

# Cardiac arrest under special circumstances. Part II: poisoning, ..., anaphylactic reaction, ..., traumatic injuries

Radosław Ziemba

Military Centre for Pharmacy and Medical Technology in Celestynów, Poland

**Author's address:**

Radosław Ziemba, Military Centre of Pharmacy and Medical Technique, ul. Wojska Polskiego 57,  
05–430 Celestynów, Poland; e-mail: zx11@op.pl

Received: 2012.07.20 • Accepted: 2012.11.22 • Published: 2012.12.08

## Summary:

In this publication, we discuss several issues related to cardiac arrest occurring under special circumstances such as: hypothermia, near drowning, poisoning, pregnancy, electric shock, anaphylactic reaction, episode of acute, severe asthma, traumatic injuries.

**Key words:** Safar's ABC scheme, resuscitation, rescue, dealing with an unconscious person.

## Introduction

This publication is a continuance of an article titled *Cardiac arrest under special circumstances. Part I: hypothermia, near drowning* published in the previous number of Military Pharmaceutics and Medicine (Issue V, No. 3). In this section, we discussed the rules of management of cardiac arrest due to: poisoning, pregnancy, electric shock, anaphylactic reaction, acute episode of severe asthma and traumatic injuries.

### Drug overdose and poisoning

Poisoning rarely leads to cardiac arrest, but is one of the main causes of death in people under 40 years of age. It is also the most frequent cause of non-traumatic coma in this age group. Suicidal poisoning with medicaments or addictive substances constitutes the main reason for hospitalizations. Accidental poisoning is most common among children. Criminal poisoning is rare. At times, it is not possible to immediately state

whether loss of consciousness or cardiac arrest is a result of poisoning. Therefore, this cause of come is important to exclude.

Large-scale exposition to chemicals or radiation may occur as a consequence of industrial accidents or warfare. In such cases, it is important that rescue services are not exposed to dangers of contamination. Proper personal protective equipment should be used. Decontamination and transporting victims into safer areas is usually the task of special services.

### Resuscitation

The basics of suicidal poisoning management ("overdose") involve supportive treatment based on the ABC scheme directed at preventing circulatory and respiratory arrest in hope that, in time, the substance will be eliminated from the system. Airway occlusion and respiratory arrest secondary to disrupted consciousness is a frequent cause

of death. Suicidal poisonings are often associated with excessive alcohol consumption.

After clearing the airway and restoring airway patency, the presence of respiration and pulse is checked. Mouth-to-mouth ventilation should not be performed when dealing with poisonings due to agents such as cyanides, hydrogen sulfide, corrosives or organophosphorous substances. Patient's lungs are ventilated through a pocket mask or a facemask set using the highest possible oxygen concentrations. Precautions should be taken in paraquat poisoning, as high oxygen concentrations can exacerbate lung damage.

Aspiration of stomach content to the lungs occurs in a considerable proportion of poisonings. Therefore, unconscious patients without pharyngeal reflexes should be intubated early. So-called fast induction with cricoid cartilage compression is performed in order to reduce the risk of aspiration. Preferentially, it should be performed by a trained anesthesiologist or a person with appropriate experience.

BLS and ALS should be commenced during cardiac arrest. Pulseless electrical activity (PEA) usually results from use of medicines exerting negative inotropic effect, but is associated with better prognosis than PEA from primary cardiac causes. Cardioversion is indicated in life-threatening tachyarrhythmias with the exception of Torsades de Pointes (look below).

Drug hypotension is a frequent phenomenon in suicidal poisoning. It usually responds to filling of vascular bed with fluids, although it sometimes requires use of inotropic drugs.

During resuscitation, we must undertake actions to identify the poison (or poisons). Patient's relatives, friends and the ambulance team can usually provide important information. Physical examination can reveal diagnostic clues (smell, puncture marks, tablets left in the oral cavity).

## Specific therapeutic actions

In poisoning management, we can formulate few specific therapeutic guidelines useful in emergency situations. Particular emphasis should be put on maintenance of vital functions,

oxygenation, compensating acid-base and electrolyte imbalances.

