Arrhythmia

Clinical problems

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Module Authors

Gorazd Voga, Medical ICU, General Hospital Celje, Celje, Slovenia

Andrej Pernat, Cardiology Department, University Medical Center, Ljubljana, Slovenia

Module Reviewers

Dermot Phelan and Janice Zimmerman

Section Editor

Jan Poelaert
**Learning Objectives**

After studying this module on Arrhythmia, you should be able to:
1. Distinguish ECG Monitoring: the normal ECG
2. Diagnose rhythm disturbances
3. Assess bradycardias, supraventricular and ventricular tachycardias
4. Treat (including pacemaker therapy) arrhythmias and conduction defects in the critically ill

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**DURATION** 7 hours

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**Key Points**

Recognition, diagnosis, interpretation and treatment of arrhythmias represents an important task and competence for every intensivist.

*Mechanisms, stepwise recognition and therapeutic management of arrhythmias, relevant to the intensive care environment.*

*Management of arrhythmias, updated with new anti-arrhythmic drugs and novel techniques such as radiofrequency catheter ablation, with consideration of the circumstances in which the intensivist might consider such options and consultation to cardiology.*

*New sections on pacemaker, implantable cardioverter defibrillator and cardiac resynchronisation therapies.*

*Specifics and potential problems encountered during the potential, escalating treatment of such patients in ICU.*
Contents

Introduction .................................................................................................................. 1

1/ Assessing the normal ECG .................................................................................. 2

   Specific indications for ECG monitoring .............................................................. 2
   Those with known cardiopulmonary disease .......................................................... 2
   Those exposed to a proarrhythmic environment ...................................................... 2
   Those with a history of unexplained collapse .......................................................... 2
   Potential problems with ECG monitoring ............................................................... 3

   General principles of ECG monitoring ................................................................. 3
   Standard limb leads .............................................................................................. 4
   The chest leads (V1-V6) ..................................................................................... 5

   Normal cardiac conduction .................................................................................... 5
   Sinoatrial node (SAN) ........................................................................................... 6
   Atrioventricular node (AVN) .................................................................................. 6
   His bundle ............................................................................................................. 6
   Bundle branches .................................................................................................... 6
   Mechanisms of cardiac arrhythmias ........................................................................ 7
   Abnormal automaticity ......................................................................................... 7
   Abnormal conduction ............................................................................................. 8

2/ Diagnosis of rhythm disturbances ......................................................................... 12

   Assessment of heart rate (= ventricular rate) ......................................................... 12
   Assessment of heart/cardiac rhythm ....................................................................... 12

3/ Managing the patient with rhythm disturbances .................................................. 17

   Ion channels ......................................................................................................... 17
       Structure .......................................................................................................... 17
       Sodium currents ............................................................................................... 17
       Calcium currents ............................................................................................. 17
       Potassium currents .......................................................................................... 17
   Electrogenic pumps ............................................................................................. 18
       The Na⁺ /K⁺ pump .......................................................................................... 18
       The Na⁺ /Ca⁺⁺ exchanger ............................................................................... 18
   Arrhythmias management in the critically ill - rules ............................................... 19
   General treatment options of arrhythmias ............................................................ 22
   Pacing ................................................................................................................... 22
   Pharmacology ...................................................................................................... 27

4/ Managing the patient with bradycardias ............................................................... 29

   Sinus node (SAN) dysfunction .............................................................................. 29
   Sinus node dysfunction in the context of acute myocardial infarction ................. 29
   Atrioventricular (AV) conduction disease .............................................................. 29
   AV node dysfunction in the context of acute myocardial infarction (MI) .................. 30
   Bundle branch block in the context of acute MI .................................................... 32
   ICU patients with implanted permanent pacemakers ............................................. 33
   Cases for special consideration .......................................................................... 34

5/ Managing supraventricular tachycardia .............................................................. 36

   Sinus tachycardia .................................................................................................. 36
   Causes ................................................................................................................. 36
   Management ........................................................................................................ 37
   Paroxysmal atrial tachycardia (PAT) ..................................................................... 37
   Causes ................................................................................................................. 38
   Management ........................................................................................................ 38
   Atrial flutter ......................................................................................................... 38
   Causes ................................................................................................................. 39
   Management ........................................................................................................ 39
   Atrial fibrillation ................................................................................................... 39
   Diagnosis ............................................................................................................. 39
   Causes .................................................................................................................. 39
INTRODUCTION

The diagnosis and management of patients with cardiac arrhythmias are undergoing rapid change. Better understanding of the mechanisms that initiate and perpetuate abnormal cardiac rhythms, and exciting new modes of treatment are the principal reasons.

NOTE
The prevalence of sustained arrhythmias in general ICU patients is 12%. Ventricular arrhythmias increase the risk of death and of neurological sequelae.


Intensive care physicians need to be thoroughly familiar with the diagnosis, appropriate investigation and treatment of patients with cardiac rhythm disturbances. They also need to be aware of the indications for specialist intervention that a cardiologist and/or cardiac surgeon might have to offer. Useful reviews are found below.


Tarditi DJ, Hollenberg SM. Cardiac arrhythmias in the intensive care unit. Semin Respir Crit Care Med 2006; 27(3): 221-229. PMID 16791756

Appropriate and timely referral for specialist opinion may be life-saving
Continuous electrocardiographic (ECG) monitoring is employed routinely in all intensive care patients. The principal goals of ECG monitoring in the ICU are to:

- Alert staff to sudden changes in cardiac rhythm which are life-threatening
- Alert staff to changes in cardiac rhythm which may herald life-threatening events
- Identify silent ischaemia

### Specific indications for ECG monitoring

ECG monitoring is mandatory in patients at risk of cardiac arrhythmia. Such patients fall into three categories.

- Those with known or suspected cardiopulmonary disease
- Those exposed to a proarrhythmic environment
- Those with a history of unexplained collapse

#### Those with known cardiopulmonary disease

Examples include:

- Known or suspected ischaemic heart disease (including unstable angina and recent myocardial infarction)
- History of past cardiac arrhythmia
- Cardiothoracic surgery
- Advanced congestive heart failure

#### Those exposed to a proarrhythmic environment

- Electrocution
- Invasive cardiac procedures
- Acute neurological disease
- Acute drug/metabolic toxicity
- Specific endocrine disease
- At risk of profound bradycardia

#### Those with a history of unexplained collapse

See the references below.


Committee, Council on Clinical Cardiology; American College of Cardiology Foundation; Heart Rhythm Society. Recommendations for the standardization and interpretation of the electrocardiogram: part I. J Am Coll Cardiol 2007; 49(10): 1109-1127. PMID 17349896

Potential problems with ECG monitoring

- Lead positioning can be difficult (e.g. the burned patient)
- Movement or muscle artefact may make tracing unreliable (e.g. shivering)
- Single-lead monitoring may make detection of ECG components (such as the P wave) difficult if axis/rhythm alters
- Single-lead monitoring may not demonstrate changes in ST-segments in an (unmonitored) ischaemic zone
- Monitoring patients nursed in the prone position
- Arrhythmia detection software may lack both sensitivity and specificity
- Diathermy interference - rare in ICU. However, may be possible on patient having emergency surgery (e.g. re-mediastinotomy in ICU after heart surgery)

**ANECDOFE** A ventilated patient was about to be DC cardioverted from ventricular fibrillation (VF). Fortunately, the disconnected lead (with transmission of ‘mains hum’ at 50 Hz) was detected in time. Always remember to check that the rhythm you observe is real: lead disconnections can cause apparent VF as well as asystole. Check the rhythm on two other leads and check the patient’s pulse or the displayed arterial tracing. Similarly, the tracing of pulse oximetry and measurement of blood pressure can be helpful.

**ANECDOFE** A patient being prone ventilated for ARDS developed ventricular fibrillation. Substantial delay ensued while the patient was turned to allow defibrillation. In such circumstances, pre-emptive application of an anterior chest ‘stick-on-pad’ prior to turning prone would have facilitated rapid defibrillation.

There is evidence of overmonitoring for arrhythmias and undermonitoring for ischaemia and QTc prolongation.


General principles of ECG monitoring

- The surface ECG records changes in electrical potential of the heart as it depolarises and repolarises; by recording in multiple directions, summation and subtraction are performed to yield a ‘net’ activity in one region of the heart only (the axis).
- Each lead on an ECG printout represents this net activity in just one region. If the net activity is towards the lead, then the resulting deflection will be upwards (positive). If away from the lead, the deflection will be predominantly downwards (negative).
- Standard recording speed is 25 mm/sec. The tracing is drawn on graph-paper which is composed of 5 mm ‘large’ squares, each lasting 0.2 sec. These are sub-divided into five ‘small’ (1 mm) squares (each lasting 0.04 sec).
- The leads which are most commonly viewed are divided in two groups, the standard limb leads (I, II, III, aVR, aVL, aVF), and the chest leads (V1-V6).

In the next ten ‘at risk’ patients undergoing ECG monitoring in your ICU, try to determine on how many occasions the monitoring affected management. What abnormal cardiac rhythms occurred and in which patients? Make a list of which factors might have predisposed to the abnormal rhythms. Address each factor in turn. Were appropriate precautions taken in each case?

See PACT module on Haemodynamic monitoring and management.

**Standard limb leads**

- Leads I and aVL ‘look at’ the left lateral surface of the heart - supplied by the left coronary artery (LCA).
- Leads II, III and aVF ‘look at’ the heart from below. These are the ‘inferior’ leads, which ‘see’ the right ventricle and the inferior part of the left ventricle, in a territory predominantly supplied by the right coronary artery (RCA).
The chest leads (V1-V6)

A 12-lead ECG allows diagnosis of the nature and origin of almost any myocardial ischaemia (an exception would be e.g. right ventricular infarction) or the origin of an arrhythmia e.g. ventricular tachycardia (VT) originating in either the right or left ventricle. Wherever possible therefore, a 12-lead ECG recording should be obtained. Serial ECGs will be required in evolving situations.

Normal cardiac conduction

To understand the principles and concepts involved in the accurate diagnosis and treatment of arrhythmias, a brief review of the anatomy and physiology of the conducting system may be useful.
Sinoatrial node (SAN)

- Sited in the subepicardium, junction of right atrium (RA) and superior vena cava (SVC)
- Extensive autonomic innervation
- Abundant blood supply via SA nodal artery (proximal branch of RCA in 55% population) or left circumflex coronary artery

Atrioventricular node (AVN)

- Subendocardial structure within interatrial septum
- Extensive autonomic innervation
- Blood supply via AV nodal artery (distal branch of RCA in 90-95% population)

His bundle

- Formed by Purkinje fibres emerging from distal AV node, forming tubular structure which runs through the membranous septum to the muscular septum and divides into the bundle branches
- Sparse autonomic innervation
- Blood supply from AV nodal artery and septal branches of LAD artery

Bundle branches

- Anatomy varies
  - Right bundle extends down right side of interventricular septum to base of anterior papillary muscle where it divides
  - Left bundle usually divides into two or three distinct fibre tracts - a left posterior and a left anterior hemibundle
- Little autonomic innervation
- Extensive blood supply from RCA and LCA

Normal conduction is initiated by the SA node, and results in a wave of depolarisation that spreads through the atria, causing atrial contraction. Atria and ventricles are electrically isolated from one another in all but one site - the AV node which serves to delay conduction between atria and ventricles, allowing time for the atrial component of ventricular filling and protect against the development of ventricular fibrillation (VF).

