

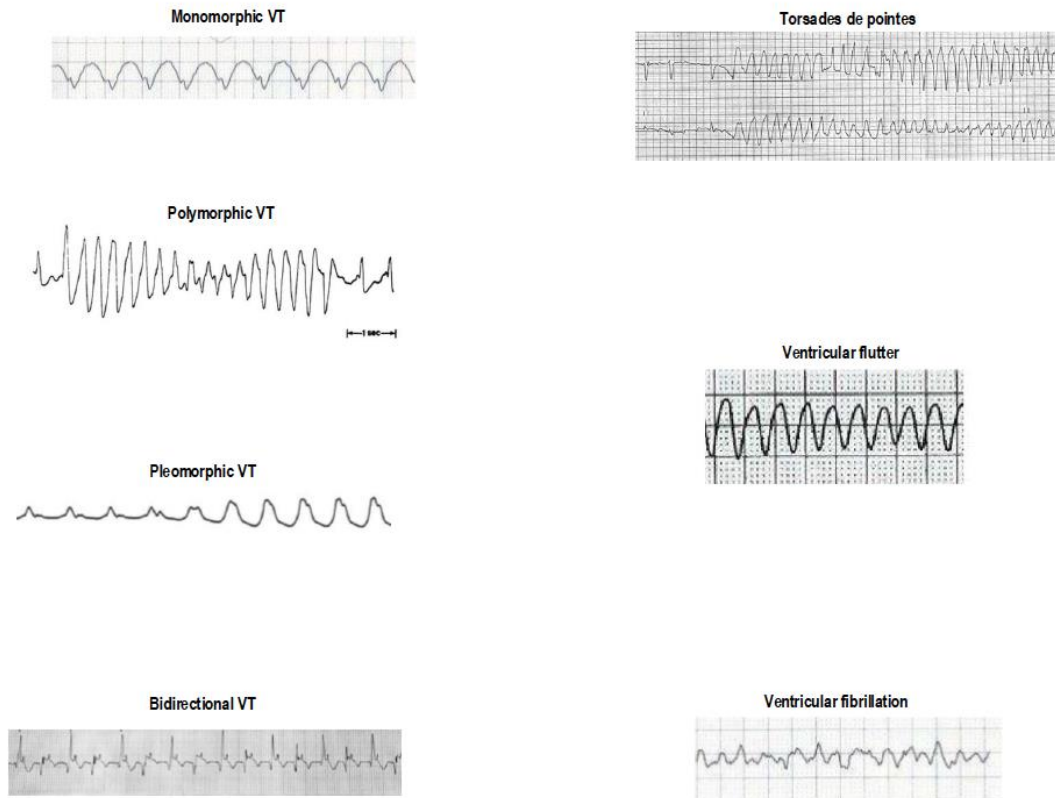


Figure 1. Types of VT.

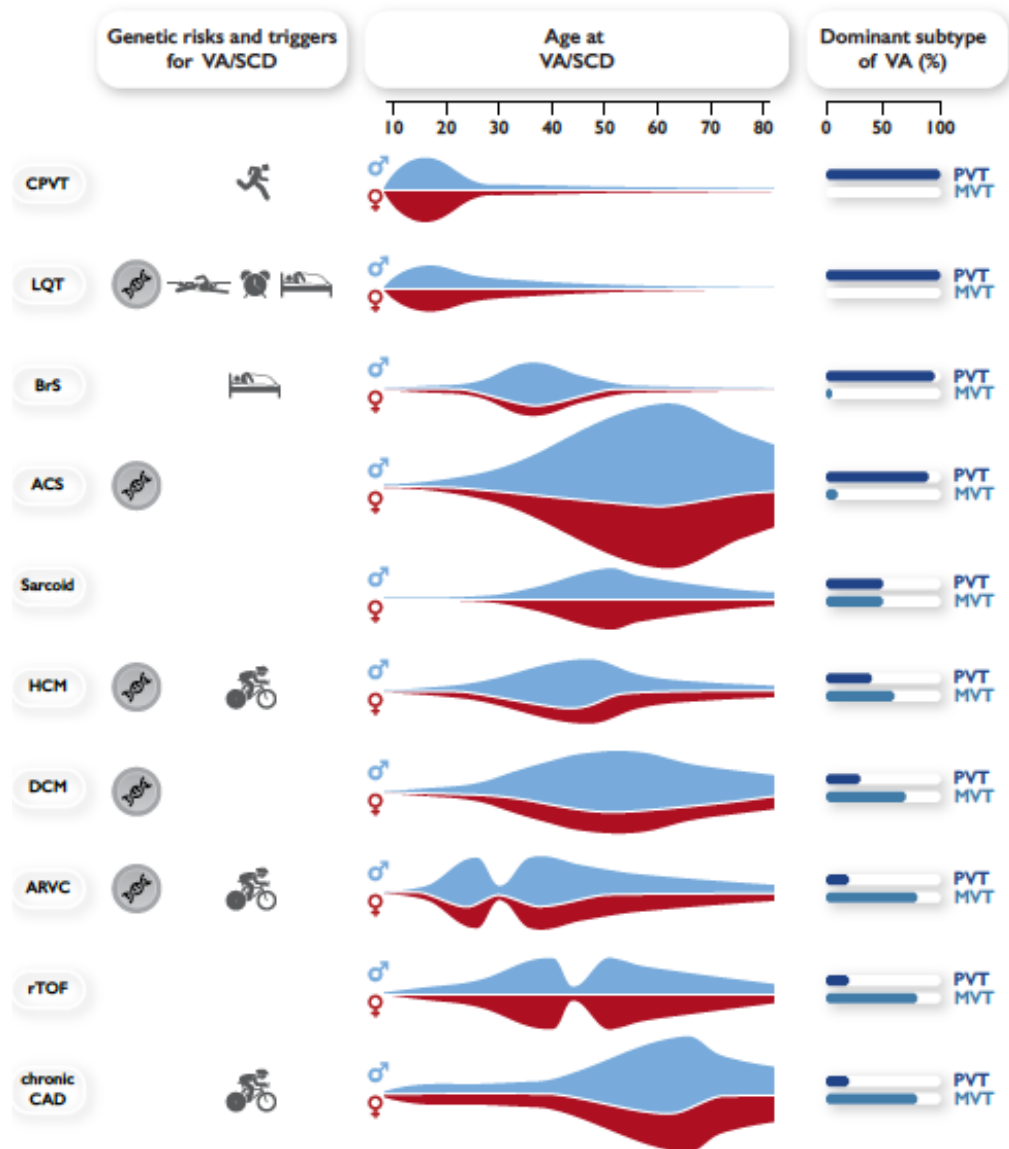


Figure 2. ESC 2022 GL on VA and SCD. Genetic risk for VA/SCD, typical triggers for VA/SCD, age at presentation with VA/SCD, sex predominance, and typical VA (PVT/VF vs. MVT) in different diseases associated with VA/SCD.

ACS, acute coronary syndrome; ARVC, arrhythmogenic right ventricular cardiomyopathy; BrS, Brugada syndrome; CAD, coronary artery disease; CPVT, catecholaminergic polymorphic ventricular tachycardia; DCM, dilated cardiomyopathy; HCM, hypertrophic cardiomyopathy; LQT, long QT syndrome; MVT, monomorphic ventricular tachycardia; PVT, polymorphic ventricular tachycardia; rTOF, repaired tetralogy of Fallot; SCD, sudden cardiac death; VA, ventricular arrhythmia; VF, ventricular fibrillation.

Table 1. ESC 2022 GL on VA and SCD. Recommendations for public basic life support and access to automated external defibrillators

Public access defibrillation should be available at sites where cardiac arrest is more likely to occur.*	I B
Prompt CPR by bystanders at OHCA.	I B
Promote community training in basic life support to increase bystander CPR rate and AED use.	I B
Mobile phone-based alerting of basic life support-trained bystander volunteers to assist nearby OHCA victims.	IIa B

*Shopping centres, stadiums, public transport stations, casinos.

Table 2. Management of Cardiac Arrest**ACC/AHA/HRS 2017 GL on VA/SCD. Recommendations for Management of Cardiac Arrest**

CPR should be performed according to published basic and advanced cardiovascular life support algorithms.	I-A
IV amiodarone in hemodynamically unstable VA that persist or recur after a maximal energy shock.	I-A
Direct current cardioversion for VA with hemodynamic instability.	I-A
Angiography with emergency revascularization in polymorphic VT or VF with ST-elevation MI,	I-B-NR
A wide-QRS tachycardia should be presumed to be VT if the diagnosis is unclear.	I C-EO
IV procainamide in hemodynamically stable VT.	IIa-A
IV lidocaine in witnessed cardiac arrest due to VF or polymorphic VT that is unresponsive to CPR, defibrillation, and vasopressor therapy.	IIa-B-R
IV beta blockers in polymorphic VT due to myocardial ischemia	IIa-B-R
IV IV beta blocker in recent MI with VT/VF that repeatedly recurs despite direct current cardioversion and antiarrhythmic medications (VT/VF storm).	IIa-B-NR
In cardiac arrest, epinephrine (1 mg every 3 to 5min) during CPR	IIb-A
IV amiodarone or sotalol in hemodynamically stable VT	IIb-B-R
In cardiac arrest, high-dose epinephrine (>1mg boluses) are not beneficial	III-A No benefit
In refractory VF not related to torsades de pointes, IV magnesium is not beneficial.	III-A No benefit
In suspected AMI, prophylactic administration of lidocaine or high-dose amiodarone for the prevention of VT is potentially harmful	III-B-R Harm
In wide QRS complex tachycardia of unknown origin, calcium	III-C-LD Harm

AHA 2025 GL on CPR. Adult Basic Life Support

Recognition of Cardiac Arrest

1	C-LD	1. If an adult is unconscious/unresponsive, with absent or abnormal breathing (ie, only gasping), the lay rescuer should assume the person is in cardiac arrest.
1	C-LD	2. If an adult is unconscious/unresponsive, with absent or abnormal breathing (ie, only gasping), the health care professional should check for a pulse for no more than 10 s and, if no definite pulse is felt, should assume the person is in cardiac arrest.

Initiation of Resuscitation: Lay Rescuer (Untrained or Trained)

1	B-NR	1. All lay rescuers should provide chest compressions for adults with presumed cardiac arrest.
1	B-NR	2. After identifying an adult in cardiac arrest, a lone responder should activate the emergency response system first, then immediately begin CPR, beginning with chest compressions.
1	C-LD	3. For adults in cardiac arrest, a lone rescuer with a mobile phone should activate the emergency response system and immediately begin CPR, beginning with chest compressions.
2a	B-R	4. For lay rescuers trained in performing CPR, it is reasonable to provide ventilation (breaths) in addition to chest compressions for an adult in cardiac arrest.
2a	C-LD	5. When immediately available, it is reasonable for rescuers to use PPE during CPR for adult cardiac arrest.

Initiation of Resuscitation: Health Care Professional

1	C-LD	1. During adult cardiac arrest, a lone health care professional should commence with chest compressions rather than with ventilation.
2a	C-LD	2. It is reasonable for health care professionals to perform chest compressions and ventilation for all adult patients in cardiac arrest from either a cardiac or noncardiac cause.

Opening the Airway in Adults in Cardiac Arrest

1	C-EO	1. For unresponsive adult patients, health care professionals and trained lay rescuers should open the airway by using a head tilt–chin lift maneuver when no cervical spine injury is suspected.
2a	C-LD	2. When managing the airway of an adult with known or suspected basal skull fracture or severe coagulopathy, an oropharyngeal airway is preferred compared with a nasopharyngeal airway.
2b	C-EO	3. The use of an airway adjunct (eg, oropharyngeal or nasopharyngeal airway) may be reasonable in unconscious (unresponsive) adult patients with no cough or gag reflex to facilitate delivery of ventilation.
3: No Benefit	C-LD	4. The routine use of cricoid pressure in adult cardiac arrest is not recommended.

Opening the Airway After Head and Neck Trauma

1	C-EO	1. In cases of head and neck trauma, trained rescuers should open the airway of an adult by using a jaw thrust without head extension.
1	C-EO	2. For an adult with head and neck trauma, if the airway cannot be opened with a jaw thrust and airway adjunct insertion, trained rescuers should open the airway by using a head tilt–chin lift.
3: Harm	C-LD	3. In the setting of an adult with head and neck trauma, lay rescuers should not use rigid cervical devices for spinal motion restriction because their use by untrained rescuers may be harmful.

Position and Location for CPR

COR	LOE	Recommendations
1	B-NR	1. In adult cardiac arrest, rescuers should perform chest compressions with the patient's torso at approximately the level of the rescuer's knees whenever possible.
1	C-LD	2. When providing chest compressions for an adult, the rescuer should place the heel of one hand on the center (middle) of the person's chest (the lower half to lower third of the sternum) and the heel of the other hand on top of the first so that the hands are overlapped.
1	C-EO	3. In adult cardiac arrest, resuscitation should generally be conducted where the person is found, as long as high-quality CPR can be administered safely and effectively.
2a	C-LD	4. In adult cardiac arrest, it is preferred to perform CPR on a firm surface and with the person in the supine position, when feasible and it does not delay chest compressions.
2b	C-LD	5. In adult cardiac arrest, rescuers may consider placing their dominant hand on the sternum when performing chest compressions.
2b	C-LD	6. For adult patients who experience cardiac arrest while in the prone position, it may be reasonable to turn the patient supine before initiating chest compressions. If the patient cannot be safely turned supine, rescuers may consider performing CPR in the prone position.

Chest Compression Fraction and Pauses

1	C-LD	1. In adult cardiac arrest, preshock and postshock pauses in chest compressions should be as short as possible.
1	C-LD	2. During rhythm checks for adult cardiac arrest, the health care professional should minimize the time taken to check for a pulse (no more than 10 s), and if the rescuer does not definitely feel a pulse, chest compressions should be restarted immediately.
2a	B-R	3. In adults with cardiac arrest, it is reasonable to immediately resume chest compressions after shock administration rather than pause CPR to perform a postshock rhythm check.
2a	B-R	4. When 2 or more rescuers are available during an adult cardiac arrest, it is reasonable to switch chest compressors approximately every 2 min (or after about 5 cycles of compressions and ventilation at a ratio of 30:2).
2a	C-LD	5. For adults in cardiac arrest receiving CPR without an advanced airway, it is reasonable to pause compressions to deliver 2 breaths, each given over 1 s.
2b	C-LD	6. During adult cardiac arrest, it may be reasonable to perform CPR with a chest compression fraction of at least 60%.
2b	C-LD	7. Outside of the advanced life support environment, where invasive monitoring is available, there are insufficient data about the value of a pulse check while performing CPR for adult cardiac arrest.

Chest Compression Depth and Rate in Adult Cardiac Arrest

1	B-NR	1. During manual CPR, rescuers should perform chest compressions to a depth of at least 2 inches, or 5 cm, for an average adult while avoiding excessive chest compression depths (>2.4 inches, or 6 cm)
2a	B-R	2. During adult cardiac arrest, it is reasonable to use audiovisual feedback devices for real-time optimization of CPR performance.
2a	B-NR	3. For adults in cardiac arrest, it is reasonable for rescuers to perform chest compressions at a rate of 100 to 120/min.
2a	C-LD	4. It can be beneficial for rescuers to allow complete chest wall recoil for adults in cardiac arrest, such as by not leaning on the chest between compressions.
2b	C-EO	5. During adult cardiac arrest, it may be reasonable to perform chest compressions so that chest compression and recoil/relaxation times are approximately equal.

Fundamentals of Ventilation During Adult Cardiac Arrest

2a	C-LD	1. In adult patients in cardiac arrest without an advanced airway, it is reasonable to deliver breaths either by mouth-to-mouth, mouth-to-mask, or bag-mask ventilation.
2a	C-LD	2. When ventilating an adult patient in cardiac arrest, it is reasonable to give enough tidal volume to produce visible chest rise.
2a	C-LD	3. When delivering ventilations by bag-mask device for an adult in cardiac arrest, it is reasonable for 1 rescuer to use 2 hands to open the airway and seal the mask to the face while a second rescuer squeezes the bag.
2b	C-LD	4. When providing breaths to an adult in cardiac arrest without an advanced airway, it may be reasonable to give each breath over 1 s.
3: Harm	C-LD	5. When providing breaths to adult patients in cardiac arrest, rescuers should avoid hypoventilation (too few breaths or too little volume) or hyperventilation (too many breaths or too large a volume).

Ventilation During Adult Cardiac Arrest: Special Circumstances

2a	C-LD	1. It is reasonable for a rescuer to use mouth-to-nose ventilation if providing breaths through the adult's mouth is impossible or impractical.
2b	C-EO	2. For an adult with a tracheal stoma who requires breaths, either mouth-to-stoma or face mask-to-stoma ventilation may be reasonable.

Ventilation of Adults With Spontaneous Circulation (Respiratory Arrest)

1	C-LD	1. For an adult with spontaneous circulation (ie, strong and easily palpable pulses) and respiratory arrest, the health care professional should provide ventilations.
2a	C-LD	2. When providing ventilations for an adult patient with respiratory arrest, it is reasonable for the health care professional to give 1 ventilation every 6 s, or 10 breaths per minute, with each ventilation creating visible chest rise.

Compression-to-Ventilation Ratio in Adult Cardiac Arrest

2a	B-NR	1. It is reasonable for lay rescuers and health care professionals to perform CPR with cycles of 30 compressions followed by 2 breaths before placement of an advanced airway (supraglottic airway or endotracheal tube).
2b	C-LD	2. For adults in cardiac arrest without an advanced airway, health care professionals may consider the use of continuous chest compressions with asynchronous breaths.

Pads for Defibrillation in Adult Cardiac Arrest

2a	C-LD	1. It is reasonable to place defibrillation paddles or pads on the exposed chest in an anterolateral or anteroposterior position, and to use a paddle or pad electrode diameter more than 8 cm for adults in cardiac arrest.
2b	C-EO	2. When placing pads for defibrillation for an adult in cardiac arrest, it might be reasonable to adjust the position of a bra instead of removing it.

CPR Before Defibrillation in Adult Cardiac Arrest

1	C-LD	1. For adults in cardiac arrest, CPR is recommended until a defibrillator or AED is applied.
2a	B-R	2. In unmonitored adult cardiac arrest, it is reasonable to provide a brief period of CPR while a defibrillator is being obtained and readied for use.
2a	C-LD	3. For adults in cardiac arrest, immediate defibrillation is reasonable for witnessed or monitored VF/pulseless ventricular tachycardia when a defibrillator is already applied or immediately available.

Foreign Body Airway Obstruction (FBAO) in Conscious Adult Patients

1	B-NR	1. For adults with severe FBAO, repeated cycles of 5 back blows (slaps) followed by 5 abdominal thrusts should be performed until the object is expelled or the person becomes unresponsive.
1	C-EO	2. Rescuers should activate the emergency response system for adults with severe FBAO.
1	C-EO	3. For adults with mild FBAO, the person should be allowed to clear the airway by coughing while being observed for signs of severe airway obstruction.

Relief of FBAO in Unconscious Adult Patients

1	C-LD	1. If adults with severe FBAO become unresponsive, rescuers should start CPR, beginning with chest compressions, and activate the emergency response system if no one has done so.
1	C-EO	2. For adults with FBAO receiving CPR, rescuers should remove any visible foreign body when opening the airway to provide breaths.
3: Harm	C-LD	3. Blind finger sweeps should not be performed for adults with FBAO.

Relief of FBAO in Special Circumstances

1	C-EO	1. If the rescuer is unable to encircle the person's abdomen, repeated cycles of 5 back blows (slaps) followed by 5 chest thrusts should be used for adults with severe FBAO until the object is expelled or the person becomes unresponsive.
1	C-EO	2. For adults in the late stages of pregnancy with severe FBAO, repeated cycles of 5 back blows (slaps) followed by 5 chest thrusts should be used until the object is expelled or the person becomes unresponsive.
2b	C-LD	3. Effectiveness and safety of suction-based airway clearance devices have not been established in adults with FBAO. There is insufficient evidence to make a recommendation.

Cardiac Arrest in Adult Patients With Obesity

1	C-LD	1. CPR for adults with obesity who are in cardiac arrest should be provided by using the same techniques as for the nonobese patient.
2a	C-LD	2. In adults with obesity who are in cardiac arrest, it is reasonable for rescuers to consider the impact of moving the patient to a firm surface on delays in initiation of chest compressions.
2b	C-EO	3. It may be reasonable for rescuers to increase the force of compressions to achieve adequate depth for adult patients with obesity who are in cardiac arrest.
3: No Benefit	C-LD	4. For adults with obesity, the use of a partial back-board (or "CPR board") during IHCA does not provide benefit over performing chest compressions on a hospital mattress.

Alternative Techniques for CPR in Adult Cardiac Arrest

2b	C-LD	1. In adult cardiac arrest, the use of mechanical CPR devices may be considered in specific settings where the delivery of high-quality manual compressions may be challenging or dangerous for the health care professional, as long as rescuers strictly limit interruptions in CPR during deployment and removal of the device.
2b	C-LD	2. For single rescuers, performing compressions from "over-the-head" of the patient may be considered in adult cardiac arrest.
2b	C-LD	3. The effectiveness of heel/foot compressions is not well established in adult cardiac arrest.
3: No Benefit	B-R	4. The routine use of mechanical CPR devices is not recommended in adult cardiac arrest.

AHA 2025 GL on CPR. Adult Advanced Life Support

Defibrillation

1	B-NR	1. In adult patients with cardiac arrest, defibrillators (using biphasic or monophasic waveforms) are recommended to treat tachyarrhythmias requiring a shock such as ventricular fibrillation or pulseless ventricular tachycardia.
2a	B-R	2. Biphasic waveform defibrillation is preferred over monophasic waveform defibrillation for treatment of tachyarrhythmias in adult patients with cardiac arrest.
2a	B-NR	3. A single shock strategy is preferred to stacked shocks for defibrillation in adult patients with cardiac arrest.
2a	C-LD	4. It is reasonable that selection of fixed versus escalating energy levels for subsequent shocks for adults in cardiac arrest with presumed shock-refractory arrhythmias be based on the specific manufacturer's instructions for that waveform.
2b	B-R	5. If using a defibrillator capable of escalating energies, higher energy for second and subsequent shocks may be considered if the initial shock fails to restore a perfusing rhythm in adult patients with cardiac arrest.
2b	C-LD	6. In the absence of conclusive evidence that one biphasic waveform is superior to another in termination of ventricular fibrillation, it may be reasonable to use the manufacturer's recommended energy dose for the first shock in adult patients with cardiac arrest. If this is not known, defibrillation at the maximal dose may be considered.

Ancillary Waveform Technology

COR	LOE	Recommendations
2b	C-LD	1. The effectiveness of artifact-filtering algorithms for analysis of electrocardiogram rhythms during chest compressions for adults in cardiac arrest has not been established.
2b	C-LD	2. The effectiveness of VF waveform analysis to guide the acute management of adults with cardiac arrest has not been established.

Vector Change and Double Sequential Defibrillation

2b	B-R	1. The usefulness of vector change for adults in cardiac arrest with persisting VF/pVT after ≥ 3 consecutive shocks has not been established.
2b	B-R	2. The usefulness of double sequential defibrillation for adults in cardiac arrest with persisting VF/pVT after ≥ 3 consecutive shocks has not been established.

Nondefibrillation Electrical Therapies for Cardiac Arrest

3: No Benefit	B-R	1. Routine use of electrical pacing is not recommended during the resuscitation of an established adult cardiac arrest.
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Vascular Access

1	A	1. It is recommended that professionals first attempt establishing intravenous access for drug administration in adult patients in cardiac arrest.
2a	A	2. Intraosseous access is reasonable if initial attempts at intravenous access are unsuccessful or not feasible for adult patients in cardiac arrest.
2b	C-LD	3. For appropriately trained professionals, central venous access may be considered for adult patients in cardiac arrest if attempts to establish intravenous and intraosseous access are unsuccessful or not feasible.

Vasopressor Medications

1	B-R	1. Administration of epinephrine is recommended for adult patients in cardiac arrest.
2a	B-R	2. It is reasonable to administer epinephrine (1 mg) every 3 to 5 min for adult patients in cardiac arrest.
2a	B-NR	3. With respect to timing, for adult patients in cardiac arrest with a nonshockable rhythm, it is reasonable to administer epinephrine as soon as feasible.
2a	B-NR	4. With respect to timing, for adult patients in cardiac arrest with a shockable rhythm, it is reasonable to administer epinephrine after initial defibrillation attempts have failed.
3: No Benefit	B-R	5. Vasopressin alone or vasopressin in combination with epinephrine offers no advantage as a substitute for epinephrine for adult patients in cardiac arrest.
3: No Benefit	B-R	6. High-dose epinephrine is not recommended for routine use for adult patients in cardiac arrest.

Nonvasopressor Medications

COR	LOE	Recommendations
2b	B-R	1. For adults in cardiac arrest amiodarone or lidocaine may be considered for ventricular fibrillation (VF) or pulseless ventricular tachycardia (pVT) that is unresponsive to defibrillation.
2b	C-LD	2. For adults in cardiac arrest, the use of β -blockers, bretylium, procainamide, or sotalol for VF/pVT unresponsive to defibrillation is of uncertain benefit.
2b	C-LD	3. For adults in cardiac arrest, the use of steroids is of uncertain benefit.
3: No Benefit	B-R	4. For adults in cardiac arrest, routine administration of calcium is not recommended.
3: No Benefit	B-R	5. For adults in cardiac arrest, routine administration of sodium bicarbonate is not recommended.
3: No Benefit	B-R	6. For adults in cardiac arrest, routine administration of magnesium is not recommended.

Adjuncts to CPR

2b	B-NR	1. An abrupt increase in end-tidal CO ₂ may be used to detect ROSC during chest compressions in adults in cardiac arrest or when a rhythm check reveals an organized rhythm.
2b	C-LD	2. Use of point-of-care ultrasonography during adult cardiac arrest resuscitation to diagnose reversible causes is not well established.
2b	C-LD	3. Use of point-of-care echocardiography to assess cardiac function for prognostication during adult cardiac arrest resuscitation is not well established.
2b	C-LD	4. When supplemental oxygen is available, it may be reasonable to use the maximal feasible inspired oxygen concentration during CPR for adults with cardiac arrest.
2b	C-LD	5. Measurement of arterial blood gases during CPR has uncertain benefit in adults with cardiac arrest.
2b	C-LD	6. In adult patients with cardiac arrest, it may be reasonable to use physiological parameters such as arterial blood pressure or end-tidal CO ₂ when feasible to monitor and optimize CPR quality.
2b	C-EO	7. Arterial pressure monitoring by arterial line may be used to detect ROSC during chest compressions or when a rhythm check reveals an organized rhythm in adults with cardiac arrest.
3: No Benefit	C-LD	8. Head-up cardiopulmonary resuscitation in adults with cardiac arrest is not recommended except in setting of clinical trials.

