Cardiopulmonary Resuscitation (CPR)

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Background: Cardiopulmonary resuscitation (CPR) is an emergency procedure to establish and maintain breathing and circulation to a person who has stopped breathing (respiratory arrest) and/or whose heart has stopped (cardiac arrest). **Objective:** The goal of performing cardiopulmonary resuscitation is to en-sure the flow of oxygen and blood to the brain and other vital organs, which seeks to preserve primarily brain function and the function of other vital organs. The aim of this technique is to save lives. Basic life support (BLS) is the backbone of effective resuscitation after cardiorespiratory arrest. The goal is to maintain adequate ventilation and circulation until the underlying acute cause of the failure can be elimi-nated, it should be emphasized that 3-4 minutes without adequate perfusion will lead to irreversible cere-bral damage. **Methods:** This paper contains description of the most important aspects of CPR which are mandatory to know and to use by health care employees or bystander response to cardiac arrest. **Results and discussion:** Resuscitation measures must be carried out until the recovery of the endangered patient or until the appearance of certain signs of death. Studies have shown that survival decreases by 10-15% for every minute of cardiac arrest without CPR. CPR that was initiated within minutes of the onset of cardiac arrest has been shown to increase survival rates 2 to 3-fold and improve neurological outcome after 1 month. The latest recommendations for cardiopulmonary resuscitation - CPR were published on October 15, 2015 by the European Resuscitation Council (ERC) and the American Society of Cardiology (AHA) based on the consensus conference of ILCOR (International Liaison Committee on Resuscita-tion–formed in 1993) held in Dallas in February 2015. **Conclusion:** Cardiac arrest and CPR knowledge are crucial to improve the response to and survival of cardiac arrest patients in public and hospital settings. High-quality CPR is associated with improved survival of cardiac arrest. Our focus should be on raising the awareness for CPR programs and optimal organization in academic settings and in the community.

Keywords: Cardiopulmonary Resuscitation (CPR), Sudden Cardiac Death, Sudden Cardiac Arrest (SCA), Acute Coronary Syndrome (ACS), Cardiac defibrillation (DC).

Professional paper, Received: Nov 10, 2019, Accepted: Dec 02, 2019, doi: 10.5455/ijbh.2019.7.71-94, Int J Biomed Healthc. 2019 Dec; 7(2): 71-94

1. ETIOLOGY AND PATHOGENESIS OF CPR

Sudden cardiac arrest (SCA) is a discontinuation of cardiac mechanical activity that leads to a sudden and unexpected loss of efficient circulation. SCA is most often the result of ventricular fibrillation, with about 400,000 cases a year in Europe. Most people who suffer from SCA become unconscious within seconds to minutes because of insufficient cerebral blood flow. There are usually no preliminary symptoms. If symptoms are present, they are not specific and include chest discomfort, palpitations, shortness of breath, and weakness. If SCA is not treated, it ends in death within minutes, and the survival rate is only 5-20%, which means that it must be responded to immediately, given that any delay carries with it a significant reduction in the chance of survival.

SCD is any natural, unexpected, sudden death caused by cardiac pathology with previous symptoms that do not last longer than 1 hour. SCD is the cause of 25% of all deaths. Coronary heart disease is the most common cause of SCD. The most common cardiac mechanisms are ventricular fibrillation (VF), asystole, pulseless electrical activity (PEA), and persistent ventricular tachycardia (VT). Other mechanisms include ventricular rupture, cardiac tamponade, acute mechanical obstruction of the flow, and acute rupture of a large blood vessel (1, 11, 13).

SCD should be distinguished from sudden cardiac arrest (SCA). The main difference between sudden cardiac death and cardiac arrest is the irreversibility of the interruption of biological functions present in SCD, while in cardiac arrest the cessation of function is potentially reversible if cardiopulmonary resuscitation and/or defibrillation is started early.

Causes of cardiac arrest can be cardiac and non-cardiac in nature:

1. Patients with acute coronary syndrome (ACS) face a high risk of fatal complications including malignant arrhythmias, cardiac arrest, and death. ACS is responsible for almost 70% of all SCDs. Cardiac arrest in these patients is mainly caused by cardiac arrhythmia (VT, VF, asystole, total AV block

and PEA) or due to active heart failure when myocardial infarction affects a large part of the heart muscle.

2. Structural heart disease not related to coronary heart disease accounts for 10% of all SCDs. This group includes cardiomyopathies (hypertrophic, dilating or arrhythmogenic), heart rhythm disorders, congenital anomalies of the coronary arteries, myocarditis, hypertensive heart disease and congestive heart failure. Congestive heart failure increases the risk of SCD fivefold.
3. Hereditary arrhythmias syndromes: arrhythmias that are not the result of structural heart disease account for 5 to 10% of sudden cardiac arrest. These are often caused by genetic disorders that lead to abnormal heart rhythms. Genetic mutations often affect specialized proteins known as ion channels that carry electrically charged particles across the cell membrane, so this group of conditions is often referred to as channelopathies. This group of inherited arrhythmias includes prolonged QT interval syndrome (LQTS), shortened QT interval syndrome (SQTS), Brugada syndrome, catecholaminergic polymorphic ventricular tachycardia (CPVT). Other conditions that induce arrhythmia but are not caused by genetic mutations include Wolff-Parkinson-White syndrome and pre-excitation syndrome (7, 11).
4. Cardiac tamponade can develop rapidly (acute) or gradually (chronic). If left untreated, cardiac tamponade can lead to cardiac arrest and eventual death. Traumatic chest injury as well as diagnostic, angiographic, and interventional cardiac procedures can lead to cardiac arrest and eventual SCD.
5. SCA due to non-cardiac causes makes up the remaining 15 to 25%. The most common non-cardiac causes are traumatic chest injuries, major bleeding (gastrointestinal bleeding, aortic rupture, or intracranial hemorrhage), hypovolemic shock, overdosing, and drowning.
6. Pulmonary embolism of various types, tension pneumothorax and pleuropericardial adhesions can cause malignant heart rhythm disorders and cardiac arrest.
7. There is a risk of cardiac or respiratory arrest during major non-cardiac surgeries, especially in patients with cardiovascular disease or with significant cardiovascular risk factors.
8. Hypothermia (body temperature <28° C) or hyperthermia (body temperature >40° C) may cause cardiac arrhythmias and consequent cardiac arrest.
9. Electrolyte disturbances (hypokalemia, hyperkalemia, hypomagnesemia, hypocalcemia and hypo or hypernatremia) and acid-base status may impair cardiac function and lead to cardiac arrest.
10. Heart failure can also be caused by poisoning: the sting of certain jellyfish, pesticides, herbicides, cocaine and opioids, amphetamines, alcohol, organophosphates, and nerve acting agents.
11. Certain medications can lead to heart failure: tricyclic antidepressants, beta blockers, calcium antagonists, theophylline, digoxin, ergometrine, antiarrhythmics, and local anesthetics.
12. Anaphylaxis, hypoglycemia, hyperglycemia, sepsis, airway obstruction and hypoxia may cause cardiac arrest.
13. Electric shocks when handling electrical appliances or in contact with a power source and lightning strikes in bad weather conditions.

It is good to know the terms H6 and T6 and their significance as an aid used to recall possible treatable or reversible causes of heart failure more easily.

The term H6 includes and means: Hypovolemia, Hypoxia, Hydrogen Ions (Acidosis), Hyperkalemia or Hypokalemia, Hypothermia and Hypoglycemia or Hyperglycemia.

The term T6 includes and means: Tablets or toxins (poisoning), Cardiac tamponade, Tension pneumothorax, Thrombosis (myocardial infarction), Thromboembolism (pulmonary embolism) and Traumatic cardiac arrest.

CPR has been the literal life saver for centuries. The first attempts to deal with sudden cardiac arrest or heart attack began in the mid-1700s in Amsterdam, where a group of wealthy civic-minded citizens organized a group called the „Society for the Recovery of the Drowned ". The organization has developed a set of rules to follow in case a person drowns. It was so successful that similar organizations were founded throughout Europe and US. In 1740, the French Academy of Sciences recommended mouth-to-mouth resuscitation to revive the drowned. In 1891, Dr. Friedrich Maass performed the first documented chest compression in humans. In 1903, Dr. George Crile reported the first successful application of external chest compression to a human.

In 1901, Igelsurd and Arbuthnoth Lane restored circulation with direct cardiac massage during surgery. In 1930, William Kouwenhoven, an electrical engineer, invented the first manual external defibrillator. In 1947, Claude Beck, a professor of surgery at Case Western Reserve University, was the first to perform a successful defibrillation on a human heart on a fourteen-year-old boy who underwent surgery because of a congenital heart defect.

Dr. Peter Safar is considered the father of modern cardiopulmonary resuscitation, he was born in 1924 in Vienna–Austria in a doctoral family. He graduated from the University of Vienna in 1948. Safar moved from Vienna to Hartford, Connecticut in the United States in 1949. He completed training in anesthesiology at the University of Pennsylvania in 1952. In 1954, he became head of the Department of Anesthesiology at Baltimore City Hospital.

Dr. Peter Safar along with Dr. James Elam identified the initial steps of CPR. These steps include a maneuver to tilt the head and chin to open the patient's airways in an unconscious state, as well as mouth-to-mouth breathing. He combined A (airway) and B (breathing) cardiopulmonary resuscitation with C (chest compression). In 1957,

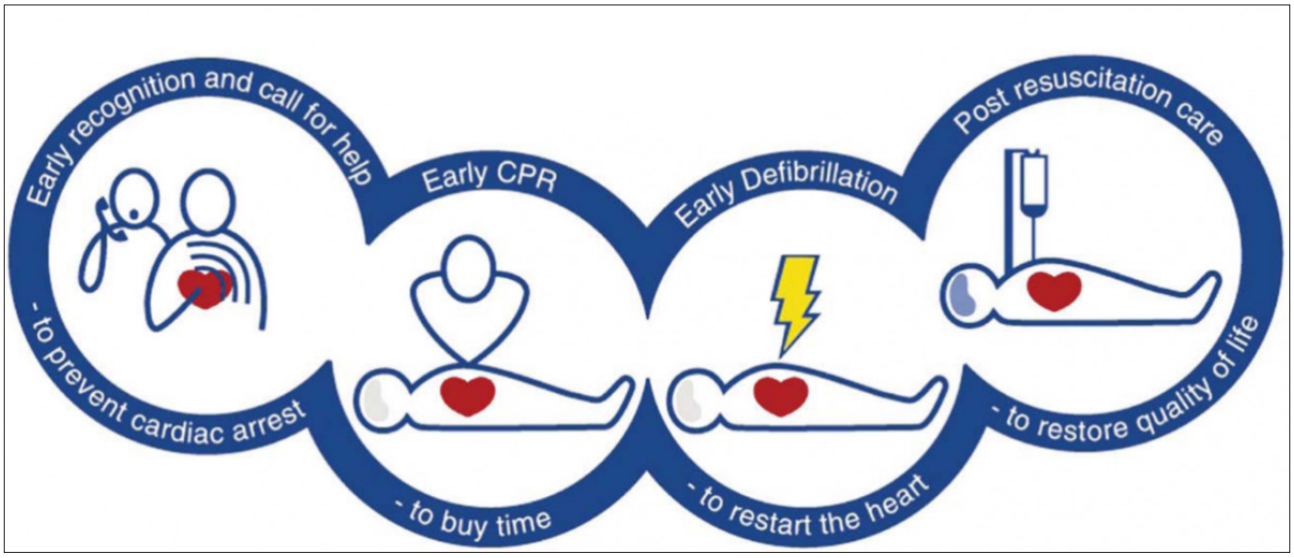

Figure 1. The chain of survival and its main features. Source: European Resuscitation Council Guidelines for Resuscitation 2015.

Peter Safar published a book called „ABC of Resuscitation ", which became a kind of „CPR bible ". This A-B-C CPR training system was later adopted by the American Heart Association. In 1961, Peter Safar introduced the combination of positive pressure ventilation with external cardiac massage. In 1963, Redding and Pearson introduce adrenaline into resuscitation procedures.

In the seventies of the twentieth century, Dr. Safar, and his colleagues-initiated study on cerebral resuscitation in cardiac arrest (by lowering the body temperature), and in 1979 Dr. Safar founded the International Resuscitation Research Center in Pittsburgh. From 1974 to 1980, Dr. Daiak (Arch Diack), Dr. Rullman, and W. Stanley Welborn developed a prototype of an automatic external defibrillator, and in 1980, a prototype heart rate monitor (pacemaker) appeared. One of the last steps in a series, in 1973, at the international conference in Washington, the American Heart Association (AHA) was authorized to standardize the measures of Basic Life Support (BLS) and Advanced Life Support (ALS). Thus, in 1974, the first standards of CPR (Standards for Cardiopulmonary Resuscitation and Emergency Cardiac Care) were published. To date, the basis of these standards has not changed much, although every 5 years at the international level they are harmonized with the latest knowledge in this area.

Today we are witnessing the significant development of highly sophisticated resuscitation equipment with monitoring systems, biphasic, automatic and implantable defibrillators, mechanical resuscitation devices (Autopulse and LUCAS), rhythm guides and pacemakers, mobile coronary units and modern emergency medical services and comprehensive programs and comprehensive programs for training of medical staff and the public in all environments (educational institutions, factories, various organizations and institutions). European guidelines indicate the need to further develop the Public Access Defibrillation (PAD) program and to set up defibrillators in public places. The entire population must be involved in education on the management of cardiac arrest in outpatient settings (1, 2, 12-18).

2. SURVIVAL CHAIN

The time from the onset of cardiac arrest to the arrival of emergency medical care is of great importance for patient survival and for reducing the consequences of congestion. A special role is played by activities such as the police, firefighters, mountain rescuers, bystanders, and everyone else–the laity. CPR performed by lay people at the scene increases the survival rate by 2-3 times. Unfortunately, lay people perform CPR in only one of 5 heart attacks.

The term „survival chain "was developed by Mary M. Newman in 1987. The American Heart Association adopted this concept in 1992 and developed it in its guidelines for cardiopulmonary resuscitation and emergency cardiac care, and in 1997 the International Liaison Committee on Resuscitation (ILCOR) repeated this concept. The chain of survival consists of four tightly connected links, the fulfillment of which is necessary to increase the success of survival after acute cardiac arrest.

According to the ERC's latest CPR guidelines today's survival chain consists of 5 links:

- Early identification and calling an emergency medical service.
- Early cardiopulmonary resuscitation, i.e., performing basic life support measures (BLS);
- Early defibrillation.
- Post reanimation integrated care.

