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Cardiopulmonary Resuscitation in Special Circumstances

Diana Carmen Cimpoesu and Tudor Ovidiu Popa

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Abstract

Cardiopulmonary resuscitation (CPR) in special circumstances includes the emergency intervention for special causes, special environments and special patients. Special causes cover the potential reversible causes of cardiac arrest that must be identified or excluded during any resuscitation, divided into two groups, 4Hs and 4Ts: hypoxia, hypo-/hyperkalaemia and other electrolyte disorders, hypo-/hyperthermia, hypervolemia, tension pneumothorax, tamponade (cardiac), thrombosis (coronary or pulmonary) and toxins. The special environments section includes recommendations for the treatment of cardiac arrest occurring in specific locations: cardiac surgery, catheterisation laboratory, dialysis unit, dental surgery, commercial airplanes or air ambulances, field of play, difficult environment (e.g. drowning, high altitude, avalanche and electrical injuries) or mass casualty incident. CPR for special patients gives guidance for the patients with severe comorbidities (asthma, heart failure with ventricular assist devices, neurological disease and obesity) and pregnancy women or elderly people.

Keywords: cardiopulmonary resuscitation, special causes, special patients, special environment

1. Introduction

According to the actual guidelines for cardiopulmonary resuscitation (CPR), early recognition and calling for help, early defibrillation, high-quality resuscitation with minimal interruption of chest compressions and treatment of reversible causes are the most important interventions that can improve the outcomes after cardiac arrest [1–4].

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The 2015 Guidelines for resuscitation published by European Resuscitation Council divide the resuscitation in special circumstances into three parts: special causes, special environments and special patients [4].

The first part covers treatment of potentially reversible causes of cardiac arrest, for which specific treatment exists and which are divided into two groups of four, called 'the 4Hs and 4Ts': hypoxia; hypo-/hyperkalaemia and other electrolyte disorders; hypo-/hyperthermia; hypovolaemia; tension pneumothorax; tamponade (cardiac tamponade); thrombosis (coronary and pulmonary) and toxins (poisoning) [1, 4].

The second part covers cardiac arrest in special environments, where universal guidelines should be modified due to specific locations or location-specific causes of cardiac arrest [1, 5].

The third part contains the recommendation for the patients with specific conditions and those with certain long-term comorbidities, where a modified approach and different treatment decisions may be necessary [4].

2. Special causes

Hypoxia is usually a consequence of asphyxia, which is defined as pathological changes caused by lack of oxygen in respired air, resulting in a deficiency of oxygen in the blood (hypoxia) and an increase in carbon dioxide in the blood and tissues (hypercapnia). Symptoms usually include irregular and disturbed respirations, or a complete absence of breathing, and pallor or cyanosis. Asphyxia may occur whenever there is an interruption in the normal exchange of oxygen and carbon dioxide between the lungs and the outside air. Some common causes are drowning, electric shock, hanging, suffocation, lodging of a foreign body in the air passages, inhalation of smoke and poisonous gases, and trauma to or disease of the lungs or air passages. Hypoxia requires ventilation with airway adjuncts that need to be verified to be placed correctly. There is necessary to check breath sounds at regular time intervals to ensure that the endotracheal tube has not slipped out of the trachea or to identify the presence of pneumothorax. Also is necessary to verify the source of oxygen (an oxygen cylinder or the piped oxygen supply).

The effective ventilation with supplementary oxygen during the early moment of resuscitation is essential during CPR. Also, it is recommended to monitor the efficacy of ventilation via capnometry which measures end-tidal CO_2. CPR is indicated to monitor the effectiveness of manoeuvres by obtaining a value between 10 and 20 mmHg. An abrupt increase of end-tidal CO_2 values indicates return of spontaneous circulation (ROSC). If after oro-tracheal intubation, there is no waveform during CPR, but a flat line, this should alert for misplacement of endotracheal tube [1, 4, 6].

Electrolyte abnormalities can cause cardiac arrhythmias or cardiac arrest. Life-threatening arrhythmias are most commonly associated with potassium disorders. The main causes of hyperkalaemia are renal failure, drugs (e.g. angiotensin-converting enzyme inhibitors, angiotensin II receptor antagonists, potassium-sparing diuretics), rhabdomyolysis, metabolic

acidosis, Addison's disease, diet. The treatment strategies for hyperkalaemia are cardiac protection, shifting potassium into cells, removing potassium from the body, monitoring serum potassium and blood glucose [1, 4, 6].

Hypothermia: accidental hypothermia is defined as an involuntary decrease of body core temperature <35°C. It is divided in five stages according to central core temperature: 1. mild hypothermia (conscious, shivering, core temperature 35–32°C); 2. moderate hypothermia (impaired consciousness without shivering, core temperature 32–28°C); 3. severe hypothermia (unconscious, vital signs present, core temperature 28–24°C); 4. cardiac arrest or low flow state (no or minimal vital signs, core temperature <24°C) and 5. death due to irreversible hypothermia (core temperature <13.7°C) [4].

Hypothermia decreases oxygen consumption at cellular level, so the heart and the brain can tolerate for a longer period of time cardiac arrest. This is the basic concept of the protective effect of hypothermia in cardiac arrest. Sometimes complete neurological recovery can be found even after prolonged cardiac arrest, but only if hypothermia was installed before respiratory arrest [1, 4, 6–9].