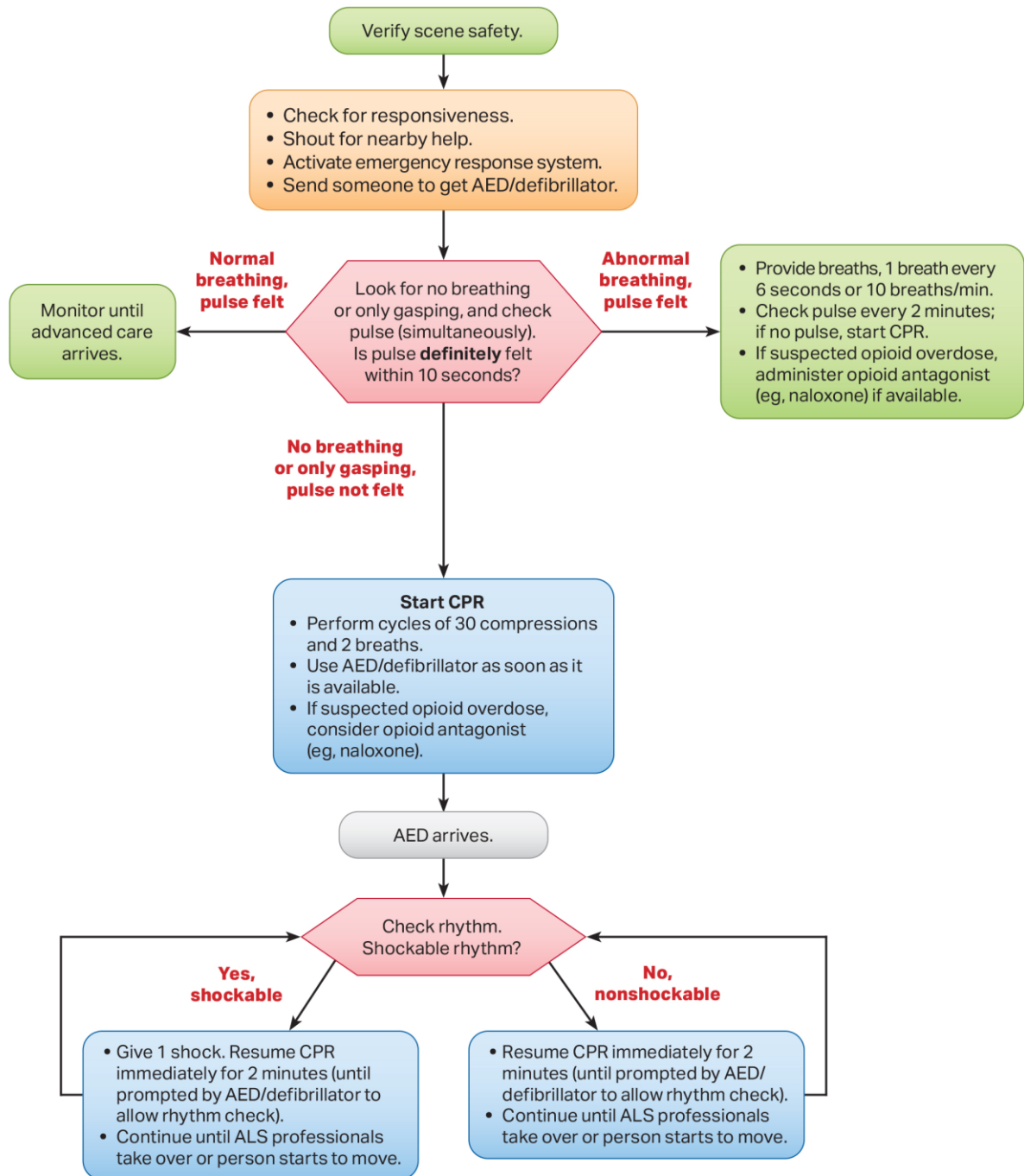
Termination of CPR

1	B-NR	1. When TOR is being considered, BLS prehospital professionals should use the BLS TOR rule for adult patients in OHCA, where ALS is not available or may be significantly delayed.
2a	B-NR	2. It is reasonable for ALS prehospital professionals to use the ALS TOR rule to terminate resuscitation efforts in the field for adult patients with OHCA.
2a	B-NR	3. In a tiered EMS system with both ALS and BLS professionals, it is reasonable to use the Universal TOR rule for adult patients with OHCA.
2b	C-LD	4. In intubated adult patients, failure to achieve an end-tidal CO ₂ of greater than 10 mm Hg by waveform capnography after 20 minutes of ALS resuscitation may be considered as a component of a multimodal approach to decide when to end resuscitative efforts.
3: Harm	C-EO	5. In nonintubated adult patients, a specific end-tidal CO ₂ cutoff value at any time during CPR should not be used as an indication to end resuscitative efforts.

Advanced Airway Placement During Resuscitation

1	B-NR	1. Frequent experience or frequent retraining is recommended for health care professionals who perform endotracheal intubation in adults with cardiac arrest.
1	C-LD	2. If advanced airway placement will interrupt chest compressions, health care professionals should defer insertion of the airway until the adult in cardiac arrest fails to respond to initial CPR and defibrillation attempts or obtains ROSC.
1	C-LD	3. Continuous waveform capnography is recommended in addition to clinical assessment as the most reliable method of confirming and monitoring correct placement of an endotracheal tube in adults with cardiac arrest.

1	C-EO	4. Emergency medical services systems that perform prehospital intubation in adults with cardiac arrest should provide a program of ongoing quality improvement to minimize complications and track overall supraglottic airway and endotracheal tube placement success rates.
2a	B-R	5. If an advanced airway is used for adults with OHCA, a supraglottic airway can be acceptable in settings with low endotracheal intubation success rate or minimal training opportunities.
2a	B-R	6. If an advanced airway is used, either a supraglottic airway or endotracheal tube can be used for adults with OHCA in settings with high endotracheal intubation success rates or optimal training opportunities for endotracheal tube placement.
2a	B-NR	7. If an advanced airway is used for adults in cardiac arrest in the in-hospital setting by expert health care professionals trained in these procedures, either a supraglottic airway or endotracheal tube can be used.
2b	B-R	8. Either bag-mask ventilation or an advanced airway strategy may be considered during CPR for adult cardiac arrest in any setting depending on the situation and skill set of the professional.
2b	C-LD	9. If an advanced airway is in place, it may be reasonable for the clinician to deliver 1 breath every 6 s (10 breaths/min) while continuous chest compressions are being performed.

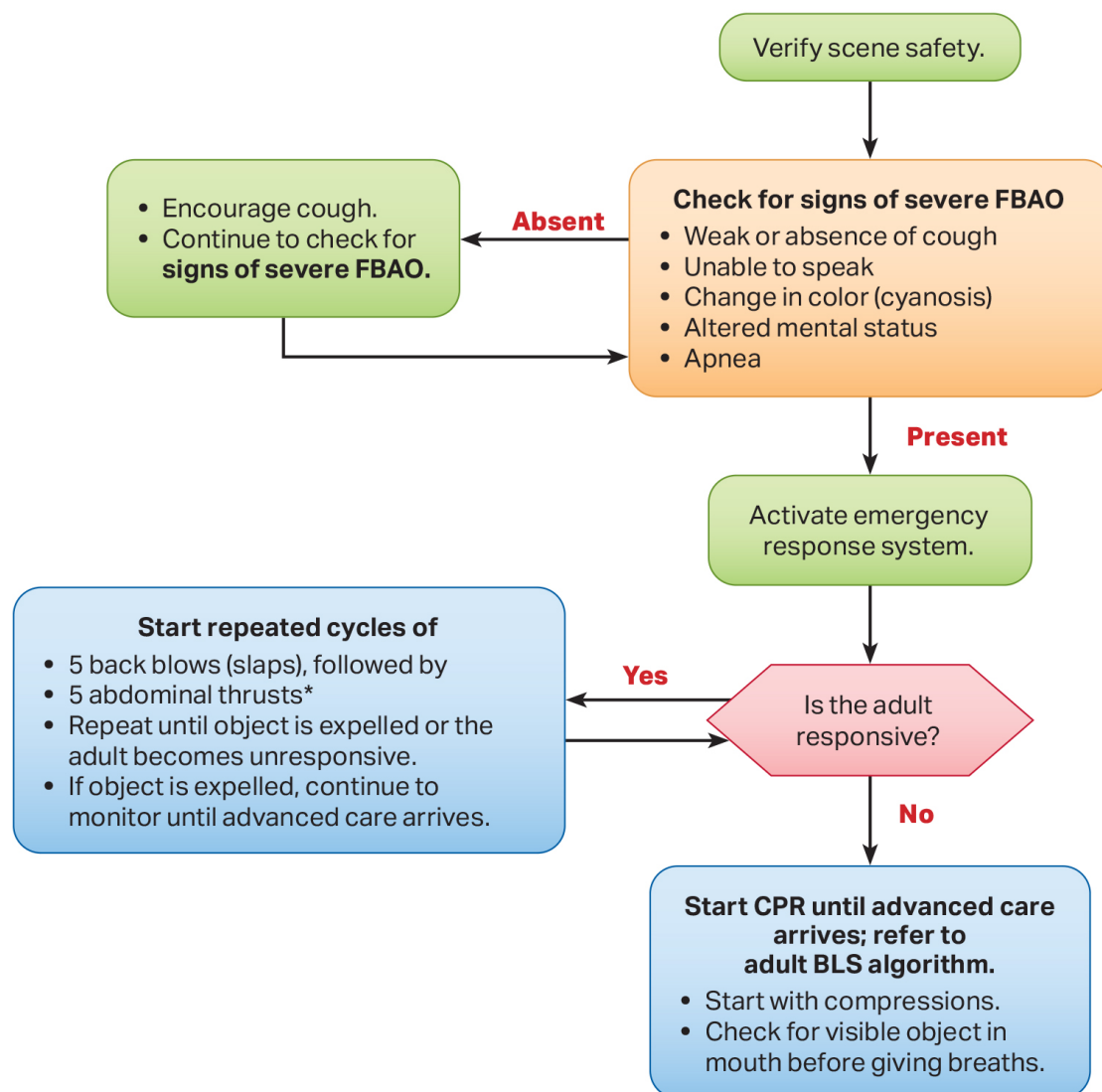


*If signs of puberty, treat as adult.

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Figure 3. AHA 2025 GL on CPR. Adult Basic Life Support Algorithm for Health Care Professionals

AED indicates automated external defibrillator; ALS, advanced life support; and CPR, cardiopulmonary resuscitation.

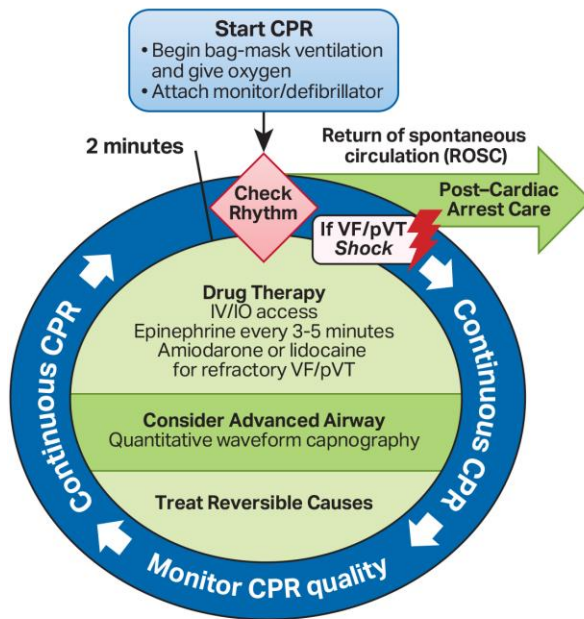


*For patients in the late stages of pregnancy, or when the rescuer is unable to encircle the patient's abdomen, 5 chest thrusts should be used instead.

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Figure 4. AHA 2025 GL on CPR. Adult Foreign-Body Airway Obstruction Algorithm.

BLS indicates basic life support; CPR, cardiopulmonary resuscitation; and FBAO, foreign-body airway obstruction.



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High-Quality CPR

- Push hard (at least 2 inches [5 cm]).
- Push fast (100-120/min) and allow complete chest recoil.
- Minimize interruptions in compressions.
- Avoid excessive ventilation.
- Change compressor every 2 minutes, or sooner if fatigued.
- If no advanced airway, 30:2 compression-ventilation ratio.
- If advanced airway in place, give 1 breath every 6 seconds (10 breaths/min) with continuous chest compressions.
- Continuous waveform capnography
 - If ETCO₂ is low or decreasing, reassess CPR quality.

Shock Energy for Defibrillation

- **Biphasic:** Manufacturer recommendation (eg, initial dose of 120-200 J); if unknown, use maximum available. Second and subsequent doses should be equivalent, and higher doses may be considered.
- **Monophasic:** 360 J

Drug Therapy

- **Epinephrine IV/IO dose:** 1 mg every 3-5 minutes
- **Amiodarone IV/IO dose:** First dose: 300 mg bolus. Second dose: 150 mg.
- or
- **Lidocaine IV/IO dose:** First dose: 1-1.5 mg/kg. Second dose: 0.5-0.75 mg/kg.

Advanced Airway

- ET intubation or supraglottic advanced airway
- Continuous waveform capnography or capnometry to confirm and monitor ET tube placement
- Once advanced airway in place, give 1 breath every 6 seconds (10 breaths/min) with continuous chest compressions

Reversible Causes

- | | |
|---------------------------|-------------------------|
| • Hypovolemia | • Tension pneumothorax |
| • Hypoxia | • Tamponade, cardiac |
| • Hydrogen ion (acidosis) | • Toxins |
| • Hypo-/hyperkalemia | • Thrombosis, pulmonary |
| • Hypothermia | • Thrombosis, coronary |

Figure 5. AHA 2025 GL on CPR. Adult Cardiac Arrest Circular Algorithm.

CPR indicates cardiopulmonary resuscitation; ET, endotracheal; IO, intraosseous; IV, intravenous; pVT, pulseless ventricular tachycardia; and VF, ventricular fibrillation.

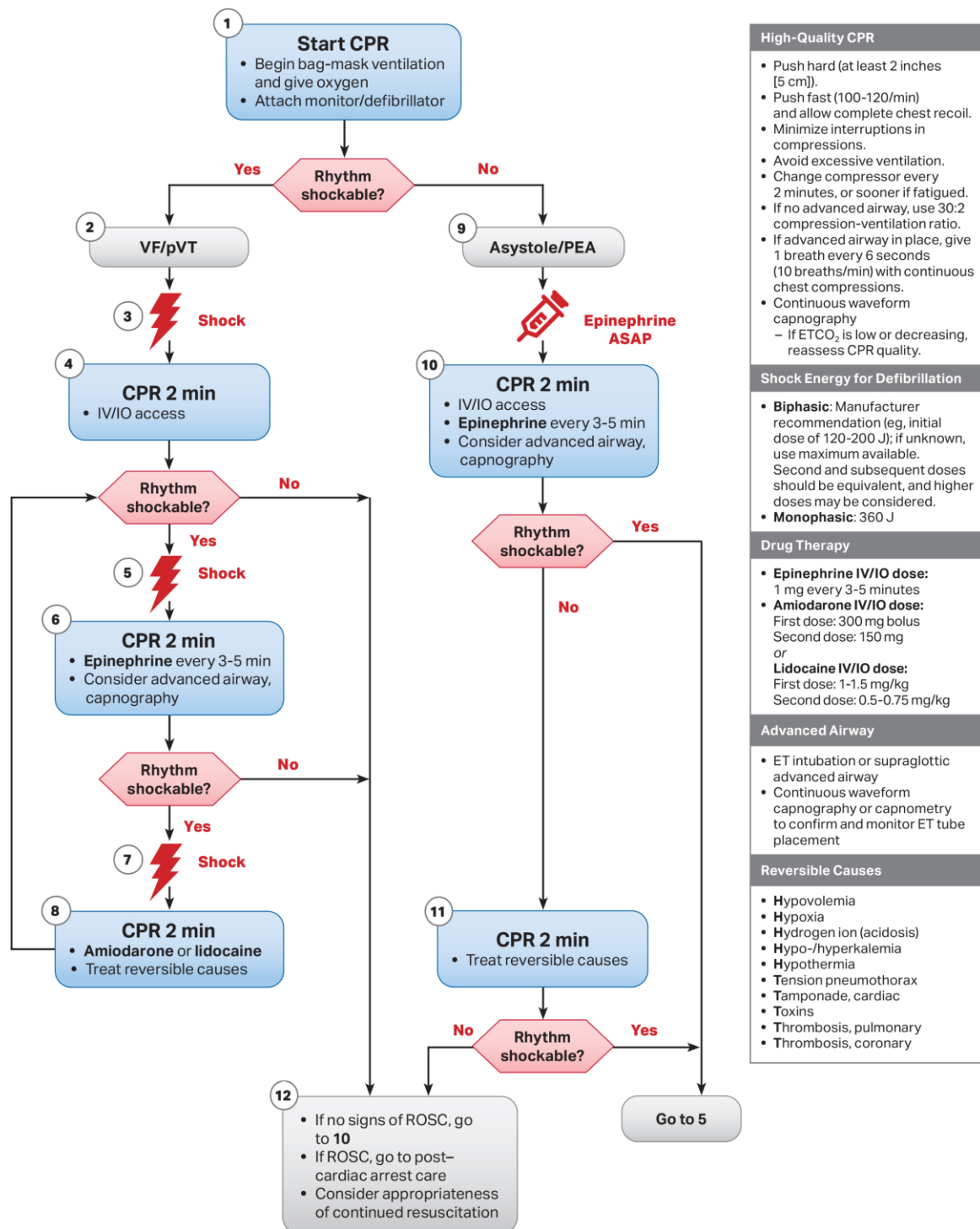


Figure 6. AHA 2025 GL on CPR. Adult Cardiac Arrest Algorithm.

CPR indicates cardiopulmonary resuscitation; ET, endotracheal; IO, intraosseous; IV, intravenous; PEA, pulseless electrical activity; pVT, pulseless ventricular tachycardia; and VF, ventricular fibrillation.

BLS Termination of Resuscitation

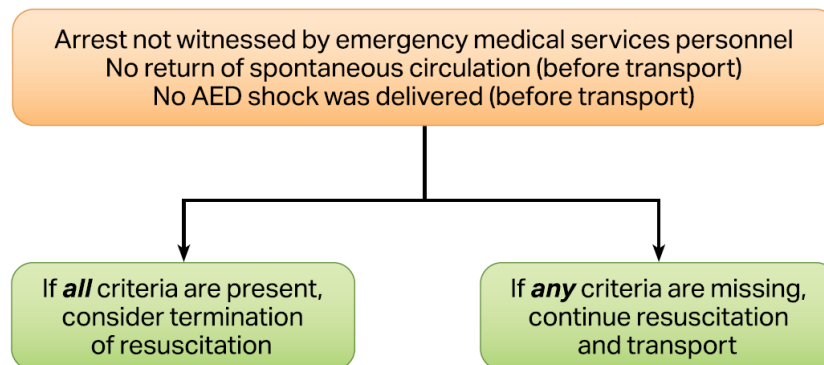
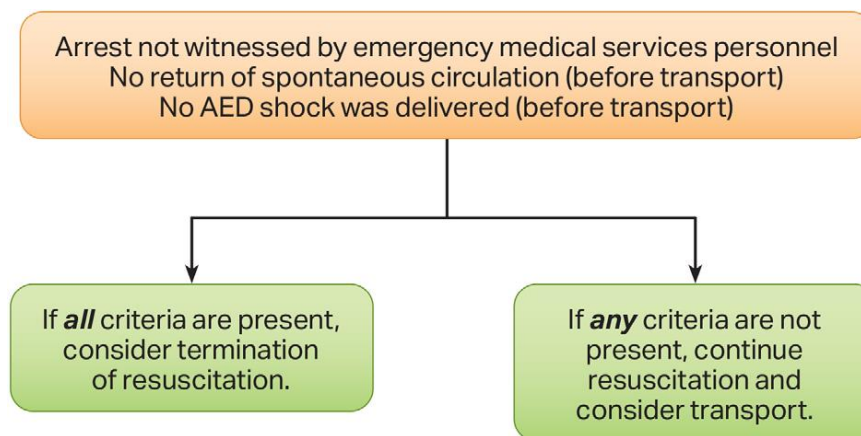


Figure 7. AHA 2025 GL on CPR. ALS Termination of Resuscitation Rule

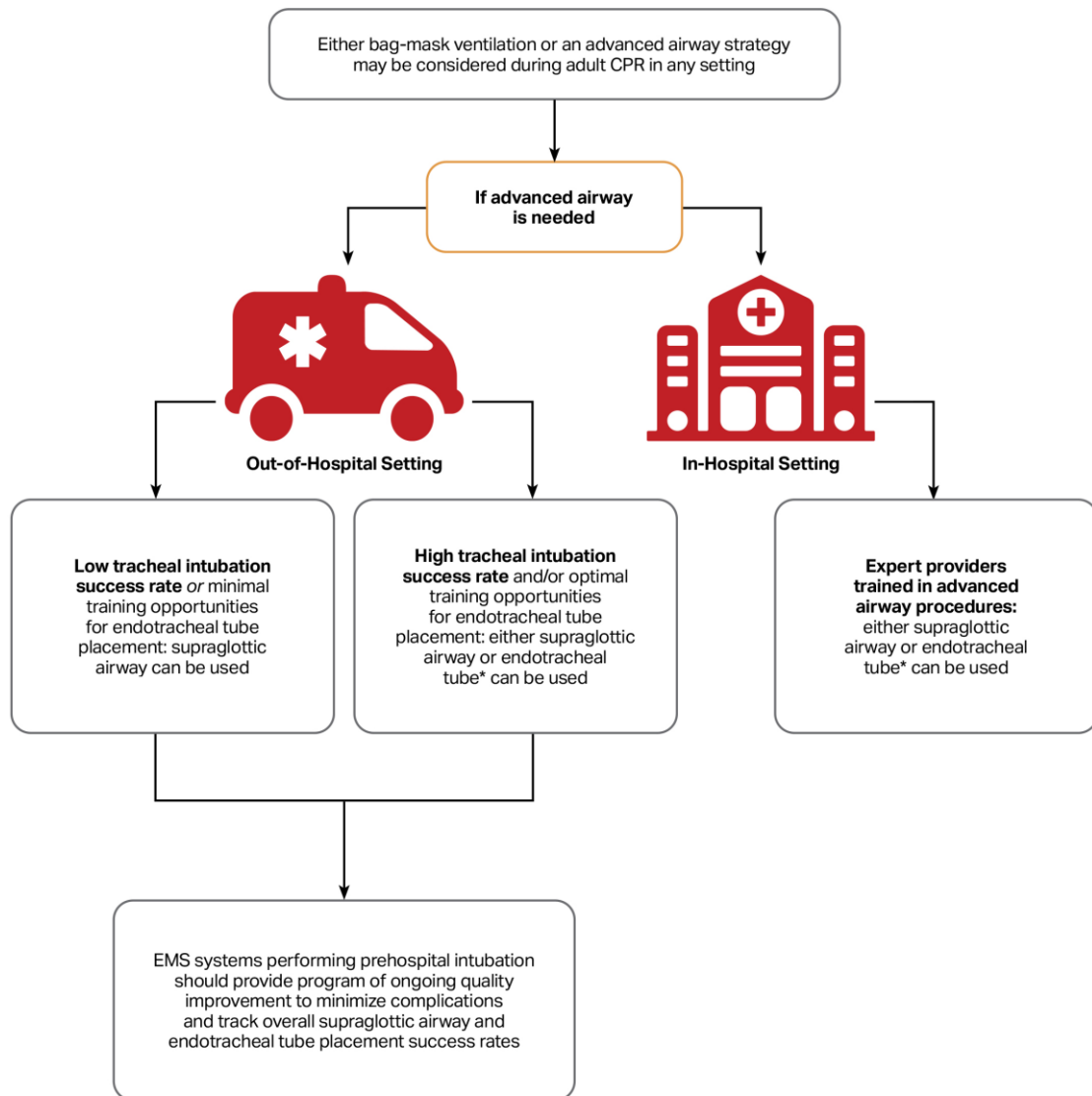
ALS indicates advanced life support; and CPR, cardiopulmonary resuscitation.



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Figure 8. AHA 2025 GL on CPR. BLS/Universal Termination of Resuscitation Rule

AED indicates automated external defibrillator; and BLS, basic life support.



*Frequent experience or frequent retraining is recommended for providers who perform endotracheal intubation.

Figure 9. AHA 2025 GL on CPR. Schematic representation of ALS recommendations for use of advanced airways during CPR.

*Frequent experience or frequent retraining is recommended for health care professionals who perform endotracheal intubation. ALS indicates advanced life support; CPR, cardiopulmonary resuscitation; and EMS, emergency medical services.

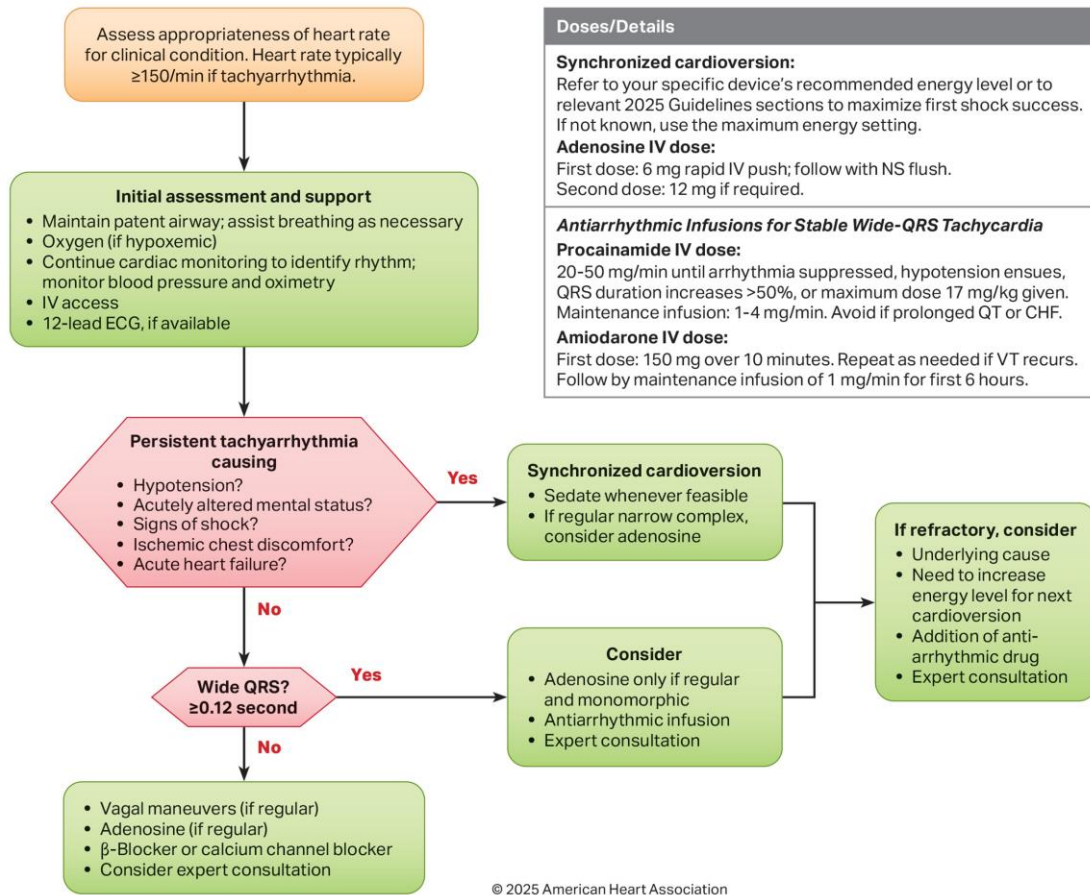
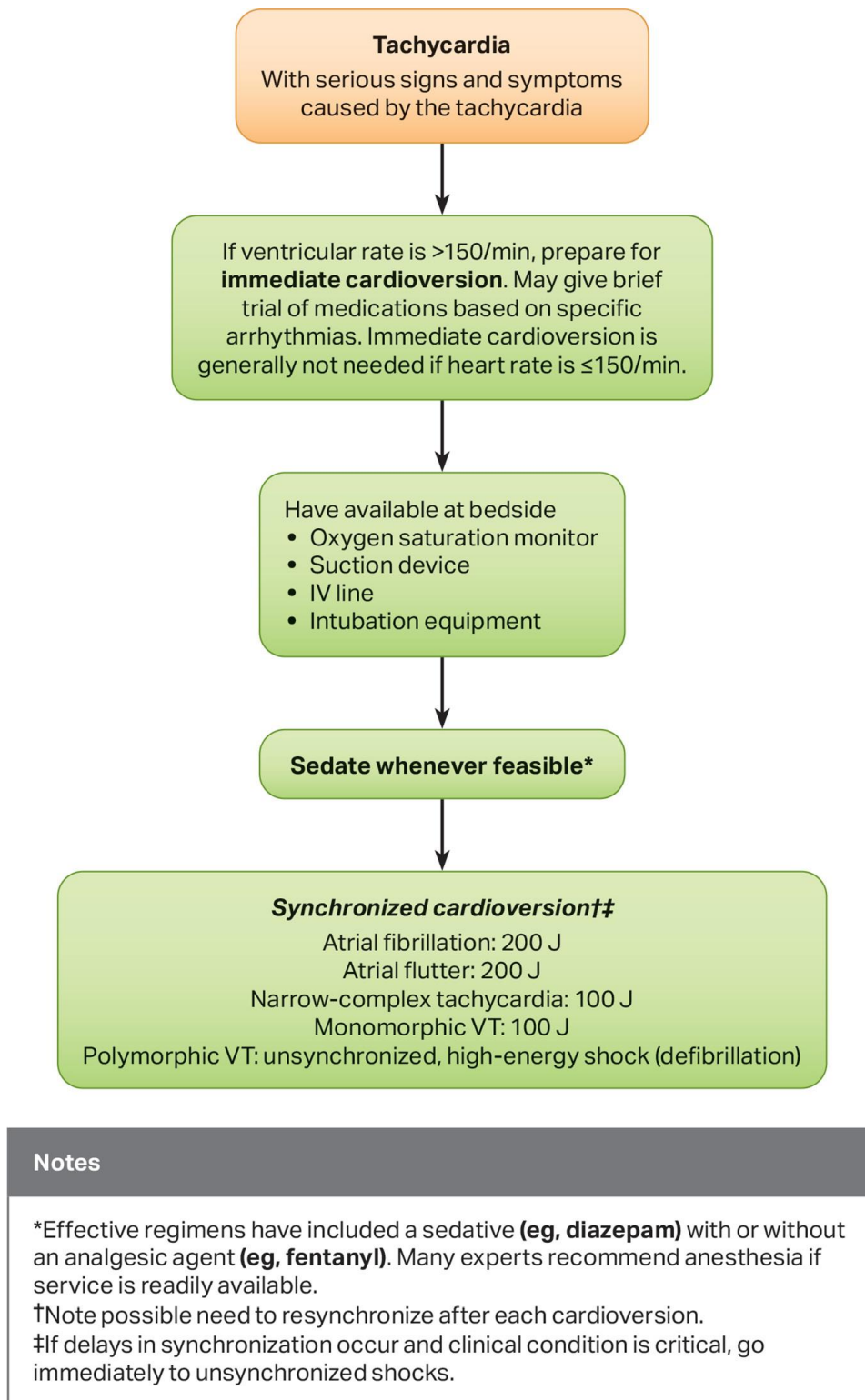


Figure 10. AHA 2025 GL on CPR. Adult Tachyarrhythmia With a Pulse Algorithm.

