# MEDICAL STUDIES IN **ENGLISH**

# CLINICAL SKILLS: **YEAR 2**

# STUDENT HANDOUT

# 2015

Contents:




Department of Clinical Skills

University of Split School of Medicine

# 1. THE GENERAL PRINCIPLES OF HISTORY TAKING

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**A Brief Overview of Clinical Medicine**

From classical Greek times interrogation of the patient has been considered most important. However, the current emphasis on the use of history taking *and* physical examination for diagnosis developed only in the 19$^{th}$ century. Until the 19$^{th}$ century, diagnosis was empirical and based on the classical Greek beliefs that all disease had a single cause, an imbalance of the four humours or body fluids (yellow bile, black bile, blood and phlegm).

In the 17$^{th}$ century physicians based their opinion on a history provided by an apothecary (assistant) and rarely saw the patients themselves. Thomas Sydenham (1624-1689) began to practise more modern bedside medicine basing his treatment on experience and not theory.

A renaissance in clinical methods began with the concept of Battista Morgagni (1682-1771) that disease was not generalised but arose in organs, a conclusion published in 1761. Leopold Auenbrugger invented chest tapping (percussion) to detect disease in the same year. The technique was forgotten for nearly half a century until Jean Corvisart (1755 -1821) translated Auenbrugger's work in 1808.

The next big step occurred with Rene Laennec (1781-1826), a student of Corvisart. He invented the stethoscope in 1816 (at first merely a roll of stiff paper) as an aid to diagnosing heart and lung disease by listening (auscultation). This revolutionised chest examination, partly because it made the chest accessible in patients too modest to allow a direct application of the examiner's ear to the chest wall!

A significant change in the approach to clinical diagnosis began in the hospitals of Paris after the French Revolution. Influenced by the philosophy of the enlightenment, which suggested that a rational approach to all problems was possible, the Paris Clinical School combined physical examination with autopsy as the basis of clinical medicine. Spectacular advances in physiology, pathology, pharmacology and microbiology in the latter half of the 19$^{th}$ century led to the development of 'clinical and laboratory medicine'. Modern imaging techniques and genetic analyses are enabling an even more detailed analysis of the human body. The modern systematic approach to diagnosis is still, however, based on taking the history and examining the patient by looking (inspecting), feeling (palpating), tapping (percussing) and listening (ausculating).

**History Taking**

An extensive knowledge of medical facts is not useful unless a doctor is able to extract accurate and succinct information from a sick person about his or her illness. Except for patients who are extremely ill, the taking of a careful medical history should precede both examination and treatment. A medical history is the first step in making a diagnosis; it will often help direct the physical examination and usually will determine what investigations are appropriate. More often than not, an accurate history suggests the correct diagnosis, whereas the physical examination and subsequent investigations merely serve to confirm this impression. The history is also, of course, the least expensive way of making a diagnosis.

**Bedside Manner**

The potential power of healing starts from the moment you establish contact with your patient, whether it's when you arrive at their bedside or welcome them into your surgery. An unkind and thoughtless approach to questioning and examining a patient can cause harm before any treatment has had the opportunity to do so. One of the axioms of the medical profession is ***primum non nocere*** (Latin, the first thing is to cause no harm). You should aim to make the patient feel better, not worse. Good communication with your patient will make the history taking rewarding as well as increase the chances of the patient complying with your recommendations. You should aim to put the patient at ease, establish rapport and gain their trust. To achieve this:

- Use helpful non-verbal communication
- Ask open questions
- Show interest and reflect back
- Empathise to show that you understand
- Avoid words that sound judgemental
- Accept, acknowledge
- Use suitable language
- Avoid commands, make suggestions

**Obtaining the History**

It is useful to make rough notes whilst questioning the patient. At the end of the consultation a detailed record is made. This record must be a sequential, accurate account of the development and course of the illness. A sick patient will sometimes emphasise irrelevant facts and forget about very important symptoms. For this reason, a systematic approach to history taking and recording is crucial (Table 1).

**Table 1** History Taking Sequence

| |
|---|
| **Presenting Complaint**/Principal Symptom (PC) |
| **History of Presenting Complaint** (HPC)<br>- Details of current illness and treatment<br>- Details of previous similar episodes<br>- Extent of functional disability |
| **Past History** (PH)<br>- Past illnesses and operations<br>- Past medications and allergies<br>- Blood transfusions |
| **Social History** (SH)<br>- Occupation, education<br>- Smoking, alcohol, drugs<br>- Overseas travel<br>- Marital status, social support, living conditions<br>- Exercise, diet |
| **Family History** (FH) |
| **Systems Review** (SR) |

Notes need to be dated and signed. This also applies to any corrections made. Try and avoid writing detailed notes while the patient is talking as this interferes with rapport.

**Introductory Questions**

Introduce yourself to the patient and shake his or her hand. Check patient's full name, date of birth and how they wish to be addressed. Sit down either beside the patient or even on the bed (with the patient's permission) so as to be close to eye level. Next, find out the patient's main complaint by asking the patient ″What has been the trouble or problem recently? ″ Encourage patients to tell their own story in their own words, from the onset of the first symptom to the present time. Some direction may be necessary to keep a patient on track. Record each presenting symptom in the patient's own words.

***Symptoms*** are subjective complaints which the clinician learns from the patient's account of his of her feelings, whereas ***signs*** are objective morbid changes detectable by the clinician. So when recording the patient's presenting complaint we are recording symptoms but when recording physical examination findings we are documenting signs.

**History of Presenting Complaint**

A number of facts have to be uncovered about every symptom. These include the duration, the mode of onset, the site and radiation (especially of pain), the character, the severity, aggravating or relieving factors and associated symptoms.

*Duration*: Ask patients when they last felt entirely well. In a patient with long-standing complaints, ask why he or she decided to come visit you today.

*Mode of Onset and Pattern*: Ask whether the symptoms came on suddenly or gradually. Ask whether the symptom has been present continuously or intermittently. Determine if the symptom is getting worse or better. Find out what the patient was doing at the time the symptom began.

