP, 2023)

Medication selection is a safety decision

Antiarrhythmic drugs can terminate or suppress VT, but they can also cause hypotension, bradycardia, conduction slowing, or new arrhythmias. A pharmacologic class is not a treatment indication. Sodium-channel effects, beta blockade, and repolarization effects often coexist within one drug. Before administration, reconcile the rhythm diagnosis, pulse status, QT interval, ventricular function, renal function, prior doses, and other rate-slowing or QT-prolonging agents. Keep the intended endpoint and stopping criteria visible at the bedside. (AHA, 2025a; Lenarczyk et al., 2024)

Procainamide: useful selection, explicit stopping points

For selected tolerated wide-QRS tachycardia, the AHA infusion is 20–50 mg/min until suppression, hypotension, QRS widening greater than 50%, or a total dose of 17 mg/kg. Maintenance is 1–4 mg/min. Avoid this pathway in prolonged QT or congestive heart failure. Renal dysfunction increases concern about accumulation of procainamide and its active metabolite,

Table 1. Adult antiarrhythmic pathways: keep pulse-present regimens separate from arrest doses.

Clinical context / drug	Verified adult regimen	Key nursing safeguards
Perfusing, selected tolerated wide-complex tachycardia: procainamide	20–50 mg/min to suppression or a stopping criterion; maximum 17 mg/kg. Maintenance 1–4 mg/min.	Stop loading for hypotension or QRS widening >50%. Avoid with prolonged QT or congestive heart failure; review renal function.
Perfusing, selected tolerated wide-complex tachycardia: amiodarone	150 mg IV over 10 minutes, then 1 mg/min for 6 hours; the reviewed injection label continues 0.5 mg/min thereafter.	Monitor blood pressure, rate, conduction, and QT. Check formulation, dilution, filter, pump, and prior doses. This is not the arrest bolus.
Refractory VF / pulseless VT: amiodarone or lidocaine	Amiodarone: 300 mg IV/IO, then 150 mg. Alternative lidocaine: 1–1.5 mg/kg IV/IO, then 0.5–0.75 mg/kg.	Use within the complete cardiac-arrest sequence. Neither drug replaces high-quality CPR or prompt defibrillation.
Stable, regular, monomorphic wide-complex rhythm of uncertain origin: adenosine	6 mg rapid IV push with flush; 12 mg if needed.	Not for unstable, irregularly irregular, or polymorphic wide-complex tachycardia. Response alone does not always establish a supraventricular origin.

Sources: AHA (2025b, 2025c); Fresenius Kabi USA (2025). Apply authorized local protocols and exact product instructions. Pediatric regimens are different.

so maintenance decisions require individualized review. A nurse should not continue loading to the maximum dose after a stopping criterion has already appeared. (AHA, 2025c; Lenarczyk et al., 2024)

In PROCAMIO, a small randomized open-label study with 62 analyzed patients, procainamide produced fewer major cardiac adverse events and more frequent termination of tolerated wide-complex tachycardia than amiodarone. The study supports thoughtful selection rather than automatic use of the most familiar agent. It does not establish procainamide as the correct drug for a patient in shock, with marked QT prolongation, or with a contraindication. The study infusion regimens also differed from the bedside AHA algorithm; do not interchange trial methods and local orders. (Ortiz et al., 2017)

Amiodarone: distinguish a perfusing patient from arrest

For stable wide-complex tachycardia, the adult AHA regimen is 150 mg IV over 10 minutes, followed by 1 mg/min for six hours. The U.S. injection label continues at 0.5 mg/min thereafter. The concentrated product reviewed requires dilution, controlled infusion, and an in-line filter; use the exact formulation instructions because ready-to-use products may differ. Check the

line, pump, concentration, compatibility, and previous loading doses. The 300-mg arrest bolus must not be substituted for a monitored 150-mg infusion in a patient with a pulse. (AHA, 2025b, 2025c; Fresenius Kabi USA, 2025)