CHF indicates congestive heart failure; ECG, electrocardiogram; IV, intravenous; NS, normal saline; and VT, ventricular tachycardia.



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Figure 11. AHA 2025 GL on CPR. Electrical Cardioversion Algorithm.

IV indicates intravenous; and VT, ventricular tachycardia.

Table 3. Cardiac arrest Survivors**ACC/AHA/HRS 2017 GL on VA/SCD. Recommendations for evaluation of sudden cardiac arrest survivors****Diagnostic evaluation**

The investigation of a SCA survivor without obvious extra-cardiac cause to be overseen by a multidisciplinary team.	I B
Coronary angiography in electrically unstable patients after SCA, with suspicion of ongoing myocardial ischaemia.	I C
In SCA survivors, brain/chest CT scan when patient characteristics, ECG, and echocardiography are not consistent with a cardiac cause.	IIa C
In SCA survivors, collection of blood samples at presentation for potential toxicology and genetic testing.	I B
Retrieval of recordings from CIEDs and wearable monitors for all SCA survivors.	I B
In SCA survivors, repeated 12-lead ECGs during stable rhythm (including high precordial lead ECG), as well as continuous cardiac monitoring.	I B
Echocardiography for evaluation of cardiac structure and function in all SCA survivors.	I C
Coronary imaging and CMR with LGE for evaluation of cardiac structure and function in all SCA survivors without a clear underlying cause.	I B
Sodium channel blocker test and exercise testing in SCA survivors without a clear underlying cause.	I B
In SCA survivors, ergonovine, acetylcholine, or hyperventilation testing for the diagnosis of coronary vasospasm.	IIb B

CIEDs, cardiac insertable electronic devices; CMR, cardiac magnetic resonance; CT, computed tomography; ECG, electrocardiogram; LGE, late gadolinium enhancement; SCA, sudden cardiac arrest.

Table 4. ESC 2022 GL on VA and SCD. Recommendations for evaluation of sudden cardiac arrest survivors

Diagnostic evaluation

The investigation of a SCA survivor without obvious extra-cardiac cause to be overseen by a multidisciplinary team.	I B
Coronary angiogram in electrically unstable patients after SCA, with suspicion of ongoing myocardial ischaemia.	I C
In SCA survivors, brain/chest CT scan when patient characteristics, ECG, and echocardiography are not consistent with a cardiac cause.	IIa C
In SCA survivors, collection of blood samples at presentation for potential toxicology and genetic testing.	I B
Retrieval of recordings from CIEDs and wearable monitors for all SCA survivors.	I B
In SCA survivors, repeated 12-lead ECGs during stable rhythm (including high precordial lead ECG), as well as continuous cardiac monitoring.	I B
Echocardiography for evaluation of cardiac structure and function in all SCA survivors.	I C
Coronary imaging and CMR with LGE for evaluation of cardiac structure and function in all SCA survivors without a clear underlying cause.	I B
Sodium channel blocker test and exercise testing in SCA survivors without a clear underlying cause.	I B
In SCA survivors, ergonovine, acetylcholine, or hyperventilation testing for the diagnosis of coronary vasospasm.	IIb B

CIEDs, cardiac insertable electronic devices; CMR, cardiac magnetic resonance; CT, computed tomography; ECG, electrocardiogram; LGE, late gadolinium enhancement; SCA, sudden cardiac arrest.

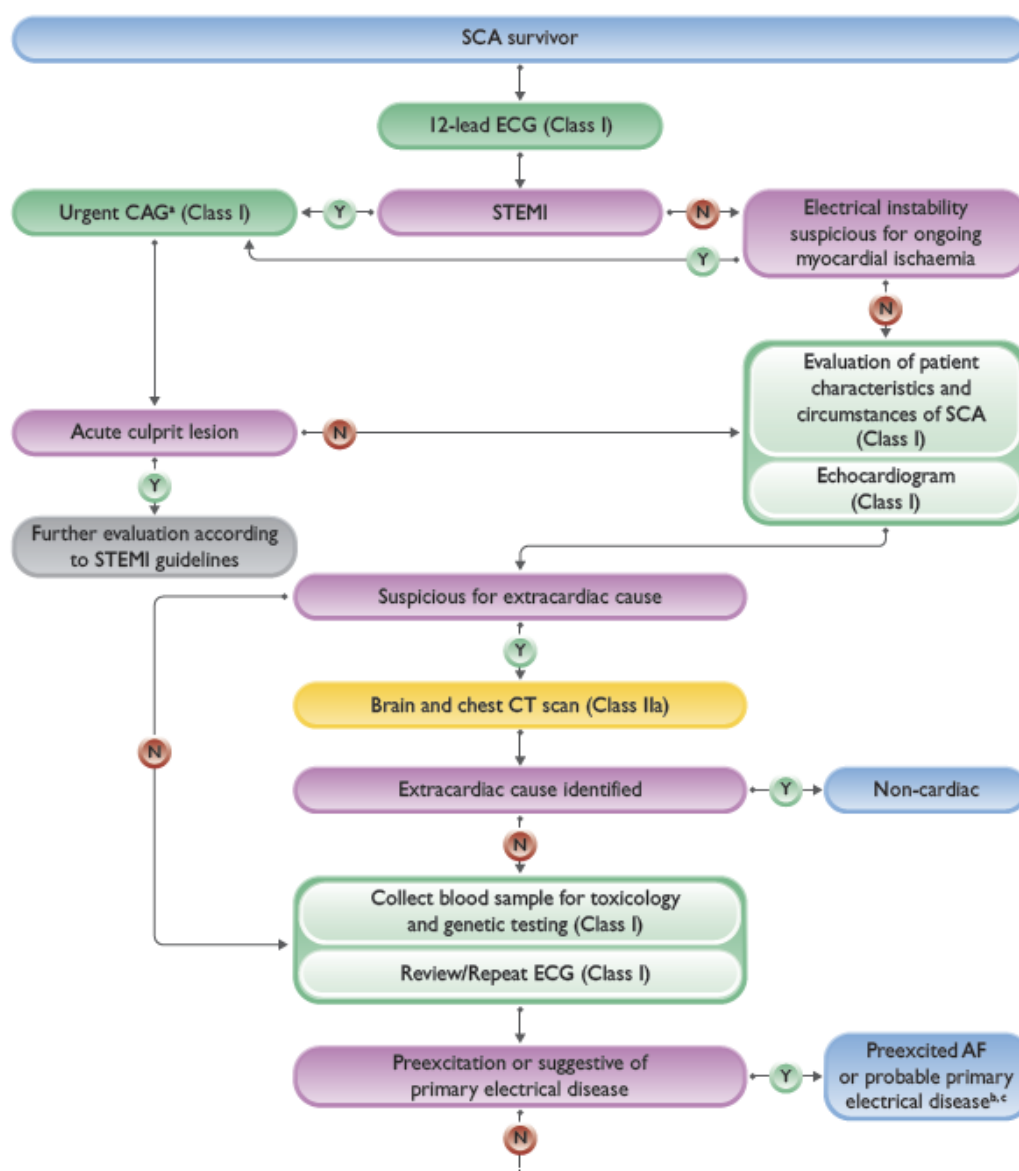


Figure 12. ESC 2022 GL on VA and SCD. Algorithm for the evaluation of sudden cardiac arrest survivors (Part1 and Part 2).

a The 2017 ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation.

b Rule out SHD according to patient age and characteristics; QT duration needs to be reassessed several days after arrest.

c Consider cardiac CT/CAG depending on patient characteristics and clinical context.

d Left ventricular function on echocardiogram needs to be reassessed several days after arrest to exclude stunning as cause of systolic dysfunction.

e In case of high clinical suspicion (typical symptoms and transient ST elevation during monitoring), it can be considered to test for coronary vasospasm earlier.

AF, atrial fibrillation; CAD, coronary artery disease; CAG, coronary angiogram; CMR, cardiac magnetic resonance; CT, computed tomography; ECG, electrocardiogram; LGE, late gadolinium enhancement; N, No; SCA, sudden cardiac arrest; SHD, structural heart disease; SMVT, sustained monomorphic ventricular tachycardia; STEMI, ST elevation myocardial infarction; VF, ventricular fibrillation; Y, Yes.

Table 5. Acute management of ventricular arrhythmias**ESC 2022 GL on VA and SCD.****Recommendations for treatment of reversible conditions**

Withdrawal of offending agents whenever drug-induced VAs are suspected.	I B
Investigation for reversible causes (e.g. electrolyte imbalances, ischaemia, hypoxaemia, fever) ^c in patients with VA.	I C
Despite a possible correctable cause for the presenting VA, consider the need for ICD implantation based on an individual evaluation of the risk of subsequent VA/ SCD.	IIa C

ICD, implantable cardioverter defibrillator; SCD, sudden cardiac death; VA, ventricular arrhythmia.

Recommendations for the acute management of sustained ventricular tachycardia

DC cardioversion for haemodynamically not-tolerated SMVT.	I B
DC cardioversion for tolerated SMVT provided that the anaesthetic/ sedation risk is low.	I C
In haemodynamically tolerated idiopathic VT, intravenous beta-blocker (RVOT VT) or verapamil (fascicular VT).	I C
In regular haemodynamically tolerated wide QRS complex tachycardia suspected for SVT, administration of adenosine or vagal manoeuvres.	IIa C
In haemodynamically tolerated SMVT and known or suspected SHD, intravenous procainamide.	IIa B
In haemodynamically tolerated SMVT in the absence of an established diagnosis, intravenous amiodarone.	IIb B
In haemodynamically tolerated SMVT in the absence of significant SHD, flecainide, ajmaline, or sotalol.	IIb C
Intravenous verapamil is not recommended in broad QRS complex tachycardia of unknown mechanism.	III B

Recommendations for the acute management of electrical storm

Mild to moderate sedation in patients with electrical storm to alleviate psychological distress and reduce sympathetic tone.	I C
Antiarrhythmic therapy with beta-blockers (non-selective preferred) in combination with intravenous amiodarone in patients with SHD and electrical storm unless contraindicated.	I B
Intravenous magnesium with supplementation of potassium in patients with TdP.	I C

Isoproterenol or transvenous pacing to increase heart rate in patients with acquired LQT syndrome and recurrent TdP despite correction of precipitating conditions and magnesium.	I C
Catheter ablation in patients presenting with incessant VT or electrical storm due to SMVT refractory to AADs.	I B
Deep sedation/intubation in patients with an intractable electrical storm refractory to drug treatment.	IIa C
Catheter ablation in patients with recurrent episodes of PVT/VF triggered by a similar PVC, non-responsive to medical treatment or coronary revascularization.	IIa C
Quinidine in patients with CAD and electrical storm due to recurrent PVT when other AAD therapy fails.	IIb C
Autonomic modulation in patients with electrical storm refractory to drug treatment and in whom catheter ablation is ineffective or not possible.	IIb C
Institution of mechanical circulatory support in the management of drug-refractory electrical storm and cardiogenic shock.	IIb C
AAD, anti-arrhythmic drug; CAD, coronary artery disease; DC, direct current; LQT, long QT; PVC, premature ventricular complex; PVT, polymorphic VT; RVOT, right ventricular outflow tract; SHD, structural heart disease; SMVT, sustained monomorphic ventricular tachycardia; SVT, supraventricular tachycardia; TdP, Torsades de pointes; VF, ventricular fibrillation; VT, ventricular tachycardia	

AHA 2025 GL on CPR. Adult Advanced Life Support

VT and VF/Wide-complex Tachycardia

1	B-NR	1. Synchronized cardioversion is recommended for acute treatment of adult patients with hemodynamically unstable wide-complex tachycardia.
1	B-NR	2. Synchronized cardioversion is recommended for acute treatment of adult patients with hemodynamically stable wide-complex tachycardia when vagal maneuvers and pharmacological therapy is ineffective or contraindicated.
2b	B-R	3. Administration of IV amiodarone, procainamide, or sotalol may be considered for the treatment of adults with wide-complex tachycardia.
2b	B-NR	4. In hemodynamically stable adult patients with regular monomorphic wide-complex tachycardia, IV adenosine may be considered for treatment or aiding rhythm diagnosis when the cause of the rhythm cannot be determined.
3: Harm	B-NR	5. Verapamil and diltiazem should not be administered for adult patients with wide-complex tachycardia.
3: Harm	C-LD	6. Adenosine should not be administered for adult patients with hemodynamically unstable, irregularly irregular, or polymorphic wide-complex tachycardia.

Polymorphic Ventricular Tachycardia

1	B-NR	1. Immediate unsynchronized shock is recommended for adults with sustained polymorphic ventricular tachycardia.
2b	C-LD	2. Magnesium may be considered for treatment of adults with recurrences of polymorphic ventricular tachycardia associated with a long QT interval (torsades de pointes).
2b	C-LD	3. IV lidocaine, amiodarone, and measures to treat myocardial ischemia may be considered to treat recurrences of polymorphic VT in adults in the absence of a prolonged QT interval.
3: No Benefit	C-LD	4. Routine use of magnesium is not recommended for the treatment of polymorphic VT in adults with a normal QT interval.

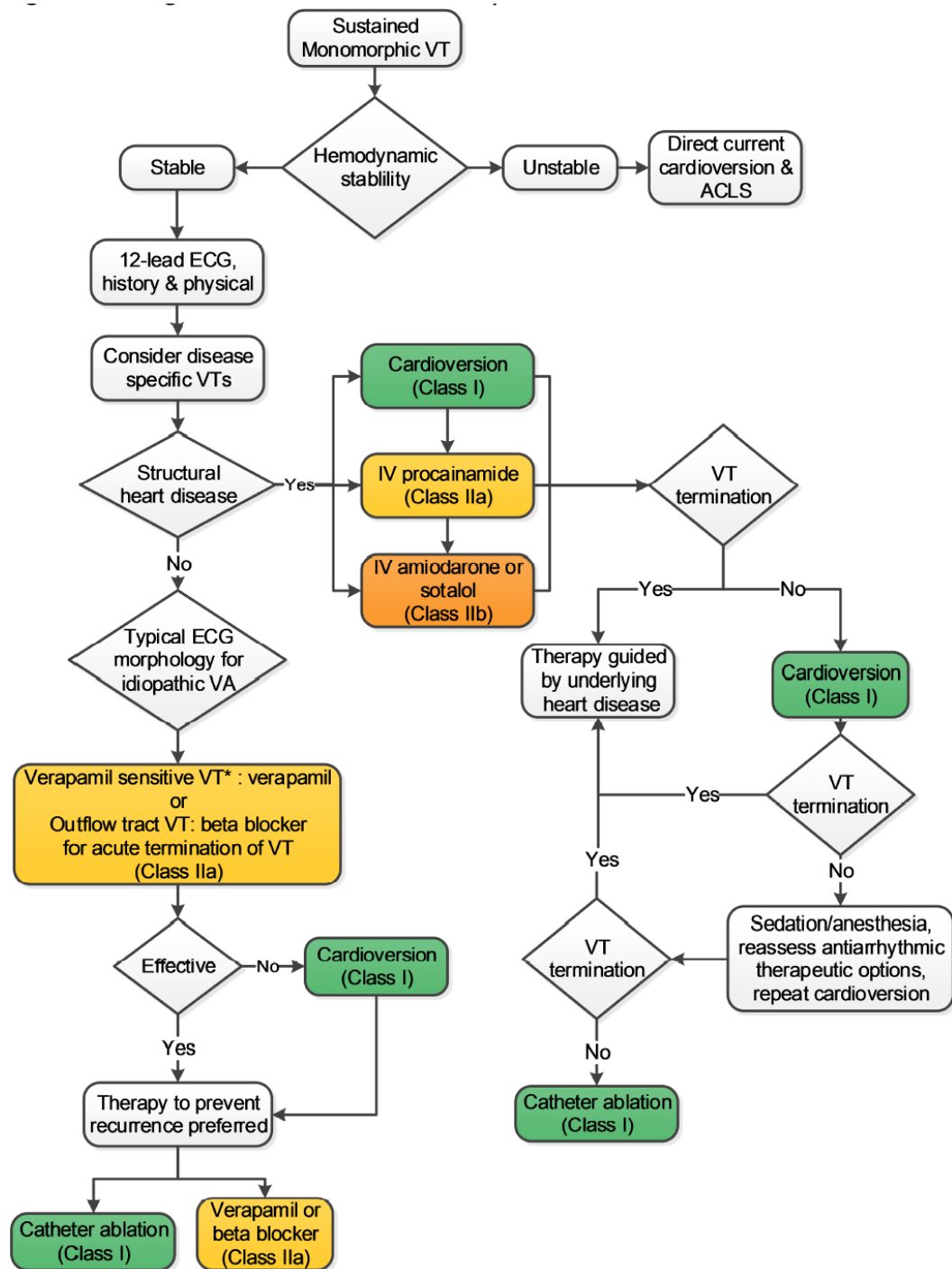


Figure 13. ACC/AHA/HRS 2017 GL on VA/SCD. Management of Sustained Monomorphic VT

*Known history of verapamil sensitive or classical electrocardiographic presentation.

ACLS indicates advanced cardiovascular life support; ECG, electrocardiogram; VA, ventricular arrhythmia; and VT, ventricular tachycardia.

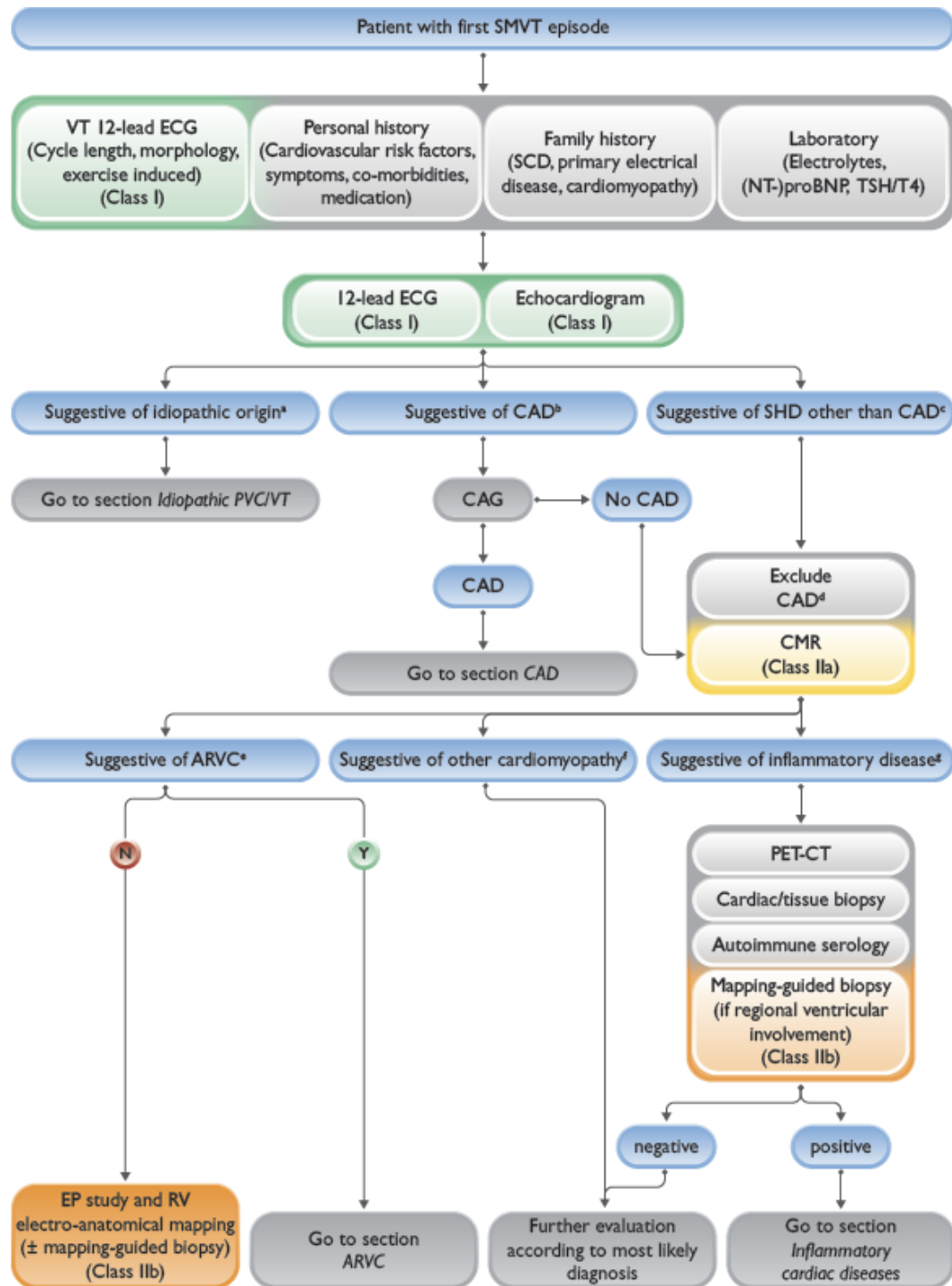


Figure 14. ESC 2022 GL on VA and SCD. Algorithm for the evaluation of patients presenting with a first sustained monomorphic ventricular tachycardia episode.

ARVC, arrhythmogenic RV cardiomyopathy; CAD, coronary artery disease; CAG, coronary angiography; CMR, cardiac magnetic resonance; ECG, electrocardiogram; EP, electrophysiological; LV, left ventricular; N, No; PET-CT, positron emission tomography and computed tomography; PVC, premature ventricular complex; RV, right ventricular; SCD, sudden cardiac death; SHD, structural

heart disease; SMVT, sustained monomorphic ventricular tachycardia; VT, ventricular tachycardia; Y, Yes.

^a ECG morphology suggestive of RV outflow tract or fascicular origin, negative family history, normal 12-lead ECG, and echocardiogram.

^b e.g. Q waves, QRS fragmentation, ST/T abnormalities, wall motion abnormalities in coronary territories.

^c e.g. atrioventricular (AV) conduction abnormalities, Q waves, broad QRS complex, T-wave inversion, abnormally high or low voltages. Ventricular dysfunction/dilatation/hypertrophy/wall thinning/wall motion abnormalities/diffuse hypokinesia. dDiagnostic test to exclude CAD according to patient profile and symptoms.

^e According to revised task force criteria.

^f e.g. AV conduction abnormalities, abnormally high or low voltages, broad QRS, ST/T wave deviations, LV dilatation and dysfunction, late gadolinium enhancement with non-ischaemic distribution.

^g e.g. AV conduction abnormalities, broad QRS, ST/T deviations, multifocal PVCs, inflammatory hyperaemia and oedema, fibrosis, LV and RV systolic dysfunction, pericardial effusion.

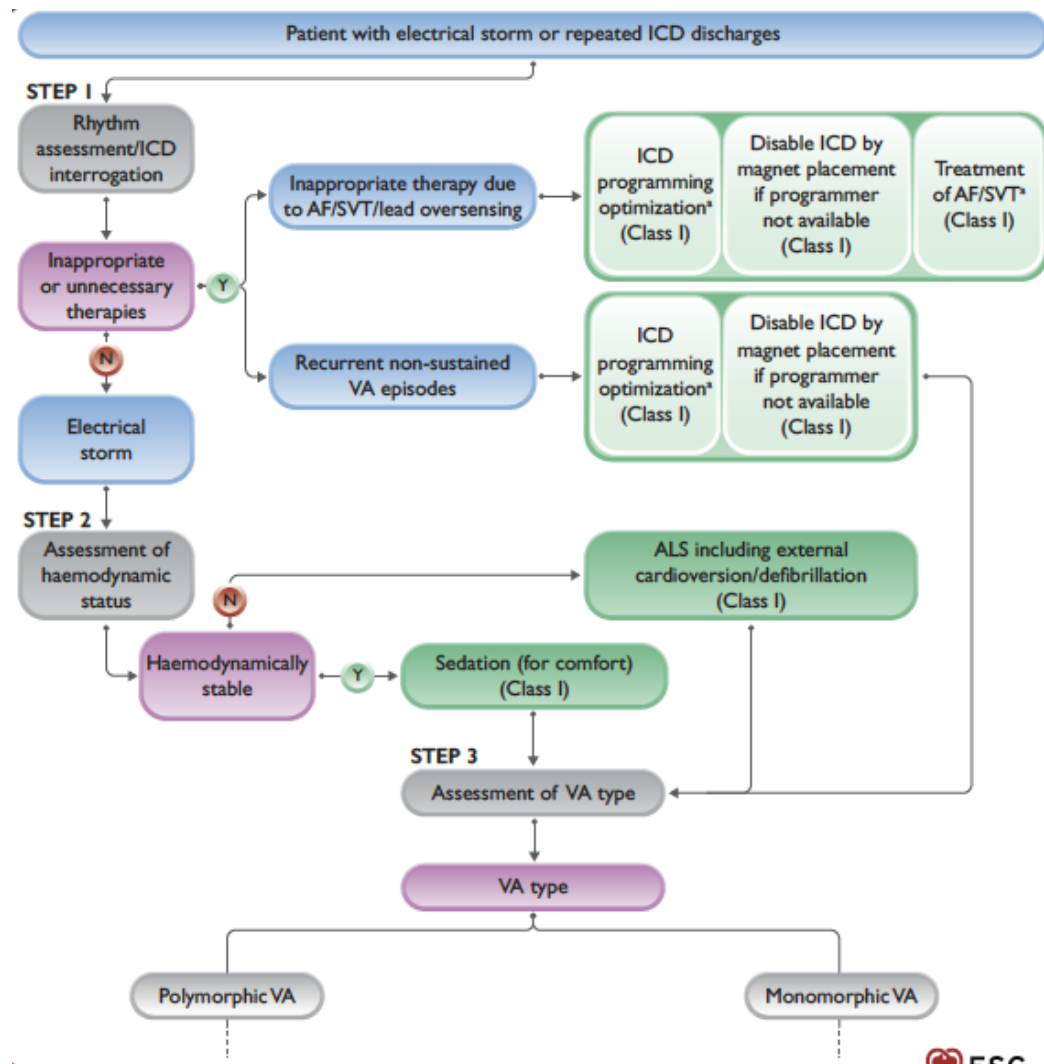


Figure 15. ESC 2022 GL on VA and SCD. Management of patients with electrical storm or repeated implantable cardioverter defibrillator discharges (Parts 1-3)

AAD, antiarrhythmic drug; AF, atrial fibrillation, ALS, Advanced life support; CPVT, catecholaminergic polymorphic ventricular tachycardia; ERS, early repolarization syndrome; ICD, implantable cardioverter defibrillator; N, No; PVC, premature ventricular complex; STEMI, ST-elevation myocardial infarction; SVT, supraventricular tachycardia; VA, ventricular arrhythmia; VF, ventricular fibrillation; Y, Yes. aSpecial aspects of device therapy section. bNo data on effect of quinidine on PVC-triggered polymorphic VAs in patients with cardiomyopathies. cHigh VA burden refers to a clinical scenario of very frequent VA episodes requiring ICD shocks, when only short periods of stable rhythm can be achieved. Low VA burden refers to a clinical scenario of repeated ATPs/ICD shocks followed by stable rhythm. dIf bradycardia or post-extrasystolic pauses facilitate initiation of PVT/VF. eOverdrive pacing (by pacing with a slightly higher rate than the baseline rhythm) can be useful to temporarily suppress slow recurrent/incessant VT.

