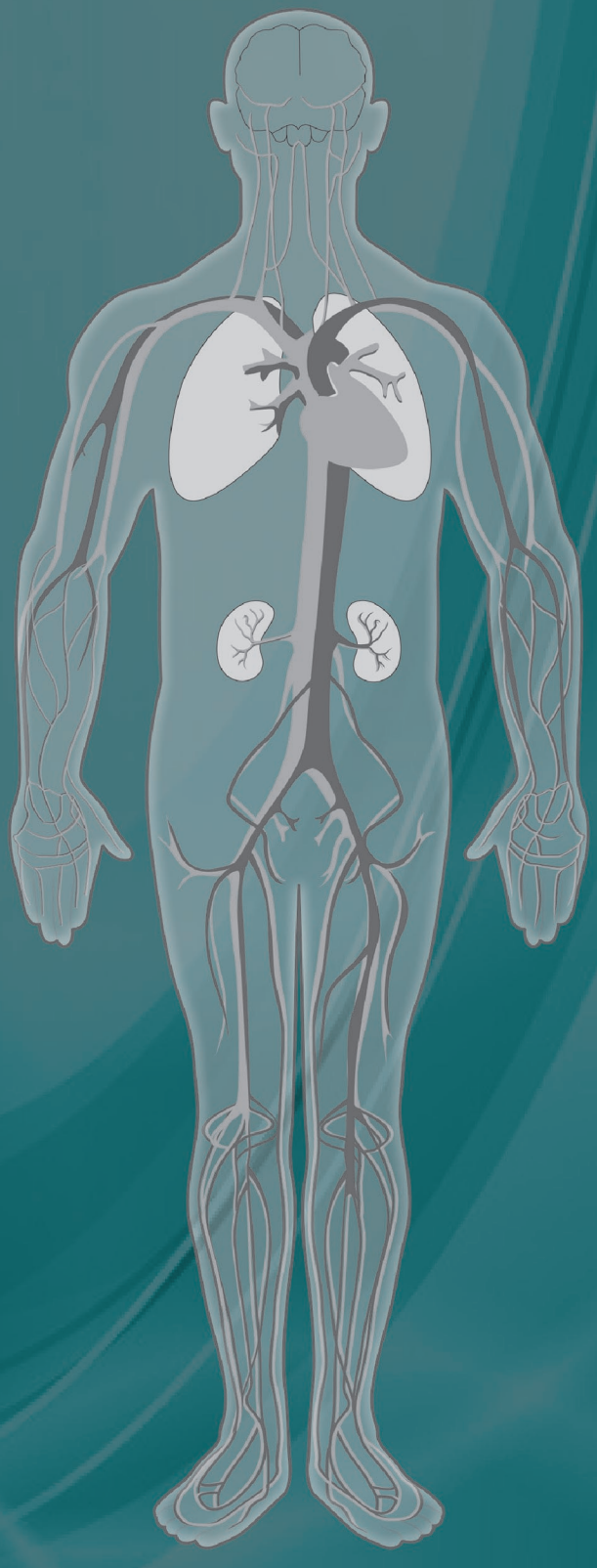
ISCCM
INDIAN SOCIETY OF CRITICAL CARE MEDICINE

Critical Care Update 2025



Editors
Pradip Kumar Bhattacharya
Srinivas Samavedam
Sheila Nainan Myatra
Subhash Todi

7th Edition

JAYPEE

Contents

SECTION 1: Respiratory

Section Editors: *Babu K Abraham, Prachee Sathe*

SECTION 2: Hemodynamics

Section Editors: *JV Divatia, Natesh Prabu R*

SECTION 3: Neurocritical Care

Section Editors: *Harsh Sapra, Kayanoosh J Kadapatti*

SECTION 4: Gastrohepatology

Section Editors: *Shivakumar Iyer, Sachin Gupta*

SECTION 5: Hemato-oncology

Section Editors: *Banani Poddar, Raymond Dominic Savio*

SECTION 6: Infections/Sepsis/AMAs

Section Editors: *Parshotam Lal Gautam, Deven Juneja*

SECTION 7: Trauma

Section Editors: *Venkat Raman Kola, TVSP Murthy*

SECTION 8: Fluids and Electrolytes

Section Editors: *Supradip Ghosh, Pankaj Anand*

SECTION 9: Endocrine Issues

Section Editors: *Susruta Bandyopadhyay, Ritesh J Shah*

SECTION 10: Transplant Critical Care

Section Editor: *Tapas Kumar Sahoo*

SECTION 11: Data Analytics and Artificial Intelligence

Section Editors: *Bharat G Jagiasi, Ketan Kargirwar*

SECTION 12: Ethics Law and End-of-Life Issues

Section Editors: *Banambar Ray, Pradeep Rangappa*

SECTION 13: Obstetric Critical Care

Section Editors: *Sunil T Pandya, Gunchan Paul*

SECTION 14: Nephro Critical Care

Section Editors: *Kanwalpreet Sodhi, Ranajit Chatterjee (Chattopadhyay)*

SECTION 15: Nutritional Support

Section Editors: *Subhal Bhalchandra Dixit, Ganshyam Jagathkar*

CHAPTER 14

State of the Art Review: Arrhythmia Management in Intensive Care Unit

Dinesh Wagh, Bhavik Vikram Shah, Shailesh Ramesh Patil

INTRODUCTION

Arrhythmias pose a frequent and significant challenge for intensivists, contributing to increased morbidity and prolonged hospitalizations. They are more likely to develop in patients with preexisting structural heart disease. Various stressors can trigger arrhythmias, such as hypoxia, infection, cardiac ischemia, excessive catecholamines (whether produced by the body or administered externally), or disturbances in electrolyte levels. Effective management involves addressing these underlying factors along with administering specific medical treatments targeting the arrhythmia.