The U.S. IV label addresses frequently recurring VF and hemodynamically unstable VT refractory to other therapy; guideline-supported use in stable wide-complex tachycardia extends beyond that narrow label. This distinction should be understood, not concealed. Infusion-related hypotension, bradycardia, atrioventricular block, liver injury, and QT prolongation require surveillance. New hypotension during a loading infusion demands immediate reassessment of both the drug and the arrhythmia. Slowing or stopping an infusion follows the order set and clinical direction; it does not replace treatment of shock. (Fresenius Kabi USA, 2025)

Long-term oral amiodarone requires a separate monitoring plan. Pulmonary symptoms, thyroid dysfunction, liver injury, visual symptoms, bradycardia, photosensitivity, and neurologic complaints deserve attention. Its long tissue persistence means adverse effects and interactions can continue after discontinuation. Review interactions with warfarin, digoxin, other bradycardic drugs, and QT-prolonging

agents. The patient should know which monitoring appointments and laboratory tests are due and should report new cough or breathlessness rather than attributing everything to heart failure. (Taro Pharmaceuticals U.S.A., 2026)

Lidocaine, beta-blockers, and selected chronic therapy

Lidocaine is an alternative to amiodarone for refractory VF/pulseless VT and may be selected for recurrent ventricular arrhythmia associated with acute ischemia. It is not a universal solution for scar-mediated VT. After a bolus, continued therapy requires an authorized infusion plan and monitoring for central nervous system toxicity, including confusion, paresthesias, tremor, or seizures. Reduced hepatic perfusion in shock or heart failure can impair clearance. Mexiletine is an oral sodium-channel blocker sometimes used as a specialist adjunct; gastrointestinal and neurologic intolerance can limit adherence. (AHA, 2025b; Lenarczyk et al., 2024; Gorennek et al., 2024)

Beta-blockade addresses sympathetic triggering and is particularly important in selected inherited syndromes, electrical storm, and heart failure. It does not replace defibrillation or cardioversion when rescue is needed. Drug selection and titration depend on perfusion, bradycardia, conduction disease, bronchospasm, and ventricular function. Do not generalize the survival benefit of evidence-based heart-failure therapy to every antiarrhythmic drug or every VT substrate. Short-acting IV agents in shock-prone patients require intensive supervision. (Lenarczyk et al., 2024; Heidenreich et al., 2022)

Sotalol combines beta-blocking and repolarization-prolonging effects. It can suppress arrhythmia while also creating torsades risk, especially with renal impairment, bradycardia, or low potassium or magnesium. The U.S. oral label reviewed requires monitored initiation or reinitiation for at least three days with resuscitation capability and creatinine-clearance assessment. Follow the product-specific QT, renal dosing, and interaction instructions. A prescription that was appropriate before acute kidney injury may be unsafe afterward. Never advise a patient to double a missed dose. (Proficient Rx LP, 2023)

Flecainide and propafenone are not routine choices for ischemic scar-related VT; important structural or ischemic heart disease changes their safety. Specialist use of flecainide in catecholaminergic polymorphic VT

is a different indication and does not erase that restriction. Verapamil can treat selected established fascicular VT, but not an undifferentiated wide-complex rhythm. Ranolazine has been studied for ventricular arrhythmia suppression, yet it should not be presented as an established replacement for an ICD or ablation. (Cronin et al., 2019; Lenarczyk et al., 2024)

Medication teaching should end with a practical reconciliation: which drug prevents recurrence, which treats the underlying disease, what must be monitored, and whom to call before starting another prescription. Ask patients to explain the plan in their own words. Include pharmacy access, affordability, refill timing, and what to do during vomiting, diarrhea, or missed doses. A technically correct prescription is not a safe long-term plan when the patient cannot obtain it or cannot recognize its most important warning symptoms.