*Site and Radiation*: Ask where the symptom is (it is best to ask the patient to point to the actual site on the body) and whether it is localised or diffuse (a patient that points to the site usually has localised pain whereas the patient who uses his/her palm to indicate where the problem is usually has diffuse pain). Ask whether the symptom radiates to a distant site (this mainly applies if the symptom is pain).

*Character*: Ask the patient to describe the nature of the symptom. This will aid in diagnosis and help avoid misunderstandings, e.g., ″indigestion″ may mean abdominal pain, excess wind or a change in bowel habit.

*Severity*: Severity is subjective so use a scale, e.g., from 0 to 10, where 0 is no pain and 10 is the worst possible pain. Another good way of assessing severity is to ask the patient to what degree does the problem interfere with daily activities.

*Aggravating or Relieving Factors*: Ask if anything makes the symptoms worse or better.

*Associated symptoms*: Here some knowledge of diseases is required as you attempt to uncover symptoms which might be expected to be associated with a particular disease. Remember that while a single symptom may provide the clue which leads to the correct diagnosis, usually it is the combination of characteristic symptoms that most reliably suggests the diagnosis.

**Medications**

When booking patients for a consultation, make sure to tell them to bring all their medications. Note the dose, length of use and indication for each medication. Ask whether the medication was taken as prescribed. Also ask the patient whether they're taking any over-the-counter medications (OTCs). Always ask specifically if a woman is taking the contraceptive pill because it is not considered a medicine by many who take it.

**Sexual/menstrual History**

The sexual history is important if a patient is complaining of genitourinary symptoms. In all cases a menstrual history should be obtained; record the first day of the last menstrual period, the age of menarche, if the periods are regular, or whether menopause has occurred. Don't forget to ask women of reproductive age if there is any possibility of pregnancy; this may preclude the use of certain investigations or medications.

**Past History**

Ask the patient about:

- Illnesses
- Operations
- Childhood diseases
- Obstetric or gynaecological problems
- Allergies

**Social History**

When asking about social habits it is important not to display personal bias; you're there to help, not criticise. When enquiring about smoking ask the patient if they've ever smoked (not if they're a smoker because they may have stopped that very day), the number of cigarettes per day and for how many years. Ask if the patient drinks alcohol, and if so, what type, how much and how often.

**Family History**

Many diseases run in families so enquire about the health and, if relevant, the causes of death and ages of death of the parents and siblings.

**Systems Review**

As well as detailed questioning in the system likely to be affected, a quick review of possible important symptoms and disorders in other systems is essential; otherwise important diseases may be missed. When recording the systems review, list important negative answers. Before completing the history, it is often valuable to ask what the patient thinks is wrong with him or her, and what he/she is most concerned about.

**Conclusion**

To obtain a useful and accurate history one must: first, establish rapport; second, ask questions in a logical sequence; third, observe non-verbal cues carefully; and fourth, properly interpret the history. This should be followed by a systematic physical examination.

**Literature:** Clinical Examination: *A Systematic Guide to Physical Diagnosis,* 7th Edition, Talley & O'Connor

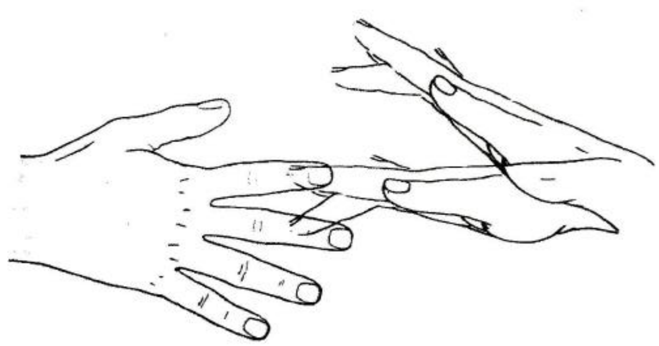
# 2. BASIC PRINCIPLES OF PHYSICAL EXAMINATION

Irena Zakarija-Grković, MD, FRACGP, IBCLC, PhD

## General Principles

The four elements which comprise the main parts of the physical examination are: looking- **inspection**; feeling- **palpation**; tapping- **percussion**; and listening- **auscultation.**

Figure 1 Percussion



*Percussion technique on the anterior or posterior chest wall (Jessica Elliott, Medical Student)*

A formal approach to physical examination is important because it ensures the examination is thorough and that important signs are not overlooked. This formal approach leads to examination of parts of the body in systems. The attention of the examining doctor is directed particularly towards those systems identified in the history as possibly being diseased, but of course proper physical examination requires that all the systems be examined. By convention, patients are always examined from the right side of the bed.

## Preparing the Patient for Examination

Ideally, patients should be examined in a well lit room that has a window in addition to artificial lighting. Privacy should be ensured for all patients by either providing covers, curtains or change rooms with gowns. Prior to the examination the doctor/student should: 1) introduce themselves; 2) confirm the patient's name and date of birth; 3) explain the purpose of the examination, and what the examination will involve; 4) obtain the patient's consent; and 5) offer a chaperone.

## General Overview of the Patient's Appearance

The danger of conducting a systematic physical examination is that time is not taken to stand back and look at the patient's general appearance, which may give many clues to the diagnosis. First, decide how sick the patient seems to be: that is, does he or she look generally ill or well. Next, if possible, ask the patient to position themselves on the examination couch at 45 degrees.  Assess the patient's colour (cyanosis, pallor, jaundice) and level of comfort.

Observe the patient's body habitus, without commenting aloud. Next observe around the bed or chair area for signs of illness (oxygen cylinders, medications, drains, urine bags...).