The clinical impact of arrhythmias largely depends on the ventricular response rate, duration of the arrhythmia, and the patient's baseline cardiac function. Bradyarrhythmias may reduce cardiac output (CO) by slowing the heart rate (HR), particularly in patients with relatively fixed stroke volumes (SV). Additionally, in patients with diastolic dysfunction, the loss of atrial contraction (or "atrial kick") can significantly increase pulmonary pressures. Conversely, tachyarrhythmias can impair diastolic filling, lowering CO and potentially causing hypotension and myocardial ischemia.

Thus, the effects of any particular arrhythmia are closely related to the patient's overall cardiac physiology and function. Consequently, the urgency and type of intervention required are determined not only by the arrhythmia itself but also by the patient's underlying cardiac status.

ASSESSMENT OF ARRHYTHMIAS

The initial crucial step in assessing arrhythmias is to confirm whether an arrhythmia is genuinely occurring. Medical device alarms can sometimes be falsely activated by artifacts, which may arise from electrical interference caused by surrounding equipment or patient movement.[1,2] It is important to thoroughly evaluate and differentiate these artifacts from actual cardiac arrhythmias, especially polymorphic ventricular tachyarrhythmias. Physicians should always align their assessment with the patient's clinical condition and meticulously examine the tracings for QRS complexes within the artifact.

The next step in the analysis is to *check for carotid pulsations* to rule out any possibility of PEA pulseless electrical activity (PEA). Determine whether the arrhythmia is new *onset* after ICU admission or was present on admission and is now aggravated by pathophysiologic insults. Determine whether the *arrhythmia* is in fact the basis for the deterioration in hemodynamics or the arrhythmia is just associated with the disease process.

The hemodynamic stability of the patient determines the next course of management[2]. Cardiac arrhythmias can disrupt CO by impairing the HR and/or SV according to the equation CO = HR × SV. The clinical presentation of cardiac arrhythmias varies widely, and they may present as (1) asymptomatic findings on an electrocardiogram (ECG) or telemetry, (2) symptoms without hemodynamic instability (e.g., palpitations, shortness of breath, syncope, or chest pain), (3) hemodynamic instability in conscious patients, or (4) cardiac arrest.

If hemodynamically stable, ascertain whether the arrhythmia is supraventricular or ventricular by analyzing examining QRS width.[2] A QRS complex <0.12 m/sec indicates supraventricular tachycardia (SVT). Be careful not to rely solely on the single rhythm strip for diagnosis. A 12 lead ECG is more useful, and comparison with previous or baseline ECG is essential to identify preexisting blocks or prolonged QT.

Carotid sinus massage and other techniques that enhance vagal tone can slow atrioventricular (AV) conduction and increase the refractory period of the AV node[2]. These methods may help reveal P waves or

TABLE 1: Vagal maneuver and its response in arrhythmia.

Arrhythmia	*Response to vagal maneuvers/ adenosine*
Sinus tachycardia	Gradual slowing with resumption of the tachycardia
Atrioventricular nodal reentrant tachycardia	Abrupt termination or only very transient slowing
Atrial fibrillation/ flutter	Increased atrioventricular block briefly with slowed ventricular response rate
Multifocal atrial tachycardia	Increased atrioventricular block briefly with slowed ventricular response rate
Ventricular tachycardia	Usually no response

terminate AV node-dependent arrhythmias such as atrioventricular nodal reentrant tachycardia (AVNRT) or atrioventricular reentrant tachycardia (AVRT). Adenosine is another option that can be used for this purpose, but clinicians should be cautious of potential side effects such as bronchospasm, asystole, or heart blocks. In cases of cardiac arrest or severely symptomatic tachycardia or bradycardia, immediate intervention following advanced cardiac life support (ACLS) protocols is necessary **(Table 1)**.

For stable arrhythmias, the next step in analysis and treatment depends on the basis of the HR, the regularity of R waves (regular and irregular), the width and morphology of the QRS complex—wide complex and narrow complex, and the length of the PR interval.

The threefold treatment aim for any arrhythmia is–RRR:[3,4]

- *R*ate control (ventricular rate)
- *R*estoration of sinus rhythm
- *R*educe complications if sinus rhythm cannot be restored. (thromboembolic phenomena)

Once you manage any causes that are reversible, major treatment options include:

- Synchronized direct current (DC) cardioversion
- Antiarrhythmic drug (AAD) therapy
- Pacing

Potential arrhythmogenicity of all the antiarrhythmic drugs is to be kept in mind, however.

TACHYARRHYTHMIAS

Tachycardia, defined as an HR >100 beats/min, can be separated into: (1) Those that arise above the ventricles, termed SVTs and (2) those that arise within the ventricles, termed *ventricular tachycardias* (VT).[6]

Supraventricular tachyarrhythmias are further distinguished as:

- Narrow complex and broad complex
- Regular and irregular

Supraventricular Tachyarrhythmias

It is clinically and therapeutically useful to distinguish between ventricular and SVTs.[7]

This is generally based on:

- Identification of P wave (atrial activation)
- Assessment of rate and morphology of P waves

The next thing is to ascertain what happens with blockades of AV nodes with drugs. If the tachycardia persists, it is *"AV node independent"* tachycardia, and if it terminates, it is *"AV node dependent"* tachycardia.

P Wave Morphology and Location

Identification of P waves is the foremost step in analyzing narrow complex tachycardias. Comparison with previous or baseline ECG for identifying any changes in the QRS complex, ST segment, and T wave that may represent atrial activity (P wave) must be done. Also, the relation of P wave in relation to QRS is of paramount importance.