The first measure is to remove the patient from cold environment, remove clothes which are usually wet or cold and try to prevent any other heat loss. Quick mobilisation of patient with hypothermia can induce arrhythmias [1, 4, 6].

Hypothermia need to be treated with gradual rewarming using normal or electric blankets and warm IV fluids. The goal is to achieve an increase of temperature with a rate of 1–1.5°C per hour. It should be used IV fluid (normal saline, for example) heated at approx. 40°C and also gastric lavage, peritoneal lavage, bladder lavage, using fluids heated at approx. 40°C.

Monitor electrolytes disorder hourly, especially hyperkalaemia, which can appear during heating manoeuvres. Oxygen should be delivered also heated and humidified at 40°C, using a mechanical ventilator, after oro-tracheal intubation [1, 4–6].

Regarding CPR manoeuvres, in case of hypothermia-associated thoracic stiffness is present. Thus, chest compressions are harder to perform, and ventilation will require higher pressures than in normal situation [1, 4, 5, 7–9].

In case of ROSC, pulse in patients with hypothermia is very weak due to peripheral vasoconstriction, so is needed to consider Doppler examination to assess correctly the presence or absence of circulation.

Hypothermic patients without signs of cardiac instability (systolic blood pressure ≥90 mmHg, absence of ventricular arrhythmias or core temperature ≥28°C) can be rewarmed externally using minimally invasive techniques (warm air and warm intravenous fluid). Patients with signs of cardiac instability should be resuscitated in the field and transferred directly to a centre capable of extracorporeal life support (ECLS) [1, 4–9].

Hypovolaemia is a potentially treatable cause of cardiac arrest that usually results from a haemorrhage, but relative hypovolaemia may also occur in patients with severe vasodilation (e.g. anaphylaxis, sepsis). In case of anaphylaxis with relative hypovolaemia, the immediate

treatment with intramuscular adrenaline is the treatment of choice, followed by IV fluids and corticoids prolonged CPR may be necessary [1, 4, 6, 10].

History of fluid or blood loss may be available. Rectal examination can identify massive lower GI bleeding; nasogastric intubation can identify massive upper GI bleeding, and bedside FAST can diagnose massive intraperitoneal bleeding. Treatment is with fluids (crystalloids, colloids), administered rapidly IV and blood products. If colloids are administered, blood samples are necessary before of this, to work compatibility cross-match blood test, because colloids can interfere with the results [1, 4, 6].

Tension pneumothorax: the mortality from traumatic cardiac arrest (TCA) is very high. The most common cause of death is haemorrhage, but the patients with trauma could have other reversible causes: hypoxia, tension pneumothorax, cardiac tamponade, and all of them must be immediately treated [1, 4, 5, 11–14].

The new treatment algorithm for traumatic cardiac arrest was developed to prioritise the sequence of life-saving measures. Chest compressions should not delay the treatment of reversible causes.

Suspect of tension pneumothorax during cardiac resuscitation if breath sounds are unequal on chest auscultation after verifying correct endotracheal tube placement. Other useful clinical sign for diagnosis are one immobile, distended hemi thorax; hyper resonance with percussion over the chest wall; trachea deviation to opposite side of tension pneumothorax and jugular veins distention. Treatment is immediate needle decompression or other technique to decompress the chest in TCA—to perform unilateral or bilateral thoracotomies in the 4th intercostal space. In the presence of positive pressure ventilation, thoracotomies are likely to be more effective than needle thoracentesis and quicker than inserting a chest tube [1, 5, 11–14].

Cardiac tamponade is the underlying cause of approximately 10% of cardiac arrest in trauma. Whereas there are TCA and penetrating trauma to the chest or epigastrium, immediate resuscitative thoracotomy (RT) can be lifesaving. If thoracotomy is not possible, consider ultrasound guided pericardiocentesis to treat cardiac arrest with cardiac tamponade. Cardiac tamponade is best identified during resuscitation by bedside transthoracic ultrasound. This requires brief interruption of chest compressions up to 10 s. Another useful ECG sign in cardiac tamponade is represented by microvoltage, in traumatic event context. Treatment of tamponade causing cardiac arrest is bedside pericardiocentesis [1, 4, 5, 12–14].

Thrombosis for the patients with out-of-hospital cardiac arrest (OHCA) of suspected cardiac origin, the transfer to the hospital with continuing CPR could be a solution in case of acute coronary syndrome–coronary thrombosis. Ground transport may be beneficial in selected patients where there is immediate hospital access to the catheterisation laboratory and an infrastructure providing prehospital and in-hospital teams experienced in mechanical or haemodynamic support and percutaneous coronary intervention (PCI) with ongoing CPR [1, 4, 15–19].

Acute coronary thrombosis or acute myocardial infarction is one of the most common causes of cardiac arrest. Risk factors are a history of coronary artery disease and initial rhythm of VF/VT. Cardiac catheterisation after resuscitation is an underused procedure. A 12-lead ECG in

the immediate postcardiac arrest state can identify an ST-elevation acute myocardial infarction and allows for arrangements of immediate coronary angiography. Myocardial and neurologic function can improve after percutaneous coronary intervention following cardiac arrest. Therefore, after ROSC, especially in the face of post-ROSC, ECG evidence of acute myocardial infarction, cardiac catheterisation and percutaneous coronary revascularisation is recommended, if available and appropriate [1, 4, 5].