Table 6. Investigations in patients with ventricular arrhythmias**ACC/AHA/HRS 2017 GL on VA/SCD.****History and physical examination**

Hospitalize patients presenting with syncope for which VA is documented, or thought to be a likely cause, for evaluation, monitoring, and management	I-B-NR
--	--------

Noninvasive evaluation**12-lead ECG and Exercise Testing**

12-lead ECG during with sustained, hemodynamically stable, wide complex tachycardia	I-B-NR
---	--------

Exercise treadmill testing in VA symptoms associated with exertion, suspected ischemic heart disease, or catecholaminergic polymorphic VT.	I-B-NR
--	--------

12-lead ECG during sinus rhythm in suspected or documented VA.	I-B-NR
--	--------

Ambulatory electrocardiography

To evaluate whether symptoms, including palpitations, presyncope, or syncope, are caused by VA	I-B-NR
--	--------

Implanted cardiac monitors

In sporadic symptoms (including syncope) suspected to be related to VA	IIa-B-R
--	---------

Noninvasive cardiac imaging

Echocardiography in known or suspected VA that may be associated with underlying structural heart disease or a risk of SCA.	I-B-NR
---	--------

Cardiac magnetic resonance imaging or computed tomography to detect and characterize underlying structural heart disease.	IIa-C-EO
---	----------

Biomarkers

BNP or N-terminal pro-BNP in patients with structural heart disease	IIa-B-NR
---	----------

Genetic considerations

Genetic counseling in patients and family members in whom genetic testing for SCA or SCD is recommended	I-C-EO
---	--------

Invasive testing**Invasive Cardiac Imaging: Cardiac Catheterization or CT Angiography**

CT or invasive coronary angiography in patients who have recovered from unexplained SCA.	I-C-EO
--	--------

Electrophysiological study

In ischemic cardiomyopathy, NICM, or adult congenital heart disease with syncope or other VA symptoms and no indications for a primary prevention ICD, for the risk of sustained VT.	IIa-B-R
--	---------

Not indicated in patients who meet criteria for ICD implantation. III-B-R
No benefit

Not recommended in long QT syndrome, catecholaminergic polymorphic ventricular tachycardia, short QT syndrome, or early repolarization syndromes. III-B-R
No benefit

ESC 2022 GL on VA and SCD. Recommendations for evaluation of patients presenting with newly documented ventricular arrhythmia.

In patients with newly documented VA (frequent PVCs, NSVT, SMVT), a baseline 12-lead ECG, recording of the VA on 12-lead ECG, whenever possible, and an echocardiogram as first-line evaluation. I C

A CMR in patients with newly documented VA (frequent PVCs, NSVT, SMVT) and suspicion of SHD other than CAD after initial evaluation,. IIa B

A ≥ 24 h Holter ECG In patients with an incidental finding of a NSVT. IIa C

CAD, coronary artery disease; CMR, cardiac magnetic resonance; ECG, electrocardiogram; NSVT, non-sustained ventricular tachycardia, PVCs, premature ventricular contractions; SHD, structural heart disease; SMVT, sustained monomorphic ventricular tachycardia; VA, ventricular arrhythmia

ESC 2022 GL on VA and SCD. Recommendations for evaluation of patients presenting with a first episode of sustained monomorphic ventricular tachycardia

In patients presenting with a first SMVT episode, electrophysiological study, Electroanatomical mapping, and mapping-guided biopsies may be for aetiological evaluation. IIb C

Table 7. ECG Score for Risk of Sudden Arrhythmic Death

ECG Score	Points
<i>Contiguous Q wave</i>	1
<i>QRS duration</i>	
80-100 ms	1
>110 ms	2
<i>LV hypertrophy</i>	1
<i>Prolonged JTc</i> (≥ 360 ms)	1
Low Risk:	0-1
Moderate Risk:	2
High Risk:	≥ 3

Chatterjee NA, et al. Simple electrocardiographic measures improve sudden arrhythmic death prediction in coronary disease. *Eur Heart J*. 2020;41:1988-1999.

Table 8. ESC 2022 GL on VA and SCD. Recommendations for genetic testing

Genetic testing when a condition is diagnosed in a living or deceased individual with a likely genetic basis and a risk of VA and SCD.	I B
When a putative causative variant is first identified, evaluation for pathogenicity using an internationally accepted framework.	I C
When a Class IV or Class V variant has been identified in a living or deceased individual with a condition that carries a risk of VA and SCD, genetic testing of first-degree and symptomatic relatives and obligate carriers.	I C
Genetic testing and counselling on its potential consequences should be undertaken by an expert multidisciplinary team.	I C
Class III (variants of uncertain significance) and Class IV variants should be evaluated for segregation in families where possible, and the variant re-evaluated periodically.	I C
It is not recommended to undertake genetic testing in index patients with insufficient evidence of a genetic disease.	III C

SCD, sudden cardiac death; VA, ventricular arrhythmia

Table 9. Long-term Management

ESC 2022 GL on VA and SCD. Recommendations for treatment with heart failure medication

Optimal medical treatment including ACE-I/ARB/ ARNIs, MRAs, beta-blockers, and SGLT2 inhibitors in all heart failure patients with reduced EF.	I A
--	-----

ACE-I, angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; ARNIs, angiotensin receptor neprilysin inhibitor; EF, ejection fraction; MRA, mineralocorticoid receptor antagonists; SGLT2, sodium–glucose co-transporter 2.

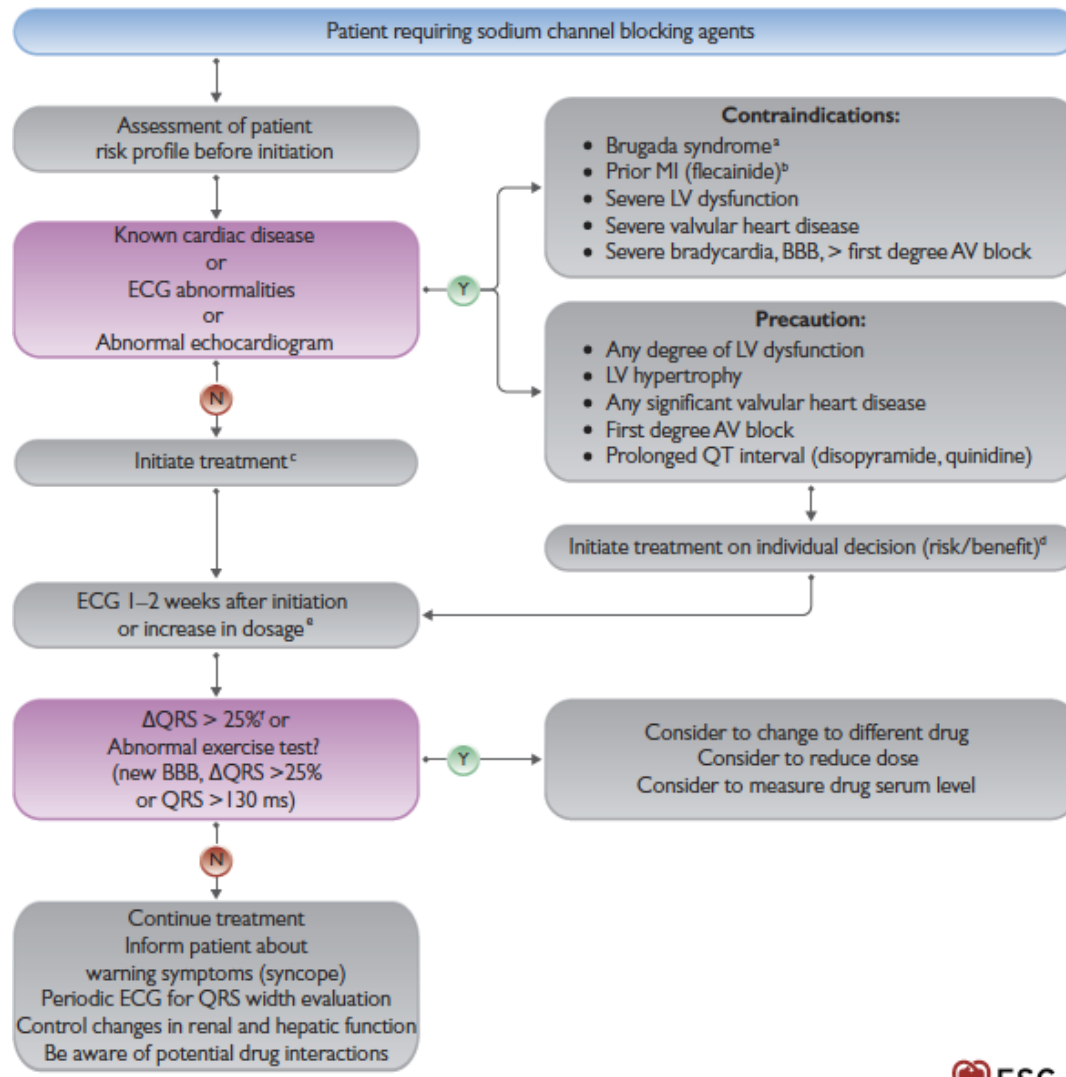


Figure 15. ESC 2022 GL on VA and SCD. Algorithm for evaluation before initiation and follow-up of patients requiring sodium channel blocking agents.

^a<http://www.brugadadrugs.org>.

^bFlecainide, encainide.

^cCo-administration of drugs with AV nodal blocking effect in patients with atrial fibrillation or atrial flutter.

^dIn implantable cardioverter defibrillator carriers, a higher risk of drug-induced pro-arrhythmia might be accepted.

^eAccording to the 2020 ESC Guidelines for the diagnosis and management of atrial fibrillation

^fA ΔQRS .25% is not an absolute cut-off value but dependent on QRS width before drug initiation and individualized patient risk–benefit considerations

AV, atrioventricular; BBB, bundle branch block; ECG, electrocardiogram; LV, left ventricular; MI, myocardial infarction; N, No; Y, Yes.

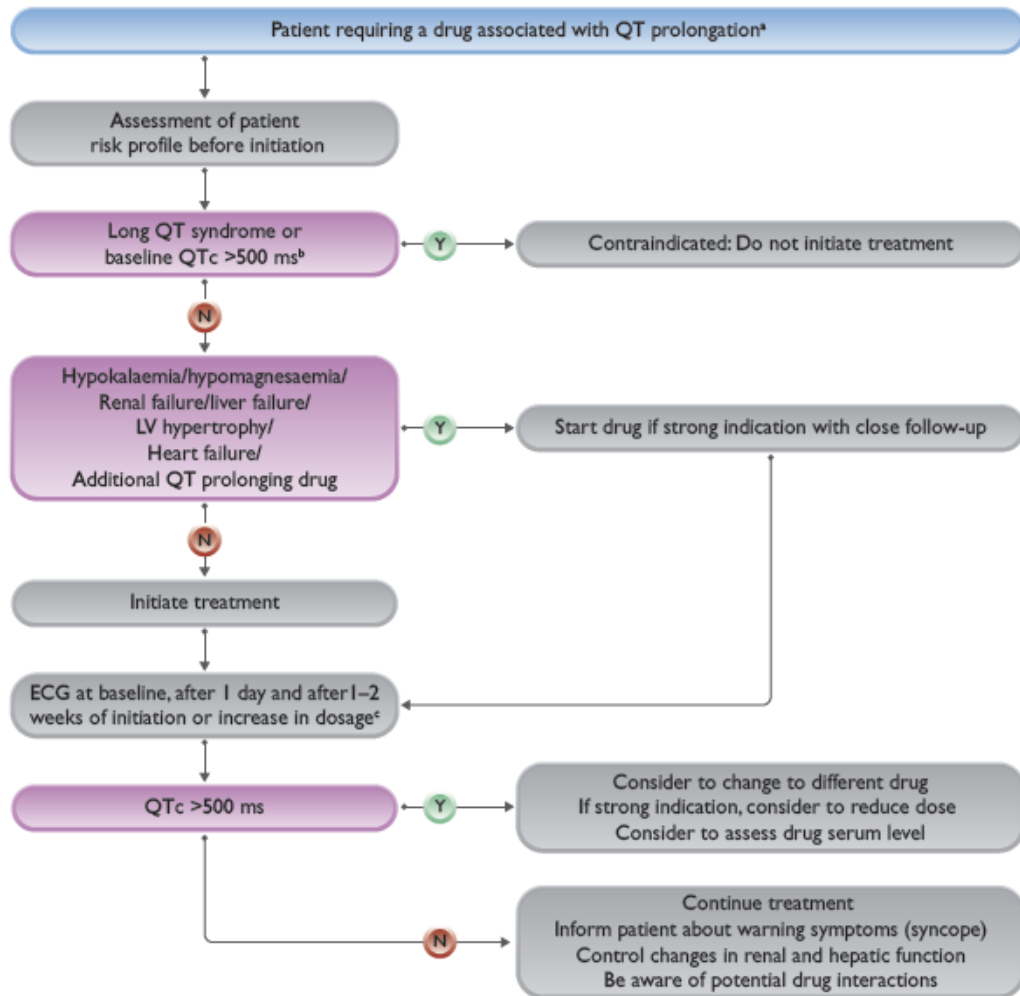


Figure 16. ESC 2022 GL on VA and SCD. Algorithm for evaluation before initiation and follow-up of patients requiring drugs associated with QT prolongation.

ECG, electrocardiogram; LV, left ventricular; N, No; Y, Yes.

^a <http://www.crediblemeds.org>.

^b If strong indication and no alternative treatment, consult a specialist.

^c According to the 2020 ESC Guidelines for the diagnosis and management of atrial fibrillation.

Table 10. Anti-arrhythmic drugs for Ventricular arrhythmias**ACC/AHA/HRS 2017 GL on VA/SCD. Pharmacological Characteristics of Available Antiarrhythmic Medications for Treating VA**

Antiarrhythmic Medication (Class) and Dose	Uses in VA/SCA	Target	Electrophysiological Effects	Pharmacological Characteristics	Common Adverse Effects
Acebutolol PO 200–1200 mg daily or upto 600 mg bid	VT, PVCs	Beta 1, Mild intrinsic sympathomimetic activity	Sinus rate slowed AV nodal refractoriness increased	Active metabolite $t_{1/2}$: 8–13 h Prolonged with renal impairment) Metab: H Excr: F 60%, U 40%	Cardiac: Bradycardia, hypotension, HF, AVB Other: Dizziness, fatigue, anxiety, impotence, hyper/hypoesthesia
Amiodarone (III) IV: 300 mg bolus for VF/pulseless VT arrest; 150-mg bolus for stable VT; 1 mg/min x 6 h, then 0.5 mg/min x 18 h PO: 400 mg* q 8 to 12 h for 1–2 wk, then 300–400 mg daily; reduce dose to 200 mg daily if possible	VT, VF, PVC,	I_{Na} , I_{Ca} , I_{Kr} , I_{K1} , I_{Ks} , I_{to} , Beta receptor, Alpha receptor nuclear T3 receptor	Sinus rate slowed QRS prolonged QTc prolonged AV nodal refractoriness increased; increased DFT	$t_{1/2}$: 26–107 d Metab: H Excr: F	Cardiac: Hypotension, bradycardia, AVB, TdP, slows VT below programmed ICD detection rate, increases defibrillation threshold Other: Corneal microdeposits, thyroid abnormalities, ataxia, nausea, emesis, constipation, photosensitivity, skin discoloration, ataxia, dizziness, peripheral neuropathy, tremor, hepatitis, cirrhosis, pulmonary fibrosis or pneumonitis
Atenolol (II) PO: 25–100 mg qd or bid	VT, PVC, ARVC, LQTS	Beta 1	Sinus rate slowed AV nodal refractoriness increased	$t_{1/2}$: 6–7 h (prolonged with renal impairment) Metab: H Excr: F 50%, U 40%	Cardiac: Bradycardia, hypotension, HF, AVB Other: Dizziness, fatigue, depression, impotence
Bisoprolol (II) PO: 2.5–10 mg once daily	VT, PVC	Beta 1 receptor	Sinus rate slowed AV nodal refractoriness increased	$t_{1/2}$: 9–12 h Metab: H Excr: U	Cardiac: Chest pain, bradycardia, AVB Other: Fatigue, insomnia, diarrhea
Carvedilol (II) PO: 3.125–25 mg q 12 h	VT, PVC	Beta 1 and 2 receptors, Alpha	Sinus rate slowed AV nodal refractoriness increased	$t_{1/2}$: 7–10 h Metab: H Excr: F	Cardiac: Bradycardia, hypotension, AVB, edema, syncope Other: Hyperglycemia, dizziness, fatigue, diarrhea
Diltiazem (IV)	VT specifically	I_{Ca-L}	Sinus rate slowed PR prolonged	$t_{1/2}$: Injection 2–5 h, immediate	Cardiac: Hypotension, edema, HF, AVB,

IV: 5-10 mg qd 15-30 min Extended release: PO: 120-360 mg/day	RVOT, idiopathic LVT		AV nodal conduction slowed	release 4.5-12 h, extended release 12 h, and severe hepatic impairment 14- 16 h Metab: H Excr: U	bradycardia, exacerbation of HFrEF Other: Headache, rash, constipation
Esmolol (II) IV: 0.5 mg/kg bolus, 0.05 mg/kg/min	VT	Beta 1 receptor	Sinus rate slowed AV nodal refractoriness increased	$t_{1/2}$: 9 min Metab: RBC esterases Excr: U	Cardiac: Bradycardia, hypotension, HF, AVB Other: Dizziness, nausea
Flecainide (IC) PO: 50-200 mg q 12 h	VT, PVC (in the absence of structural heart disease). Has a role in treating patients with CPVT	I_{Na} , I_{Kr} , I_{Kur}	PR prolonged QRS prolonged; increased DFT	$t_{1/2}$: 7-22 h Metab: H Excr: U	Cardiac: Sinus node dysfunction, AVB, drug- induced Brugada syndrome. monomorphic VT in patients with a myocardial scar, exacerbation of HFrEF Other: Dizziness, tremor, vision disturbance, dyspnea, nausea
Lidocaine (IB) IV: 1 mg/kg bolus, 1-3 mg/min 1-1.5 mg/kg. Repeat 0.5- 0.75 mg/kg bolus every 5- 10 min (max cumulative dose 3 mg/kg). Maintenance infusion is 1-4 mg/min although one could start at 0.5 mg/min	VT, VF	I_{Na}	No marked effect on most intervals; QTc can slightly shorten	Initial $t_{1/2}$ 7-30 min; terminal 90-120 min. Prolonged in HF, liver disease, shock, severe renal disease Metab: H Excr: U	Cardiac : Bradycardia, hemodynamic collapse, AVB, sinus arrest Other: Delirium, psychosis, seizure, nausea, tinnitus, dyspnea, bronchospasm
Metoprolol (II) IV: 5 mg q 5 min up to 3 doses PO: 25-100 mg Extended release qd or q	VT, PVC	Beta 1 receptor	Sinus rate slowed AV nodal refractoriness increased	$t_{1/2}$: 3-4 h Metab: H Excr: U	Cardiac: Bradycardia, hypotension, AVB Other: Dizziness, fatigue, diarrhea, depression, dyspnea

12 h					
Mexiletine (IB) PO: 150–300 mg q 8 h or q 12 h	T, VF, PVC, has a role in patients with LQT3	I _{Na}	No marked effect on most intervals; QTc can slightly shorten	t _{1/2} : 10–14 h Metab: H Excr: U	Cardiac: HF, AVB Other: Lightheaded, tremor, ataxia, paresthesias, nausea, blood dyscrasias
Nadolol (II) PO: 40–320 mg daily	VT, PVC, LQTS, CPVT	Beta 1 and 2 receptors	Sinus rate slowed AV nodal refractoriness increased	t _{1/2} : 20–24 h Metab: none Excr: U	Cardiac: Bradycardia, hypotension, HF, AVB Other: Edema, dizziness, cold extremities, bronchospasm
Procainamide (IA) IV: loading dose 10–17 mg/kg at 20–50 mg/min Maintenance dose: 1–4 mg/min PO (SR preparation): 500–1250 mg q 6 h	VT	I _{Na} , I _{Kr}	QRS prolonged QTc prolonged; increased DFT	Metab: H t _{1/2} : 2–5 h; NAPA 6–8 h t _{1/2} prolonged in renal dysfunction. Anephric: proc 11 h and NAPA 42 h Excr: U	Cardiac: TdP; AVB, hypotension and exacerbation of HFrEF Other: Lupus symptoms, diarrhea, nausea, blood dyscrasias
Propafenone (IC) PO: Immediate release 150–300 mg q 8 h Extended release 225–425 mg q 12 h	VT, PVC (in the absence of structural heart disease)	I _{Na} , I _{Kr} , I _{Kur} , Beta receptor, Alpha receptor	PR prolonged QRS prolonged; increased DFT	t _{1/2} : 2–10 h or 10–32 h t _{1/2} : extensive metabolizers 2–10 h; poor metabolizers 10–32 h. Metab: H Excr: U	Cardiac: HF, AVB, drug-induced Brugada syndrome Other: Dizziness, fatigue, nausea, diarrhea, xerostomia, tremor, blurred vision
Propranolol (II) IV: 1–3 mg q 5 min to a total of 5 mg PO: Immediate release 10–40 mg q 6 h; Extended release 60–160 mg q 12 h	VT, PVC, LQTS	Beta 1 and 2 receptors, I _{Na}	Sinus rate slowed AV nodal refractoriness increased	t _{1/2} : Immediate release 3–6 h Extended release 8–10 h Metab: H Excr: U	Cardiac: Bradycardia, hypotension, HF, AVB Other: Sleep disorder, dizziness, nightmares, hyperglycemia, diarrhea, bronchospasm
Quinidine (IA) PO: sulfate salt 200–600 mg q 6 h to q 12 h gluconate salt	T, VF, (including short QT syndrome, Brugada)	I _{Na} , I _{to} , I _{Kr} , M, Alpha receptor	QRS prolonged QTc prolonged; increased DFT	t _{1/2} : 6–8 h longer in HF, liver cirrhosis, and with older age Metab: H Excr: U	Cardiac: Syncope, TdP, AVB Other: Dizziness, diarrhea, nausea, esophagitis, emesis, tinnitus, blurred vision, rash, weakness,

324–648 mg q 8 h to q 12 h					tremor; blood dyscrasias
IV: loading dose: 800 mg in 50 mL infused at 50 mg/min					
Ranolazine (not classified)	VT	I_{Na} , I_{Kr}	Sinus rate slowed Tc prolonged	$t_{1/2}$: 7 h Metab: H Excr: U 75%, F 25%	Cardiac: Bradycardia, hypotension Other: Headache, dizziness, syncope, nausea, dyspnea
PO: 500–1000 mg q 12 h					
Sotalolol (III)	VT, VF, PVC	I_{Kr} , Beta 1 and 2 receptor	Sinus rate slowed QTc prolonged AV nodal refractoriness increased; decreased DFT	$t_{1/2}$: 12 h Metab: none Excr: U	Cardiac: Bradycardia, hypotension, HF, syncope, TdP Other: Fatigue, dizziness, weakness, dyspnea, bronchitis, depression, nausea, diarrhea
IV: 75 mg q 12 h					
PO: 80–120 mg q 12 h, may increase dose every 3 d; max 320 mg/d					
Verapamil (IV)	VT (specifically RVOT, verapamil- sensitive idiopathic LVT)	I_{Ca-L}	Sinus rate slowed PR prolonged AV nodal conduction slowed	$t_{1/2}$: 3–7 h Metab: H Excr: U	Cardiac: Hypotension, edema, HF, AVB, bradycardia, exacerbation of HFrEF Other: Headache, rash, gingival hyperplasia, constipation, dyspepsia
IV: 2.5–5 mg q 15–30 min					
Sustained release PO: 240–480 mg/d					

*Although up to 800 mg every 8 h might be used, higher doses of amiodarone are associated with a higher risk of adverse events.

Alpha indicates alpha-adrenergic receptor; ARVC, arrhythmogenic right ventricular cardiomyopathy; AV, atrioventricular; AVB, atrioventricular block; Beta, beta-adrenergic receptor; HF, heart failure; CPVT, catecholaminergic polymorphic ventricular tachycardia; DFT, defibrillation threshold; F, feces; H, hepatic; I_{Ca} , L-type calcium channel current; I_{K1} , inward rectifier potassium channel; I_{KACH} , muscarinic receptor-gated potassium channel; I_{KATP} , adenosine-activated potassium channel; I_{Kr} , rapid delayed rectifier potassium current; I_{Ks} , slow delayed rectifier potassium current; I_{Kur} , ultra-rapid delayed rectifier potassium current; I_{Na} , fast inward sodium current; I_{to} , transient outward potassium current; LQTS, long-QT syndrome; LVT, left ventricular tachycardia; M, muscarinic; Metab, metabolism; NAPA, n-acetyl procainamide; PVC, premature ventricular complex; QTc, corrected QT interval; $t_{1/2}$, half-life; RVOT, right ventricular outflow tract; T3, triiodothyronine; TdP, torsades de pointes; U, urine; VT, ventricular tachycardia; and VF, ventricular fibrillation.