Figure 1 illustrates the survival chain, which shows the importance of the connection of the „chain links" or separate interventions. In outpatient settings, early recognition of the importance of chest pain will direct the patient or observer to seek emergency medical attention. The first link indicates the importance of early recognition of the risk of cardiac arrest and calling the emergency medical service, with the hope that early intervention will prevent cardiac death. Hope is justified since signs of physical deterioration in 80% of people may be present

for up to an hour before cardiac arrest. The second and third links indicate the integration of early CPR and early defibrillation as fundamental components of survival and return-to- life attempts and the fourth last link points to the importance of effective post-resuscitation integrated care aimed at stabilizing and preserving vital life functions and advocating the restoration and improvement of quality of life. It should be noted that according to the American Heart Association (AHA) the survival chain consists of 5 links as opposed to the ERC's survival chain. Namely, after early defibrillation, Americans consider early implementation of advanced life support (ALS) measures during the transport of the patient as an important link, and the last 5 links in that chain are integrated post-resuscitation care. Survival of cardiac arrest depends on the sequence of interventions, and parts of the chain are time sensitive and need to be optimized with each other to increase the chance of survival. The survival chain is a recognizable symbol of resuscitation in many countries around the world, and although the design of the chain changes over time, its message has remained unchanged and clear (1, 5, 7).

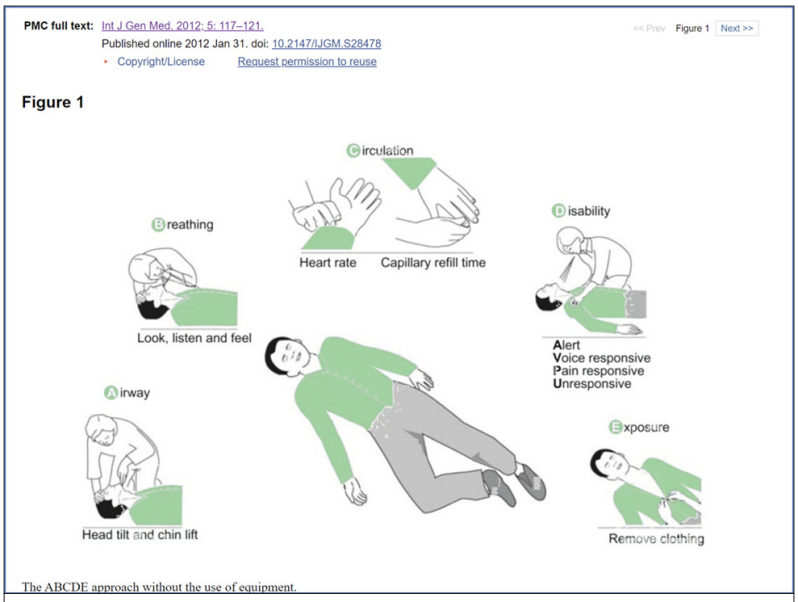

Figure 2. ABCDE Algorithm: A: Airways, B: Breathing, C: Circulation, D: Disability-rapid neurological assessment, E: Exposure. Source: Thim T et al. Int J Gen Med. 2012; 5: 117-121.

Early recognition and initial assessment of the patient's condition: A physician, medical professional, or eyewitness who is trained and capable should quickly assess the condition of the patient who has collapsed to determine if the collapse was due to cardiac arrest. It is necessary to first determine whether the patient is answering or not answering the call, i.e., whether the patient has lost consciousness, whether he/she is breathing normally or not, or whether there has been a cessation of breathing. The patient who does not answer the call and does not breathe normally is in cardiac arrest and should start cardiopulmonary resuscitation immediately. Discoloration of the skin and visible mucous membranes in the patient may lead to marked pallor (bleeding) or cyanosis (anoxia or ischemia), auscultation may reveal inaudible heart sounds, palpation of the main arteries (carotid and femoral arteries) may reveal pulse loss over large blood vessels, peripheral pulses are not palpable when arterial pressure is lower than 40 mmHg. Based on these criteria, cardiac arrest should be suspected as soon as possible without wasting time. It should always be borne in mind that after 3-5 minutes, tissue anchovies and irreversible damage to brain cells occur, after which any resuscitation is unsuccessful because brain death has occurred. This period can be prolonged (in hypothermia, in patients on cerebroprotective therapy as well as in young children whose brain is resistant to ischemia and hypoxia) or shortened (in chronic patients with diseases of the respiratory and cardiovascular systems, in hyperthyroidism, and in pregnant women). That is why the so-called „arrest time "is particularly important-the time from the moment of cardiac arrest to the moment of CPR onset, in which eyewitnesses or witnesses of cardiac arrest play a key role, whether they are medical lay people or health workers (1, 7-9).

The first minutes after the formation of SCA are critical. With each minute of delayed CPR or defibrillation, survival is reduced by 10%.

1.4. ABCDE Algorithm

The ABDCE algorithm stands for A: Airways, B: Breathing, C: Circulation, D: Disability, rapid neurological assessment, E: Exposure. We always start the examination of the patient with approach A, B, C, D, E. In case of visible and significant bleeding, we approach the patient according to the principle of C, A, B–so stop the bleeding first. The entire patient examination protocol takes place as a team. The doctor is the leader of the team and he schedules the tasks and examines the patient person at the same time. The ABCDE approach is applicable in all clinical emergencies for immediate assessment and treatment. This approach can be used on the street without equipment or, in a more advanced form, upon the arrival of an ambulance, in an emergency medical service, in a general hospital ward, or in an intensive care unit. This approach is widely accepted by emergency medicine experts. This approach improves outcome because it helps healthcare professionals focus on life-threatening clinical problems. In an acute setting, possessing the high-quality ABCDE approach skill in all medical team members will significantly save valuable time and improve their work, which will ultimately have a positive impact on the outcome of the patient's treatment (3, 4, 11).

Before performing cardiopulmonary resuscitation, it is necessary to do the following:

First of all, it should be ensured that the environment in which the patient is located is safe and secure. First look at the patient in general to see if the patient is not feeling

Figure 3. Proper performance of external cardiac massage

well. If the patient is awake, ask „how are you? " If the patient is unconscious, shake the patients and ask, „Are you okay?". This first quick examination of the patient's „**look, listen, feel**" should take about 30 seconds and will often indicate whether the patient is critically ill and whether there is a need for urgent intervention.

If the patient is unconscious, unresponsive, and is not breathing normally (occasional inhalations are not normal) CPR should be initiated according to resuscitation guidelines. If you are a health care professional who is educated and trained to provide CPR, palpate the pulse to determine if the patient has respiratory arrest. If you are not sure if the patient has a palpable pulse, CPR should be started. Vital signs should be monitored early. Connect a pulse oximeter, ECG monitor and non-invasive blood pressure monitor as soon as possible for all critically ill patients. Place the intravenous cannula as soon as possible. Take blood for laboratory analysis when placing an intravenous cannula.

A = Airway

1. Look for signs of airway obstruction: airway obstruction causes paradoxical movements in the chest and abdomen („seesaw "breathing) and the use of auxiliary respiratory muscles. Central cyanosis is a late sign of airway obstruction. With complete airway obstruction, there is no audible breathing through the mouth or nose. In partial obstruction the air intake is reduced and often noisy. In a critically ill patient, the disturbance of consciousness often leads to airway obstruction.
2. Treat airway obstruction as an urgent medical condition: untreated airway obstruction causes hypoxemia (low PaO_2) with the risk of hypoxic brain, kidney, and heart injury, cardiac arrest, and even death. In most cases, only simple airway cleansing methods are required (e.g., airway opening maneuvers, airway suction, oropharyngeal, or nasopharyngeal tube insertion). Sometimes tracheal intubation is required if the above methods were not successful.
3. Provide oxygen in high concentration using an oxygen tank mask. Ensure that the oxygen flow is sufficient (usually 15 L/min.) To prevent the tank from collapsing during inspiration. If the patient is intubated, give high-concentration oxygen with a self-blowing bag. In acute respiratory insufficiency, 94-98% oxygen saturation should be maintained. In patients at risk of hypercapnic respiratory failure, the goal is oxygen saturation of 88 to 92%.

B = Breathing

During the immediate assessment of respiration, it is vital to diagnose and treat life-threatening conditions immediately (e.g., acute severe asthma, pulmonary edema, tension pneumothorax, and massive hemothorax) (1, 3, 7, 18, 19)..

1. If you notice that the patient produces snoring sounds, open the airway by opening the mouth, lifting the chin and lower jaw, manually remove larger parts of foreign bodies (teeth, chewing gum...), aspirate the liquid contents. After cleaning the airway and reaching the open airway, the patient is administered oxygen 10-15 L/min. by mask.
2. If the patient does not breathe even after airway cleansing, an oropharyngeal tube is placed and the patient is ventilated through an Ambu mask, or endotracheal intubation is performed, with which we must be careful because at the moment of intubation there is an increase in intracranial pressure.
3. Look, listen, and feel the general signs of respiratory distress: sweating, central cyanosis, use of auxiliary respiratory muscles, and abdominal breathing.
4. Determine the respiratory rate. The normal rate is 12-20 breaths/min. High (>25 min.) Respiratory rate indicates disease and warns that the patient's condition may deteriorate abruptly. A respiration rate of less than 10 and greater than 29 requires ventilation support.
5. Assess the depth of each breath, the rhythm of breathing, and whether the expansion of the chest

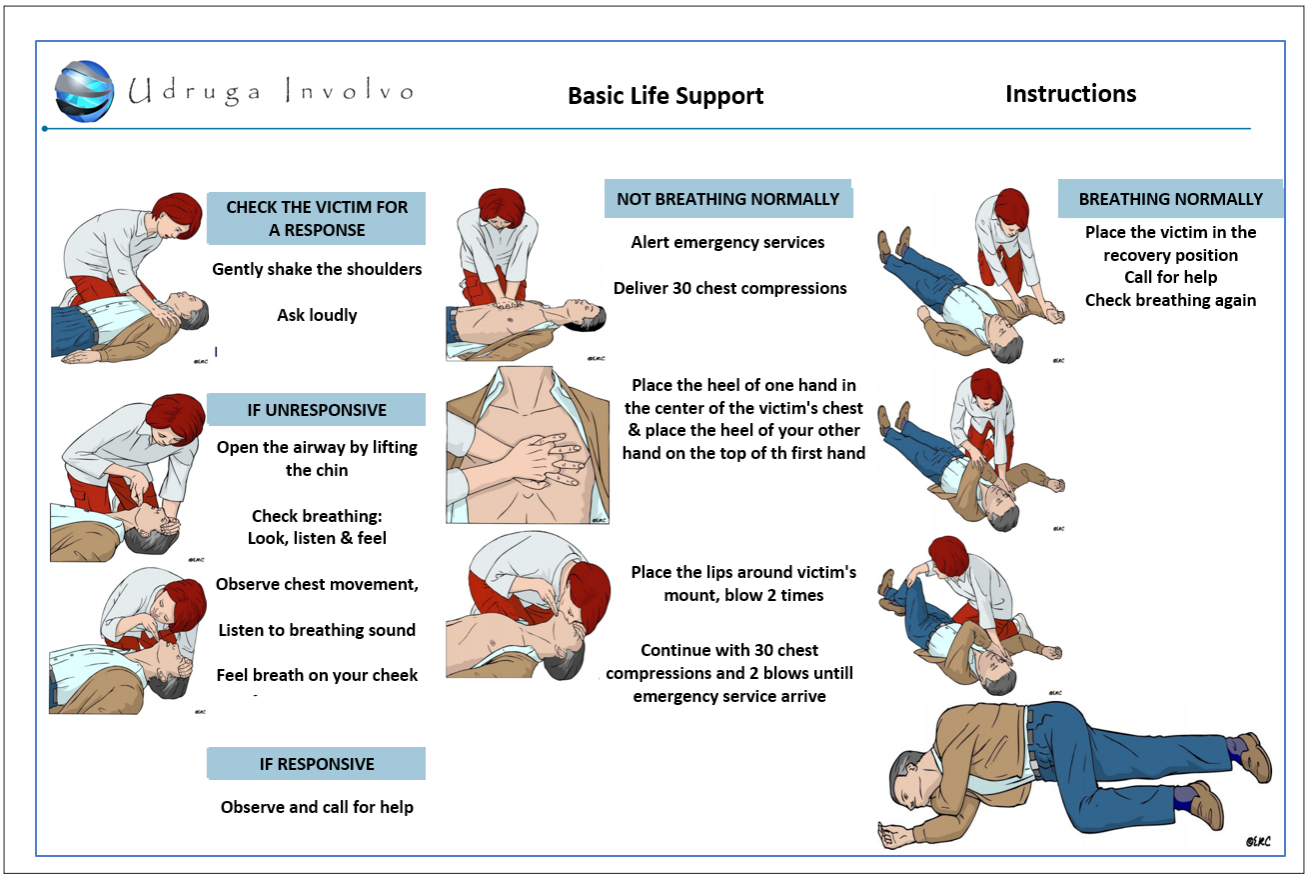

Figure 4. Basic life support steps and lateral stable position for patient recovery. Source: European Resuscitation Council Guidelines for Resuscitation 2015

is equal on both sides.

6. Pay attention to any deformity of the chest (this may increase the risk of deteriorating the ability to breathe normally); look for an elevated jugular venous pulse (JVP) (e.g., in acute severe asthma or tension pneumothorax); remember that abdominal distension can restrict diaphragm movements, which will worsen respiratory distress.
7. Listen to how the patient's breathing sounds at a short distance from the face, wheezing indicates the presence of secretions in the airways, and are usually caused by the patient's inability to cough enough or take a deep breath. Stridor or wheezing suggests partial but significant airway obstruction.
8. Percuss the chest, a hyperresonant sound indicates pneumothorax, and a dull sound goes in favor of lung consolidation (infiltrative changes or pleural effusion).
9. Auscultate the chest, listen to breathing with a stethoscope in 6 places on the chest. Bronchial respiration indicates consolidation of the lungs with airway patency, absent or attenuated respiratory murmur suggests pneumothorax or pleural effusion, or consolidation of the lungs due to complete obstruction.
10. Check the position of the trachea in the suprasternal incision: a deviation to one side indicates a displacement of the mediastinum (e.g., pneumothorax, pulmonary fibrosis, or pleural effusion).
11. The specific treatment of respiratory disorders depends on the cause. However, all critically ill patients should be given oxygen. In a subgroup of patients with COPD, high oxygen concentrations can depress respiration (i.e., they are at risk of hypercapnic respiratory arrest–often referred to as type 2 respiratory failure). However, these patients will also suffer end organ damage or heart failure if there is a decrease in blood oxygen pressures. In this group it is necessary to strive for lower saturation of PaO2 and oxygen than usual. Give oxygen through a Venturi 28% mask (4 L/min.) Or 24% Venturi mask (4 L/min.) At the beginning and re-evaluate. Target range of SpO2 88-92% in most patients with COPD.
12. If the patient's breathing depth or speed is judged to be inadequate or absent, use an Ambu mask or pocket mask to improve oxygenation and ventilation, and seek professional help immediately. In patients without airway obstruction, the use of non-invasive ventilation (NIV) should be considered. In patients with acute exacerbation of COPD, the use of NIV is often beneficial and prevents the need for endotracheal intubation and invasive ventilation.