Acute pulmonary embolism will be suspected by clinical symptoms such as dyspnoea, chest pain and syncope, either only one of this or in combination. Most frequent patient-related predisposing factors for developing pulmonary embolism include age, history of previous deep vein thrombosis, active cancer, neurological disease with extremity paresis, medical condition causing prolonged bed rest, such as heart or acute respiratory failure or post-surgery, congenital or acquired thrombophilia, hormone replacement therapy and oral contraceptive therapy [1, 4, 5, 15–17].

Electrocardiographic signs of RV strain, such as inversion of T waves in leads V1–V4, a QR pattern in lead V1, the classic pattern of S1Q3T3 ECG type and incomplete or complete right bundle-branch block, may be helpful also to raise the suspicion for pulmonary embolism.

The administration of fibrinolytic when pulmonary embolism is the suspected cause of cardiac arrest remains the actual recommendation. Pulmonary embolism causing cardiac arrest requires fibrinolysis or embolectomy. However, the diagnosis is rarely made at time of collapse, and even then, most systems are not geared to make such prompt diagnosis and initiate the necessary procedures for embolectomy [1, 4, 5, 15, 16, 19].

Fibrinolytic agents could be considered during cardiac arrest from suspected pulmonary embolism on a case-by-case basis. Factors suggestive of pulmonary embolism causing cardiac arrest include two of three signs/symptoms (prearrest respiratory distress, altered mental status or shock); arrest witnessed by a physician or emergency medical technician and PEA as the first or primary arrest rhythm. Ongoing CPR is not a contraindication to fibrinolysis, and after fibrinolytic drug is administered, CPR should be continued for at least 60–90 min before terminating resuscitation attempts [1, 4, 15–18, 20].

Toxics: airway obstruction and respiratory arrest secondary to a decreased conscious level is a common cause of cardiac arrest after accidental or self-poisoning. There are few specific therapeutic measures for poisoning that are useful immediately and during cardiopulmonary resuscitation and improve outcomes: decontamination, enhancing elimination and the use of specific antidotes.

The preferred method of gastrointestinal decontamination in patients with protected airways is activated charcoal but is most effective only if given within first hour from ingestion. Drug overdose is rarely identified as a cause of cardiac arrest during the resuscitation process. In the event of antidepressant overdose, administer IV sodium bicarbonate. Lipid emulsion infusion may be useful in cardiac arrest associated with cyclic antidepressants or local anaesthetics.

Opioid poisoning causes respiratory depression followed by respiratory insufficiency or respiratory arrest. The use of naloxone can prevent the need for intubation. The initial doses of

naloxone are 0.4–2 mg IV, IO, IM or SC and may be repeated every 2–3 min. Additional doses may be needed every 20–60 min. Titrate the dose until the victim is breathing adequately and has protective airway reflexes [4, 5, 21].

3. Special environment

The special environments include cardiac arrest in specific locations: operating theatre, cardiac surgery, catheterisation laboratory, dialysis unit, dental surgery, commercial airplanes or medical helicopters, field of play, outside environment (e.g. drowning, remote area, high altitude, avalanche, lightning strike and electrical injuries) or the scene of a mass casualty incident.

Cardiac arrest following major **cardiac surgery** is relatively common in the immediate postoperative phase. Perioperative cardiac arrest may be caused by the physiological effects of the surgery, bleeding, general anaesthesia (failure of ventilation, medication-related events, complications associated with central venous access, drugs or blood administration, perioperative myocardial infarction) [22–24] or complications relating to pre-existing comorbidities.

The management of perioperative cardiac arrest starts with advanced life support (ALS) algorithm, but with appropriate modifications depending on the cause identified. Key to successful resuscitation is recognition of the need to perform emergency re-sternotomy, especially in the context of tamponade or haemorrhage, where external chest compressions may be ineffective. Re-sternotomy should be performed within 5 min if other interventions have failed.

Cardiac arrest may occur during percutaneous coronary intervention (PCI) for ST-elevation myocardial infarction (STEMI) or non-STEMI, or it may be a complication during angiography as air or thrombus embolism in the coronary artery, artery intima dissection or pericardial tamponade. A defibrillator must be available in the angiography room, and self-adhesive defibrillation pads may already be placed at the beginning of the procedure in high-risk patients.

Cardiac arrest from shockable rhythms (ventricular fibrillation or pulseless ventricular tachycardia) during **cardiac catheterisation** should immediately be treated with up to three stacked shocks before starting chest compressions. Use of mechanical chest compression devices during angiography is recommended to ensure high-quality chest compressions and reduce the radiation of the personnel during angiography with ongoing CPR.

Most of the standard reversible causes (4Hs and 4Ts) apply to **dialysis patients**. Electrolyte disorders, particularly hyperkalaemia and hypoxia, due to fluid overload with pulmonary edema are most common causes of cardiac arrest in dialysis unit.

During cardiopulmonary resuscitation, it follows the universal ALS algorithm, and the dialysis access open for drug administration A shockable rhythm (VF/pulseless VT) is more common in patients undergoing haemodialysis than in the general population [25–29]. So the delay in delivering defibrillation must be minimised.