Electrical storm requires coordinated escalation

Electrical storm is generally defined as at least three sustained ventricular arrhythmia episodes within 24 hours, separated by at least five minutes and requiring an intervention. Repeated ICD therapies, even with spontaneous recovery between episodes, require urgent assessment. The episode count should trigger escalation, not a wait until the patient meets every formal definition. Recurrent VT can reduce cardiac output, while repeated shocks, pain, and anxiety intensify adrenergic activation. (Lenarczyk et al., 2024)

A coordinated response treats the rhythm, the trigger, and the distressed patient. Obtain continuous monitoring and external defibrillation capability, involve electrophysiology and critical care, correct ischemia and metabolic problems, and review medication exposure. Device interrogation distinguishes appropriate treatment of VT from inappropriate therapy caused by another rhythm or sensing problem. Preserve electrograms and event times. A nurse should not assume that every shock represents VT or that a device is functioning normally simply because it delivered energy. (Lenarczyk et al., 2024)

Analgesia and sedation can reduce distress and sympathetic activation; appropriate beta-blockade and antiarrhythmic therapy may be needed. Deep sedation, mechanical circulatory support, stellate ganglion blockade, cardiac sympathetic denervation, or urgent ablation are specialist options for refractory cases. They are not interchangeable routine treatments, and the

evidence varies. Repeatedly adding QT-prolonging drugs without reassessing the substrate, hemodynamics, and drug accumulation can make the emergency worse. (Lenarczyk et al., 2024)

Device therapies should be adjusted only by trained personnel with the appropriate monitoring and backup. A magnet can suspend tachyarrhythmia treatment in many ICDs, but the response depends on the device and programming. It is not a universal nursing intervention for painful shocks. Unsupervised application during true recurrent VT can remove protection. If therapies are temporarily disabled for an appropriate clinical reason, the team must maintain external rescue capability and explicitly document restoration before transfer. (Lenarczyk et al., 2024)

CASE 4

The ICD treated the episodes, not the substrate

A 61-year-old with a healed infarction and an ICD receives three appropriate therapies for monomorphic VT over eight hours despite prescribed antiarrhythmic treatment. The patient now has a perfusing rhythm but is frightened and exhausted. The nurse arranges urgent monitored evaluation rather than a routine outpatient callback, retrieves event timing and medication history, and prepares for device interrogation. The team evaluates reversible triggers and discusses ablation with electrophysiology. Successful shocks do not mean the problem has resolved; the recurrence pattern requires a prevention strategy. (Lenarczyk et al., 2024; Sapp et al., 2025)

Prevent sudden death and reduce recurrence

Long-term management asks two different questions: how should the patient be protected from a future lethal episode, and how can the number of episodes be reduced? An ICD provides rescue capability; antiarrhythmic therapy and ablation can reduce recurrence; disease-directed treatment addresses ischemia, ventricular dysfunction, inflammation, or inherited risk. These approaches often complement one another. A patient may still need an ICD after apparently successful ablation because termination of one circuit does not eliminate every future risk. (Cronin et al., 2019; Lenarczyk et al., 2024)

ICD assessment is individualized

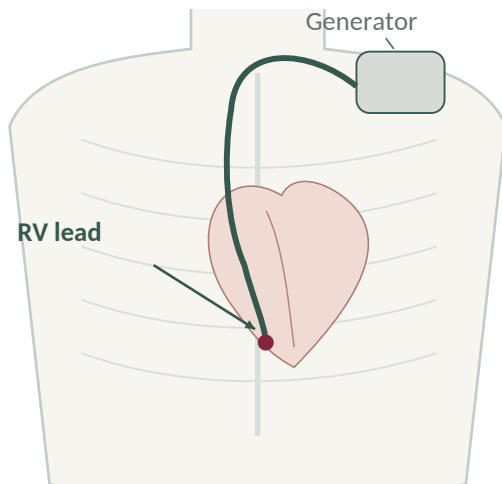
Survivors of cardiac arrest or patients with clinically important sustained VT need assessment for secondary-prevention ICD therapy, including whether a reversible cause fully explains the episode and whether meaningful survival and goals support implantation. Primary prevention is different: ventricular function, the underlying diagnosis, heart-failure treatment, recovery time, and competing mortality risk matter. A single episode of nonsustained VT is not an automatic ICD indication. Recent myocardial infarction or revascularization should not be managed by copying chronic primary-prevention thresholds without checking the required reassessment period. (Heidenreich et al., 2022; Cronin et al., 2019)