C = Circulation

In almost all medical and surgical emergencies, hypovolemia is the underlying cause of shock until proven otherwise. Unless there are obvious signs of a heart cause, give intravenous fluid to any patient with a cold periphery and a fast heartbeat. In surgical patients, quickly

rule out bleeding (obvious or hidden). Keep in mind that breathing problems, such as tension pneumothorax, can also endanger the patient's circulatory condition. This should be treated earlier during the assessment of the patient's condition.

1. Look at the color of the hands and fingers: are they blue, pink, pale, or mottled? By touching the patient, we assess the quality of the skin: cold, warm, wet, dry and see if the skin is pale or cyanotic (cyanosis is a sign of decompensated shock).
2. Asses the body temperature of the limbs by touching the patient's hands: are they cold or warm?
3. Measure the capillary refill time (CRT). By pressing on the nail or sternum for 5 seconds to cause blanching (pallor) and calculate the time it takes for the skin to return to the surrounding skin color and thus estimate the CRT which is usually <2 seconds, and over 4 seconds is a sign of hypovolemia–suspected bleeding and hemorrhagic shock.
4. Assess the condition of the veins: they may be insufficiently filled or collapse when hypovolemia is present.
5. Determine the patient's heart rate (or preferably heart rate by listening to the heart with a stethoscope).
6. Palpate peripheral and central pulses, assessing presence, speed, quality, regularity, and evenness. Barely palpable central pulses suggest poor cardiac output. Palpation of the radial pulse immediately leads to the data that the systolic pressure is above 90 mmHg.
7. Measure the patient's blood pressure. Even in shock, blood pressure may be normal, as compensatory mechanisms increase peripheral resistance in response to decreased cardiac output. Low diastolic blood pressure suggests arterial vasodilation (in anaphylaxis or sepsis). Narrow pulse pressure (difference between systolic and diastolic pressure; usually 35-45 mmHg) indicates arterial vasoconstriction (cardiogenic shock or hypovolemia) and may occur with rapid tachyarrhythmia.
8. Auscultate the heart. Is there noise or pericardial friction? Is it hard to hear heart tones? Does the audible heart rate correspond to the heart rate?
9. Look for other signs of poor cardiac output, such as decreased level of consciousness, and if the patient has a urinary catheter, oliguria (urine volume <0.5 ml/kg/h).
10. Inspection of clothes for bleeding is a quick examination and if you notice significant bleeding, stop the bleeding immediately with the method of digital compression–finger pressure, compressive bandage, Esmarch bandage, elevation of the extremities only if there are no signs of a fracture.
11. Carefully look for external bleeding from wounds or drainage or evidence of occult bleeding (e.g., thoracic, intra-peritoneal, retroperitoneal). Intrathoracic, intra-abdominal, or pelvic blood loss can be significant, even if the drains are empty.
12. The specific treatment of cardiovascular collapse depends on the cause, but should focus on fluid replacement, bleeding control, and restoration of tissue perfusion. Look for signs of a condition that are immediately life-threatening (e.g., cardiac tamponade, massive or continuous bleeding, septic shock) and treat it urgently.
13. Place one or more large (14 or 16G) intravenous cannulas. Use short, wide tubular cannulas as they allow maximum flow.
14. Before injecting intravenous fluid, take blood from the cannula for routine hematological, biochemical, coagulation, and microbiological tests and blood group and Rh factor determination.
15. Apply a bolus of 500 ml of warmed crystalloid solution (e.g., Hartmann's solution or 0.9% sodium chloride) within 15 minutes if the patient is hypotensive. Use smaller amounts (e.g., 250 ml) for patients with known heart failure or trauma under close supervision. (listen to the lungs and determine if there is a squeak or crackle after each bolus).
16. Regularly assess heart rate and blood pressure (every 5 min.) and aim for the patient's blood pressure to be normal or aim for systolic BP to be >100 mmHg.
17. If the patient's condition does not improve, repeat the fluid delivery procedure. Seek professional help if the patient's reaction to repeated fluid boluses is lacking.
18. If symptoms and signs of heart failure occur (dyspnea, rapid heartbeat, elevated JVP, third heart tone, and pulmonary flutter), reduce the rate of fluid infusion or stop fluid completely. Look for alternative means to improve tissue perfusion (e.g., inotropic or vasopressor drugs).
19. Since traumatized patients are often hemodynamically unstable during treatment, vital parameters should be constantly evaluated, airway, respiration, circulation checked, and problems diagnosed. Place a urinary catheter. In addition to fluid replacement with infusion solutions, correction of hypoxia, the patient should be given analgesia and warmed due to the tendency of traumatized patients to hypothermia (1, 3, 14).

D = Disability: Rapid neurological assessment

The assessment of the awareness of the patient person is part of the initial assessment and is performed when we have already assessed whether the place is secured and there is no danger to the rescuer and the person we are helping. The most common causes of unconsciousness are profound hypoxia, hypercapnia, cerebral hypoperfusion, or recent administration of sedatives or analgesics (1, 3, 5, 8).

1. Look the ABC steps first and treat according to that: exclude or include hypoxia and hypotension.
2. Check the medications the patient is using and

whether they may be reversible causes of the disturbance of consciousness. Give an antagonist if appropriate (e.g., naloxone for opioid toxicity).

- Make a quick initial assessment of the patient's consciousness using the **AVPU scale: A** (patient is awake and responsive), **V** (responds to verbal stimuli), **P** (unconscious but responds to painful stimuli when pinched) or **U** (does not respond to all stimuli at all)). Alternatively, the Glasgow Coma Scale Score (GCS) with a maximum score of 15 and a minimum score of 3 can be used **(Table 1)**.

3. If the patient responds, are aware, has maintained circulation and oxygenation, an open airway, enough air volume to utter, and enough cerebral perfusion to understand and respond. As they answer we listen to whether they answer the whole sentence, whether they produce any sounds, which may indicate partial airway obstruction and a foreign body. Assessment of the patient's speech also tells us about adequate or inadequate brain perfusion, if the patient cannot compose a sentence, we suspect hypoxia and an increase in intracranial pressure.
4. If unresponsive, check and open the airway and if breathing normally turn the victim to the recovery position (if the airway is not protected).
5. Notice the possible existence of abnormal flexion or abnormal extension of the extremities–signs of decerebration.
6. Examine the pupils (size, equality, and reaction to light).
7. Measure blood glucose to exclude hypoglycemia with a rapid finger test, and in a patient with arrest, use a venous or arterial blood sample to measure glucose, as measuring glucose from the finger may be unreliable. Follow approved hypoglycemic management protocols. For example, if the blood sugar is less than 4.0 mmol/L in an unconscious patient, give an initial dose of 50ml of 50% glucose solution or 100ml of 20% glucose solution intravenously. If necessary, give additional doses of intravenous 10% glucose every minute until the patient is fully conscious or receives a total of 250ml of 10% glucose.

Table 1. Glasgow Coma Scale (GCS)

Eyes opening		Motor response		Speech response	
Spontaneously	4	Obeys commands	6	Oriented	5
To voice	3	Localizing pain	5	Confused	4
To pain	2	Withdrawal from pain	4	Inappropriate	3
No response	1	Abnormal flexion to pain	3	Incomprehensible	2
		Abnormal extension to pain	2	No response	1
		No response	1		

E = Exposure. Sometimes it is necessary to take off all clothing, for the patient to be properly examined. Cover the patient with a blanket and respect his dignity and minimize heat loss. Examine the patient's entire body, including the groin and back, for injuries, bleeding, or rashes. Check the temperature of the patient. It is necessary to warm the patient if he is in hypothermia and there is no indication for therapeutic cooling (Figure 2).

3. PRINCIPLES OF CARDIOPULMONAL RESUSCITATION

A person who has had a sudden cessation of breathing and heart function requires emergency measures called resuscitation measures, or cardiopulmonary resuscitation. The cessation of heart functioning is actually the cessation of blood circulation, that is, the interruption in the supply of oxygen to tissues and organs. In that sense, the most sensitive is the brain, whose cells are subject to permanent damage after only 3-4 minutes in hypoxic conditions.

The goal of CPR is to maintain life through the delivery of oxygen to the brain and other vital organs until the establishment of spontaneous breathing and heart function or until the intervention of professionals. This procedure will prevent the brain cells death and other vital organs and thus save the life of the patient person with the application of ethical principles. According to research, the highest survival rates are in people who have received CPR and defibrillation within the first 3-5 minutes of losing consciousness. In each subsequent minute, the chance of survival decreases by 10% (1, 8, 12, 14).

After the first link in the chain of survival, which includes the initial assessment of the condition of the patient, it is necessary to start with cardiopulmonary resuscitation, which is divided into:

1. Early Cardiopulmonary Resuscitation–Basic Life Support (BLS)
2. Advanced Cardiopulmonary Resuscitation–Advanced Life Support (ALS)
3. Post reanimation integrated care.

Early Cardiopulmonary Resuscitation–Basic Life Support (BLS)

According to the ERC 2015 CPR guidelines, a person who does not respond to a call and who is not breathing is presumed to have cardiac arrest. The basis of life support is a combined CPR technique, which involves alternately performing external cardiac massage and artificial respiration in a ratio of 30 compressions and 2 ventilations (30:2 ratio). Be sure to wait for the chest to fully return to its original position between two breaths as well as after compression.

The following situations should ALWAYS be considered when performing an early CPR:

- Is the patient's head tilted far enough? (with the aim of opening the airway)
- Is the patient's nose sufficiently closed or the mouth well covered? (so that the air does not „leak" into the environment)?
- Does the patient lie on a hard surface?

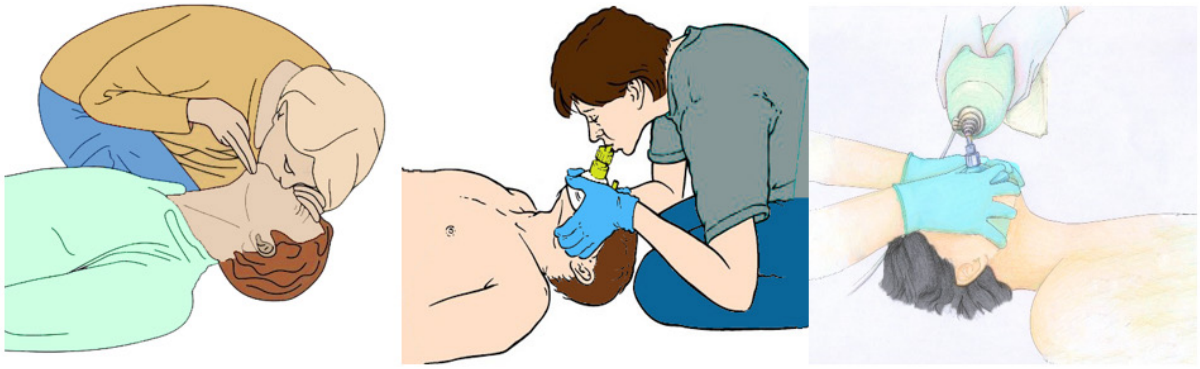

Figure 5. Artificial respiration: mouth-to-mouth technique, with help of the pocket mask and Ambu mask Source: European Resuscitation Council Guidelines for Resuscitation.

- Is there too little or too much air?
- Is the rate of air blowing (insufflation) adequate?
- Is the position of the hands on the patient's chest adequate?
- Is the pressure on the sternum too weak or too strong (4-5cm)?
- Is the frequency of chest compressions appropriate?
- Does the arms bend at the elbows during compression?
- Does the rescuer separate the palms from the chest wall?

External Cardiac Massage

External Cardiac Massage is a mechanical effect on the heart muscle to maintain blood flow through the large blood vessels of the body at the time of stopping the heartbeat caused by a particular disease. Cardiac massage involves the compression of the heart with a certain frequency, which, above all, stimulates the artificial circulation of blood, and secondly, activates its own electrical activity, which together helps to restore the work of the heart. Continuous cardiac massage has proven to be a more effective method than the classic resuscitation technique, which combines artificial respiration (mouth-to-mouth) and cardiac massage, this is the conclusion of the conducted studies. It has been found that even non-medical professionals can be more lifesaving if a person who has had a heart attack is given a continuous cardiac massage.

How is external cardiac massage performed?

1. The patient person should lie on their back and it is necessary to place the patient on a hard surface.
2. The rescuer should kneel next to the patient's shoulder between his knees.
3. Determine the position on the sternum for massage, i.e., the lower half of the sternum, i.e., the junction of the upper two thirds and the lower third of the sternum (place the root of the left hand with raised fingers in the middle of the sternum in the intermammillary line above the xyphoid extension, place the second palm over the palm of the first hand and knit the fingers). Be sure to apply pressure to the sternum and not to the ribs to prevent rib fracture.
4. With the arms outstretched at the elbows, pressure is applied to the sternum so that the chest is pushed by 4-5 cm, pressing the sternum with a frequency of 100-120 compressions per minute. Push the sternum for about 0.5 seconds, then quickly loosen. Compress the sternum rhythmically using the weight of the whole body with the elbows outstretched. Pressure movements are done from the „hips" to get less tired and the quality of the massage was better. To avoid fatigue and maintain this pace, it is recommended not to use the muscles of the arms, but the weight of the whole body and to „fall" and thus create pressure on the chest and thus perform proper compression.
5. The punctum maximum of compression force is at the root of the hand with the fingers raised to avoid rib fractures.
6. After each compression, release the pressure, allow the chest to return to its original position, do not separate the hands from the patient's chest; compression and release should last equally.
7. If resuscitation is provided by only one person, then the rescuer will bend their head back after 30 compressions, raise their lower jaw, place their hand on the patient's forehead and gently tilt their head, close the patient's nose with the thumb and forefinger of the same hand, place the fingertips of the other open the airways by lifting the chin and then perform two puffs of air into the patient's mouth–press your mouth to the patient's mouth, inhale the amount of air as in a normal breath, for 1 second, separate your mouth from the patient's and allow „passive exhalation"), insufflation should

not last longer than 5 seconds. After 2 breaths, continue with the compression and repeat this rhythm after every 30 compressions or until the arrival of another rescuer.