For sinus and ectopic atrial tachycardias (EAT), the P wave is normal or near normal located with regular PR interval. For tachycardias involving AV node or junction, QRS complex frequently obscures the P wave or it can be seen in the terminal portion of the QRS waves. In accessory pathway-mediated tachycardias, the P waves are frequently present in the ST segment as the conduction through the accessory pathway is more rapid. SVTs are frequently classified as "SHORT R-P" (AV nodal atrial tachycardias and orthodromic AV reentry tachycardias) and "LONG R-P" (most commonly atrial tachycardias) **(Fig. 1)**.

Next, look at the morphology of the P waves. Atrial activation is generally classified as "high-low" if the P waves are upright in the lead II III arteriovenous fistula AVF (inferior leads) and low-high if the P waves are inverted. Inverted P waves suggest atrial activation is initiated from the AV junction or mitral and tricuspid annulus and upright P waves suggest activation from a superior location.

Atrioventricular Node Dependent and Independent Tachycardias

Atrial tachycardias do not depend on the AV node for their conduction and perpetuation. In contrast, AV nodal

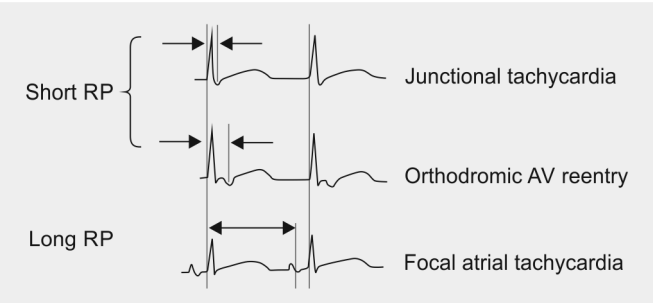


Fig. 1: Diagram illustrating the location of P wave for different tachycardias.

reentrant and accessory pathway-mediated tachycardia are AV node dependent, and hence the tachycardia terminates if AV block is done with drugs or vagal maneuvers.

Narrow Complex Tachycardia

Regular narrow complex SVTs include sinus tachycardia, AVNRT, AVRT, or EAT.

Irregular narrow complex SVTs include AF, multifocal atrial tachycardia (MAT), atrial flutter (AFl) with variable block, and sinus tachycardia with frequent atrial premature complexes (APCs).

Regular narrow complex rhythms

Sinus tachycardia: Sinus tachycardia is often a physiological response to a sympathetic stimulus in the form of hypoxia hypovolemia, hypothyroidism, dehydration, catecholamines infusions. Exclude an iatrogenic etiology by reviewing the medication sheet. The treatment is focused on identification of the cause and treating it. The point to keep in mind is that sinus tachycardia may be an appropriate physiological response to hypotension or hypovolemia and undo treatment can reduce CO.

Atrioventricular nodal reentrant tachycardia: This is so called as the *reentry mechanism* is in the AV node. Also known as junctional tachycardia, this typically occurs with a sudden onset of 140–180 beats/min and is indicated by absent P waves. AVNRT typically involves dual AV nodal pathways with (1) slow conduction antegrade and (2) retrograde conduction by the second pathway which is transiently refractory.

The main aim of treatment is to delay or block AV conduction—either by vagal maneuvers or by AV nodal blocking drugs such as adenosine, beta-blockers, digoxin, or non-dihydropyridine (DHP) calcium channel blockers. Long-term treatment includes beta-blockers or radioactive ablation of one of the pathways.

Atrioventricular reentrant tachycardia: Atrioventricular reentrant tachycardia occurs due to the presence of an accessory pathway that bypasses the AV node, leading to premature ventricular activation. This early activation creates the characteristic delta wave and a shortened PR interval (<0.12 seconds). The term Wolff-Parkinson-White (WPW) syndrome) refers to the presence of preexcitation along with episodes of tachycardia.[8]

Atrioventricular reentrant tachycardia can present as either narrow or wide complex tachycardia (WCT). If the electrical impulse travels down the AV node and returns via the accessory pathway, it produces a narrow complex tachycardia. Conversely, when the impulse travels down the accessory pathway and returns through the AV node (antidromic conduction), a WCT occurs.

In cases of atrial fibrillation (AF) with AVRT, the bypassing of the AV node results in a rapid ventricular response, which can progress to ventricular fibrillation (VF). This leads to poor perfusion, and immediate synchronized DC cardioversion is necessary. However, if the patient is stable, treatment should focus on prolonging the refractory period of the accessory pathway relative to the AV node. This slows the transmission of impulses through the accessory pathway, thereby reducing the ventricular rate.

In contrast, treating AF in patients without WPW involves prolonging the refractory period of the AV node. As a result, medications commonly used to manage AF, such as adenosine, verapamil, and digoxin, should be avoided in WPW patients. Instead, agents such as procainamide, ibutilide, and flecainide are preferred as they slow conduction through the accessory pathway.

Amiodarone is also considered a safe option for treating WPW.

Atrial flutter: This is a micro reentrant arrhythmia identified by flutter waves most prominently seen in the inferior leads and at the rate of 250–350 beats/min with variable AV block. Most commonly, the patient presents with 2:1 AV block, leading to a fixed ventricular rate of 150 beats/min.

The immediate and midterm treatment is very similar to the management of atrial fibrillation (AF), i.e., AV nodal blocking drugs for rate control. DC cardioversion with 50 joules is usually sufficient if required.