In **dental surgery**, causes of cardiac arrest are related to pre-existing comorbidities (acute myocardial infarction, grand mal seizures or exacerbation of asthma), loss of airway patency

related to the primary pathology or complications of the procedure (e.g. bleeding, secretions, tissue swelling) or anaphylaxis to local anaesthetics.

The patient will not be moved from the dental chair to start CPR, the dental chair will be reclined into a horizontal position or a stool will be placed under the head to increase its stability during CPR. Consider the over-the-head technique of CPR, if access of the chest is limited [30–32].

Cardiopulmonary resuscitation on the airplane: in case of cardiac arrest, universal algorithm for adult basic life support and automated external defibrillation (AED) will be followed, but performance of CPR is limited in an aircraft due to space restriction, so consider the transfer of the patient to a larger space. Consider an over-the-head technique of CPR if access precludes conventional PR [30–32].

If the CPR equipment is available, attach oxygen to the facemask or self-inflating bag. Request immediate flight diversion to the nearest appropriate airport. The in-flight use of AEDs aboard commercial airplanes can result in up to 50% survival to hospital discharge [4]. AEDs and appropriate CPR equipment should be mandatory on board of all commercial aircraft in Europe, including regional and low-cost carriers [33].

The incidence of cardiac arrest on board of **helicopter emergency medical services** (HEMS) and air ambulances is low. Cardiac arrest may occur in-flight, both in patients being transported from a primary intervention site and also critical patients transferred between hospital [34]. The pre-flight preparation is important for the patients with high risk of cardiac arrest and use of mechanical chest compression devices are emphasised [4, 5].

Sudden and unexpected collapse of a sportsman **during exercises or on the field of play** is likely to be cardiac in origin and requires rapid recognition, initiating basic life support (BLS) and early defibrillation. If the athlete responds to resuscitation, then he/she must be transported immediately to the nearest cardiac centre for further evaluation and treatment [4, 5].

For **drowning patients**, bystanders play an essential role in early rescue and high-quality resuscitation. The victim needs to be removed from the water promptly. Resuscitation strategies for those in respiratory or cardiac arrest continue to prioritise oxygenation and ventilation. Inflation should take about 1 s and be sufficient to see the chest rise [4, 5].

Rescue breaths/ventilation will continue until an ALS team arrives and is ready to intubate the victim. Palpation of the pulse is not always reliable. As soon as possible, use information from monitoring modalities such as the ECG, end-tidal CO_2, and echocardiography to confirm the diagnosis of cardiac arrest. If the drowning victim is hypothermic or hypovolaemic, modify the ALS approach in accordance with the treatment of hypothermia and give IV warm fluid.

The chances of good outcome from cardiac arrest in **difficult terrain or mountains** may be reduced because of delayed access and prolonged transport. There is a recognised role of air rescue and availability of AEDs in remote but often-visited locations [4, 35]. Resuscitation at high altitude does not differ from standard CPR. CPR is more exhausting for a single rescuer than at sea level, due to lower pO_2, and the average number of effective chest compressions may decrease within the first minute [36].

For **avalanche victims** in cardiac arrest, prolonged CPR and extracorporeal rewarming are indicated. Cardiac arrest secondary to avalanche is mainly due to asphyxia associated with trauma and hypothermia. In all cases, extricate the body gently and use spinal precautions. Extracorporeal life support (ECLS) is indicated if the duration of burial is >60 min, core temperature at extrication is <30°C and serum potassium at hospital admission is ≤8 mmol L^{-1} [4, 35, 37].

Safety measures are essential for providing CPR to the victim of an **electrical injury** [4]. Factors influencing the severity of electrical injury include the current type alternating (AC) or direct (DC), voltage, magnitude of energy delivered, resistance to current flow, the area and duration of contact. As with industrial and domestic electric shock, after lightning strikes death is caused by cardiac or respiratory arrest [38–41].

Ensure that any power source is switched off and approach the casualty only if it is safe and start standard BLS and ALS without delay. Airway management may be difficult, and early tracheal intubation is needed if there are electrical burns around the face and neck. Head and spine trauma can occur after electrocution, and the spine immobilisation must be performed.

VF is the commonest initial arrhythmia after high-voltage AC shock, and prompt defibrillation is essential. Asystole is more common after DC shock with standard ALS protocols.

Unlike normal circumstances, CPR is not usually initiated in **mass casualty incidents** (MCI), in order to avoid delaying potentially effective treatment for the critically ill but salvageable victims. This critical decision depends on available medical and paramedical resources in relation to the number of casualties.

A triage system should be used to prioritise treatment and, if the number of casualties overwhelms the prehospital medical resources, withhold CPR for the patients without signs of life [1, 5]. For triage, the START triage is used. The first step is that everyone able to walk is directed to clear the scene, and respiratory status of nonwalking patients is assessed. If the casualty does not breathe, open the airway using head tilt and chin lift or jaw thrust. Assess breathing for no more than 10 s and if a patient does not begin breathing is declared dead. If an unresponsive victim is breathing normally, turn them into the recovery position and label as red-highest priority for treatment. The same goes for the patient with sign of hemodynamic instability.