8. When two rescuers are present, then one performs an external heart massage at a rate of 100/min., and the other ventilates 2 insufflations after 30 compressions and resuscitators are alternated every 2 minutes.
9. Check the position of the hands on the sternum and the depth of the external heart massage from time to time (so that the pressure on the sternum does not loosen over time). Do not stop compressions at all–if possible.

With a successful external cardiac massage, we achieve 20-30% of normal heart rate with more than 80-90 mmHg of systolic pressure, which can satisfy the minimal metabolic needs of vital organs, primarily the brain and heart. Successful resuscitation leads to to blood supply to the skin and visible mucous membranes, narrowing of the pupils, the appearance of a spontaneous pulse and the appearance of spontaneous breathing. The victim is then placed in a lateral recovery position so as not to suffocate due to aspiration of vomit or drooping tongue.

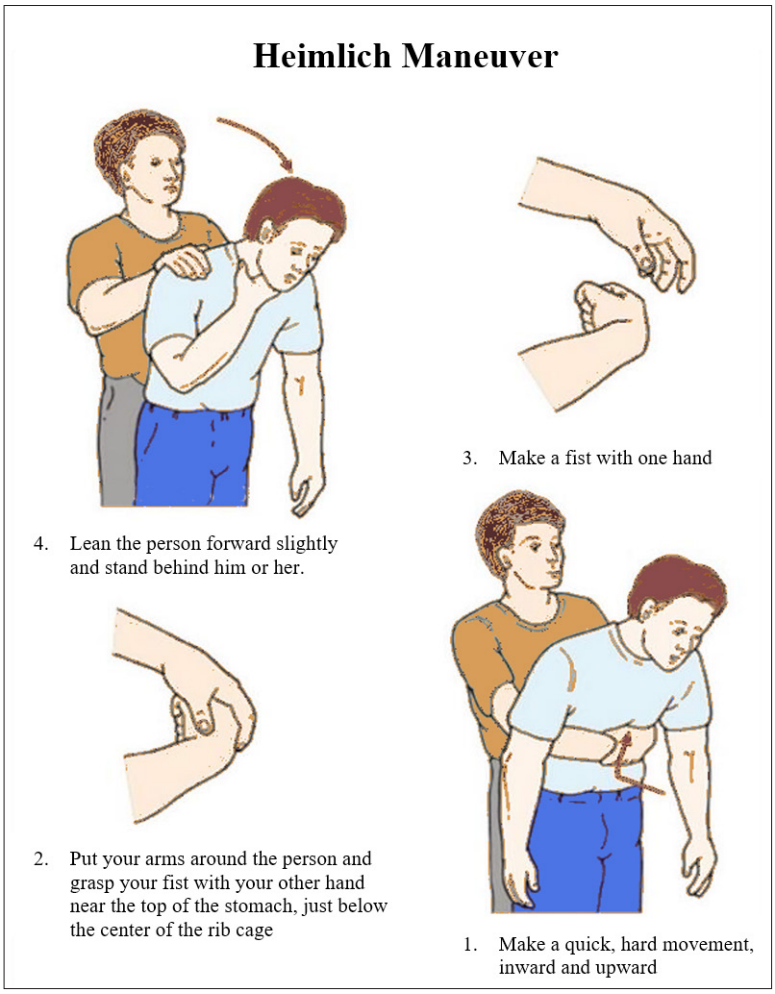

Figure 6. Performing the Heimlich Maneuver

Certain complications can occur during external cardiac massage. Complications are most often the result of improper chest compression and insufflation of too much inhaled air. Distension of the stomach by air during artificial respiration due to sagging of the lower esophageal sphincter and rapid insufflation of too much air, in some cases there may be rupture of the stomach, aspiration of gastric contents and the development of aspiration pneumonia. Sternum fractures, rib fractures are among the most common complications that can cause pneumothorax, pulmonary embolism, or pericardial effusion. Pressure on the lower chest can in rare cases lead to laceration or rupture of the liver, rupture of the spleen, rupture of a cardiac aneurysm or aorta (1, 2, 5, 18) (Figure 3).

Recovery position–Stable lateral position

This position is recommended for all unconscious patients who breathe spontaneously because in this position the possibility of airway obstruction by the base of the tongue is eliminated, and at the same time the contents can be drained from the mouth (saliva, vomit, etc.).

It is performed by turning the patient on their side. Putting in this position is more a skill than strength (as are all other resuscitation measures):

- Kneel near the patient's waist.
- Place the patient's hand that is closer to you at a right angle to the elbow (so that it looks like it is 'waving').
- Grasp the other hand of the patient, rest it with the back on the cheek of the patient which is closer to you.
- Raise the patient person's far leg at the knee so that the foot is on the ground.
- Using your knee as a lever, pull the person to your side.
- Tilt their head back facing down so that their position can be stable.

If a cervical spine injury is suspected (this should be considered), the head should be held while turning. The rotation of the body should be followed by the simultaneous rotation of the head. In addition, something should be placed under the head to rest on the base without „breaking "the neck (Figure 4).

Artificial ventilation

The heart and lungs are connected anatomically and functionally, and their function-related disorders are also causally connected. If cardiac arrest occurs within a maximum of 30 seconds, there will be a cessation of breathing. If breathing stops earlier, cardiac arrest will occur within a maximum of 3 minutes. Thus, regardless of what the primary disorder is, the consequences are always the same: there is a complete blockage of blood circulation (circulation), so the tissues are left without the

necessary amount of oxygen. The brain is most sensitive to lack of oxygen (although it makes up only 2% of total body weight, it consumes more than 20% of the oxygen delivered by the blood to organs and tissues per unit time).

Cessation of breathing occurs very rapidly after cardiac arrest. Complete cessation of breathing may be preceded by attempts to breathe called „catching air ". We recognize the cessation of breathing by the absence of chest movement (watch), the absence of breathing noise in front of the patient's mouth and nose (listen), and the absence of feeling the air current on his own cheek in front of the patient's mouth and nose (feel).

It is necessary to ensure airway patency before performing resuscitation, which may include cleaning the oral cavity of foreign contents (vomited food, blood, dentures, mucus, etc.), and then tilting the patient's head and raising the lower jaw to prevent the root of the tongue from falling and airway obstruction.

Resuscitation is performed by direct insufflation (blowing) of air from the lungs of the rescuer into the lungs of the patient. The amount of air inhaled should be appropriate to the patient's size (6-7 ml/kg) and should produce a chest lifting effect and should be delivered within 1 second. Insufflation should not be faster as it can lead to gastric distension with all the consequences (vomiting, entry of vomit into the airways). The frequency of blowing should be 8-10/min. The rescuer's exhaled air contains 16-18% O_2 and this satisfies the need of the afflicted, and the volume of insufflated air should be 700-1000 ml, which causes a well-visible chest lift. The recent ERC recommendations continue with the 2015 recommendations that the compression-ventilation ratio remain 30: 2 regardless of whether resuscitation is performed by one or two people. When using advanced means in airway maintenance, compression is performed continuously with 10 ventilations/min (1, 2, 12, 13).

The techniques of artificial respiration by direct insufflation of air into the patient's lungs are:

Mouth-to-mouth resuscitation technique: the rescuer kneels on the victim's side and throws their head back. With one hand they raise their chin and keep their mouth open, and with the other they keep their nostrils closed and keep their head tilted back. They take a deep breath, cover the patient's mouth with his mouth and blow in the air briefly (for 1 second) and evenly. During ventilation, the rescuer should monitor the patient's chest movements and adjust the volume of air that is blown accordingly. Namely, this volume should be large enough to lift the chest as in spontaneous breathing.

Mouth-to-nose resuscitation technique: resuscitation is more difficult to perform and is performed only when we cannot open the patient's mouth (trismus, convulsions) or the presence of obstructions in the oral cavity, when we cannot cover them with our mouth or when there are facial injuries such that mouth-to-mouth breathing is impossible. The head tilted back as in the first. With the hand on the chin, the lower jaw is raised but also pushed towards the upper jaw to close the patient's mouth. They

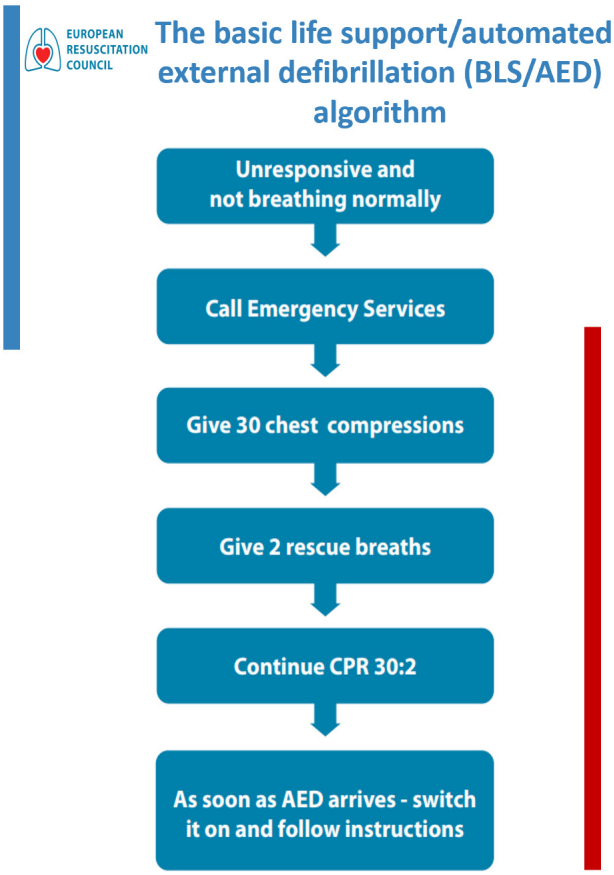

Figure 7. Basic life support using an automatic external defibrillator/AED). Source: European Resuscitation Council Guidelines for Resuscitation 2015.

take a deep breath, cover the patient's nose with their lips and inhale the required volume of exhaled air into the patient's lungs. When exhaling, open the patient's mouth for easier spontaneous exhalation of air. The patient's chest is monitored to assess the effectiveness of artificial respiration and the adequacy of the volume of air delivered. Inhalation rate required volume of exhaled air, and respiratory rate are the same as in mouth-to-mouth breathing.

Today, first aid kits usually contain masks or tubes through which resuscitation can be successfully performed without endangering the health of the rescuer.

Mouth to mask resuscitation technique: The pocket mask can be used by trained non-professional personnel; it does not improve the efficiency of ventilation compared to the mouth-to-mouth technique. The advantage is the enrichment of the insufflation air with oxygen (O_2) 50-100%. Masks should be transparent (detection of cyanosis and vomiting) with an O_2 application connector and a strap for attaching to the head.

Valve balloon and Ambu mask resuscitation technique: applied when the patient is apneic or breathing insufficiently. An oxygen supply (O_2) of 10-15 L can be connected to the balloon to achieve a FiO_2 of 0.5. A tank that increases FiO_2 to 0.9 (90%) can also be connected. Masks should be transparent, of various sizes. Technique: a) an oropharyngeal airway is placed on the patient b) a mask is placed over the patient's mouth and nose, fastened with one hand, the head is thrown back slightly, and air is in-

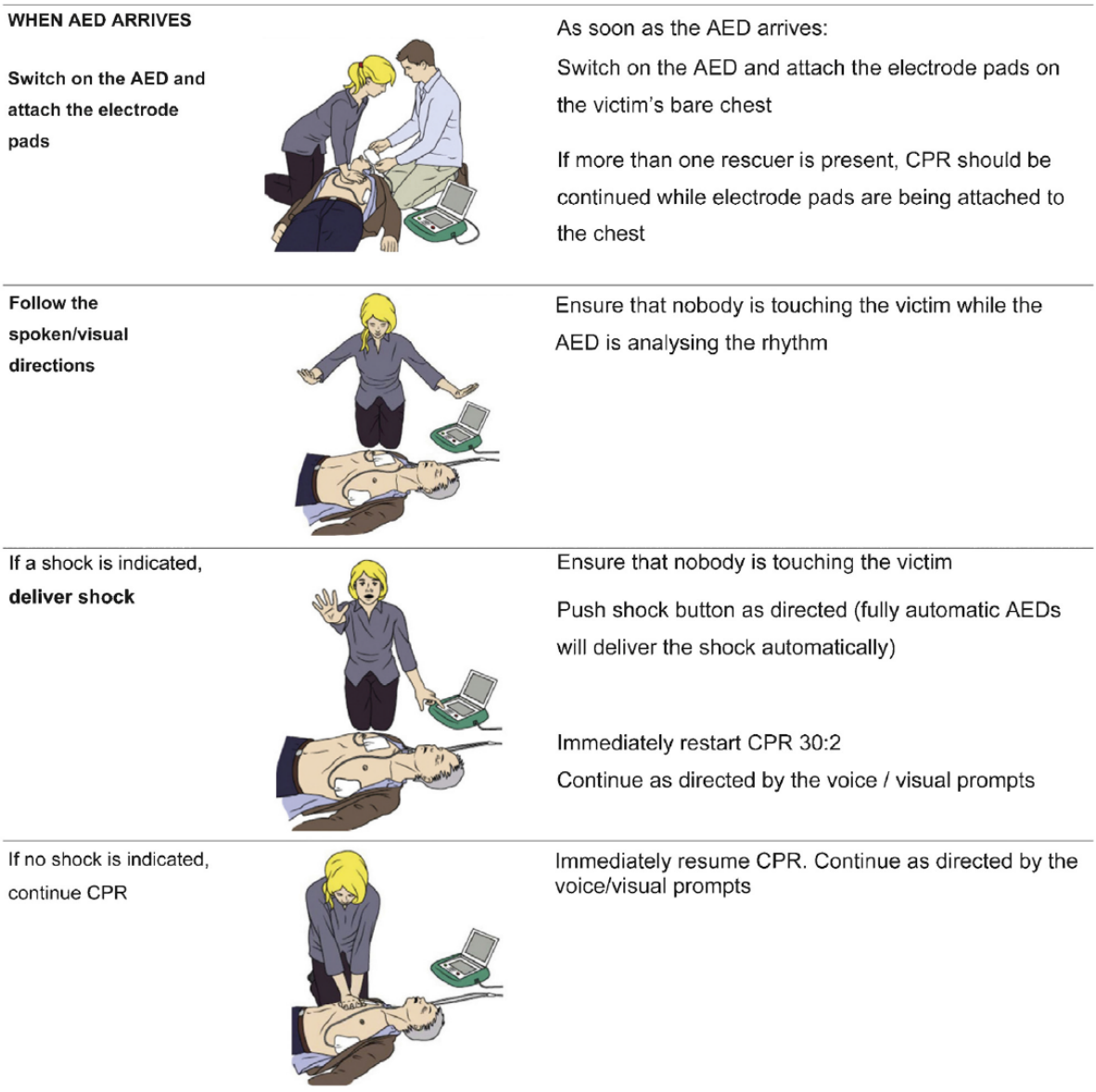

Fig. 2.4. (*Continued*).