ESC 2022 GL on VA and SCD. Anti-arrhythmic drugs (acute and chronic treatment)

Anti-arrhythmic drug	Effects on ECG	Indications (specific indication)	Oral dose per day (i.v. dose)	Side effects	Contraindications, precautions, other considerations
Amiodarone	Decreases sinus node frequency, prolongs QT interval ^a	PVC, VT, VF	200–400 mg Loading dose: 600–1200 mg/24 h 8–10 days. (Loading dose: 5 mg/kg in 20 min–2 h, 2–3 times in 24 h, then 600–1200 mg/24 h 8–10 days)	<i>Cardiac</i> : Bradycardia, TdP (infrequent) <i>Extracardiac</i> : Photosensitivity, corneal deposits, hypothyroidism, hyperthyroidism, pulmonary toxicity, hepatotoxicity, polyneuropathy, skin discoloration	<i>Precautions</i> : Sinus node dysfunction, severe AV conduction disturbances, hyperthyroidism <i>Other considerations</i> : Can be used in patients with heart failure. Increases the risk of myopathy when used with statins
Adenosine	Transitory AV block	Regular wide complex tachycardia of unknown origin (outflow tract VT)	No oral use (6–18 mg bolus)	Chest pain, flushing, bronchoconstriction	<i>Contraindications</i> : Severe asthma, pre-excited AF <i>Other considerations</i> : Antagonist: theophylline
Ajmaline	Prolongs QRS duration and QT interval ^a	VT (unmasking BrS ECG)	No oral use (1 mg/kg over 5–10 min (maximum dose 100 mg) or 1 mg/kg at 10 mg/min)	<i>Cardiac</i> : VF (rare in suspected BrS), occasional TdP, negative inotrope <i>Extracardiac</i> : Cholestatic jaundice, headache, nausea, thrombocytopenia	<i>Contraindications</i> : BrS ECG type I, QT prolongation
Beta-blocker	Decreases sinus node frequency, prolongs PR interval, shortens QT interval	PVC, VT (LQTS, CPVT)	Various (various)	<i>Cardiac</i> : Bradycardia, AV block, hypotension, negative inotrope <i>Extracardiac</i> : Fatigue, bronchospasm, sexual disturbances, depression, cold extremities	<i>Contraindications</i> : Severe sinus node dysfunction, severe AV conduction disturbances, decompensated heart failure, coronary vasospasm, severe asthma, BrS
Landiolol (Ultra-short acting β_1 -selective blocker)	See beta-blocker	VT, electrical storm	No oral use 100 μ g/kg bolus in 1 min, infusion 10–40 μ g/kg/min (max 80 μ g/kg/min; max 24 h total dose 57.6 mg/kg/day)	See beta-blocker	<i>Contraindications</i> : See beta-blocker. Bradycardia, hypotension <i>Other considerations</i> : Limited experience with its use beyond 24 h
Nadolol (Non-selective $\beta_1\beta_2$ blocker)	See beta-blocker	PVC, VT (LQTS, CPVT)	40–120 mg	See beta-blocker	<i>Contraindications</i> : See beta-blocker <i>Other considerations</i> : Plasma half-life 20–24 h
Propranolol (Non-selective $\beta_1\beta_2$ blocker)	See beta-blocker	PVC, VT (electrical storm, LQTS, CPVT)	80–320 mg (160 mg/24 h)	See beta-blocker	<i>Contraindications</i> : See beta-blocker
Disopyramide	Increases sinus node frequency and prolongs PR interval, QRS duration, and QT interval ^a	PVC, VT	250–750 mg	<i>Cardiac</i> : Negative inotrope, AV block, pro-arrhythmia (MVT, occasional TdP)	<i>Contraindications</i> : Severe sinus node dysfunction, severe AV conduction disturbances, severe intraventricular

				<i>Extracardiac:</i> Anticholinergic effects	conduction disturbances, prior MI, significant SHD, hypotension <i>Other considerations:</i> Reduces LV outflow tract obstruction and symptoms in HCM
Flecainide	Prolongs PR interval, QRS duration, and QT interval ^a	PVC, VT (unmasking BrS ECG ^b)	200–400 mg (1–2 mg/kg over 10 min)	<i>Cardiac:</i> Pro-arrhythmia (MVT, occasional TdP), negative inotrope, sinus bradycardia, AV block, 1:1 AV conduction during flutter <i>Extracardiac:</i> Central nervous system effects (e.g. drowsiness, diplopia, headache)	<i>Contraindications:</i> Prior MI, significant SHD, BrS, severe sinus node dysfunction, severe AV or intraventricular conduction disturbances, inherited LQTS (other than LQTS3), severe kidney disease (CrCl <35 mL/min/1.73 m ²) <i>Other considerations:</i> Discontinue if QRS widening >25% or bundle branch block
Isoproterenol	Increases sinus node frequency, shortens QT interval	(Electrical storm in BrS, idiopathic VF, and ERS, TdP, beta-blocker overdose; acquired LQTS)	No oral use (0.5–10 µg/min)	<i>Cardiac:</i> Sinus tachycardia, vasodilation <i>Extracardiac:</i> Headache, sweating, tremor	<i>Contraindications:</i> ACS, LQTS <i>Other considerations:</i> Short plasma half time (2 min)
Lidocaine	No significant effects	(VT/VF associated with ACS)	No oral use (50–200 mg bolus, then 2–4 mg/min)	<i>Cardiac:</i> Sinoatrial arrest <i>Extracardiac:</i> Central nervous system effects (e.g. drowsiness, dizziness)	<i>Precautions:</i> Reduced dose with reduced liver blood flow (e.g. shock, β-blockade, severe heart failure) <i>Other considerations:</i> More effective with high potassium level. Few haemodynamic side effects
Mexiletine	No significant effects	PVC, VT (LQT3)	600–1200 mg Loading dose: 400 mg initially followed by 600 mg in the first 24 h	<i>Cardiac:</i> Sinus bradycardia in sinus node dysfunction, hypotension <i>Extracardiac:</i> Central nervous system effects (e.g. tremor, dysarthria, dizziness), gastrointestinal complaints	<i>Contraindications:</i> Sinus node dysfunction, severe AV conduction disturbances, severe heart failure
Procainamide	Prolongs PR interval, QRS duration, and QT interval ^a	VT	(100 mg bolus, can be repeated after 5 min if no effect, max 500–750 mg [max 50 mg/min]. Then, 2–6 mg/min)	<i>Cardiac:</i> Sinus bradycardia, hypotension, TdP <i>Extracardiac:</i> Rash, myalgia, vasculitis, systemic lupus, agranulocytosis	<i>Contraindications:</i> Severe sinus node dysfunction, severe AV conduction disturbances, severe intraventricular conduction disturbances, severe LV dysfunction hypotension, BrS
Propafenone	Prolongs PR interval, QRS duration, and QT interval ^a	PVC, VT	450–900 mg	<i>Cardiac:</i> Sinus bradycardia, AV block, negative inotrope, pro-arrhythmia (MVT, occasional TdP) <i>Extracardiac:</i> Gastrointestinal disturbances, headache, dry mouth	<i>Contraindications:</i> Prior MI, significant SHD, BrS, severe sinus node dysfunction, severe AV or intraventricular conduction disturbances, LQTS, significant renal or liver disease <i>Other considerations:</i> Discontinue if QRS widening >25% or bundle branch block

Quinidine	Increases sinus node frequency and prolongs PR interval, QRS duration, and QT interval ^a	(VF ^c , BrS, SQTS)	600–1600 mg Loading dose: Start 200 mg every 3 h until effect, max. 3 g in first 24 h	<i>Cardiac</i> Hypotension, TdP ^d <i>Extracardiac</i> Gastrointestinal disturbances, auditory and visual disturbances, confusion, leukopenia, haemolytic anaemia, thrombocytopenia, anaphylaxis	<i>Contraindications</i> Severe sinus node dysfunction, severe AV or intraventricular conduction disturbances, previous MI, significant SHD, hypotension, LQTS
Ranolazine	Decreases sinus node frequency, prolongs QT interval ^a	VT (LQTS3)	750–2000 mg	<i>Cardiac</i> Sinus bradycardia, hypotension <i>Extracardiac</i> Dizziness, nausea, constipation, gastrointestinal disturbance, headache, rash	<i>Contraindications</i> Severe sinus node dysfunction, severe heart failure, LQTS (other than LQTS3) <i>Precautions</i> Concomitant treatments associated with QT interval prolongation
Sotalol	Decreases sinus node frequency, prolongs QT interval ^a	VT	160–640 mg (0.5–1.5 mg/kg in 10 min. If necessary, can be repeated after 6 h)	See beta-blockers, TdP ^d (>2% of patients, close monitoring of QT interval and CrCl)	<i>Contraindications</i> Severe sinus node dysfunction, severe AV conduction disturbances, severe heart failure with reduced LVEF, significant LVH, CrCl <30 ml/min, coronary vasospasm, LQTS <i>Precautions</i> Concomitant treatments associated with QT interval prolongation, hypokalaemia <i>Other considerations</i> Potassium channel blocker effects require higher dose than beta-blocker effects
Verapamil	Prolongs PR interval	(LV fascicular tachycardia)	120–480 mg (5–10 mg in slow bolus. If necessary, can be repeated in 30 min)	<i>Cardiac</i> Sinus bradycardia in sinus node dysfunction, AV block, negative inotrope, hypotension <i>Extracardiac</i> Gastrointestinal disturbances, peripheral oedema, flushing	<i>Contraindications</i> Heart failure with reduced LVEF, severe sinus node dysfunction, and severe AV conduction disturbances, VT of unknown origin, ACS, WPW syndrome <i>Other considerations</i> Increase the risk of myopathy when used with statins

ACS, acute coronary syndrome; AF, atrial fibrillation; AV, atrioventricular; BrS, Brugada syndrome; CPVT, catecholaminergic polymorphic ventricular tachycardia; CrCl, creatinine clearance; ECG, electrocardiogram; ERS, early repolarization syndrome; HCM, hypertrophic cardiomyopathy; LQTS, long QT syndrome; LV, left ventricular; LVEF, left ventricular ejection fraction; LVH, left ventricular hypertrophy; MI, myocardial infarction; MVT, monomorphic ventricular tachycardia; PVC, premature ventricular complex; SHD, structural heart disease; SQTS, short QT syndrome; TdP, torsades de pointes; VF, ventricular fibrillation; VT, ventricular tachycardia; WPW, Wolff–Parkinson–White.

^a Precaution with concomitant conditions or drugs that prolong QT interval. Should be discontinued if QTc >500 ms. See Algorithm for evaluation before initiation and follow-up of patients requiring drugs associated with QT prolongation.

^b If ajmaline not available.

^c Subacute MI, multifocal ectopic Purkinje-related premature contractions, ERS, idiopathic VF.

^d Proarrhythmic side effects require strong indication in patients without ICD.

Table 11. ICD: General principles**ESC 2022 GL on VA and SCD.****Recommendations for implantable cardioverter defibrillator implantation (general aspects)**

Implantation of a cardioverter defibrillator is only recommended in patients who have an expectation of good quality survival >1 year. I C

It is not recommended to implant an ICD in patients with incessant VAs until the VA is controlled. III C

ICD, implantable cardioverter defibrillator; VA, ventricular arrhythmia.

Recommendations for secondary prevention of sudden cardiac death

ICD implantation in patients with documented VF or haemodynamically not-tolerated VT in the absence of reversible causes. I A

In patients with VT/VF, an indication for ICD, and no contraindication for amiodarone, Amiodarone when an ICD is not available, contraindicated for concurrent medical reasons, or declined by the patient. IIb C

In patients with SMVT or SPVT/VF triggered by aPVC with similar morphology and an indication for ICD, catheter ablation when an ICD is not available, contraindicated for concurrent medical reasons, or declined by the patient. IIb C

PVC, premature ventricular complex; SMVT, sustained monomorphic VT; SPVT, sustained polymorphic VT; VF, ventricular fibrillation; VT, ventricular tachycardia.

Recommendations for subcutaneous implantable cardioverter defibrillator

Subcutaneous defibrillator as an alternative to transvenous defibrillator in patients with an indication for an ICD when pacing therapy for bradycardia, cardiac resynchronization, or ATP is not needed. IIa B

ATP, anti-tachycardia pacing

Recommendations for adding cardiac resynchronization therapy to implantable cardioverter defibrillator

When an ICD is indicated, evaluate whether the patient could benefit from CRT-defibrillator. I C

CRT, cardiac resynchronization therapy

Recommendations for wearable cardioverter defibrillator

WCD for adult patients with a secondary prevention ICD indication, who are temporarily not candidates for ICD implantation. IIa C

WCD in the early phase after MI in selected patients. IIb B

ICD, implantable cardioverter defibrillator; WCD, wearable cardioverter defibrillator.
aClass of recommendation.

Recommendations for concomitant treatment to avoid inappropriate implantable cardioverter defibrillator therapy

Catheter ablation for ICD patients with recurrent SVT resulting in inappropriate ICD therapies. I C

Pharmacological treatment or catheter ablation in patients with AF-related inappropriate ICD therapies despite optimal ICD programming. I C

AF, atrial fibrillation; SVT, supraventricular tachycardia.

Recommendations for psychosocial management after implantable cardioverter defibrillator implantation

Assessment of psychological status and treatment of distress is recommended in ICD patients. I C

Communication between patient and physician/ healthcare professional is recommended to address ICD-related concerns and to discuss quality-of-life issues before ICD implantation and during disease progression. I C

Recommendations for implantable cardioverter defibrillator implantation in left ventricular assist device recipients

ICD implantation in LVAD recipients with symptomatic sustained VAs. IIa B

LVAD, left ventricular assist devices

Recommendations for prevention of implantable cardioverter defibrillator complications

Single-chamber ICD over dual-chamber ICD in primary prevention patients without current or expected indication for atrial or atrioventricular sequential pacing due to a lower risk of device-related complications. I A

Use of single-coil leads over dual-coil ICD leads due to lower complication rate during transvenous lead extraction. IIa C

Recommendations for end-of-life issues in implantable cardioverter defibrillator carriers

Informed discussion with patient and family about ICD deactivation options and shared decision-making prior to implantation and in case of significant health status deterioration. I C

ACC/AHA/HRS 2017 GL on VA/SCD.

Terminal Care

At the time of ICD implantation or replacement, and during advance care planning, patients should be informed that their ICD shock therapy can be deactivated at any time if it is consistent with their goals and preferences. I-C-EO

In patients with refractory HF symptoms, refractory sustained VA, or nearing the end of life from other illness, clinicians should discuss ICD shock I-C-EO

deactivation and consider the patients' goals and preferences.

Shared decision-making.

In patients with VA or at increased risk for SCD, clinicians should adopt a shared decision-making approach in which treatment decisions are based not only on the best available evidence but also on the patients' health goals, preferences, and values. I-B-NR

Patients considering implantation of a new ICD or replacement of an existing ICD for a low battery should be informed of their individual risk of SCD and nonsudden death from HF or noncardiac conditions and the effectiveness, safety, and potential complications of the ICD in light of their health goals, preferences and values. I-B-NR

Subcutaneous Implantable Cardioverter-Defibrillator

In patients who meet criteria for an ICD who have inadequate vascular access or are at high risk for infection, and in whom pacing for bradycardia or VT termination or as part of CRT is neither needed nor anticipated. I-B-NR

In patients who meet indication for an ICD, if pacing for bradycardia or VT termination or as part of CRT is neither needed nor anticipated. IIa-B-NR

In patients with an indication for bradycardia pacing or CRT, or for whom antitachycardia pacing for VT termination is required, a subcutaneous ICD should not be implanted III-B-NR (Harm)

Wearable Cardioverter Defibrillator

In patients with an ICD and a history of SCA or sustained VA in whom removal of the ICD is required (as with infection). IIa-B-NR

In patients at an increased risk of SCD but who are not ineligible for an ICD, such as awaiting cardiac transplant, having an LVEF of 35% or less and are within 40 days from an MI, or have newly diagnosed NICM, revascularization within the past 90 days, myocarditis or secondary cardiomyopathy or a systemic infection. IIb-B-NR

Table 12. Specific therapies

ACC/AHA/HRS 2017 GL on VA/SCD.

Autonomic Modulation

Bet blockers in symptomatic, non–life-threatening VA IIa-C-LD

Cardiac sympathetic denervation for patients with VT/VF storm in whom a beta blocker, other antiarrhythmic medications, and catheter ablation are ineffective, not tolerated, or not possible. IIb-C-LD

Special Considerations for Catheter Ablation

Catheter ablation is useful for reducing the risk of recurrent VT and ICD shocks in bundle-branch reentrant VT I-C-LD

Epicardial catheter ablation in patients with structural heart disease who have failed endocardial catheter ablation. IIa-B-NR

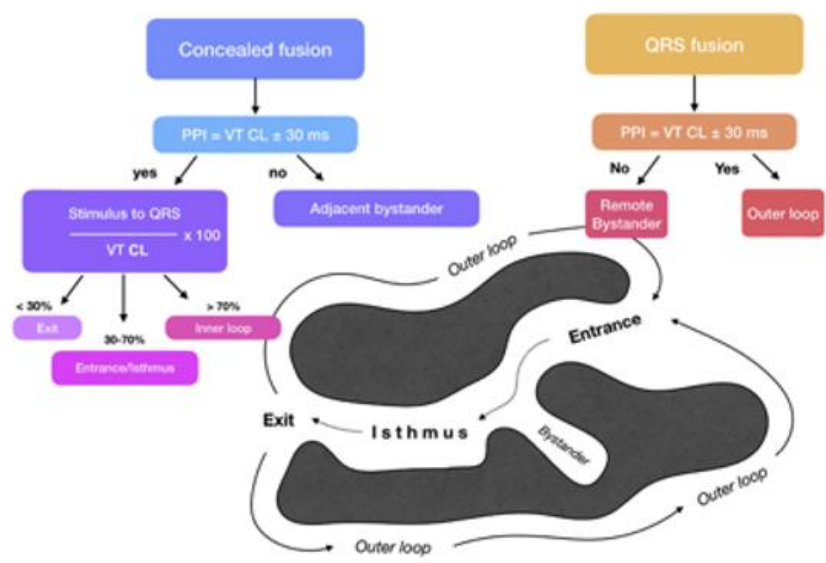


Figure 17. Entrainment responses from components of reentrant VT circuit.

CL 5 cycle length; PPI 5 postpacing interval; VT 5 ventricular tachycardia.

Cronin EM, Bogun FM, Maury P, et al. 2019 HRS/EHRA/APHRS/LAHRS expert consensus statement on catheter ablation of ventricular arrhythmias: Executive summary. *Heart Rhythm*. 2020;17(1):e155-e205.

Table 13. Management of patients with nonsustained VT

Clinical setting	Investigations	Therapy
Idiopathic NSVT	EP testing differentiates from ARVC	Beta blockers, calcium channel blockers; RF ablation if inducible sustained VT, progressively reduced LVEF, or symptoms
Arrhythmogenic ventricular cardiomyopathy	Value of EP testing not established NSVT indicates intermediate arrhythmic risk (<2% per year)	Not established. Perhaps amiodarone or sotalolol; ICD frequently considered
Hypertension, valve disease	No need for specific management	Optimal antihypertensive therapy, including beta blockers
Non-STE ACS, NSVT >48 h after admission	Meticulous ischaemia testing	Revascularization and optimal medical therapy ¹
Acute MI, NSVT >24 h till pre-discharge	Routine for acute MI	Revascularization and optimal medical therapy
Previous MI with LVEF = 31–40%	Ischaemia testing, EP testing ²	Revascularization and optimal medical therapy. If EP-inducible monomorphic VT or VF ³ : ICD ⁴
Previous MI with LVEF ≤30% or LVEF ≤35% and NYHA I/II	Ischaemia testing. No EP needed ²	Revascularization and optimal medical therapy ICD ⁴
Asymptomatic CAD with EF >40%	Ischaemia testing	Revascularization and optimal medical therapy. No need for specific NSVT therapy
Syncope in CAD with EF >40%	Ischaemia testing, EP testing ²	Revascularization and optimal medical therapy. If EP-inducible monomorphic VT or VF ³ : ICD
Non-ischaemic dilated cardiomyopathy	Value of EP testing not established	Optimal CCF therapy (medical and CRT if indicated). Ablation for bundle branch reentry. ICD for syncope or LVEF ≤30–35% and NYHA I/II
Hypertrophic cardiomyopathy	Evaluate additional risk factors: - Previous cardiac arrest - Unexplained syncope - Massive LV hypertrophy (≥30 mm) - Hypotensive or attenuated blood pressure response to upright exercise	Beta blockers, ICD, especially with frequent and prolonged (>10 beats) episodes of NSVT
Congenital heart disease (usually repaired Fallot)	EP testing	Predictive value of NSVT not established. Consider corrective surgery. If VT inducible: ablation and ICD
Long QT syndrome	Genotype analysis useful	Beta blockers, ICD if syncope despite beta blockers
Catecholaminergic polymorphic VT	Value of EP testing not established	Beta blockers and perhaps calcium channel blockers. ICD if cardiac arrest
Brugada syndrome	Value of EP testing disputed	Possibly quinidine (more data needed). ICD if cardiac arrest

¹ Optimal medical therapy for NSVT in the setting of coronary artery disease is defined as administration of beta blockers (essential, unless absolutely contraindicated), aspirin, statins, and angiotensin-converting enzyme inhibitors or angiotensin receptor blockers.

² If conditions remain the same after indicated revascularization.

³ Prognostic significance of inducible monomorphic VT induced by three extrastimuli is similar to that of VT induced by one or two extrastimuli. Monomorphic VT, as opposed to VF, usually does not respond to revascularization. VF induced by three extrastimuli may represent a non-specific response.

⁴ ICD is recommended if indications exist at least 40 days after MI.

EP, electrophysiology study; ARVC, arrhythmogenic right ventricular cardiomyopathy; non-STE ACS, non-ST elevation acute coronary syndrome (non-STEMI or unstable angina); MI, myocardial infarction; LVEF, left ventricular ejection fraction; ICD, implantable cardioverter-defibrillator; CAD, coronary artery disease; CCF, congestive cardiac failure; CRT, cardiac resynchronization therapy.

Katritsis D, et al., Non-sustained ventricular tachycardia. *J Am Coll Cardiol*. 2012;60:1993–2004 with permission from Elsevier.

Table 14. ESC 2020 GL on Sports. Recommendations for exercise in individuals with premature ventricular contractions or non-sustained ventricular tachycardia

Thorough evaluation (including a detailed family history) to exclude underlying structural or arrhythmogenic conditions in exercising individuals with $>_2$ PVCs on a baseline ECG (or $>_1$ PVC in the case of high-endurance athletes)	I C
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Among individuals with frequent PVCs and nonsustained VT a thorough investigation with Holter monitoring, 12-lead ECG, exercise test, and suitable imaging	I C
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All competitive and leisure-time sports activities are permitted, with periodic re-evaluation in individuals without familial or structural underlying disease.	I C
---	-----

ECG = electrocardiogram; PVC = premature ventricular contractions; VT = ventricular tachycardia.

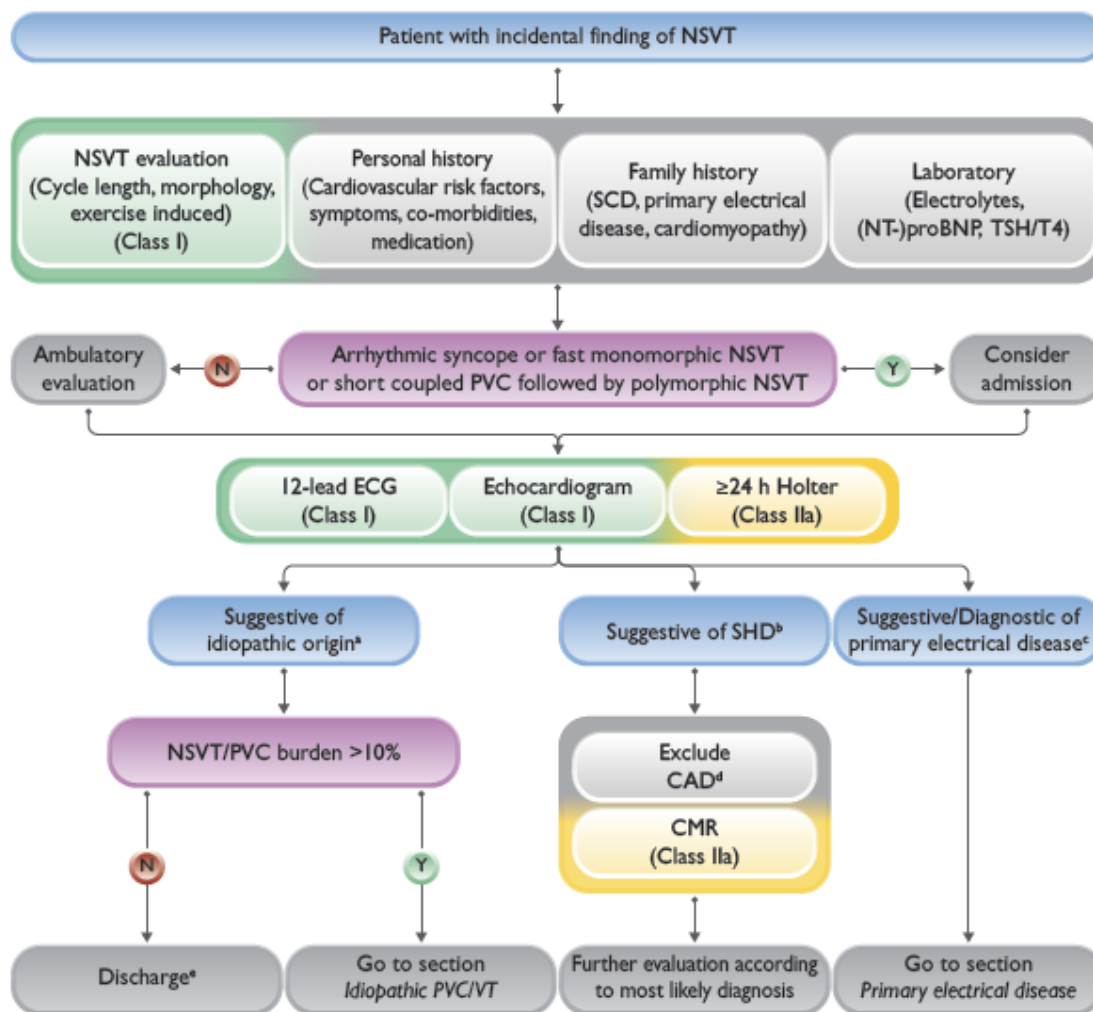


Figure 18. ESC 2022 GL on VA and SCD. Algorithm for the evaluation of patients presenting with an incidental finding of non-sustained ventricular tachycardia.

CAD, coronary artery disease; CMR, cardiac magnetic resonance; ECG, electrocardiogram; N, No; NSVT, non-sustained ventricular tachycardia; PVC, premature ventricular complex; SCD, sudden cardiac death; SHD, structural heart disease; Y, Yes.

^a ECG morphology suggestive of RVOT or fascicular origin, negative family history, normal 12-lead ECG, and echocardiogram.

^b e.g. atrioventricular conduction abnormalities, Q waves, broad QRS complex, ST/T waves deviations, abnormally high or low voltages. Ventricular dysfunction/dilatation/hypertrophy/wall thinning, wall motion abnormalities, multitopic PVCs/NSVTs/increasing ventricular arrhythmia (VA) burden with exercise.

^c e.g. Brugada pattern, long/short QT, polymorphic/bidirectional VA with exercise.

^d Diagnostic test to exclude CAD according to patient profile and symptoms.

^e Consider re-evaluation in case of new symptoms or changes in patient clinical condition.