Gastric lavage with addition of activated carbon is justified up to 1 hour after poison ingestion. It is usually performed following intubation. Late gastric lavage exerts little influence on poison absorption and can even induce its movement further along the gastrointestinal tract. Elimination of poison from the system can be accelerated through hemodialysis or hemoperfusion.

Effective specific antidotes include:

- 1) N-acetylcysteine in paracetamol poisoning;
- 2) high doses of atropine in organophosphorous insecticide poisoning;
- 3) sodium nitrate, sodium thiosulfate or EDTA in cyanide poisoning, digoxin-specific antibodies in digoxin poisoning;
- 4) flumazenil in benzodiazepine poisoning, naloxone in opioid overdose.

## Tricyclic antidepressants

Suicidal tricyclic antidepressant overdoses are frequent and may lead to convulsions and arrhythmias. Threat to patient's life exists within the first 6 hours following ingestion. Widening of QRS complexes (over 0.16 seconds) indicates elevated risk of arrhythmias. Sodium bicarbonate can provide some cardiac protection and prevent arrhythmias in high-risk patients.

## Opiates

Opiate overdose causes respiratory depression, pinpoint constriction of pupils and coma. Pethidine overdose may result in convulsions. Naloxone is a specific opiate antagonist. The recommended dose is 0.4-0.8 mg i.v. (drug is administered slowly in the amount that is needed to obtain an effect) or 0.8 to 1.2 mg in an intramuscular or subcutaneous injection (which is easier in drug abusers with difficult venous access). Time of action of naloxone is shorter (45-70 minutes) than opiates (up to several hours), necessitating administration of additional doses at times.

## Cocaine

The following may occur in cocaine poisoning due to excessive sympathetic stimulation:

tachycardia, hypertensive crisis and cardiac ischemia. Small doses of benzodiazepines (midazolam, diazepam, lorazepam) constitute first-line treatment. Nitrates are used as second-line treatment – they counteract cardiac ischemia. Tachycardia and sudden blood pressure elevation caused by toxic effect of cocaine can be alleviated by labetalol (alfa and beta receptor blocker).

### Drug-induced bradycardias

They may respond to intravenous atropine at doses that do not exceed 3 mg (although higher doses are needed in organophosphate poisoning) or temporary external electrostimulation. Glucagon can be used in bradycardia induced by beta-blockers, improving cardiac contractility and increasing the heart rate.

### Torsades de Pointes

This phenomenon is associated with toxicity of various substances administered for therapeutic or suicidal purposes. The most important principles of management of such cases include intravenous administration of magnesium, correction of electrolyte imbalance and overdrive pacing.

### Further management and prognosis

Persisting loss of consciousness without changing body position can lead to sore formation and rhabdomyolysis. Electrolyte (particularly potassium) and glucose concentrations, as well as arterial blood gases should be closely monitored. Body temperature should also be overseen, as disturbances of thermoregulation occur frequently. Overdose of some substances may lead to either hypo – or hyperthermia. It is important to preserve blood and urine samples for further biochemical tests. We should be constantly prepared for prolonged resuscitation, particularly in young people, as the poison can be metabolized or excreted during that time.

### Pregnancy

Resuscitation of a pregnant woman involves two people. However, the emphasis is put on effective actions aimed at saving the life of a mother. At the same time, it is the best mode of action to maintain the wellbeing of a fetus. Sudden cardiac arrest in a mother is most often associated with

changes occurring in woman's organism in the third trimester of pregnancy. Causes of cardiac arrest in a mother include bleeding, pulmonary embolism, amniotic fluid embolism, premature placental detachment, eclampsia and drug toxicity. Cooperation with an obstetrician and a neonatologist should be established early.

