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EMERGENCY MANAGEMENT OF PATIENTS WITH CARDIAC ARREST

Methodical recommendations
to prepare 5th year students for practical classes
Discipline "Emergency and urgent medical care"

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The methodical recommendations set out the main aspects of the management of patients with cardiac arrest. For 5th year students to prepare for practical classes in the discipline " Emergency and urgent medical care".

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Навчальне видання

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**НЕВІДКЛАДНА ДОПОМОГА ПАЦІЄНТАМ
З ЗУПИНКОЮ СЕРЦЯ**

Методичні вказівки до практичних занять для здобувачів вищої медичної освіти 5-го року навчання з дисципліни «Екстрена та невідкладна медична допомога»
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LIST OF ABBREVIATIONS

SCA	Sudden cardiac arrest
CPR	Cardiopulmonary resuscitation
AHA	American Heart Association
ILCOR	The International Liaison Committee on Resuscitation
ERC	European Resuscitation Council
OHCA	Out-of-hospital cardiac arrest
BLS	Basic Life Support
ALS	Advanced Life Support
PRLS	Post-Resuscitation Life Support
VF	Ventricular fibrillation
VT	Ventricular tachycardia
PEA	Pulseless electrical activity
ROSC	Return of spontaneous circulation
ICU	Intensive Care Unit
IV	Intravenous
IO	Intra osseus
$ETCO_2$	End-tidal carbon dioxide
TTM	Targeted temperature management

BASIC KNOWLEDGE, SKILLS, HABITS NECESSARY FOR STUDYING THE TOPIC

Names of previous disciplines	Acquired skills
Foreign Language	Be able to work with foreign sources to obtain up-to-date data on methods of diagnosis and treatment cardiac arrest
Medical informatics	Apply modern computer programs and be able to work with them, have statistical methods for processing the results of clinical trials, analyze research results, be able to evaluate and interpret the results of clinical trials
Human anatomy Normal physiology Histology, cytology and embryology	Know the normal structure, functions and regulation of the cardiovascular, pulmonary, central nervous system understand and determine the relationship of its structure and functions with other organs and systems of the human body
Pathomorphology Pathophysiology	Know the typical pathological processes: mechanisms of development, changes in the human body, compensatory reactions of the body, the development of connections that have the character of "cause-effect", in the pathology of the whole organism.
Pharmacology	Be able to navigate the range of drugs. Know the mechanisms of action of drugs, their pharmacodynamics, indications and contraindications to their use. Know the features of clinical pharmacology of drugs used in the treatment of cardiac arrest and post-cardiac-arrest syndrome, features of the pharmacological action of these drugs in different categories of patients. Make a reasonable choice of individual drugs and treatment regimens taking into account the principles of evidence-based medicine, optimization of treatment regimens, evaluate the effectiveness and safety of pharmacotherapy taking into account the individual characteristics of the patient, the presence of comorbidities.
Propaedeutics of internal medicine	Conduct a physical examination of patients, analyze the results of basic laboratory and instrumental research methods. Identify the leading syndromes and symptoms. Be able to make a differential diagnosis, substantiate and formulate a diagnosis on the basis of physical examination and data of additional methods.

THE STUDENT SHOULD KNOW:

- anatomy and functions of the cardiovascular sistem;
- anatomy and functions of the pulmonary sistem;
- definition and epidemiology of sudden cardiac arrest;
- main causes and pathogenesis of cardiac arrest;
- diagnostic criteria for cardiac arrest;
- standards of treatment for cardia arrest;
- prevention and prognosis of cardiac arrest;
- cardio-pulmonary resuscitation;
- stages of cardio-pulmonary resuscitation;
- Basic Life Support;
- when and how to use an AED;
- Advanced Life Support;
- Airway management and Ventilation;
- assisting the Circulation;
- post-resuscitation life support.

The student should be able to:

- conduct a diagnostic of cardiac arrest in unconscious patients;
- to make a plan for cardio-pulmonary resuscitation of the patient;
- to make a differential diagnosis of cardiac arrest, syncope and respiratory arrest;
- to determine the tactics Airway management and Ventilation in each case;
- to make assisting the Circulation in patient with cardiac arrest.

INTRODUCTION

Today, sudden cardiac arrest (death) (SCA) is an urgent medical and social problem.

Cardiopulmonary resuscitation (CPR) refers to a series of emergency lifesaving actions which is performed in an effort to manually resuscitate a person in cardiac arrest. As CPR requires various emergency treatments in short time, the essential treatment procedures had been established as a standardized guideline. In 1962, the American Heart Association (AHA) had established "A Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care" for the first time and since then the efforts to medically improve CPR has been continued up to now, and it has been applied continually to CPR based on the research results known from many clinical studies. Thereafter, the

International Liaison Committee on Resuscitation (ILCOR) which was constituted in 1993 had performed its tasks to apply new scientific grounds which were periodically accumulated at each 5 year from 2000 to the Guidelines for Cardiopulmonary Resuscitation and came to present an Integrated Guidelines, providing each country scientific grounds for revising or establishing their own CPR guidelines.

In late 2021, the new recommendations of the European Resuscitation Council (ERC) were published.

CARDIAC ARREST

Epidemiology of cardiac arrest. Sudden cardiac death refers to an unexpected death from cardiac arrest. According to the WHO, the incidence of sudden cardiac death is 30 cases per week per 1 million population. It is the third leading cause of death in Europe. Worldwide, SCA is the most common cause of death accounting for 17 million deaths every year or 25% of all global mortality. Out-of-hospital cardiac arrest (OHCA) is a global health issue with incidence reported as 40.6 per 100,000 person-years in Europe, 47.3 in North America, 45.9 in Asia, and 51.1 in Australia.

The incidence of sudden cardiac death varies in different age groups. Thus, people aged 60 to 69 years suffer from it 8 times more often than people aged 30-39 years. In men, SCA observed 3-4 times more often than in women.

Etiology. *In adults*, sudden cardiac arrest results primarily from cardiac disease (of all types, but especially coronary artery disease). In a significant percentage of patients, sudden cardiac arrest is the first manifestation of heart disease. Other causes include circulatory shock due to noncardiac disorders (especially pulmonary embolism , gastrointestinal hemorrhage , or trauma), ventilatory failure, and metabolic disturbance (including drug overdose).

In infants and children, cardiac causes of sudden cardiac arrest are less common than in adults. The predominant cause of sudden cardiac arrest in infants and children is respiratory failure due to various respiratory disorders (eg, airway obstruction, smoke inhalation, drowning , infection, sudden infant death syndrome). Other causes of sudden cardiac arrest include trauma and poisoning.

The main causes and pathogenesis. Potential causes or aggravating factors for which specific treatment exists must be considered during all cardiac arrests. For ease of memory, these are divided into two groups of four, based upon their initial letter: either H or T:

- Hypoxia
- Hypovolaemia

- Hyperkalaemia, hypokalaemia, hypoglycaemia, hypocalcaemia, acidaemia and other metabolic disorders
- Hypothermia
- Thrombosis (coronary or pulmonary)
- Tension pneumothorax
- Tamponade – cardiac
- Toxins

<u>The four 'Hs'</u>

Minimise the risk of **hypoxia** by ensuring that the patient's lungs are ventilated adequately with the maximal possible inspired oxygen during CPR. Make sure there is adequate chest rise and bilateral breath sounds. Using the techniques described below, check carefully that the tracheal tube is not misplaced in a bronchus or the oesophagus.

Pulseless electrical activity caused by **hypovolaemia** is due usually to severe haemorrhage. This may be precipitated by trauma, gastrointestinal bleeding or rupture of an aortic aneurysm. Stop the haemorrhage and restore intravascular volume with fluid and blood products.

Hyperkalaemia, hypokalaemia, hypocalcaemia, acidaemia and other metabolic disorders are detected by biochemical tests or suggested by the patient's medical history (e.g. renal failure). Give IV calcium chloride in the presence of hyperkalaemia, hypocalcaemia and calcium channel-blocker overdose.

Hypothermia should be suspected based on the history such as cardiac arrest associated with drowning.

<u>The four 'Ts'</u>

Coronary **thrombosis** associated with an acute coronary syndrome or ischaemic heart disease is the most common cause of sudden cardiac arrest. An acute coronary syndrome is usually diagnosed and treated after ROSC is achieved. If an acute coronary syndrome is suspected, and ROSC has not been achieved, consider urgent coronary angiography when feasible and, if required, percutaneous coronary intervention. Mechanical chest compression devices and extracorporeal CPR can help facilitate this (see below).

