

Accident and Emergency Department

TOPIC:

Peri arrest arrhytmias in emergency department "early assessment and treatment to prevent cardiac arrest"

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OBJECTIVES:

At the end of this presentation you will be able:

To define: - Peri arrest arrhytmias

- Early assessment of Peri arrest arrhytmias
- -Treatment of Peri arrest arrhytmias
- Prevention of cardiac arrest and decisions about CPR

What is peri arrest period?

The recognized **period**, either just before or just after a full cardiac **arrest**, when the patient's condition is very unstable and care must be taken to prevent progression or regression into a full cardiac **arrest**.

Cardiac arrhythmias are relatively common in the 'peri-arrest' period. An arrhythmia may precede the development of ventricular fibrillation (VF) or asystole or may develop after successful defibrillation. Although arrhythmias are common in the setting of acute myocardial infarction and there are many other causes. Some rhythm abnormalities are usually benign and others usually dangerous; each rhythm encountered requires assessment and treatment in the context of the individual clinical circumstances at the time.

The most common cause of sudden cardiac arrest in adults is pulseless **ventricular tachycardia** (**VT**) or ventricular fibrillation (VF). **VT** can also occur in the presence of a **pulse**; often it is the precursor to VF. **VT** is characterized by rapid, wide (greater than 0.12 seconds) QRS complexes.

Usually, **VT** results from underlying cardiac diseases such as myocardial infarction or cardiomyopathy, but it **can** also be idiopathic or iatrogenic. ... In the acute setting, management of **VT** may **require immediate cardioversion**, defibrillation, or administration of antiarrhythmic drugs.

CONT'

If a patient with an arrhythmia is not acutely ill there may be other treatment options, including the use of drugs (oral or parenteral), that are less familiar to the non-expert. In this situation advice should be sought from the most appropriate experts (e.g. cardiologists).

The treatment algorithms described in this section have been designed to enable the non-specialist advanced life support (ALS) provider to treat a patient effectively and safely in an emergency; for this reason they have been kept as simple as possible. They are based on current national and international guidelines for management of arrhythmia

Sequence of actions

Assess a patient with a suspected arrhythmia using the ABCDE approach

In particular, note the presence or absence of 'adverse features'

- Give oxygen immediately to hypoxemic patients and adjust delivery according to observed arterial oxygen saturations
- Insert an intravenous (IV) cannula

- ▶ Whenever possible, record a 12-lead ECG; this will help identify the precise rhythm, which may guide immediate treatment and/or be crucial to planning later treatment
- ► Correct any electrolyte abnormalities (e.g. K⁺, Mg²⁺, Ca²⁺).

When you assess and treat any arrhythmia address two factors

- The condition of the patient (stable versus unstable determined by the absence or presence respectively of adverse features)
- The nature of the arrhythmia.

Adverse features

The presence or absence of adverse symptoms or signs will dictate the appropriate immediate treatment for most arrhythmias. The following adverse features indicate that a patient is at high risk of early deterioration and death ('unstable'), either because of the arrhythmia itself or because of underlying heart disease with the arrhythmia superimposed

- Shock hypotension (systolic blood pressure <90 mm Hg), pallor, sweating, cold, clammy extremities, confusion or impaired consciousness</p>
- Syncope transient loss of consciousness due to global reduction in blood flow to the brain
- Myocardial ischemia typical ischemic chest pain and/or evidence of myocardial ischemia on 12-lead ECG
- Heart failure pulmonary edema and/or raised jugular venous pressure (with or without peripheral edema and liver enlargement).

