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 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 2010 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

Learning objectives

After reading this article, you should be able to:

- recognize the clinical features of cardiac arrhythmias in the critically ill patient that demands immediate and swift action
- have a systematic approach in the assessment and management of arrhythmias in critically ill patients
- appreciate the changes in the updated 2010 European Resuscitation Council guidelines

Arrhythmias are common amongst patients who are critically ill (Table 1). Their clinical manifestation may range from patients being completely asymptomatic to cardiorespiratory arrest. The variety of arrhythmias is considerable and the underlying cause is often multifactorial, hence the management and resuscitation of patients must be performed in a systematic and methodical manner. This article aims to provide a framework for the appropriate management of the more frequently encountered cardiac arrhythmias in the critically ill and illustrate them with sample ECGs.

Causes of arrhythmias

Arrhythmias can be the result of 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 mechanisms of arrhythmias in the critically ill.

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Assessing the patient with cardiac arrhythmia

The management of critically ill patients with arrhythmias has to include supportive, diagnostic, therapeutic and, possibly, resuscitative measures not only for the cause of the arrhythmia but also systemic effects, including impaired end-organ perfusion and function.

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:

- rapid assessment of the circulation, including the presence of a pulse
- enrichment of the oxygen supply
- establishment of secure venous access
- application of monitors
  - peripheral oxygen saturation (SpO_2)
  - ECG
  - non-invasive blood pressure (NIBP)
- the application of stick-on defibrillation pads
  - right infraclavicular and left anterior axillary line over the fifth/sixth intercostal spaces attached to a compatible defibrillator might have the potential for external cardiac pacing if the patient is bradycardic.

And if the clinical condition warrants it:

- the recording of a 12-lead ECG
- the removal or withdrawal of precipitants
- measurement of 'plasma' K^+, Mg^{2+} and Ca^{2+}

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

Question 2: Is the heart rate slow or fast?

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

Features of a compromised circulation

- Absent pulse
- Signs of low cardiac output
- Hypotension (systolic blood pressure <90 mmHg)
- Heart rate <40 or >150
- Chest pain
- Ischaemia on the ECG
- Heart failure
- Reduced level of consciousness

Box 1

Question 3: Is this a primary arrhythmia or secondary to another disease process?

Are the cardiac arrhythmias normal and appropriate responses to alterations of physiology or are they 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 (ECG 1). At other times, determining the cause of the arrhythmia might require electrophysiological studies (EPS). Categorizations based on the answers resulting from Questions 2 and 3 are shown in Table 2.

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. Table 3 describes the diagnoses fitting into each category.

The appropriate treatment has to be made in the clinical setting. Asymptomatic second-degree heart block in a non-compromised patient, for example, is rarely going to warrant invasive intervention; however, in the compromised patient, atropine 500 mg (repeated up to 3 mg) or an epinephrine infusion will temporize the circulation before single- or dual-chamber pacing. The indications for pacing are shown in Box 2 and the Resuscitation Council's 2010 algorithm for bradycardias is shown in Figure 2.

Indications for temporary cardiac pacing

- Symptomatic bradycardia unresponsive to atropine
- Acute conduction disturbances following MI
  - Anterior MI associated with complete AV block, Mobitz type I or type II AV block, non-adjacent bifascicular or trifascicular block requires prophylactic pacing
- Pacing might be required in inferior MI with complete AV block
- Complete AV block with rate <50
- Asystole with P wave activity
- Suppression of drug-resistant tachyarrhythmias — 'over-drive pacing'
- During or after cardiac surgery (especially in aortic surgery, tricuspid surgery, ventricular septal defect closure and ostium primum repair)
- Preoperatively in patients with
  - Second-degree or complete AV block
  - Intermittent AV block
  - Trifascicular block
  - First-degree AV block and LBBB

AV, atrioventricular; LBBB, left bundle branch block; MI, myocardial infarction.

Box 2

Tachycardias can be difficult to interpret because the P wave can be absorbed into the QRS complex. Furthermore, retrograde P waves can be seen which might represent retrograde conduction of a ventricular tachycardia (VT) via the atrioventricular (AV) node and the apparent PR interval might be normal. Table 4 describes the possible relationships of the P wave to the QRS complex.