> Perform life-saving interventions in patients triaged as red (highest priority) to prevent cardiac arrest: control major haemorrhage, open airway using basic techniques, perform chest decompression for tension pneumothorax, use antidotes and consider initial rescue breaths in a nonbreathing child [42].

4. Special patients

Special patients with special guidance for CPR are considered to be the patients with severe comorbidities: asthma, heart failure with ventricular assist devices, neurological disease, obesity and those with specific physiological conditions (pregnancy, elderly people).

Cardiac arrest in a patient with **asthma** is often a terminal event after a hypoxemic period or it may be sudden. CA is linked to:

- severe bronchospasm and mucous plugging leading to asphyxia;

- cardiac arrhythmias caused by hypoxia, by stimulant drugs (beta-adrenergic agonists, aminophylline) or electrolyte abnormalities;

- dynamic hyperinflation, that is, auto positive end-expiratory pressure (auto-PEEP), can occur in mechanically ventilated asthmatics. Gradual build-up of pressure occurs and reduces venous return and blood pressure and determines heart failure;

- tension pneumothorax uni/bilateral.

These high-risk patients should be treated to prevent deterioration with oxygen to achieve an SpO_2 94–98%, inhaled beta-2 agonists (salbutamol 5 mg) or intravenous beta-2 agonists for those patients in whom inhaled therapy cannot be used reliably, nebulised anticholinergics (ipratropium, 0.5 mg 4–6 hourly), nebulised magnesium sulphate, intravenous corticosteroids, intravenous bronchodilators and aminophylline, a dose of 5 mg kg^{-1} over 20–30 min. In cases of severe asthma associated with dehydration and hypovolemia IV fluids are necessary.

In case of cardiac arrest BLS is performed according to standard guidelines. Ventilation could be difficult because of increased airway resistance.

Modifications to standard ALS guidelines include the need for early tracheal intubation. The peak airway pressures recorded during ventilation of patients with severe asthma are significantly higher than the normal lower oesophageal sphincter pressure (approximately 20 cm H_2O) [43, 44].

Respiratory rates of 8–10 breaths/min and a tidal volume required for a normal chest rise during CPR should minimise dynamic hyperinflation of the lungs [air trapping].

Tidal volume depends on inspiratory time and inspiratory flow. Lung emptying depends on expiratory time and expiratory flow. In mechanically ventilated severe asthmatics, increasing the expiratory time (achieved by reducing the respiratory rate) provides only moderate gains in terms of reduced gas trapping when a minute volume of less than 10 L min^{-1} is used [44].

Dynamic hyperinflation increases transthoracic impedance [45] but modern impedance-compensated biphasic defibrillation waveforms are no less effective in patients with higher impedance. Consider increasing defibrillation energy if the first shock is unsuccessful, and a manual defibrillator is available [4].

There is no good evidence for the use of open-chest cardiac compressions in patients with asthma-associated cardiac arrest. Looking through the four H's and four T's will identify potentially reversible causes of asthma-related cardiac arrest, often tension pneumothorax. This pathologic situation may be indicated by unilateral expansion of the chest wall, shifting of the trachea and subcutaneous emphysema. If a pneumothorax is suspected, perform needle decompression using a large-gauge cannula, followed by insertion of a chest tube.

In patients with **ventricular assist devices** (VADs), confirmation of cardiac arrest may be difficult. The management of patients with VADs is more complex, in that a cardiac arrest may be due to mechanical failure and may be actions specific to the device that are required. In any cases, external chest compression in patients with ventricular assist devices is not successful without damage to the VAD.

Transthoracic/transesophageal echocardiography, capnography or Doppler flow in a major artery may assist in the cardiac arrest diagnosis. If cardiac arrest is confirmed, start CPR, check the rhythm and perform defibrillation for shockable rhythms (VF/VT), start pacing for asystole. If during the first 10 days of surgery, cardiac arrest does not respond to defibrillation, perform re-sternotomy immediately.

Cardiac arrest associated with **acute neurological disease** is relatively uncommon and can appear in subarachnoid haemorrhage, intracerebral haemorrhage, epileptic seizures and ischemic stroke and in brain injury associated with trauma [46].

The mechanism of cardiac arrest in neurological disease is related to:

(a) loss of consciousness, causing airway obstruction, hypoxemia and respiratory arrest followed by cardiac arrest, or an increased risk of aspiration of gastric contents into the lungs

(b) respiratory and cardiac depression caused by compression of the brain stem

(c) arrhythmias and myocardial dysfunction associated with acute neurological injury (e.g. sub-arachnoid haemorrhage)

(d) Sudden unexpected death in epilepsy [4].

Patients with subarachnoid haemorrhage may have ECG changes that suggest an acute coronary syndrome. Whether a computed tomography brain scan is done before or after coronary angiography will depend on clinical judgement regarding the likelihood of a subarachnoid haemorrhage versus acute coronary syndrome.

For resuscitation of **obese patients**, in order to maintain sufficient depth of chest compressions (approximately 5 cm but no more than 6 cm), consider changing rescuers more frequently than the standard 2-min interval. Early tracheal intubation by an experienced physician is recommended. Use of mechanical resuscitation devices is limited by the slope of the anterior chest wall and thoracic dimensions.