Treatment options

- Depending on the nature of the underlying arrhythmia and clinical status of the patient (in particular the presence or absence of adverse features) immediate treatment options can be categorized under four headings:
- No treatment needed
- Simple clinical intervention (e.g. vagal maneuvers, fist pacing)
- Pharmacological (drug treatment)
- Electrical (cardioversion for tachyarrhythmia or pacing for Brady arrhythmia

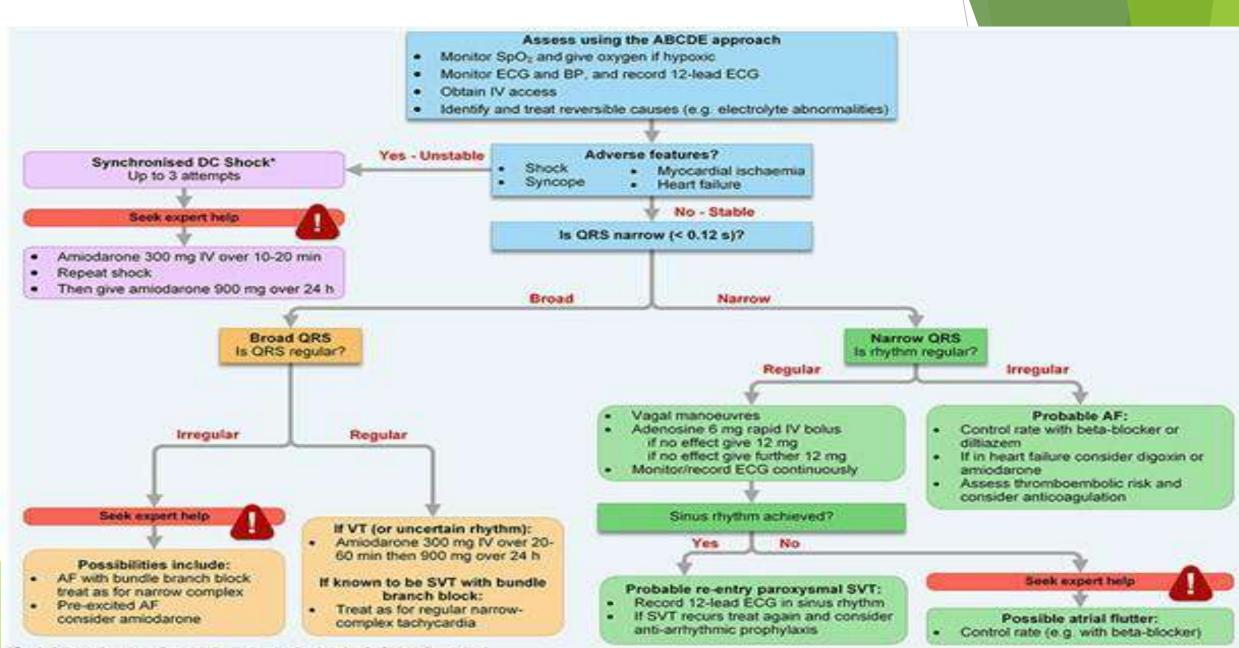
Most drugs act more slowly and less reliably than electrical treatments, so electrical treatment is usually the preferred treatment for an unstable patient with adverse features.

If a patient develops an arrhythmia during, or as a complication of some other condition (e.g. infection, acute myocardial infarction, heart failure), make sure that the underlying condition is assessed and treated appropriately, involving relevant experts if necessary.

Once an arrhythmia has been treated successfully, continue to assess the patient (ABCDE) and repeat a 12-lead ECG to detect any other abnormalities that may require treatment, either immediately or in the longer term.

Tachycardia

The approach to an adult with tachycardia and a palpable pulse is shown in the Adult Tachycardia (with pulse) algorithm (Figure 1).



^{*}Conscious patients require sedation or general anaesthesia for cardioversion

If a patient is unstable

If a patient with a tachyarrhythmia is unstable (i.e. has adverse features likely to be caused or made worse by the tachycardia) synchronized cardioversion is the treatment of choice. In patients with otherwise normal hearts adverse symptoms and signs are uncommon from arrhythmia with ventricular rate <150 min⁻¹.

Synchronized cardioversion

- If the patient is conscious, carry out cardioversion under sedation or general anesthesia, administered by a healthcare professional competent in the technique being used. Ensure that the defibrillator is set to synchronized mode.
- For a broad-complex tachycardia or atrial fibrillation, start with 120–150 J and increase in increments if this fails.
- Atrial flutter and regular narrow-complex tachycardia will often be terminated by lower energies: start with 70–120 J.