A transvenous ICD can provide antitachycardia pacing as well as shocks; some systems also provide bradycardia pacing or cardiac resynchronization when indicated. A conventional standalone subcutaneous ICD avoids an intracardiac lead but does not provide chronic pacing or antitachycardia pacing. Figure 4 compares these two established configurations; it is not an inventory of every newer device platform. Recurrent pace-terminable monomorphic VT, venous access, infection risk, anatomy, and pacing needs influence selection. Device benefits and lead-related risks should be discussed in language the patient understands. (Cronin et al., 2019)

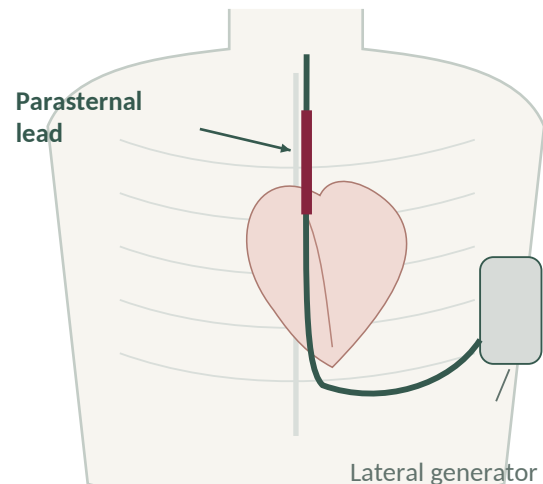
Antitachycardia pacing can interrupt a reentrant circuit without a painful shock, but programming requires individualized detection and treatment zones. Drugs may slow VT below previously programmed detection rates. A wearable defibrillator may be considered for selected temporary-risk or bridge situations; its protection depends on correct use and adherence. It is not a substitute for resolving the long-term treatment question, and a poor prognosis alone does not establish an indication. Record the plan for reassessment, not merely that a device was discussed. (Cronin et al., 2019; Lenarczyk et al., 2024)

Earlier ablation: interpret the evidence in its population

VANISH2 randomized 416 patients with prior myocardial infarction and clinically significant VT, all with ICD protection, to initial catheter ablation or antiarrhythmic therapy. Over a median 4.3 years, its composite outcome occurred in 50.7% with ablation and 60.6% with drug therapy, with a hazard ratio of 0.75.

IMPLANTED DEFIBRILLATORS: LEAD LOCATION CHANGES CAPABILITY**Transvenous ICD****Shock therapy + antitachycardia pacing**

Pacing capabilities depend on the selected system and clinical indication.

Standalone subcutaneous ICD**Shock therapy without an intracardiac lead**

No chronic pacing or antitachycardia pacing from this standalone configuration.

Simplified anterior views. Newer platforms and combined systems require device-specific assessment.

Figure 4. Two established ICD configurations. Lead paths are simplified and not to scale; the device team selects the system according to anatomy and pacing needs.

Original educational schematics based on Cronin et al. (2019) and Lenarczyk et al. (2024).