Incidence and impact of arrhythmias in patients in the intensive care unit[1]

	Sustained supraventricular arrhythmias %	Sustained ventricular arrhythmias %	Conduction abnormalities %	No arrhythmias %
Incidence	8	2	2	88
Unadjusted in-hospital death rates	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

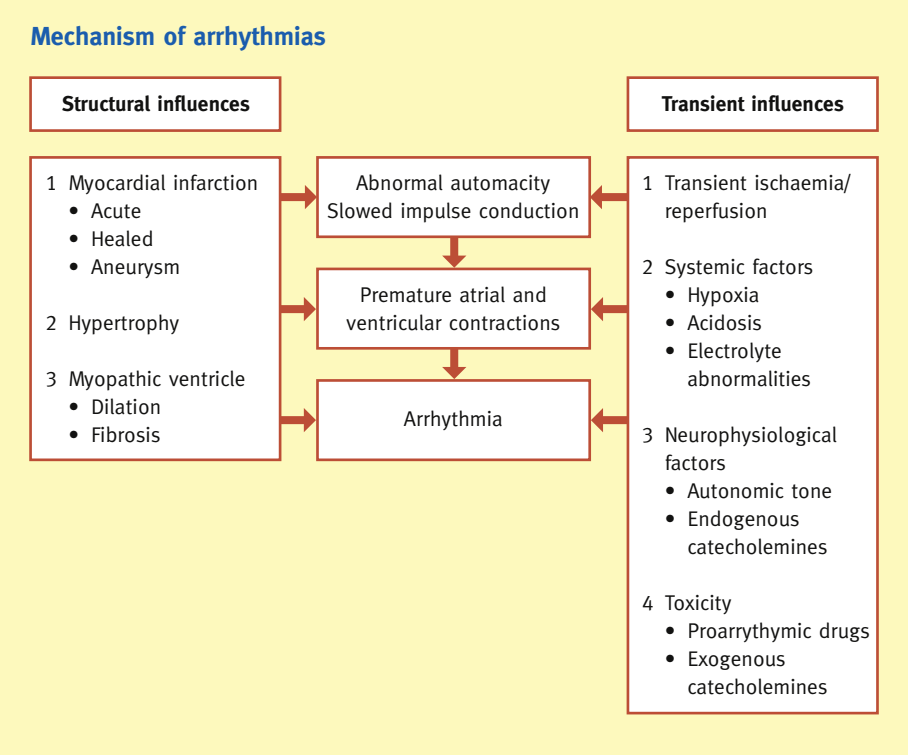


Figure 1

Some causes of arrhythmias in the critically ill

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 processes

- Hypothermia
- Hyperkalaemia
- Head injury and raised intracranial pressure
- Drugs, e.g. β-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

AV, atrioventricular; MI, myocardial infarction.

Table 2

Question 5: How can a supraventricular tachycardia with aberrant conduction be distinguished from a ventricular tachycardia?

A broad complex tachycardia is either VT or a supraventricular tachycardia (SVT) with aberrant conduction (left bundle branch block or right bundle branch block). 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 these are important in terms of their subsequent management. However, if there is any doubt, treat the arrhythmia as VT because this has the propensity to progress to ventricular fibrillation. Clinically, a changeable pulse pressure, irregular cannon waves in the jugular waveform and a variable first heart sound are features common to VT. A useful scheme based on the morphology of the QRS complex is shown in Figure 3.

Additional electrocardiographic features suggestive of a VT are as follows.

- **Fusion beat**: a sinus beat conducts via the AV node to the ventricles but fuses with a beat arising in the ventricle; the complex is intermediate between a normal beat and a tachycardia beat.
- **Capture beat**: when an atrial impulse 'captures' the normal conduction system and an early, narrow complex is seen.
- **QRS duration**: longer than 140 ms.
- **Axis**: extreme left axis.