**Assessing the Patient's Hands**

Changes occur in the hands in many different diseases, therefore they warrant careful inspection. Pay particular attention to:

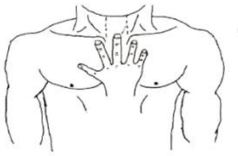
1. Nails: splinter haemorrhages, nicotine staining, pitting, leuconychia...
2. Hands: colour, warmth, capillary return, pallor, palmar erythema, contractures...
3. Clubbing
4. Tremor
5. Turgor
6. Radial pulse (rate and rhythm)
7. Respiratory rate

**Assessing the Patient's Face**

1. Eyes: Check the conjunctiva for pallor and sclera for jaundice; and look for xanthelasma around the eyes.
2. Mouth: Check the patient's lips and base of tongue for central cyanosis; check for any breath smells, apthous ulcers, angular stomatitis, glossitis, dentition, dentures.

**Assessing the Patient's Neck**

1. Assess the cervical and supraclavicular lymph nodes
2. Assess the jugular venous pressure (JVP)
3. Palpate the carotid pulse (one at a time using the index or middle finger over the sternocleidomastoid-lower half to avoid pressing on the carotid sinus)
4. Palpate the trachea placing your index and ring fingers on the two clavicular heads, palpate the trachea with the middle finger.


Locating the trachea (Sam Hey, Medical Student)

5. Palpate the thyroid.

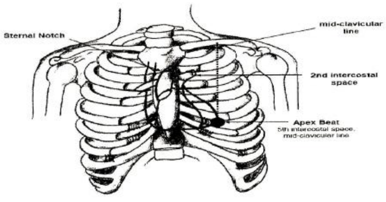
**Assess the Patient's Chest Wall**

Inspection:

1. Scars, rashes, spider naevi
2. Chest shape
3. Respiratory Pattern
4. Deformities
5. Pulsations
6. Gynaecomastia

Palpation:

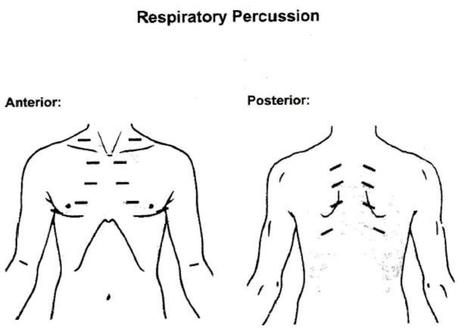
1. Feel for expansion of the patient's chest. Using both hands on the anterior chest wall, ask the patient to take deep breaths in and out. There should be equal expansion on both sides of the chest.
2. Locate the patient's apex beat (5th intercostals space, mid-clavicular line)




*Location of apex beat (Areej Paracha, Medical Student)*

Percussion:

1. Tap your middle finger against your middle finger
2. Your finger should be positioned flat between the patient's ribs
3. Action is from your wrist
4. Compare each side, to assess for discrepancy
5. Solid/liquid = dull sound
6. Air = resonant sound
7. Examination for the anterior chest can be above the clavicles, clavicles, three areas of the anterior chest wall on either side, and the sides.




Auscultation:

1. Ear pieces should be angled forward
2. Bell- best for high pitched sounds
3. Diaphragm best for low pitched sounds
4. Listen in the same areas as percussed (with the diaphragm), but using a bell for above the clavicles;
5. Sounds should be symmetrical
6. Any abnormal sounds should be described (character, location, inspiratory/expiratory)
7. Listen to heart sounds
8. 1st heart sound
   - Loudest at the apex
   - Mainly caused by mitral valve closure
   - Precedes systole
   - Generates a low pitched sound, longer in duration than S2
9. 2nd heart sound
   - Loudest at left sternal edge in the 2nd intercostals space
   - Caused by closure of the aortic and pulmonary valves

- Creates a higher pitched sound, shorter than S1
- Precedes diastole
- During inspiration the pulmonary valve closure is delayed causing S2 to split

**Examine the Patient's Abdomen**

Inspection:

1. Ask the patient to lie flat if possible.
2. Ask the patient for consent to expose their abdomen.
3. Observe the patient's face (for expressions of pain or discomfort) while examining their abdomen, and ask the patient to let you know if it feels too uncomfortable or painful at any point.
4. Inspect the abdomen for scars, distension, swellings, distended veins, visible peristalsis and abdominal wall movement.

Palpation:

1. All abdominal regions should be assessed systematically, starting furthest from where the patient has identified any pain.
2. Use the flat of palmar surfaces of your fingers to palpate the patient's abdominal wall.
3. Initially, palpate superficially, and then deeply.
4. The patient's liver, spleen, kidneys and aorta should all be palpated, using the edge of the index finger to detect organ edges.
5. Start in the right iliac fossa for liver and spleen.
6. The liver descends towards the right iliac fossa on inspiration.
7. The spleen descends infero-medially on inspiration towards the right iliac fossa.
8. Palpate the patient's kidney by balloting in the patient's loins
9. Normally, the patient's abdomen is felt as soft and elastic, sometimes with mild tenderness. Deep palpation is possible.

Percussion:

1. The patient's general abdomen should be percussed, and should be resonant due to gas.
2. The patient's liver is percussed from the right iliac fossa.
3. The patient's spleen is percussed diagonally from the right iliac fossa.
4. The patient's bladder is percussed working down from the umbilicus.

Ausculation:

1. Assess for bowel sounds using the diaphragm of the stethoscope.

Examine the groins for inguinal lymphadenopathy, hernial orifices and femoral pulse.
In men palpate the testes, if indicated.

**Assess the Patient's Legs**

Look for peripheral oedema (swelling) and leg ulcers. Feel all the peripheral pulses.

**Neurological Examination**

Neurological examination should be performed separately and is not within the scope of this handout.

**Literature**

Clinical Examination: *A Systematic Guide to Physical Diagnosis*, 7th Edition, Talley & O'Connor

## 3. OVERVIEW OF A PATIENT CONSULTATION/CLINICAL ENCOUNTER

Irena Zakarija-Grković, MD, FRACGP, IBCLC, PhD

**Introduction**

Time management is crucial in medicine, especially if you're a busy clinician who sees many patients, therefore is it important to establish a routine consultation that will flow easily and cover all the essentials. The following format is recommended, with an emphasis on 'patient-centred care'.