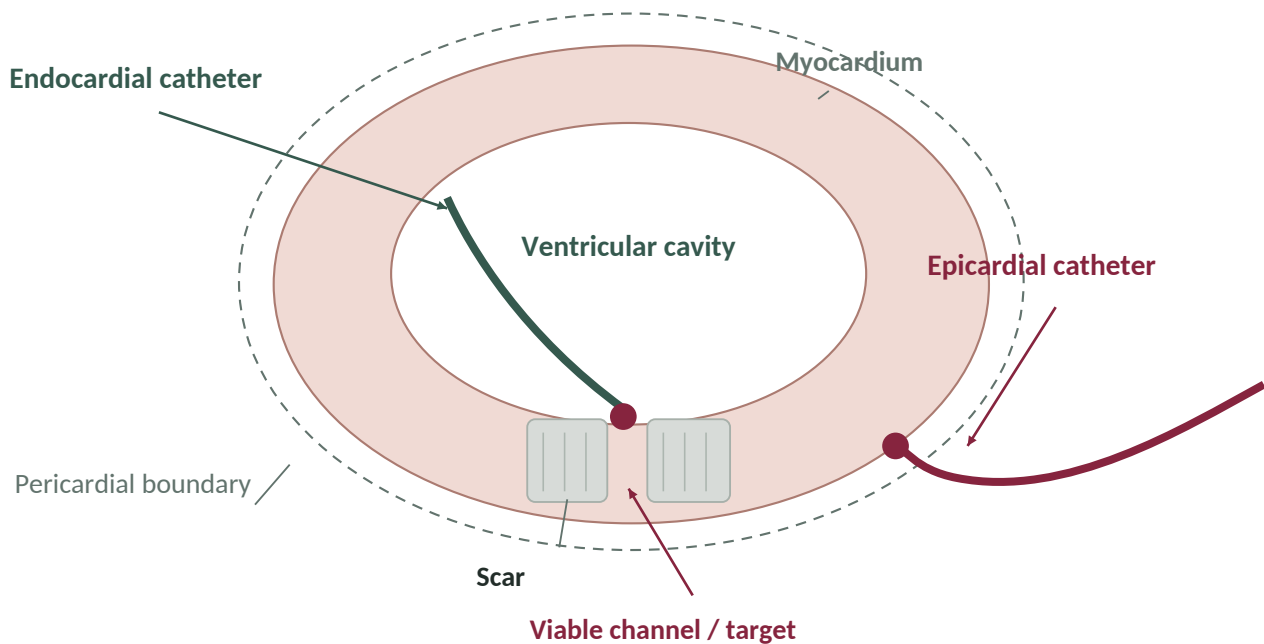
The composite included death and specified recurrent arrhythmia outcomes. Ablation had procedural risks; drug treatment had important adverse effects. These findings support discussing ablation earlier in appropriately selected ischemic VT, rather than assuming that multiple drug failures must always occur first. They do not prove a mortality benefit alone or establish the same strategy for every pediatric, idiopathic, or nonischemic VT population. (Sapp et al., 2025)

Patients need a balanced explanation of what success means. Fewer shocks, fewer hospital visits, and less arrhythmia burden can be meaningful even when no therapy guarantees a cure. The decision includes procedural risk, kidney and lung disease, frailty, ventricular function, the likely substrate, patient preferences, and the experience of the treating center. Nursing assessment can reveal that a patient accepts recurrent admissions because they mistakenly believe ablation requires open-heart surgery, or avoids a needed consultation because one earlier procedure was unsuccessful.

How ventricular ablation changes the substrate

Catheter ablation identifies an arrhythmia focus or a critical part of a circuit and creates a lesion intended to interrupt it. For focal idiopathic VT, the target differs from the surviving myocardial channels of scar-related VT. Activation mapping during tolerated VT can clarify the circuit, but unstable VT may be approached with substrate mapping during sinus rhythm or pacing. A surface ECG often reflects the circuit exit and does not precisely identify every protected isthmus. Figure 5 illustrates endocardial and epicardial access without implying that one approach suits every substrate. (Cronin et al., 2019)

Right-sided targets are often reached through venous access. Left ventricular endocardial work may require a transseptal or retrograde aortic approach. Epicardial mapping requires access to the pericardial space and is selected when the suspected substrate and clinical history justify it. Prior surgery or pericarditis can create adhesions that limit percutaneous access. Coronary arteries and the phrenic nerve must be protected. Deep

VENTRICULAR ABLATION TARGETS A FOCUS OR CRITICAL CIRCUIT

Monitor for bleeding, tamponade, vascular injury, and new neurologic deficits after the procedure.