If the patient is stable

► If a patient with a tachyarrhythmia has no adverse features consider whether any treatment is required. If so, consider using drug treatment in the first instance

Assess the ECG and determine the QRS duration. If the QRS duration is 0.12 s or greater (3 small squares on standard ECG paper speed of 25 mm s⁻¹) this is a broad-complex tachycardia. If the QRS duration is less than 0.12 s it is a narrow-complex tachycardia.

Broad-complex tachycardia

Many broad-complex tachycardia (QRS ≥ 0.12 s) are ventricular in origin. In other cases broad-complex tachycardia may be a supraventricular rhythm with aberrant conduction (bundle branch block). In an unstable patient assume that the rhythm is ventricular in origin and attempt synchronized cardioversion as described above. Conversely, if a patient with broad-complex tachycardia is stable, the next step is to determine from the ECG if the rhythm is regular or irregular.

Regular broad-complex tachycardia

A regular broad-complex tachycardia is likely to be ventricular tachycardia (VT) or a regular supraventricular rhythm with bundle branch block.

► In a stable patient, if the broad-complex tachycardia is thought to be VT, treat with amiodarone 300 mg IV over 20–60 min, followed by an infusion of 900 mg over 24 h

Irregular broad-complex tachycardia

This is most likely to be atrial fibrillation (AF) with bundle branch block, but careful examination of a 12-lead ECG (if necessary by an expert) may enable confident identification of the rhythm. Other possible causes are AF with ventricular pre-excitation (in patients with Wolff-Parkinson-White [WPW] syndrome), or polymorphic VT (e.g. torsade de pointes), but sustained polymorphic VT is unlikely to be present without adverse features. Seek expert help with the assessment and treatment of irregular broad-complex tachyarrhythmia.

- Treat torsade de pointes VT immediately by stopping all drugs known to prolong the QT interval. Do not give amiodarone for definite torsade de pointes. Correct electrolyte abnormalities, especially hypokalemia. Give magnesium sulfate 2 g IV over 10 min (= 8 mmol, 4 mL of 50%
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Narrow-complex tachycardia

- Examine the ECG to determine if the rhythm is regular or irregular. Regular narrow-complex tachycardias include:
- sinus tachycardia
- ► AV nodal re-entry tachycardia (AVNRT) the commonest type of regular narrow-complex tachyarrhythmia
- ► AV re-entry tachycardia (AVRT) due to WPW syndrome
- ▶ atrial flutter with regular AV conduction (usually 2:1).
- Irregular narrow-complex tachycardia is most likely to be AF or sometimes atrial flutter with variable AV conduction ('variable block').

Regular narrow-complex tachycardia

Sinus tachycardia : is not an arrhythmia. This is a common physiological response to stimuli such as exercise or anxiety. In a sick patient it may occur in response to many conditions including pain, infection, anemia, blood loss, and heart failure. Treatment is directed at the underlying cause. Trying to slow sinus tachycardia that has occurred in response to most of these conditions will usually make the situation worse. Do not attempt to treat sinus tachycardia with cardioversion or anti-arrhythmic drugs.

Treatment of regular narrow-complex tachycardia

If the patient is unstable, with adverse features caused by the arrhythmia, attempt synchronized electrical cardioversion. It is reasonable to apply vagal maneuvers and/or give adenosine to an unstable patient with a regular narrow-complex tachycardia while preparations are being made urgently for synchronized cardioversion. Do not delay electrical cardioversion if adenosine fails to restore sinus rhythm.

In the absence of adverse features:

Start with vagal maneuvers:

Carotid sinus massage or the Valsalva maneuver will terminate up to a quarter of episodes of paroxysmal SVT. Record an ECG (preferably multi-lead) during each maneuver. If the rhythm is atrial flutter, slowing of the ventricular response will often occur and reveal flutter waves.