Irregular narrow complex rhythms

Atrial fibrillation: Atrial fibrillation is the most common narrow complex tachyarrhythmia in the ICU, second only to VT overall.[9,10] Its prevalence rises significantly with age. Common risk factors for AF include structural heart disease, hypertension, valvular disease, and left ventricular hypertrophy. Postoperative AF is also frequent, especially after cardiac surgery, with an incidence ranging from 25% to 40%, typically peaking around day 2 or 3. In most cases, postoperative AF resolves spontaneously, with normal rhythm being restored within 6–8 weeks.[11]

Data on the best approach to managing AF in critically ill patients are limited. A rapid ventricular rate and the loss of atrial contraction can result in significant hemodynamic instability. If DC cardioversion is needed, using an anterior-posterior electrode placement with biphasic waveforms is more effective than lateral electrode positioning. In patients who have undergone thoracic surgery, a DC shock of 200 joules should be attempted due to the higher impedance and suboptimal electrode placement. However, DC cardioversion in critically ill patients with AF is often unsuccessful, with success rates ranging between 30% and 37%.

For hemodynamically stable patients, treatment decisions should take into account the duration of AF onset. In the ICU, new-onset AF is often a temporary consequence of the patient's critical condition and may spontaneously revert to sinus rhythm. In these cases, focusing on adequate ventricular rate control and addressing the underlying causes may be an appropriate first-line approach. Pharmacological cardioversion should be reserved for patients with persistent symptoms or when rate control is insufficient.

For acute rate control, beta-blockers, non-DHP calcium channel blockers, and digoxin are commonly used. Beta-blockers are particularly effective in ICU settings where sympathetic tone is heightened. Amiodarone can help with both rhythm control and rate management until cardioversion occurs. Digoxin can be considered as a third-line option.[12,13]

Once AF is confirmed, thromboprophylaxis should be initiated regardless of whether rate or rhythm control is planned. Stroke and bleeding risks should be evaluated using the CHA2DS2-VASc and HAS-BLED scores. In stable patients with AF lasting 48 hours or more, anticoagulation with warfarin (target INR 2–3) or a novel oral anticoagulant (NOAC) is recommended for at least 3 weeks before and 4 weeks after cardioversion, regardless of the method used to restore sinus rhythm, whether electrical or pharmacological.[14,15]

Alternatively, transesophageal echocardiography (TEE) can be performed to rule out a cardiac thrombus, allowing cardioversion without the need for 3 weeks of preprocedural anticoagulation. This must still be followed by 4 weeks of anticoagulation. The HAS-BLED score should be used to identify patients at high risk for bleeding and ensure appropriate follow-up.

Multifocal atrial tachycardia: Multifocal atrial tachycardia is an irregular atrial tachycardia manifested by 3 or more P wave morphology and PR intervals.[5] It is commonly seen to be associated with cardiopulmonary disease, metabolic derangements and drugs such as theophylline derivatives. Treatment consists of correcting hypoxia and electrolyte abnormalities and the use of AV nodal-blocking drugs as required.

Ventricular Tachyarrhythmias

These are distinguished as:

- Sustained monomorphic VT
- Nonsustained monomorphic VT
- VF
- Polymorphic ventricular tachycardia-torsades de pointes
- Electrical storm

Sustained Monomorphic Ventricular Tachycardia

Wide complex tachycardia exhibits QRS duration of >120 m/sec and a rate of >100 beats/min.

Any WCT on ECG should be assumed as VT unless proven otherwise. Assess hemodynamic stability and obtain a thorough history. A prior history of myocardial infarction predisposes the patient to WCT secondary to ventricular etiology. Also, history regarding the use of QRS prolonging drugs and pacemakers[16] if present is critical to distinguish between other causes of WCT. Signs of AV dissociation such as irregular cannon A waves and low systolic BP with respect to heartbeats favor VT over SVT. Response to vagal maneuvers such as carotid sinus massage may suggest SVT.

Four points to consider:

1. Absent RS complex in any precordial leads, i.e., precordial leads all monophasic R waves or all S waves = positive concordance or negative concordance. (21% sensitive, 100% specific) **(Fig. 2)**

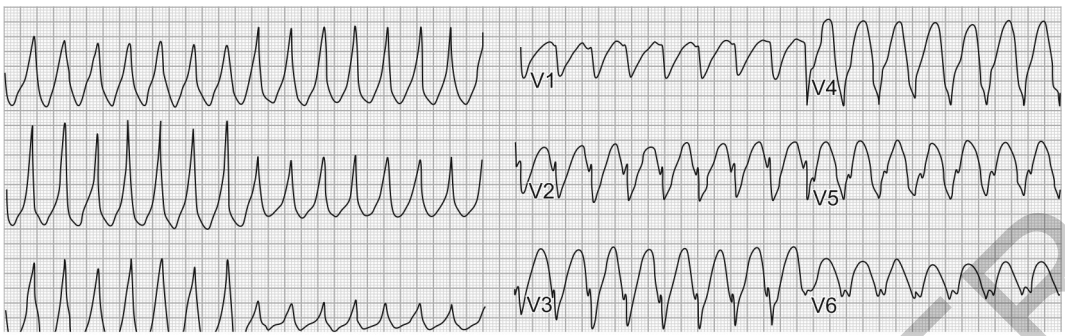


Fig. 2: Absent RS complex in precordial leads.

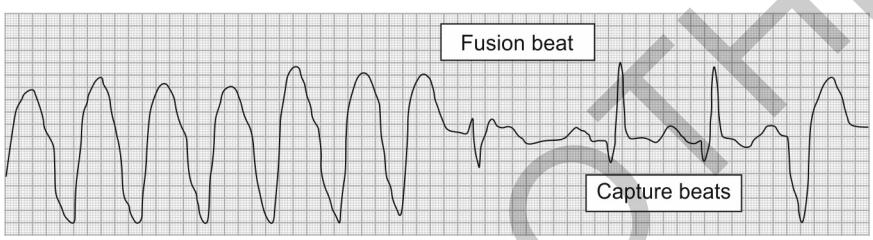
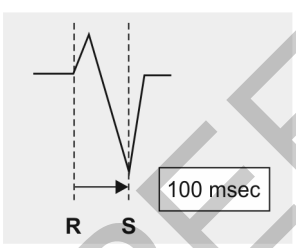
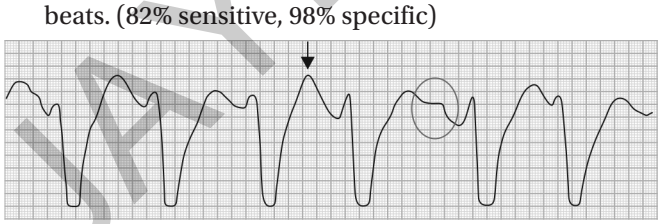


Fig. 3: Fusion beat.