### Course of resuscitation

All rules of BLS and ALS apply to pregnant patients. Delayed stomach emptying occurs in the first trimester of pregnancy, which increases the risk of aspiration of gastric contents. Therefore, early intubation is recommended, preferably with an assistant applying pressure on cricoid cartilage. Intubation may be sometimes difficult due to anatomical changes taking place during pregnancy (short and wide neck, large mammary glands, edema of epiglottis). Diaphragm is elevated and its mobility is reduced by the enlarged uterus during the last trimester of pregnancy and higher ventilation pressures are required for effective ventilation.

In order to improve venous return and cardiac output, it is necessary to reduce the pressure exerted by the uterus on inferior vena cava and aorta (aortocaval compression) through:

- 1) placing a sand-filled sac, a pillow or a prefabricated wedge (Cardiff type) under the right buttock and lumbar area;
- 2) manually moving the uterus leftward;
- 3) tilting the patient to the left on an operating table or a long board.

Chest compressions are performed in a standard manner, although they are more difficult to execute due to mammary gland enlargement and diaphragmatic stiffening.

Circulating blood volume in a pregnant woman is large, but cardiac arrest may occur as a result of hypovolemia due to an occult internal hemorrhage. Blood is drawn for cross matching and intravenous fluid administration is commenced. Early surgical treatment aimed at stopping the hemorrhage is of most importance.

### Arrhythmias

Cardiac arrhythmias are treated according to standard management schemes.

## Further management

Immediate cesarean section is indicated following five minutes of ineffective resuscitation, improving the likelihood of survival of the mother as well as the fetus. It is a difficult decision, but it must be made without unnecessary delay. Extraction of a fetus removes the aortocaval compression. ALS should be continued during and after the operation.

## Electric shock

Electric shock can occur at home, in a factory or as a result of a lightning strike. Most traumatic injuries caused by electricity in adults take place at work. On the other hand, children exposed to the greatest risk at home. In any given moment, there are 2000 thunderstorms around the globe and 1000 people around the world die because of it every year.

Severity of injury caused by electricity depends on the type of current (alternating or direct), its voltage, amount of energy it produces, resistance to current flow, path of the current through the patient as well as the surface area and time of contact. Skin resistance decreases due to moisture, increasing the likelihood of injury.

Contact with alternating current can lead to tetanic skeletal muscle spasm. It prevents detachment from the source of current and may lead to respiratory arrest. Alternating current is able to induce ventricular fibrillation if it acts on a cardiac muscle during a vulnerable period, analogously to a phenomenon called R-on-T. Sometimes, electrical current causes cardiac ischemia due to coronary artery constriction. Flow of current across the chest (from one upper limb to another) is more often fatal than a vertical flow path (from an arm to a foot) or astride (from one foot to another). Diffuse tissue damage may occur on the path the flowing current.

A lightning strike causes acute and massive discharge of DC current, leading to depolarization of the entire cardiac muscle. It poses a threat of asystole or ventricular fibrillation. Due to heart's automatism, hemodynamically effective sinus rhythm returns sometimes. Respiratory muscle paresis may be the reason for respiratory arrest and secondary cardiac arrest occurs

if appropriate actions are not taken. Lightning may also cause diffuse neurological damage, including encephalopathy and peripheral nerve damage.

## Diagnosis

Circumstances of an accident are not always clear to a rescuer and he should pay special attention to the presence of contact burns at the point of current entrance and exit.

## Rescue actions

The rescue team must make sure that all sources of electrical current are turned off and cannot approach the victim until it is completely safe. One should remember that high-voltage current (higher than that in home power outlets) might flow through the ground within a diameter few meters from the victim. On the other hand, it is safe to approach victims of a lightning strike, although it is reasonable to move them to a safer place.

## Course of resuscitation

BLS and ALS should be commenced immediately. Restoration of airway patency is sometimes difficult if there are electrical burns around the face and neck. In such cases, early intubation should be performed, as diffuse soft tissue edema develops quickly, leading to airway obstruction. Electrocutation can result in head and vertebral injury. Therefore, the vertebra should be immobilized until full clinical assessment. Muscle paresis, especially following high-voltage current discharge (industrial conditions), can persist up to 30 minutes and ventilatory support may be necessary during that time.