The commonest cause of thromboembolic or mechanical circulatory obstruction is massive pulmonary embolism. If pulmonary embolism is thought to be the cause of cardiac arrest consider giving a fibrinolytic drug immediately. Following fibrinolysis during CPR for acute pulmonary embolism, survival and good neurological outcome have been reported, even in cases requiring in excess of 60 min of CPR. If a fibrinolytic drug is given in these circumstances, consider performing CPR for at least 60–90 min before termination of resuscitation attempts. In some settings extracorporeal CPR, and/or surgical or mechanical thrombectomy can also be used to treat pulmonary embolism.

A **tension pneumothorax** can be the primary cause of PEA and may be associated with trauma. The diagnosis is made clinically or by ultrasound. Decompress rapidly by thoracostomy or needle thoracocentesis, and then insert a chest drain.

Cardiac **tamponade** is difficult to diagnose because the typical signs of distended neck veins and hypotension are usually obscured by the arrest itself. Cardiac arrest after penetrating chest trauma is highly suggestive of tamponade and is an indication for resuscitative thoracotomy. The use of ultrasound will make the diagnosis of cardiac tamponade much more reliable.

In the absence of a specific history, the accidental or deliberate ingestion of therapeutic or **toxic** substances may be revealed only by laboratory investigations. Where available, the appropriate antidotes should be used, but most often treatment is supportive and standard ALS protocols should be followed.

Symptoms and signs. In critically or terminally ill patients, cardiac arrest is often preceded by a period of clinical deterioration with rapid, shallow breathing, arterial hypotension, and a progressive decrease in mental alertness. In sudden cardiac arrest, collapse occurs without warning, occasionally accompanied by a brief (< 5 seconds) seizure.

Diagnosis. Diagnosis of cardiac arrest is by clinical findings of apnea (1), pulselessness (2), and unconsciousness (3) **(TRIAD)**. Arterial pressure is not measurable. Pupils dilate and become unreactive to light after several minutes. The following may also be seen:
(a) occasional, ineffectual (agonal) gasps
(b) pallor or cyanosis
(c) dilated pupils
(d) brief tonic grand mal seizure.

A cardiac monitor should be applied; it may indicate ventricular fibrillation (VF), ventricular tachycardia (VT), or asystole. Sometimes a perfusing rhythm (eg, extreme bradycardia) is present; this rhythm may represent true pulseless electrical activity (PEA, or electromechanical dissociation) or extreme hypotension with failure to detect a pulse.

Diagnosis of cardiac arrest must be carried out as quickly as possible (For no more **10 seconds**) for the immediate start of resuscitation, because if the critical period of 3-5 min of clinical death is overlooked, an irreversible damage to the brain begins.

Prognosis. Survival to hospital discharge, particularly neurologically intact survival, is a more meaningful outcome than simply return of spontaneous circulation.

Survival rates vary significantly; favorable factors include

- Early and effective bystander-initiated CPR
- Witnessed arrest

- In-hospital location (particularly a monitored unit)
- Initial rhythm of VF or VT
- Early defibrillation (of VT or VF after initial chest compression)
- Postresuscitative care, including circulatory support and access to cardiac catheterization
- In adults, targeted temperature management (body temperature of 32 to 36°C) and avoidance of hyperthermia

If many factors are favorable (eg, VF is witnessed in an ICU or emergency department), about 50% of adults survive to hospital discharge. Overall, in-hospital arrest (VT/VF and asystole/PEA) survival is about 25%.

When factors are uniformly unfavorable (eg, patient in asystole after unwitnessed, out-of-hospital arrest), survival is unlikely. Overall, reported survival after out-of-hospital arrest is about 10%.

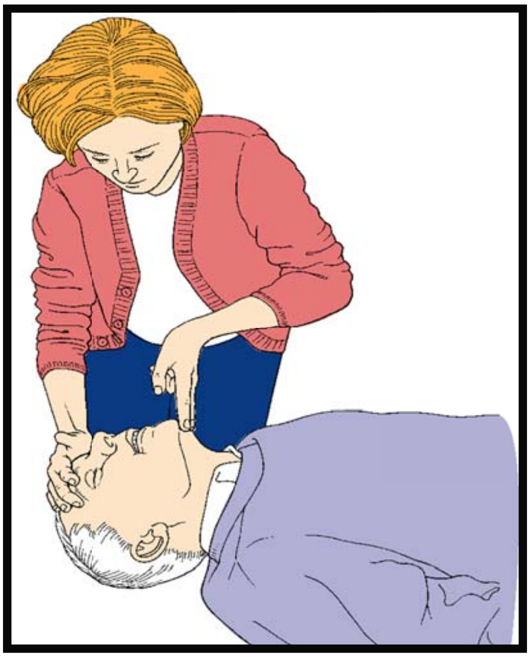
Only about 10% of all cardiac arrest survivors have good central nervous system function at hospital discharge.

CARDIO-PULMONARY RESUSCITATION

Stages of CPR. Safar divided the whole CPR complex into 3 stages:

Stage I. Basic Life Support (BLS)
Stage II. Advanced Life Support (ALS)
Stage III. Post-Resuscitation Life Support (PRLS)

How to recognise cardiac arrest. Start CPR in any unresponsive person with absent or abnormal breathing. Slow, laboured breathing (agonal breathing) should be considered a sign of cardiac arrest. A short period of seizure-like movements can occur at the start of cardiac arrest. Assess the person after the seizure has stopped: if unresponsive and with absent or abnormal breathing, start CPR.



I. BASIC LIFE SUPPORT

Basic Life Support (BLS) refers to the care healthcare workers and public safety professionals provide to patients who are experiencing respiratory arrest, cardiac arrest or airway obstruction. As emergencies might happens anywhere, even in hospitals, it is therefore important for the first responder to have the knowledge and skill to perform BLS.

The BLS algorithm is presented in Fig. 1 and step-by-step instructions are provided in Fig. 2.

Airway

Open the airway using the head tilt and chin lift technique whilst assessing whether the person is breathing normally. Do not delay assessment by checking for obstructions in the airway. The jaw thrust and finger sweep are not recommended for the lay provider.