2. Onset of R wave to the nadir of S > 100 m/s in any of V1–V6 (21% sensitive, 100% specific)



3. If RS < 100 m/s, signs of AV dissociation should be sought such as notching at any point in QRS complex due to superimposed P wave, fusion beats, and capture beats. (82% sensitive, 98% specific)



Notching of QRS

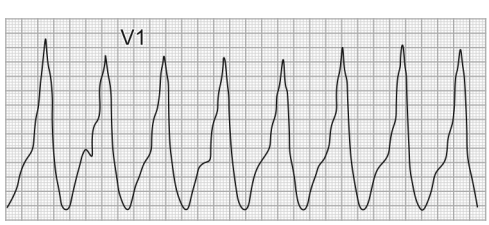
Fusion beats: Supraventricular impulse reaches the AV node at the same time as ventricular impulse **(Fig. 3)**.

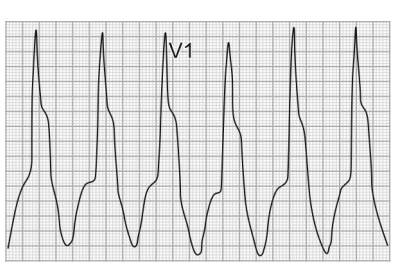
Capture beats: Supraventricular impulse dominates over ventricular impulse and therefore normal QRS is seen in between wide complexes **(Fig. 3)**.

4. See the morphology of QRS complexes—VT frequently have right bundle branch block (RBBB) or left bundle branch block (LBBB) morphology (98% sensitive, 97% specific)

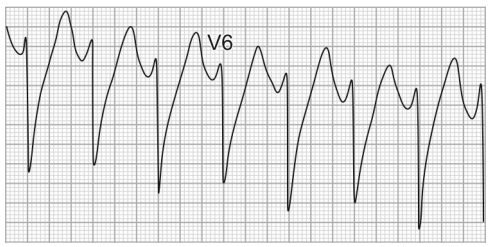
In lead V1, if there is dominant R wave assess for RBBB morphology, if:

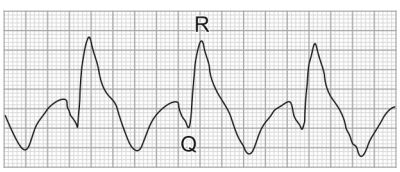
- In V1-V2 monophasic R wave, taller left rabbit ear, qR complex in V1, as shown in below figures:

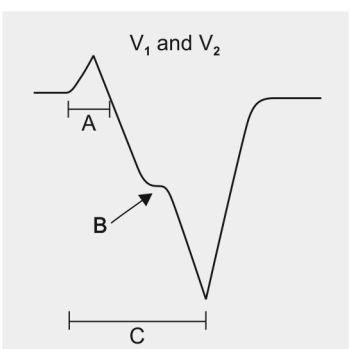




- In V6 lead QS complex (no R wave) and rS (indicates VT if left axis deviation present)

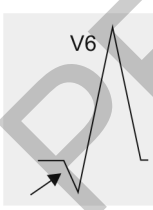


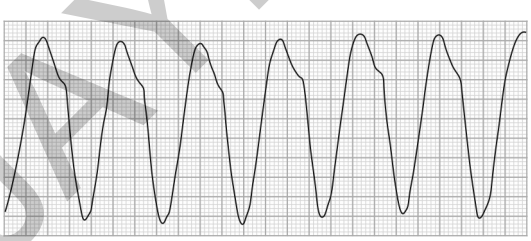




In lead V1 if there is dominant S wave then look for LBBB morphology:

- If in V1-V2 you find an initial R wave >30 m/s, notching or slurring of S wave, RS >30 m/s it suggests VT.
- If in V6 lead, QS or qR, it suggests VT.





If the patient has pulse, synchronized cardioversion is preferred as it will be coordinated with the heart's electrical activity (QRS complex) to reduce the risk of inducing cardiac arrest. If the patient does not have a pulse, then this requires immediate defibrillation.

Electrical cardioversion with 150–200 joules in biphasic defibrillators can also be used for stable sustained VT with no response to antiarrhythmics. Electrophysiology is indicated prior to discharge to decide on implantable cardioverter-defibrillator (ICD) implantation or VT ablation.[17]

Hemodynamically stable sustained VT can be treated with intravenous amiodarone 300 mg over hour followed by 900 mg/24 hours or lignocaine.

It is important to identify the correct rhythm. If VT is mistakenly diagnosed as AF and treated with electrical cardioversion, this could have some serious consequences. When the atria beat irregularly, this increases the risk of clot formation on the atrial walls. The patient who is not on anticoagulation and receives cardioversion in the setting of AF, there is a risk stroke due to clot dislodgement.

Similarly, if VT is mistakenly treated with AV node blocking agents assuming it as SVT, conduction through alternative pathways which can either be outside of the AV node or within the AV node due to their faster activation of the ventricular myocardium can create a positive feedback loop and lead to AF, VF, or torsades de pointes (TdP).