If the arrhythmia persists and is not atrial flutter, give adenosine 6 mg as a rapid IV bolus. Use a relatively large cannula and large (e.g. antecubital vein. Warn the patient that they will feel unwell and probably experience chest discomfort for a few seconds after the injection, Record an ECG (preferably multi-lead) during the injection If the ventricular rate slows transiently, but then speeds up again, look for atrial activity, such as atrial flutter or other atrial tachycardia, and treat accordingly. If there is no response (i.e. no transient slowing or termination of the tachyarrhythmia) to adenosine 6 mg IV, give a 12 mg IV bolus. If there is no response give one further 12 mg IV bolus. Apparent lack of response to adenosine will occur if the bolus is given too slowly or into a peripheral vein.

- Vagal maneuvers or adenosine will terminate almost all AVNRT or AVRT within seconds. Termination of a regular narrow-complex tachycardia in these ways identifies it as being AVNRT or AVRT. Failure to terminate a regular narrow-complex tachycardia with adenosine suggests an atrial tachycardia such as atrial flutter (unless the adenosine has been injected too slowly or into a small peripheral vein).
- If adenosine is contra-indicated, or fails to terminate a regular narrow-complex tachycardia without demonstrating that it is atrial flutter, consider giving verapamil 2.5–5 mg IV over 2 min.

- An irregular narrow-complex tachycardia is most likely to be AF with an uncontrolled ventricular response or, less commonly, atrial flutter with variable AV block.
- Record a 12-lead ECG to identify the rhythm. If the patient is unstable, with adverse features caused or made worse by the arrhythmia, start antithrombotic therapy and attempt synchronized cardioversion.

- ▶ If there are no adverse features, immediate treatment options include:
- no treatment
- rate control by drug therapy
- rhythm control using drugs to encourage chemical cardioversion
- rhythm control by electrical cardioversion
- treatment to prevent complications (e.g. anticoagulation).

• Obtain expert help to determine the most appropriate treatment for the individual patient. The longer a patient remains in AF the greater is the likelihood of atrial thrombus developing. In general, patients who have been in AF for more than 48 h should not be treated by cardioversion (electrical or chemical) until they have been fully anticoagulated for at least three weeks, or unless trans-oesophageal echocardiography has shown the absence of atrial thrombus.

If the clinical situation dictates that cardioversion is needed more urgently, give either low-molecular-weight heparin in weight-adjusted therapeutic dose or an intravenous bolus injection of unfractionated heparin followed by a continuous infusion to maintain the activated partial thromboplastin time (APTT) at 1.5 to 2 times the reference control value. Continue heparin therapy and commence oral anticoagulation after attempted cardioversion, whether or not it is successful. Seek expert advice on the duration of anticoagulation, which should be a minimum of four weeks, often substantially longer, unless the risk of bleeding is prohibitive.

- If the aim is to control heart rate, the usual drug of choice is a beta-blocker. Diltiazem or verapamil may be used in patients in whom beta-blockade is contraindicated or not tolerated
- If the duration of AF is less than 48 h and rhythm control is considered appropriate, chemical cardioversion may be attempted. Seek expert help with the use of drugs such as flecainide or propafenone. Do not use flecainide or propafenone in the presence of heart failure, known left ventricular impairment or ischemic heart disease, or a prolonged QT interval