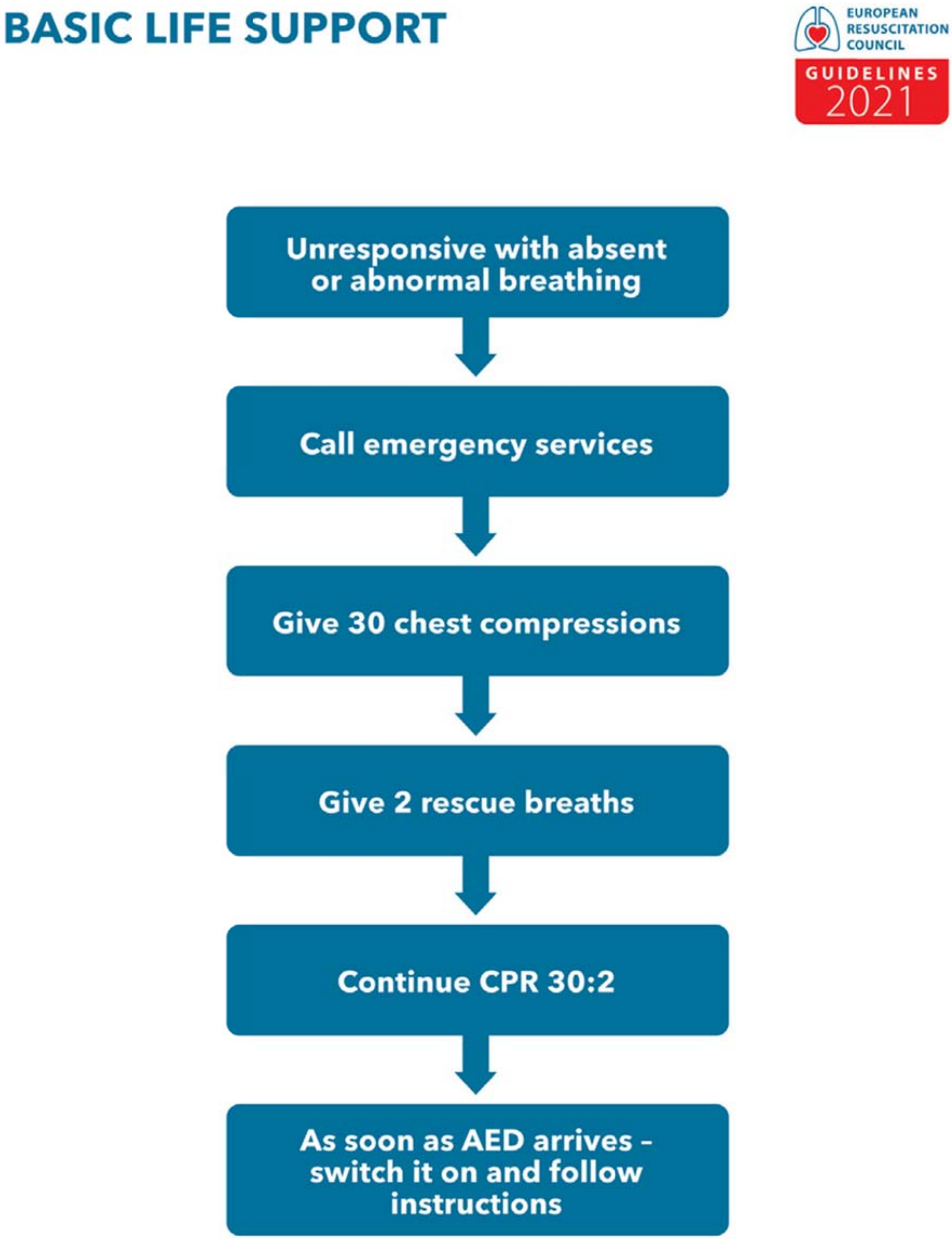


Fig. 1. BLS algorithm

SEQUENCE	TECHNICAL DESCRIPTION
SAFETY	***Make sure you, the victim and any bystanders are safe***
RESPONSE	***Check the victim for a response*** - Gently shake his shoulders and ask loudly: "Are you all right?" - If he responds leave him in the position in which you find him, provided there is no further danger; try to find out what is wrong with him and get help if needed; reassess him regularly
AIRWAY	***Open the airway*** - Turn the victim onto his back - Place your hand on his forehead and gently tilt his head back; with your fingertips under the point of the victim's chin, lift the chin to open the airway
BREATHING	***Look, listen and feel for normal breathing for no more than 10 seconds*** - A victim who is barely breathing, or taking infrequent, slow and noisy gasps. Do not confuse this with normal breathing, is **NOT** breathing **normally**
ABSENT OR ABNORMAL BREATHING	***Alert emergency services.*** - If breathing is absent or abnormal, ask a helper to call the emergency services or call them yourself - Stay with the victim if possible - Activate the speaker function or hands-free option on the telephone so that you can start CPR whilst talking to the dispatcher
SEND FOR AED	***Send someone to get an AED if available*** - If you are on your own, **do not leave** the victim, but start CPR
CIRCULATION	***Start chest compressions*** - Kneel by the side of the victim - Place the heel of one hand in the centre of the victim's chest; (which is the lower half of the victim's breastbone (sternum)) - Place the heel of your other hand on top of the first hand - Interlock the fingers of your hands and ensure that pressure is not applied over the victim's ribs - Keep your arms straight - Do not apply any pressure over the upper abdomen or the bottom end of the bony sternum (breastbone) - Position your shoulders vertically above the victim's chest and press down on the sternum at least 5 cm (but no more than 6 cm) - After each compression, release all the pressure on the chest without losing contact between your hands and the sternum; - Repeat at a rate of 100–120 min^{-1}

COMBINE RESCUE BREATHING WITH CHEST COMPRESSIONS	***If you are traned to do so, after 30 compressions, open the airway again, using head tilt and chin lift*** - Pinch the soft part of the nose closed, using the index finger and thumb of your hand on the forehead - Allow the victim's mouth to open, but maintain chin lift - Take a normal breath and place your lips around the victim's mouth, making sure that you have an airtight seal - Blow steadily into the mouth while watching for the chest to rise, taking about 1 second as in normal breathing; this is an effective rescue breath - Maintaining head tilt and chin lift, take your mouth away from the victim and watch for the chest to fall as air comes out - Take another normal breath and blow into the victim's mouth once more to achieve a total of two effective rescue breaths. - Do not interrupt compressions by more than 10 seconds to deliver two breaths even if one or both are not effective. - Then return your hands without delay to the correct position on the sternum and give a further 30 chest compressions - Continue with chest compressions and rescue breaths in a ratio of 30:2
COMPRESSION-ONLY CPR	***If you are untrained or unable to give rescue breathes,*** give chest-compression-only CPR (continuous compressions at a rate of 100-120 min^{-1})
WHEN AED ARRIVES	***Switch on the AED and attach the electrode pads on the victim's bare chest*** - If more than one rescuer is present, CPR should be continued while electrode pads are being attached to the chest
FOLLOW THE SPOKEN/VISUAL DIRECTIONS	- Follow the spoken and visual directions given by the AED - ***If a shock is advised,*** ensure that neither you nor anyone else is touching the victim - Push shock button as directed (fully automatic AEDs will deliver the shock automatically) - Immediately restart CPR at a ratio of 30:2 - Continue as directed by the voice/visual prompts - ***If no shock is advised,*** immediately resume CPR and ***c***ontinue as directed by AED
IF NO AED IS AVAILANLE	***- If no AED is availanle, OR whilst waiting for one to arrive, continue CPR*** ***- Do not interrupt resuscitation until:*** • A health professional tells you to stop • The victim is definitely waking up, moving, opening eyes and breathing normally • You become exhausted - It is rare for CPR alone to restart the heart. Unless you are certain the person has recovered continue CPR

	- Signs that the victim has recovered • Waking-up • Moving • Opening eyes • Breathing normally
IF UNRESPONSIVE BUT BREATHING NORMALLY	***- If you are certain the victim is breathing normally but is still unresponsive, place in the recovery position*** - Remove the victim's glasses, if worn - Kneel beside the victim and make sure that both his legs are straight - Place the arm nearest to you out at right angles to his body, elbow bent with the hand palm-up - Bring the far arm across the chest, and hold the back of the hand against the victim's cheek nearest to you - With your other hand, grasp the far leg just above the knee and pull it up, keeping the foot on the ground - Keeping his hand pressed against his cheek, pull on the far leg to roll the victim towards you on to his side - Adjust the upper leg so that both the hip and knee are bent at right angles - Tilt the head back to make sure that the airway remains open - If necessary, adjust the hand under the cheek to keep the head tilted and facing downwards to allow liquid material to drain from the mouth - Check breathing regularly Be prepared to restart CPR immediately if the victim deteriorates or stops breathing normally

Fig. 2. BLS steps-by-step instructions

Breathing.

- Alternate between providing 30 compressions and 2 rescue breaths.
- If you are unable to provide ventilations, give continuous chest compressions.

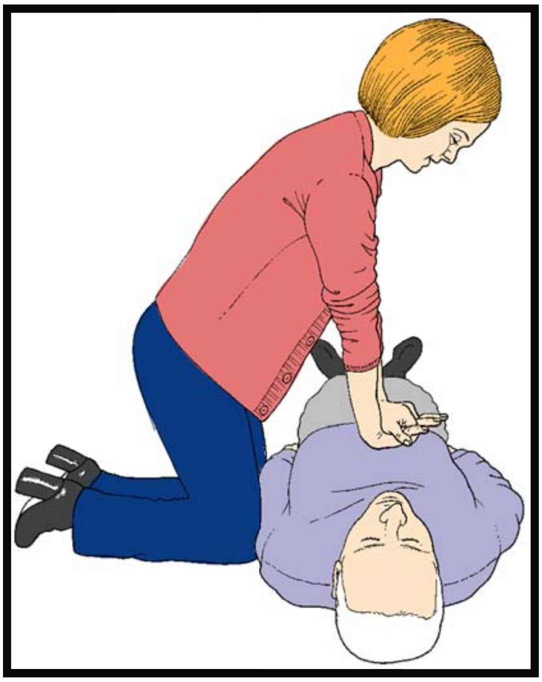
CPR providers should give rescue breaths with an inflation duration of 1 second and provide sufficient volume to make the victim's chest rise. Avoid rapid or forceful breaths. The maximum interruption in chest compression to give two breaths should not exceed 10 seconds.

Mouth-to-nose ventilation

Mouth-to-nose ventilation is an acceptable alternative to mouth-to-mouth ventilation. It may be considered if the victim's mouth is seriously injured or cannot be opened, the CPR provider is assisting a victim in the water, or a mouth-to-mouth seal is difficult to achieve.

Mouth-to-tracheostomy ventilation

Mouth-to-tracheostomy ventilation may be used for a victim with a tracheostomy tube or tracheal stoma who requires rescue breathing.



Barrier devices for use with rescue breaths

Barrier devices decrease transmission of bacteria during rescue breathing in controlled laboratory settings. Their effectiveness in clinical practice is unknown.

If a barrier device is used, care should be taken to avoid unnecessary interruptions in CPR. Manikin studies indicate that the quality of CPR is improved when a pocket mask is used, compared to a bag-mask or simple face shield during basic life support.