This is the normal situation. Consider bypass (or ‘accessory’) tracts which allow ‘aberrant conduction’.

The impulse is then carried through the His-Purkinje system, and results in ventricular depolarisation.


Ellenbogen KA, Kay GN, Wilkoff BL, editors.: Clinical Cardiac Pacing and Defibrillation. 2nd ed. WB Saunders Company; 2000. ISBN 0721676839
Q. In the table below, insert the normal values of the following ECG intervals: 0.12; 0.12-0.20; 0.38-0.42; 0.012.

<table>
<thead>
<tr>
<th>Name</th>
<th>Represents</th>
<th>Normal values [sec]</th>
</tr>
</thead>
<tbody>
<tr>
<td>P wave</td>
<td>Atrial depolarisation</td>
<td>0.012</td>
</tr>
<tr>
<td>PR interval</td>
<td>Time to conduct through AVN and His bundle system</td>
<td>0.12-0.20</td>
</tr>
<tr>
<td>QRS duration</td>
<td>Depolarisation of septum and ventricular tissue</td>
<td>0.12</td>
</tr>
<tr>
<td>QT interval (QTc)</td>
<td>Ventricular repolarisation</td>
<td>0.38-0.42</td>
</tr>
</tbody>
</table>

A. P wave, Atrial depolarisation, 0.012
PR interval, Time to conduct through AVN and His bundle system, 0.12-0.20
QRS duration, Depolarisation of septum and ventricular tissue, 0.12
QT interval (QTc), Ventricular repolarisation, 0.38-0.42

NOTE SAN, AVN, and His bundle depolarisation are electrically silent on the ECG. An increase in QRS duration is seen when:
The conducting system is damaged (leading to bundle branch block)
The ventricle is being activated through an alternative AV pathway (e.g. an accessory or non-His-AV bundle)
Ventricular activation originates in the ventricle itself (e.g. a ventricular escape focus, or VT).

NOTE All areas of the heart have the capacity for automaticity, and therefore can become pacemakers. In general, the rate of pacemaker activity is lower the further down the heart one goes:
An atrial pacemaker may ‘fire’ at rates of <250 bpm
The SA node may have re-entrant cycles producing similar rates of <200 bpm
The common His bundle generally ‘fires’ at rates of <70 bpm. Lower His bundle pacemaker sites fire at slower rates, and the apices of the ventricles at rates of 35-40 bpm and will produce wide complexes.

Mechanisms of cardiac arrhythmias

Abnormal automaticity and abnormal conduction are two major causes of cardiac arrhythmias.

Abnormal automaticity

Automatic arrhythmias, such as automatic atrial tachycardia, require no specific stimulus for initiation and may be persistent. Enhanced phase 4 depolarisation (for details refer to the Section 3, Function of ion channels, and corresponding references) would provoke such arrhythmias.
Abnormal conduction

Abnormal conduction may promote re-entry in heart muscle. Re-entry is responsible for most clinically important arrhythmias including VT associated with coronary artery disease, atrial flutter, AV nodal re-entrant tachycardia, atrioventricular re-entry tachycardia as observed in the Wolff-Parkinson-White Syndrome (see Task 5).

NOTE The following section assumes a degree of competence in ECG interpretation. You will find the following references particularly helpful.


http://www.ecglibrary.com/

Consider apprenticing yourself to a cardiological colleague, particularly while doing ‘ECG reporting’, until you have ready familiarity with common ECG abnormalities.

Q. This ECG was obtained from a 55-year-old man with typical ischaemic chest pain. Analyze and explain the ECG findings.
A. The ECG is consistent with acute inferior wall myocardial infarction (ST elevation in the inferior leads II, III, aVF). High-degree AV nodal block is present suggesting a proximal location of the RCA obstruction and a consequent large inferior wall infarction. Right ventricular involvement may be identified by additional recording of lead V4R.

**Q. Does the high-degree nodal block in this patient have implications for patient mortality?**

A. When high-degree AV nodal block occurs in acute myocardial infarction the in-hospital mortality rate is two and a half times that of inferior myocardial infarction without high-degree AV block.

**Q. What treatment option is optimal?**

A. This patient should be referred for primary percutaneous coronary intervention (PCI) due to ST elevation myocardial infarction (STEMI) when resources are available or transferred promptly to another suitable institution. (Unless in a country where early PCI is not the routine practice in STEMI patients, the majority of patients with STEMI should be treated with immediate PCI).

See the PACT module on Acute myocardial ischaemia.

**Q. This ECG was obtained from a 60-year-old man with typical ischaemic chest pain. He was known to have a normal routine pre-operative ECG one year ago. Outline the ECG findings.**

A. This case illustrates an acute anterior myocardial infarction complicated by a left bundle branch block.

**Q. Does the development of bundle branch block during the acute phase of anterior myocardial infarction have a diagnostic implication?**
A. Yes. It indicates extensive anterior wall infarction, because such conduction problems indicate an occlusion proximally in the LAD coronary artery.

Q. Is there a prognostic implication?

A. Yes. When anterior myocardial infarction is complicated by bundle branch block, early death can occur because of pump failure and ventricular tachycardia or fibrillation.

Q. The finding of a bundle branch block as a complication of anterior wall infarction calls for aggressive treatment. Please outline.

A. This patient should be referred for primary percutaneous coronary intervention. Development of second or third degree heart block in association with bundle branch block during anterior MI would necessitate temporary pacing.

Q. This ECG was obtained from a 22-year-old woman with a history of pyrexia, and rigors. On examination there were profuse splinter haemorrhages and a short early diastolic murmur at the lower left sternal edge. What do the clinical findings suggest as the most likely diagnosis?

A. The clinical features in this patient are those of an active infective endocarditis of the aortic valve. The development of high-degree atrioventricular block suggests the development of an aortic root abscess.

Q. How do you interpret the QRS voltage in the ECG?

A. Low voltage is present in the limb leads as the amplitude of the QRS complex in each of the three standard limb leads (I, II, and III) is <5 mm. However, definition of low voltage in all leads requires the average voltage in the limb leads to be <5 mm and the average voltage in the chest leads to be <10 mm.
Q. Outline some causes that might account for the low voltage ECG in this or other patients?

A. It may be caused by a pericardial effusion in this patient. Other causes to be considered are obesity, anasarca, underlying myocardial disease (amyloidosis), lung diseases, severe hypothyroidism.

Q. Using knowledge of the anatomical relationships between the valve cusps and adjacent structures, what will an echocardiographer examine in this patient?

A. The echocardiographer can more clearly define the perivalvular extension of infection. Extension of the infection beyond the valve leaflets into the surrounding tissue is an ominous step in the progression of infective endocarditis.

Q. In patients with endocarditis complicated by perivalvular extension of infection, what additional management option should be considered?

A. Cardiac surgery should be considered to debride invasive infection, ablate abscesses and reconstruct anatomical damage.

Q. This ECG was obtained from a 62-year-old lady admitted to ICU following a cardiac arrest. The only past medical history of note was treatment with antidepressants which are known to be associated with orthostatic hypotension and arrhythmias. What is the relevant ECG abnormality that may have predisposed to her cardiac arrest?

A. Tricyclic antidepressants (like quinidine) may prolong the Q-T interval as in this patient and predispose to ventricular arrhythmias.

Torsade de pointes was the underlying cause of this patient’s cardiac arrest.

We have now assessed a number of causes of an abnormal ECG. Understanding of the normal anatomy and physiology of the conducting system has been emphasised, in order to allow interpretation of ECG changes in pathological conditions. These skills will now be used to interpret arrhythmias.
Assessment of heart rate (= ventricular rate)

As the standard recording speed is 25 mm/second, an estimate of rate can be most easily determined by

- dividing the number of ‘large’ squares between QRS complexes into 300
- counting the number of QRS complexes over 30 large squares (i.e. the number in 6 seconds) and multiplying by ten

By convention (in adults), a rate of >100 bpm is classified as a tachycardia and <60 bpm as a bradycardia.

**ANECDOTE** A 22-year-old fit rugby player was admitted with abdominal pain. His heart rate was 74 bpm, which reassured his carers. Several hours later, he collapsed suddenly. At laparotomy, 400 mL of pus was drained from an appendiceal abscess.

In this case tachycardia was expected due to pain and infection. The ‘normal’ heart rate in this case was not normal (probably due to athlete’s heart and vagal stimulation) and was misleading to his clinical carers.

**ANECDOTE** A 27-year-old patient was brought to the Accident and Emergency Department, complaining of a severe headache. She would intermittently scream inconsolably. Neurological examination was unremarkable. Heart rate was 65 bpm. Two hours later after CT of the brain, neurosurgeons performed a shunt to treat her hydrocephalus.

In this case tachycardia was expected because of severe headache but the normal heart rate in her condition raised the suspicion of increased intracranial pressure.

**Warning** Remember, the heart rate must be put in the context of the individual patient. A ‘normal’ heart rate may be abnormal in certain circumstances.

**Assessment of heart/cardiac rhythm**

- Assessment of regularity.
- Assessment of QRS duration.
- If P waves present - implies co-ordinated atrial electrical activity. See algorithm A below.
- If P waves absent - implies absence of co-ordinated atrial electrical activity. See algorithm B below. (Ensure however that they are not ‘hidden’ after or within QRS complex).
- Atrial flutter - atrial activity at 300 bpm - ‘saw-tooth’ baseline to ECG. Consider this if QRS rate is regular and is a division of 300. Atrial Flutter with 2:1 a-v block (ventricular rate 150/min) or with 3:1 a-v block (ventricular rate 100/min) is sometimes confused with sinus rhythm.
- Atrial fibrillation (AF) - chaotic atrial activity and irregular QRS rate.

Algorithm A: P wave activity present

**ARE THERE P WAVES BEFORE EVERY QRS COMPLEX AND NOWHERE ELSE?**

- **YES**
  - Is there always a constant relationship to each QRS?
    - **YES**
      - Low-normal rate, with morphology and axis similar to previous ECGs.
        - **YES**
          - Sinus rhythm
        - **NO**
          - Paroxysmal atrial tachycardia
    - **NO**
      - What is the relationship to QRS complexes?
        - None: P waves are entirely independent of the QRS complexes
          - V rate < 60
            - Complete heart block
          - V rate > 120
            - Consider ventricular tachycardia
        - Conducted PR intervals constant, with intermittent loss of conduction
          - Mobitz type II heart block
        - Progressive lengthening of PR interval, a dropped beat, then a repeat cycle
          - Mobitz type I heart block

- **NO**
  - QRS complex usually wide
  - QRS complex usually narrow

Algorithm B: no clear P wave activity present

**NO VISIble P WAVES**

- Sinus arrest
  - SA block
  - AFib
  - A Flutter

- ?No clearly visible P waves (low voltage P waves, obese subject, pericardial effusion)
  - P waves buried at the same point within each QRS complex?
    - Consider retrograde activation of the atria from an impulse arising below them
  - P waves actually occur independently of the QRS complexes but are just hard to see

**Narrow complex tachycardia?**

- Tachycardia (SVT)

**Broad complex tachycardia?**

- Possible VT
  - **YES**
    - Complete heart block
  - **NO**
    - Complete heart block
Q. ECG 1. What is the rhythm?