**Opening the consultation**

1. Introduce yourself
2. Check the patient's full name, date of birth, how they wish to be addressed
3. Explain confidentiality
4. Obtain consent for the consultation and examination (if you're a student)

**Listening to the patient's history**

1. Gather information relevant to the patient's presenting complaint
2. Seek to explore and understand what the patient thinks, feels and does in relation to the presenting complaint: health beliefs (including motivation); coping; social support; change in activities of daily living
3. Elicit the patient's ideas, concerns, expectations and feelings

**During the examination**

1. Obtain consent to begin the examination, and ask the patient if they'd like a chaperone
2. Check that the patient is comfortable and not in any pain
3. Explain each stage of the examination to the patient, in non-technical language, seeking consent throughout
4. Inspect the patient
5. Observe the patient's hands and face
6. Check vital functions
7. Perform a routine examination

**Closing the examination**

1. Summarise the discussion
2. Check if there's anything else
3. Provide information/advice/reassurance as appropriate
4. Signpost the closing
5. Thank the patient for their time and participation (if you're a student)

**Throughout the consultation**

1. Build rapport and maintain the relationship with the patient

2. Provide structure and manage the flow (using summaries, signposts)
3. Involve the patient in the consultation and explain the process to them
4. Attend to the patient's dignity and comfort

# 4. MONITORING VITAL SIGNS

Nenad Karanović, MD, PhD

**Introduction**

With the ocurrence of the first intensive care units some 40 years ago, the need has arisen for more intensive monitoring of patients' vital signs. Unfortunately at this stage of development of the ICU's, the most difficult patients were monitored only intermittently by nurses and other medical staff. Continuous measurement was unavailable or required a number of invasive procedures.
The rapid development of technology and the wide use of computers have significantly changed the potentiality of intensive care and monitoring within a few decades. There is no single part of the hospital where the patient is monitored better, more intense and with more continuity than in ICU's and operating theatres. Today all the vital signs can be tracked accurately and continuously, either by invasive or non-invasive procedures.
The role of most of the surveillance and monitoring of patients is to alert the staff, physicians and nursing staff about possible threatening disorders to the patient's condition.

**Monitoring**: represents the process by which medical staff, primarily physician, identifies and evaluates the (estimate) physiologic and pathophysiologic changes, noting trends during treatment. Efficient monitoring reduces any unfavorable treatment outcome, suggesting disturbances before they result in serious or irreversible damage. With their appearance the monitors increased specificity and accuracy of clinical assessment.
The first goal of monitoring is to point out the pathophysiological events (abnormalities) in patients at high risk of developing these events and disturbances. Another equally important role of monitoring is enabling timely and meaningful treatment. Final, but not the least important role in monitoring is the assessment of improvement of the patient condition.
The monitoring itself may be clinical supervision or with the help of various technical and technological resources, and laboratory.
**Clinical supervision or monitoring** is carried out with the help of our own senses (sight, hearing, touch, smell, etc.). The main characteristic is that it is always available, but unfortunately not sufficiently objective. It depends on many factors, such as the speed of perception, the ability of individual senses, fatigue etc.
**Monitoring with the help of technical and technological means** is more accurate, provides more data, is continuous and devoid of subjectivity. With certain computer software it is also capable of very complex diagnosis, not just surveillance. The possibility of malfunction is a risk. However, there are certain programs that warn of such a situation.
**Laboratory monitoring** with the aforementioned types of monitoring also enables the review of the complete state of the patient.
**Selection of monitoring** depends on the general status of the patient, the type of illness or

injury and the intended way of treatment or possible intervention.
**Basic monitoring**: ECG and pulse, non-invasive measured blood pressure, body temperature, oxygen saturation of peripheral blood and state of consciousness.

**Monitoring of the cardiovascular system**

**1. Electrocardiography**: Monitoring of cardiac electrical activity. Continuous electrocardiography provides observation of heart rate, rhythm disturbances detection and tracking the function of pacemakers. It can help with the spotting of cardiac ischaemia and some electrolyte imbalances.
**Indications:**
Cardiac patients - patients with possible development of arrhythmias, myocardial infarction or angina pectoris.
Patients in whom there is a possibility of bleeding to death or the need for replacement of fluids, blood and blood derivatives.
Diabetics - damage to blood vessels (coronary and other arteries) - arrhythmias and electrolyte disturbances.
All others that are likely to expect any kind of rhythm disturbances (bradycardia, tachycardia, other arrhythmias)
Placement: Leads II and V5 are most commonly used (3 electrodes). They are positioned on the shoulders and the front axillary line at the level of the tip of the heart xyphoid.
Complications: Malfunction due to possible technical errors: old, dry or insufficiently well attached electrodes. Interruptions of the cables that drains the signal from the electrodes are possible.
Normal values of heart rate: 60-100 beats/min. Lowering or raising the ST up to 1 mm.

2. Blood pressure measurement: Because the systemic blood pressure is dependent on cardiac function and peripheral circulation, arterial blood pressure monitoring provides "rough data" on the total cardio-circulatory function.
Blood pressure measurement is a standard procedure and an absolute requirement for all endangered patients.
However, the type and frequency of measurements depend on the individual condition and diagnosis of individual patients. Blood pressure depends on cardiac output (CO) and the systemic vascular resistance (SVR). Blood pressure can be measured either directly - with the instrumentation vascular areas (invasive) or indirectly (non-invasive) - techniques that include cuff for arterial occlusion.