Figure 8. Defibrillation mode using an automatic external defibrillator (AED). Source: European resuscitation Council Guidelines for Resuscitation 2015.

sufflated by pressing the balloon until the patient's chest is raised spontaneously and the pressure on the balloon is released (1, 5, 7).

Laryngeal Mask (LMA) resuscitation technique: The laryngeal mask (LMA) is placed blindly, without laryngoscopy. When placed, the tip is in the upper part of the esophagus, and the inflatable cuff clings around the glottis. It is designed for patients who breathe spontaneously but can be used in artificial ventilation if the airway pressure does not increase above 25 cm of water column (H_20). Advantages of LMA placement: can be placed while the neck is in neutral position and with collar, artificial ventilation is better than over the mask, easier to place, and placement skill is easier to learn, aspiration prevention is important although not absolute, placement is usually successful when assumes difficult intubation, no muscle relaxants are required for placement.

LMA placement technique: cuff is blown out and the back of the mask is smeared with lubricant, the LMA is held in the dominant hand using the index finger as a guide, the mouth is opened, and the LMA is placed behind the upper incisors along the hard palate with the open side facing the tongue but not touching the tongue, the mask is further placed towards the pharynx using the index finger, when the index finger can no longer be forward, the mask is held with the other hand and the index finger is removed, then the mask is still placed until resistance is felt and finally cuff is inflated.

Mechanical ventilation: There are smaller portable mechanical ventilators with adjustable pressure that are successfully used for ventilation of intubated patients in ambulances. For controlled ventilation of a comatose patient, when it is impossible to determine gases in the blood, ventilate with oxygen with a respiratory volume of approximately 10 ml/kg (700 ml per 70 kg), with a frequency of about 12/minute (Figure 5).

The presence of a foreign body in the airway

When the patient is not breathing after airway release, and we cannot even ventilate he/she by resuscitation (mouth to mouth, mouth to nose, etc.), it is reasonable to suspect airway obstruction by a foreign body. A foreign body can be removed from the airway in several ways:

a) **Fingers-**but we must be careful, especially with children, not to push it into the deeper airways. We can imitate tweezers (forefinger + middle finger) or hook (bent forefinger) with our fingers. If we wrap our finger in gauze or a handkerchief, we will also have the effect of a sponge.
b) **By turning the head to the side**–this way we can allow the liquid to flow out of the mouth (caution when suspecting a cervical spine injury)
c) **Back blows or Heimlich's maneuver–**in a conscious patient or chest compressions in an unconscious patient. Namely, we can help a conscious patient who is suffocating due to a foreign body in the airway to expel a foreign body with 5 strong blows with the palm of the hand in the back and / or with 5 strong pressures on the abdominal wall (Heimlich's maneuver).

Performing the Heimlich maneuver:

In the early 1970s, the American surgeon Henry J. Heimlich, after whom the maneuver was named, noticed that people who suffocate often could not be helped only by hits to the back. The Heimlich maneuver is performed as follows: 1. If the person choking is sitting and fully conscious, tell him to stand up. Let him know that you will help him with Heimlich's maneuver. 2. Stand behind the victim, tilt him slightly forward and place one arm across the person's chest for support. Bend the person over at the waist so that the upper body is parallel with the ground. Deliver five separate back blows between the victim's shoulder blades with the heel of your hand. 3. Then apply the Heimlich maneuver: Stand behind the person. Place one foot slightly in front of the other for balance. Wrap your arms around the waist. Tip the person forward slightly. If a child is choking, kneel behind the child. Make a fist with one hand. 4. 4. Place it in the abdominal cavity of the victim, just slightly above the person's navel. 5. Grasp the fist with the other hand. Press hard into the abdomen with a quick, upward thrust–as if trying to lift the person up. 6. Give five abdominal thrusts. Repeat until the object is ejected and the victim can breathe or cough on their own.

If the victim lies on its back and cannot get up, then the head should be turned to the side, and the pressure on the chest is performed abruptly, as in a heart massage. In the case of children or infants, they grab their legs with their head turned down and, by pressing their fingers on their chest or light blows to the back, expel the foreign body from the airways (4, 5, 7) (Figure 6).

Defibrillation

The most common arrhythmia in adult cardiac arrest is ventricular fibrillation (VF), so the most important is the urgent conversion to a rhythm that maintains perfusion. The same approach is for ventricular tachycardia (VT) without a pulse.

The precordial thumb (single sharp blow to the patient's mid-sternum using the medial aspect of a clenched fist from a height of 20–30 cm."). It is advised only if a defibrillator is not available. A sharp blow to the precordium can lead to the mechanical conversion of VF or VT to a normal heart rhythm. However, it is not recommended for children. One or two strokes are directed between the middle and lower third of the sternum with a clenched fist 20-35 cm away from the chest.

Defibrillation involves the passage of electricity through the heart muscle, most often through the chest wall, with the intention of interrupting the most dangerous heart rhythm disorders-ventricular fibrillation (VF) or ventricular tachycardia without pulse (pVT). It is assumed that these heart rhythm disorders are the cause of cardiac arrest in about 80% of cases. The basis of the physiological action of defibrillation is that the externally supplied current, if strong enough, simultaneously depolarizes most of the heart muscle (about 75% or more) and thus allows the sinus node to regain control of a calm or electrically discharged heart. The likelihood of a sinus node taking control of cardiac function after defibrillation is most causally related to the duration of VF, which is why defibrillation is used today at the first aid level (previously it could only be performed by medical staff).

Emergency direct current (DC) cardioversion is more effective than antiarrhythmics; the success of defibrillation, however, depends on time and decreases by about 10% with each minute of duration of VF (or VT without pulse). An automatic external defibrillator (AED) also allows poorly trained rescuers to interrupt VT or VF. Their availability to the first to arrive (police and firefighters) and in public places seems to increase the rate of successful resuscitations.

Only defibrillation can interrupt a fatal heart rhythm disorder. Survival has been shown to increase significantly if resuscitation and defibrillation are started within 3-5 minutes of cardiac arrest.

There are two types of defibrillators: AED or automatic external defibrillator and manual external defibrillator (used by emergency medical teams and hospital health workers). Without prompt intervention by immediate eyewitnesses, the survival rate for sudden cardiac arrest is less than ten percent, while intervention within three to five minutes from the moment of cardiac arrest increases the chance of survival to more than 50%. Therefore, knowledge of the skills of external cardiac massage and artificial respiration and the use of an automatic external defibrillator (AED) is extremely important. A person who has experienced a sudden cardiac arrest can also be helped by lay people without medical knowledge, thus saving his life in the fateful minutes until the arrival of the emergency medical service. The AED device is easy to operate, it assesses the heart rhythm and gives instructions in the national language for further treatment (deliver or not an electric shock) (1, 2, 12, 13) (Figure 7).

Automatic External Defibrillator (AED)

During resuscitation, in most cases, we use external defibrillation-defibrillation with a closed chest. Lay people (non-medical staff) use the automatic external defibrillator (AED). AED is a lightweight, simple, inexpensive, portable device that delivers electric shock through the chest to the heart. Automatic external defibrillators have microprocessors that analyze several features of the ECG, including frequency and amplitude. Some AEDs are programmed to detect spontaneous movements of patients or others. The development of technology will soon allow AEDs to provide information on the frequency and depth of external cardiac massage during CPR, which can improve the resuscitation performed by all rescuers. Automatic external defibrillators have been extensively tested on stored and recorded rhythms and in many trials in adults and children. The sensitivity of HF detection by AED is 96-100% and the specificity is 100%, so they are extremely accurate in rhythm analysis. AED is a computerized defibrillator that can:

- **Analyze heart rhythm.**
- Recognize the rhythm that requires electric shock and
- Advise the rescuer (by voice and/or written message, and sometimes by a light signal) when an electric shock is required.

AEDs deliver low-energy biphasic electroshocks that automatically adjust to measured values of resistance to the passage of current through the chest wall. These are relatively inexpensive devices that are easy to maintain and that laymen learn to use after a short workout. Use by laymen enables the so-called early defibrillation, i.e., defibrillation within 5 minutes of emergency medical services (EMS) notification of cardiac arrest. That is why today AEDs are found in airplanes, in squares, stadiums, in public buildings, nursing homes or in workplaces with a lot of people (3, 4, 5).

AED is used in the following manner:

1. Place self-adhesive paddles to the patient's chest according to the attached picture.
2. Turn on the device.
3. Basically, the AED itself analyzes the rhythm as soon as it turns on, and for some it is necessary to press the ANALYSE button. During the analysis, which lasts about 10 seconds, resuscitation and transport of the patient should be stopped!
4. If the rhythm analysis determines that defibrillation is required, most AEDs automatically charge themselves, and some require you to press the CHARGE button after a voice message or message on the display.
5. While charging the defibrillator, a message appears on the display to move everyone away from the patient or the same message is broadcast by voice. If a shock is to be delivered, we must make sure that no one, including us, touches the patient person during the delivery of the shock.
6. After charging is complete, the device warns us that we may defibrillate. By pressing the SHOCK button, defibrillation is performed, after the patient's environment has been previously checked. After that, the device will guide us with voice messages through the entire resuscitation process!
7. If the AED says the shock should not be delivered, the voice messages it provides should be followed.
8. Resuscitation should be applied until an ambulance arrives, the person begins to show signs of life (such as coughing, moving, opening the eyes, and breathing normally, etc.) or until we become tired.
9. After the resuscitation procedure, the portable AED defibrillator returns to charge to its original location from which it was taken.

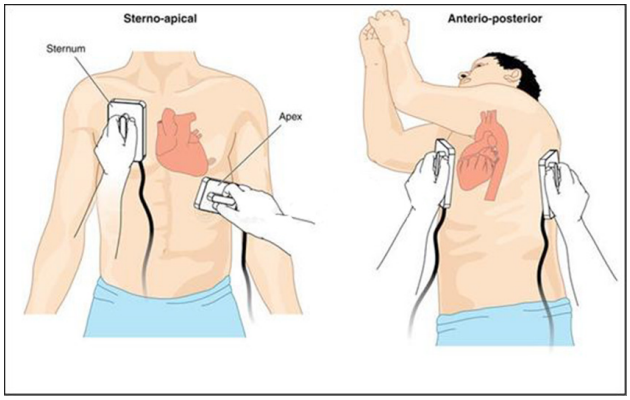

Figure 9. Demonstration of 2 paddles position when using a conventional defibrillator. Source: dreamstime.com.

The advantage of the AED device is that it can also be used by lay people who, through short and targeted education, successfully master its use. So, the use of an AED is not reserved for healthcare professionals-anyone can be educated to use an AED. Numerous studies have shown that AEDs are simple and safe enough devices that they can be used by anyone, even a child older than 7 years. An educated layman can in no way intentionally or unintentionally harm someone by using an AED (3-5).

Manual external defibrillator

Manual defibrillator defibrillation may only be performed by trained medical personnel. The paddles must be coated with an electrolyte gel to reduce the resistance between the paddles and the skin. The gel is placed on one paddle and then rubbed against the entire surface of the metal disk. When placing the paddles on the chest, the defibrillator selector must be turned to the ECG position. When performing defibrillation, the paddles must be firmly attached to the skin of the chest. An ECG can then be read on the defibrillator screen.

Monophasic or biphasic defibrillators are used for defibrillation purposes. Unlike monophasic, biphasic defibrillators deliver an electric shock that goes in a positive direction over a period of time before returning in a negative direction for the remaining few milliseconds. When using manual defibrillators, the efficiency of the first defibrillation shock in PF / VT without pulse is higher when

using biphasic than monophasic form of defibrillation wave and therefore newer defibrillators with biphasic pulse wave are recommended (1-7).

Paddle placement and defibrillation

The defibrillator for adults has two round paddles measuring 8-12 cm. One paddle is placed externally in relation to the apex of the heart, on the middle axillary line at the level of the V6 electrode of the ECG, i.e., at the level of the left mammilla, and the other paddle on the right side of the chest just below the clavicle (collarbone) in the right second intercostal space. Larger paddles reduce chest resistance, but care should be taken again not to be too large. They must not be connected. Pediatric electrodes are usually 4.5 cm in diameter and can also be used for infants.

The antero-lateral paddles position is most used in outpatient settings. One paddle (usually marked or marked with STERNUM) is placed below the patient's right clavicle along the sternum and the other paddle (also marked and usually written APEX) is placed in the mid-axillary line of the other side of the patient's chest approximately at the V6 paddle position. which we use when we do a 12-channel ECG. This is the standard position of the defibrillation paddles. Of course, we can also use self-adhesive paddles by placing them in the same or antero-posterior position.

When performing defibrillation, the defibrillator must be turned on, remove the paddles from their sockets on the device, adjust the energy to the desired level (e.g. 360 J), lubricate the surface of the paddles with gel as good contact between the paddles and the skin is essential gel does not form a current between the electrodes over the patient's skin (burns), the paddles are placed so that the positive paddle (APEX) above the top of the heart, and the negative paddle (STERNUM) below the right clavicle, the paddles are pressed firmly to the chest using body weight 6-7 kg per paddles and check the ECG on the monitor, warn the attendees to move away from the patient and the metal parts of the bed and then deliver DC shock by pressing the buttons located on the paddles handles, check the ECG curves and pulse on the carotids. After establishing the heart rhythm, measures of support with medication and care of the patient in hospital conditions are approached (1-7).

Energy of the electric shock:

Single-phase defibrillators: 360 J (first defibrillation and all other defibrillations)

Biphasic defibrillators: 150-200 J (for the first defibrillation) and 150-360 J (for the second defibrillation as well as all the others), i.e., the same or higher energy is applied, depending on the capabilities of the device.