A. Atrial fibrillation: irregular ventricular rhythm, normal heart rate, narrow QRS complexes, no visible P waves

Q. ECG 2. What is the rhythm?

A. Atrial flutter: regular ventricular rhythm, narrow QRS complexes, undulation waves evident with 3:1 (atrioventricular conduction) block.
**Q. ECG 3. What is the rhythm?**

A. VT - broad complex tachycardia: regular ventricular rhythm, broad QRS complexes, no visible P waves.

**Q. ECG 4. What is the rhythm?**

A. Complete heart block: regular ventricular and atrial rhythm, no constant relation between P waves and QRS complexes. Narrow QRS complexes indicates ventricular stimulation origin high in the His bundle. In the majority of patients with complete heart block, QRS complexes are broad and the ventricular rate is slower.
We have outlined a method for assessing cardiac rhythm disturbance in a systematic way. We will now consider the underlying causes for rhythm disturbance, and the potential treatment strategies available.
Knowledge of the ionic currents responsible for the action potential and the nature of cell-to-cell electrical transmission are important for a comprehensive understanding of the cardiac action potential and the interaction of drugs and hormones with the ion channels.

### Ion channels

**Structure**

Ion channels are proteins that traverse the plasma membrane. The major function of ion channels is the rapid and selective movement of ions in and out the cell.

The selective permeability of a channel for a particular ion in preference to others is the basis for the classification of ion channels into Na⁺, K⁺, and Ca²⁺ channels among others.

**Sodium currents**

The sodium current is primarily responsible for the depolarisation phase of the action potential.

**Calcium currents**

There are two major Ca²⁺ currents in cardiac cells, the L-type (slow inward current) and the T-type which is faster and smaller than the L-type current.

**Potassium currents**

Several K⁺ currents are important in the cardiac tissue. Two key currents are involved in the process of repolarisation (phase 3) during the action potential and diastolic depolarisation (phase 4). You will find the following references useful in this connection.


Electrogenic pumps

In addition to the various ion channels, there are electrogenic transporters which contribute to the membrane potential.

*The Na⁺ /K⁺ pump*

Adenosine triphosphatase (ATPase) dependent, inhibited by digitalis glycosides, exchanges two potassium ions for three sodium ions. The pump is electrogenic and increases the intracellular negative potential. It promotes repolarisation and maintains a low Na⁺ and high K⁺ inside the cell.

*The Na⁺ /Ca⁺⁺ exchanger*

The Na⁺/Ca⁺⁺ exchanger extrudes three Na⁺ ions for each entering Ca⁺⁺ ion when the membrane potential is more positive than −40 mV, thereby increasing intracellular negativity.

Conditions that increase intracellular Ca⁺⁺ also increase the extrusion of Ca⁺⁺; this depolarising process may produce early and/or late after-depolarisations.

Digitalis inhibits the enzyme sodium-potassium adenosine triphosphatase (ATPase) and thus interferes with the sodium pump, causing sodium to accumulate in the cell, which in turns alters sodium-calcium exchange. Digitalis intoxication results in enhanced impulse formation, based on triggered activity (early after-depolarisations). Common digitalis arrhythmias are atrial tachycardia, junctional tachycardia and VT. Digitalis also causes SA and AV block.

Q. Describe those conditions that may promote expressions of digitalis intoxication.

A. Conditions that may promote arrhythmic expression of digitalis toxicity are increased sympathetic stimulation (which includes intracellular calcium overload), hypokalaemia, hypercalcaemia, hypomagnesaemia, diuretics, ischaemia/reperfusion, increased wall tension and heart failure. All of these are independently capable of producing triggered activity.

Q. Outline your approach to the treatment of digitalis intoxication?

A. Treatment consists of discontinuation of digitalis, bedrest (no sympathetic stimulation) and correction of potassium-magnesium deficits. If haemodynamically unstable, phenytoin is indicated unless digitalis antibodies are available. During treatment with phenytoin, ventricular pacing may be indicated since suppression of the tachycardia may be followed by asystole.

Q. There are a number of factors (fourteen are listed below) that increase the likelihood of the arrhythmias which are commonly encountered in the intensive care setting. List the ones of which you are aware:

**NOTE** The assessment and treatment of any cardiac arrhythmia must include full clinical assessment of the patient.
A. 
- Pre-existing cardiac disease
- Treatment with anti-arrhythmics (this is with reference to proarrhythmic potential e.g. class Ic agents)
- Recent macrovascular (i.e. occlusive coronary) event
- Microvascular disease causing ischaemia (e.g. diabetes mellitus, sepsis)
- Altered acid-base status
- High PaCO₂
- Abnormal electrolyte balance (especially hypokalaemia and hypomagnesaemia)
- Endogenous catecholamines (pain, anxiety)
- Exogenous catecholamines (inotropes, vaspressors)
- Presence of intracardiac catheters or pacing wires
- Suctioning, bronchoscopy, airway manipulation
- Deep anaesthesia (especially young patients but should be unusual in ICU)
- Anaesthetic drugs e.g. pancuronium, methoxamine
- Other drugs

Determine, in the next ten patients admitted to your ICU, which proarrhythmic drugs are used and make a list of concomitant factors that might provoke arrhythmias.

**Arrhythmias management in the critically ill - rules**

As management is complex, some safe and simple rules are outlined below:

1. **Not all arrhythmias need to be treated**

The following questions can help in determining the urgency of intervention.

---

_all anti-arrhythmic drugs are potentially proarrhythmic._

Is the patient haemodynamically compromised? If so, act now (e.g. wide complex tachycardia or profound bradycardia with significant hypotension).

Is the patient likely to become haemodynamically compromised? (e.g. AF with impaired ventricular function). Wait and watch.

If the patient is haemodynamically stable, do you need to intervene at all?

---

Keep a diary of interventions for cardiac arrhythmias in your ICU for one month. Was the correct diagnosis of the underlying arrhythmia made in each case? Was intervention necessary on each occasion?
2. ‘Electricity’ is generally safer than drugs

In general, every anti-arrhythmic drug is proarrhythmic, nearly all are negatively inotropic, and many have long half-lives. If in doubt, ‘electricity’ is safer and quicker. Thus, when the patient is haemodynamically compromised:

If the rhythm and rate are fast, consider cardioversion.
If the rhythm and rate are slow, consider pacing.

3. Correct all correctable abnormalities

Do this simultaneously with other treatment if the patient is haemodynamically compromised; otherwise, do this first:

Check arterial blood gases and electrolytes.

‘Normal’ isn’t necessarily normal e.g. in patients with cardiac disease or recent cardiac surgery, maintaining potassium levels at the upper range of normal reduces the risk of arrhythmia (this does not apply after cardiac transplantation).

Magnesium deficiency is common in ICU patients. Blood levels do not reflect tissue levels, generous replacement/therapy may be useful. However, particularly in renal failure patients, care should be taken in replacement, as side effects occur with high levels.

**ANECDOFE** Shortly after arrival in the ICU, a patient with acute renal failure developed refractory VF. Defibrillation was unsuccessful until calcium gluconate, bicarbonate and intravenous insulin and glucose had been administered.

**ANECDOFE** A patient with severe burns suffered repeated runs of AF, despite all measured electrolytes being normal. The patient responded well to 4 mmol intravenous magnesium.


4. Treat all treatable ischaemia

Myocardial ischaemia as a cause of arrhythmia must always be considered, and if thought to be the cause, must be treated. Consider:
• Pharmacotherapy (β-blockade, nitrates)
• Normalise arterial pressure (afterload) and preload
• Mechanical support (intra-aortic balloon pump or other devices)
• Temporary transvenous (or external) pacing
• Percutaneous intervention (angioplasty ± stent)
• Surgery

Ischaemia in the presence of hypotension and VT/atrial tachycardia may need additional urgent non-pharmacological intervention: inotropes increase BP (and hence coronary perfusion pressure) but in the presence of flow-limiting coronary disease, the increased coronary flow may be offset by increased cardiac work and the persistence of ischaemia. Moreover, the tachycardia may be perpetuated. In these situations, use of intra-aortic balloon counterpulsation may be considered.

5. Consider your intravascular catheters

Is a central venous pressure (CVP) or pulmonary artery (PA) catheter ‘tickling’ the right atrium, tricuspid valve or right ventricle? If in doubt, withdraw it a few centimetres. Similarly, you may need to remove a PA catheter or reposition temporary pacing wires.

Is the patient ‘under- or over-filled’? Atrial fibrillation (AF), in particular, responds to altered blood volume status.

6. Consider drug toxicity

All anti-arrhythmics may be proarrhythmic. Many other drugs (often those which affect neurotransmission e.g. antidepressants, anticonvulsants and some anaesthetic agents) may be proarrhythmic.


A patient with paroxysmal AF was treated with oral amiodarone. Four months later she presented in sustained AF. Increasing dosage resulted in repeated VT arrests. This was the ECG on admission to the ICU.

Q. What is the ECG abnormality and the cause of her arrhythmia?

A. Long QT syndrome, with possible torsade de pointes.
Q. The emergence of a sustained ventricular tachycardia after starting a Class IC anti-arrhythmic drug or after an increase in drug dose suggests an adverse drug effect. What electrophysiological abnormality may have occurred? What treatment would you consider?

A. Lengthening of the membrane action potential duration prolongs the QT interval and may therefore promote the occurrence of early after-depolarisations and polymorphic ventricular arrhythmias (torsade de pointes).

Q. Which electrolyte abnormalities may exacerbate this pathology?

A. Hyperkalaemia and hypomagnesaemia.

Q. In this setting, detection and treatment should be early and relatively straightforward. Outline your approach.

A. The offending drug should be stopped and electrolyte disturbances (potassium, magnesium) corrected. Intravenous magnesium 4-8 mmol by intravenous bolus over five minutes is suggested even in normomagnesaemia.

Q. If the above (including intravenous magnesium) is unsuccessful, what might you consider next?

A. An increase in the basic heart rate with isoproterenol or by ventricular pacing may be necessary.

General treatment options of arrhythmias

- Electricity – pacing, cardioversion, defibrillation
- Pharmacology
- Other (catheter ablation, Implantable cardiac defibrillator (ICD), surgical intervention). See Task 6

Pacing

Cardiac pacing is a definitive life-saving treatment for bradyarrhythmias, in addition to its role for the termination of certain tachyarrhythmias. The modes available are:

- Mechanical
- Transcutaneous electrical
- Transvenous electrical
- Transoesophageal
- Transthoracic

Mechanical pacing

Before addressing the electrical methods, a brief note on Percussion (and cough) pacing may be warranted.
Percussion pacing is thought to work by mechanical stimulation of excitable myocardial tissue, either by direct or transmitted physical forces - performed with serial chest thumps, or cough-induced.

**NOTE**

- Performed using serial thumps to the sternum with clenched fist thumps from a height of 20-30 cm above the sternum
- 1/4-1/3 of the force used to cardiovert VF
- Rate: 60-90 bpm
- Efficacy monitored/confirmed by a palpable peripheral pulse or displayed arterial tracing (ECG can be misleading)


Cough pacing (also known as cough CPR): Precise mechanism of action unclear - possibly via mechanical stimulation. It is suggested, in a conscious patient suffering a witnessed haemodynamically important bradycardia, that the patient be instructed to cough forcefully every 1-3 seconds.

**Mechanical pacing** can offer instantaneous and effective maintenance of cardiac output. Most successful when used early in witnessed arrests. In most instances secondary cardioversion/defibrillation/pacing is the ultimate solution.

Although the above manoeuvres are of interest, most serious arrhythmias, in the ICU context, require rapid initiation of more definitive (electrical) measures.