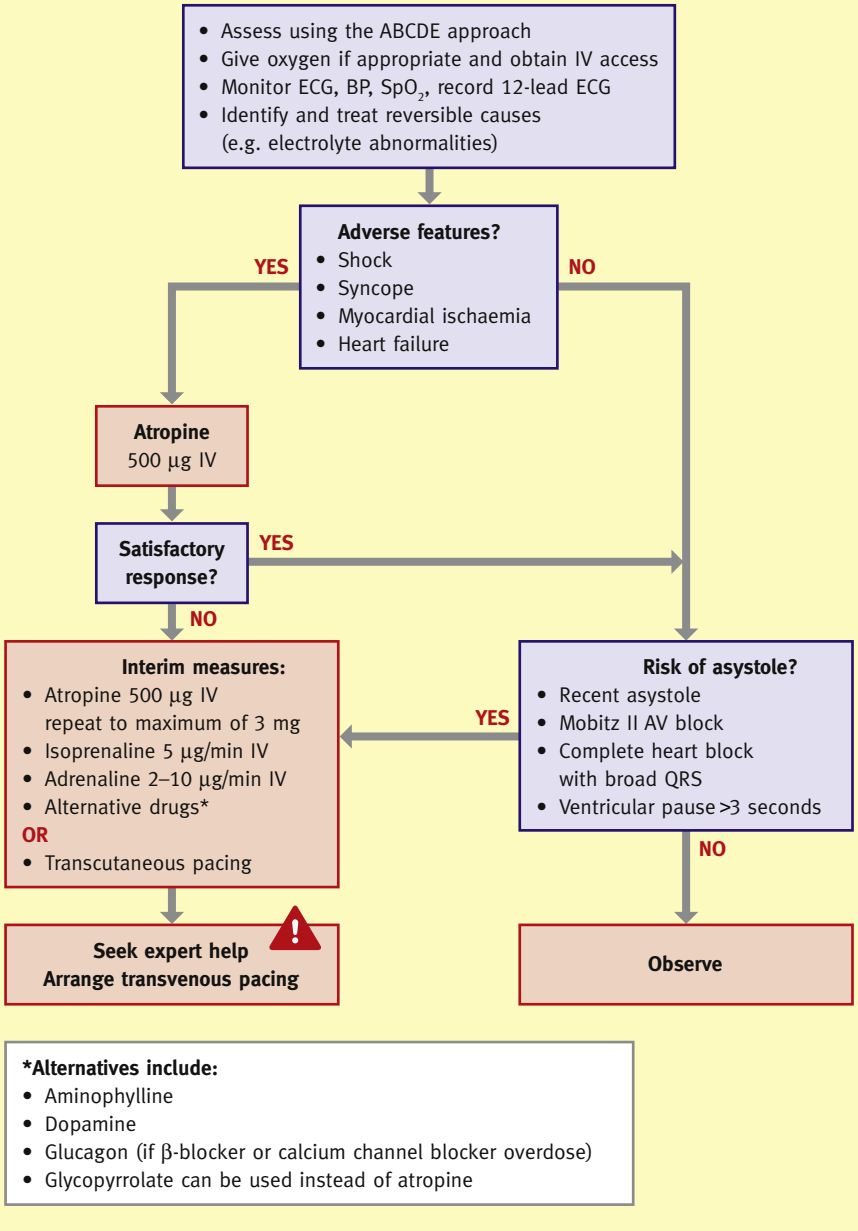
Common bradycardias classified according to their relationship to the P wave

Regular relationship	Irregular relationship	No P waves visible
Sinus bradycardia	Complete HB	Atrial fibrillation with slow ventricular response
First-degree HB	Möbitz type II second-degree HB	Atrial flutter with slow ventricular response
Möbitz type I second-degree HB		

HB, heart block.

Table 3

Adult bradycardia algorithm 2010 Resuscitation Guidelines



Resuscitation Council UK. Reproduced with kind permission. AV, atrioventricular; BP, blood pressure; ECG, electrocardiography; IV, intravenous.