Circulation

In adults needing CPR, there is a high probability of a primary cardiac cause for their cardiac arrest. When blood flow stops after cardiac arrest, the blood in the lungs and arterial system remains oxygenated for some minutes. To emphasise the priority of chest compressions, start CPR with chest compressions rather than initial ventilations.

High quality chest compressions.

- Start chest compressions as soon as possible.
- Deliver compressions on the lower half of the sternum (‘in the centre of the chest’).
- Compress to a depth of at least 5 cm but not more than 6 cm.
- Compress the chest at a rate of 100 - 120 min^{-1} with as few interruptions as possible.
- Allow the chest to recoil completely after each compression; do not lean on the chest.
- Perform chest compressions on a firm surface whenever feasible.

AED

AEDs are safe and effective when used by laypeople, including if they have had minimal or no training. AEDs may make it possible to defibrillate many minutes before professional help arrives. CPR providers should continue CPR with minimal interruption to chest compressions both while attaching an AED and during its use. CPR providers should concentrate on following the voice prompts, particularly when instructed to resume CPR, and minimising interruptions in chest compression.

When and how to use an AED

- As soon as the AED arrives, or if one is already available at the site of the cardiac arrest, switch it on.
- Attach the electrode pads to the victim's bare chest according to the position shown on the AED or on the pads.
- If more than one rescuer is present, continue CPR whilst the pads are being attached.
- Follow the spoken (and/or visual) prompts from the AED.
- Ensure that nobody is touching the victim whilst the AED is analysing the heart rhythm.
- If a shock is indicated, ensure that nobody is touching the victim. Push the shock button as prompted. Immediately restart CPR with 30 compressions.
- If no shock is indicated, immediately restart CPR with 30 compressions.
- In either case, continue with CPR as prompted by the AED. There will be a period of CPR (commonly 2 min) before the AED prompts for a further pause in CPR for rhythm analysis.

II. ADVANCED LIFE SUPPORT

Advanced life support (ALS) includes advanced interventions after basic life support has started and when appropriate an automated external defibrillator (AED) has been used (Fig. 3). The transition between basic and advanced life support should be seamless as BLS will continue during and overlap with ALS interventions.

The division between basic life support and advanced life support is arbitrary – the resuscitation process is a continuum.

Top messages of ALS 2021:

1. High quality chest compressions with minimal interruption and early defibrillation, and treatment of reversible causes remain the priority.
2. Premonitory signs and symptoms often occur before cardiac arrest is preventable in many patients.
3. Use basic or advanced airway technique – only rescuers with a high success rate should use tracheal intubation.
4. Use adrenaline early for non-shockable cardiac arrest.
5. In select patients, if feasible, consider extracorporeal CPR (eCPR) as a rescue therapy when conventional ALS is failing.

Heart rhythms associated with cardiac arrest are **divided into two groups**: *shockable rhythms* (ventricular fibrillation/pulseless ventricular tachycardia (VF/pVT)) and *non-shockable rhythms* (asystole and pulseless electrical activity (PEA)). The main difference in the treatment of these two groups is the need for attempted defibrillation in patients with VF/pVT.

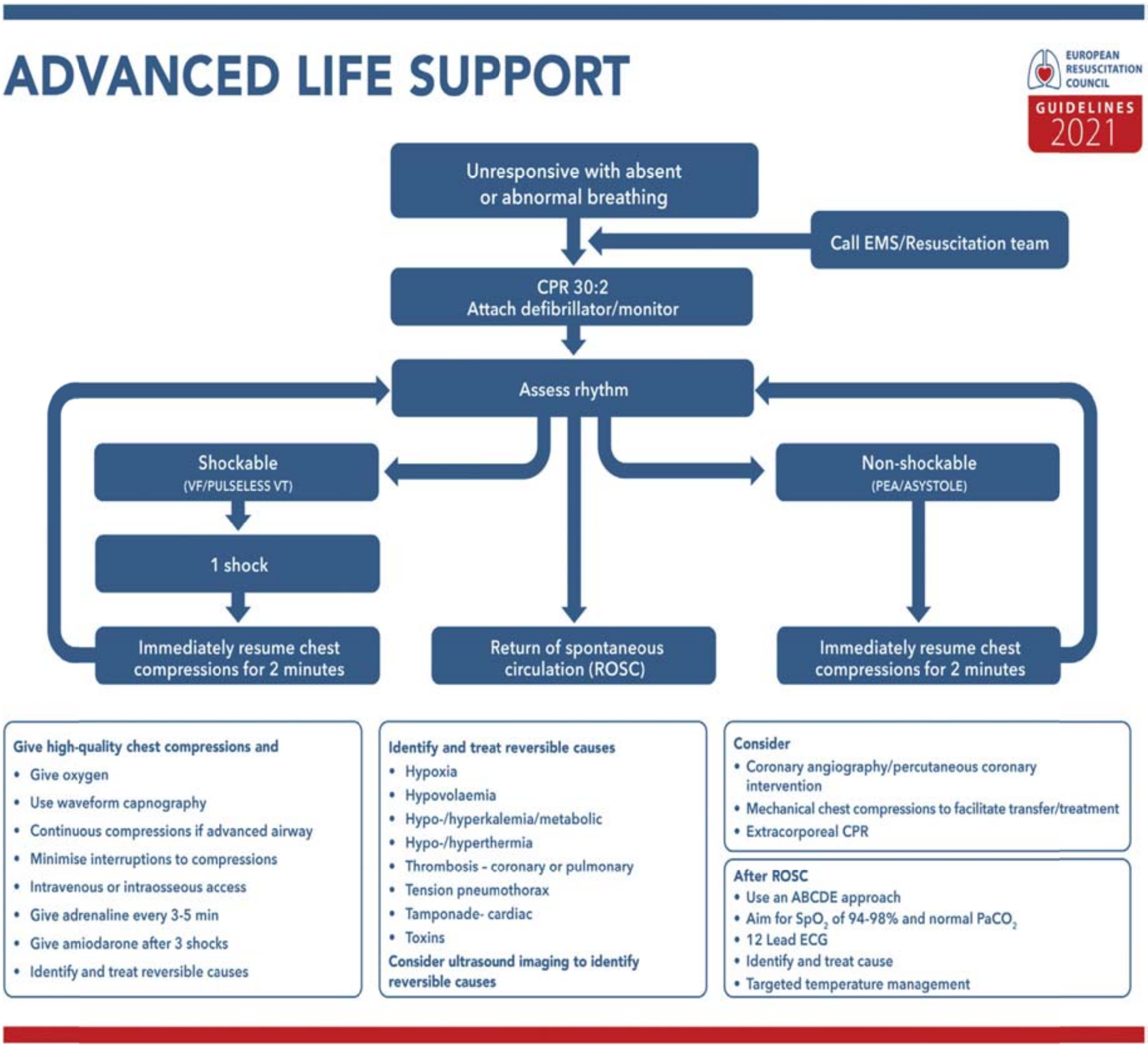


Fig. 3. ALS algorithm

Other actions, including chest compression, airway management and ventilation, vascular access, administration of adrenaline, and the identification and correction of reversible factors, are common to both groups. The ALS algorithm provides a standardised approach to the management of adult patients in cardiac arrest.

The first monitored rhythm is VF/pVT in approximately 20% of both in-hospital and out-of-hospital cardiac arrests. Ventricular fibrillation/pulseless ventricular tachycardia will also occur at some stage during resuscitation in about 25% of cardiac arrests with an initial documented rhythm of asystole or PEA.

Pulseless electrical activity (PEA) is defined as cardiac arrest in the presence of electrical activity (other than ventricular tachyarrhythmia) that would normally be associated with a palpable pulse. These patients often have some mechanical myocardial contractions, but these are too weak to produce a detectable pulse or blood pressure – this is sometimes described as ‘pseudo-PEA’ (see below). PEA can be caused by reversible conditions that can be treated if they are identified and corrected. Survival following cardiac arrest with asystole or PEA is unlikely unless a reversible cause can be found and treated effectively.

Airway management and ventilation

Airway and ventilation. During CPR, start with basic airway techniques and progress stepwise according to the skills of the rescuer until effective ventilation is achieved. If an advanced airway is required, rescuers with a high tracheal intubation success rate should use tracheal intubation. The expert consensus is that a high success rate is over 95% within two attempts at intubation. Aim for less than a 5 s interruption in chest compression for tracheal intubation. Use direct or video laryngoscopy for tracheal intubation according to local protocols and rescuer experience. Use waveform capnography to confirm tracheal tube position. Give the highest feasible inspired oxygen during CPR. Give each breath over 1 s to achieve a visible chest rise. Once a tracheal tube or a supraglottic airway (SGA) has been inserted, ventilate the lungs at a rate of 10 min^{-1} and continue chest compressions without pausing during ventilations. With a SGA, if gas leakage results in inadequate ventilation, pause compressions for ventilation using a compression-ventilation ratio of 30:2.