Optimal defibrillation energy levels in obese patients are unknown [4]. So the recommended energy remains the same (150–360 J). Unlike monophasic defibrillators, modern biphasic defibrillators are impedance compensated and adjust their output according to the patient's impedance. Defibrillation protocols for obese patients should therefore follow those recommended for patients with a normal BMI and consider higher shock energies for defibrillation if initial defibrillation attempts fail.

For the **pregnant woman** in cardiac arrest, high-quality CPR with manual uterine displacement, early ALS and emergent delivery of the foetus if early return of spontaneous circulation (ROSC) is not achieved remain key interventions.

Foetal survival usually depends on maternal survival and initial resuscitation efforts should focus on the pregnant mother.

From 20 weeks' gestation, the uterus can compress the inferior vena cava (IVC) and aorta, impeding venous return and cardiac output and therefore can cause pre-arrest hypotension or shock and, in the critically ill patient, may precipitate cardiac arrest [47, [49]]. During cardiac arrest, the compromise in venous return and cardiac output by the gravid uterus limits the effectiveness of chest compressions. Manually displace the uterus to the left is recommended to reduce IVC compression.

During CPR, the hand position for chest compressions may need to be slightly higher on the sternum for patients with advanced pregnancy—third trimester [48].

During BLS and ALS, pregnant patients are at risk of aspiration and oxygenation and ventilation are the priority over aspiration prevention. Early tracheal intubation (using a tracheal tube 0.5–1 mm internal diameter smaller than that used for a non-pregnant woman) with mechanical ventilation will however make ventilation of the lungs easier in the presence of increased intra-abdominal pressure.

The 4Hs and 4Ts approach helps identify all the common causes of cardiac arrest in pregnancy. The most important causes are

- Haemorrhage: postpartum haemorrhage, ectopic pregnancy, placental abruption, placenta praevia, placenta accrete, uterine rupture. The key steps for treatment are stop the bleeding, fluid resuscitation, including use of rapid transfusion system and cell salvage [50] and correction of coagulopathy including use of tranexamic acid and/or recombinant activated factor VII.

- Cardiovascular disease: acute myocardial infarction and aneurysm or dissection of the aorta and peripartum cardiomyopathy, pulmonary embolism, amniotic fluid embolism cause most deaths from acquired cardiac disease.

- Pre-eclampsia and eclampsia need magnesium sulphate infusion for the treatment in peri-arrest situation.

Consider the need for an emergency hysterectomy or Caesarean section as soon as a pregnant woman goes into cardiac arrest. Delivery will relieve IVC compression and may improve chances of maternal resuscitation. Seek for help and ask gynaecologist and neonatologist to start preparing for emergency caesarean section—the foetus will need to be delivered if initial resuscitation efforts fail. The caesarean delivery also enables access to the infant so that newborn resuscitation can begin [4].

Elderly people have an increased incidence of cardiac causes of arrest because the incidence of coronary heart disease and chronic heart failure increases with age. The incidence of PEA as the first recorded rhythm increases significantly with age with a decrease of the incidence of shockable rhythms (VF/pulseless VT) [4]. No modifications of standard resuscitation protocols are needed for aged patients in cardiac arrest. Rescuers should be aware that the risk of both sternal and rib fractures is higher in elderly and the incidence of CPR-related injuries

increases with duration of CPR [51]. When deciding to resuscitate elderly patients, age alone should not be the element to consider and other more established criteria (witnessed arrest, resuscitation times, first recorded rhythm, the degree of autonomy, quality of life, mental status and presence of major comorbidities) are important factors. Whenever possible, a decision to resuscitate or not should be discussed in advance with the patient and his family.

Special circumstances in cardiac arrest need special interventions with an appropriate approach of guidelines for cardiopulmonary resuscitation.

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References

[1] Cimpoesu D. [coordinator], Rotaru L, Petris A et al. Current protocols and guidelines in emergency medicine. UMF "Gr. T. Popa" Publishing House, Iasi 2011.

[2] Nikolaos Nikolaou, Maaret Castrén, Koenraad G. Monsieurs, Diana Cimpoesu, Marios Georgiou, Violetta Raffay, Rudolph Koster, Silvija Hunyadi-Anticevi, Anatolij Truhlárˇ, Leo Bossaert, The EUROCALL investigators. Time delays to reach dispatch centres in different regions in Europe. Are we losing the window of opportunity? — The EUROCALL study. Resuscitation 111 [2017] 8-13

[3] Jan-Thorsten Gräsner, Leo L.Bossaert, Diana Cimpoesu et al., EuReCa ONE – 27 Nations, ONE Europe, ONE Registry. A prospective one month analysis of out-of-hospital cardiac arrest outcomes in 27 countries in Europe, Resuscitation, Vol. 105:188-195, http://dx.doi.org/10.1016/j.resuscitation.2016.06.004

[4] European R7esuscitation Council Guidelines for Resuscitation 2015, Truhlář A, Barelli, Al et al. Resuscitation, Volume 95, 148-201

[5] Soar J, Perkins GD, Abbas G, et al. European Resuscitation Council Guidelines for Resuscitation 2010 Section 8. Cardiac arrest in special circumstances: electrolyte abnormalities, poisoning, drowning, accidental hypothermia, hyperthermia, asthma, anaphylaxis, cardiac surgery, trauma, pregnancy, electrocution. Resuscitation 2010;**81**:1400-33.