Bradycardia

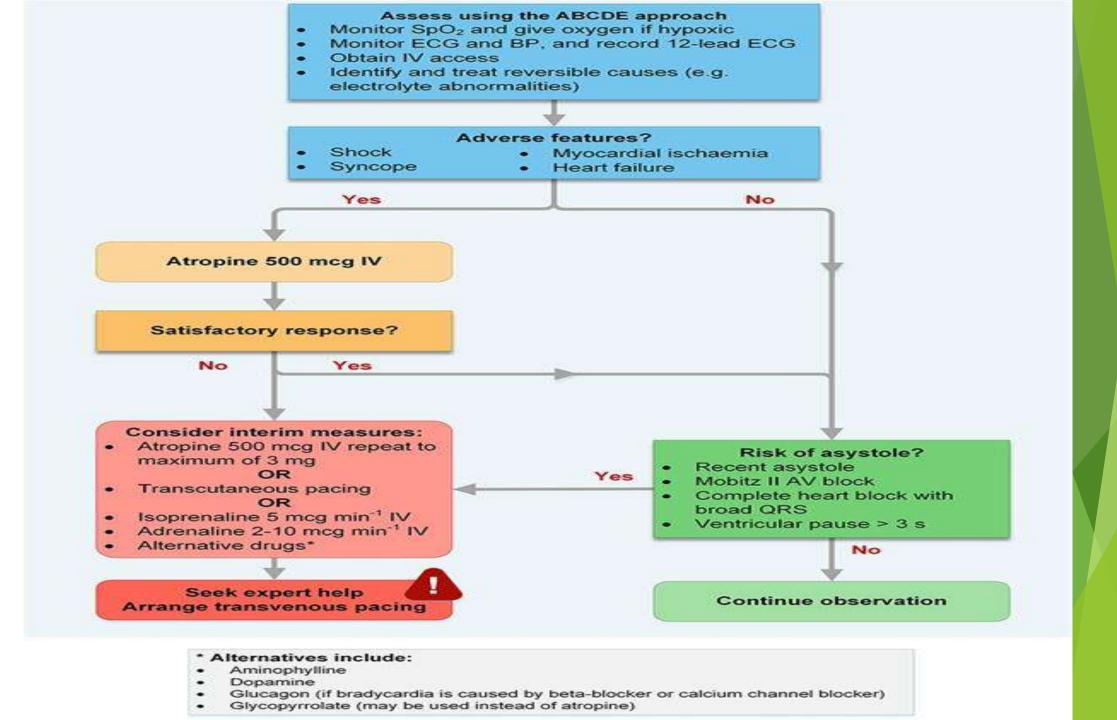
The approach to an adult with bradycardia and a palpable pulse is shown in the Adult Bradycardia algorithm (Figure 2).

Bradycardia is defined as a heart rate in an adult of <60 min⁻¹. Causes include:

- physiological (e.g. during sleep, in athletes)
- cardiac causes (e.g. atrioventricular block or sinus node disease)
- non-cardiac causes (e.g. vasovagal, hypothermia, hypothyroidism, hyperkalemia)
- drugs (e.g. beta-blockade, diltiazem, digoxin, amiodarone) in therapeutic use or overdose.

Assess a patient with bradycardia using the ABCDE approach. Consider the potential cause of the bradycardia and look for adverse features. If no adverse features are present, continue to monitor and reassess the patient (ABCDE). Seek expert help to plan any necessary further assessment and treatment.

Consider treating any reversible causes of bradycardia identified in the initial assessment. If adverse features signs are present start to treat the bradycardia. Initial treatment for most patients is pharmacological; pacing is indicated for patients unresponsive to pharmacological treatment or with risks factors for asystole.



Treatment using drugs

If adverse features are present, give atropine 500 mcg IV and, if necessary, repeat every 3–5 min to a total of 3 mg. Doses of atropine of less than 500 mcg can cause paradoxical slowing of the heart rate. In healthy volunteers a dose of 3 mg produces the maximum achievable increase in resting heart rate. Use atropine cautiously in the presence of acute myocardial ischemia or infarction; the resulting increase in heart rate may worsen ischemia or increase the zone of infarction. Do not give atropine to patients with cardiac transplants. Their hearts are denervated and will not respond to vagal blockade by atropine, which may cause paradoxical sinus arrest or high-grade AV block in these patients.

If bradycardia with adverse signs persists despite atropine, consider cardiac pacing. If pacing cannot be achieved promptly consider the use of second-line drugs. Seek expert help to select the most appropriate choice. In some clinical settings second-line drugs may be appropriate before the use of cardiac pacing. For example consider giving intravenous glucagon if a beta blocker or calcium channel blocker is a likely cause of the bradycardia

Consider using digoxin-specific antibody fragments for bradycardia due to digoxin toxicity. Serious cases of digoxin or other drug toxicity should be discussed with the National Poisons Information Service. Consider . using theophylline (100–200 mg by slow IV injection) for bradycardia complicating acute inferior wall myocardial infarction, spinal cord injury or cardiac transplantation.