**A. Non-invasive blood pressure measurement**
- Palpation - method is limited, insufficiently precise and enables only the measurement of systolic blood pressure.
- Auscultation (Riva-Rocci method) - more reliable than palpation. Imprecise. In comparison to the intra-arterial measurement, systolic blood pressure varies from 1-8 mm Hg, while the diastolic differs 8-10 mmHg. In comparison to intra-arterial measurement auscultatory method shows higher values at a pressure lower than 120 mm Hg, and lower values at the

systemic pressure higher than 120 mmHg.
- Oscillometry - uses two cuffs. This method is the only non-invasive one that allows determination of the mean arterial pressure. The disadvantage is the inability of accurate measurement in patients with arrhythmias and those with poor circulation and reduced volume.
- Plethysmography - based on the fact that arterial pulsations lead to changes in the volume of the limb. Such changes may be determined by finger plethysmograph. Insufficient accuracy in stress situations (vasoconstriction) and at reduced intravascular volume.
- Doppler - ultrasonics. It is measured by ultrasound probe placed onto the artery distal to the cuff. Arterial pressure obtained by this method are often higher than those obtained by palpation and lower than those obtained by invasive measurements. However, they give very good data at low pressures. Sensitivity to movement is a drawback, as well as the need for exact placement of the probe and the use of ultrasound jelly (gel).
- Tonometry - based on the detection of occlusive pressure required to stop the flow through surface artery compressed to the bone structures.
The good side is the continuous monitoring where the resulting wave pulsations are very similar to those obtained by invasive measurements.

**B. Invasive blood pressure measurement:** A catheter placed into an artery provides the most accurate blood pressure measurement nowadays. Such catheters are connected to pressure transducers, which convert pressure into electrical signals. Care must be taken that the air does not reach the system, which can lead to incorrect measurements since the air is more pressurable than water.
**Indication**: The need for continuous monitoring of pressure and its variations in different clinical settings, and during the use of vasoactive drugs.
**Clinical application:** Radial, ulnar, dorsalis pedis, posterior tibial, femoral and axillary arteries are used most frequently. Radial artery is preferred for ease of puncture and less possibility of severe complications (vascular insufficiency of the hand caused by thrombosis or arterial vasospasm). These complications are more common when using the cannulas larger than 22 G. Women have less potential for development of arterial thrombosis than men, out of unknown reasons. Infection is frequent complication.
Advantages - possibility of frequent blood sampling

**3. Measurement of central venous pressure** (CVP): CVP reflects changes in the right heart and may indicate about the state of pulmonary circulation and left heart (only in cardiac and pulmonary uncompromised persons) only secondarily.
Indications: Assessment of the volume load (in patients without heart disease). Assessment of the right heart condition.
Place of the catheter application: internal jugular, subclavian and both sides femoral veins. Femoral vein is avoided because of frequent thrombosis and greater risk for infection.
Complications: arterial puncture, pneumothorax, perforation of superior vena cava (mortality 67%), laceration of the right ventricle (100% mortality), cardiac tamponade, damage to the brachial plexus, ganglion stelatum and phrenic nerve, air embolism (rare), venous thrombosis, infection.

**4. Catheterization of the pulmonary artery:** It was initially used as a way to measure intracardial pressures and to assess the left heart function, supplemented with CVP measurement. Today it is considered a "golden standard". Provides information regarding the condition of the left heart and indirectly of certain other hemodynamic values.
Indications: From cardiac disease to various pulmonary and circulatory disorders.
Complications: the same as when placing a catheter to measure CVP along with the additional possibility to cause significant cardiac arrhythmias (VF and VES), creation of a node on the catheter, rupture of the pulmonary artery (41% mortality).
Subspecies of hemodynamic monitoring, not so invasive as the one obtained by pulmonary catheter are PiCCO (Pulse Contour Cardiac Output) and LIDCO(Lithium Dilution Cardiac Output) method. Cardiac output (CO) can be observed together with various other values necessary for the proper supervision of circulatory system.

**5.Doppler echocardiography**: Ultrasonic monitoring of cardiac function. Recently constantly used by transoesophageal approach during cardiac surgical procedures. Significant because of its ability to diagnose acute myocardial infarction within ten seconds, unlike other methods. However, there are contraindications to its use (transoesophageal).

**6. Monitoring of tissue perfusion**: For now, there are two well-established methods.
a. Tonometry of the gastrointestinal tract mucosa: Allows an indirect measurement of the partial pressure of mucosal carbon dioxide ($pCO_2$) and pH calculation.
b. The saturation of mixed venous blood: To be taken from the pulmonary artery with the assistance of Swan-Ganz catheter. Based on the obtained values it provides estimation of the state of consumption of oxygen and perfusion.

**Monitoring of the respiratory system**
**1. Auscultation using a stethoscope** is the oldest method that is also very often used nowadays. Not objective enough.
**2. Pulse oximetry**: Allows for a non-invasive assessment of arterial oxygenation of peripheral blood. It is based on the change in the light absorption (red and infrared) which passes through the pulsating arterial vasculature. It belongs to the usual and standard patient monitoring. Not disabled by hyperpigmentation (black race), hyperbilirubinemia or anemia (with exception of severe anemia).
**Limitations**: Incorrect values in serious hypoxemia (below 75%), in cases of abnormal arterial pulsations, hypoperfusion, vasoconstriction, hypotension. In case of carbon monoxide poisoning cannot demonstrate appropriate values (shows higher values). It cannot completely replace the arterial blood gases analysis.
**Indications**: broad application in ICU's. Control of oxygenation in patients on mechanical ventilation, and during the various procedures and interventions such as bronchoscopy, gastrointestinal endoscopy, cardioversion, etc.
**Sensor placement sites**: the peaks of the nose and fingers and ears.

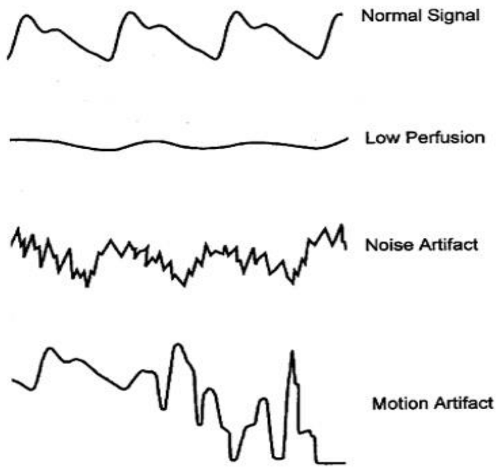



Image 1. Various signals received by pulse oximetry

**3. Monitoring of $CO_2$ - capnography**: Continuous monitoring of $CO_2$ concentration during each exhalation. The concentration of carbon dioxide at the end of exhalation ($E_TCO_2$) is approximately equal to the concentration of this gas in arterial blood ($PaCO_2$) in patients with normal pulmonary function. Large differences between $E_TCO_2$ and $PaCO_2$ can be caused by poor lung perfusion or intrapulmonary shunts. The progressive increase in $E_TCO_2$ may indicate hypoventilation, airway obstruction or increased metabolism.