**Transcutaneous electrical pacing**

Application of self-adhesive surface patch electrodes coated with high-impedance conductive gel allows rapid establishment of pacing.

- Skin should be cleaned to improve contact
- Patches secured anteriorly (negative) and posteriorly
- Safe to perform external cardiac massage during pacing
- Prominent muscle twitches in the unconscious patient may make palpation of the pulse difficult - confirm efficacy using intra-arterial trace or Doppler

Most patient require analgesia and, some of them, sedation.

During transcutaneous pacing, sedation should be considered. Pacing thresholds increase significantly after cardiac and thoracic surgery.

Q. Which features of cardiac and/or thoracic surgery would you expect to cause an increase in pacing thresholds during transcutaneous pacing?

A. Increased intrathoracic air (pneumothorax), pericardial effusion (drain), myocardial ischaemia/electrolyte disturbances.

Q. How would you treat them?

A. Taking the above conditions in turn, reduce positive pressure ventilation and drain the pneumothorax, drain the pericardial effusion and correct metabolic abnormalities and factors that might aggravate ischaemia such as hypoxaemia. CPR may be necessary if the cardiac output is inadequate.

Transvenous pacing

Used for atrial and/or ventricular pacing, this provides the most reliable means of temporary pacing, although it requires a degree of operator skill and may take some minutes to initiate.

- Right internal jugular approach is the fastest and easiest
- Femoral approach is difficult and, given the reported higher incidence of infection, is avoided where possible
- In urgent situations, the most practical route is likely the correct route
- The more distal the approach, the higher the risk of lead displacement
- Defibrillator needs to be at hand
- Usually ventricular, single chamber pacing is adequate
- Performed under fluoroscopic guidance normally - aim to place ventricular lead in the right ventricular apex
- A simple ‘pacing-only’ balloon tipped catheter may be used
- If a pulmonary artery catheter insertion is planned, a catheter with special pace port for insertion of a pacing wire may be used.

Confirm correct position with measurement of threshold parameters, stability, chest radiograph (see figure below) and 12-lead ECG recording.
Avoid the subclavian approach if possible, in patients likely to require subsequent permanent pacemaker implantation.

Chest radiograph showing correct position of ventricular pacing wire.

Q. What pattern of conduction block would you expect to see in a 12-lead ECG, presuming a correctly positioned ventricular lead?

A. Since ventricular pacing is from the right ventricle, the QRS complexes have the left bundle branch block configuration.

In addition to checking stability, pacing thresholds and chest radiographic position at the time of temporary wire insertion, ensure the lead(s) is/are secure and then check the pacing thresholds daily. Pacemaker output is usually set at three times threshold value.

In a patient who is undergoing temporary ventricular pacing, make sure you know how to assess the sensing mode, and the pacing threshold. In one such patient, if safe and appropriate: Adjust the rate of pacing, while looking at the haemodynamic monitoring. Is the patient’s haemodynamic status improved with a faster rate, or paradoxically worse? Practically, the importance of atrial systole and its contribution to the stroke volume should be assessed individually. Some patients have better cardiac output with sinus bradycardia than with a faster paced ventricular rhythm.
Drugs such as isoprenaline/isoproterenol or dobutamine may also be considered but pacing is the preferred option. Increased heart rate by pacing increases myocardial oxygen consumption less than the same increase by drugs, since pacing has no effect on the contractility. Instruction for checking pacing thresholds may be found in the following reference.


**THINK** If, despite successful (ventricular) pacing, the patient remains haemodynamically compromised, consider whether AV pacing would be more appropriate or whether there is another reason for instability.

### Causes for malfunctioning in pacing

<table>
<thead>
<tr>
<th>Potential cause</th>
<th>Investigation/evaluation</th>
<th>Management</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lead displacement (macro or micro displacement)</td>
<td>Check chest radiograph appearance. ECG morphology (paced)</td>
<td>Reposition lead (temporarily may increase output)</td>
</tr>
<tr>
<td>Myocardial infarction/oedema/inflammation in area of lead contact</td>
<td>ECG for evidence of previous/ongoing ischaemia/infarction</td>
<td>Reposition lead</td>
</tr>
<tr>
<td>Metabolic/electrolyte disturbance</td>
<td>Lab investigations</td>
<td>Correct as appropriate</td>
</tr>
<tr>
<td>Battery/lead malfunction</td>
<td>Ensure position of leads unchanged and adequate, check leads and battery in turn</td>
<td>Replace/reconnect as appropriate</td>
</tr>
<tr>
<td>Rupture of IV septum or RV free wall</td>
<td>ECG morphology change. Check CXR. Echocardiography for lead position and pericardial effusion/tamponade</td>
<td>Lead reposition/replacement Pericardiocentesis</td>
</tr>
</tbody>
</table>

**Antitachycardia pacing**

In addition to its widespread use in the treatment of bradycardias, pacing may be used in the treatment of tachycardias:

- To prevent emergence of rhythm disturbances which occur during episodes of relative bradycardia.
- Also used in arrhythmias due to repolarisation abnormalities - atrial flutter (Type I), AV node re-entry, AV re-entry, atrial tachycardia, VT.
- Overdrive pacing - 20-30% faster than the tachycardia rate.
• Underdrive pacing - pace asynchronously at a rate lower than the tachycardia (only useful if tachycardia rate >150 bpm).

Antitachycardia pacing may terminate, re-initiate or accelerate existing tachycardias. The risks of acceleration or fibrillation increase as the rate and duration of antitachycardia pacing increase.

New modalities of pacing

Simultaneous right and left ventricular pacing - resynchronisation pacing has been shown to improve morbidity and mortality of heart failure in non-ICU patients.

It represents a long-term treatment option but its role in the setting of acute heart failure is unclear. The beneficial effect on morbidity and mortality has been shown for the following patients:

• NYHA function class III/IV, LVEF ≤35%, QRS duration ≥120 ms
• NYHA function class II, LVEF <35 %, QRS duration >150 ms

Pharmacology

No large clinical trials have been performed of the use of anti-arrhythmic therapies in the general ICU population. Care must be taken therefore when applying the results found in the non-critically ill and those on the coronary care unit, to those in the ICU setting. The choice of drugs, however, remains the same.

<table>
<thead>
<tr>
<th></th>
<th>Class 1</th>
<th>Class 2</th>
<th>Class 3</th>
<th>Class 4</th>
</tr>
</thead>
<tbody>
<tr>
<td>Method of action</td>
<td>Blocks fast sodium channel</td>
<td>β-blockade</td>
<td>Increases APD</td>
<td>Slow calcium channel blockade</td>
</tr>
<tr>
<td>1a APD↑</td>
<td>1b APD↓</td>
<td>1c APD same</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Primary site of action</td>
<td>A, V, AP</td>
<td>V</td>
<td>His, V, AP</td>
<td>SAN, AVN</td>
</tr>
<tr>
<td>AVN and His bundle effect</td>
<td>↑His RP</td>
<td>↓His RP</td>
<td>↑His RP</td>
<td>↑AVN RP and ↑His RP</td>
</tr>
</tbody>
</table>

Drug classification-related abbreviations.
APD: action potential duration, RP: refractory period, AVN: atrioventricular node, SAN: sinoatrial node

All anti-arrhythmic drugs are potentially proarrhythmogenic.
Examples

- Class 1a: Quinidine, procainamide
- Class 1b: Lidocaine, phenytoin
- Class 1c: Flecainide, encainide
- Class 2: β-blockers
- Class 3: Amiodarone, disopyramide, sotalol (high dose only), dronaderone
- Class 4: Verapamil, diltiazem
- Other: Adenosine, digoxin (vernakalant Class I and III)

Although drug classification is easy, it is better to choose the drug for the diagnosis, rather than fit the diagnosis to the drug.

Make a list of the most commonly used anti-arrhythmic drugs in your department and look for potential drug interactions.

It is extremely important when caring for critically ill patients to be familiar with rhythm disturbances and be able to institute appropriate therapy. Optimal management of bradyarrhythmias and tachycardias requires expertise in electrocardiography, pharmacodynamics, pharmacokinetics and bedside clinical acumen.
These are common, but usually only transient in the ICU setting (i.e. related to airway manipulation, hypoxaemia etc). In this section we will cover diagnosis of the more common bradyarrhythmias and consider their treatment in the ICU.

**Sinus node (SAN) dysfunction**

‘Sinus node dysfunction’ encompasses a heterogeneous group of conditions, including:

- Sinus bradycardia
- Sinus arrest
- Sinoatrial block
- Sick sinus syndrome

Sinus node dysfunction may be exacerbated by many medications, but rarely needs treatment in the ICU setting.

*Sinus node dysfunction in the context of acute myocardial infarction*

This is a relatively common finding (5–30%) and is often associated with concomitant AV nodal block. Usually no treatment is required, unless in the case of cardiac failure, significant hypotension, or continuing myocardial ischaemia.

**NOTE**

Intermittent sinus node dysfunction may respond to small doses of atropine (note: rate response is unpredictable). Consider temporary pacing if the bradycardia is:

- Prolonged
- Severe
- Aggravating ventricular irritability
- Not responding to atropine and isoprenaline

**Atrioventricular (AV) conduction disease**

The nomenclature of AV conduction disease refers to the ECG patterns and not to the underlying pathophysiology or anatomy. Treatment based on an ECG diagnosis is considered in the clinical context.

**1st degree AV block**

This refers to prolongation of the PR interval (>0.21 sec), and is strictly speaking not conduction block, merely conduction delay. The QRS duration is normal (narrow QRS). See the ECG below.
2nd degree AV block

This results from intermittent failure of atrial depolarisation to reach the ventricles. Ventricular beats that do occur result from normal conduction pathways.

Type I (Mobitz I or Wenckebach)

- Progressive prolongation of the PR interval, then a ‘dropped beat’
- Commonly occurs at the level of the AV node (narrow QRS)

Type II (Mobitz II)

- Normal, constant PR interval, with intermittent ‘dropped beats’
- Commonly occurs at the level of the AV node (narrow QRS)

Q. What degree of AV block is shown in the ECG rhythm strip?

![ECG rhythm strip]

A. Second degree AV block - Mobitz type I (Wenckebach phenomenon).

3rd degree AV block (complete heart block)

In complete heart block, although the atria depolarise normally, none of the atrial depolarisations reach the ventricles, which beat independently in response to an infranodal pacemaker (wide QRS). There is AV dissociation. See the ECG.

![ECG rhythm strip]

AV node dysfunction in the context of acute myocardial infarction (MI)

A degree of complete AV block occurs in 12-25% of patients with acute myocardial infarction, most commonly in the context of inferoposterior MI (with right ventricular involvement). AV block in this context usually results from AV nodal ischaemia, is usually transient and usually resolves. In anterior MI, AV nodal block usually occurs in the bundles and can progress suddenly and without warning to complete AV block.

Risk of progression to higher degrees of heart block/asystole, and therefore requirement for temporary backup pacing varies.
Risk of progression to high-grade block

**NOTE** 1st and 2nd degree (type I) block rarely require pacing (low risk of progression). However, 2nd degree (type I) block associated with a wide QRS (especially in the context of anterior myocardial infarction) is an indication for temporary backup pacing.

**THINK** In this context, what is the significance of the widened QRS complex, and what does it imply?

Type II 2nd degree heart block (with a wide QRS) and type II 2nd degree heart block in the context of anterior myocardial infarction (with wide or narrow QRS complex) are indications for temporary backup pacing.