Upper airway obstruction relieved by head tilt, chin lift, or jaw thrust. In infants and children, a simple suctioning of the airway will help with the clearance. The bulb syringe or any other mechanical suction device can clear the airway. Suction must not last for over 10 seconds.

Advanced Airway are supraglottic devices (laryngeal mask airway, laryngeal tube, esophageal-tracheal) and endotracheal tube. They come in different sizes. This facilitates the rapid transportation to the hospital. Surgical Airway are cricothyrotomy and tracheostomy. If it is impossible to ventilate an apnoeic patient with a bag-mask, or to pass a tracheal tube or alternative airway device, delivery of oxygen through a cannula or surgical cricothyroidotomy may be life saving. A tracheostomy is contraindicated in an emergency, as it is time consuming, hazardous and requires considerable surgical skill and equipment.

It is first imperative to understand some basic terms to understand mechanical ventilation.

- Ventilation: Exchange of air between the lungs and the air (ambient or delivered by a ventilator), in other words, it is the process of moving air in and out of the lungs. Its most important effect is the removal of carbon dioxide (CO2) from the body, not on increasing blood oxygen content. Ventilation is measured as minute ventilation in the clinical setting, and it is calculated as respiratory rate (RR) times tidal volume (Vt). In a mechanically ventilated patient, the CO2 content of the blood can be modified by changing the tidal volume or the respiratory rate.
- Oxygenation: Interventions that provide greater oxygen supply to the lungs, thus the circulation. In a mechanically ventilated patient, this can be achieved by increasing the fraction of inspired oxygen (FiO 2%) or the positive end-expiratory pressure (PEEP).
- FiO2: Percentage of oxygen in the air mixture that is delivered to the patient. During CPR, give the maximal feasible inspired oxygen concentration.

The options for airway management and ventilation during CPR vary according to patient factors, the phase of the resuscitation attempt (during CPR, after ROSC), and the skills of rescuers.[55] They include: no airway and no ventilation (compression-only CPR), compression-only CPR with the airway held open (with or without supplementary oxygen), mouth-to-mouth breaths, mouth-to-mask, bag-mask ventilation with simple airway adjuncts, supraglottic airways (SGAs), and tracheal intubation (inserted with the aid of direct laryngoscopy or videolaryngoscopy, or via a SGA).

Provide artificial ventilation as soon as possible in any patient in whom spontaneous ventilation is inadequate or absent. Expired air ventilation (rescue breathing) is effective but the rescuer’s expired oxygen concentration is only 16–17%, so it must be replaced as soon as possible by ventilation with oxygen-enriched air. A pocket resuscitation mask enables mouth-to-mask ventilation and some enable supplemental oxygen to be given. Use a two-hand technique to maximise the seal with the patient’s face. A self-inflating bag can be connected to a face mask, tracheal tube, or SGA. The two-person technique for bag-mask ventilation is preferable. Deliver each breath over approximately 1 second and give a volume that corresponds to normal chest movement; this represents a compromise between giving an adequate volume, minimising the risk of gastric inflation, and allowing adequate time for chest compression.

Assisting the Circulation

Defibrillation strategy. Continue CPR while a defibrillator is retrieved and pads applied. Give a shock as early as possible when appropriate. Deliver shocks with minimal interruption to chest compression, and minimise the pre-shock and post-shock pause. This is achieved by continuing chest compressions during defibrillator charging, deliver- ing defibrillation with an interruption in chest compressions of less than 5 s and then immediately resuming chest compressions. Immediately resume chest compressions after shock delivery. If there is a combination of clinical and physiological signs of return of spontaneous circulation (ROSC) such as waking, purposeful movement, arterial waveform or a sharp rise in end-tidal carbon dioxide (ETCO2), consider stopping chest compressions for rhythm analysis, and if appropriate a pulse check.

Use single shocks where indicated, followed by a 2 min cycle of chest compressions. The use of up to three-stacked shocks may be considered only if initial ventricular fibrillation/pulseless ventricular tachycardia (VF/pVT) occurs during a witnessed, monitored cardiac arrest with a defibrillator immediately available e.g. during cardiac catheter-isation or in a high dependency area. Defibrillation shock energy levels are: - for biphasic waveforms (rectilinear biphasic or biphasic truncated exponential), deliver the first shock with an energy of at least 150 J; - for pulsed biphasic waveforms, deliver the first shock at 120-150 J. If the rescuer is unaware of the recommended energy settings of the defibrillator, for an adult use the highest energy setting for all shocks.

Vascular access. Consistent with ILCOR, the ERC suggests attempting intra vascular (IV) access first to enable drug delivery in adults in cardiac arrest. Intra osseus (IO) access may be considered if unable to obtain IV access in adults in cardiac arrest.

Vasopressors. The ERC recommends adrenaline 1 mg IV (IO) is administered as soon as possible for adult patients in cardiac arrest with a non-shockable rhythm. For patients with a shockable rhythm persisting after 3 initial shocks, give adrenaline 1 mg IV (IO). Repeat adrenaline 1 mg IV (IO) every 3-5 min whilst ALS continues. The ERC does not support the use of vasopressin during cardiac arrest.

Antiarrhythmic drugs. The ERC recommend that amiodarone should be given after three defibrillation attempts, irrespective of whether they are consecutive shocks, or interrupted by CPR, or for recurrent VF/pVT during cardiac arrest. The initial recommended dose is amiodarone 300 mg; a further dose of 150 mg may be given after five defibrillation attempts. Lidocaine 100 mg may be used as an alternative if amiodarone is not available, or a local decision has been made to use lidocaine instead of amiodarone. An additional bolus of lidocaine 50 mg can also be given after five defibrillation attempts.

Thrombolytic therapy. The ERC does not support the routine use of thrombolytic drugs in cardiac arrest, unless the cause is suspected or confirmed PE.

Fluid therapy. The ERC maintains its recommendation to avoid the routine infusion of large volume fluids in the absence of evidence of suspicion of a hypovolaemic cause of the cardiac arrest.

Atropine. Several recent studies have failed to demonstrate any benefit from atropine in out-of-hospital or in-hospital cardiac arrests and its routine use for asystole or PEA is no longer recommended.

Magnesium. Although the benefits of giving magnesium in known hypomagnesaemic states are recognized, the benefit of giving magnesium routinely during cardiac arrest is unproven. Give an initial i.v. dose of 2 g (= 8 mmol or 4 mL of 50% magnesium sulphate) for refractory VF if there is any suspicion of hypomagnesaemia (e.g. patients on potassium-losing diuretics); it may be repeated after 10–15 min. Other indications are: ventricular tachyarrhythmias in the presence of possible hypomagnesaemia; torsade de pointes VT; digoxin toxicity.

Bicarbonate. Cardiac arrest causes a combined respiratory and metabolic acidosis because pulmonary gas exchange ceases and cellular metabolism becomes anaerobic. The best treatment of acidaemia in cardiac arrest is chest compression; some additional benefit is gained by ventilation. During cardiac arrest, arterial blood gas values may be misleading and bear little relationship to the tissue acid–base state – analysis of central venous blood may provide a better estimation of tissue pH. Giving sodium bicarbonate routinely during cardiac arrest and CPR, or after ROSC, is not recommended. Give sodium bicarbonate 50 mmol if cardiac arrest is associated with hyperkalaemia or tricyclic antidepressant overdose. Repeat the dose according to the clinical condition of the patient and the results of repeated blood gas analysis.

Calcium. There are no data supporting any beneficial action for calcium after most cases of cardiac arrest. High plasma concentrations achieved after injection may be harmful to the ischaemic myocardium and may impair cerebral recovery. Give calcium during resuscitation only when indicated specifically (cardiac arrest caused by hyperkalaemia, hypocalcaemia, or overdose of calcium channel blocking drugs).

Mechanical chest compression devices. Consider mechanical chest compressions only if high-quality manual chest compression is not practical or compromises provider safety. When a mechanical chest compression device is used, minimise interruptions to chest compression during device use by using only trained teams familiar with the device.

Extracorporeal CPR. Consider extracorporeal CPR (eCPR) as a rescue therapy for selected patients with cardiac arrest when conventional ALS measures are failing or to facilitate specific interventions (e.g. coronary angiography and percutaneous coronary intervention (PCI), pulmonary thrombectomy for massive pulmonary embo-lism, rewarming after hypothermic cardiac arrest) in settings in which it can be implemented.