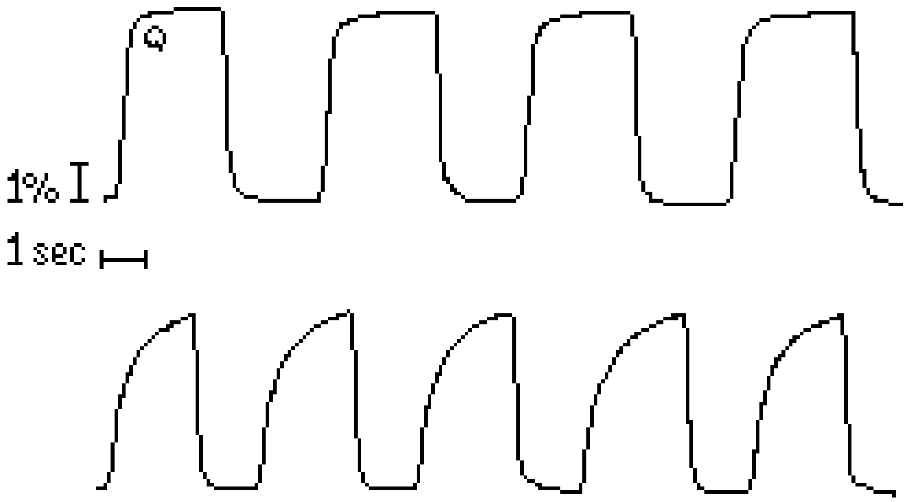



Image 2. Capnograph measured normally (above) and during an asthmatic episode (below)

**4. Transcutaneous measurement of blood gases**: It is based on the determination of the partial pressure of oxygen and carbon dioxide in the tissue using the infrared part of the light ($CO_2$) while heating the electrodes to 43-45°C ($O_2$). Correlates well with the partial pressure of oxygen in arterial blood. Routinely used in the neonatal intensive care unit.
Note: Monitoring location needs to be changed every 4-6 hours because of possible thermal injury.

**5. Respiratory mechanics monitoring**: Includes directly measured values and those calculated from these values. The most commonly measured values are tidal volume, minute volume, airway pressure, intrathoracic pressure. Typical derived values are lung compliance, airway resistance, work of breathing.

**Monitoring of the nervous system**

1. **GCS - Glasgow coma score -** a scale that allows evaluation of the state of consciousness. The minimum number of points is 3, and the maximum 15. It is estimated on the basis of eye opening, motor activity and verbal responses.

Table 1: Glasgow coma score

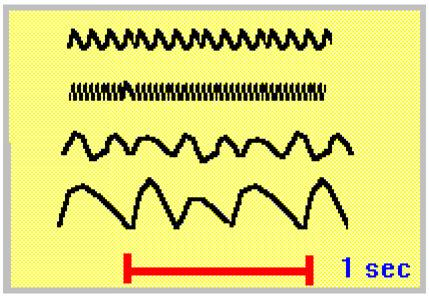
| **Eye opening** | **score** |
|---|---|
| Spontaneous | 4 |
| On call | 3 |
| On pain stimulus | 2 |
| Absent | 1 |
| **Best motoric response** | |
| Following commands | 6 |
| Localising pain | 5 |
| Retraction on pain stimulus | 4 |
| Decortication flexion | 3 |
| Extension | 2 |
| Absence of response | 1 |
| **Best verbal response** | |
| Oriented | 5 |
| Confused | 4 |
| Inadequate | 3 |
| Indistinguishable speech | 2 |
| Absence of response | 1 |
| **Total** | 3-15 |

**2. Neurological status**: Follows the presence or absence of reflexes, pathological phenomena, pupil width and reaction to light, motor power.

**3. Intracranial pressure:** Used to confirm the diagnosis of elevated intracranial pressure and to monitor the success of treatment. It is indicated in head injury with GCS <7 or pathologic CT. Complications: Infection, bleeding, especially in patients with coagulopathy or during difficult insertions.

**4. Electroencephalography:** EEG reflects changes in cortical electrical activity. It records spontaneous activity. This activity is dependent on cerebral perfusion and oxygenation.

Conventional EEG can be used intermittently, but is expensive and impractical. Therefore the so-called Cerebral function nonitor (CFM) is often used in the ICU. CFM is single channel. The so called "Bispectral index" monitor or BIS (anesthesia depth monitor) is also designed on the basis of the EEG. It is used during anesthesia in surgical procedures.
Image 3: EEG waveforms, from top to bottom: α waves, β waves , θ waves, δ waves




**5. Evoked potentials:** Electrical signals that occur in the nerve pathways after periodic external stimulation. They are divided into auditory, visual and somatosensoric.

**6. Cerebral blood flow:**
a. Transcranial Doppler ultrasound is based on ultrasound, like other echo-based diagnostic tools. It is used in certain cardiac surgery procedures.
b. Radioisotope imaging, usually xenon, which is administered intravenously or by inhalation. The drawback is a need of ptient transportation to the laboratory with the gamma camera, which is highly impractical, so far used only as a diagnostic tool.

**7. Cerebral oxygenation monitoring** is performed in two ways
a. saturation of venous blood in the internal jugular bulbus
b. "Near-infra-red» spectroscopy (NIRS) based on the absorption of infrared light wavelengths of 700-1000 nm, by hemoglobin, myoglobin and cytochrome aa3.

**Laboratory monitoring**
Different biochemical tests associated with particular organs function (liver, kidneys, heart, brain), the degradation products, electrolytes, enzymes in the blood. Condition of the immune system; specific and non-specific indicators and acute phase proteins (eg. CRP: C-reactive protein). Coagulation status. Circulatory condition - exsanguinity. Nutritional status. Blood gas analysis. Microbiological and toxicological tests. Biochemical markers of cardiac and brain injury etc.