Figure 2

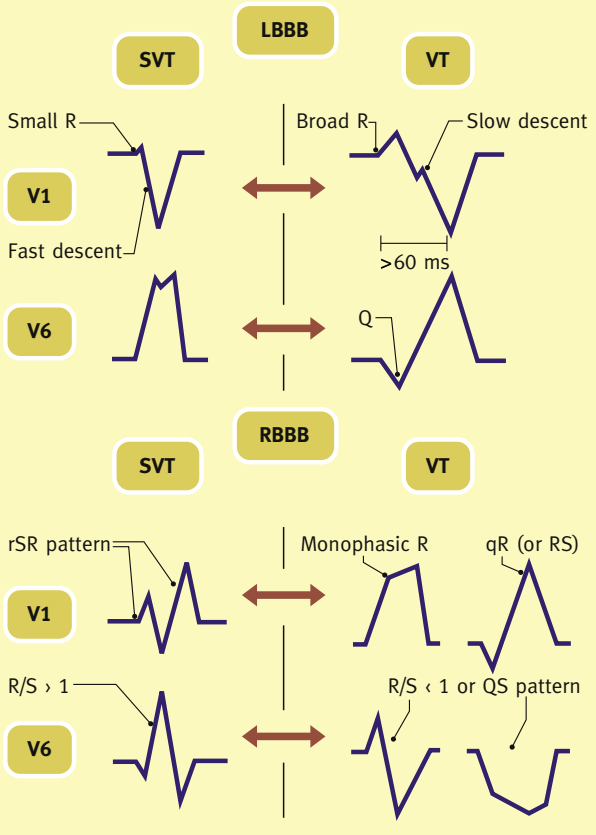
Common tachycardias classified according to their relationship to the P wave

Single P wave per QRS complex	No apparent relationship between P wave and QRS complex	
	QRS < 120 ms	QRS > 120 ms
Sinus tachycardia	Regular RR	SVT with aberrant conduction
Sinus node re-entry (HR 130)	SVT	VT
Intra-atrial re-entry (HR 140–240)	AV re-entrant	Fascicular tachycardia
Ectopic atrial tachycardia	AV nodal re-entrant	
	Irregular RR interval	
	Atrial fibrillation	

AV, atrioventricular; HR, heart rate; RR, RR interval = distance from the onset of one QRS to the onset of the next; SVT, supraventricular tachycardia; VT, ventricular tachycardia.

Table 4

Distinguishing features of supraventricular tachycardia with aberrant conduction from ventricular tachycardia based on the morphology of the QRS complex



LBBB, left bundle branch block; RBBB, right bundle branch block; SVT, supraventricular tachycardia; VT, ventricular tachycardia.

Reproduced with permission from Eckardt et al.[2]

Figure 3

- **Concordance**: all QRS complexes in the chest leads are either positive or negative.
- **AV dissociation**: present in 20–50% of VTs and almost never in an SVT.

The electrocardiographic characteristics of common atrial tachycardias are presented in Box 3.

Electrocardiographic characteristics of atrial tachycardias

- Sinus tachycardia
  - P waves have normal morphology
  - Atrial rate 100–200 bpm
  - Regular ventricular rhythm
  - Ventricular rate 100–200 bpm
  - One P wave precedes every QRS complex
- Atrial tachycardia
  - Abnormal P wave morphology
  - Atrial rate 100–250 bpm
  - Ventricular rhythm usually regular
  - Variable ventricular rate
- Atrial flutter
  - Undulating saw-toothed baseline F (flutter) waves
  - Atrial rate 250–350 bpm
  - Regular ventricular rhythm
  - Ventricular rate typically 150 bpm (with 2:1, 3:1 or 4:1 atrioventricular conduction)
  - 1:1 conduction is uncommon
- Atrial fibrillation
  - P waves absent; oscillating baseline f (fibrillation) waves
  - Atrial rate 350–600 bpm
  - Irregular ventricular rhythm
  - Ventricular rate 100–180 bpm

Reproduced with permission from Goodacre and Irons.[3]

Box 3

Treatment of tachyarrhythmias

Anti-arrhythmic drugs and the Vaughan Williams classification: the Vaughan Williams classification (see also Drugs acting on the heart: anti-arrhythmics, on pp 374–377 of this issue) of anti-arrhythmic drugs has both physiological and therapeutic uses. In the first instance, by describing the electrophysiological mode of action (see Table 2 on p 377 of this issue); in the latter instance, if a drug in one class is ineffective treatment for an arrhythmia, a drug from another class is often chosen.

The parenteral route of administration is usually the preferred route of administration in the critically ill, thus, the repertoire of drugs is relatively limited.

The 2010 revision of the Resuscitation Council (UK) guidelines for the treatment of tachyarrhythmias are shown in Figure 4.

Question 6: Is the arrhythmia associated with congenital heart disease or prolongation of the QT interval?