Pacing

Transcutaneous pacing

Initiate transcutaneous pacing immediately if there is no response to atropine, or if atropine is contraindicated. Transcutaneous pacing can be painful and may fail to achieve effective electrical capture (i.e. a QRS complex after each pacing stimulus) or fail to achieve a mechanical response (i.e. palpable pulse). Check for electrical capture on the monitor or ECG and check that it is producing a pulse. Reassess the patient's condition (ABCDE). Use analgesia and sedation as necessary to control pain; sedation may compromise respiratory effort so continue to reassess the patient at frequent intervals.

Fist pacing

If atropine is ineffective and transcutaneous pacing is not immediately available, fist pacing can be attempted for life-threatening, extreme bradycardia, while waiting for pacing equipment or personnel. Give repeated rhythmic thumps with the side of a closed fist over the left lower edge of the sternum to stimulate the heart at a rate of 50–70 min⁻¹.

Transvenous pacing

Seek expert help to assess the need for temporary transvenous pacing and to initiate this when appropriate. Temporary transvenous pacing should be considered if there is documented recent asystole (ventricular standstill of more than 3 s), Mobitz type II AV block or complete (third-degree) AV block (especially with broad QRS or initial heart rate <40 beats min⁻¹).

Prevention of cardiac arrest and decisions about

Prevention of cardiac arrest is the first link in the Chain of Survival.³ This section of the Resuscitation Council guidelines stresses the importance of preventing cardiac arrest in all age groups, and the decision-making process when cardiopulmonary resuscitation (CPR) is inappropriate.

Prevention of out-of-hospital cardiac arrest

Recognizing and responding to cardiac chest pain

Most sudden cardiac death (SCD) victims have a history of heart disease and warning symptoms, most commonly chest pain, in the hour before cardiac arrest.⁸ Early recognition of cardiac chest pain and rapid activation of the EMS is vitally important.^{9,10} When a call to the EMS is made before a cardiac arrest victim collapses, the ambulance arrives significantly sooner after collapse, and the chance of survival is higher

Prompt assessment of people with acute chest pain by the EMS, including recording and interpretation of a 12-lead ECG, enables appropriate treatment of acute coronary syndromes with a minimum of delay (especially reperfusion therapy, usually by primary percutaneous coronary intervention [PPCI] for ST-segment myocardial infarction [STEMI]), reducing the risk of early cardiac arrest and death and of subsequent complications, including death.

Recognising and responding to other causes of cardiac arrest and sudden death

Coronary artery disease is the commonest cause of SCD in people over the age of 35 years. Other causes of SCD include cardiomyopathies, valve disease, inherited ion channel disorders (e.g. long and short QT syndromes, Brugada syndrome, catecholaminergic polymorphic ventricular tachycardia) and congenital heart disease.^{8,15} Whilst cardiac arrest and SCD are relatively uncommon in people younger than 35, coronary disease is also less common in this age group, so an inherited condition is more likely to be the cause when a younger person suffers unexpected cardiac arrest or SCD.

In patients known to have heart disease, syncope (with or without prodrome – particularly recent or recurrent) is an independent risk factor for increased risk of death.⁸ Apparently healthy children and young adults who suffer SCD may also have symptoms and signs (e.g. syncope/pre-syncope, chest pain, palpitation, heart murmur) that should alert healthcare professionals to seek expert help to prevent cardiac arrest in those at risk. Features that indicate a high probability of arrhythmic syncope (and potential risk of SCD) include:

- syncope in the supine position
- syncope occurring during or after exercise (although syncope after exercise is often vasovagal)
- syncope with no or only brief prodromal symptoms
- repeated episodes of unexplained syncope
- syncope in individuals with a family history of sudden death or inherited cardiac condition.