Duration of resuscitation attempt. The decision to stop CPR requires clinical judgement and a careful assessment of the likelihood of achieving ROSC. If it was considered appropriate to start resuscitation, it is usually considered worthwhile continuing, as long as the patient remains in VF/pVT, or there is a potentially reversible cause than can be treated. The use of mechanical compression devices and extracorporeal CPR techniques make prolonged attempts at resuscitation feasible in selected patients. It is generally accepted that asystole for more than 20 minutes in the absence of a reversible cause and with ongoing ALS constitutes a reasonable ground for stopping further resuscitation attempts.

III. POST-RESUSCITATION LIFE SUPPORT

Successful return of spontaneous circulation (ROSC) is the first step towards the goal of complete recovery from cardiac arrest. The complex pathophysiological processes that occur following whole body ischaemia during cardiac arrest and the subsequent reperfusion response during CPR and following successful resuscitation have been termed the post-cardiac arrest syndrome. Depending on the cause of the arrest, and the severity of the post-cardiac arrest syndrome, many patients will require multiple organ support and the treatment they receive during this post-resuscitation period influences significantly the overall outcome and particularly the quality of neurological recovery. The post-resuscitation phase starts at the location where ROSC is achieved but, once stabilised, the patient is transferred to the most appropriate high-care area (e.g. emergency room, cardiac catheterisation laboratory or intensive care unit (ICU)) for continued diagnosis, monitoring and treatment. The post-resuscitation care algorithm (Fig. 4) outlines some of the key interventions required to optimise outcome for these patients.

Of those comatose patients admitted to ICUs after cardiac arrest, as many as 40–50% survive to be discharged from hospital depending on the cause of arrest, system and quality of care. Of the patients who survive to hospital discharge, the vast majority have a good neurological outcome although many have subtle cognitive impairment.

The Post-Cardiac-Arrest Syndrome, which comprises post-cardiac-arrest brain injury, post-cardiac-arrest myocardial dysfunction, the systemic ischaemia/ reperfusion response, and persistence of the precipitating pathology, often complicates the post-resuscitation phase.

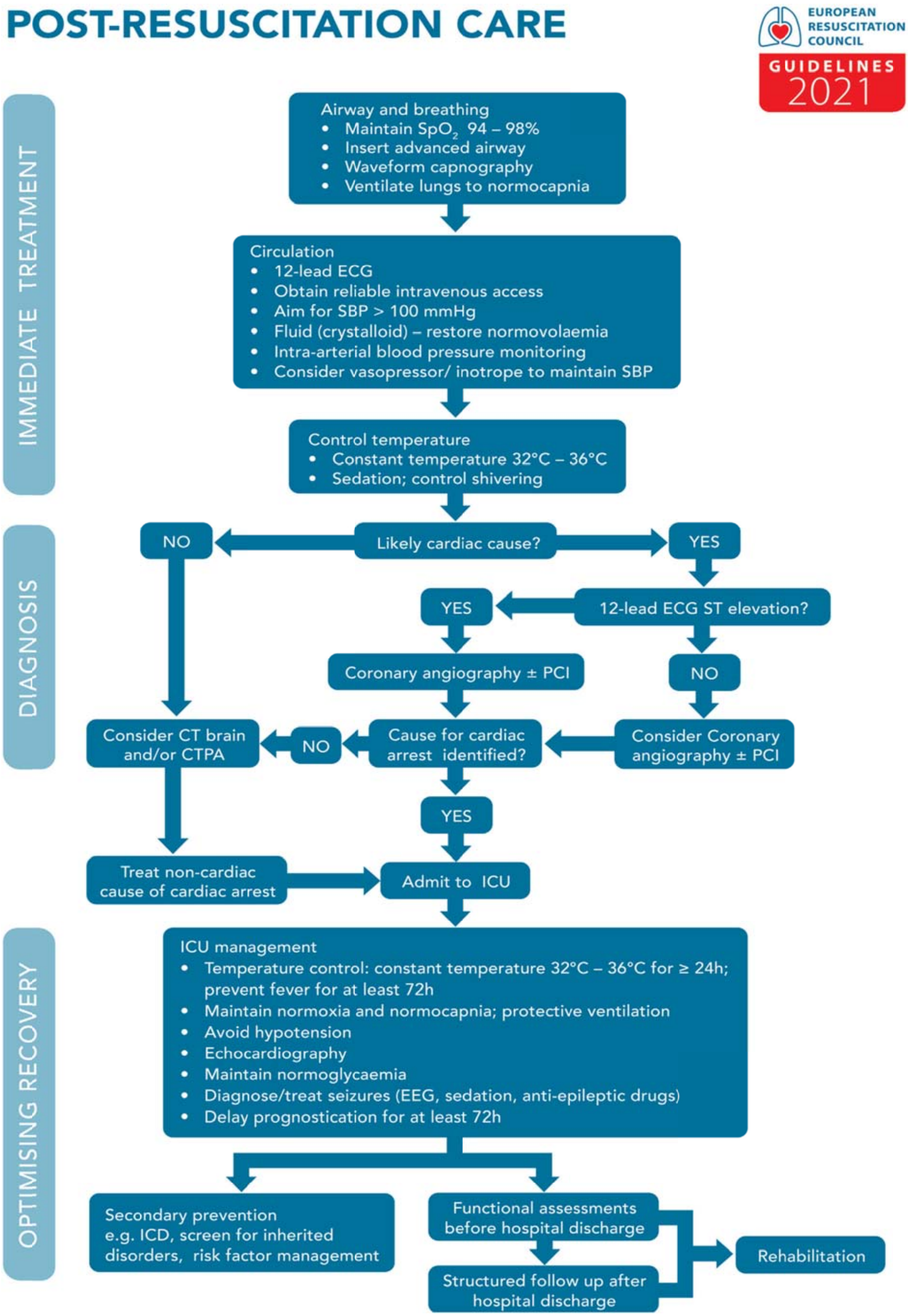


Fig. 4. Post resuscitation care algorithm

Post-cardiac-arrest brain injury manifests as coma, seizures, varying degrees of neurocognitive dysfunction and brain death. Post-cardiac-arrest brain injury may be exacerbated by microcirculatory failure, impaired autoregulation, hypercarbia, hyperoxia, pyrexia, hyperglycaemia and seizures.
Significant myocardial dysfunction is common after cardiac arrest but typically recovers by 2–3 days.

Total systematic ischaemia occurs during cardiac arrest and is only partly reversed by CPR (which achieves a cardiac output of only 25% of normal). After ROSC, myocardial dysfunction and microcirculatory failure will prolong the period of inadequate tissue oxygen delivery. Restoration of tissue oxygenation generates reactive oxygen species and reperfusion injury, causing multiple organ failure and increased risk of infection. This condition resembles sepsis, with high concentrations of inflammatory mediators and activation of coagulation and fibrinolysis. Clinical manifestations of this systemic ischaemic–reperfusion response include intravascular volume depletion, impaired vasoregulation, impaired oxygen delivery and utilization, and increased susceptibility to infection
Post-cardiac arrest care is started immediately after ROSC has been achieved, irrespective of location. An 'ABCDE' (Airway, Breathing, Circulation, Disability, Exposure) systems approach is used to identify and treat physiological abnormalities and organ injury.

Airway and Breathing. Hypoxaemia and hypercarbia both increase the likelihood of a further cardiac arrest and may contribute to secondary brain injury. Hyperoxaemia increases the formation of reactive oxygen species and increases post-ischaemic oxidative injury and cellular death. A clinical registry study documented that post-resuscitation hyperoxaemia was associated with worse outcome, compared with both normoxaemia and hypoxaemia. As soon as arterial blood oxygen saturation can be monitored reliably (by blood gas analysis and/or pulseoximetry), titrate the inspired oxygen concentration to maintain the arterial blood oxygen saturation in the range of 94–98%. Consider tracheal intubation, sedation and controlled ventilation in any patient with obtunded cerebral function.

Ventilation is adjusted to achieve normocapnia and monitored using end-tidal carbon dioxide (CO_2) with waveform capnography and ABGs. Avoid hyperventilation, which will cause cerebral vasoconstriction and possible cerebral ischaemia. It seems rational to apply protective lung ventilation: tidal volume 6–8 $mL\ kg^{-1}$ ideal body weight and positive end expiratory pressure 4–8 cm H2O.