In the ERC guidelines from 2015, the level of delivered energy used is unchanged compared to 2010. When delivering the first biphasic defibrillation wave, the recommended energy is at least 150 J. The consensus determined energy level for the second and subsequent biphasic defibrillation shocks is in the range of 150-360 J. If the initial electric shock was unsuccessful, the second and subsequent defibrillations are performed with higher energy levels. The energy applied to each biphasic defibrillator must be based on the manufacturer's instructions-clearly indicated on the defibrillator cover. The new recommendations do not even mention single-phase defibrillators because they are considered to have been replaced by biphasic ones in most EU countries. Since this is still not the case in our hospitals, the recommendations from 2010 apply to the use of single-phase defibrillators, the first and each subsequent defibrillation are performed with a current of 360 J.

The defibrillation strategy in the 2015 ERC guides has changed slightly from 2010. Defibrillation should be performed as soon as possible without routine CPR measures except when necessary (e.g., in drowning, in hypoxia, when the defibrillator is not immediately available). Defibrillation should not be delayed longer than necessary to prepare and charge the defibrillator.

One shock strategy: immediately after the first defibrillation, without checking the rhythm and palpation of the carotid pulse, CPR–5 cycles are continued for two minutes, before defibrillating a second time, if indicated. Even if the first defibrillation was successful, rarely will the pulse immediately after defibrillation be palpable and therefore palpation of the pulse is a waste of time. Chest compression will not worsen any established perfusion heart rhythm. If a perfusion rhythm is established, chest compression will not increase the possibility of VF recurrence.

Early defibrillation within 3-5 minutes of loss of consciousness ensures a survival success rate of 50-70%. This can be achieved with the availability of AEDs in public places. AED represents the greatest technological advancement in resuscitation medicine. With the introduction of AEDs, defibrillation has become an integral component of basic life support measures (BLS) and can be used by lay people and non-medical staff in hospital and outpatient settings. In that sense, a program of mass application of defibrillation (PAD program–Public Access Defibrillation) has been developed in Europe. The use of early defibrillation has increased the survival rate from 7 to 19%, depending on the individual areas of application in the world and in our country. The success rate of resuscitation in hospital conditions with classic DC devices and CPR kits is achieved in 20-50%, and with new AED devices and 50-80%. The success rate of outpatient resuscitation with classic equipment of emergency medical services is 2-5%, and with AED devices up to 20%. It is necessary to develop and implement a program of publicly available early defibrillation, which would have three main goals: Public health promotion of the importance of early defibrillation in cases of sudden cardiac arrest, and public health education to raise awareness and involve the general public in the program, increasing the availability of AEDs (AED networks) in places with high population density and educating as many lay people as possible to carry out resuscitation procedures using AEDs (1-7) (Figures 8 and 9).

Advanced Adult Life Support (ALS)

Important notes in the guidelines on advanced life support are the use of a rapid response system for patient care, minimal cessation of chest compressions, and the use of self-adhesive electrodes instead of pedals. The recent guidelines also refer to monitoring during advanced life support with an emphasis on the use of wave capnography, to detect the return of spontaneous circulation (ROSC), monitor the quality of chest compressions and validate the position of the endotracheal tube. The algorithm itself (Figure No.) begins by determining the absence of vital parameters followed by a call from the resuscitation team. Cardiac arrhythmias 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)). is the need to attempt defibrillation in patients with VF / pVT. The team assesses the patient's rhythm on the monitor and decides whether it is a defibrillation rhythm **(shockable rhythm)** or a non-defibrillation rhythm **(non-shockable rhythm) (2, 5, 7).**

If it is a case of rhythm suitable for defibrillation, the goal is to deliver DC shock to the patient's heart as soon as possible with as few interruptions of compression to the chest as possible. After the defibrillation rhythm is repeated three times and DC shock is delivered, adrenaline is repeated every 3-5 minutes and amiodarone is applied once. In the case of a rhythm that is not for defibrillation, compressions to the chest and blowing air are continued, and adrenaline is applied, which is repeated every 3-5 minutes.

Regardless of the patient's heart rhythm, the goal is always to make as few interruptions in chest compressions as possible during resuscitation, to adequately secure the airway as early as possible, preferably with an endotracheal tube, and to monitor the patient's compression and ventilation performance. After the return of spontaneous circulation, it is necessary to adequately care for the patient by following the guidelines for post-resuscitation care of the patient, which achieves longer survival, but also to find and treat the cause of cardiac arrest so that it does not recur (Figure 10).

Shockable rhythm and non-shockable rhythm

Treatment of the shockable rhythm

During cardiac arrest, two types of shockable rhythm that require defibrillation can be registered: 1. Ventricular fibrillation (VF). 2. Pulseless ventricular tachycardia (pVT).

Ventricular fibrillation (VF) is a rhythm disorder characterized by rapid, chaotic electrical impulses with incomplete ventricular contractions leading to loss of pulse and blood pressure. FV occurs in 70% of patients in cardiac arrest and is therefore a terminal event in many cardiac and other disorders. VF/ pVT will also occur at some stage during resuscitation in about 25% of cardiac arrests with an initial documented rhythm of asystole or PEA. In general, most patients with VF have underlying heart disease (usually ischemic, but also hypertrophic or dilated cardiomyopathy, arrhythmogenic right ventricular dysplasia (ARVD), or Brugada syndrome). The risk of developing VF in any heart disease is increased with electrolyte disorders, acidosis, hypoxemia, or ischemia. The easiest way to remember the most common causes of HF are the abbreviations H6 and T6.

Adrenaline (Epinephrine) has been a key component of advanced life support algorithms for many years. Its mechanism of action–stimulation of α1 receptors in vascular smooth muscle–causes vasoconstriction. This increases diastolic pressure in the aorta, which increases coronary perfusion pressure (CPP) and cerebral perfusion pressure (CePP). CPP is strongly associated with the return of spontaneous circulation (ROSC).

Treatment of VF/pVT primarily involves cardiopulmonary resuscitation combined with early defibrillation. The amount of energy delivered through biphasic defibrillators is 150-360 J.

- It is necessary to perform continuous chest compressions. Place electrodes or self-adhesive defibrillation electrodes at the defibrillation sites: one Cap below the right clavicle and the other in the V6 position in the mid-axillary line.
- Stop chest compressions; confirm VF/pVT on the monitor. This break in chest compressions should be short and not longer than 5 seconds. The more you shorten the time from stopping chest compression because even a delay of 5–10 seconds. reduces the chances of defibrillation being successful.
- Select the amount of energy to be delivered (150-300 J for biphasic defibrillators) and after the safety check is completed deliver the first defibrillation. The user can follow the manufacturer's instructions for a specific defibrillator. If you are not sure what amount of energy is needed, then choose the highest available energy on the defibrillator.
- After delivery of DC shock, without pause to check rhythm or pulse, continue with CPR using a ratio of 30:2 immediately after the first defibrillation, continue with chest compression to limit post shock break and total peri shock break time (which should not be longer of 5 seconds).
- Continue CPR for 2 min., then take a short pause to check the rhythm: in case VF / pVT persists, perform a second defibrillation (150-360 J biphasic defibrillator). Without a pause to check rhythm or pulse, continue CPR using a 30: 2 ratios immediately after DC shock.
- After CPR for 2 minutes, take a short break to check the rhythm: in case VF / pVT persists then deliver a third defibrillation (150-360 J biphasic defibrillator).
- If there is no effect, without checking rhythm or pulse, continue CPR (30:2 ratio) and if an in-

travenous route is established give adrenaline 1 mg (repeated every 3-5 minutes) or 300 mg of Amiodarone once.

- If the pulse is not present, continue with CPR. A second dose of 150 mg amiodarone is administered after the fifth (5) failed defibrillation, during the two-minute CPR that follows.
- The interval between compression stop and shock delivery must be minimized. Prolonged chest compressions reduce the possibility of shock by restoring spontaneous circulation. Chest compressions continue immediately after shock delivery (without rhythm or pulse check), because even if a defibrillation attempt is successful in restoring perfusion rhythm, it is very rare for the pulse to be palpable immediately after defibrillation. The duration of asystole before ROSC can be longer than 2 min in as many as 25% of successful shocks. If the shock was successful, immediate resumption of chest compression does not increase the risk of VF recurrence. Furthermore, delaying the attempt if you palpate the pulse will further compromise the myocardium if no perfusion rhythm is established.
- The use of a capnography curve can allow the detection of spontaneous circulation return (ROSC) without pausing chest compression. Several studies in humans have shown that there is a significant increase in the final respiratory volume of carbon dioxide (ETCO2) when ROSC occurs. If ROSC is suspected to exist during CPR, do not give adrenaline. Give adrenaline if cardiac arrest is confirmed at the next heart rate control.
- According to the 2015 ERC guidelines, in the event of VF/pVT patients being monitored and in the presence of a healthcare professional (catheterization room, coronary unit, Intensive Care Unit or monitoring after cardiac surgery) in case a manual defibrillator is immediately available: Confirm cardiac stop and call for help. In case the initial rhythm of VF / pVT is to perform three shocks one after the other. Quickly check rhythm after each shock if ROSC occurs discontinue defibrillation. In case the third defibrillation is also unsuccessful, continue with CPR for the next 2 min. Although there is insufficient data to support the strategy of three consecutive shocks, in any situation, it does not appear that chest compression will increase the chances of ROSC compared to defibrillation performed early in the electrical phase, immediately after VF.
- Regardless of the rhythm of cardiac arrest, after giving the initial dose of adrenaline, give further adrenaline (epinephrine) in a dose of 1mg every 3-5 minutes until ROSC is achieved, in practice it will be approximately once in two cycles of this algorithm. If signs of life return during CPR (e.g., conscious body movements, normal breathing, or coughing) or there is an increase in final respiratory volume of carbon dioxide (ETCO2), check the monitor; if an organized rhythm is present, check the pulse. If the pulse is palpable, start post-resuscitation care. If there is no pulse, continue with the CPR procedure.
- Give amiodarone 300 mg intravenously after three (3) defibrillation attempts whether consecutive shocks or CPR with defibrillation interruptions. Consider a further dose of amiodarone 150 mg i.v. after a total of five (5) defibrillation attempts. Lidocaine 1 -1.5 mg/kg can be used as an alternative if amiodarone is not available, but do not give lidocaine if amiodarone is already administered.
- If rhythm changes to asystole or pulseless electrical activity (PEA), continue resuscitation according to a non-shockable algorithm (2, 5, 7, 12).

Treatment of non-shockable rhythm

Heart rhythms in the case of cardiac arrest that are not shockable are asystole and pulseless electrical activity (PEA).

Recommendations for the treatment of asystole

- According to the algorithm for advanced resuscitation, first the patient's rhythm should be checked, it takes less than 10 seconds to assess.
- It is necessary to check the presence of asystole on at least two leads.
- Continue CPR at a compression rate of 100-120 per minute. Rotate team members every 2 minutes to maintain high CPR quality.
- If a medical team that is educated and experienced is present, the patient should then be intubated.
- Check the heart rhythm.
- If there is no electrical activity (the patient is in asystole), continue with CPR.
- During CPR, look for and treat possible causes.
- Allow intravenous route, give vasopressors: adrenaline or epinephrine 1 mg i.v. or i.o. each 3 -5 minutes.
- Do not stop CPR to administer medications.
- If electrical activity is present, see if the patient has a pulse.
- If the patient has no pulse or there is any suspicion of pulse, continue with CPR.
- If there is no effect, the patient can be connected to a temporary external pacemaker.
- If a good pulse is present and the rhythm is organized, start with post-resuscitation care.
- The intravenous approach is a priority over advanced airway management. If an advanced

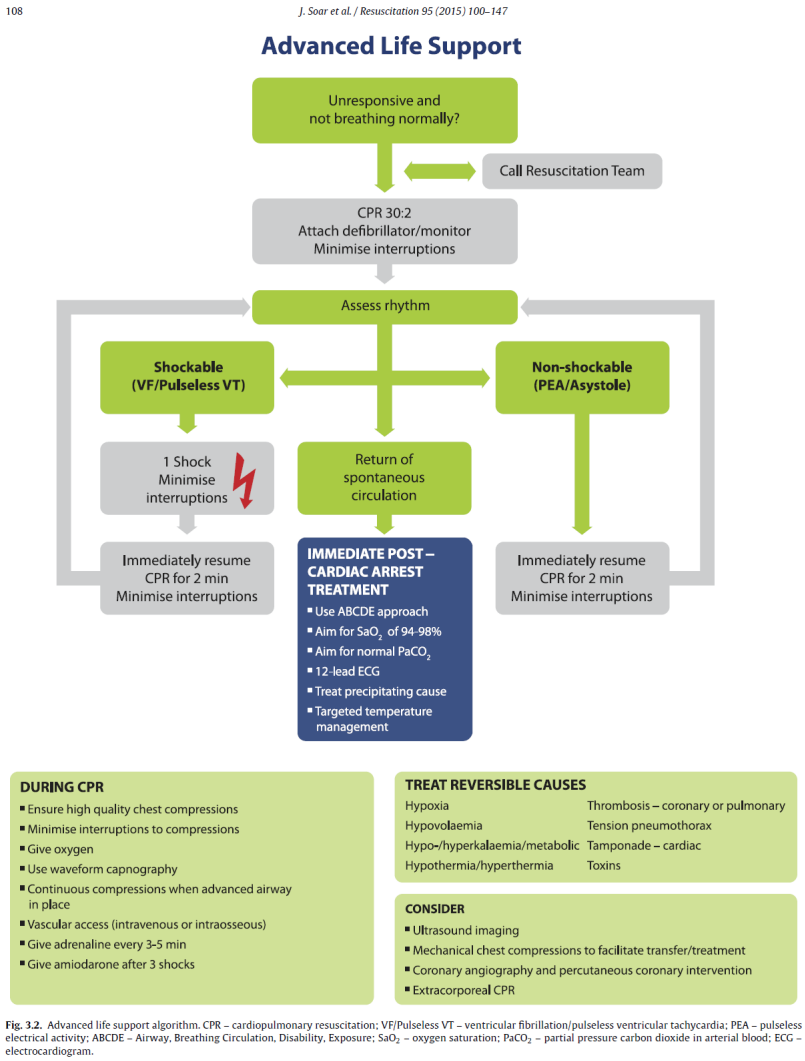

Figure 10. Advanced Life Support. Source: European Resuscitation Council Guidelines for Resuscitation 2015.

airway is placed, switch to continuous chest compressions without a pause for inhalation. Give 10 breaths per minute (once every 6 seconds) and control your heart rate every 2 minutes.