Anterior MI with anything more than low-grade block may exhibit abrupt transition to high-grade block with a slow, unreliable ventricular escape rhythm. This combination is associated with severe left ventricular dysfunction and high mortality.

**Bundle branch block (BBB)**

In the case of failure of conduction in the right or left bundles of His, the ECG changes are of bundle branch block (BBB). In the intensive care setting, the diagnosis of BBB on the ECG does not by itself usually indicate that any specific action is necessary, although the development of new BBB should alert the physician to the possibility of myocardial infarction.

See PACT module on Acute myocardial ischaemia.

**Right bundle branch block (RBBB)**

Due to delayed depolarisation of the right ventricular free wall:

- QRS duration >0.12 seconds
- Secondary positive deflection in lead V1 (rsR)
- Deep slurred S wave in I, aVL, V4-6
- Secondary ST, T changes in V1-V3

See the ECG below.

**NOTE** The appearance of new RBBB should raise suspicion for acute cor pulmonale and requires further diagnostic procedures (echocardiography).
Left bundle branch block (LBBB)

Due to depolarisation of free wall of the left ventricle, together with reversal of the direction of septal depolarisation:

- QRS duration >0.12 seconds
- Absence of septal Q waves in I, aVL and V4-V6
- Absence of secondary R in V1

See the ECG below.

Bundle branch block in the context of acute MI

Development of BBB in anterior MI signifies a poorer prognosis (due to large infarct size, left ventricular dysfunction and conduction abnormalities). It is, however, difficult to predict those patients who will need temporary pacing. Insertion of a backup temporary pacing wire should be considered in the case of bifascicular and trifascicular block and alternating BBB.
ICU patients with implanted permanent pacemakers

Potential issues in patients with permanent pacemakers relevant to ICU doctors include those that are procedure related and occur mostly acutely, and those that are a consequence of pacemaker battery or lead malfunction. Most common issues are listed in the table, below:
### Complications in patients with permanent pacemakers

#### Recent pacemaker implantation

<table>
<thead>
<tr>
<th>Problem</th>
<th>Cause</th>
<th>Management</th>
</tr>
</thead>
<tbody>
<tr>
<td>Haematothorax/Pneumothorax</td>
<td>Procedure related injury of vessels or lung</td>
<td>Thoracic drainage, surgical haemostasis as necessary</td>
</tr>
<tr>
<td>Tamponade</td>
<td>Lead perforation</td>
<td>Pericardiocentesis, lead repositioning</td>
</tr>
<tr>
<td>Infection</td>
<td></td>
<td>Treat infection and sepsis, Device removal if systemic infection</td>
</tr>
</tbody>
</table>

#### Non-recent pacemaker implantation

<table>
<thead>
<tr>
<th>Problem</th>
<th>Cause</th>
<th>Management</th>
</tr>
</thead>
<tbody>
<tr>
<td>Muscle twitching/Hiccups</td>
<td>Lead dislodgement/migration causing muscle or phrenic nerve stimulation</td>
<td>Reprogramming, lead repositioning</td>
</tr>
</tbody>
</table>

#### Pacemaker malfunction resulting in bradycardia

<table>
<thead>
<tr>
<th>Problem</th>
<th>Cause</th>
<th>Management</th>
</tr>
</thead>
<tbody>
<tr>
<td>Failure to output</td>
<td>Battery depletion</td>
<td>Temporary transvenous or external pacing if patient severely bradycardic and compromised Battery or lead replacement Pacemaker reprogramming</td>
</tr>
<tr>
<td>(Pacing artefacts</td>
<td>Lead fracture/disconnection Pacemaker inhibition by oversensing of intracardiac or extracardiac signals</td>
<td></td>
</tr>
<tr>
<td>intermittently or permanently absent on the ECG)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Failure to capture</td>
<td>Lead insulation tear</td>
<td>Temporary transvenous or external pacing if patient severely bradycardic and compromised Lead repositioning or replacement Correction of potentially reversible causes of elevated pacing threshold</td>
</tr>
<tr>
<td>(Pacing artefacts not resulting</td>
<td>Lead dislodgement Pacemaker inhibition by oversensing of intracardiac or extracardiac signals</td>
<td></td>
</tr>
<tr>
<td>in depolarisation, and dissociated from QRS complexes or P waves on the ECG)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Inappropriate high pacing rate</td>
<td>Tracking of atrial flutter/fibrillation Pacemaker-mediated tachycardia Runaway pacemaker</td>
<td>Magnet over pacemaker battery for acute termination of rapid pacing rates Adenosine or carotid sinus massage when magnet not available Reprogramming of pacemaker as definitive solution</td>
</tr>
</tbody>
</table>

#### Pacemaker malfunction resulting in tachycardia

<table>
<thead>
<tr>
<th>Problem</th>
<th>Cause</th>
<th>Management</th>
</tr>
</thead>
<tbody>
<tr>
<td>Undersensing</td>
<td>Lead dislodgement</td>
<td>Lead replacement, repositioning Increase sensitivity setting</td>
</tr>
<tr>
<td>(Pacing artefacts resulting in depolarisation, and dissociated from QRS complexes or P waves on the ECG). Usually not associated with tachycardia, but non-synchronised pacing may trigger VT</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Inappropriate high pacing rate</td>
<td>Tracking of atrial flutter/fibrillation Pacemaker-mediated tachycardia Runaway pacemaker</td>
<td></td>
</tr>
</tbody>
</table>

### Cases for special consideration

Certain conditions in the ICU merit special consideration when considering bradyarrhythmias.
Infective endocarditis

**NOTE** Development of new AV block/BBB in a patient with infective endocarditis implies an aortic root abscess (usually the non-coronary cusp). Patients with aortic valve endocarditis are regularly assessed for conduction abnormalities e.g. by ECG monitoring/daily 12-lead ECGs performed specifically to look for conduction abnormalities.

![Warning symbol] This complication carries significant risk of abrupt development of high-grade block. Immediate temporary pacing is indicated, usually by temporary pacing wire insertion, together with discussion with a cardiologist and cardiac surgeon.

Lyme disease

The commonest manifestation of the myocarditis in this condition is AV block. This frequently resolves with antibiotic treatment, but may require temporary pacing wire insertion.

Bradycardias in the ICU are commonly found also in patients with sick sinus syndrome, but these require specific treatment only occasionally.

Bradycardias in the post cardiac surgery patients (particularly after valve surgery) require temporary pacing. Wires are usually placed epicardially during the operation.
This section will deal exclusively with tachyarrhythmias arising above the AV node. By definition, the heart rate is >100 bpm for all causes, although in the critically ill, rates around and above 100 bpm are common, and may be entirely appropriate.

**MANAGING SUPRAVENTRICULAR TACHYCARDIA**

All supraventricular tachycardias may be caused and/or exacerbated by proarrrhythmic agents e.g. inotropic drugs - especially in patients with inadequate preload. If possible, concomitant with treating the arrhythmia, proarrrhythmic drugs should be reduced.

Various clinical skills may be useful in the diagnosis of supraventricular tachycardias, in addition to interpretation of the ECG:

Carotid sinus massage (CSM) may increase AV block, and help in distinguishing some tachycardias. It is common clinical practice to only perform CSM if both carotid pulses are present and of equal strength and there are no bruits. Perform gently to one side only but consider the risks in the older patient or those with a history of transient ischaemic attacks or other manifestations of cerebrovascular disease.

Intravenous adenosine increases AV block and may help in diagnosis.

Examination of the CVP line tracing (simultaneously with ECG) may be helpful in revealing the absence of an a-wave (for instance in AF), or the presence of cannon waves (in the case of AV dissociation).

If the patient has temporary pacing wires inserted (either epicardially at time of surgery, or transvenously as endocardial wires), simultaneous recordings can be made from these to aid in diagnosis. For instance, the absence of P waves can confirm atrial flutter or fibrillation in difficult cases. Retrograde P waves - occurring after the onset of each ventricular depolarisation - can also be identified (via the atrial ECG recording) indicating AV nodal or AV junctional re-entrant tachycardia.

**Sinus tachycardia**

The ECG will show normal P waves, normal QRS complexes (in the absence of BBB) with a constant PR interval between each beat.

**Causes**

Consider common factors that are associated with an increase in sympathetic activity, or catecholamine drive:

- β-agonist activity (such as inotropes, salbutamol, aminophylline)
  - Pain and anxiety
  - Hypovolaemia, left ventricular dysfunction, or any cause of reduced cardiac output
- Sepsis or other cause of vasodilation, causing a reflex chronotropic response
  - Anaemia
  - Fever
  - Thyrotoxicosis

**Sinus tachycardia in the ICU**

Patient can be the result of patient condition, physician intervention or both.
Management

Correct the underlying cause. Rarely, if ever, are small doses of β blocker indicated. Ensure that the diagnosis is not paroxysmal atrial tachycardia or atrial flutter with 2:1 or 3:1 block (see below).

NOTE In a patient with severe left ventricular diastolic dysfunction and poor cardiac output, a tachycardia (even if only moderate) may significantly impair ventricular filling. Consider, in this situation, cautious use of a short-acting β-blocking agent in a tightly monitored situation.

Q. What form of monitoring/investigation might be useful diagnostically in the analysis of an ICU patient with sinus tachycardia?

A. Transthoracic or transoesophageal echocardiography (TTE or TOE) can be a useful for basic haemodynamic assessment and to establish aetiology of cardiac disease.

See PACT module on Haemodynamic monitoring and management.

Paroxysmal atrial tachycardia (PAT)

NOTE The terminology used here is different from that commonly used in the US, in which the term paroxysmal SVT is preferred to PAT.

Paroxysmal SVTs are divided into those arising from an automatic focus and those resulting from re-entry. Of these, 8–10% result from increased automaticity, about 60% from AV nodal re-entry, and 30% from AV re-entry involving an accessory pathway, often concealed. Junctional tachycardia refers to accelerated junctional activity, and is uncommon except with digoxin toxicity.

Atrial tachycardia occurs when a site of atrial automaticity is discharging (an ‘ectopic atrial focus’). Sudden abrupt rises in heart rate are seen, and P wave morphology or axis may change, but tachycardia may also be incessant. If the P wave morphology is constantly changing, the tachycardia is known as multifocal atrial tachycardia (MAT).
Paroxysmal supraventricular tachycardia

Causes

May derive from a number of general proarrhythmic factors in ICU patients, or underlying structural heart disease.

Management

Adenosine has been known to cardiovert some patients. In others, it will unmask focal atrial tachycardia or atrial flutter. If tolerated, intravenous β-blockers are effective. Note, however, that since chronic obstructive pulmonary disease is a common cause of MAT, β-blockers may not be the best choice. In such patients, verapamil or diltiazem may be an alternative, with necessary precautions. In all cases, stop digoxin and treat toxicity if necessary. Cardioversion should be considered in patients with resistant and haemodynamically important PAT.

Atrial flutter

Consider in any patient where the heart rate is a division of 300, and constant. Diagnosis is made on the characteristic ‘saw-tooth’ (not always) flutter waves on the 12-lead ECG. Not every impulse will be conducted. If every second impulse is conducted, then a heart rate of 150 bpm will be seen. If every third is conducted,
then a heart rate of 100 results. Blockade may be variable, thus mimicking atrial fibrillation.

**Causes**

In addition to the causes described above, specific additional causes to remember include under/overfilling, and pulmonary embolism. Atrial flutter may be resistant to chemical cardioversion.