Figure 5. Endocardial and epicardial targets lie on different myocardial surfaces. Access route and lesion location depend on the patient's substrate; a combined approach is not routine for every patient.

Original conceptual visualization based on Cronin et al. (2019).

intramural circuits may remain difficult to reach even when both surfaces can be mapped. (Cronin et al., 2019)

Before the procedure, clarify ventricular function, imaging findings, anticoagulant and antiarrhythmic instructions, renal status, prior access problems, implanted hardware, and the anesthesia plan. Some patients are vulnerable to circulatory deterioration during VT induction or prolonged procedures. Decisions about mechanical support belong to the multidisciplinary team and should not be represented as routine for all ablations. The nurse can identify discrepancies between the medication list, referral record, and actual doses before they become procedural hazards. (Cronin et al., 2019)

After ablation, monitor rhythm and circulation, vascular sites, distal perfusion, neurologic status, pain, urine output, and signs of bleeding or tamponade. New hypotension, dyspnea, progressive chest discomfort, a focal neurologic deficit, or an enlarging access-site hematoma requires prompt escalation. Epicardial procedures add specific concerns about pericardial bleeding and inflammation. The postprocedural

antithrombotic regimen depends on the approach, extent of left-sided ablation, and other indications; do not apply one universal regimen to every VT ablation. (Cronin et al., 2019)

Confirm which antiarrhythmics continue, whether device programming changed, and how recurrence will be evaluated. Follow-up often combines symptom review, device or ambulatory monitoring, ventricular-function reassessment, and medication surveillance. Explain that recurrence can reflect an incompletely treated circuit, another circuit, or progression of the underlying disease. It does not automatically prove that the patient failed to follow instructions. Early reporting helps the electrophysiology team distinguish these possibilities.

Surgical ablation has a selective role when catheter access is inadequate, the substrate is inaccessible, or another cardiac operation provides an appropriate opportunity. Revascularization treats coronary ischemia but does not reliably erase a healed scar circuit. Autonomic interventions are used in selected refractory cases at experienced centers. Noninvasive cardiac

radioablation and other novel energy strategies should be described as evolving specialist approaches rather than routine alternatives with established long-term safety for all patients. (Cronin et al., 2019; Lenarczyk et al., 2024)

Ventricular ectopy and arrhythmia-induced cardiomyopathy

Frequent premature ventricular complexes (PVCs) or incessant VT can contribute to left ventricular dysfunction, while an existing cardiomyopathy can itself produce ectopy. The direction of causality is not always clear at first presentation. Evaluate burden, morphology, duration, symptoms, ventricular function, and other potential causes. Ambulatory monitoring quantifies the burden, but day-to-day variation means that one short recording may not represent the usual exposure. Symptoms alone are also unreliable: substantial ectopy can occur without palpitations. (Cronin et al., 2019)

A PVC burden around 10% or more warrants attention to ventricular function and follow-up, especially when the burden is persistent. No single percentage proves or excludes PVC-induced cardiomyopathy in an individual. The purpose of a threshold is to trigger investigation, not to label all frequent PVCs as dangerous or to recommend prophylactic elimination in every asymptomatic patient. Suppression or ablation may improve function when the arrhythmia contributes materially to the dysfunction. Recovery supports the diagnosis but requires documented reassessment. (Cronin et al., 2019)

