Cardiac arrhythmias in the critically ill

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Abstract

Arrhythmias are a common problem in the critically ill and they can have significant effects on patient outcome. They often require immediate and swift action and it is, therefore, essential that clinicians have a structured approach to the recognition and management of arrhythmias. Here, we provide a framework for the appropriate management of the more frequently encountered cardiac arrhythmias in critical care. We include the algorithms from the 2015 Resuscitation Council Guidelines for reference.

Keywords Arrhythmias; atrial fibrillation; bradycardia; congenital heart disease; ion channelopathies; prolonged QT supraventricular tachycardia; temporary cardiac pacing; therapeutic hypothermia; ventricular tachycardia

Royal College of Anaesthetists CPD Matrix: 3G00

Learning objectives

After reading this article, you should be able to:

- recognize the clinical features of cardiac arrhythmias in the critically ill patient that demand immediate and swift action
- understand the various causes and mechanisms of arrhythmias in the critically ill
- have a systematic approach in the assessment and management of arrhythmias in critically ill patients
- appreciate the changes in the updated 2015 European Resuscitation Council guidelines

Arrhythmias are common in critically ill patients (Table 1) and their clinical manifestation may range from patients being completely asymptomatic to cardiorespiratory arrest. The underlying causes are often multifactorial, hence the management and resuscitation of patients must be performed in a systematic and methodical manner. The aim of this article is to provide a framework for the appropriate management of the more frequently encountered cardiac arrhythmias in the critically ill.

Causes of arrhythmias (Table 2)

Arrhythmias can result from primary pathology in the cardiac conductive pathways or abnormalities in other organ systems. In the critically ill patient, arrhythmias can be caused or potentiated by increased catecholamine levels (endogenous or exogenous), hypoxia, hypercarbia, severe acidosis, gross electrolyte disturbance, and pain or anxiety. The end result is a combination of decreased cardiac output and/or increased myocardial oxygen demand. Treatment should be targeted at the underlying cause, often revealed through a thorough history, examination and relevant investigations. Figure 1 summarizes the various mechanism of arrhythmias in the critically ill.

Assessing the patient with cardiac arrhythmia

The evaluation and treatment of critically ill patients with arrhythmias has to include supportive, diagnostic, therapeutic and, possibly, resuscitative measures encompassing not just the cause of the arrhythmia but also systemic effects, including impaired end-organ perfusion and function. Establishing the presence of structural heart disease and/or the history of arrhythmias is essential prior to assessing the arrhythmia. The increasing incidence of adult congenital heart disease adds complexity to arrhythmia management and we emphasize the need for early involvement of specialist cardiologists in the management of these patients.

In this article we present a rational, step-wise sequence of questions to guide management.

Question 1: Is the patient compromised by the arrhythmia?

The answer to this question determines the speed of treatment: the greater the compromise, the swifter the response needs to be. Irrespective of the instantaneous degree of compromise, the ever present risk of further cardiovascular deterioration or the development of life-threatening arrhythmias mandates:

- enrichment of the oxygen supply in hypoxaemic patients
- rapid ABCDE assessment
- establishment of secure venous access
- application of monitors
  - peripheral oxygen saturation (SpO2)
  - ECG
  - non-invasive blood pressure (NIBP)
- application of stick-on defibrillation pads
  - right infraclavicular and left anterior axillary line over the 5th/6th intercostal spaces attached to a compatible defibrillator to also allow for external cardiac pacing if the patient is bradycardic. With a history of heart failure or if the heart is suspected to be dilated a more lateral position of the left defibrillator pad will allow a greater chance of successful defibrillation
- recording a 12-lead ECG as soon as the clinical situation allows.

And if the clinical condition warrants it:

- removal or withdrawal of precipitants

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Incidence and impact of arrhythmias in patients in the intensive care unit

	Sustained supraventricular arrhythmias %	Sustained ventricular arrhythmias %	Conduction abnormalities %	No arrhythmias %
Incidence	8	2	2	88
Unadjusted in-hospital death rate	29	73	60	17
Neurological sequelae among survivors	15	38	17	6

After adjustment for prognostic factors and propensity scores, only ventricular arrhythmias increased mortality (odds ratio 3.53, 95% confidence interval 1.19—10.42).

Table 1

Some causes of arrhythmias in the critically unwell

Arrhythmias arising from primary cardiac disease

Bradycardias

- Second-degree and complete heart block
- Following inferior MI
- Excessive vagal tone
- Chronotropic incompetence
- Congenital heart disease

Tachycardias

- Myopathies
  - Atrial fibrillation with rapid ventricular response
  - Cardiomyopathies
- Conducting system
  - Nodal re-entrant
  - AV nodal re-entrant
  - Channelopathies
- Pacemaker generated
- Congenital heart disease

Arrhythmias arising secondary to systemic disease process

- Hypothermia
- Hyperkalaemia
- Head injury and raised intracranial pressure
- Drugs, e.g. B-blockade, calcium channel antagonists
- Organophosphorus poisoning
- Excessive endogenous catecholamines (pain)
- Excessive exogenous catecholamines
- Pyrexia
- Sepsis
- Thyrotoxicosis
- Hypercarbia
- Hypoxia
- Hypovolaemia
- Tension pneumothorax
- Pulmonary embolism
- Drug or alcohol withdrawal
- Vagolytic drugs

Table 2

- measurement of 'plasma' K^+, Mg^{2+} and Ca^{2+}.

The relevant clinical signs of compromise are described in Box 1.

Question 2: Is this a primary arrhythmia or secondary to another disease process?

Is the cardiac rhythm a normal and appropriate response to alterations of physiology or is it pathological? The answer to this question might be swiftly obtained by means of the history, examination and appropriate investigations; for example, the profound bradycardia associated with hypothermia. At other times, determining the cause of the arrhythmia might require electrophysiological studies (EPS).

Question 3: Is the heart rate slow or fast?

The normal heart rate (HR) is between 60 and 100 beats per minute (bpm). A HR below 60 is termed a bradycardia, and above 100 a tachycardia; however, this needs to be taken in context.

Question 4: What is the relationship of the P wave to the QRS complex?

Although this is easier to answer if the HR is slow, all 12 leads need to be scrutinized as the P wave might be evident in some leads but not in others. The relationship might be regular, irregular or there might be no P waves visible.

Bradycardia

Table 3 describes common bradycardias and classifies them according to their relationship to the P wave. The appropriate treatment decision has to be made in the clinical setting. Asymptomatic second-degree heart block in a non-compromised patient, for example, is going to warrant invasive intervention but this may not be immediately required; however, in the compromised patient, atropine 500 mg (repeated up to 3 mg), isoprenaline or adrenaline as bolus or infusion will temporise the circulation before single- or dual-chamber pacing. The indications for pacing are shown in Box 2 and the ResuscitationCouncil's 2015 algorithm for bradycardias is shown in Figure 2.

Tachycardia

Tachycardias can be difficult to interpret because the P wave may be absorbed into the QRS complex. Furthermore, retrograde P waves could be present which represent retrograde conduction of a ventricular tachycardia (VT) via the atrioventricular (AV) node. Table 4 describes the possible relationships of the P wave to the QRS complex.

A broad complex tachycardia is either VT or a supraventricular tachycardia (SVT) with aberrant conduction (left bundle branch block or right bundle branch block). It can be complicated to distinguish between these. The majority of broad complex tachycardias will be VT, and in patients with known structural or ischaemic heart disease these are almost always VTs. Distinguishing between VT and aberrant SVT is important in terms of their subsequent management; however, if there is

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