- If a VF appears on the monitor during a two-minute CPR cycle, complete the CPR cycle and deliver DC shock if necessary, this strategy will minimize the interruption in chest compression.
- In case you are in a dilemma whether it is a case of fine fibrillation or asystole (sometimes it is difficult to distinguish them), do not perform defibrillation, but continue with chest compression and ventilation. Fine VF cannot be defibrillated and performing chest compression can increase the amplitude and frequency of HF and increase the chance of successful defibrillation and perfusion rhythm.
- If a VF/pVT occurs during a CPR during a CPR, then follow the recommendations and algorithm for treating the shockable rhythm (1-7) (Figure 11).

Recommendations for the PEA treatment

Pulseless electrical activity (PEA) occurs when you see a rhythm on the monitor that would otherwise be related to the pulse, but the patient is pulseless. Patients with PEA have a poor prognosis. These patients often have some mechanical myocardial contractions, but they are too weak to produce a detectable pulse or blood pressure. Their best chance to return to the perfusion rhythm is quick identification of the underlying reversible cause and correct treatment. While using the algorithm to manage a patient with PEA, remember to consider all H6 and T6 as the most common causes of asystole and PEA. Also look for drug overdoses or poisonings.

Guidelines for CPR (ALS/BLS) advise that cardiopulmonary resuscitation begins immediately to maintain cardiac output until PEA is treated. PEA can be caused by reversible conditions that can be treated if they are identified and treated. Survival after cardiac arrest with asystole or PEA is unlikely unless a reversible cause is found and effectively treated.

The approach to treating PEA is to treat the underlying cause, if known (e.g., treatment of tension pneumothorax). If the underlying cause of PEA cannot be identified and / or eliminated, treatment of pulseless electrical activity is like that for asystole. There is no evidence that external cardiac compression can increase cardiac output in any of the many PEA scenarios, such as bleeding, in which cardiac filling disorder is the underlying mechanism that creates a detectable pulse loss.

There are two priorities during PEA management: 1. Maintaining high quality CPR and 2. Simultaneously looking for a cause that can be remedied or adequately treated. The priority in resuscitation is to provide an intravenous route for drug administration. The main drug therapy for PEA is adrenaline (epinephrine) at a dose of 1 mg every 3-5 minutes. Although atropine was previously recommended for the treatment of PEA/asystole, this recommendation was withdrawn by the American Society of Cardiology in 2010 due to a lack of evidence of therapeutic benefit. Adrenaline also has limited scientific evidence, so it is recommended based on its mechanism of action. Sodium bicarbonate 1meq/kg may also be considered at this rate, although there is little evidence to support this

practice. Its routine use is not recommended for patients in this context, except in special situations (e.g., pre-existing metabolic acidosis, hyperkalemia, overdose of tricyclic antidepressants). All these medications should be administered in conjunction with appropriate CPR techniques. CPR is performed at a rate of 100 to 120 per minute during continuous resuscitation lasting more than 5–10 seconds to assess rhythm and pulse. Ventilate the patient with an Ambu mask at a frequency of 10/minute. If pulse and/or signs of life appear, start post-resuscitation care, if there is no pulse and / or no signs of life (PEA or asystole) CPR should be continued. If the patient during CPR develops VF/pVT during the rhythm check, then it is necessary to follow the recommendations for the treatment of shock rhythm (2, 4, 11, 12, 15).

Extracorporeal cardiopulmonary resuscitation (ECPR)

Extracorporeal cardiopulmonary resuscitation (ECPR) should be considered life-saving therapy in patients in whom initial advanced resuscitation measures are unsuccessful, and/or to facilitate specific interventions (e.g., coronary angiography and percutaneous coronary intervention (PCI) or pulmonary thrombectomy in massive pulmonary emboli). ECPR refers to venoarterial extracorporeal membrane oxygenation (ECMO) during cardiac arrest. Extracorporeal technique requires a vascular approach and thus can ensure oxygenated blood circulation to restore tissue perfusion. This made it possible to buy time to establish proper spontaneous circulation and treat reversible underlying causes. This technique is becoming increasingly common and is used in hospitals and outpatient settings despite limited observational data in selected groups of patients. Observational studies show that eCPR in cardiac arrest is associated with improved survival when there is a reversible cause of heart failure (e.g., myocardial infarction, pulmonary embolism, severe hypothermia, poisoning) especially in individuals who have high quality CPR immediately and when ECPR is administered early 1 hour from the event). Implementing ECPR requires significant resources and training. Compared with manual or mechanical CPR, ECPR was associated with improved survival in selected patients who had intrahospital cardiac arrest (IHCA). Outcomes in outpatient cardiac arrest (OHCA) with standard and ECPR are less favorable. The duration of standard CPR and patient selection are important success factors prior to the ECPR resuscitation method (20).

Post resuscitation care

Post resuscitation care is a critical component of advanced life support. Most deaths occur within the first 24 hours after cardiac arrest. More than 60% of those successfully resuscitated do not experience discharge from the hospital. The goal of post-resuscitation care after cardiac arrest is to optimize systemic perfusion, restore metabolic homeostasis, and support organ function to increase the likelihood of intact neurological survival. The period after cardiac arrest is often marked by hemodynamic instability as well as metabolic disorders. Support and treatment of acute myocardial dysfunction and acute myocardial ischemia may increase the likelihood of survival. Interventions to reduce secondary brain injury, such as therapeutic hypothermia, can improve survival and neurological recovery. Every organ system is at risk during this period, and patients are at risk of developing multiorgan dysfunction (2, 6, 9, 10).

The complex pathophysiological mechanisms that result from generalized ischemia during cardiac arrest and the consequent reperfusion response that occurs after successful resuscitation are referred to as Post Cardiac Arrest Syndrome (PCAS). Such patients need multiple organic support in the post-resuscitation period, which has a significant impact on their neurological outcome.

Comprehensive treatment of various problems after heart failure includes multidisciplinary aspects of intensive care, cardiology, pulmonology, hematology, and neurology. For this reason, it is important to admit patients to appropriate intensive care units with a comprehensive care plan that will anticipate, monitor, and treat each of these different problems (21, 22).

ACS is a common cause of heart failure. The clinician should evaluate the patient's 12-channel ECG and cardiac markers after ROSC. Urgent treatment of ST-elevation myocardial infarction (STEMI) should be initiated as in patients with non-cardiac arrest, regardless of coma or induced hypothermia. Recognition of myocardial infarction is urgently important for the urgent application of reperfusion therapy (e.g., thrombolysis, percutaneous coronary intervention). Therapeutic hypothermia can be safely combined with primary PCI after cardiac arrest caused by acute myocardial infarction.

It is necessary to perform laboratory analyzes of blood, from the tests, ABS, complete blood count, electrolytes, blood glucose, urea, creatinine and cardiac markers (Troponin) are determined. PaO_2 should be as close as possible to normal values (80 -100 mmHg). Hematocrits should be ≥ 30, glycaemia should be between 7.7–9.9 mmol/L, and electrolytes, especially potassium, should be within the reference range. In the new ERC 2015 guidelines for CPR, there is a recommendation for glycemic control in the post-arrest period: in adults after acute cardiac arrest, hyperglycemia ≥10 mmol/L must be treated, but hypoglycemia must be avoided.

Blood pressure support: current recommendations are to maintain a mean arterial pressure (MAP) of > 80 mmHg in elderly adults or > 60 mmHg in younger and previously healthy patients. Blood pressure maintenance support includes i.v. administration of 0.9% saline NaCl, sometimes inotropic or vasopressor drugs, rarely intra-aortic balloon pump (IABP).

Pulmonary dysfunction is common after cardiac arrest. The etiology includes hydrostatic pulmonary edema due to left ventricular dysfunction; noncardiogenic edema from inflammatory, infectious, or physical injuries; severe pulmonary atelectasis; or aspirations that occur during cardiac arrest or resuscitation. Patients often develop a regional mismatch of ventilation and per-

fusion, contributing to a reduced arterial oxygen content. The severity of pulmonary dysfunction is often measured in the PaO_2/FiO_2 ratio. A PaO_2/FiO_2 ratio of ≤ 300 mmHg usually defines acute lung injury. Published studies indicate that patients after cardiac arrest are at risk of acute lung injury and ARDS, but refractory hypoxemia is not a common cause of death after cardiac arrest, and that there is no reason to recommend hyperventilation and „permissive hypercapnia" to these patients (hypoventilation), and normocapnia should be considered standard. There is also no data to recommend a unique ventilation strategy in this population other than the usual care of other mechanically ventilated patients who are at risk for acute lung injury and ARDS. Until recently, it was the rule that the patient in the old age period should be mechanically ventilated in the next 24-48 hours, with sedation and frequent muscle relaxation. After acute cardiac arrest, hypocapnia caused by hyperventilation leads to cerebral ischemia. Routine hyperventilation is detrimental both during Cardiopulmonary Cerebral Resuscitation (CPCR) and in the post arrest period. There is no doubt about the usefulness of 100% O_2 during CPR. Short-term therapy with 100% O_2 is useful and non-toxic. However, during ROSC, routine clinical practice is often present to continue administering 100% O_2 for the next few hours, although arterial blood gas analysis and pulse oximetry do not require it. Post reanimation hyperoxemia is associated with a poor neurological response. Therefore, O_2 saturation (via gas analysis or pulse oximetry) should be monitored as soon as possible to titrate FiO_2 to achieve 94-98% arterial O_2 saturation.

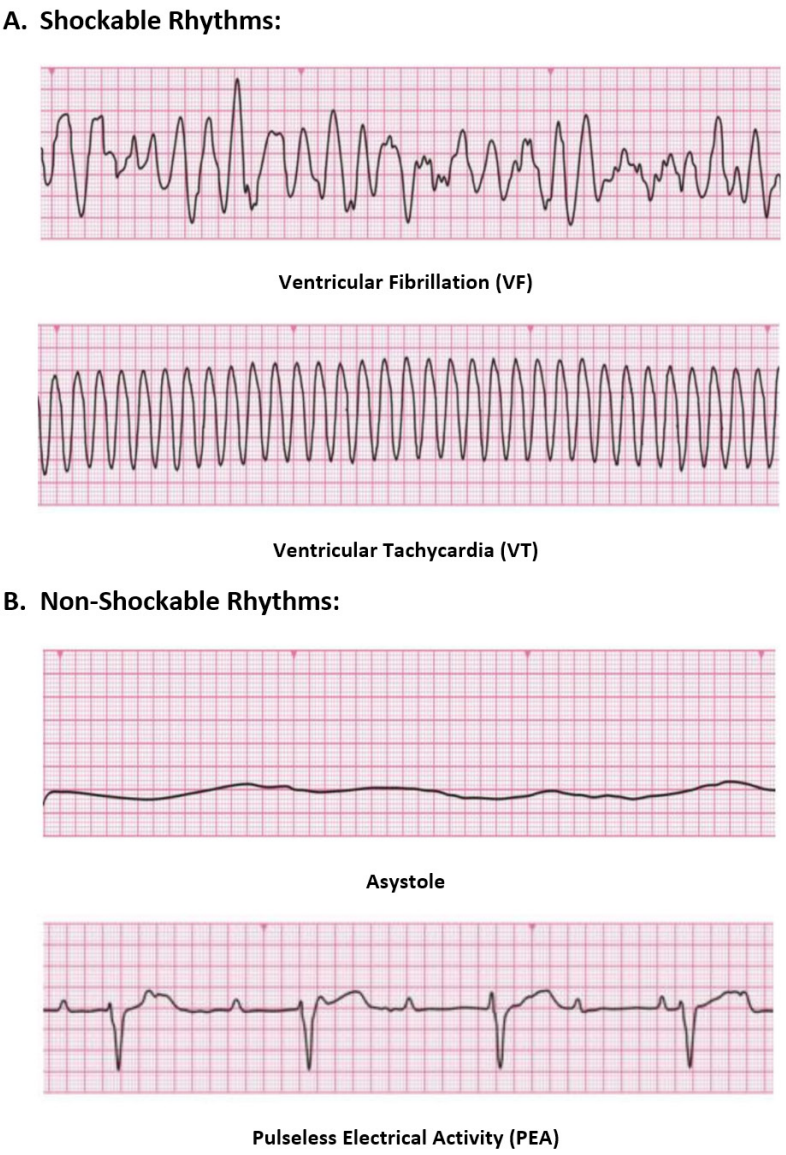

Figure 11. ECG display of shockable (VF, VT) and non-shockable (Asystole, PEA) rhythms.

Brain injury is a common cause of morbidity and mortality in patients after cardiac arrest. Brain injury is the cause of death in 68% of patients after outpatient cardiac arrest and in 23% after nosocomial cardiac arrest. The pathophysiology of brain damage after cardiac arrest involves a complex cascade of molecular events triggered by ischemia and reperfusion that lasts for hours to days after ROSC. Events and conditions in the period after cardiac arrest can potentially worsen or alleviate these pathways of injury and affect final outcomes. Clinical manifestations of brain damage after cardiac arrest include coma, convulsions, myoclonus, varying degrees of neurocognitive dysfunction (ranging from memory deficits to permanent vegetative state), and brain death.

Induced hypothermia to protect the brain and other organs is a useful therapeutic approach in patients after cardiac arrest. Mild hypothermia (32–34 C°) for 12–24 hours after cardiac arrest has a neuroprotective effect and improves the outcome after global cerebral hypoxia. According to the new recommendations, instead of the current term „Moderate controlled hypothermia ", it is more appropriate to use the term „Achieving the target temperature " -TTM (target temperature management) or „temperature control". ILCOR has made several recommendations for TTM accepted by ERC guidelines: Maintain a constant target temperature between 32 and 36 C° for all patients using TTM. TTM is recommended in adult patients after outpatient cardiac arrest with initial shock rhythm who are unconscious after ROSC establishment, in patients after outpatient cardiac arrest with initial non-shock rhythm who are unconscious after ROSC establishment and in patients after nosocomial cardiac arrest with any initial rhythm, who are unconscious after the estab-

lishment of ROSC. In case TTM is used, it is suggested that its duration is at least 24h. External or internal cooling techniques can be used, although more precise control of body temperature is achieved by intravascular cooling. Patients receiving TTM require continuous monitoring and treatment in the intensive care unit due to due to numerous complications that may occur. Be sure to treat shivering with muscle relaxants, sedatives or $MgSO_4$.(1-10)

Implantable Cardioverter Defibrillator (ICD)

An implantable cardioverter defibrillator is an electronic device powered by a special battery like a classic pacemaker that is installed to detect and interrupt malignant, life-threatening heart rhythm disorders. The device cannot prevent the occurrence of dangerous arrhythmias, but the ICD treats arrhythmias in the following ways: stimulation (anti-tachycardia pacing) or delivery of electroshocks (cardioversion, defibrillation), which significantly improves survival in these patients. ICD therapy prevents sudden death and prolongs the life of high-risk patients, provided the patient does not suffer from other conditions that limit life expectancy to 1-2 years.