Atrial fibrillation, atrial flutter, and incessant supraventricular tachycardia can also cause or worsen arrhythmia-mediated ventricular dysfunction. These are important differential and comorbidity considerations, particularly in children and congenital disease, but their rate-control or atrial-ablation pathways are not treatments for undifferentiated VT. Keep the arrhythmia diagnosis explicit when reading a referral or discharge summary. A statement that a patient had an ablation is incomplete unless the target and purpose are known. (Cronin et al., 2019)

Continue appropriate disease-directed heart-failure care during reassessment. An improved ejection fraction does not automatically justify stopping guideline-directed treatment in a patient with previous reduced-ejection-fraction heart failure. The heart-failure

guideline recommends continued therapy to reduce relapse risk, even when symptoms improve. Medication withdrawal, changes after ablation, and the need for continuing device protection are individualized decisions rather than automatic consequences of a single normal echocardiogram. (Heidenreich et al., 2022)

Children, congenital anatomy, and uncommon substrates

Pediatric VT is not an adult dose in a smaller patient

Children may present with pallor, poor feeding, irritability, exercise intolerance, syncope, or shock rather than a clear description of palpitations. The differential includes supraventricular tachycardia with aberrancy, myocarditis, cardiomyopathy, inherited electrical disease, drug toxicity, and postoperative substrates. The 2025 pediatric tachyarrhythmia algorithm uses QRS duration greater than 0.09 second for a wide complex; do not substitute the adult 0.12-second threshold. Seek pediatric electrophysiology or cardiology advice early. (AHA & American Academy of Pediatrics [AAP], 2025a, 2025b)

For a child with wide-complex tachycardia, a pulse, and cardiopulmonary compromise, synchronized cardioversion begins at 0.5–1 J/kg and increases to 2 J/kg if ineffective. Sedation must not delay treatment. For a regular monomorphic wide-complex rhythm without compromise, expert consultation is recommended and adenosine may be considered. Irregular or polymorphic rhythms are not candidates for an indiscriminate adenosine trial. Pulseless VT requires the pediatric cardiac-arrest protocol, including weight-based defibrillation rather than the synchronized energies above. (AHA & AAP, 2025a, 2025b)

CASE 5

Weight and physiology determine the pediatric pathway

A 10-year-old weighing 28 kg, with previously repaired tetralogy of Fallot, develops regular wide-complex tachycardia, lethargy, weak pulses, and blood pressure 62/38 mm Hg. A definite pulse is present. The team prepares synchronized cardioversion at 14–28 J, calculated from 0.5–1 J/kg, and prepares 56 J if the first attempt fails. The nurse uses closed-loop confirmation of weight, energy, synchronization, and pulse status. Congenital expertise is requested, but emergency

treatment is not delayed while operative records are located. If the pulse disappears, the team changes to the pediatric arrest pathway. (AHA & AAP, 2025b)

Congenital repairs can create long-term substrates

Adults with repaired congenital heart disease may develop VT many years after surgery. In repaired tetralogy of Fallot, regions between patches, surgical scars, and anatomical boundaries can form slow-conducting isthmuses. Ventricular dilation, residual hemodynamic lesions, and scar also influence risk. Operative anatomy, prior interventions, ventricular function, rhythm documentation, and symptoms need to be considered together. A repaired defect is not equivalent to a structurally normal heart. (Cronin et al., 2019)

Atrial tachyarrhythmias are also common in congenital disease. Atrial septal defects and prior atrial surgery may be associated with flutter or macroreentry, while Ebstein anomaly may involve accessory pathways. These facts matter because a rapid wide-complex rhythm could be pre-excited or supraventricular, not because every congenital arrhythmia should be labeled VT. Complex venous pathways, baffles, conduits, and prior surgery can complicate access and ablation. Transfer information should include the exact congenital diagnosis and repair whenever available. (Cronin et al., 2019)

The 2025 adult congenital-heart-disease guideline emphasizes specialist-center involvement and multidisciplinary planning, particularly for moderate or complex anatomy and procedures. Nursing coordination should connect the emergency team, congenital cardiologist, electrophysiologist, imaging specialists, and the patient's established center. Explain why the referral is important even after rhythm stabilization; local familiarity with ordinary ischemic VT does not substitute for understanding an unusual surgical circulation. (AHA, 2025e)