The most common initial arrhythmia following high-voltage alternating current discharge is ventricular fibrillation, which needs to be treated with a defibrillation attempt. Direct current discharge more frequently leads to asystole. Standard management should be undertaken in arrhythmias. Smoldering clothing and footwear should be removed in order to avoid further thermal damage. In case of diffuse tissue injury, it is sometimes necessary to commence intense intravenous fluid resuscitation. It is important to maintain proper urine excretion, which enables

systemic excretion of myoglobin, potassium and other products released by damaged tissues.

Patients with serious thermal injuries often require surgical intervention.

### Further management and prognosis

Immediate commencement of resuscitation in young patients with cardiac arrest caused by electric shock often brings positive outcome. There are reports of effective resuscitation even after prolonged ALS. All patients after serious electric shock and patients with circulatory or respiratory problems, loss of consciousness, cardiac arrest, electrocardiographic abnormalities, soft tissue injuries and burns require hospital monitoring.

### Anaphylactic shock

It seems that phenomena related to anaphylaxis are increasingly more common. It is certainly associated with growing frequency of allergies over the course of two or three past decades.

### Diagnosis of anaphylactic reactions

There is no generally accepted definition of anaphylactic reaction. The term “anaphylaxis” usually refers to immunoglobulin E (IgE)-mediated hypersensitivity reactions occurring in typical situations. Anaphylactoid reactions are similar, but are not associated with hypersensitivity. For simplicity, we will use the term anaphylaxis for both types of reactions unless they are clearly distinguished. Their symptoms and management are similar, so this distinction is only important when considering further treatment. Both of those reactions may be associated with various degrees of angioedema, urticaria, dyspnea and hypotension. Some patients die due to acute, irreversible bronchospasm or laryngeal edema. Among other symptoms are the following: rhinitis, conjunctivitis, abdominal pain, vomiting, diarrhea, sense of unrest. There is usually skin discoloration: patient’s face becomes red or pale.

Cardiovascular depression is a common symptom, particularly when it comes to reactions to intravenous agents or insect stings. It is caused by vascular dilatation and movement of plasma into the extravascular space. Circulatory failure or arrhythmias are associated mainly with a drop in

blood pressure and are rarely caused by primary heart disease or intravenous administration of adrenaline. Anaphylactic reactions present with various degrees of severity and can develop quickly, slowly or, rarely, in a biphasic manner. Rarely, symptoms may be delayed (it happens in case of latex allergies) or persist over 24 hours. Such reactions may be associated with exposition to various agents. The most frequent causes include insect bites, reactions to drugs, contrast agents or some foods. Peanut and hazelnut allergies are particularly dangerous.

Muscle relaxants can induce anaphylaxis and anesthetic agents constitute an important cause of anaphylactoid reactions. Absence of established symptoms and wide scope of clinical picture can pose diagnostic difficulties. In any case, it is necessary to acquire full medical history (with particular focus on past allergic reactions) and perform physical examination. Special attention should be paid to the condition of the skin, heart rate, blood pressure, upper airways and auscultation.

If possible, peak expiratory flow should be measured and documented. Distinguishing between anaphylaxis, panic attack or vasovagal episode can be sometimes difficult. All of these phenomena can occur, e.g. after vaccination. Full clinical assessment facilitates making this distinction.

### Comments on management

There is a common agreement that adrenaline is the most important drug used in management of anaphylactic reactions. As an alpha receptor antagonist, it abolishes peripheral vessel dilatation and reduces the edema. Its activity toward beta-receptors causes airway dilatation, increases contractility of cardiac muscle and inhibits histamine and leukotriene release.

Adrenaline is most effective if administered immediately after the occurrence of a reaction, but is not devoid of risk, particularly when given intravenously. Intramuscular adrenaline is a very safe drug. Undesirable effects are incredibly rare and the only case of myocardial infarction following its intramuscular administration involved a patient with high risk of coronary artery disease. At times, there is doubt whether the complication (e.g. myocardial ischemia) is a

result of the allergen itself or adrenaline administered for therapeutic purposes.