***Other monitoring methods***
**1.Temperature.** Detecting increased or decreased body temperature. It has significance in diagnosing certain conditions and pathological processes (sepsis, hyper- or hypothermia).
**2. Diuresis.** Along with partial kidney function it often indicates the state of cardio-vascular system.
(low blood pressure or low volume -exsanguinity, low urine output). The possibility of infections due to urinary catheters.
**3. Presence of peristalsis**. Tube retention (naso- or orogastric) refers to the condition and

possible complications of the gastrointestinal system, particularly after surgical procedures. Intraabdominal pressure facilitates decision making for surgical procedure; eg in pancreatitis.

# 5. STRUCTURED APPROACH TO THE SERIOUSLY INJURED

**Mihajlo Lojpur, M.D., Ph.D.**

## INTRODUCTION

The severity of injuries can range from minor to life-threatening. Namely, they may affect many parts of the body, including the brain, the extremities and internal organs. Injuries of vital organs are more serious than injuries of limbs, although the injuries of the limbs can also be the cause of serious morbidity and mortality.

The multitude of possible injuries to various organ systems makes major trauma a complex problem. A systematic, organized approach to each patient, following International Trauma Life Support **(ITLS)** guidelines, is necessary to determine the patient's condition and appropriate interventions. This, as well as rapid transport will have the greatest impact on trauma survival in the pre-hospital setting.

## EPIDEMIOLOGY

In developed countries, every year about 3% of the total population experiences a serious trauma. Of the total number injured, 4% are permanently disabled and 1.5% dies. It is important to note that death and disability due to trauma affects mostly the young adult segment of the population (people ages 1-45).

## STRUCTURED APPROACH TO THE SERIOUSLY INJURED

Whether you are a bystander when an accident occurs, or working in the ED when a Trauma Alert is called, a quick, thorough, ITLS patient assessment is the essential first step in effective patient management. It is important to have a systematic way to approach trauma patients, to ensure that nothing is missed.

Steps of ITLS Patient Assessment are:

1. Primary Survey
2. Secondary Survey, and
3. Ongoing Exam

## PRIMARY SURVEY

The **ITLS primary survey** is a rapid examination used to identify and intervene in other life-threatening conditions and injuries and to make transport decisions. It is divided into 3 parts:

**1.** The Scene Survey,
**2.** The Initial Assessment, and
**3.** The Rapid Trauma Survey or Focused Exam.

The primary survey should take no more than 1.5-2 minutes and should be interrupted only for safety, airway obstruction and/or cardiac arrest.

SCENE SURVEY includes 5 components:

1. **Personal Protective Equipment -** Keeping yourself protected is the first priority of any first aider. The key skill for this is awareness of your surroundings and the changing situation. Once you are aware of the hazards, you can then take steps to minimize the risk

to oneself. One of the key dangers to a first aider is bodily fluids, such as blood, vomit, urine and feces, which pose a risk of cross contamination. Body fluids can carry infections and diseases, including, but not limited to, HIV and hepatitis.

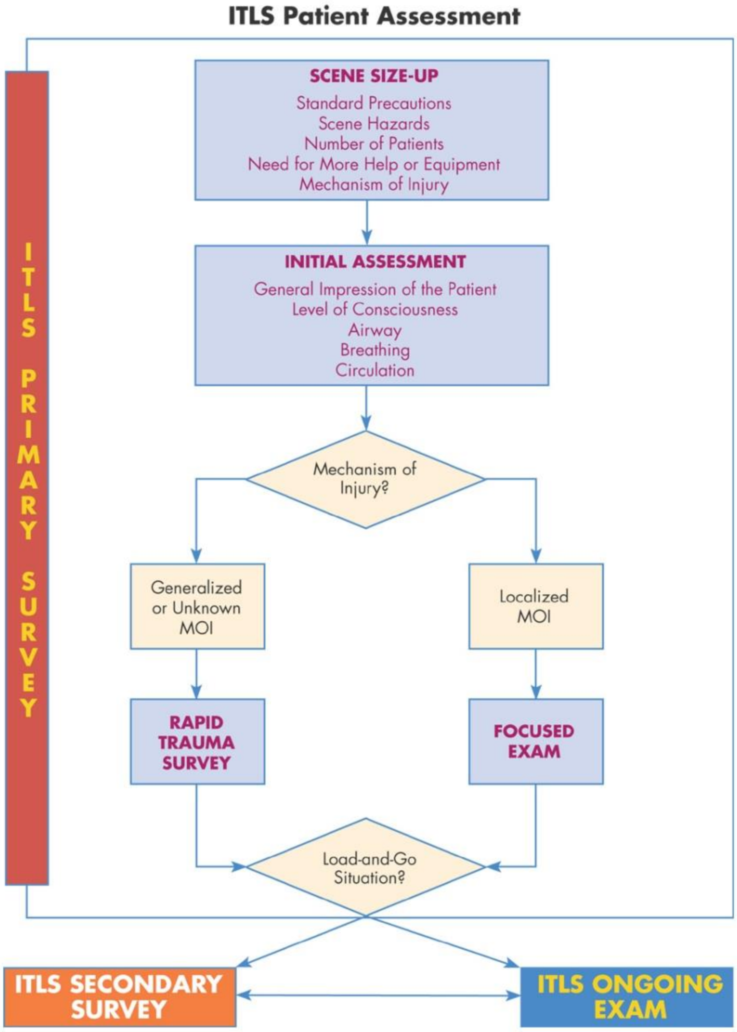



*Figure 1. ITLS algorithm*

The main tool of the first aider to avoid this risk is a pair of impermeable gloves. Gloves protect the key contact point with the victim (i.e. the hands) and allow you to work in increased safety. They protect not only from bodily fluids, but from any dermatological infections or parasites that the victim may have.