Nonsustained Ventricular Tachycardia

This does not require any specific treatment other than correction of precipitating causes such as electrolyte abnormalities. Lignocaine and beta-blockade can be used in symptomatic patients.

Ventricular Fibrillation

Immediate defibrillation is needed to treat VF. If VF or pulseless VT is refractory to 2–3 shocks intravenous (IV) amiodarone is considered to be the first-line AAD. Lignocaine can be used if amiodarone is not available. Full cardiac evaluation and decision on the need for ICD to be decided in long-term management plan.

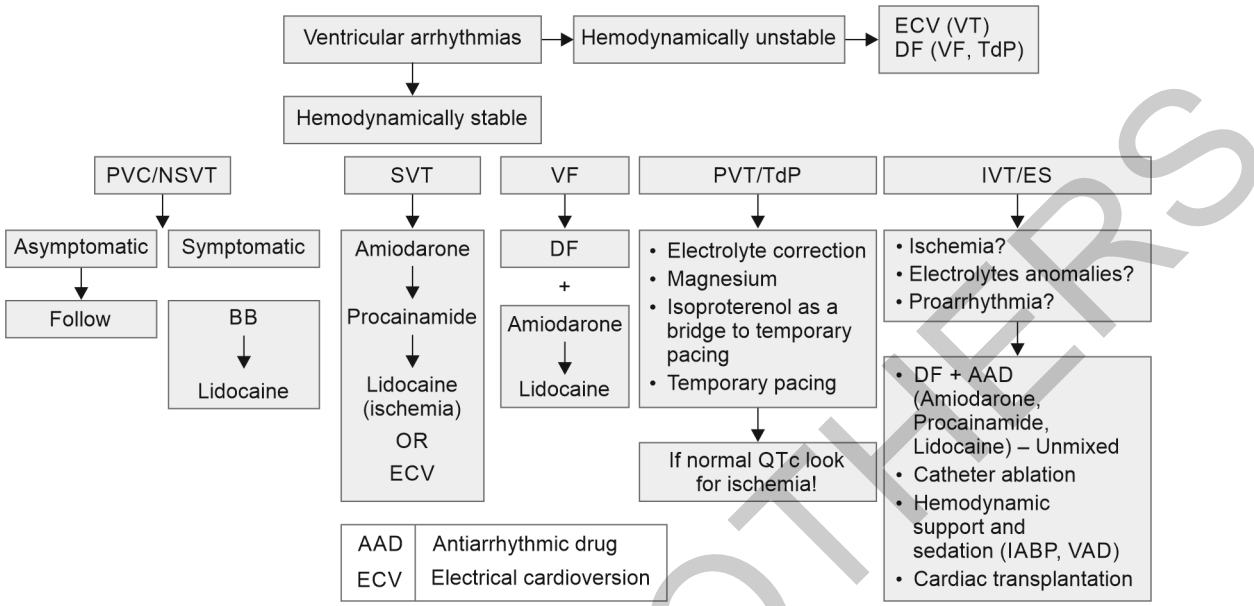
Polymorphic Ventricular Tachycardia or Torsades de Pointes

Torsades de pointes is associated with QT prolongation. Treatment constitutes stopping all QT-prolonging drugs and correction of electrolytes and temporary pacing may be needed.

Electrical Storm or Incessant Ventricular Tachycardia

An electrical storm is a recurrence of VT >3 times/24 hours. Correction of reversible causes, ongoing ischemia,

Flowchart 1: Electrical Storm or incessant ventricular tachycardia.



(ES: electrical storm; IABP: intra-aortic balloon pump; IVT: incessant ventricular tachycardia; NSVT: nonsustained ventricular tachycardia; PVC: premature ventricular contractions; PVT: polymorphic ventricular tachycardia; SVT: sustained ventricular tachycardia; TdP: torsade de pointes; VAD: ventricular assist device; VF: ventricular fibrillation)

and stopping the proarrhythmic drugs is the mainstay in treatment. The patient may require intra-aortic balloon pump (IABP) for hemodynamic support. Sedation and general anesthesia are required in some cases **(Flowchart 1)**.

BRADYARRHYTHMIAS

Bradyarrhythmia is a type of cardiac arrhythmia characterized by an abnormally slow HR, specifically an HR of <60 beats/min in adults.

Significant bradyarrhythmia is uncommon in the ICU, accounting for only 10% of all arrhythmias in this setting.[17-23] In the ICU or cardiac care unit (CCU), severe or untreated bradyarrhythmia can lead to more serious conditions, including heart failure, decreased CO, and an increased risk of sudden cardiac arrest.

Patients with bradyarrhythmia in the ICU may present with a range of etiologies, from simple to complex multifaceted conditions. Understanding the causes of bradyarrhythmia in this context is crucial for effective management.

Emergent perspectives on important causes encompass a variety of physiological and systemic factors. Hypervagotonic states can arise from vasovagal activity triggered by pain, as well as from procedures such as endotracheal suctioning or episodes of emesis. Respiratory issues, such as hypoxia, and temperature regulation problems, such as hypothermia, also play significant roles.

Electrolyte and metabolic disturbances, including electrolyte imbalances, acidosis, and hypothyroidism, can complicate the clinical picture. Cardiovascular concerns, particularly acute coronary syndromes, must be considered alongside systemic conditions such as sepsis and trauma. Additionally, substance-related factors, including medications and intoxications, can significantly impact patient outcomes in emergent situations.

Classification

Disorders of impulse generation:

- *Sinus arrhythmia:* Change in beat-to-beat P-P interval without change in the morphology of P waves
- *Sinus bradycardia:* Reduced automaticity of the SA node due to alterations in vagal tone
- *Sinus node dysfunction (sick sinus syndrome):* Intrinsic dysfunction of the SA node leads to inappropriate automaticity, which can manifest as tachy-brady syndrome.