In rare cases adrenaline may fail to abolish clinical symptoms of anaphylaxis, particularly in late reactions or in patients treated with beta-blockers. In such instance, other means of management grow in significance, especially replenishing of circulating blood volume. Antihistamines (H<sub>1</sub> receptor blockers) should be routinely used in all cases of anaphylactic reaction, which facilitates attenuation of vasodilatation occurring as an effect of histamine action. These drugs may not be effective in some anaphylactoid reactions that are partially induced by other mediators, but their use is safe. It should be emphasized that use of antihistamines only may not be sufficient for saving patient's life. Administration of H<sub>2</sub> receptor blockers should also be considered.

Corticosteroids are thought to work too slowly, as the effect of their administration may appear as long as 4-6 hours following intravenous administration. However, they may help in immediate control of an acute episode and play a significant role in prevention or shortening the time of prolonged reactions.

## Resuscitation

All victims should be placed in a comfortable, supine position. Suspected allergen is removed (e.g. drug infusion or blood transfusion). Supine position with or without leg elevation may facilitate correction of hypotension, but makes breathing difficult. If conditions allow, high-flow oxygen should be administered (10-15 l/min).

BLS or ALS is commenced in case of cardiac arrest. During resuscitation, it may be necessary to administer higher doses of adrenaline. Infusion of large amounts of fluids is sometimes necessary.

In all patients with clinical signs of shock, airway edema or apparent breathing disturbances, adrenaline should be administered intramuscularly, which accelerates its absorption. Signs such as inspiratory wheezing, rhonchi, cyanosis, severe tachycardia and poor capillary return should evoke a suspicion of severe reaction. Adults should receive 0.5 ml of adrenaline in a 1:1000 (500 micrograms) solution. This dose should be repeated after about 5 minutes if clinical signs

persist or become more severe (particularly when disturbances of consciousness are present as a result of hypotension). In many cases there may be a necessity of administering several doses, especially when improvement is short-lasting.

Intravenous administration of adrenaline in a 1:10 000 (under no circumstances should it be 1:1000) solution is associated with complications and it should be reserved for patients in deep shock posing an immediate threat to life, and to special situations, e.g. during anaesthesia. Even greater dilution of adrenaline in a ratio of 1: 100 000 allows for more precise dosing and increases the safety, as the risk of unwanted effects is reduced. This drug is given under constant heart rate and ECG supervision, which constitutes the minimum monitoring. Doctors experienced at intravenous adrenaline administration may choose this route of administration in patients with signs of severe anaphylaxis.

Airway occlusion may occur as a result of soft tissue edema. Early intubation should be performed in such cases. Any delay can make it extremely difficult. Antihistamine drug acting on H<sub>1</sub> receptors [e.g. dimetindene (Fenistil®)] should be administered in slow intravenous injection, but H<sub>2</sub> blockers can also be used (e.g. ranitidine).

Hydrocortisone (sodium succinate preparation) should be used following a severe episode and prevents the late sequelae from occurring. It is particularly important for patients suffering from asthma (who are at greater risk of severe or even fatal anaphylaxis) if they were treated with corticosteroids before. Hydrocortisone is administered in a slow intravenous injection. Fluid infusion should be commenced if there is significant blood pressure reduction and patient does not respond quickly to administered medicines.

## Anaphylactic reactions in adults – treatment by first-aid providers

Patients who suffered an episode of anaphylaxis, even of moderate degree, should be warned against the possibility of early return of symptoms and, in some circumstances, they need to be hospitalized for observation for next 8-24 hours. It refers especially to cases of:

- 1) severe reactions with slow onset – evidence of idiopathic anaphylaxis,

- 2) reactions in patients with severe asthma or strong asthmatic component,
- 3) reactions associated with the possibility of continuous exposure to the allergen,
- 4) patients who underwent biphasic reactions in the past.