[6] Judith E. Tintinalli, et. al.-Tintinalli's Emergency Medicine A Comprehensive Study Guide, 8th edition McGraw-Hill Publishing House, NewYork, 2016, ISBN: 007179476X

[7] Lexow K. Severe accidental hypothermia: survival after 6 hours 30 minutes of cardiopulmonary resuscitation. Arctic Med Res 1991;**50**:112-4.44.

[8] Pasquier M, Zurron N, Weith B, et al. Deep accidental hypothermia with coretemperature below 24 degrees c presenting with vital signs. High Alt Med Biol 2014;**15**:58-63.

[9] Wood S. Interactions between hypoxia and hypothermia. Annu Rev Physiol 1991;**53**:71-85.

[10] Simpson CR, Sheikh A. Adrenaline is first line treatment for the emergency treatment of anaphylaxis. Resuscitation 2010;**81**:641-2.

[11] Kleber C, Giesecke MT, Lindner T, Haas NP, Buschmann CT. Requirement for a structured algorithm in cardiac arrest following major trauma: epidemiology, management errors, and preventability of traumatic deaths in Berlin. Resuscitation 2014;**85**:405-10.

[12] Warner KJ, Copass MK, Bulger EM. Paramedic use of needle thoracostomy inthe prehospital environment. Prehosp Emerg Care 2008;**12**:162-8.

[13] Escott ME, Gleisberg GR, Kimmel K, Karrer A, Cosper J, Monroe BJ. Simple thoracostomy. Moving beyond needle decompression in traumatic cardiac arrest. JEMS 2014;**39**:26-32.

[14] Pokorna M, Necas E, Skripsky R, Kratochvil J, Andrlik M, Franek O. How accurately can the aetiology of cardiac arrest be established in an out-of-hospital setting? Analysis by "concordance in diagnosis crosscheck tables". Resuscitation 2011;**82**:391-7

[15] Cimpoesu D Pulmonary Thromboembolism : prehospital and Emergencu Department approach in Petris A, Tînt D, Tatu-Chitoiu G, Pop C [coordinator]. Pulmonary thromboembolism: a modern approche. Ed. PIM, Iasi 2015

[16] Cimpoesu D, Petris A, Tatu-Chitoiu G, Pop C, Tînt D. ESC 2008 Guidelines for diagnosis and treatment of pulmonary embolism – vademecum for Emergency Department. Edited by Sandesc D, Bedreag O, Papurica M. in Recommendations and protocols in anesthesia, intensive care and emergency medicine 2010, Mirton Publishing House, Timisoara 2010:533-550. ISBN 978-973-52-0914-8

[17] Cimpoesu D, Rotaru L, Diaconu C, Petris O, Petris A. Protocol approach to chest pain in emergency. Edited by Sandesc D, Bedreag O, Papurica M in Recommendations and protocols in anesthesia, intensive care and emergency medicine, Mirton Publishing House, Timisoara 2009:591-614. ISBN 978-973-52-0736-6

[18] Tatu-Chitoiu G, Dorobantu M, Petris A, Deleanu D, Arafat R, Cizmaru O, Cimpoesu D, Georgescu V, Oprisan M, Dimitriu I, PopescuL, Costinas M, Oprita B. Guidelines for diagnosis and treatment in pre-hospital phase of acute myocardial infarction with ST segment elevation. Curtea Veche Publishing House, Bucharest 2009. ISBN 978-973-1983-14-1, 216p

[19] Petris AO, Tatu-Chitoiu G, Cimpoesu D, Costache I, Tînt D.Thrombolysis for intra-hospital cardiac arrest related to pulmonary embolism-study design. Biomedical Research 2017;**28**(1):106-110

[20] Bottiger BW, Arntz HR, Chamberlain DA, et al. Thrombolysis during resuscitation for out-of-hospital cardiac arrest. N Engl J Med 2008;**359**:2651-62

[21] Mowry JB, Spyker DA, Cantilena Jr LR, McMillan N, Ford M. 2013 Annual Report of the American Association of Poison Control Centers' National Poison Data System [NPDS]: 31st Annual Report. Clin Toxicol [Phila] 2014;**52**:1032-283.

[22] Nunnally ME, O'Connor MF, Kordylewski H, Westlake B, Dutton RP. The incidence and risk factors for perioperative cardiac arrest observed in the national anesthesia clinical outcomes registry. Anesth Analg 2015;**120**:364-70

[23] Sprung J, Warner ME, Contreras MG, et al. Predictors of survival following cardiac arrest in patients undergoing noncardiac surgery: a study of 518,294 patients at a tertiary referral center. Anesthesiology 2003;**99**:259-69.

[24] Newland MC, Ellis SJ, Lydiatt CA, et al. Anesthetic-related cardiac arrest and its mortality: a report covering 72,959 anesthetics over 10 years from a US teaching hospital. Anesthesiology 2002;**97**:108-15.

[25] Davis TR, Young BA, Eisenberg MS, Rea TD, Copass MK, Cobb LA. Outcome of cardiac arrests attended by emergency medical services staff at community outpatient dialysis centers. Kidney international 2008;**73**:933-9.

[26] Lafrance JP, Nolin L, Senecal L, Leblanc M. Predictors and outcome of cardiopulmonary resuscitation [CPR] calls in a large haemodialysis unit over a seven-year period. Nephrol Dial Transplant 2006;**21**:1006-12.