According to European and US guidelines, indications for ICD implantation are divided into 2 broad categories of patients for secondary or primary prevention of sudden cardiac death:

1. Secondary prevention of sudden cardiac death in patients who have suffered sudden cardiac arrest due to ventricular tachycardia (VT) or ventricular fibrillation (VF), hemodynamically unstable VT, stable VT, unexplained syncope plus inducible permanent monomorphic VT at the electrophysiological study. The results of randomized clinical trials have shown that ICD for secondary prevention is better than antiarrhythmic drug therapy in patients with a history of life-threatening VT/VF.
2. Primary prevention in patients with heart failure as part of ischemic heart disease (ischemic cardiomyopathy) with reduced left ventricular ejection fraction LVEF ≤ 35%, NYHA class II, III despite the use of necessary pharmacological therapy, and in patients with primary dilated hemorrhagic or hypertensive) with reduced left ventricular ejection fraction LVEF ≤ 35%. Randomized trials (MADIT I and II, SCD-HeFT, DANISH) have consistently shown that ICD implantation reduces mortality in both groups of patients (1, 5, 11, 12).

Medications under CPR

Medications that are used in CPR can be divided into three groups: 1. Vasopressors. 2. Antiarrhythmics. 3. Other drugs.

Vasopressors

Adrenaline

According to the 2015 ERC recommendations for CPR in the treatment of cardiac arrest. Adrenaline has a combined alpha adrenergic and beta-adrenergic effects. Alpha-adrenergic effects may increase coronary diastolic pressure, thus increasing subendocardial perfusion during chest compression. Adrenaline also increases the chances of successful defibrillation. However, beta-adrenergic effects can be harmful because they increase oxygen needs (especially of the heart) and cause vasodilation. Intracardiac injection of adrenaline is not recommended because, in addition to interrupting chest compressions, pneumothorax, coronary artery laceration, and development of cardiac tamponade may occur. 1 mg of adrenaline is administered immediately after the establishment of the vascular path in non-shock rhythms, i.e., after the third defibrillation in shockable rhythms, during the chest compression that follows, and then every 3-5 minutes. (i.e., during every other CPR cycle). Adrenaline should be given dissolved in 10 ml of saline to reach the central circulation as soon as possible. Although there is thought to be a benefit in short-term survival (return of spontaneous circulation–ROSC and survival during transport to hospital), there are no reliable data on the beneficial and harmful effects of adrenaline in terms of long-term survival and neurological consequences (1-13).

Vasopressin

The new CPR recommendations mention vasopressin as a future possible alternative to adrenaline. One dose of vasopressin 40 units that has an activity of 40 minutes–its half-life is 10-20 minutes. Vasopressin is a powerful vasoconstrictor that manifests its effects by stimulating the V1 receptors of vascular smooth muscle, while acidosis does not affect its action. Vasopressin is an alternative to adrenaline (adults only). However, it is not more effective than adrenaline and is therefore no longer recommended in the guidelines of the American Heart Association (AHA), however, in case of lack of adrenaline during CPR, vasopressin may be a substitution. New recommendations suggest that vasopressin should not be used instead of adrenaline in cardiac arrest.

Antiarrhythmics

Amiodarone

In terms of the use of antiarrhythmics, the new recommendations for CPR bring essentially nothing new. Amiodarone 300 mg is is given after the third failed defibrillation in refractory VF/pVT shock, increasing short-term survival until hospitalization compared to placebo or lidocaine. Amiodarone 300 mg can be given once if defibrillation is not successful after adrenaline, followed by 1 dose of 150 mg. There is no convincing evidence that this increases survival. Amiodarone should be dissolved in a 20 ml syringe with 5% glucose and given over the vein of a larger lumen due to the possibility of developing thrombophlebitis. The second dose of 150 mg amiodarone is repeated after the fifth failed defibrillation, and at the old age period the continuous infusion can be continued up to a total of 900 mg/24 h. Lidocaine should not be given after amiodarone (12,14).

Lidocaine

Lidocaine is not recommended for routine use during cardiac arrest. However, it may be a useful alternative to

amiodarone for VF or VT that does not respond to defibrillation (in children) or after ROSC due to VF or VT (in adults). Lidocaine is given at a dose of 1-1.5 mg/kg body weight up to a maximum of 3 mg/kg i.v. as a bolus and may recur after 3-5 minutes up to a total dose of 3mg/kg, maintenance therapy is continued within 24-48 hours.

Procainamide is no longer recommended as an antiarrhythmic drug in the new 2015 recommendations.

Phenytoin can rarely be used to treat VF or VT, but only when VF or VT occurs due to digitalis intoxication that is refractory to other drugs. A dose of 50 to 100 mg/minute every 5 minutes is given until the rhythm improves, or the total dose reaches 20 mg / kg.

Other medication: Atropine, Calcium Chloride ($CaCl_2$), Magnesium Sulfate ($MgSO_4$), Sodium Bicarbonate, Thrombolytic Therapy.

Cerebral resuscitation

Between 8 and 20% of patients of cardiac arrest show some degree of brain dysfunction after resuscitation. Brain damage results from hypoxic neuronal lesions and brain edema. These lesions develop within 48-72 h after resuscitation. Cardiopulmonary-cerebral resuscitation represents clearly defined procedures and application of certain medications with the aim of enabling tissue oxygenation in a person with cardiac arrest, establishing spontaneous circulation and performing restitution of brain function and other vital systems in the body. The brain is the organ most sensitive to hypoxia. The brain cannot store oxygen and has a limited capacity for anaerobic metabolism. Permanent brain damage will occur after three to four minutes of complete hypoxia at normal temperatures.

The etiology of brain death includes two groups of brain damage: primary and secondary brain damage. Primary brain damage are traumatic brain lesions, spontaneous intracerebral hemorrhage, ischemic brain lesion, decompensated primary brain tumor and inflammation of the central nervous system. Secondary brain damage is ischemic-anoxic brain damage caused after cardiac arrest, respiratory failure, and prolonged circulatory shock. Irreversible failure of neurological functions sooner or later leads to cessation of respiration, cardiac function, and the onset of death.

The term „cerebral resuscitation" describes a series of measures designed to protect and restore neurological function after acute brain injury. Many of these measures were originally developed for patients with head trauma and other specific conditions but were later applied to a variety of cerebral conditions, including global ischemia and hypoxia, focal ischemia, subarachnoid hemorrhage, and other events of intracranial hemorrhage, metabolic coma, and encephalopathy. The use of such measures assumes that persistent neurological deficits result not only from this and other initial cerebral injuries, but also from secondary changes that occur during the resuscitation period.

Cardiac arrest is a condition of global ischemia and the brain is extremely susceptible to this condition. Only 5-6 seconds after the beginning of the interruption of circulation, the patient loses consciousness. At the end of the first minute, the basal brain functions are interrupted, the respirations become agonal, and the pupils are stiff or dilated. Without a blood supply, the oxygen concentration in the brain tissue is constantly declining and reaches zero after 2 minutes. At the same time, the energy of neurons in terms of glucose and adenosine triphosphate (ATP) is depleted and metabolites, such as adenosine, lactate, and hydrogen ions, accumulate in brain cells. Dysfunction of cell membrane ion pumps leads to severe disruption of cell homeostasis. One consequence is the large accumulation of calcium in the cellular cytosol. This calcium overload is considered a key factor in brain cell intoxication. Irreversible brain changes occur after 4-5 minutes.

If the ischemia lasts long enough, necrosis of the neurons eventually occurs throughout the brain. However, neural energy is rapidly restored after reperfusion due to CPR application and return of spontaneous circulation. The main feature of the reperfusion period is that refilling with adenosine triphosphate gives the cell the ability to actively respond to damage. Thus, reperfusion stops neuronal degeneration to some extent; but this does not necessarily fully restore function. During reperfusion, free radicals are formed when the oxygen supply is restored, which can even worsen cell damage. It is called „post reanimation syndrome "which implies variability of flow since in the first hour after reperfusion, reactive hyperemia is accompanied by a global decrease in cerebral blood flow (delayed hypoperfusion), which is caused by cerebral vasospasm due to dysfunction of endothelin and nitric oxide metabolism, erythrocyte rigidity, platelet aggregation, pericapillary cellular edema, and abnormal calcium ion flow. If the blood flow in the brain is not established for a longer period (18-24 hours), there will be the development of functional disorders with progressive ischemic damage to brain cells and the occurrence of brain death. The accumulation of calcium ions causes intoxication of brain cells due to vasospasm, double oxidative phosphorylation and destruction of cell membranes, and the production and accumulation of toxic metabolites such as: prostaglandins, leukotrienes and free radicals (1, 2, 9, 20).

Treatment recommendations in cerebral resuscitation

The first goal of all therapeutic measures should be to establish an optimal environment for brain recovery. The basis of treatment for all patients is to maintain proper gas exchange and perfusion of the brain, to avoid secondary brain damage. Decisive early treatment of hypoxia, hypercapnia, hypotension, and elevated intracranial pressure helps to avoid secondary complications. Other complications, the development of which should be checked and prevented, are hyperthermia, hyponatremia, hyperglycemia, and fluid imbalance. International CPR guidelines recommend the maintenance of normotension, which means a mean blood pressure (MAP) value

according to current recommendations > 80 mmHg in elderly adults or > 60 mmHg in younger and previously healthy patients. Blood pressure maintenance support includes i.v. administration of 0.9% saline NaCl, vasoactive substances (inotropic or vasopressor drugs), in rare cases using intraaortic balloon pumps. It is necessary to monitor blood saturation with oxygen through gas analysis or pulse oximetry, in order to titrate FiO_2 to achieve arterial saturation O_2 from 94-98%. Pulse oximetry and determination of arterial blood gases, i.e., ABS (if possible, CO_2 at the end of exhalation), can be used to assess oxygenation and ventilation. The goal is to achieve a normal level of $PaCO_2$ (5–5.6 kPa, i.e., 38-42 mmHg). Maintenance of normoglycemia is another important recommendation of current guidelines so that blood glucose values are between 7.7–9.9 mmol/L, normalization of hematocrit ≥ 30 is an important step, it is necessary to correct electrolytes, especially potassium and magnesium, it is especially important to correct acid-base status and maintenance of arterial pH between 7.3-7.5. In case of cerebral edema or signs of increased intracranial pressure, osmotic therapy is required, and osmotic diuretics (mannitol and glycerol) can be used to reduce intracranial pressure and maintain serum osmolality with mannitol or glycerolol. Normoosmolarity or mild hyperosmolarity should be maintained in patients (target serum osmolality is 295–320 mOsm/kg). If necessary, the patient should be immobilized due to neuromuscular paralysis. Sometimes sedation of patients using diazepam or morphine is required. In case of convulsions, the use of anticonvulsant drugs (diazepam, phenytoin, and barbiturates) is necessary. Mild hypothermia (32-34 C°) for 12-24 hours after cardiac arrest has a neuroprotective effect and improves the outcome after global cerebral hypoxia and hypoperfusion (2, 6, 9, 11, 19).

Ethics in resuscitation and end-of-life decisions

In all medical practice, there is no more exciting and dramatic situation than when a bystander, family member or healthcare worker finds himself next to a patient who has had a sudden disturbance of his vital functions. In times past, little could be done to prolong human life. The power of today's medicine to delay death has created difficult moral and ethical questions. Deciding when to start, when to stop or give up on starting cardiopulmonary resuscitation is an important problem for medical professionals, patients, family members and lawyers.

Ethics is a science that studies morality, its expressions, development, theories, principles, and norms. Today it is becoming more and more obvious that moral action is not only necessary in some places but is the foundation of human life. In recent decades, huge progress has been made in the field of medical sciences, especially in terms of better knowledge of the etiology and pathogenesis of the disease, faster and more accurate diagnosis and application of modern treatments thanks to the development of medicine treatment. The decision on when to stop resuscitation procedures is complex and often, in addition to medical ones, also reads through moral, ethical, legal, cultural, and religious components. Guided by medical attitudes, CPR measures should be discontinued when the following criteria are met when the safety of the rescuer is compromised, when fatal injuries or certain signs of death are present, when there is an objective suggestion from a higher instance–team leader, when each further CPR is futile and useless, when the asystole is longer than 20 min despite the ALS measures performed in the absence of reversible causes.

Patients in the final stage of an incurable disease should have care that ensures their right to dignified care and death. It is necessary to provide treatment to reduce their suffering associated with pain, dyspnea, delirium, convulsions, and other terminal complications. For such patients, it is ethically acceptable that CPR measures should not be applied when it is clinically clear that it is a terminal stage of non-communicable disease. While the sad reality is that most of those who experience cardiac arrest do not survive, recent research provides evidence of continued improvement in outcomes, especially where survival procedures are well applied. Specific cases of refractory cardiac arrest, which would have been fatal in the past, may benefit from additional interventional approaches. Further improvement in survival can be expected by applying clear instructions for initiating, not initiating, stopping, or denying resuscitation attempts, and identifying refractory cases that may respond to advanced interventions. In line with the above, a new chapter on ethics in resuscitation has been added to the new ERC guidelines for cardiopulmonary resuscitation. The chapter contains a detailed discussion of the ethical principles associated with cardiopulmonary resuscitation in the context of patient-centered health care: respect for his or her wishes; benefit and harmlessness of resuscitation procedures; fairness and equal access to CPR; pre-defined provisions; non-initiation or termination of CPR; transport of patients to a hospital with ongoing CPR; organ donation decisions; cardiac arrest in children; family presence during CPR; provider security; education of health workers in connection with making decisions on not starting resuscitation; practicing procedures on the recently deceased; research and consent of informed patients; audit of hospital resuscitations and analysis of national and international registries. In Europe, there is still a need for harmonization in legislation, powers, terminology, and practice, so the mission of the ERC and its guidelines is precisely to contribute to that harmonization (10, 22-25).

- ***Author's contribution: Both authors were involved in all steps of preparation this article. Final proofreading was made by the first author.***
- ***Conflict of interest: None declared.***
- ***Financial support and sponsorship: Nil***

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