If there is bronchospasm not responding to standard management, inhalation of beta-2 receptor antagonist, e.g. salbutamol, may be of some help.

### Additional tests and further management

Measuring mast cell tryptase levels can retrospectively facilitate the diagnosis of anaphylaxis. Ten milliliters of blood should be drawn to a tube containing clot activator between 45 minutes to 6 hours after the episode.

Following successful management of anaphylactic reaction, it is important to identify the allergen in order to avoid recurrence of the condition in the future. Therefore, patient should be referred to a specialist outpatient clinic. Patients at great risk of anaphylactic reaction can keep at all times a special, adrenaline-filled syringe for self-administration and wear a warning bracelet.

### Acute episode of severe asthma

Acute episode of severe asthma is almost always a reversible state and we must assume that such patients can be saved. The majority of deaths occur outside of the hospital. Several factors contribute to it, such as:

- 1) patient and his relatives do not recognize the severity of asthmatic episode and turn to medical help too late;
- 2) emergency services and family doctors do not always act fast enough;
- 3) patients with mild asthmatic episodes are discharged home after being provided with medical assistance and suffer from sudden deterioration of their condition.

It is important to treat all asthmatic exacerbations aggressively in order to prevent future life-threatening recurrences and cardiac arrest. National guidelines were published on management of acute asthma exacerbations based on early oxygen administration, use of beta-2 receptor blockers (salbutamol), corticosteroids and aminophylline.

Cardiac arrest in patients with severe asthma may occur as a result of:

- 1) hypoxia due to severe bronchospasm and airway obstruction by secretions;
- 2) arrhythmias due to hypoxia or beta receptor agonist and aminophylline toxicity;
- 3) tension pneumothorax.

Life-threatening asthmatic episode is recognized based on the absence of breath sounds, cyanosis and poor respiratory drive. It may be accompanied by bradycardia and hypotension. Patient appears exhausted, confused or falls into a coma. Arterial blood gas measurement reveals hypoxia, acidosis accompanied by normal or elevated carbon dioxide partial pressures.

### Acute management

Patient status will quickly deteriorate, leading to respiratory arrest and secondary cardiac arrest if proper management is not immediately commenced. Such patient should be quickly moved to a facility where proper care and monitoring is available. High oxygen concentrations are administered. First-line therapy in acute asthma is inhalation of beta-2 agonists. It usually begins with administration of salbutamol (5 mg in 5 ml of saline) in nebulization mixed with oxygen, or in 4-6 puffs using an inhaler with a spacer. This dose can be repeated at 15-minute intervals or, if necessary, it can be administered continuously. Corticosteroid therapy should be commenced at an early stage (during first 30 minutes). Prednisolone at a dose of 30-60 mg orally, 200 mg of hydrocortisone i.v. or both drugs should be given if patient's condition is very severe.

If this treatment gives no effects, subcutaneous administration of adrenaline (0.3 mg) can prevent the necessity of artificial ventilation. The same dose of adrenaline (0.3 mg) can be repeated twice in 20-minute intervals.

If drugs administered so far are not effective, other actions are taken such as: inhalations with anticholinergic drugs (ipratropium 0.5 mg in nebulization), intravenous infusion of aminophylline (5 mg/kg infusion in 30-45 minutes), intravenous magnesium sulfate (2-3g) or administration of a breathing mixture containing helium and oxygen in a 70:30 ratio. Performing chest x-ray examination early is instrumental.

Such patients often suffer from substantial dehydration and intravenous infusion of fluids is beneficial.

Mechanical ventilation is only considered when all conservative methods fail. Decision regarding commencement of mechanical ventilation should be based on the degree of patient exhaustion, not on blood gas analysis. Noninvasive ventilation may prevent the necessity of tracheal intubation and invasive mechanical ventilation.

Achieving normal blood gas values during mechanical ventilation can be difficult due to high airway resistance. It is sometimes necessary to use special techniques of assisted ventilation with patient sedation (e.g. using anesthetic gases or ketamine).