[27] Meaney PA, Nadkarni VM, Kern KB, Indik JH, Halperin HR, Berg RA. Rhythms and outcomes of adult in-hospital cardiac arrest. Critical care medicine 2010;**38**:101-8.

[28] Girotra S, Nallamothu BK, Spertus JA, et al. Trends in survival after in-hospital cardiac arrest. The New England journal of medicine 2012;**367**:1912-20.

[29] Bird S, Petley GW, Deakin CD, Clewlow F. Defibrillation during renal dialysis: a survey of UK practice and procedural recommendations. Resuscitation 2007;**73**:347-53.

[30] Perkins GD, Stephenson BT, Smith CM, Gao F. A comparison between over-the-head and standard cardiopulmonary resuscitation. Resuscitation 2004;**61**:155-61.

[31] Handley AJ, Handley JA. Performing chest compressions in a confined space. Resuscitation 2004;**61**:55-61.

[32] Maisch S, Issleib M, Kuhls B, et al. A comparison between over-the-head and standard cardiopulmonary resuscitation performed by two rescuers: a simulation study. The Journal of emergency medicine. 2010;**39**:369-76.

[33] Hung KK, Cocks RA, Poon WK, Chan EY, Rainer TH, Graham CA. Medical volunteers in commercial flight medical diversions. Aviat Space Environ Med. 2013;**84**:491-7.

[34] Lyon RM, Nelson MJ. Helicopter emergency medical services [HEMS] response to out-of-hospital cardiac arrest. Scandinavian journal of trauma, resuscitation and emergency medicine 2013;**21**:1.

[35] Brugger H, Paal P, Boyd J. Prehospital resuscitation of the buried avalanche victim. High Alt Med Biol 2011;**12**:199-205.

[36] Wang JC, Tsai SH, Chen YL, et al. The physiological effects and quality of chest compressions during CPR at sea level and high altitude. Am J Emerg Med 2014;**32**:1183-8.

[37] Tomazin I, Ellerton J, Reisten O, Soteras I, Avbelj M, International Commission for Mountain Emergency Medicine. Medical standards for mountain rescue operations using helicopters: official consensus recommendations of the International Commission for Mountain Emergency Medicine [ICAR MEDCOM].High Alt Med Biol 2011;**12**:335-41.

[38] Fahmy FS, Brinsden MD, Smith J, Frame JD. Lightning: the multisystem group injuries. The Journal of trauma 1999;**46**:937-40.

[39] Browne BJ, Gaasch WR. Electrical injuries and lightning. Emerg Med Clin North Am 1992;**10**:211-29.

[40] Kleiner JP, Wilkin JH. Cardiac effects of lightning stroke. Jama 1978;**240**:2757-9.

[41] Lichtenberg R, Dries D, Ward K, Marshall W, Scanlon P. Cardiovascular effects of lightning strikes. J Am Coll Cardiol 1993;**21**:531-6.

[42] SALT mass casualty triage: concept endorsed by the American College of Emergency Physicians, American College of Surgeons Committee on Trauma, American Trauma Society, National Association of EMS Physicians, National Disaster Life Support Education Consortium, and State and Territorial Injury Prevention Directors Association. Disaster Med Public Health Prep 2008;**2**:245-6.

[43] Lim WJ, Mohammed Akram R, Carson KV, et al. Non-invasive positive pressure ventilation for treatment of respiratory failure due to severe acute exacerbations of asthma. The Cochrane database of systematic reviews 2012;**12**:CD004360.

[44] Leatherman JW, McArthur C, Shapiro RS. Effect of prolongation of expiratory time on dynamic hyperinflation in mechanically ventilated patients with severe asthma. Critical care medicine 2004;**32**:1542-5.

[45] Deakin CD, McLaren RM, Petley GW, Clewlow F, Dalrymple-Hay MJ. Effects of positive end-expiratory pressure on transthoracic impedance-- implications for defibrillation. Resuscitation 1998;**37**:9-12.

[46] Hubner P, Meron G, Kurkciyan I, et al. Neurologic causes of cardiac arrest and outcomes. The Journal of emergency medicine 2014;**47**:660-7.

[47] Page-Rodriguez A, Gonzalez-Sanchez JA. Perimortem cesarean section of twin pregnancy: case report and review of the literature. Academic emergency medicine : official journal of the Society for Academic Emergency Medicine 1999;**6**:1072-4.

[48] Lipman S, Cohen S, Einav S, et al. The Society for Obstetric Anesthesia and Perinatology consensus statement on the management of cardiac arrest in pregnancy. Anesthesia and analgesia 2014;**118**:1003-16.

[49] Nolan JP, Soar J, Wenzel V, Paal P. Cardiopulmonary resuscitation and management of cardiac arrest. Nat Rev Cardiol 2012;**9**:499-511.

[50] Geoghegan J, Daniels JP, Moore PA, Thompson PJ, Khan KS, Gulmezoglu AM. Cell salvage at caesarean section: the need for an evidence-based approach. BJOG 2009;**116**:743-7.

[51] Kashiwagi Y, Sasakawa T, Tampo A, et al. Computed tomography findings of complications resulting from cardiopulmonary resuscitation. Resuscitation 2015;**88**:86-91.