**Management**

Digoxin is sometimes helpful in converting atrial flutter to atrial fibrillation, which is easier to manage. Note, however, that the primary rationale for using digoxin is to increase AV blockade. Overdrive atrial pacing may be used to cause cardioversion, if an atrial wire is in use. Otherwise, management is similar to that of atrial fibrillation.

Atrial flutter is usually easily converted by synchronised electrical cardioversion, and this option warrants consideration as it is generally regarded as preferable to chemical cardioversion. Atrial flutter carries a risk of embolisation – anticoagulation may be advisable before and after cardioversion (same guidelines as AF).

**Design a clinical flow chart for the treatment of patients with persistent atrial flutter.**

**Atrial fibrillation**

**Diagnosis**

If the atrial activity is chaotic, then atrial fibrillation is said to be present. The AVN is bombarded with erratic electrical activity, and impulses are intermittently conducted. An ‘irregularly irregular’ heart rhythm results: you cannot tell when a beat is coming next.

Q. Give two reasons why atrial fibrillation may cause haemodynamic deterioration?

A. Because of loss of the benefit of atrial systole (to stroke volume) and the decrease in diastolic filling time caused by a rapid heart rate.

**Causes**

Specific causes to remember include under/overfilling, and pulmonary embolism. Fever and sepsis are common causes for atrial fibrillation in the ICU population.
Hyperthyroidism/thyrotoxicosis should always be excluded.

**Treatment**

Therapeutic objectives in patients with atrial fibrillation, in order of importance, are:

- Heart rate control
- Conversion to sinus rhythm
- Prevention of embolic complications
- Treatment of underlying (precipitating) cause

**Electrical cardioversion**

DC cardioversion is safe and effective, and always the first option in haemodynamically unstable patients.

**NOTE** The natural history of AF in ICU patients, in a large proportion of cases, is spontaneous reversion to SR. Cardioversion is often unsuccessful unless the underlying cause is corrected.

You can find further information in the following references.


**Prevention of embolic complications in acute AF - Anticoagulation**

- Consider anticoagulation e.g. to achieve an international normalised ratio (INR) of 2.0-3.0. The risk of cardioversion-associated embolisation with AF of >48 hours duration is 5.5% without anticoagulation; <1% with 3 weeks of anticoagulation.
- Transoesophageal echocardiography (TOE) (to exclude thrombus in the atrial appendage and to allow immediate cardioversion in the absence of three weeks anticoagulation) may be useful.
- If cardioversion fails, amiodarone (300 mg/30 min) may increase the likelihood of success.

**Chemical cardioversion**

- Anti-arrhythmics for acute cardioversion should only be considered in haemodynamically stable patients.
- All anti-arrhythmic drugs have potential proarrhythmogenic effects, and many exhibit negative inotropic action.
- In the presence of significant heart disease, amiodarone is the anti-
arrhythmic with the most acceptable safety profile. Amiodarone (5 mg/kg slow ‘push’, followed by 75 mg/h for 6 hours) is used for heart rate control and may result in cardioversion to sinus rhythm. The use of amiodarone can be associated with early and late complications.

- In the perioperative state, magnesium-sulphate (34 mg/kg over 20 min, 0.1 mmol/kg) may be effective, but the data are controversial.
- Propafenone (2 mg/kg slow intravenous injection over 10 min) is also an option with relatively rapid time to cardioversion (30 min to 2 hours). However it shares similar precautions and contraindications as flecainide (see below).

Flecainide is contraindicated in patients with left ventricular dysfunction or ischaemic heart disease. Up to 10% of patients may develop acceleration of rate, or a proarrhythmic response.

Two newer drugs (Vernakalant and Ibutilide) have shown promise in non-ICU patient studies of the management of AF and may have possible future relevance to critical care research and practice.

Vernakalant (atrial selective potassium channel blocker) was found superior to amiodarone for cardioversion. It has been used as a 3mg/kg infusion over 10 min, followed by 2 mg/kg after 15 min, as necessary. It has a potential for rapid cardioversion of AF of <7 days duration and it was superior to amiodarone in achieving cardioversion within 90 minutes of administration. It is contraindicated in advanced heart failure and in hypotensive patients with SBP <100 mmHg.


Clinical trials (but note again, NOT in the ICU population) have demonstrated increased success rate of transthoracic electrical cardioversion for AF with the class III potassium channel blocker, Ibutilide, but note the increased risk of torsade de pointes.

Rate control

To achieve rate control in atrial fibrillation acutely, digoxin has the slowest onset of action and is not the drug of choice. Amiodarone is rapidly effective in the control of the ventricular response to AF in the ICU population. Intravenous β-blockers, verapamil (0.075 mg/kg as a slow push) and diltiazem (0.25 mg/kg in 2 minutes bolus and continuous infusion 5-15 mg/hour) provide rapid rate response, but are negatively inotropic. In the non-ICU population, digoxin together with atenolol has been shown to be effective in controlling ventricular response rate in AF.

If ventricular response is uncontrolled, causing significant haemodynamic compromise, and resistant to all conventional manoeuvres, discussion with an
electrophysiologist of the potential for catheter radiofrequency ablation of atrial fibrillation (and restitution of sinus rhythm) or AV nodal ablation and insertion of a permanent pacemaker, may be helpful.

Q. Are there situations when verapamil should not normally be used or used only with extreme caution?

A. Verapamil should, in principle, not be used with a β-blocker, or if an accessory pathway is known or suspected. Extreme caution is also necessary in patients with profound hypotension.


http://www.escardio.org/guidelines-surveys/esc-guidelines/GuidelinesDocuments/Guidelines_Focused_Update_Atrial_Fibrillation.pdf

Aside from considerations of thromboprophylaxis, rhythm control in the haemodynamic treatment of AF in non-ICU patients has no advantage over rate control, but there are no data in ICU patients. The treatment objective depends primarily on haemodynamic impact of the atrial fibrillation.


Atrial fibrillation after cardiac and thoracic surgery

Postoperative AF is a significant problem in the ICU patient, and many trials have attempted to address this issue.

Currently, the use of prophylactic drugs at the time of cardiac surgery is not routine. Some of the evidence is summarised below.

- Amiodarone (pre-operatively, 600 mg by mouth for 1 week prior to cardiac surgery, and continued at 200 mg by mouth until discharge) reduces the risk of AF.
- Amiodarone (intravenous immediately post-operatively and continued for 48 hours) also reduces the risk of AF.
- Ibutilide successfully cardioverts patients with AF following cardiac surgery.

Treatment of postoperative AF is similar to treatment of acute onset atrial fibrillation in other situations (see above). For data regarding the use of β-blockers and sotalol in this context read:


Rhythm control did not show beneficial effect over rate control in CTS patients.


For data on contemporary prevention and treatment of post cardiac or thoracic surgery associated AF and other arrhythmias read:


Junctional tachycardias

AV nodal re-entrant tachycardia

These are usually based upon re-entry, two separate pathways within the AV node having two different refractory periods and different conduction velocities. These
two pathways are connected proximally (close to the atrium) and distally (close to the His bundle).

**Diagnosis**

Fast regular rhythm (classically rates of >150 bpm), paroxysmal, narrow QRS (less than 0.12 sec). There will be no P waves preceding the QRS complex: most often P waves are hidden within the QRS complex (common form), although (retrogradely-conducted) negative P waves may sometimes be seen following the QRS complex in leads (II, III, aVF) with a RP interval that is equal to or longer than the PR interval (rare form).

**Treatment**

Carotid sinus massage or adenosine may both slow the rhythm, or cardiovert it. There are three possible outcomes following adenosine injection:

1. Typically, tachycardia will stop with adenosine.
2. Temporary slowing of SVT following adenosine injection without conversion usually indicates sinus tachycardia or atrial tachycardia.
3. Temporary AV block (2 to 3 seconds) unmasking underlying atrial flutter or rapid atrial tachycardia.

If the PSVT recurs, then verapamil and diltiazem are effective at terminating the rhythm and preventing recurrence. Flecainide, β-blockers, and propafenone are also effective.

**Circus movement tachycardia (CMT) Wolff-Parkinson-White (WPW) syndrome**

These are synonyms for tachycardia based upon the existence of an accessory AV connection (Accessory Pathway, AP) between the atria and the ventricles. These pathways not only lead to earlier activation of the ventricle, following a supraventricular impulse, than during normal AV node conduction (so-called pre-excitation), but they also create the substrate for the re-entry circuit (CMT).

**Diagnosis**

Only patients with anterograde conduction have a delta wave on the electrocardiogram. This ECG manifestation of pre-excitation is seen in approximately 3/1000 ECGs. (See ECG below)

CMT may result in narrow or wide QRS tachycardia.

Antidromic CMT: broad QRS tachycardia occurring in patients with evident pre-excitation (delta waves) on the ECG during sinus rhythm.

Orthodromic CMT: most often narrow QRS tachycardia unless pre-existing bundle branch block, paroxysmal, regular rhythm, P waves are always separate from QRS: usually RP<PR (fast conducting AP), RP>PR (slow conducting AP). May not be differentiated from AV nodal tachycardia from the surface ECG.
An ECG of the patient with pre-excitation. Delta waves are evident in all leads. These patients will typically present with a broad QRS complex tachycardia.

Treatment

Acute treatment depends on the type of tachycardia. Orthodromic tachycardia is treated the same way as AV nodal reciprocating tachycardia.

Antidromic tachycardia (wide QRS tachycardia) may be terminated with adenosine, although there is possibility of induction of atrial fibrillation with consequent very rapid ventricular rate.

Drugs that slow the conduction through the accessory pathway (ibutilide, propafenone, flecainide) are therefore preferable for acute termination of wide QRS, pre-excited tachycardia.

Cardioversion is safe and effective and should be considered as a first choice in unstable patients.

⚠️ Digoxin is not given in the WPW syndrome because it may shorten the refractory period of the AP (accessory pathway) as well as the atrium. Such a caution is necessary for all drugs that slow the conduction through the AV node (verapamil, beta blockers) because they may accelerate the ventricular rate during atrial fibrillation in a WPW syndrome.

http://www.escardio.org/guidelines-surveys/esc-guidelines/GuidelinesDocuments/guidelines-SVA-FT.pdf

As with all tachycardias, the ECG diagnosis and subsequent action taken need to take into account the clinical context. Any underlying or exacerbating factors require attention and successful treatment of the tachycardia is more likely if an absence of high-dose inotropic therapy can be achieved.

**Ventricular extrasystoles**

In the context of the ICU, these should alert the physician to the possibility of cardiac disease or irritability (mechanical or chemical) of the heart. Appropriate management includes:

- Rigorous attention to correcting electrolyte imbalance
- Consider repositioning of any intracardiac catheters
- In patients who have undergone cardiac surgery, or with underlying ischaemic heart disease, potassium and magnesium should be supplemented
- Beta blockers should be used in coronary artery disease

**Ventricular tachycardia**

Always consider the possibility of ventricular tachycardia (VT) in a wide complex rhythm (even if the heart rate is below 100 bpm, especially if the patient is being, or has been, treated with anti-arrhythmic drugs) and also in the context of known or suspected ischaemic heart disease.

A wide complex tachycardia may be due to:

- Ventricular tachycardia
- Supraventricular tachycardia with aberrant conduction
- Pre-excited AV reciprocating tachycardia in a patient with WPW syndrome

---

**Do not panic when confronted with a broad QRS tachycardia.** Try to obtain a 12-lead ECG. If the patient is haemodynamically compromised with a broad complex tachycardia, it is safer to assume VT, and treat with cardioversion.