Inherited disease, inflammation, and tumors

Exercise- or emotion-associated syncope, unexplained family death, or polymorphic/bidirectional VT in an apparently normal heart should prompt inherited-arrhythmia evaluation. Long-QT syndrome, catecholaminergic polymorphic VT, and other channelopathies differ in triggers and treatment. Family assessment, genetic counseling, medication avoidance,

and individualized activity advice may be needed. Do not reassure a patient solely because resting imaging is normal, and do not impose a universal exercise rule without the specialist's disease-specific assessment. (Cronin et al., 2019; Lenarczyk et al., 2024)

Myocarditis and other inflammatory or infiltrative processes may produce both active electrical instability and later scar-mediated VT. New ventricular arrhythmia with chest pain, a recent inflammatory illness, or unexplained ventricular dysfunction warrants evaluation of the underlying disease. The treatment question extends beyond suppressing the rhythm: imaging, hemodynamic support, disease-directed therapy, and the timing of longer-term protection require specialist assessment. (Peretto et al., 2020)

Cardiac tumors are uncommon but clinically relevant substrates. A ventricular mass may disrupt conduction, invade tissue, or create a reentrant border. Acute rescue follows the patient's pulse, perfusion, and rhythm, not the tumor's presumed benign or malignant label. Imaging and multidisciplinary evaluation determine whether tumor-directed therapy, resection, antiarrhythmics, or selected ablation is appropriate. Rhabdomyomas may regress, whereas another mass may require a different approach; neither spontaneous regression nor successful tumor treatment guarantees immediate freedom from arrhythmia. Published experience is largely observational, and individual case reports must not be converted into a universal chemotherapy, device, or surgical recommendation. (Pengju et al., 2025)

Safer transitions, follow-up, and clinical synthesis

Discharge planning should specify the arrhythmia diagnosis, probable cause, unresolved investigations, medication changes, monitoring schedule, device status, and who owns follow-up. Record the QT and renal issues that influenced treatment, the actual ablation target if a procedure occurred, and any postprocedural anticoagulation instructions. A transfer summary should distinguish sustained VT, nonsustained episodes, inappropriate ICD therapy, and uncertain monitor events. These are not interchangeable entries in a problem list.

Give patients a written action plan for recurrent palpitations, syncope, chest discomfort, breathlessness, and ICD shocks. Persistent symptoms, collapse, or repeated shocks require emergency services. After a

single shock without ongoing symptoms, the patient should promptly follow the individualized device-clinic instructions rather than assuming no review is needed. Teach device identification, remote-monitoring expectations, wound care when relevant, and avoidance of unapproved medication changes. Driving and occupational restrictions vary by jurisdiction, event, and device indication; the treating team must provide the applicable advice. (Lenarczyk et al., 2024)

Ask about fear of shocks, sleep disruption, activity avoidance, and caregiver distress. A patient may stop exercising, traveling, or sleeping alone even when the rhythm is controlled. Clear explanations, an emergency plan, and appropriate psychosocial support are part of recovery. Discuss goals of care and future device decisions in advanced illness before a crisis forces the conversation. Decisions about deactivating shock therapy require informed, individualized discussion and do not mean that all other treatment or comfort-focused care stops. (Heidenreich et al., 2022)

The central nursing task is to connect changing physiology with the next safe action. Confirm circulation before choosing a pathway; distinguish monomorphic from polymorphic rhythms; use the QT context to prevent recurrence; and check medication safety beyond the name on the syringe. After rescue, preserve diagnostic evidence and ensure that the patient reaches an appropriate prevention pathway. ICDs, ablation, disease-directed treatment, and patient education address different parts of the same problem. A quiet monitor is reassuring, but a complete care plan requires more than the disappearance of VT.

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