Circulation. After cardiac arrest, patients often have haemodynamic instability and arrhythmias associated with reversible myocardial dysfunction. Echocardiography often shows global impairment with both systolic and diastolic dysfunction. Although systemic vascular resistance (SVR) may be high initially, the release of inflammatory cytokines associated with the post-cardiac arrest syndrome (PCAS) causes vasodilation. Treatment with fluids, inotropes, and vasopressors is guided by blood pressure, heart rate, urine output, rate of plasma lactate clearance, central venous oxygen saturations, and cardiac output monitoring (typically non-invasive). Given the possibility of cerebral hypoperfusion in the post-cardiac arrest patient, it is reasonable to maintain mean arterial pressure (MAP) >65 mmHg. Target the mean arterial blood pressure to achieve an adequate urine output (1 mL kg^{-1} h^{-1}) and normal or decreasing plasma lactate values, taking into consideration the patient's usual blood pressure (if known).

Patients who survive a cardiac arrest caused by VF/pVT and who have no evidence of a disease that can be treated effectively (e.g. coronary revascularization) are considered for an implantable cardioverter defibrillator (ICD) before leaving hospital.

Temperature Control.

Treatment of Hyperpyrexia. A period of hyperthermia (hyperpyrexia) is common in the first 48 h after cardiac arrest and this is associated with worse neurological outcome. Treat hyperthermia occurring after cardiac arrest with antipyretics or active cooling.

We recommend targeted temperature management (TTM) for adults. Maintain a target temperature at a constant value between 32 ^{0}C and 36 ^{0}C for at least 24 h. Avoid fever (>37.7 ^{0}C) for at least 72 h after ROSC in patients who remain in coma.

Therapeutic Hypothermia. Animal and human data indicate that mild hypothermia is neuroprotective and improves outcome after a period of global cerebral hypoxia-ischaemia. Cooling suppresses many of the pathways leading to delayed cell death, including apoptosis (programmed cell death). Hypothermia decreases the cerebral metabolic rate for oxygen by about 6% for each 1 °C reduction in temperature and this may reduce the release of excitatory amino acids and free radicals.

All studies of post-cardiac-arrest therapeutic hypothermia have included only patients in coma. There is good evidence supporting the use of induced hypothermia in comatose survivors of out-of-hospital cardiac arrest caused by VF and there is lower level evidence supporting cooling after cardiac arrest from non-shockable rhythms and after in-hospital cardiac arrest. Cooling should be started as soon as possible after ROSC (pre-ROSC cooling is being investigated) with the aim of maintaining temperature in the range of 32–34 °C for 24 h.

The practical application of therapeutic hypothermia is divided into three phases: induction, maintenance, and rewarming. External and/or internal cooling techniques can be used to initiate cooling. An infusion of 30 mL kg^{-}

[1] of 4 °C 0.9% sodium chloride or Hartmann's solution decreases core temperature by approximately 1.5 °C and can easily be started prehospital. Other methods of inducing and/or maintaining hypothermia include: simple ice packs and/or wet towels; cooling blankets or pads; intranasal cooling; water or air circulating blankets; water circulating gel-coated pads; intravascular heat exchanger; and cardiopulmonary bypass.

In the maintenance phase, a cooling method with effective temperature monitoring that avoids temperature fluctuations is preferred. This is best achieved with external or internal cooling devices that include continuous temperature feedback to achieve a set target temperature. Plasma electrolyte concentrations, effective intravascular volume and metabolic rate can change rapidly during both cooling and rewarming. Control rewarming at 0.25–0.5 °C per hour.

General intensive care management.

- Use short acting sedatives and opioids.
- Avoid using a neuromuscular blocking drug routinely in patients undergoing TTM, but it may be considered in case of severe shivering during TTM.
- Provide stress ulcer prophylaxis routinely in cardiac arrest patients.
- Provide deep venous thrombosis prophylaxis.
- Target a blood glucose of 7.8 -10 mmol L^{-1} (140 -180 mg dL^{-1}) using an infusion of insulin if required; avoid hypoglycaemia (<4.0 mmol L^{-1} (<70 mg dL^{-1}).
- Start enteral feeding at low rates (trophic feeding) during TTM and increase after rewarming if indicated. If TTM of 36 ^{0}C is used as the target temperature, gastric feeding rates may be increased early during TTM.
- We do not recommend using prophylactic antibiotics routinely.

EEG electroencephalography; NSE neuron specific enolase; SSEP somatosensory evoked potential; ROSC return of spontaneous circulation

Prognostication. Two thirds of those dying after admission to ICU after out-of-hospital cardiac arrest die from neurological injury. A quarter of those dying after admission to ICU after in-hospital cardiac arrest die from neurological injury. A means of predicting neurological outcome that can be applied to individual comatose patients is required. Many studies have focused on prediction of poor long term outcome (vegetative state or death), based on clinical or test findings that indicate irreversible brain injury, to enable clinicians to limit care or withdraw organ support (Fig. 5). The implications of these prognostic tests are such that they should have 100% specificity or zero false positive rate, i.e. no individuals eventually have a 'good' long-term outcome despite the prediction of a poor outcome.

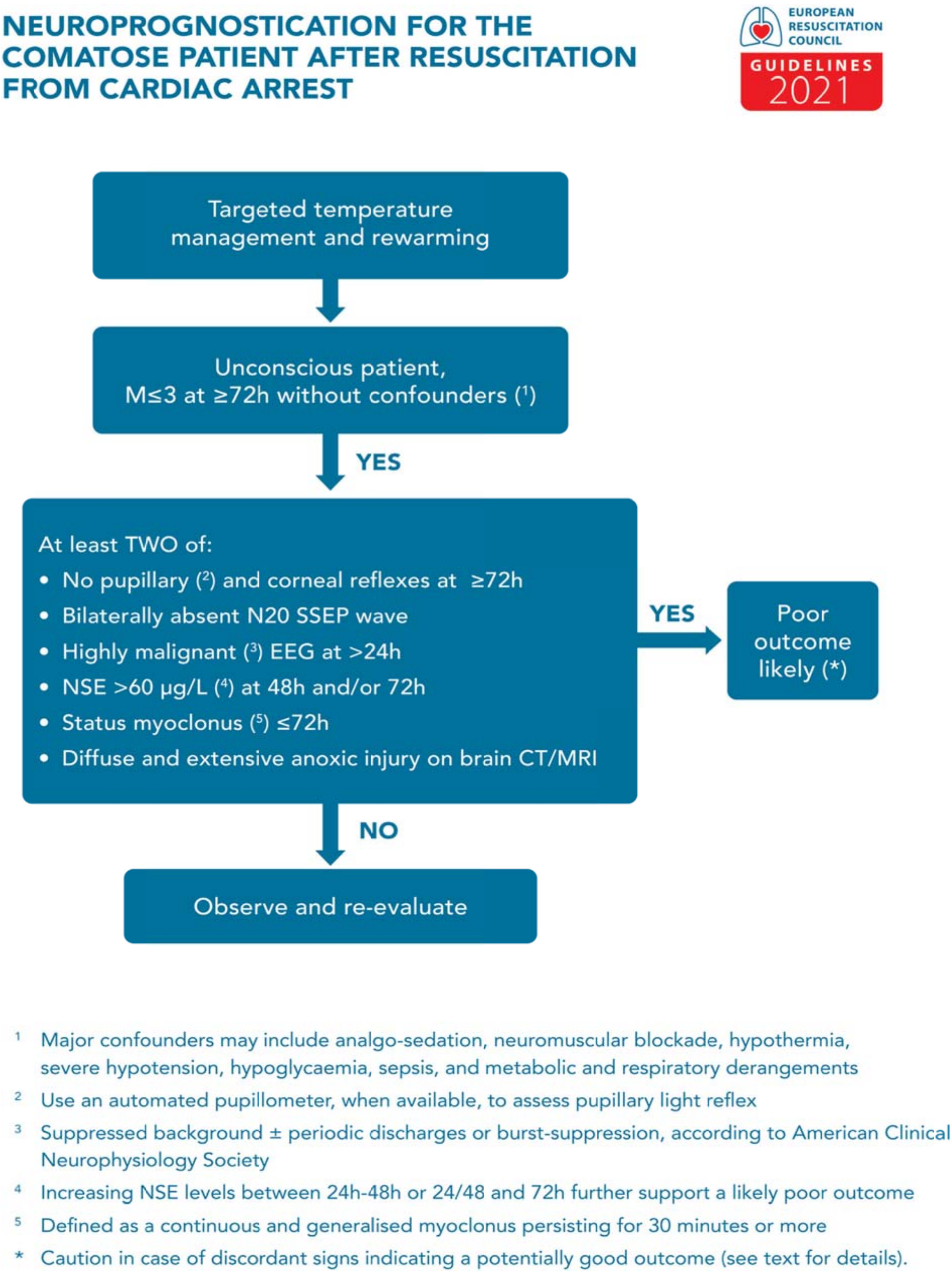


Fig. 5. Prognostication strategy algorithm

CONCLUSION

A successful outcome from cardiac arrest depends on an intact and efficient chain of survival. Immediate activation of the EMS (out-of-hospital) or resuscitation team (in-hospital) is critically important. While BLS will sustain some oxygen delivery to the heart and brain and will help to slow the rate of deterioration in these vital organs, it is important to achieve ROSC as soon as possible. Once ROSC is achieved, the quality of post-cardiac arrest management will influence the patient's final neurological outcome. Many of these patients will develop PCAS, which may result in multiple organ failure. Interventions in this phase aim to restore myocardial function and minimize neurological injury.