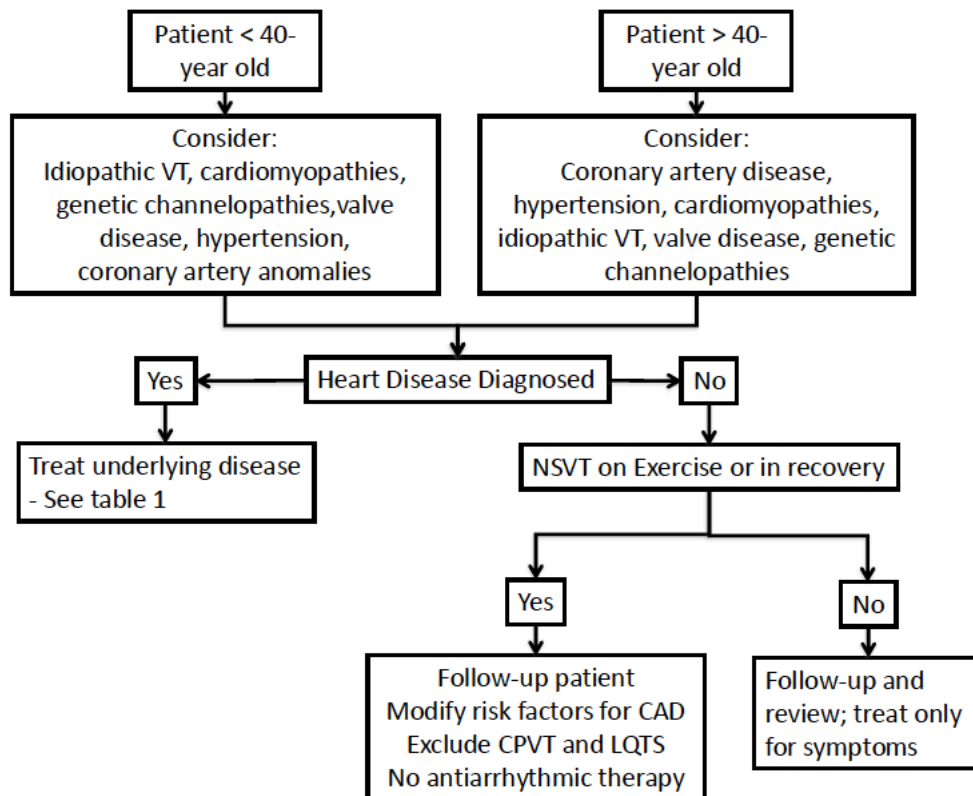


Figure 19. Clinical approach to the patient with NSVT.

(CAD: coronary artery disease, CPVT: catecholaminergic polymorphic ventricular tachycardia, LQTS: long QT syndromes)

Katritsis D, et al. Non-sustained ventricular tachycardia. JACC 2012;60:1993-2004.

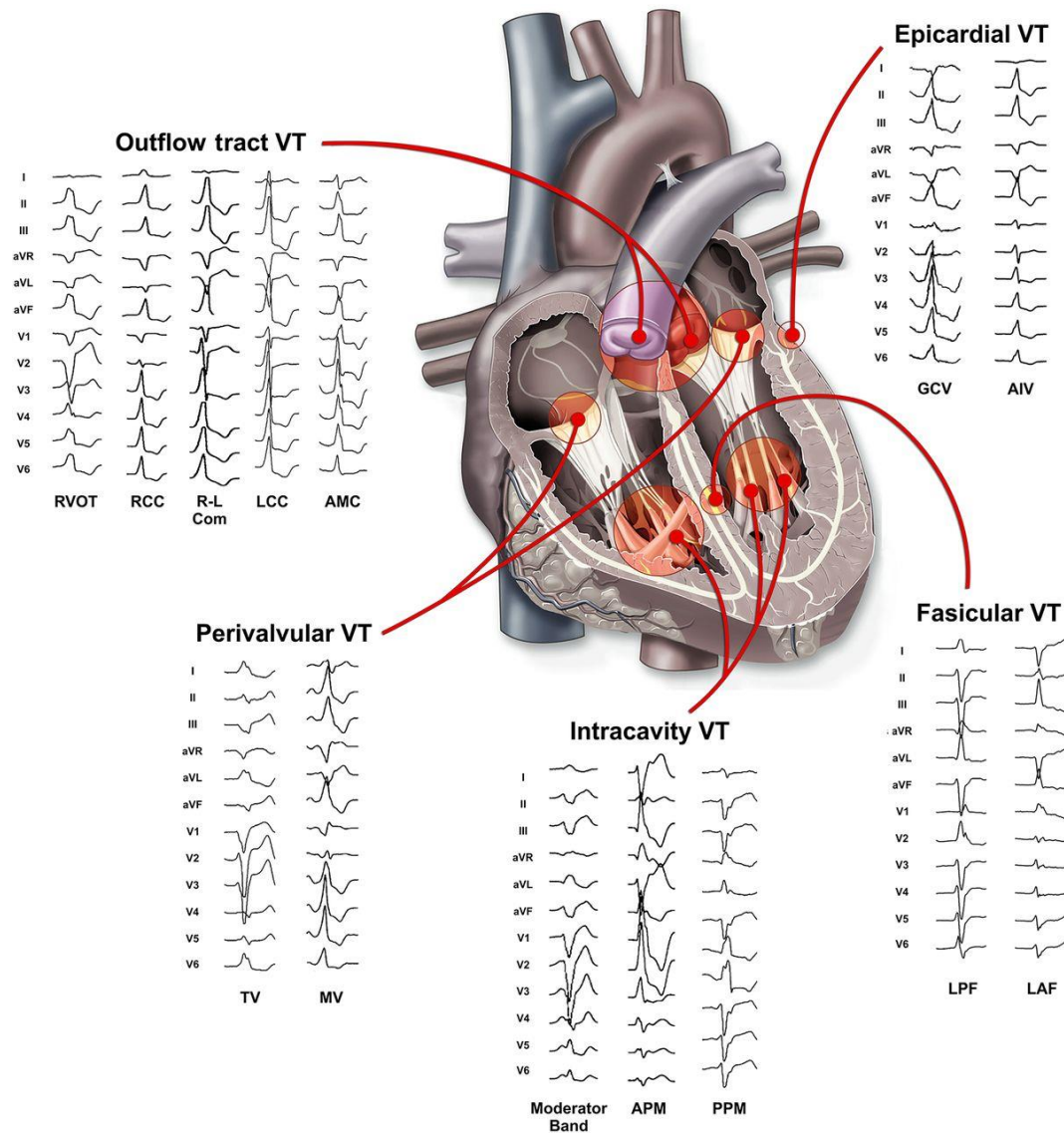


Figure 20. Twelve-lead electrocardiogram morphology of different sites of origin in idiopathic ventricular tachycardia (RVOT, right ventricular outflow tract; RCC, right coronary cusp; R-L com, right-left coronary cusp commissure; LCC, left coronary cusp; AMC, aortomitral continuity; TV, tricuspid annulus; MV, mitral annulus; APM, anterior PAP; PPM, posterior PAP; LPF, left posterior fascicle; LAF, left anterior fascicle; GCV, greater cardiac vein; AIV, anterior inter-ventricular vein).

Table 15. Ventricular arrhythmias in the structurally normal heart**ACC/AHA/HRS 2017 GL on VA/HRS.****VA in the structurally normal heart.**

Beta blocker or nondihydropyridine calcium channel blocker in symptomatic PVCs to reduce recurrent arrhythmias and improve symptoms	I-B-R
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In symptomatic VA, antiarrhythmic medication to reduce recurrent arrhythmias and improve symptoms if beta blockers and nondihydropyridine calcium channel blockers are ineffective or not tolerated	Ia-B-R
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Outflow Tract and Atrioventricular Annular VA

Catheter ablation for symptomatic outflow tract VA when antiarrhythmic medications are ineffective, not tolerated, or not the patient's preference.	I-B-NR
---	--------

Beta blocker or calcium channel blocker for symptomatic outflow tract VT in an otherwise normal heart.	I-B-NR
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Papillary Muscle VA (PVCs and VT)

Catheter ablation for symptomatic VA arising from the papillary muscles when antiarrhythmic medications are ineffective, not tolerated, or not the patient's preference.	I-B-NR
--	--------

Interfascicular Reentrant VT (Belhassen Tachycardia)

Catheter ablation for verapamil-sensitive, idiopathic LVT related to interfascicular reentry when antiarrhythmic medications are ineffective, not tolerated, or not the patient's preference.	I-B-NR
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IV verapamil for termination sustained hemodynamically tolerated verapamil-sensitive, idiopathic LVT related to interfascicular reentry.	I-B-NR
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Oral verapamil for recurrent verapamil-sensitive idiopathic LVT	Ia-C-LD
---	---------

PVC-induced cardiomyopathy

Catheter ablation for patients who require arrhythmia suppression for symptoms or declining ventricular function suspected to be due to frequent PVCs (generally >15% of beats and predominately of 1 morphology) and for whom Antiarrhythmic medications are ineffective, not tolerated, or not the patient's preference.	I-B-NR
--	--------

Pharmacological treatment (e.g., beta blocker, amiodarone) PVC-induced cardiomyopathy to reduce recurrent arrhythmias and improve symptoms and LV function.	Ia-B-NR
---	---------

Table 16. ESC 2022 GL on VA and SCD. Recommendations for the management of patients with idiopathic premature ventricular complexes/ventricular tachycardia

General recommendations

Regular assessment of ventricular function of patients with PVC burden >10% and normal ventricular function is indicated. I C

CMR despite a normal echocardiogram in patients with PVCs/VT and a presentation not typical for an idiopathic origin,^a IIa C

Treatment

Catheter ablation as first-line treatment for symptomatic idiopathic VT/PVCs from the RVOT or the left fascicles.^b I B

Beta-blockers or non-dihydropyridine CCBs in symptomatic patients with idiopathic VT/PVCs from an origin other than the RVOT or the left fascicles. I C

Beta-blockers, non-dihydropyridine CCBs, or flecainide when catheter ablation is not available, desired, or is particularly risky in symptomatic patients with idiopathic VT/PVCs from the RVOT or the left fascicles. IIa B

Catheter ablation or flecainide in symptomatic patients with idiopathic VT/PVCs from an origin other than the RVOT or the left fascicles. IIa C

Catheter ablation for idiopathic VT/PVCs in asymptomatic patients with repeatedly more than 20% of PVCs per day at follow-up. IIb B

Catheter ablation of idiopathic VT/PVCs is not recommended in children <5 years of age or <10 kg weight except when previous medical therapy fails or when VT is not haemodynamically tolerated. III C

Amiodarone as a first-line treatment is not recommended in patients with idiopathic VTs/PVCs. III C

Verapamil is not recommended in children <1 year of age with PVC/VT, particularly if they have signs of heart failure or concurrent use of other AADs. III C

AAD, anti-arrhythmic drug; CCB, calcium channel blocker; CMR, cardiac magnetic resonance; PVC, premature ventricular complex; RVOT, right ventricular outflow tract; SMVT, sustained monomorphic ventricular tachycardia; VT, ventricular tachycardia.

^a Including but not limited to older age, right bundle branch block (RBBB) morphology, SMVT consistent with re-entry.

^b Level of evidence C for VT/PVCs from left fascicles.

Table 17. ESC 2022 GL on VA and SCD. Summary of the recommendations for the treatment of patients with frequent idiopathic premature ventricular complexes/ventricular tachycardia or premature ventricular complex-induced cardiomyopathy.

	Ablation	Beta-blocker	CCB	Flecainide	Amiodarone
RVOT/fascicular PVC/VT: Symptomatic, normal LV function	Class I	Class IIa	Class IIa	Class IIa	Class III
PVC/VT other than RVOT/fascicular: Symptomatic, normal LV function	Class IIa	Class I	Class I	Class IIa	Class III
RVOT/fascicular PVC/VT: LV dysfunction	Class I	Class IIa	Class III ^a	Class IIa ^b	Class IIa
PVC/VT other than RVOT/fascicular: LV dysfunction	Class I	Class IIa	Class III ^a	Class IIa ^b	Class IIa
PVC: Burden >20%, asymptomatic, normal LV function	Class IIb				Class III

CCB, calcium channel blocker; LV, left ventricular; PVC, premature ventricular complex; RVOT, right ventricular outflow tract; VT, ventricular tachycardia.

^aIntravenous calcium channel blockers.

^bIn selected patients (only moderate LV dysfunction).

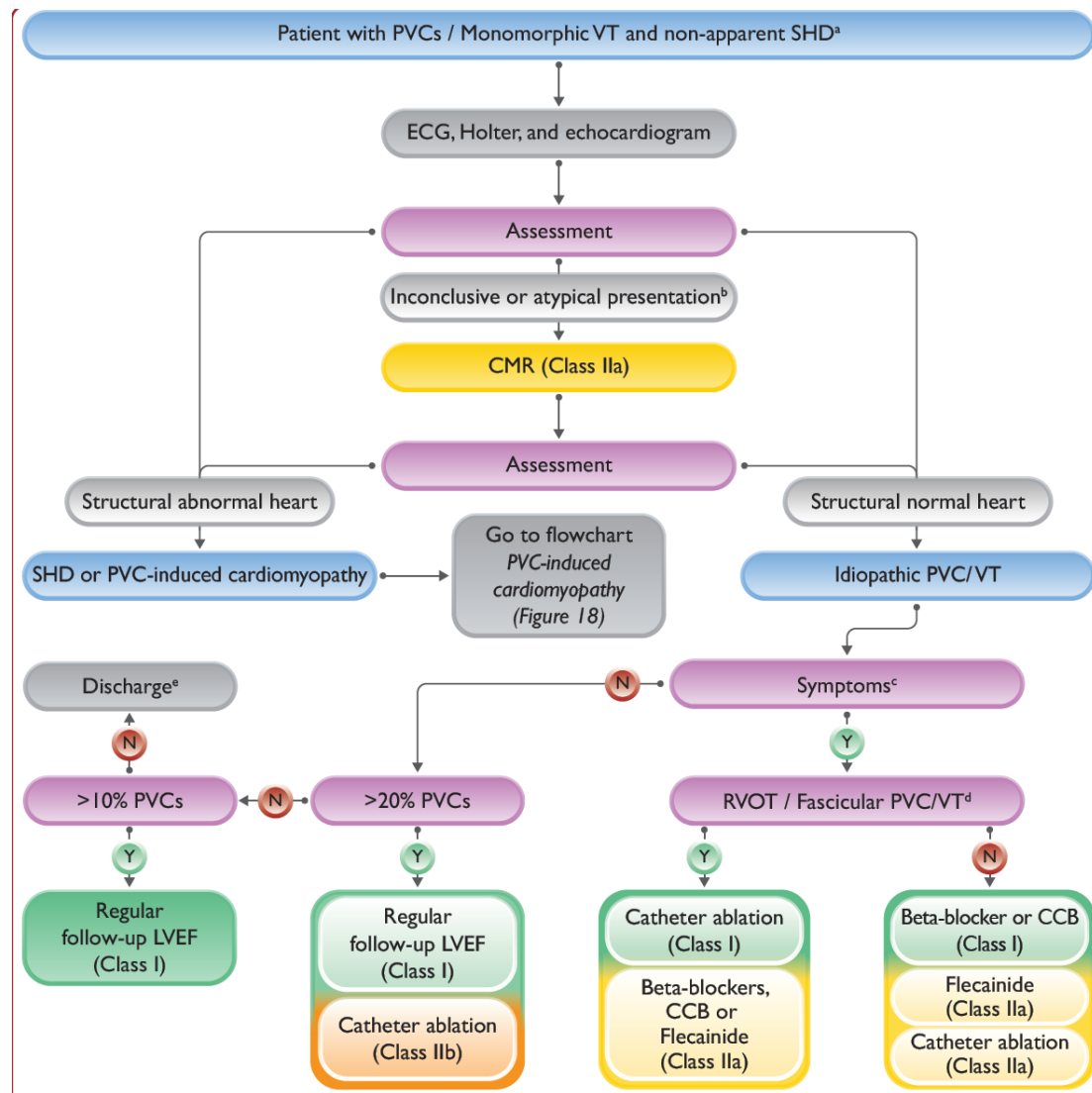


Figure 21. ESC 2022 GL on VA and SCD. Algorithm for the management of patients with idiopathic premature ventricular complexes/ventricular tachycardia and non-apparent structural heart disease.

CCB, calcium channel blocker; CMR, cardiac magnetic resonance; ECG, electrocardiogram; LVEF, left ventricular ejection fraction; N, No; PVC, premature ventricular complex; RVOT, right ventricular outflow tract; SHD, structural heart disease; VT, ventricular tachycardia; Y, Yes.

^a Non-apparent SHD is defined by lack of significant abnormalities in physical examination, basal ECG, and echocardiogram.

^b Atypical presentation: e.g. older age, right bundle branch block morphology, sustained monomorphic VT consistent with re-entry.

^c Symptoms should be relevant and related to PVC/VT.

^d Origin suspected by ECG or confirmed during electrophysiological evaluation. ^eConsider re-evaluation in case of new symptoms or changes in patient clinical condition.

Table 18. Idiopathic VF**ACC/AHA/HRS 2017 GL on VA/HRS. Idiopathic Polymorphic VT/VF**

Evaluation for genetic arrhythmia syndromes in young patients (<40 years of age) with unexplained SCA, unexplained near drowning, or recurrent exertional syncope, who do not have ischemic or other structural heart disease.	I-B-NR
ICD* in patients resuscitated from SCA due to idiopathic polymorphic VT or VF.	I-B-NR
Catheter ablation for recurrent episodes of idiopathic VF initiated by PVCs with a consistent QRS morphology.	I-B-NR

*: If meaningful survival greater than 1 year is expected

ESC 2022 GL on VA and SCD. Recommendations for the management of patients with idiopathic ventricular fibrillation***Diagnostic evaluation***

Idiopathic VF is diagnosed in a SCA survivor, preferably with documentation of VF, after exclusion of an underlying structural, channelopathic, metabolic, or toxicological aetiology.	I B
Clinical testing (history, ECG and high precordial lead ECG, exercise test, echocardiogram) of first-degree family members of idiopathic VF patients.	IIb B
Genetic testing of genes related to channelopathy and cardiomyopathy.	IIb B

Secondary prevention of SCD and treatment of VA

ICD implantation in idiopathic VF.	I B
Isoproterenol infusion, verapamil, or quinidine for acute treatment of an electrical storm or recurrent ICD discharges in idiopathic VF.	IIa C
Quinidine should be considered for chronic therapy to suppress an electrical storm or recurrent ICD discharges in idiopathic VF.	IIa B
Catheter ablation by experienced electrophysiologists in idiopathic VF patients with recurrent episodes of VF triggered by a similar PVC non-responsive to medical treatment.	IIa C

ECG, electrocardiogram; ICD, implantable cardioverter defibrillator; PVC, premature ventricular complex; SCA, sudden cardiac arrest; SCD, sudden cardiac death; VA, ventricular arrhythmia; VF, ventricular fibrillation.

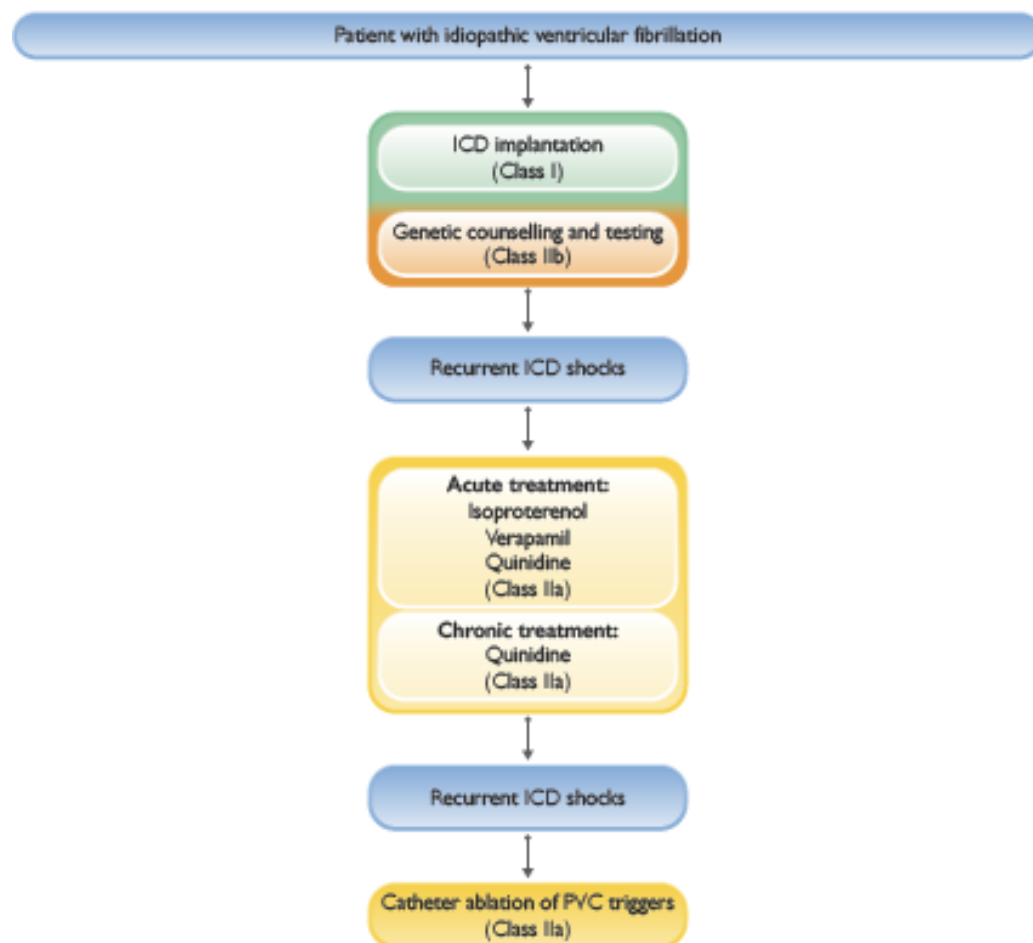


Figure 22. ESC 2022 GL on VA and SCD. Algorithm for the management of patients with idiopathic ventricular fibrillation.

ICD, implantable cardioverter defibrillator; PVC, premature ventricular complex.

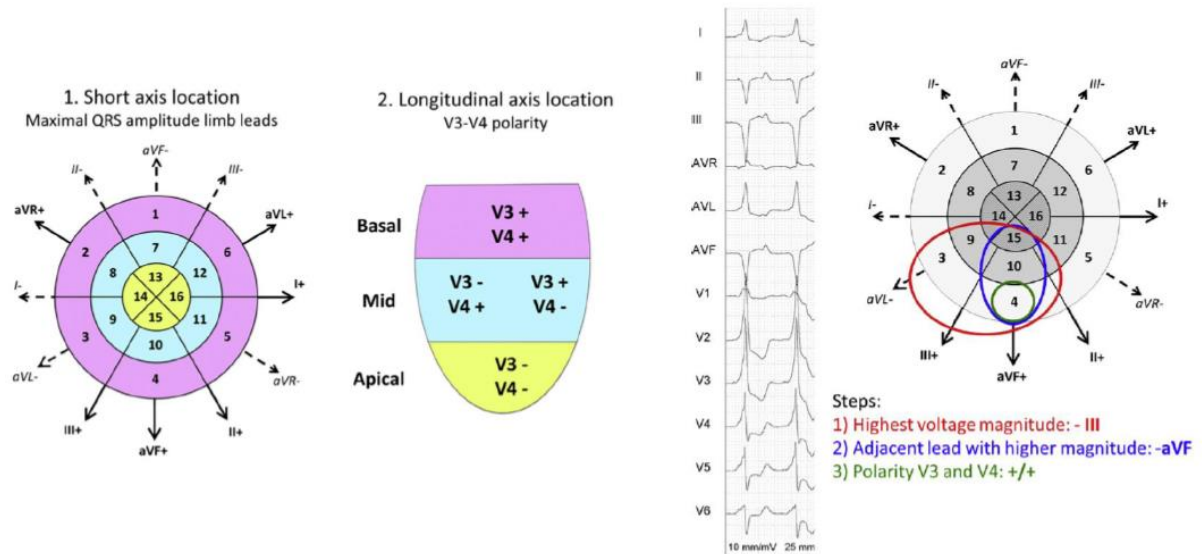


Figure 23. Localization of the origin of scar-related VT.

Left panel: The QRS axis-based algorithm. The figure shows the 17-segment model from the American Heart Association superimposed on a representation of the QRS axis and all the limb leads. Steps to identify the segment of origin of a ventricular arrhythmia: (1) Identify the limb lead with the highest voltage magnitude (positive or negative). If this magnitude is in lead I, II, or III, analyze the adjacent leads. The adjacent limb lead with higher magnitude will determine the group of segments suggested as a possible site of origin. (2) Identify the positivity or negativity of the precordial leads V3 and V4; concordance indicates a basal or apical origin, respectively. Other combinations indicate a medial origin.

Right panel: Example of the application of the QRS axis-based algorithm. A: An ECG of VT. B: The application of the QRS axis-based graphical algorithm. In the ECG of panel A, the lead with the highest voltage magnitude is III2. Applying the first step of the algorithm, the segment of origin of VT is one of the groups selected by the red circle. By analyzing the voltage magnitude of the adjacent leads of III2 (aVF2 and aVL1), it is possible to determine that aVF2 has more voltage magnitude than aVL1. As a result, the group of segments selected by the algorithm is the one marked with the blue circle. The last step of the algorithm (positivity or negativity in leads V3 and V4) indicates a basal origin of VT (leads V3 and V4 are both more positive than negative). The final selected segment by the algorithm is 4, marked with the green circle. ECG 5 electrocardiogram; VT 5 ventricular tachycardia.

Andreu D, et al. A QRS axis-based algorithm to identify the origin of scar-related ventricular tachycardia in the 17-segment American Heart Association model. *Heart Rhythm*. 2018;15:1491-1497.