Find out whether there is agreement in your unit concerning the above approach to broad QRS tachycardia. If not, what alternative approaches are suggested?

The likelihood of VT (vs SVT with aberrant conduction) increases if:

- Heart rate $>$170 bpm
- QRS duration $>$0.14 seconds (see diagnosis below)
The likelihood of SVT with aberrant conduction (vs VT) increases if the morphology of the QRS complex on the 12-lead ECG is identical to that seen prior to the onset of tachycardia.

NOTE The history of MI (myocardial infarction) prevails over ECG criteria in the differential diagnosis (of VT versus SVT, with aberrant conduction) and strongly suggests ventricular tachycardia.

Drew BJ, Scheinman MM. ECG criteria to distinguish between aberrantly conducted supraventricular tachycardia and ventricular tachycardia: practical aspects for the immediate care setting. Pacing Clin Electrophysiol 1995; 18 (12 pt 1): 2194-2208. PMID 8771133

**ECG diagnosis**

<table>
<thead>
<tr>
<th>AV dissociation?</th>
<th>Present</th>
<th>→ favours VT</th>
</tr>
</thead>
<tbody>
<tr>
<td>QRS width?</td>
<td>&gt;140 msec</td>
<td>→ VT</td>
</tr>
<tr>
<td>Left axis deviation</td>
<td>Present</td>
<td>→ favours VT</td>
</tr>
</tbody>
</table>

**Analysis of QRS configuration**

Variation of RR interval is typically seen in SVTs, but minor variation (up to 20 ms) may also occur in VT

<table>
<thead>
<tr>
<th>RBBB shaped</th>
<th>V1</th>
<th>mono- or biphasic QRS suggests VT</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>V6</td>
<td>R/S &lt;1 suggests VT (the S wave is larger than the R wave in the V6 lead)</td>
</tr>
<tr>
<td>LBBB shaped</td>
<td>V1</td>
<td>( R_{\text{tachy}} &lt; R_{\text{SR}} ) suggests SVT</td>
</tr>
<tr>
<td></td>
<td>V1,2</td>
<td>( R_{\text{tachy}} &gt; R_{\text{SR}} ) suggests VT</td>
</tr>
<tr>
<td></td>
<td>V1,2</td>
<td>notching downslope S wave suggests VT</td>
</tr>
<tr>
<td></td>
<td>V1,2</td>
<td>beginning QRS to upslope S wave &gt;0.70 sec suggests VT</td>
</tr>
<tr>
<td></td>
<td>V6</td>
<td>QR suggests VT</td>
</tr>
</tbody>
</table>

\( R_{\text{tachy}} \): amplitude of R wave during tachycardia; \( R_{\text{SR}} \): amplitude of R wave during sinus rhythm; QR: a QR pattern characterised by deep Q wave followed by R wave.

A negative praecordial pattern (concordance in all praecordial leads) is always a VT.

**Management**

**Non-sustained VT**

Asymptomatic, normal left ventricular function - low risk of sudden death or serious ventricular arrhythmias. Treat as for ventricular extrasystoles.
Ischaemic heart disease with left ventricular ejection fraction <40% - high risk of sudden death or serious ventricular arrhythmias. Address all treatable exacerbating factors, seek cardiological opinion regarding catheterisation, possible intervention (angioplasty or surgical referral), choice of anti-arrhythmic agent and consideration for implantable cardioverter defibrillator (ICD).

Recurrent non-sustained VT causing haemodynamic compromise. Address all treatable exacerbating factors, consider amiodarone infusion or ventricular pacing (especially if VT emerges during a period of relative bradycardia).

American Heart Association; American College of Cardiology; North American Society for Pacing and Electrophysiology 2002 Guideline Update for Implantation of Cardiac Pacemakers and Antiarrhythmia Devices:
http://circ.ahajournals.org/cgi/content/full/106/16/2145

Zipes DP, Camm AJ, Borggrefe M, Buxton AE, Chaitman B, Fromer M, et al; American College of Cardiology; American Heart Association Task Force; European Society of Cardiology Committee for Practice Guidelines; European Heart Rhythm Association; Heart Rhythm Society. ACC/AHA/ESC 2006 guidelines for management of patients with ventricular arrhythmias and the prevention of sudden cardiac death: a report of the American College of Cardiology/American Heart Association Task Force and the European Society of Cardiology Committee for Practice Guidelines (Writing Committee to Develop guidelines for management of patients with ventricular arrhythmias and the prevention of sudden cardiac death) developed in collaboration with the European Heart Rhythm Association and the Heart Rhythm Society. Europace 2006; 8(9): 746-837. PMID 16935866


Sustained VT

- Synchronised DC cardioversion is indicated whenever VT is associated with haemodynamic compromise.
- In the uncompromised and awake patient, consider intravenous amiodarone (150 mg iv over 10 minutes) as first line drug (effective in 24-40% only).
- It is worthwhile to try a beta blocker challenge whenever underlying ischaemia is suspected.
- If amiodarone fails, proceed rapidly to DC cardioversion.
- In sustained polymorphic VT 24-48 hours post-infarction, and in the context of myocardial ischaemia, intravenous beta blockers or amiodarone are the treatments of choice.
- In recurrent polymorphic VT, always make sure to exclude QT interval prolongation. If present, consider appropriate treatment (see Torsade de Pointes section below).
Refractory ventricular tachycardia and fibrillation

Refractory monomorphic VT and VF typically occur in two settings. First, in the context of myocardial ischaemia, myocardial infarction and following coronary artery bypass grafting. Second, they may occur in patients with ischaemic or dilated cardiomyopathy without ongoing ischaemia. In both cases, arrhythmias may be refractory to intravenous amiodarone, therefore consider:

- Overdrive or underdrive pacing
- Intra-aortic balloon counterpulsion
- Coronary revascularisation
- Intubation and mechanical ventilation
- Cardiac assist device

NOTE: In cases of intractable recurrent VT/VF, consult an electrophysiologist. Sometimes, radiofrequency catheter ablation can abolish or eliminate the arrhythmia.

In the presence of significant left ventricular dysfunction, this group of patients have a very poor outlook, and no intervention may be appropriate.

Any patient who develops VT or VF >48h after myocardial infarction in the absence of ongoing myocardial ischaemia is at high risk for malignant arrhythmias and should be referred for cardiological assessment.

Consult with your cardiological colleagues as to what they would consider in the event of such a referral.

Drugs given for the treatment of supraventricular tachycardia may be dangerous in a patient with ventricular tachycardia. When in doubt about the origin of the tachycardia, do not use verapamil; use amiodarone instead. Severe haemodynamic deterioration is a complication of IV verapamil if administered during ventricular tachycardia.


Newer interventions for the management of VT/VF

Implantable cardioverter defibrillator (ICD)

Recently, a new type of device-based treatment for heart failure has emerged - resynchronisation pacing. In many cases such modality is combined with ICD function.
in one device, a CRT-D (cardiac resynchronisation therapy with defibrillator function).

From a standpoint of an intensivist, these two types of devices share the same basic characteristics:

ICD/CRT-Ds:

- Implantable subcutaneously (pre-pectoral), with transvenous leads
- Able to diagnose ventricular tachycardia and ventricular fibrillation
- Able to deliver antitachycardia pacing and/or defibrillation
- Can be interrogated to determine number and length of arrhythmic episodes
- May be deactivated by placing a magnet directly over the generator site
- The presence of the device does not preclude an operator delivering standard cardioversion/defibrillation transcutaneously (take care not to place paddles over the device)

Patients with improved survival with ICDs include (most common indications):

- Patients following myocardial infarction and LV ejection fraction <35%
- Patients with dilated cardiomyopathy and LV ejection fraction <35%
- Patients with unexplained syncope and VT/VF induced during electrophysiological testing
- Survivors of arrests attributed to sustained VT with syncope, or VF without identifiable reversible cause
- Structural heart disease and spontaneous sustained VT

Patients with conventional indications for an ICD and concomitant heart failure are candidates for a CRT-D device when NYHA function class III/IV, LVEF ≤35%, QRS ≥120 ms.

**NOTE** Above recommendations may not be relevant for acute management of VT/VF, but familiarity with them will be useful to the intensivist in making appropriate Cardiology referral when the acute phase of illness is resolved or following ICU discharge.

As increasing numbers of patients are fitted with these devices, and those with ICDs are likely to come under the care of critical care physicians at some stage during the course of their illness, it is important that critical care physicians have some knowledge of their potential, functions and problems.

Most frequent ICD/CRT-D related problems that an intensivist might confront include:

<table>
<thead>
<tr>
<th>Problem</th>
<th>Possible cause</th>
<th>Solution</th>
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<tbody>
<tr>
<td>Procedures causing electromagnetic interference (electrocautery during surgery, etc)</td>
<td></td>
<td>Temporary reprogramming of ICD/CRT-D - Disabling ICD discharge - Deactivate rate responsive pacing</td>
</tr>
<tr>
<td>No ICD intervention during VT</td>
<td>ICD battery/lead failure VT rate below preset threshold Completed cycle, exhaustion</td>
<td>Acutely - Terminate VT with external DC cardioversion, and/or anti-</td>
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<td>Suspected device infection</td>
<td>Early post-implant</td>
<td>Acute</td>
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<tr>
<td></td>
<td>- Procedure related</td>
<td>- Treat infection and sepsis</td>
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<td>Late (&gt;60 days post-implant)</td>
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<td>- from bacteraemia, pouch skin erosion, or delayed onset of peri-procedurally acquired infection</td>
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<td></td>
<td></td>
<td>Definitive</td>
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<td></td>
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<td>- Complete ICD system replacement</td>
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<tr>
<th>Repetitive ICD shocks</th>
<th>Appropriate</th>
<th>Acutely</th>
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<tr>
<td></td>
<td>- Frequently recurring VT/VF</td>
<td>- Intensify anti-arrhythmic therapy, treat potential triggers</td>
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<td></td>
<td></td>
<td>- Temporary device deactivation if VT not haemodynamically compromising</td>
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<tr>
<td></td>
<td>- Shocks do not terminate VT reliably</td>
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<td></td>
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<td>Definitive</td>
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<tr>
<td></td>
<td>- Missensing of supraventricular arrhythmias</td>
<td>- Oral anti-arrhythmic drug optimisation</td>
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<td></td>
<td>- Antitachycardia pacing</td>
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<td></td>
<td>- Catheter ablation of VT</td>
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<tr>
<td>Inappropriate</td>
<td>- Oversensing of intracardiac potentials (T wave) or myopotentials</td>
<td>Acutely</td>
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<td>- Termination of SVT/AF by external cardioversion or anti-arrhythmics</td>
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<td>- Temporary ICD deactivation if SVT/AF frequently recurs</td>
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<td></td>
<td>Definitive</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Appropriate ICD reprogramming, upgrade to dual chamber device</td>
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<td></td>
<td></td>
<td>- Modification of anti-arrhythmic regime</td>
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<td>- Catheter ablation of SVT/AF</td>
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<td>- Reprogramming</td>
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<tr>
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<td>- ICD interrogation</td>
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<td></td>
<td>- Battery/lead replacement</td>
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<td>- Reprogramming</td>
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| Definitive                          | - Oral anti-arrhythmic drug optimisation |
| Definitive                          | - Antitachycardia pacing |
| Definitive                          | - Catheter ablation of VT |

| Acutely                             | - Termination of SVT/AF by external cardioversion or anti-arrhythmics |
| Acutely                             | - Temporary ICD deactivation if SVT/AF frequently recurs |

| Acutely                             | - Temporary device deactivation |
| Acutely                             | - Reprogramming                |

| Definitive                          | - Appropriate ICD reprogramming, upgrade to dual chamber device |
| Definitive                          | - Modification of anti-arrhythmic regime |
| Definitive                          | - Catheter ablation of SVT/AF |

| Definitive                          | - Temporary device deactivation |
| Definitive                          | - Reprogramming                |

| Definitive                          | - Oral anti-arrhythmic drug optimisation |
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| Definitive                          | - Temporary device deactivation |
| Definitive                          | - Reprogramming                |
A question of need for an ICD/CRT device to be turned off following the death of a patient sometimes arises in ICU. Typically, pacing function is not turned off after the death. Nowadays, ICDs are programmed such as to give maximum of five consecutive defibrillation shocks, and then this function is aborted. This obviates the need for turning off an ICD following the death, as well as preventing the deceased patient from receiving continuous shocks if VF recurs after resuscitation interventions have been aborted.