Arrhythmias in patients with congenital heart disease

The majority of patients with congenital heart disease (CHD) survive to adulthood. The longer the survival, the greater the chance of the development of arrhythmias.

The arrhythmias in adults with CHD are:

- macro-re-entry tachycardia
  - associated with thrombosis and sudden death
- atrial fibrillation
- ventricular arrhythmias
  - often in the setting of ventricular incision and scarring
  - associated with sudden death
- sinus node dysfunction
  - associated with paroxysmal atrial tachycardias
- heart block
  - associated with surgery to the ventricular septum
- pre-excitation
  - associated with Ebstein's anomaly.

The patients most likely to have arrhythmias are those having had:

- Senning or Mustard procedures
- Fontan procedures
- repair of tetralogy of Fallot.

The anatomical substrates initiating arrhythmias include:

- ventricular hypertrophy
- myocardial fibrosis
- chronic pressure and volume overload
- surgical scars.

Because arrhythmias worsen cardiac function and vice versa, a vicious cycle of clinical deterioration can ensue.

The following factors make management decisions difficult:

- nature of the CHD
- difficulty titrating anti-arrhythmic therapy with effect
- possibility that multiple arrhythmias can simultaneously co-exist
- difficulty distinguishing intra-atrial re-entrant tachycardia with sinus node dysfunction from VT
- associated risks of thrombosis and sudden death
- increasing use of catheter or surgical ablative therapy
- consideration of cardiac valve replacement to prevent further deterioration in cardiac function
- possible presence of implantable defibrillators.

It is therefore in the patient's best interests to call for advice from those with special expertise in the longer term management of adults with CHD.

Prolongation of the QT interval

The normal QT interval is from the onset of the QRS to the end of the T wave and is normally 0.35–0.45 s (QTc 0.38–0.42).

Calculation of the QT corrected for heart rate (QTc) is as follows:

Bazetts formula $\mathrm{QTc} = \mathrm{QT\ interval}/(\sqrt{\mathrm{RR\ interval}})$

RR interval = the onset of one QRS to the onset of the next

Note that Bazett's formula tends to over-correct at high heart rates and under-correct at low.

Although Vaughan Williams class 3 anti-arrhythmic drugs prolong the QT interval (measured while in sinus rhythm), paradoxically other drugs capable of the same effect are proarrhythmic, the sensitivity for which is markedly increased in those patients with the long QT syndrome (LQTS). The list of such drugs is formidable, and the reader is referred to www.azcert.org.

The mode of action common to many of these drugs is to alter the inward rectifying current (I_{Kr}), predisposing to polymorphic ventricular tachycardia (torsade de pointes), VF and sudden death.

Other acquired causes of prolonged QT interval are hypokalaemia, hypomagnesaemia and hypocalcaemia.

Ion channelopathies

Ion channelopathy is the term used to cover mutations of ion channels, which within the current context predispose to malignant arrhythmias and sudden cardiac death. The molecular basis for the pro-arrhythmic state include mutations of the:

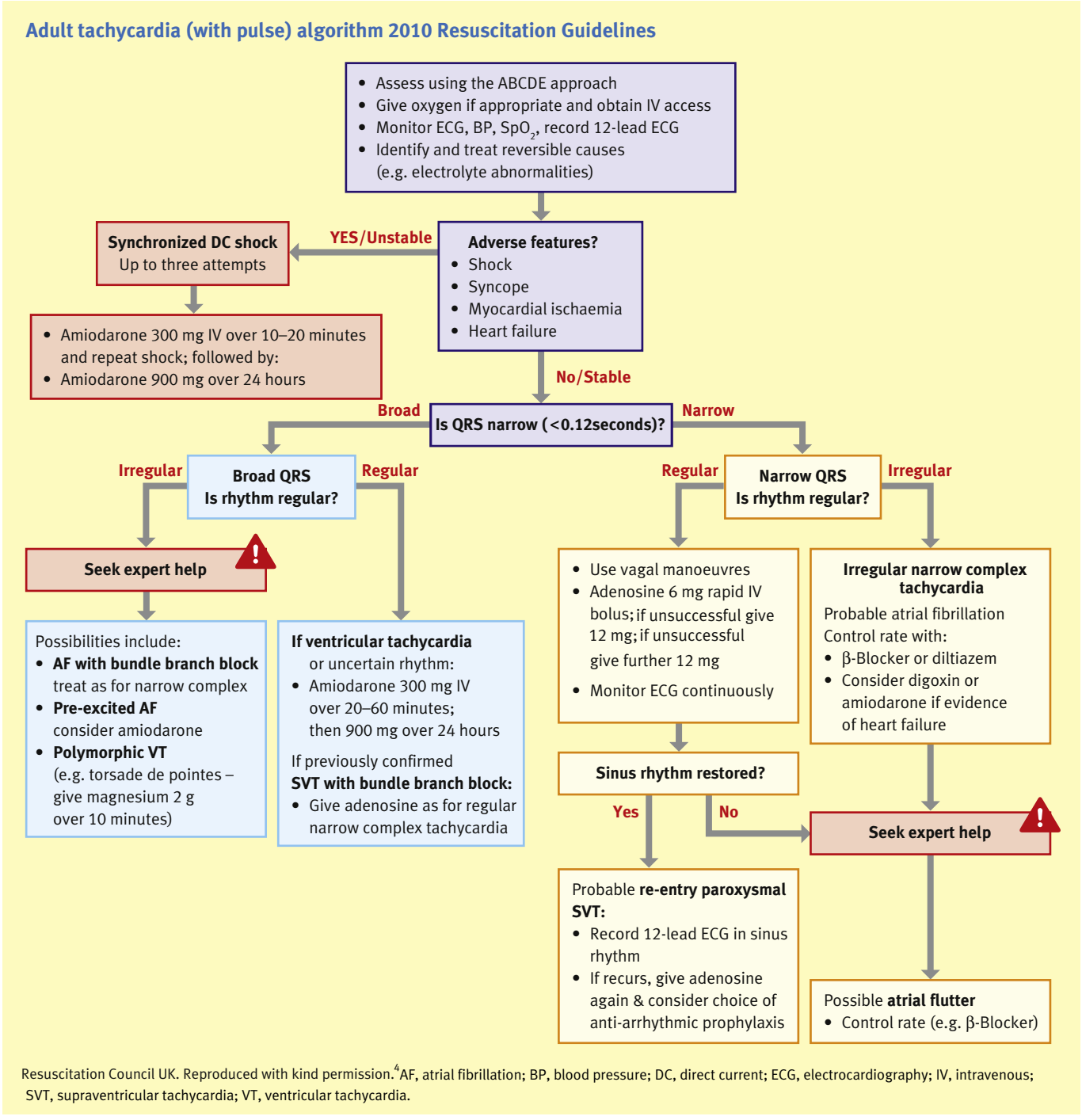
- *KCNE1* and *KCNQ1* gene: this results in a slowing of the function of the slow cardiac outward delayed rectifier K^+ current (I_{Ks}) (LQT-1)
- *HERG* (human ether-a-go-go-related gene, also known as *KCNH2*): this results in a reduction in the HERG channel current and the I_{Kr} (LQT-2)
- *SCN5A* gene: causes a slowing of the inactivation fast Na^+ current, I_{Na}, prolonging the Na influx during depolarization (LQT-3 and in some cases of the Brugada syndrome).

This is a rapidly changing arena with an increasing number of recognized variations, the common feature (consequent upon a prolongation of the QT interval) being an increased vulnerability to ectopic beats. Importantly, amiodarone in these patients can further aggravate the arrhythmia.

Question 7: Should I call for expert help to manage the patient and the arrhythmia?

Within the context of critical care, management objectives are directed towards the rapid restoration of the circulation and minimization of complications. What is evident is:

- the cause of the arrhythmia can be complex
- the drug therapies might worsen the arrhythmia
- definitive investigation and treatment might involve EPS, ablation or implantable devices (implantable cardioverter defibrillator; ICD).



Resuscitation Council UK. Reproduced with kind permission.[4] AF, atrial fibrillation; BP, blood pressure; DC, direct current; ECG, electrocardiography; IV, intravenous; SVT, supraventricular tachycardia; VT, ventricular tachycardia.