Disorders of impulse propagation:

- *Sinoatrial block:*
  - *First degree:* Delayed conduction within the SA node

- *Second degree:* Intermittent conduction of SA impulse
- *Third degree:* Presents as sinus arrest

- *Atrioventricular block:*
  - *First degree:* Prolongation of the PR interval to >200 m/s.
  - *Second degree:*
    - *Type I (Wenckebach):* Progressive prolongation of the PR interval before conduction of the P wave fails.
    - *Type II:* Failure in the propagation of the atrial impulse without change in the PR interval.
  - *Third degree:* Complete failure of atrial impulse leading to AV dissociation

Management of Bradyarrhythmia

Asymptomatic bradyarrhythmias generally do not carry a poor prognosis and typically do not warrant therapy. Only symptomatic bradycardia needs to be treated. The recommended initial therapy for bradycardia causing end-organ perfusion issues is intravenous atropine at a dose of 1.0 mg. Atropine may have limited effect on high-grade AV blocks (i.e., infra-Hisian disease) and has a short half-life, so a subsequent treatment plan should be prepared.

According to the ACLS guidelines, β-adrenergic agonists such as epinephrine and dopamine, as well as cardiac pacing, are recommended as the first or next steps after a trial of atropine for hemodynamically unstable bradyarrhythmia. Additionally, isoproterenol infusion may be used to stimulate HR via β-1 agonism. Care should be taken when using chronotropic infusions, as they can exacerbate ischemia in the context of cardiogenic shock by reducing coronary perfusion. Other treatments can be tailored to address the factors precipitating the bradyarrhythmia.

The presence of syncope, heart failure, or other symptoms accompanying bradycardia is an indication for pacemaker implantation.[24] Second-degree and third-degree AV blocks, often referred to as high-grade AV blocks, indicate abnormal propagation at the infra-Hisian level, which can be unstable and may require temporary or permanent pacing depending on their reversibility.

According to the guidelines for managing ST-elevation myocardial infarction (STEMI), pacing is indicated (class I) for complete AV block, symptomatic bradyarrhythmias refractory to drug therapy, and trifascicular block, including alternating bundle branch block and bifascicular block with Mobitz type II second-degree AV block.

Temporary pacing is most easily accomplished via the transcutaneous route, using large surface area skin electrodes placed in either the anterior-posterior position (cathode over the cardiac apex and the anode between the right scapula and spine) or the anterior-anterior position (cathode over the apex and anode on the right chest). Transcutaneous pacing is effective (>90%) but cannot be used for prolonged periods due to pain, muscle stimulation, and loss of capture from impedance changes. Transvenous pacing is generally required if continuous pacing is needed for >20–30 minutes.

For patients with symptomatic AV block in the absence of a transient cause of the AV conduction disturbance, a permanent pacemaker (PM) is usually implanted. Candidates for PM implantation typically have an infra-Hisian block associated with anterior myocardial infarction rather than inferior myocardial infarction.

For more complex cases, consulting cardiology or electrophysiology is advised.

CONCLUSION

The management of arrhythmias in the ICU is a multi-faceted process that involves accurate diagnosis, targeted treatment, and continuous monitoring. By addressing the unique needs of ICU patients and employing advanced monitoring techniques, healthcare providers can effectively manage arrhythmias and improve patient outcomes. A comprehensive approach that integrates clinical expertise, multidisciplinary collaboration, and proactive measures is essential for optimizing care and ensuring the best possible outcomes for critically ill patients.

REFERENCES

1. Drew BJ, Harris P, Zegre-Hemsey JK, Mammone T, Schindler D, Salas-Boni R. Insights into the problem of alarm fatigue with physiologic monitor devices: a comprehensive observational study of consecutive intensive care unit patients. PLoS One. 2014;9:e110274.
2. Wellens HJ. Electrophysiology: ventricular tachycardia: diagnosis of broad QRS complex tachycardia. Heart. 2001;86:579-85.
3. Annane D, Sebille V, Duboc D, Le Heuzey JY, Sadoul N, Bouvier E, et al. Incidence and prognosis of sustained arrhythmias in critically ill patients. Am J Respir Crit Care Med. 2008;178:20-5.
4. Trappe HJ, Brandts B, Weismueller P. Arrhythmias in the intensive care patient. Curr Opin Crit Care. 2003;9:345-55.
5. Wang K, Goldfarb BL, Gobel FL, Richman HG. Multifocal atrial tachycardia. Arch Intern Med. 1977;137:161-4.