Table 19. ACC/AHA/HRS 2017 GL on VA/SCD. Ischaemic heart disease.**Surgery and Revascularization Procedures in Patients With Ischemic Heart Disease**

Evaluated for ischemic heart disease, and revascularize patients with sustained VA and survivors of SCA	I-B-NR
---	--------

Repair or revascularization in anomalous origin of a coronary artery suspected to be the cause of SCA	I-C-EO
---	--------

Surgery for Arrhythmia Management

Surgical ablation in monomorphic VT refractory to antiarrhythmic medications and attempts at catheter ablation.	IIb-C-LD
---	----------

Secondary Prevention of SCD in Patients With Ischemic Heart Disease

ICD if meaningful survival greater than 1 year is expected in:

Survivors of SCA due to VT/VF or in hemodynamically unstable VT	I-B-R
---	-------

Stable VT not due to reversible causes	I-B-NR
--	--------

Unexplained syncope and inducible sustained monomorphic VT	I-B-NR
--	--------

A transvenous ICD provides intermediate value in the secondary prevention of SCD particularly when the patient's risk of death due to a VA is deemed high and the risk of nonarrhythmic death (either cardiac or noncardiac) is deemed low based on the patient's burden of comorbidities and functional status	Value statement (B-R)
---	-----------------------

Primary Prevention of SCD in Patients With Ischemic Heart Disease

ICD if meaningful survival of greater than 1 year is expected in:

LVEF $\leq$ 35% due to ischemic heart, at least 40 days' post-MI and at least 90 days postrevascularization, and with NYHA class I-III HF despite GDMT.	I-A
---	-----

NSVT due to prior MI, LVEF $\leq$ 40% and inducible sustained VT or VF at electrophysiological study	I-B-R
--	-------

Nonhospitalized patients with NYHA class IV who are candidates for cardiac transplantation or an LVAD	IIa-B-NR
---	----------

An ICD is not indicated for NYHA class IV patients with medication-refractory HF who are not also candidates for cardiac transplantation, an LVAD, or a CRT defibrillator that incorporates both pacing and defibrillation capabilities.	III-C-EO No benefit
--	------------------------

A transvenous ICD provides intermediate value in the secondary prevention of SCD particularly when the patient's risk of death due to a VA is deemed high and the risk of nonarrhythmic death (either cardiac or noncardiac) is deemed low based on the patient's burden of comorbidities and functional status	Value statement (B-R)
---	-----------------------

Recurrent VA in Patients With Ischemic Heart Disease

Amiodarone or sotalol in significant symptoms or ICD shocks despite optimal device programming and treatment with a beta blocker recurrent	I-B-R
Catheter ablation in prior MI and recurrent episodes of symptomatic sustained VT, or presentation with VT or VF storm and failure or intolerance of:	
amiodarone	I-B-R
other antiarrhythmic medications	I-B-NR
Catheter ablation in ischemic heart disease and ICD shocks for sustained monomorphic VT or recurrent, hemodynamically tolerated sustained monomorphic VT	IIb-C-LD
In prior MI, class IC antiarrhythmic medications (e.g., flecainide and propafenone) should not be used	III-B-R Harm
In incessant VT or VF, an ICD should not be implanted until sufficient control of the VA is achieved to prevent repeated ICD shocks	III-C-LD Harm
Coronary revascularization alone is an ineffective therapy to prevent recurrent sustained monomorphic VT	III-C-LD No benefit

ACC/AHA/SCAI 2021 GL on Revascularization. Revascularization in Patients to Reduce Ventricular Arrhythmias

In patients with ventricular fibrillation, polymorphic ventricular tachycardia (VT), or cardiac arrest, revascularization of significant CAD to improve survival.	I B-NR
In patients with CAD and suspected scar mediated sustained monomorphic VT, revascularization is not recommended for the sole purpose of preventing recurrent VT.	III C-LD (No benefit)

Table 20. ESC 2022 GL on VA and SCD. Ischaemic heart disease and coronary artery anomalies

Recommendations for risk stratification, sudden cardiac death prevention, and treatment of ventricular arrhythmias in chronic coronary artery disease

Risk stratification and primary prevention of SCD

In patients with syncope and previous STEMI, PES hen syncope remains unexplained after non-invasive evaluation.	I C
ICD therapy in patients with CAD, symptomatic heart failure (NYHA class II–III), and LVEF $\leq 35\%$ despite ≥ 3 months of OMT.	I A
ICD therapy in patients with CAD, NYHA class I, and LVEF $\leq 30\%$ despite ≥ 3 months of OMT.	IIa B
ICD implantation in patients with CAD, LVEF $\leq 40\%$ despite ≥ 3 months of OMT, and NSVT, if they are inducible for SMVT by PES.	IIa B
In patients with CAD, prophylactic treatment with AADs other than beta-blockers is not recommended.	III A

Secondary prevention of SCD and treatment of VAs

ICD implantation in patients without ongoing ischaemia with documented VF or haemodynamically not-tolerated VT occurring later than 48 h after MI.	I A
In patients with CAD and recurrent, symptomatic SMVT, or ICD shocks for SMVT despite chronic amiodarone therapy, catheter ablation in preference to escalating AAD therapy.	I B
Addition of oral amiodarone or beta-blocker replacement by sotalol in patients with CAD with recurrent, symptomatic SMVT, or ICD shocks for SMVT while on beta-blocker treatment.	IIa B
In patients with CAD and haemodynamically well-tolerated SMVT and LVEF $\geq 40\%$, Catheter ablation in experienced centres as an alternative to ICD therapy, provided that established endpoints have been reached.*	IIa C
ICD implantation in patients with a haemodynamically tolerated SMVT and an LVEF $\geq 40\%$ if VT ablation fails, is not available, or is not desired.	IIa C
Catheter ablation in patients with CAD and recurrent, symptomatic SMVT, or ICD shocks for SMVT despite beta-blockers or sotalol treatment.	IIa C
In patients with CAD eligible for ICD implantation, catheter ablation just before (or immediately after) ICD implantation to decrease subsequent VT burden and ICD shocks.	IIb C

AAD, anti-arrhythmic drug; CAD, coronary artery disease; ICD, implantable cardioverter defibrillator; LVEF, left ventricular ejection fraction; MI, myocardial infarction; NSVT, non-sustained ventricular tachycardia; NYHA, New York Heart Association; OMT, optimal medical therapy; PES, programmed electrical stimulation; SCD, sudden cardiac death; SMVT, sustained monomorphic ventricular tachycardia; STEMI, ST elevation myocardial infarction; VA, ventricular arrhythmia; VF, ventricular fibrillation; VT, ventricular tachycardia.

*: VT non-inducibility and elimination of electrograms consistent with conduction delay.

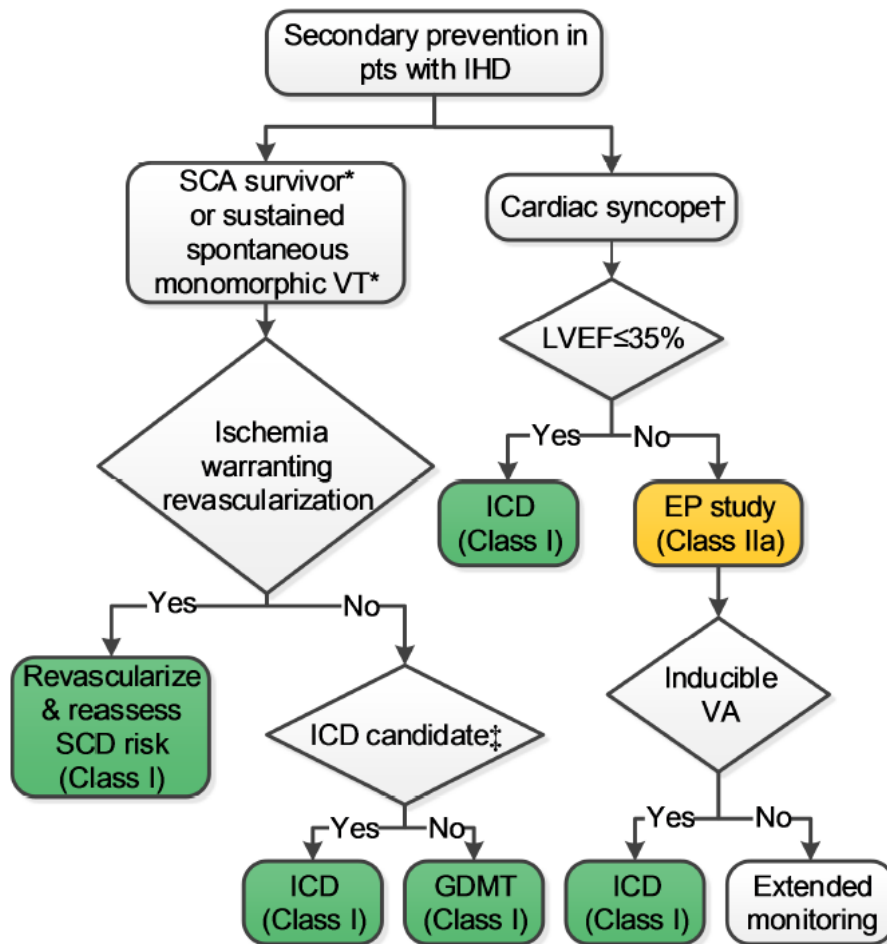


Figure 24. ACC/AHA/HRS 2017. Secondary Prevention in Patients With Ischemic Heart Disease

*Exclude reversible causes.

†History consistent with an arrhythmic etiology for syncope.

‡ICD candidacy as determined by functional status, life expectancy, or patient preference.

EP indicates electrophysiological; GDMT, guideline-directed management and therapy; ICD, implantable cardioverter defibrillator; IHD, ischemic heart disease; LVEF, left ventricular ejection fraction; pts, patients; SCA, sudden cardiac arrest; SCD, sudden cardiac death; and VT, ventricular tachycardia.

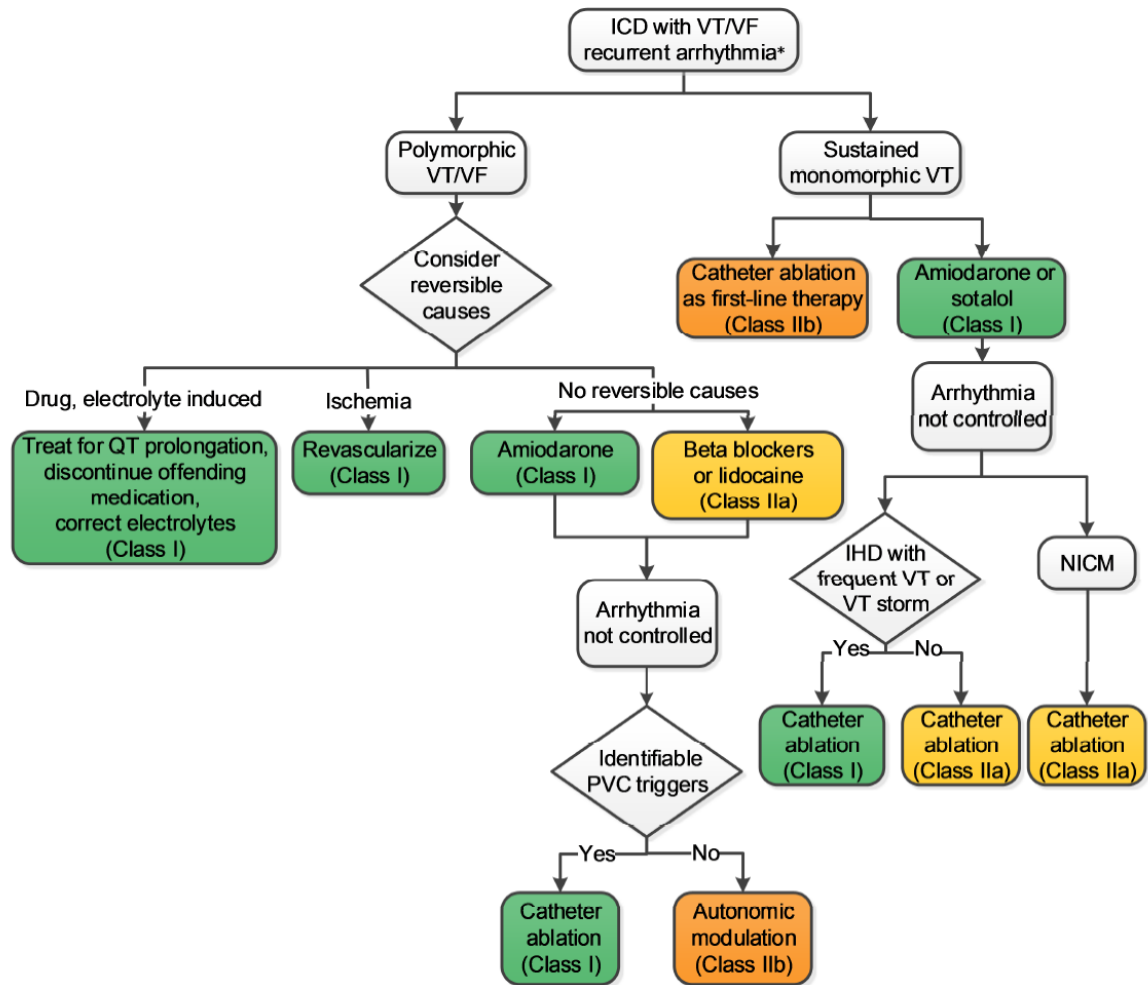


Figure 25. ACC/AHA/HRS 2017. Treatment of Recurrent VA in Patients With Ischemic Heart Disease or NICM

*Management should start with ensuring that the ICD is programmed appropriately and that potential precipitating causes, including heart failure exacerbation, are addressed. For information regarding optimal ICD programming, refer to the 2015 HRS/EHRA/APHRS/SOLAECE expert consensus statement.

APHRS indicates Asia Pacific Heart Rhythm Society; EHRA, European Heart Rhythm Association; HRS, Heart Rhythm Society; IHD, ischemic heart disease; ICD, implantable cardioverter-defibrillator; PVC, premature ventricular complex; NICM, nonischemic cardiomyopathy; SOLAECE, Sociedad Latinoamericana de Estimulación Cardíaca y Electrofisiología; VF, ventricular fibrillation; and VT, ventricular tachycardia.

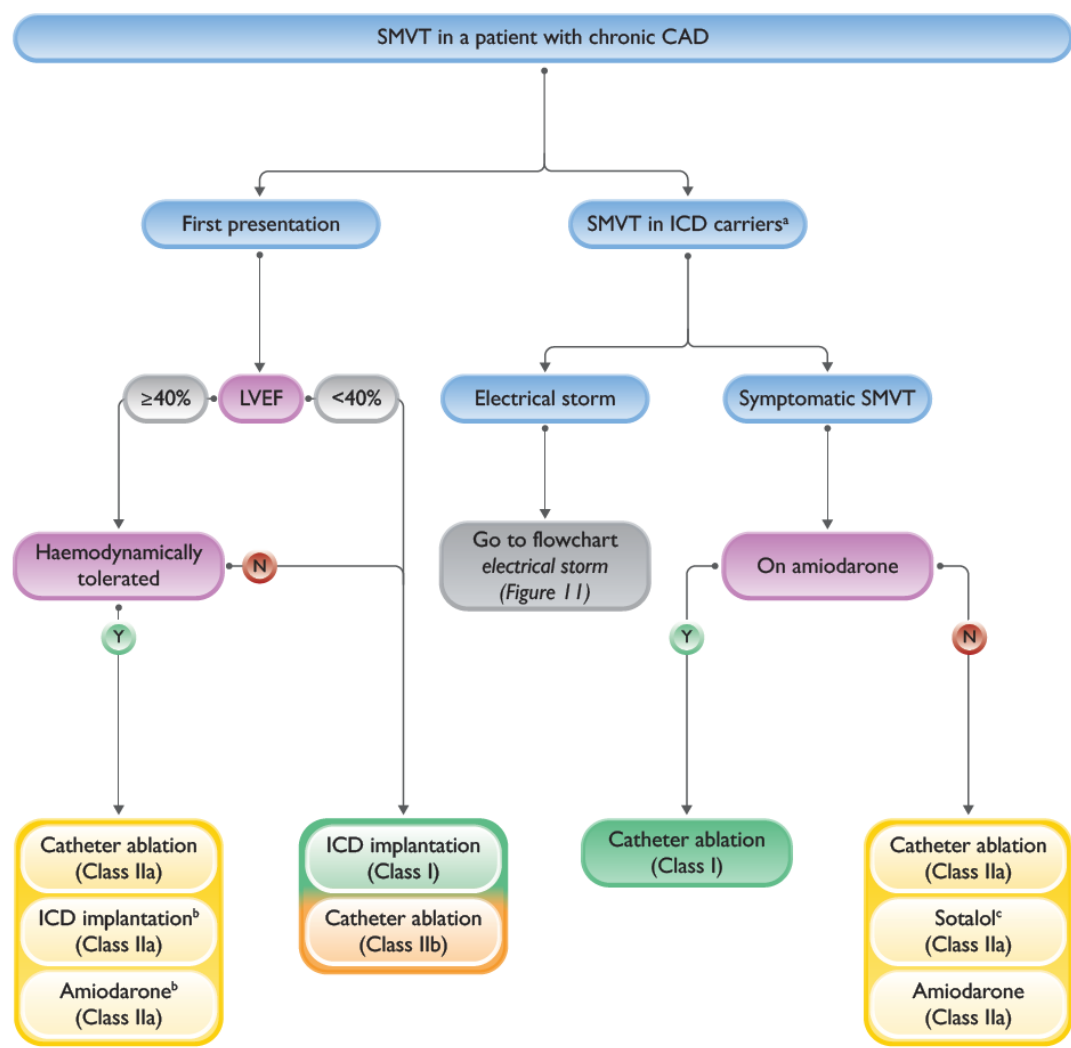


Figure 26. ESC 2022 GL on VA and SCD. Algorithm for the management of sustained monomorphic ventricular tachycardia in patients with chronic coronary artery disease.

CAD, coronary artery disease; ICD, implantable cardioverter defibrillator; LVEF, left ventricular ejection fraction; N, No; SMVT, sustained monomorphic ventricular tachycardia; Y, Yes.

^a Incessant ventricular tachycardia in monitor zone: consider catheter ablation.

^b If catheter ablation is not available, not successful or not desired by the patient.

^c To reduce ICD shocks.

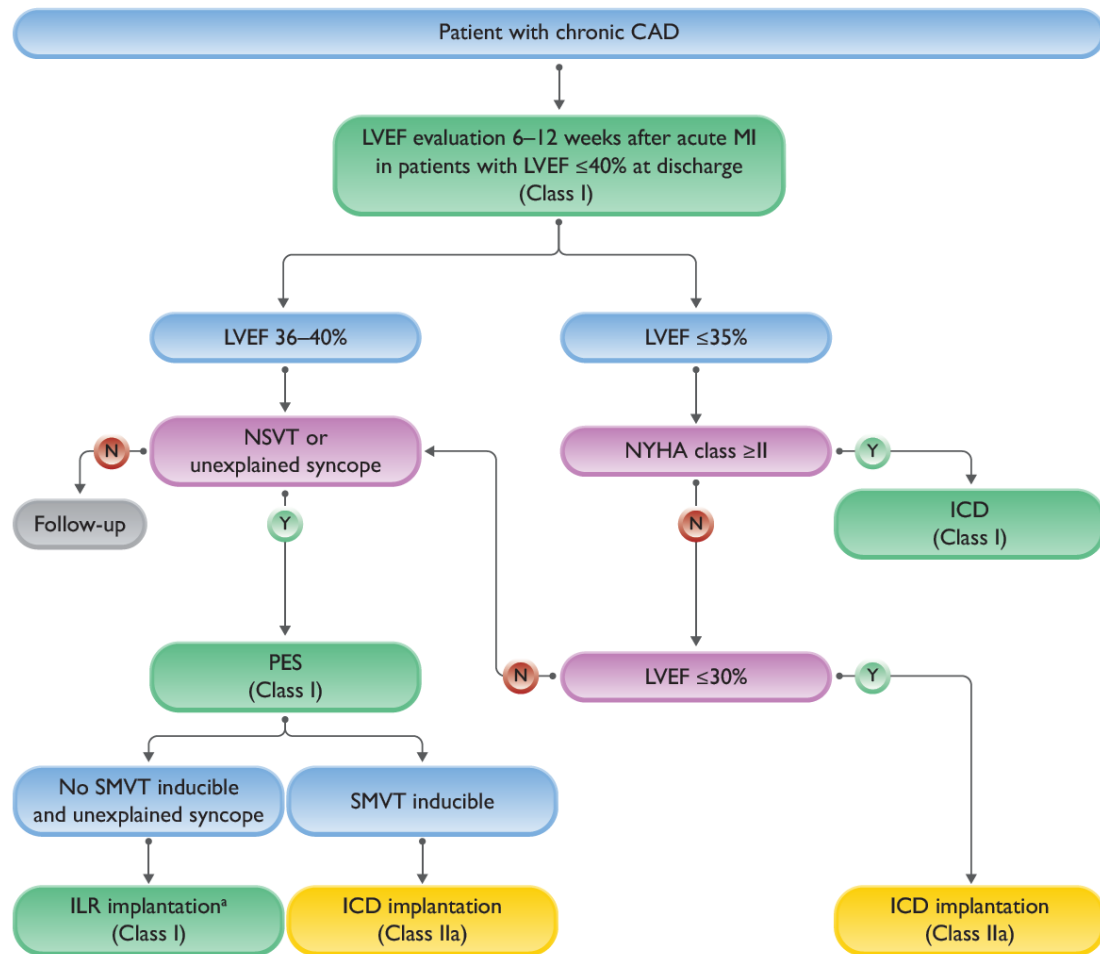


Figure 28. ESC 20022 GL on VA and SCD. Algorithm for risk stratification and primary prevention of sudden cardiac death in patients with chronic coronary artery disease and reduced ejection fraction.

CAD, coronary artery disease; ICD, implantable cardioverter defibrillator; ILR, implantable loop recorder; LVEF, left ventricular ejection fraction; MI, myocardial infarction; N, No; NSVT, non-sustained ventricular tachycardia; NYHA, New York Heart Association; PES, programmed electrical stimulation; SMVT, sustained monomorphic ventricular tachycardia; Y, Yes.

^aThe 2018 ESC Guidelines for the diagnosis and management of syncope.

Table 21. QT Interval–Prolonging Drugs Known to Cause TdP

Drug Class	Drug	Incidence, %	Mechanism
Anesthetic	Propofol	4.93/million	I_{Ks} and I_{to} inhibition
	Sevoflurane	...	
Antiarrhythmic	Amiodarone	0.7–1.5*	I_{Kr} inhibition
	Disopyramide	...	I_{Kr} inhibition
	Dofetilide	4–10†	I_{Kr} inhibition, I_{Na-L} augmentation
	Dronedarone	<0.1	I_{Kr} inhibition
	Flecainide	...	I_{Kr} inhibition
	Hydroquinidine	...	I_{Kr} inhibition
	Ibutilide	1.2–11.5	I_{Kr} inhibition, I_{Na-L} augmentation
	Procainamide	0.3–6	I_{Kr} inhibition‡
	Quinidine	2–12	I_{Kr} , I_{K1} and I_{to} inhibition
	Sotalol	0.2–23.6	I_{Kr} inhibition,§ I_{Na-L} augmentation§
Antibiotic	Azithromycin	0.97¶	I_{Kr} inhibition, I_{Na-L} augmentation
	Ciprofloxacin	...	I_{Kr} inhibition
	Clarithromycin	...	I_{Kr} inhibition
	Erythromycin	0.4	I_{Kr} inhibition, I_{Na-L} augmentation
	Levofloxacin	0.2	I_{Kr} inhibition
	Moxifloxacin	...	I_{Kr} inhibition
	Roxithromycin	...	I_{Kr} inhibition
Anticancer	Aclarubicin	...	—
	Arsenic trioxide	...	Inhibition of I_{Kr} trafficking
	Oxaliplatin	0.07	...
	Vandetanib	1.4–2.1	I_{Kr} inhibition
Antidepressant (SSRI)	Citalopram	...	I_{Kr} inhibition and inhibition of I_{Kr} trafficking
	Escitalopram	...	I_{Kr} inhibition and inhibition of I_{Kr} trafficking
Antiemetic	Domperidone	...	I_{Kr} inhibition
	Droperidol	<0.1	I_{Kr} inhibition
	Ondansetron	...	I_{Kr} inhibition
Antifungal	Fluconazole	...	I_{Kr} inhibition and inhibition of I_{Kr} trafficking
	Pentamidine	Up to 21	Inhibition of I_{Kr} trafficking
Antimalarial	Chloroquine	...	I_{Kr} inhibition
	Hydroxychloroquine	...	I_{Kr} inhibition
	Halofantrine	...	I_{Kr} inhibition
Antipsychotic	Chlorpromazine	...	I_{Kr} inhibition
	Haloperidol	3.6	I_{Kr} inhibition
	Levomepromazine	...	...
	Levosulpride	...	...
	Pimozide	...	I_{Kr} inhibition
	Sulpiride	...	...
	Sultopride	...	...
	Thioridazine	...	I_{Kr} inhibition, I_{Na-L} augmentation
Cholinesterase inhibitor	Donepezil	...	I_{Kr} inhibition
Coronary vasodilator	Papaverine HCL	...	I_{Kr} inhibition¶
Illicit	Cocaine	...	I_{Kr} inhibition
Muscle relaxant	Terodiline	...	I_{Kr} inhibition
Opioid agonist	Methadone	...	I_{Kr} inhibition
Phosphodiesterase-3 inhibitor	Anagrelide	...	...
	Cilostazol	...	...
Psychoactive	Ibogaine	...	I_{Kr} inhibition
Toxin	Cesium chloride	...	I_{Kr} inhibition
Vasoconstrictor	Terlipressin	...	...

HCL, hydrochloride; I_{Kr} , rapid component of delayed rectifier potassium current; I_{Ks} , slow component of delayed rectifier potassium current; I_{K1} , inwardly rectifying potassium current; I_{NaL} , late sodium current; I_{to} , transient outward potassium current; SSRI, selective serotonin reuptake inhibitor; TdP, torsades de pointes; and ..., unknown.

*0.7% to 0.8% with oral administration; 1.5% associated with intravenous administration.

†Incidence is higher in patients with heart failure with reduced ejection fraction and can be as high as 10% if higher-than-recommended doses are administered or if dose is not appropriately adjusted in patients with kidney disease.

‡Primarily by the active metabolite n-acetylprocainamide.

§d-Sotalol enantiomer.

||0.97% of patients with heart rate-corrected QT interval >450 milliseconds during azithromycin therapy.

¶Possibly because of the preservative chlorbutanol.

Drug-Induced Arrhythmias: A Scientific Statement From the American Heart Association. *Circulation*. 2020

Table 22. Management of Drug-Induced Arrhythmias

ACC/AHA/HRS 2017 GL on VA/SCD. Medication-Induced Arrhythmias

Digoxin

Digoxin antibodies for sustained VA potentially due to digoxin toxicity	I-B-NR
---	--------

Medication-Induced QT Prolongation and Torsades de Pointes

Increase the heart rate with atrial or ventricular pacing or isoproterenol in recurrent torsades de pointes associated with acquired QT prolongation and bradycardia that cannot be suppressed with IV magnesium.	I-B-NR
---	--------

IV magnesium for QT prolongation due to a medication, hypokalemia, hypomagnesemia, or other acquired factor and recurrent torsades de pointes.	I-C-LD
--	--------

Potassium repletion to ≥ 4.0 mmol/L and magnesium repletion to normal values (e.g., ≥ 2.0 mmol/L) torsades de pointes associated with acquired QT.	I-C-LD
--	--------

Sodium Channel Blocker–Related Toxicity

In patients taking sodium channel blockers who present with elevated defibrillation or pacing thresholds, discontinue the presumed responsible medication or reprogram the device.	IIa-C-LD
--	----------

In patients with congenital or acquired long QT syndrome, QT-prolonging medications are potentially harmful	III-B-NR Harm
---	------------------

Table 23. ESC 2022 GL on cardio-oncology.

Risk factors for drug-induced QT prolongation and torsade de pointes

Correctable	Non-correctable
QT-prolonging drugs ^a • Antiarrhythmics • Antibiotics • Antidepressants • Antifungals • Antiemetics • Antihistamines • Antipsychotics • Loop diuretics • Opioids (methadone) Bradyarrhythmia Electrolyte imbalance/abnormalities • Hypokalaemia (≤ 3.5 mEq/L) • Hypomagnesaemia (≤ 1.6 mEq/L) • Hypocalcaemia (≤ 8.5 mEq/L) Inadequate dose adjustment of renal or hepatic cleared QT-prolonging drugs	Acute myocardial ischaemia Age > 65 years Baseline QTc interval prolongation ^b Family history of sudden death (congenital LQTS or genetic polymorphism) Female sex Impaired renal function (for renally excreted drugs) Liver disease (for hepatically excreted drugs) Personal history of syncope or drug-induced TdP Pre-existing CVD (CAD, HF, LV hypertrophy)

CAD, coronary artery disease; CVD, cardiovascular disease; HF, heart failure; LQTS, long QT syndrome; LV, left ventricular; QTc, corrected QT interval; TdP, torsade de pointes.

^aSee <https://www.crediblemeds.org>.

^bQTc using Fridericia correction ($QTcF = QT / \sqrt[3]{RR}$) is recommended in patients with cancer.

Classification of corrected QT interval prolongation induced by cancer drug therapy

Classification	Drugs
High risk: QTcF prolongation ≥ 10 ms and risk of TdP	• Acarubicin • Arsenic trioxide • Glasdegib • Nilotinib • Oxaliplatin • Pazopanib • Ribociclib • Sunitinib • Toremifene • Vandetanib
Moderate risk: QTcF prolongation ≥ 10 ms and low or no risk of TdP (or uncertain)	• Abarelix • Belinostat • Brigantini • Carbozantinib • Ceritinib • Crizotinib • Dovitinib • Entrectinib • Eribulin • Gilteritinib • Ivosidenib • Lapatinib • Lenvatinib • Osimertinib • Panobinostat • Rucaparib • Selpercatinib • Sorafenib • Tipiracil/ trifluridine • Vemurafenib
Low risk: QTcF prolongation < 10 ms ^a	• ADT • Afatinib • Axitinib • Binimetinib • Bortezomib • Bosutinib • Carfilzomib • Dabrafenib • Dasatinib • Encorafenib • Midostaurin • Pertuzumab • Ponatinib • Romidepsin • Quizartinib • Tamoxifen • Vorinostat

ADT, androgen deprivation therapy; QTcF, corrected QT interval using Fridericia correction; TdP, torsade de pointes.