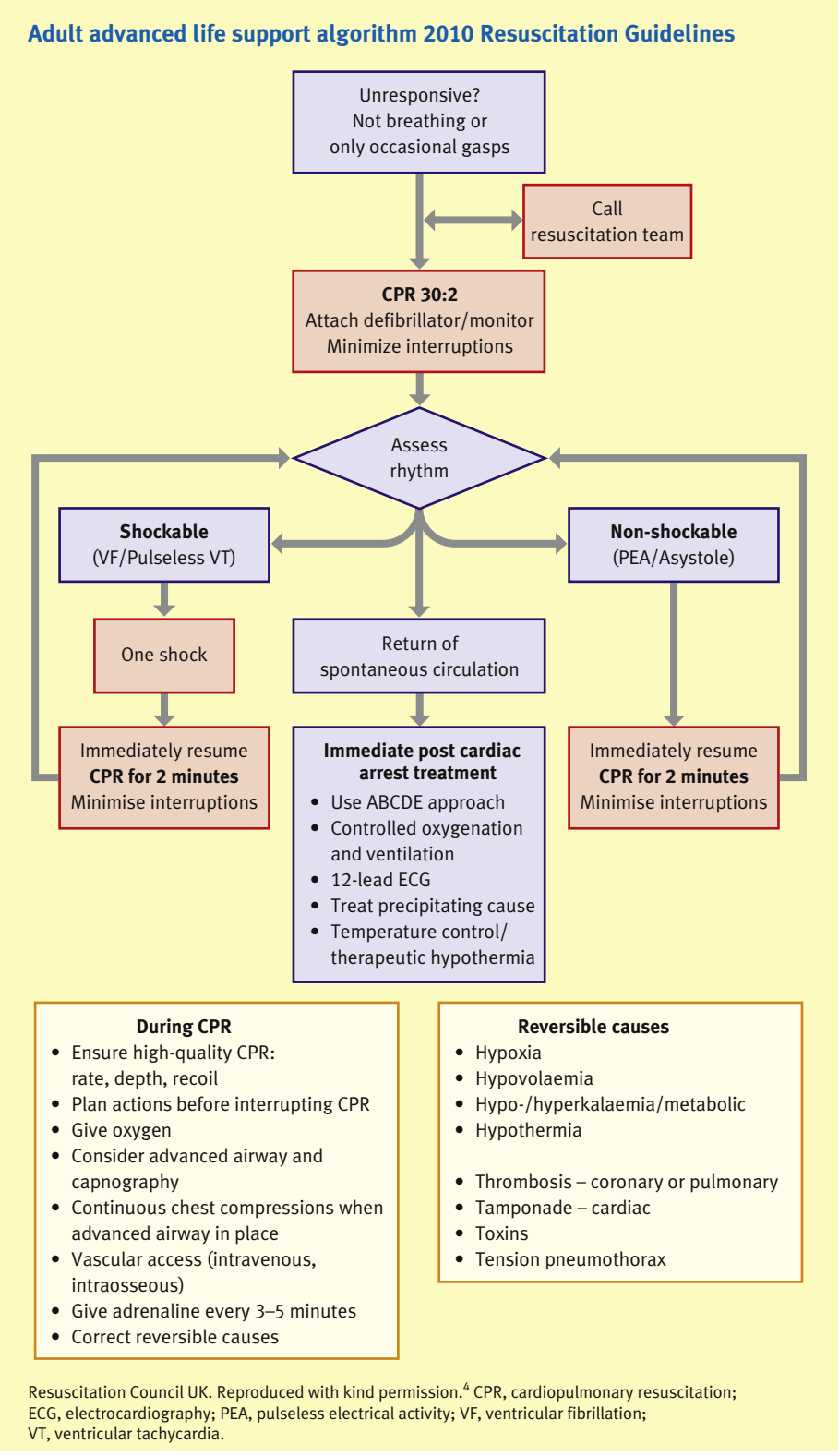
Figure 4

The broad circumstances when advice needs to be sought are if:

- the arrhythmia is in the setting of congenital heart disease
- the arrhythmia is recurrent
- the arrhythmia has failed to respond to first-line treatments
- the QT interval is prolonged
- the arrhythmia has resulted in an episode of 'failed sudden death'
- temporary pacing is indicated
- the patient has been fitted with an ICD.