6. Wagner G. Marriott's Practical Electrocardiography. Philadelphia, PA: Lippincott Williams and Wilkins; 2001.
7. Josephson ME, Wellens HJ. Differential diagnosis of supraventricular tachycardia. Cardiol Clin. 1990;8:411-42.
8. Wellens HJ, Bar FW, Lie KI. The value of the electrocardiogram in the differential diagnosis of a tachycardia with widened QRS complex. Am J Med. 1978;64:27-33.
9. Reinelt P, Karth GD, Geppert A, Heinz G. Incidence and type of cardiac arrhythmias in critically ill patients: a single center experience in a medical-cardiological ICU. Intensive Care Med. 2001;27:1466-73.
10. Thompson A, Balser JR. Perioperative cardiac arrhythmias. Br J Anaesth. 2004;93:86-94.
11. Seguin P, Laviolle B, Maurice A, Leclercq C, Malledant Y. Atrial fibrillation in trauma patients requiring intensive care. Intensive Care Med. 2006;32:398-404.
12. Duby JJ, Heintz SJ, Bajorek SA, Heintz BH, Durbin-Johnson BP, Cocanour CS. Prevalence and course of atrial fibrillation in critically ill trauma patients. J Intensive Care Med. 2017;32:140-5.
13. Fleisher LA, Fleischmann KE, Auerbach AD, Barnason SA, Beckman JA, Bozkurt B, et al. 2014 ACC/AHA guideline on perioperative cardiovascular evaluation and management of patients undergoing noncardiac surgery: a report of the American College of Cardiology/American Heart Association Task Force on practice guidelines. J Am Coll Cardiol. 2014;64:e77-137.
14. Hadjizacharia P, O'Keeffe T, Brown CVR, Inaba K, Salim A, Chan LS, et al. Incidence, risk factors, and outcomes for atrial arrhythmias in trauma patients. Am Surg. 2011;77:634-9.
15. Sauaia A, Moore FA, Moore EE. Postinjury inflammation and organ dysfunction. Crit Care Clin. 2017;33:167-91.
16. Gregoratos G, Cheitlin MD, Conill A, Epstein AE, Fellows C, Ferguson TB Jr, et al. ACC/AHA guidelines for implantation of cardiac pacemakers and antiarrhythmia devices: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee on Pacemaker Implantation). J Am Coll Cardiol. 1998;31:1175-12093.
17. Panchal AR, Berg KM, Kudenchuk PJ, Del Rios M, Hirsch KG, Link MS, et al. 2018 American Heart Association focused update on advanced cardiovascular life support use of antiarrhythmic drugs during and immediately after cardiac arrest: an update to the American Heart Association guidelines for cardiopulmonary resuscitation and emergency cardiovascular care. Circulation. 2018;138(23):e740-9.
18. Guidelines for the management of patients with ST-elevation acute myocardial infarction (JCS 2013). http://www.j-circ.or.jp/guideline/pdf/ JCS2013_kimura_h.pdf.
19. Obando MA, Marra EM. Wide QRS Complex Tachycardia. In: StatPearls. Treasure Island (FL): StatPearls Publishing; 2024.
20. Boriani G, Fauchier L, Aguinaga L, Beattie JM, Blomstrom Lundqvist C, Cohen A, et al. European Heart Rhythm Association (EHRA) consensus document on management of arrhythmias and cardiac electronic devices in the critically ill and post-surgery patient, endorsed by Heart Rhythm Society (HRS), Asia Pacific Heart Rhythm Society (APHRS), Cardiac Arrhythmia Society of Southern Africa (CASSA), and Latin American Heart Rhythm Society (LAHRS). Europace. 2019;21:7-8.
21. Kashou AH, Noseworthy PA, DeSimone CV, Deshmukh AJ, Asirvatham SJ, May AM, et al. Wide Complex Tachycardia Differentiation: A Reappraisal of the State-of-the-Art. J Am Heart Assoc. 2020 Jun 2;9(11):e016598.
22. Reinelt P, Karth GD, Geppert A, Heinz G. Incidence and type of cardiac arrhythmias in critically ill patients: a single center experience in a medical-cardiological ICU. Intensive Care Med. 2001;27:1466-73.
23. Gregoratos G, Cheitlin MD, Conill A, Epstein AE, Fellows C, Ferguson TB Jr, et al. ACC/AHA guidelines for implantation of cardiac pacemakers and antiarrhythmia devices: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee on Pacemaker Implantation). J Am Coll Cardiol. 1998;31:1175-209.
24. Guidelines for the management of patients with ST-elevation acute myocardial infarction (JCS 2013). http://www.j-circ.or.jp/guideline/pdf/ JCS2013_kimura_h.pdf.

Critical Care Update 2025

Salient Features

- Contemporary review of recent advances and challenges in critical care
- Fifteen sections covering 101 key topics of interest
- Seasoned author pool with key opinion leaders on the editorial board
- Focused section on Ethics Law and End-of-Life Issues
- Dedicated section on Data Analytics and Artificial Intelligence.

Pradip Kumar Bhattacharya
MD ACME FICCM FCCCM FCCM
Professor and Head
Department of Critical Care Medicine
In-Charge (Trauma and Emergency)
Dean Research and PhD Coordinator
Rajendra Institute of Medical Sciences
Ranchi, Jharkhand, India
ESICM National Representative from India
President, ISCCM National (2024–2025)
Chancellor, Indian College of Critical Care Medicine

Srinivas Samavedam
MD DNB FRCP FNB EDIC FICCM DMLE MBA
Chief Intensivist
Department of Critical Care
Ramdev Rao Hospital
Hyderabad, Telangana, India
President Elect, ISCCM (2024–2025)
Associate Editor, IJCCM

Sheila Nainan Myatra
MD FCCM FICCM
Professor of Critical Care Medicine
Department of Anesthesiology, Critical Care and Pain
Tata Memorial Hospital
Mumbai, Maharashtra, India
Immediate Past President, ISCCM
Chair, Intensive and Critical Care Medicine Committee
World Federation of Societies of Anaesthesiologists (WFSA)

Subhash Todi
MD MRCP
Director (Critical Care)
Manipal Hospitals
Kolkata, West Bengal, India
Current Editor, Indian Journal of Critical Care Case Report
Current Chair Guideline Committee, ISCCM
Past Chair Research Committee, ISCCM
Editor, ICU Protocol

Printed in India

Available at all medical bookstores or buy online at www.ejaypee.com

JAYPEE BROTHERS
Medical Publishers (P) Ltd.
EMCA House, 23/23-B, Ansari Road,
Daryaganj, New Delhi - 110 002, INDIA
www.jaypeebrothers.com

Join us on facebook.com/JaypeeMedicalPublishers
Follow us on instagram.com/JaypeeMedicalPublishers

Shelving Recommendations
CRITICAL CARE, ANESTHESIOLOGY

ISBN 978-93-6616-506-6