^a ADT may prolong the QTc interval (GnRH agonist, GnRH antagonist, bicalutamide, flutamide, apalutamide, darolutamide, enzalutamide, and abiraterone).

Developed from EMA prescribing information, DA prescribing information, and AZCERT.

Table 24. ESC 2022 GL on cardio-oncology. Recommendations for the management of long corrected QT interval and ventricular arrhythmias in patients receiving anticancer treatment

How to manage QTc prolongation in patients with cancer

Discontinuation of QTc-prolonging cancer therapy in patients who develop TdP or sustained ventricular tachyarrhythmias during treatment. I C

Temporary interruption of QTc-prolonging cancer therapy in patients who develop asymptomatic QTcF ≥ 500 ms and an ECG should be repeated every 24 h until resolution of the QTcF prolongation. I C

Immediate withdrawal of any offending drug and correction of electrolyte abnormalities and other risk factors^a in patients with cancer who develop QTcF ≥ 500 ms. I C

Weekly ECG monitoring in asymptomatic patients with cancer with QTcF 480–500 ms who are treated with a QTc-prolonging cancer therapy. I C

A 12-lead ECG after any dose increase of QTc-prolonging cancer therapy. I C

Restarting QTc-prolonging cancer therapy

A multidisciplinary discussion before restarting QTc-prolonging drugs in patients who have developed significant QTcF prolongation, to discuss alternative cancer treatments. I C

In patients who experienced significant QTcF prolongation, restarting the culprit QTc-prolonging cancer treatment ideally at a reduced dose according to each drug recommendation. IIb C

Weekly ECG monitoring during the first 4–6 weeks and then monthly thereafter in patients with cancer after restarting QTc-prolonging cancer therapy. I C

ECG, electrocardiogram; QTc, corrected QT interval; QTcF, corrected QT interval using Fridericia correction; TdP, torsade de pointes.

^a See <https://www.crediblemeds.org> and Table 20.

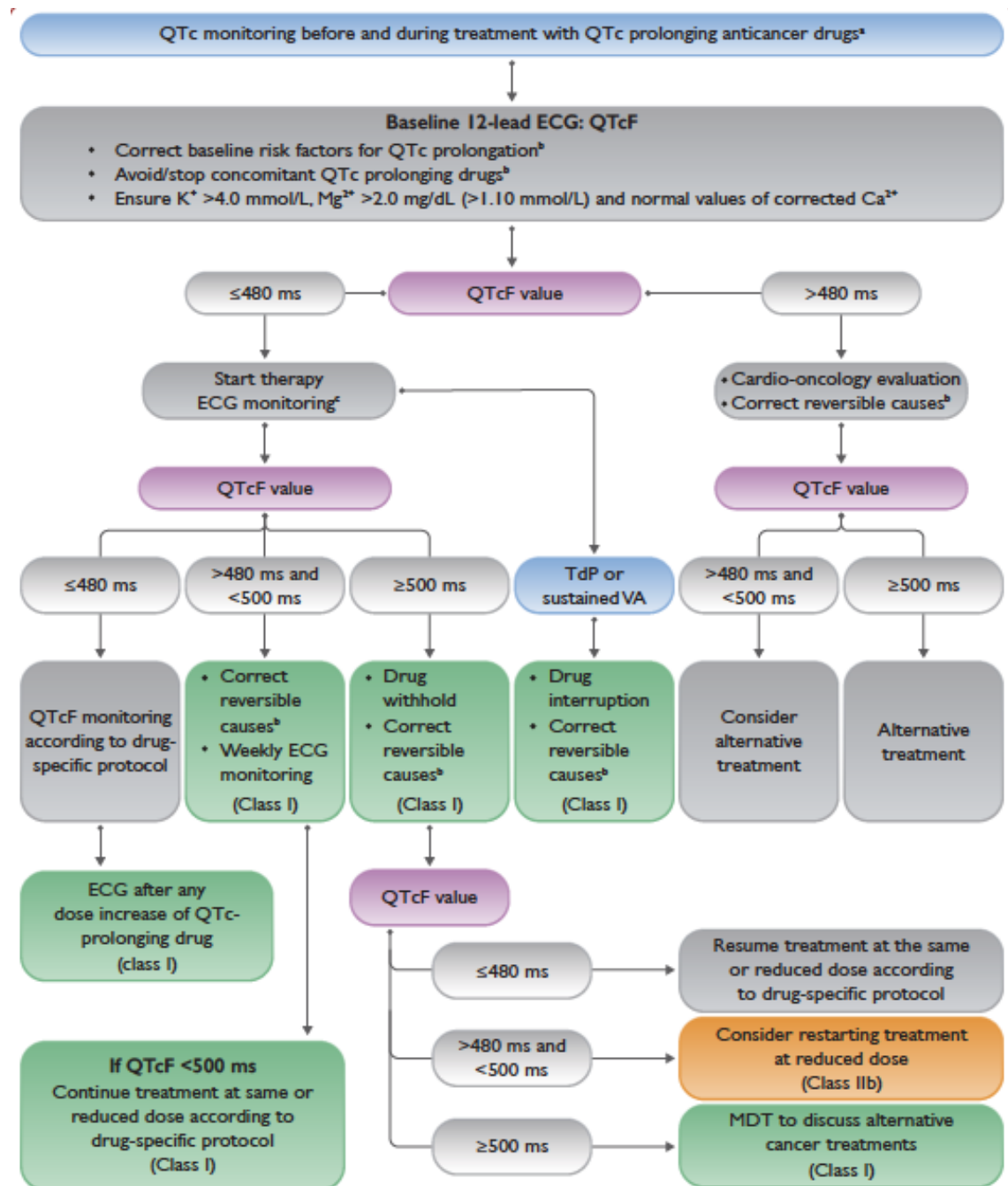


Figure 29. ESC 2022 GL on cardio-oncology. Corrected QT interval monitoring before and during treatment with corrected QT interval-prolonging anticancer drugs.

Ca^{2+} , calcium; ECG, electrocardiogram; K^+ , potassium; MDT, multidisciplinary team; Mg^{2+} , magnesium; QTc, corrected QT interval; QTcF, corrected QT interval using Fridericia correction; TdP, Torsade de pointes; VA, ventricular arrhythmias. QT interval using Fridericia correction ($QTcF = QT/3\sqrt{RR}$) is recommended in patients with cancer. Upper 99% limits of normal for QTc values in the general population are 450 ms for men and 460 ms for women.

a Table 20

b Table 20 and <https://www.crediblemeds.org>.

c ECG monitoring at baseline, once steady-state anticancer drug levels have been achieved, after each dose modification, or any treatment interruption >2 weeks; monthly for the first 3 months, and then periodically during treatment depending on patient-specific risk factors and cancer treatment.

Table 25. Effect of Cocaine and Methamphetamines on Ions Channels and EKG

	Cocaine	Methamphet-amine
Effect of agent on cardiac ion channels		
Na ⁺ (I _{Na})	↓	—
K ⁺	↓ (K _{ATP} , I _{Kr})	↓ (K _{to} and K _{ir})
Ca ²⁺	↓ & ↑ (I _{CaL} =increases with low cocaine concentration, I _{CaL} =decreases with high cocaine concentrations)	↓/↑ (I _{CaL})
Effect of agent on EKG		
PR interval	—	—
QRS duration	↑	
QTc interval	↑	+
ST-T wave changes	+	+
Atrial dysrhythmias		
Sinus tachycardia	+	+
Atrial fibrillation	+	±
Atrial tachycardias (including SVT)	+	+
Ventricular arrhythmias		
Ventricular tachycardia	+	+
Ventricular fibrillation	+	?
Torsade de pointes	+	+
Bradyarrhythmias		
Bradycardia	+	—
Heart block	+	—

↑ indicates increase in current or ECG parameter; ↓, decrease in current or ECG parameter; +, a known effect of the drug on the EKG parameters or cardiac arrhythmias; —, no effect or unknown effect; ?, unknown or controversial data on effect; I_{CaL}, L-type calcium channels; I_{Kr}= Rapidly activating delayed rectifier potassium channels; K_{ATP}, ATP sensitive potassium channels; K_{ir}, inwardly rectifying potassium channels; and K_{to}, transient outward potassium channel.

Dominic P, et al. Stimulant Drugs of Abuse and Cardiac Arrhythmias. *Circ Arrhythm Electrophysiol*. 2021;CIRCEP121010273.

Table 26. ESC 2022 GL on non-cardiac surgery. Recommendations for management of known or newly diagnosed arrhythmias

Ventricular arrhythmias

In patients with symptomatic, monomorphic, sustained VT associated with myocardial scar, recurring despite optimal medical therapy, ablation of arrhythmia before elective NCS I B

It is not recommended to initiate treatment of asymptomatic PVC during NCS. III C

NCS: non-cardiac surgery

Table 27. Ventricular arrhythmias in Pregnancy

ACC/AHA/HRS 2017 GL on VA/SCD

In mothers with long QT syndrome, a beta blocker should be continued during pregnancy and throughout the postpartum period including in women who are breastfeeding. I-B-NR

Electrical cardioversion sustained VA is safe and effective and should be used with standard electrode configuration. I- C-EO

In pregnant patients needing an ICD or VT ablation, undergo these procedures during pregnancy, preferably after the first trimester IIa-B-NR

ESC 2025 GL on Pregnancy. Recommendations for ventricular tachycardia, device implantation, catheter ablation, and pregnancy

Immediate electrical cardioversion is recommended for both unstable and stable ventricular tachycardias. ²⁵²	I	C
Beta-blockers or verapamil are recommended for the prevention of idiopathic sustained VT.	I	C
If an ICD, pacemaker, or resynchronization therapy device is indicated during pregnancy, implantation is recommended with optimal radiation protection. ^{667,675}	I	C
In idiopathic RVOT-VT, flecainide should be considered if beta-blockers fail, to prevent recurrence.	IIa	C
For acute conversion of haemodynamically stable sustained VTs during pregnancy, i.v. beta-blocker, adenosine (idiopathic RVOT-VT), verapamil (fascicular VT), procainamide, or overdrive ventricular pacing (ICD lead) should be considered. ^{252,653,676–678}	IIa	C
When performing catheter ablation during pregnancy, the use of non-fluoroscopic mapping and navigation systems should be considered. ^{663–665}	IIa	C
Catheter ablation with electro-anatomical mapping systems may be considered in experienced centres in the case of sustained drug-refractory, recurrent, and/or poorly tolerated VT if there are no other alternatives.	IIb	C

ICD, implantable cardioverter defibrillator; i.v., intravenous; RVOT, right ventricular outflow tract; VT ventricular tachycardia.

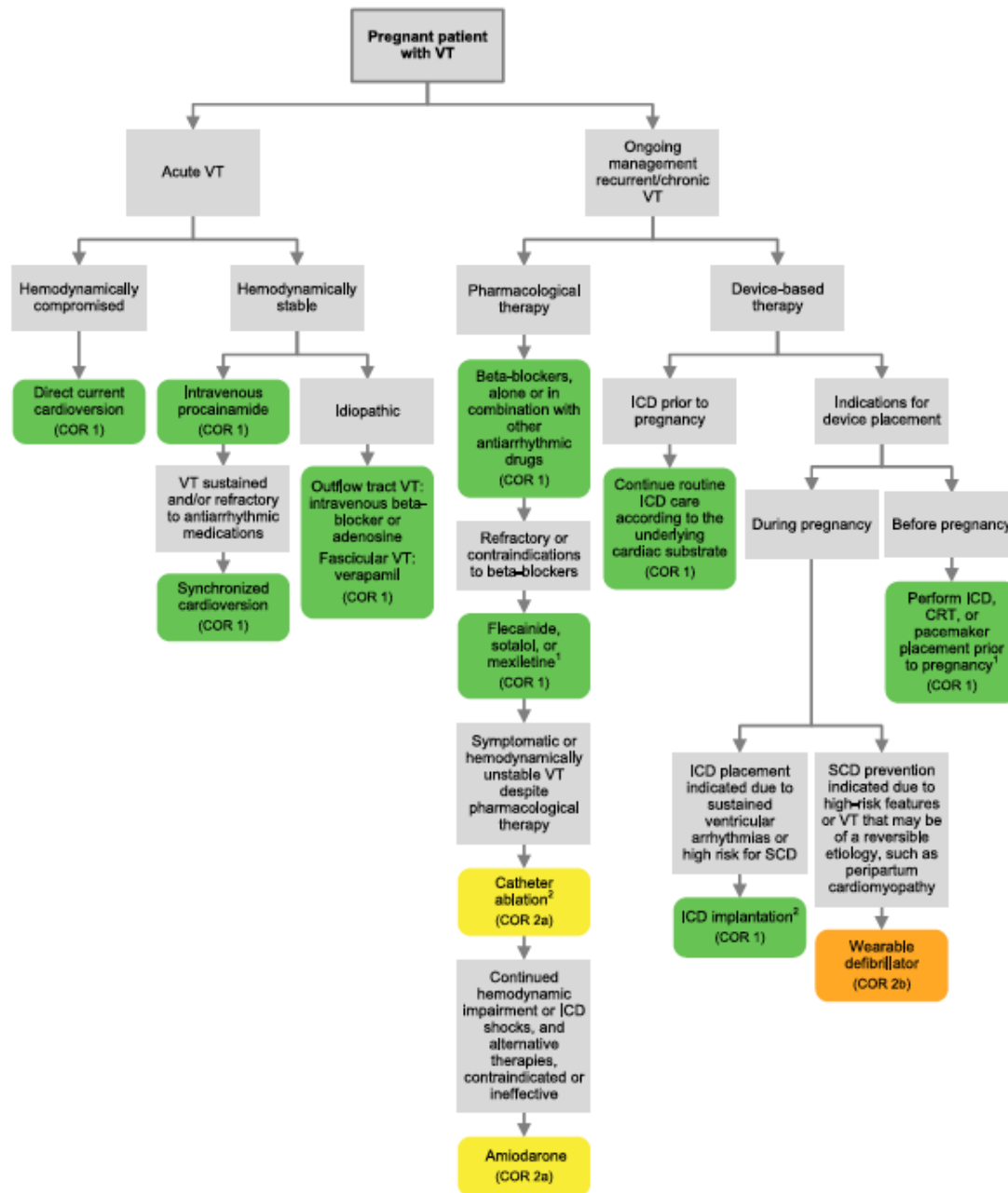


Figure 30. 2023 HRS expert consensus statement on the management of arrhythmias during pregnancy. Algorithm of recommendations for the management of pregnant patients with ventricular arrhythmias.

1 Choice of drug dependent on the underlying cardiac substrate.

2 With techniques for minimizing fluoroscopy as low as reasonably achievable.

CRT: cardiac resynchronization therapy, ICD: implantable cardioverter-defibrillator, SCD: sudden cardiac death, VT: ventricular tachycardia.

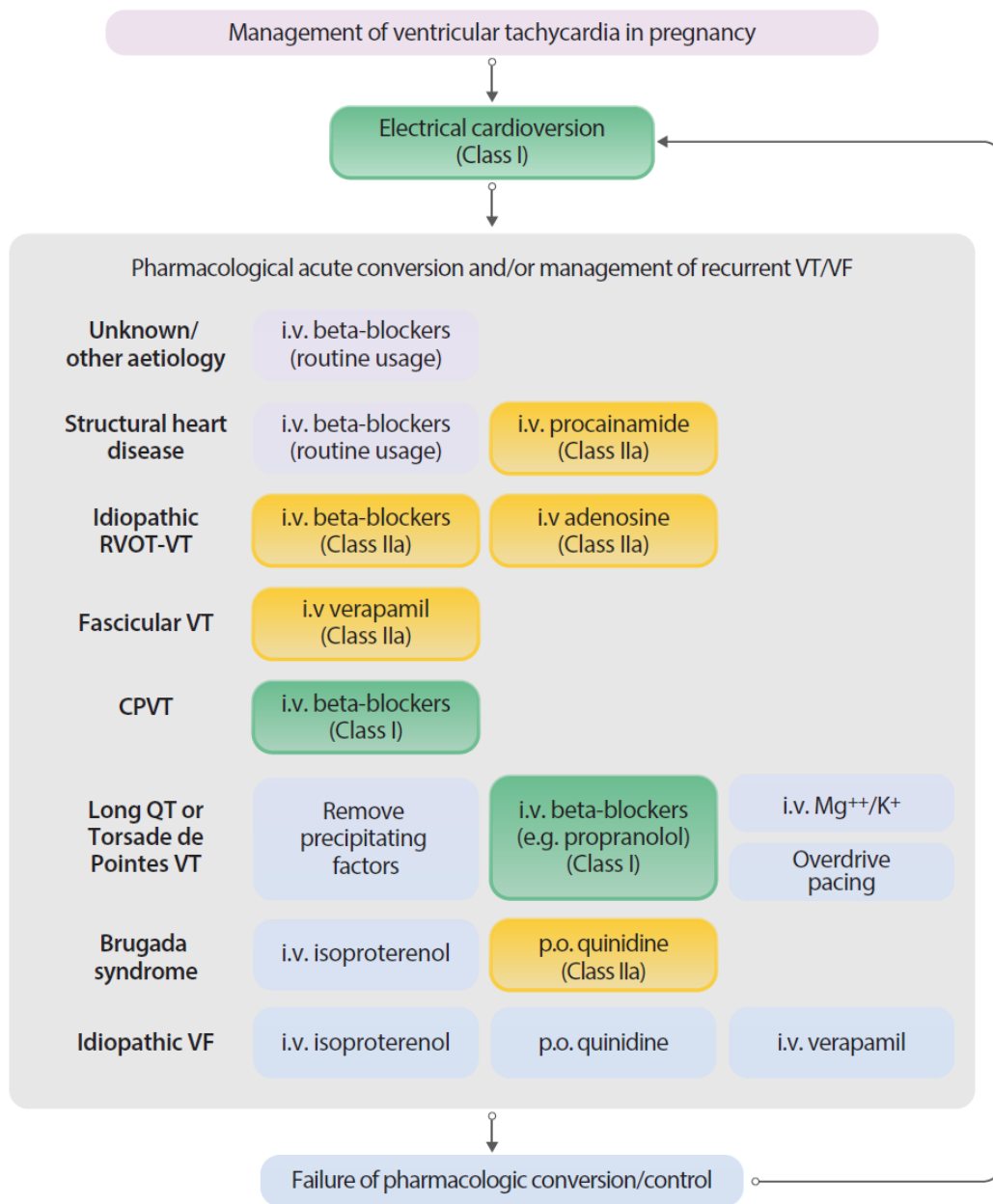


Figure 31. ESC 2025 GL on Pregnancy. Management of ventricular tachycardias in pregnancy.

CPVT, catecholaminergic polymorphic ventricular tachycardia; i.v., intravenous; N, no;

RVOT, right ventricular outflow tract; VF, ventricular fibrillation; VT, ventricular tachycardia; Y, yes.

Table 28. ESC 2025 GL on Pregnancy. Recommendations for cardiac arrest and pregnancy

Continuous manual left uterine displacement during CPR in pregnant women (≥ 20 weeks) with cardiac arrest is recommended to relieve aortocaval compression. ⁶¹⁴	I	C
It is recommended to establish i.v. access above the diaphragm to ensure that the i.v. therapy is not obstructed by the gravid uterus. ⁶¹⁴	I	C
It is recommended to perform the same chest compressions and defibrillation protocols in pregnant as in non-pregnant women. ^{554,614,687}	I	C
Anterolateral defibrillator pad placement is recommended with the lateral pad placed under the breast. ^{614,660}	I	C
It is recommended that no drugs are withheld in pregnant women with cardiac arrest due to concerns of teratogenicity. ⁶⁸⁶	I	C
Immediate caesarean section at the site of the arrest should be considered and immediately prepared if ROSC has not been achieved in the mother after 4 min of resuscitative efforts and if the foetus is viable, taking gestational age, comorbidities, and the available level of medical care into account. ^{614,688,689}	IIa	C

CPR, cardiopulmonary resuscitation; i.v., intravenous; ROSC, return of spontaneous circulation.

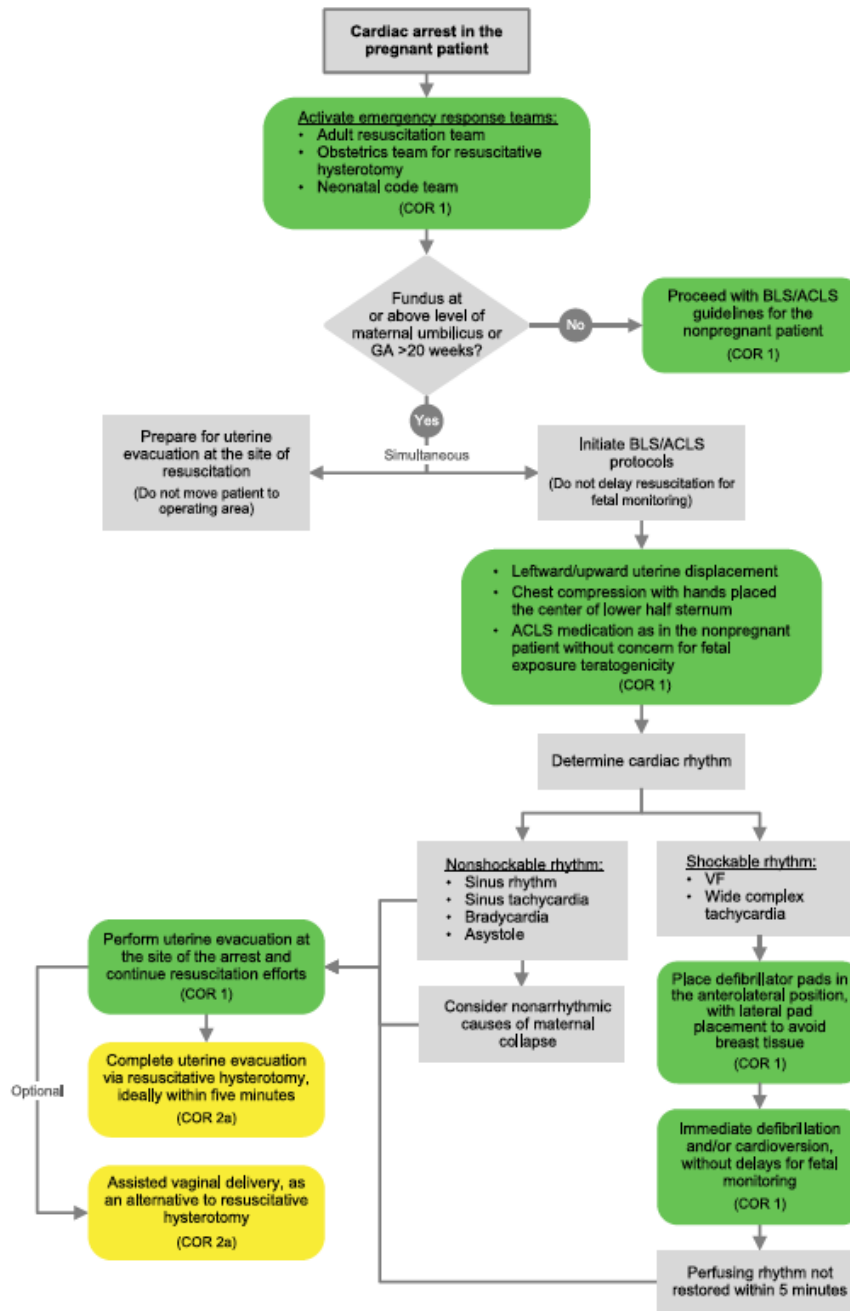


Figure 32. 2023 HRS expert consensus statement on the management of arrhythmias during pregnancy. Management of cardiac arrest in the pregnant patient.

ACLS: advanced cardiac life support, BLS: basic life support, GA: gestational age, VF: ventricular fibrillation.

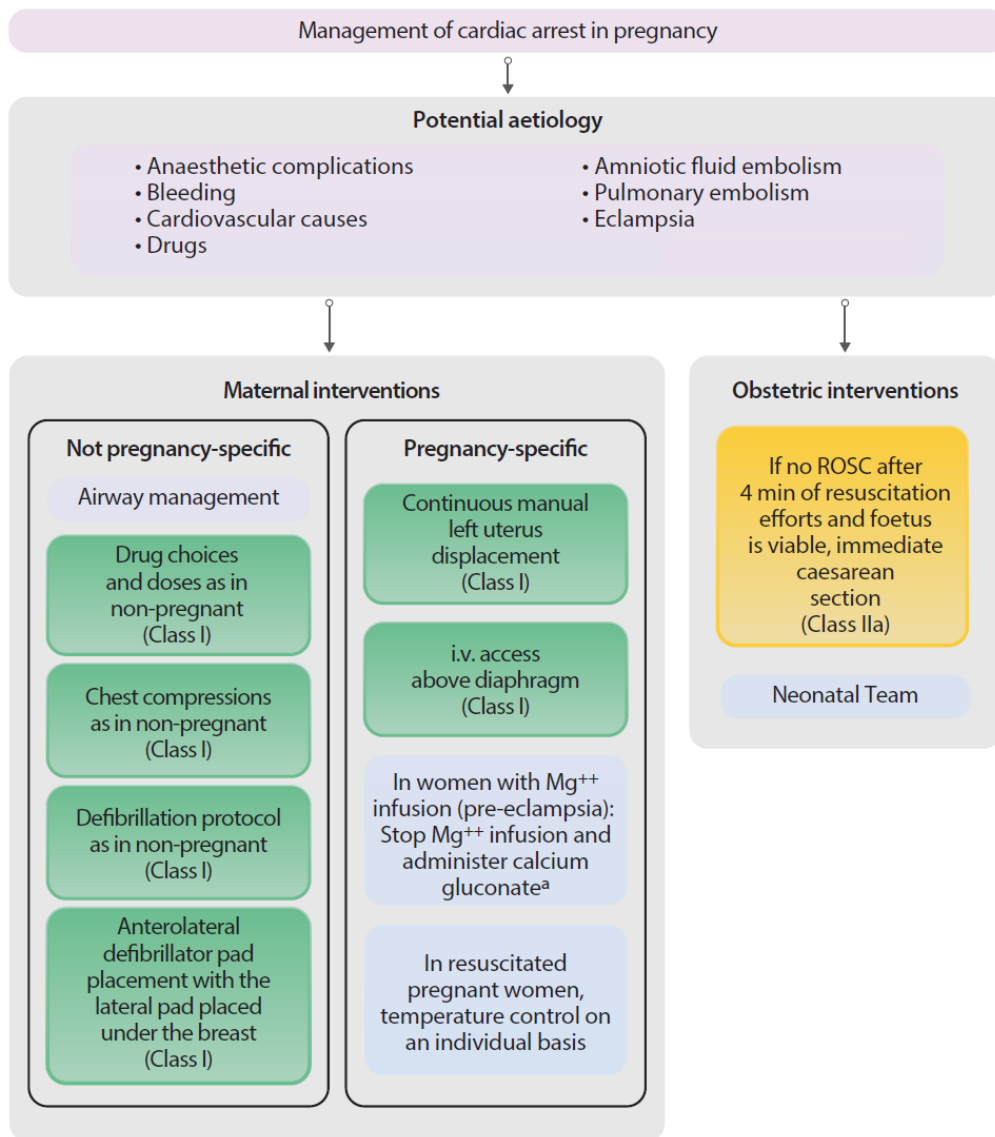


Figure 33. ESC 2025 GL on Pregnancy. Management of cardiac arrest in pregnancy.

i.v., intravenous; ROSC, return of spontaneous circulation.

^a30 mL calcium gluconate 10% solution.