Management of cardiac arrest

In 2010, the International Liaison Committee of Resuscitation updated the guidelines for the management of cardiac arrest based on the latest evidence and expert opinion (Figure 5). Like the 2005 guidelines, there is a further emphasis on early, good quality, uninterrupted chest compressions. These should be performed at a ratio of 30 compressions to two ventilatory breaths. There should be minimal interruptions for rhythm checks, drug administration and the delivery of shocks. Ideally this period should be



Resuscitation Council UK. Reproduced with kind permission.[4] CPR, cardiopulmonary resuscitation; ECG, electrocardiography; PEA, pulseless electrical activity; VF, ventricular fibrillation; VT, ventricular tachycardia.

Figure 5

less than 5 seconds. Chest compressions should be continued as the defibrillator is charging. The use of the pre-cordial thump is de-emphasized.

With regard to drug therapy used in cardiac arrest, several changes have been made compared to the 2005 guidelines. Drug delivery via the endotracheal route is no longer recommended. If intravenous access cannot be established, the intra-osseous route is recommended. Atropine is no longer recommended for routine use in the treatment of asystolic or pulseless electrical activity (PEA). In the treatment of VF and pulseless VT, adrenaline is

Cardiovascular effects of hypothermia (adapted from Polderman, 2004)

Temperature	Effect
35–36°C	Tachycardia
<35°C	Bradycardia
	Increased central venous pressure
	Decreased CO
	Increased or unchanged mixed venous saturation
<34°C	Slight increase in blood pressure
<33°C	ECG changes: Increased PR interval, widening of QRS complex, increased QT interval
<28–30°C	Tachyarrhythmias, beginning with atrial fibrillation
<28°C	Ventricular fibrillation/ventricular tachycardia

Table 5

given after the third shock at the same time as 300 mg of amiodarone.

The use of three successive shocks in VF and pulseless VT is only recommended if used during cardiac catheterization in the laboratory or in the period immediately following cardiac surgery.

Therapeutic hypothermia and arrhythmias

Therapeutic hypothermia (TH) is being used with increasing frequency to provide protection for the brain, spinal cord and perhaps other organs, such as the heart, against ischaemic and post-traumatic injury. Interest in TH started around the 1950s for cardiovascular surgery and neurological injuries but was largely abandoned over the next 30 years. The publication of two pivotal randomized, controlled trials in 2002 confirmed the efficacy of TH in improving neurological outcomes post-VF/VT cardiac arrest. This then led to the International Liaison Committee on Resuscitation recommending that unconscious adult patients with spontaneous circulation after out-of-hospital cardiac arrest should be cooled to 32–34°C for 12–24 hours when the initial rhythm was VF/VT. Although there is uncertainty concerning the use of TH in non-VF/VT arrest, more recent evidence from the literature would suggest that favourable outcomes have also been achieved in this group.

Hypothermia tends to result in a bradycardia due to decreased depolarization of cardiac pacemaker cells and overall decrease in metabolism. Atropine is usually unhelpful as the bradycardia is not mediated by the vagus nerve. Mean arterial pressure and cardiac output decrease, and an ECG may show characteristic J or Osborne waves. Hypothermia can also cause atrial and ventricular arrhythmias, below a core temperature of 28°C, asystole and ventricular fibrillation have been noted to begin spontaneously (Table 5).

Arrhythmias are common in the post-resuscitation phase in these patients due to the underlying nature of the disease. At the initiation of TH, there is an increase in myocardial demand and irritability due to the increase adrenaline and noradrenaline levels. Surprisingly both the original Hypothermia After Cardiac Arrest Trial (HACA 2008) and subsequent studies found no difference in incidence of arrhythmia in control and patients treated with therapeutic hypothermia.[5] Up to one-third of patients post-cardiac arrest will have clinical significant arrhythmias in both treatment groups. In a survey of UK intensive care units (ICUs), 42% of the ICUs that use therapeutic hypothermia would rewarm patients before the end of the target time if complications occurred, particularly cardiac instability or bradycardia.

It should be remembered that the rewarming phase of TH can also provoke arrhythmias. The maximum acceptable rewarming rate is 0.5°C per hour.

In addition, hypothermia induces renal tubular dysfunction and electrolyte shifts. Indeed it is not uncommon for patients treated with TH to undergo a marked diuresis and hypokalaemia. ◆

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