Prevention of in-hospital cardiorespiratory arrest

Rates of survival and complete physiological recovery following in-hospital cardiac arrest are poor in all age groups. For example, fewer than 20% of adult patients having an in-hospital cardiac arrest (IHCA) will survive to go home.¹⁸ Cardiac arrest is rare in both pregnant women and children, but outcomes in these groups after in-hospital arrest are also poor. **Prevention of in-hospital cardiac arrest requires staff education, monitoring of patients, recognition of patient deterioration, a system to call for help and an effective response**

Adults

Most adult survivors of in-hospital cardiac arrest have a witnessed and monitored ventricular fibrillation (VF) arrest and are defibrillated immediately.²⁰ The underlying cause of arrest in this group is usually myocardial ischemia. In comparison, cardiac arrest in patients in unmonitored ward areas is usually a predictable event not caused by heart disease.²¹ In this group, cardiac arrest often follows a period of slow and progressive physiological deterioration involving unrecognized or inadequately treated hypoxemia and hypotension.²² The cardiac arrest rhythm is usually asystole or PEA, and the chance of survival to hospital discharge is extremely poor unless a reversible cause is identified and treated immediately.

Recognition of 'at-risk', or critically ill, adult patients

When patients deteriorate, they display common signs that represent failing respiratory, cardiovascular, and nervous systems. This is the basis for monitoring patients' vital signs. Abnormal physiology is common on general wards,²⁹ yet the important physiological observations of sick patients are measured and recorded less frequently than is desirable

Children

In children, cardiorespiratory arrest is more often caused by profound hypoxemia and/or hypotension than by heart disease. Ventricular fibrillation is less common than asystole or pulseless electrical activity. As with adults, there may be opportunities to introduce strategies that will prevent arrest.

Resuscitation decisions

CPR was originally developed to save the lives of people dying unexpectedly when acute myocardial infarction (AMI) caused sudden cardiac arrest in ventricular fibrillation – 'hearts too good to die'. As awareness of CPR increased and resuscitation equipment became more widely available and more portable, attempts at CPR became very common in situations other than a sudden cardiac arrest due to AMI

When to consider making decisions about CPR

Recognition of an 'at-risk' or critically ill/deteriorating patient should trigger consideration of whether or not attempted CPR would be successful and/or in the patient's best interests. Critical care outreach and medical emergency teams may contribute to a 'reduction' in cardiorespiratory arrests by triggering such consideration and thereby avoiding inappropriate CPR attempts However, a crisis situation when someone is acutely unwell and has been admitted to hospital, is not the optimal time to make anticipatory decisions about CPR for most people who have advanced medical conditions and are approaching the end of life. Early consideration of CPR decisions is recommended in the context of broader 'advance care planning' (see 'Discussing decisions about CPR' below).

Decisions about CPR should be considered, discussed and recorded:

At the request of a person with capacity.

As an important element of end-of-life care for a person who is terminally ill from an advanced and irreversible disease.

As an important element of care of a patient with an acute severe illness, who continues to deteriorate towards death despite all appropriate treatment or who has suffered a sudden catastrophic event from which no recovery can be reasonably expected.

As an element of care of people recognized by healthcare professionals as approaching the end of their lives (i.e. within the last year of life).

Recording decisions about CPR

All considerations, discussions and decisions about CPR must be recorded fully and clearly, together with details of the reasons for any decision. Such decisions should also be communicated clearly, where necessary in writing, to all those involved in the patient's care

Reviewing decisions about CPR

Just as every hospital patient should have a plan detailing their individual needs for type and frequency of vital-sign measurement, so every person with a CPR decision should have a recorded plan detailing their individual need for review of that decision

Recorded decisions about CPR should be reviewed:

if the patient requests review

if those close to the patient request review

whenever there is a significant change in the patient's clinical condition

when the patient moves from one care setting to another (including transfer between wards or teams in a hospital).

Finally, Peri arrest arritymias are common in severely admitted patients in Accident and Emergency, most of them are in life threatening conditions and must be quickly recognized and treated to avoid fatal cardiac arrest. The treatment can be pharmacological or Electrical cardioversion based on whether patient is stable or unstable,

In addition, for Cardio arrest prevention require Recognition and responding to cardiac chest pain for out of hospital patient and for in hospital patients occure the staff education, monitoring of patients, recognition of patient deterioration, a system to call for help and an effective response for in-hospital patients,

In case of CPR in Emergency the decision making is taken by Emergency medical staff due to clinical history and prognosis of the patient.

THANK YOU FOR YOUR KIND ATTENTION

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