TEST QUESTIONS FOR SELF-CONTROL

1. What does CPR stand for?
   a) Chest Pressure Response;
   b) Chest Pre Resuscitation;
   c) Cardiopulmonary Resuscitation;
   d) Cardio Press Resuscitation.

2. What is the correct depth of compressions performed on a child during high quality CPR?
   a) 5 cm;
   b) about 4 cm;
   c) 5-6 cm;
   d) 4-6 cm;
   e) 15 compressions 2 breaths.

3. What does the acronym A.E.D stand for?
   a) Swich rescuers about every 2 min;
   b) Automated External Defibrilation;
   c) it restores regular cardic rhythm;
   d) 15 compressions 2 breaths;
   e) Academy for Educational Development.

4. What would you do if a person is unresponsive?
   a) ABCD;
   b) victim recovers, help arrives, ordered tp stop, scene is unsafe;
   c) call 911, get the first aid kit and AED, check for breathing, start CPR and use an AED.

5. Rescue breaths for an ADULT should be one breath every
   a) when the chest rises;
   b) 5 cm;
   c) 5-6 seconds;
   d) 3-5 seconds.

6. Chest compressions should be started within ________ of recongtion of cardiac arrest
   a) 2 seconds;
   b) 15 seconds;
   c) 10 seconds;
   d) 20 seconds.

7. After you assess a victim's pulse and do not feel one, what is the next step?
   a) Activate the emergency response system;
   b) Start high quality chest compressions at a rate of 100-120/min;
   c) Start high quality chest compressions at a rate of 60-100/min;
   d) Give 2 rescue breaths.

8. How do you know the victim is receiving adequate breaths during CPR?
   a) The victim's chin begins to move forward;
   b) There is no definite way to tell with out medical intervention;
   c) The victim's stomach rises;
   d) The victim's chest rises.

9. During 2 rescuer CPR on an adult victim what is the compression and breath ratio?
   a) 30:1;
   b) 30:2;
   c) 15:2;
   d) 15:1.

10. The preferred way to check for a person for breathing is to:
   a) Put your hand on the chest and see if it moves with any respiration;
   b) Tickle the person in a sensitive spot and see if they respond;
   c) Observe their chest to see if it rises whilst also listening and feeling for air coming from their nose or mouth;
   d) Place your ear to their mouth.

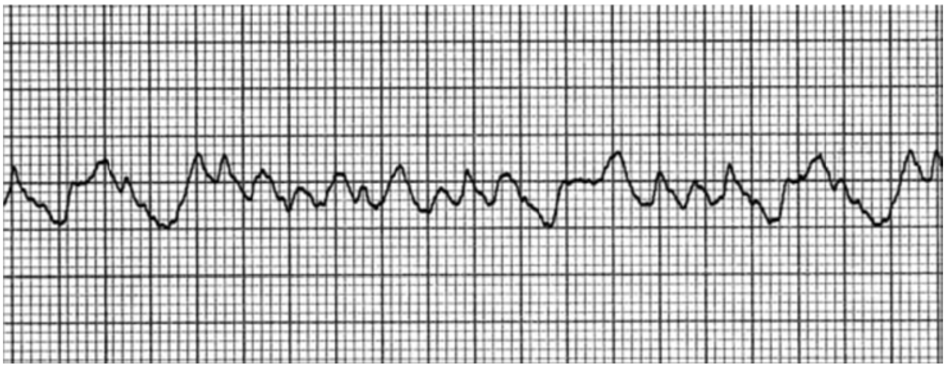
Keys: 1c, 2c, 3b, 4a, 5c, 6c, 7b, 8d, 9b, 10c.

SITUATIONAL TASKS

1. An 80-year-old patient presents with severe chest pain. Heart rate is 30 and blood pressure is 60/P mmHg. The monitor shows sinus bradycardia. Which drug is indicated first?

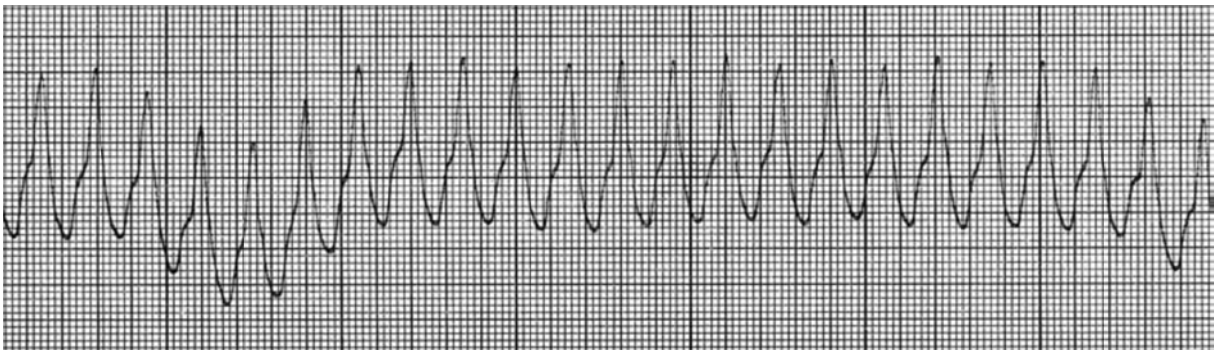
a. lidocaine 75 mg IV bolus
b. isoproterenol infusion at 2–10 mcg/min
c. atropine 0.5–1 mg IV
d. morphine 2–5 mg IV

2. CPR is in progress. Immediately upon diagnosing the following rhythm, what is the treatment?



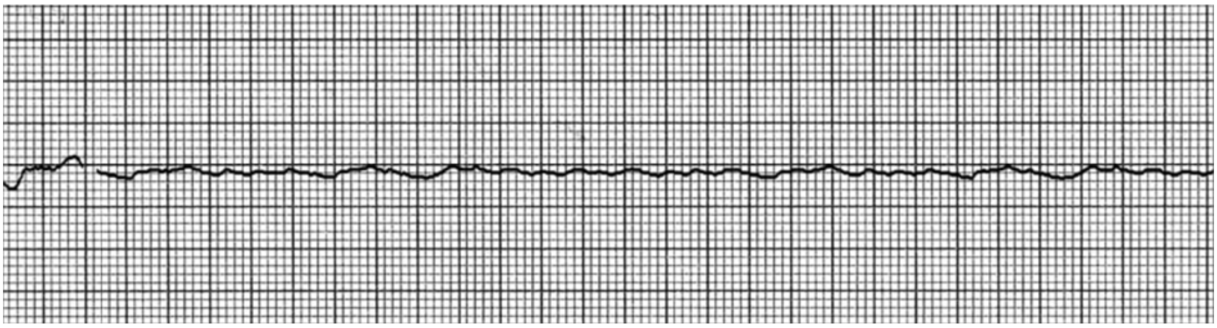
a. administer lidocaine
b. precordial thump
c. administer epinephrine
d. shock with 200 J

3. A patient with a myocardial infarction develops the following rhythm and loses consciousness. The patient is pulseless and not breathing. What is the preferred immediate treatment?



a. administer bretylium
b. administer amiodarone
c. defibrilation
d. administer magnesium

4. Paramedics arrive at the scene and find an unresponsive patient. EMTs have already established an IV and CPR is in progress. There is no pulse. The monitor shows the following rhythm. What should the paramedics do?



a. immediate defibrillation followed by setup of the automatic external defibrillator
b. administer epinephrine, 1 mL
c. administer epinephrine, 10 mL, then defibrillate
d. administer sodium bicarbonate 50 mEq IV bolus

5. A 25-year-old male is evaluated by paramedics for "difficulty breathing." Paramedics find the male not breathing, is pulseless, and apneic. The monitor shows a bradyarrhythmia at 30 beats/min. What should be the first intervention?
a. check the airway
b. start chest compressions
c. defibrillation
d. administer epinephrine, 1 mL

Standards of answers: 1 C, 2 D, 3 C, 4 A, 5 A.

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