## Resuscitation

BLS and ALS rules apply during cardiac arrest. However, there are some additional requirements, which should be remembered: patient ventilation is sometimes difficult due to high airway resistance. Under such circumstances, ventilation with a facemask is associated with high risk of stomach distension. Therefore, it is important to perform tracheal intubation early. High airway pressures necessary to maintain proper minute ventilation increase the risk of tension pneumothorax. Prolonging inspiration and expiration time is often necessary in order to avoid increase in intrinsic end-expiratory pressure (i-PEEP or auto PEEP). Indirect cardiac massage is difficult or impossible in a patient with hyperinflated chest. Prolonging the expiration time may partially overcome this difficulty. If experienced personnel is present, opening of thoracic cavity for direct cardiac massage should be considered. Arrhythmias are treated according to standard therapeutic schemes.

## Traumatic injuries

Cardiac arrest secondary to blunt trauma is associated with very poor prognosis. In cases of cardiac arrest after penetrating trauma, patient can be sometimes saved if conditions for undertaking therapy by personnel experienced in direct cardiac massage (with opening of thoracic cavity) are met.

Causes of cardiac arrest following trauma include:

- 1) severe brain injury,
- 2) hypovolemia due to massive blood loss,
- 3) hypoxia secondary to respiratory arrest,
- 4) direct injury to vital organs (heart or great vessels),
- 5) comorbidities (e.g. cardiac arrest in a driver, which preceded the traffic accident),
- 6) tension pneumothorax,
- 7) cardiac tamponade.

## Resuscitation

Early assessment and commencement of appropriate actions can prevent cardiac arrest. It is important to identify and undertake appropriate management of life-threatening injuries as soon as they are diagnosed. Fast transport to a hospital is crucial, as immediate surgery is often necessary. Rules of BLS and ALS in trauma patients are the same as in cardiac arrest due to other causes. However, one should remember that: cervical spine should be protected during restoration of airway patency.

It is important to exclude the presence of tension pneumothorax. This complication may be indicated by worsening lung compliance or hyperresonant percussion sound. In such instance, pleural cavity should be immediately punctured with a needle (in the second intercostal space, in the midclavicular line).

Pulseless electrical activity (PEA) due to hypoxia, hypovolemia or both, constitutes the most common mechanism of cardiac arrest in trauma patients. Therefore, administration of 100% oxygen, replacement of circulating blood volume or attempt at stopping the hemorrhage (direct compression, surgery) are necessary.

Thoracic cavity can be opened in a small proportion of patients with penetrating chest trauma and PEA in order to commence direct cardiac massage and simultaneous management of cardiac tamponade and control of hemorrhage. Immediate and proper management of the discussed conditions can prevent occurrence of cardiac arrest. Resuscitation method may have to be modified if cardiac arrest takes place in special situations described above.



## References:

1. R.G. Wilcox: How to cope with cardiac arrest.  
Źródło: <http://www.libramed.com.pl/wpg/NumeryArchiwalne/05/04.html>
2. Marzena Wojewódzka-Żeleznikowicz, Sławomir Lech Czaban, Piotr Szczesiul, Anna Nielepiec-Jałosieńska, Jerzy Robert Ładny: Hipotermia poresuscytacyjna – wskazania, sposób prowadzenia, skuteczność kliniczna, powikłania stosowania, *Postępy Nauk Medycznych*, 12/2009
3. White L. Audit of cardiac arrest procedure and outcome. A thesis submitted in partial fulfilment of the requirements for the degree of Bachelor Medical Sciences, University of Nottingham Medical School 1992.
4. European Resuscitation Council Working Party, Adult advanced cardiac life support: the European Resuscitation Council Guidelines 1992 (abridged). *BMJ*1993; 306: 1589
5. Niemann JT. Cardiopulmonary resuscitation. *N Engl J Med.* 1992; 327: 1075-1080.
6. Praca zbiorowa *Anestezjologia i intensywne opieka*, red. Laura Wołowicka, Danuta Dyk, Wydawnictwo Lekarskie PZWL, 2008.