http://content.onlinejacc.org/article.aspx?articleid=1486116


http://www.escardio.org/guidelines-surveys/esc-guidelinesDocuments/guidelines-CRT-HF.pdf


Catheter ablation for VT

Catheter ablation of VT is curative and first line treatment in some forms of VT associated with a structurally normal heart. The following provides an insight into the considerations pertaining to its clinical application. Consider referral for cardiological opinion in:

Right ventricular outflow tract (RVOT) VT

Consider in a young patient with RVOT VT (LBBB, right axis deviation) that may terminate with adenosine, in the context of a structurally normal heart.

Idiopathic left ventricular tachycardia

Consider in a structurally normal heart when VT occurs with RBBB and left axis morphology terminates with verapamil - as a specific diagnostic test usually in the electrophysiology laboratory situation.
Bundle branch re-entrant VT

Consider in a patient with LBBB, syncope and dilated cardiomyopathy.

In addition, catheter ablation may provide a treatment option in some forms of intractable recurrent VT in context of ischaemic or dilative cardiomyopathy.

Electrophysiologist consultation is warranted in:

- Patients with very frequent (>3/24h) VT/VF following acute myocardial infarction - electrical storm
- Patients with ischaemic or dilated cardiomyopathy and recurrent sustained monomorphic VT


Torsade de pointes

This ECG is a polymorphic ventricular tachycardia with a sinusoidal electrocardiographic appearance due to the QRS complex undulating around the baseline. The arrhythmia arises from prolonged myocardial repolarisation (seen on the surface ECG as a prolonged QTc), which may be congenital or acquired. The tachycardia is paroxysmal and may result in VF and sudden death.

Causes

- Electrolyte abnormalities especially hypomagnesaemia
- Anti-arrhythmic agents
- Hereditary long QT syndrome
- Bradyarrhythmias
- Myocardial ischaemia
- Neurological events (ischaemic stroke, intracerebral haemorrhage)
- Neuroleptics
- Certain antibiotics
- Toxins
Q. Give examples of relevant antibiotics and neuroleptics that cause torsade de pointes?

A. Antibiotics (e.g. macrolides), neuroleptics (e.g. haloperidol).

Management

Make the diagnosis and correct all exacerbating or causative factors. Consider temporary pacing and intravenous magnesium. ICD placement (although rarely necessary for torsades except in patients with hereditary long QT) may be considered.

CONCLUSION

We have now covered the diagnosis, assessment and management of the major arrhythmias occurring in the intensive care unit. A systematic and informed approach to the 12-lead electrocardiogram contributes to better outcome for many of our patients. The correct diagnosis of the underlying cause is the first step to optimal treatment. From time to time the development of arrhythmias may lead to the need for implementation of resuscitation protocols following cardiac arrest. The following guidelines are valuable resources for necessary emergency skills.

**PATIENT CHALLENGES**

A 32-year-old male patient is transferred from the Emergency Department in your hospital to the Cardiac Intensive Care Unit suffering from wide complex tachycardia and dyspnoea.

The patient, a well-trained sportsman, was well until one month earlier when he presented to his GP with progressive exertional dyspnoea and fatigue, accompanied by a feeling of heaviness in the limbs and the right upper quadrant. There was no history of chest pain. On the day of admission he awoke with a ‘racing’ heart, profuse sweating, nausea, vomiting and dyspnoea at rest.

On admission the patient’s core temperature is 36.8 °C, pulse rate estimated at >150 bpm (pulse deficit detected), respiratory rate 26 breaths per minute and arterial blood pressure 106/82 mmHg. Arterial oxygen saturation is 100 percent with supplementary oxygen by mask.

On examination the patient is pale and sweaty. The jugular venous pressure is 14 cm H₂O. Rales are heard at both lung bases. The heart sounds appear normal but rapid.

A wide complex tachycardia with a ventricular rate of 160 bpm is evident on the 5-lead ECG monitoring record of the Emergency Department.

**Learning Issues**

Diagnosis of rhythm disturbances


Q. In this patient, suffering a regular broad (QRS) complex tachycardia, what immediate standard diagnostic test should be done?

A. First, a 12-lead ECG is obtained.

Having performed a 12-lead ECG, you note a regular wide (broad) complex tachycardia with a left bundle-branch-configuration at a rate of 162 bpm (figure shown below).
Q. Viewing the ECG (presuming the patient remains acceptably stable), what is your next diagnostic priority?

A. To differentiate between ventricular tachycardia and supraventricular tachycardia with aberrant conduction (bundle branch block).

The emergency response to the diagnosis of a broad QRS tachycardia demands a calm, informed and systematic approach. Certain morphological findings on this standard ECG and on the Lead 2 rhythm strip above e.g. the ‘fusion beats’ on the rhythm strip can quickly and accurately pinpoint ventricular tachycardia.

**Learning Issues**

‘Fusion beats’ as evidence of A-V dissociation (see ‘criteria’ below)
Criteria for the ECG diagnosis of VT (Ventricular Tachcardia - diagnosis -Task 6)

Because of the compromised haemodynamic status of this patient on admission to the Emergency Department, you opt, in discussion with the attending cardiologist, to resort to immediate cardioversion under controlled sedation. Measurement of arterial blood gases and serum electrolytes was first performed by the bedside and electrolyte disturbance was excluded. The patient was transferred (after successful cardioversion) to the Cardiac ICU/CCU in sinus rhythm for ongoing management.

Q. A trainee colleague from the department of Emergency Medicine follows with interest these activities and asks you how to proceed when confronted with a patient suffering from a broad (QRS) complex tachycardia in an emergency department situation.

A. Your advice is that if the patient is haemodynamically unstable, cardiovert.
A simple rule in the treatment of arrhythmias: cardiovert if haemodynamically unstable.

Q. The colleague then asks how to approach the stable patient with a broad complex tachycardia.
A. If stable, determine the type of tachycardia by obtaining a history to establish the absence or presence of heart disease, by doing a physical examination and by systematic evaluation of the 12-lead ECG. Careful clinical assessment of the patient in this manner will help you differentiate between a supraventricular and a ventricular tachycardia.

Q. If ventricular tachycardia is the diagnosis, what drug therapy is suggested?
A. Amiodarone is the anti-arrhythmic drug of choice. If medical therapy is unsuccessful, cardiovert.

Q. If there is certainty regarding the diagnosis of supraventricular tachycardia, what agent might be utilised?
A. Intravenous verapamil is only considered in the treatment of broad QRS complex tachycardia when the diagnosis of supraventricular tachycardia is 100% certain.

Importance of careful history when confronted with a broad (wide) QRS tachycardia
Management of SVT
Management of VT

Q. The presence of regular independent P waves cannot clearly be demonstrated on the 12-lead ECG recording in your patient. Indeed, P wave identification is not always possible on the standard ECG recordings. How can we ‘unmask’ P waves ‘hidden’ after or within the QRS complex?
A. In this situation, intravenous adenosine can be useful to differentiate ventricular from supraventricular tachycardia with aberrant ventricular conduction.

Presence/absence of P waves
Diagnosis of supraventricular tachycardia

At the time of admission to the Cardiac ICU/CCU, an echocardiogram on your patient showed a dilated, diffuse hypo- to akinetic left ventricle (ejection fraction 20-25%) with a moderate to severe mitral valve insufficiency (see figure below). Further investigation could not elucidate the cause of his cardiomyopathy and a coronary angiogram showed absence of marked coronary artery disease.
The patient suffered recurrent episodes of sustained monomorphic VT despite continuing anti-arrhythmic drug therapy (amiodarone). An implantable cardioverter defibrillator (ICD) was placed one month later and ongoing treatment with ACE-inhibitors, anticoagulants, loop-diuretics and a beta blocker was initiated/maintained.

The patient remained stable until six months later when he was readmitted after he experienced multiple shocks from the ICD at which time he was suffering from concomitant acute laryngotracheobronchitis with a core temperature <40°C.

**Learning Issues**

Refresh your Echo interpretative skills - see the PACT module on Haemodynamic monitoring and management

Q. What is the immediate course of action when confronted with electrical instability in your patient with an implantable cardioverter defibrillator?

A. The sudden occurrence of multiple shocks requires immediate evaluation and hospitalisation of your patient.
Q. What is the differential diagnosis of the multiple ICD-induced shocks?
A. The differential diagnosis includes: recurrent ventricular arrhythmias, supraventricular arrhythmias, or ICD/lead malfunction.

Revision of the ICD in the electrophysiology laboratory, however, did not in this instance, identify any system failure. Antitachycardia pacing function and VT detection criteria were considered appropriate.

**NOTE** There are important ICD interactions with anti-arrhythmic drugs that require recognition by physicians who care for these patients.

**Learning Issues**

**Implantable cardioverter defibrillator (ICD)**

You confirm that all shocks of the ICD were appropriate and all correctable abnormalities (electrolyte imbalance, fever etc.) have been addressed and further conclude that additional oral β-blocker therapy would not be appropriate because of the haemodynamically poor acute condition of the patient.

Q. Is there any place for changing or initiating (additional) anti-arrhythmic drugs in this hospitalised patient?
A. Amiodarone may be given by continuous intravenous infusion.

Q. Would additional intravenous β-blockade be appropriate in the acute circumstance?
A. The haemodynamically poor condition of your patient precludes intravenous β-blockade except in very specific, exceptional circumstances.

**Learning Issues**

**Refractory VT**


Despite this treatment, the ventricular electrical storm cannot be controlled. Your patient needs to be sedated and ventilated. To assist in the management of the evolving cardiogenic shock and the mitral regurgitation, an intra-aortic balloon pump is placed; also a pulmonary artery monitoring catheter. The patient is put on the waiting list for urgent heart transplantation.
On reflection, the clinical course of this patient was complicated and involved progressively more sophisticated treatment. The intensive care physician is involved particularly in the management of the acute, potentially life-threatening arrhythmias and in the management of the related cardiogenic shock. From the outset you will have appreciated that interpretation of electrocardiographic findings is done in light of your patient’s history and clinical condition. Knowing when and how to act quickly and when to seek specialist opinion is important. When conservative measures fail, life support measures may be required to sustain the patient until specialist interventions e.g. heart transplantation can be effected.