The other key piece of protective equipment that should be in every first aid kit is an adjunct for helping to perform safe mouth-to-mouth resuscitation. With mouth-to-mouth resuscitation, there is a high probability of bodily fluid contact, especially with regurgitated stomach contents and mouth borne infections. A suitable mask will protect the rescuer from infections the victim may carry (and to some extent, protect the victim from the rescuer). It also makes the performance of CPR less onerous (not wishing to perform mouth to mouth is a key reason cited for bystanders not attempting CPR).

CPR adjuncts come in a variety of forms, from small keyrings with a nitrile plastic shield, up to a fitted rescue 'pocket mask' such as the one pictured.

Larger first aid kits, or those in high risk areas could contain additional equipment such as:

• **Safety glasses** - Prevents spurting or pooled fluid which could splay from coming in contact with the eyes.

• **Apron or gown** - Disposable aprons are common items in larger kits, and help protect the rescuers clothing from contamination.

• **Filter breathing mask** - Some large kits, especially in high risk areas such as chemical plants, may contain breathing masks which filter out harmful chemicals or pathogens. These can be useful in normal first aid kits for dealing with victim who are suffering from communicable respiratory infections such as tuberculosis.
Often, all of these will be included as a part of a larger kit. The kit should have a list of instructions on how to properly don/don off the equipment. Follow these instructions to prevent an accidental exposure.


Figure 2. *Nitrile Glove* *Resusciation Face Shield* *CPR pocket mask, with carrying case*

2. **Scene hazards** - As you approach a scene, you need to be aware of the dangers which might be posed to you as a first aider, or to the victim. These can include obviously dangerous factors such as traffic, gas or chemical leaks, live electrical items, buildings on fire or falling objects.

There are also human factors, such as bystanders in the way, victim not being co-operative, or an aggressor in the vicinity who may have inflicted the injuries on the victim. If these factors are present, have the police called to control the situation.
If there are dangers which you cannot mitigate by your actions (such as falling masonry), then STAY CLEAR and call the emergency services. Remember to **never put yourself in harms way**.

3. **Number of patients** - Determine the total number of patients

4. **Need for more help or equipment** – If there are more patients than the responding unit can effectively handle, initiate a mass casualty plan. Begin triage.

5. **Mechanisms of injury (Generalized or focused? Potentially life – threatening?)** - determine from the patient, family, or bystanders and inspection of the scene (Examples: Vehicle Crash – Speed at impact? Rollover /Head-on, Rear end?; Fall – How high and onto what?; Stabbing – What caliber gun?)

INITIAL ASSESSMENT AND RESUSCITATION OF THE INJURED PATIENT

In severe trauma, assessment and resuscitation should be performed simultaneously. The purpose of the initial evaluation and management is to diagnose and address life-threatening problems, which can cause death or serious morbidity if not treated early.

The initial assessment includes 5 components, which should always be followed in strict order.

1. **General impression of the patient** – The general impression will help you decide the seriousness of the patient's condition based on his level of distress and mental status.
   The general impression contains the following elements: approximate age, sex, and level of distress or responsiveness.
   Examples of a typical general impression may look something like the following: I have an approximately 30-year-old male in moderate distress. Or I have an approximately 60-year-old female who appears to be unresponsive.
   Emergency Medical Responders have always formed a general impression when they first see a patient, even if they are not immediately aware of doing so. With experience, you may form one on intuition alone. You may notice if the patient looks very ill, pale, or cyanotic. You may notice unusual details such as odors, temperature, and living conditions. You may immediately see serious injuries or that the patient looks stable.
   This impression forms an early opinion of how seriously ill or injured the patient is. Your decision to request immediate transport or to continue assessing the patient may be based solely on your general impression.

2. **Level of consciousness (LOC)** – our actual assessment of a patient begins by determining the patient's level of responsiveness. You must quickly determine if he is responsive or unresponsive. A responsive patient may be obviously awake and interacting with those around him. An unresponsive person may not be so obvious.

You must kneel beside the patient, tap his shoulder, and state loudly something like, “Are you okay?” or “Can you hear me?” If he responds, you know he is not totally unresponsive. You will then categorize his level of responsiveness based on the AVPU scale, the letters of which stand for alert, verbal, painful, and unresponsive.

***A — Alert.*** The alert patient will be awake, responsive, oriented, and talking with you.

***V — Verbal.*** This is a patient who appears to be unresponsive at first but will respond to a loud verbal stimulus from you.

***P — Painful.*** If the patient does not respond to verbal stimuli, he may respond to painful stimuli, such as a sternal (breastbone) rub or a gentle pinch to the shoulder.

***U — Unresponsive.*** If the patient does not respond to either verbal or painful stimuli, he is said to be unresponsive.

3. **Airway assessment and protection** (maintain cervical spine stabilization when appropriate)

4. **Breathing and ventilation assessment** (maintain adequate oxygenation)
   If the patient is unresponsive, check for adequate breathing by observing the chest rise and fall. The patient is not breathing if there is no chest movement. Gasping respirations are called *agonal respirations*. They should not be considered normal respirations.

5. **Circulation assessment** (control hemorrhage and maintain adequate end-organ perfusion)
   ***a. Check for a Pulse***
   If the patient is not breathing, check for a **carotid pulse** at the neck to determine if blood is circulating. The pulse at the neck is considered more reliable than the pulse at the wrist. A pulse at the wrist (the **radial pulse**) may not be present if the patient is in shock.
   Check for a carotid pulse for 5 to 10 seconds. It is not important during the primary assessment to count the exact rate of the pulse.You only want to confirm the presence of a pulse.
   - If the pulse is very rapid or weak, the patient may be in shock.
   - If there is no pulse, alert dispatch and begin CPR.
   - If the patient is not breathing but does have a pulse, the patient may have an airway obstruction or he may be in respiratory arrest. You must take immediate action to ventilate the patient before the heart stops.

   ***b. Check for Serious Bleeding***
   The next step in the primary assessment is checking for serious bleeding. While any uncontrolled bleeding may eventually become life threatening, you will only be concerned with profuse bleeding during the primary assessment. Blood that is bright red and spurting may be coming from an *artery.* Because blood in arteries is under a great deal of pressure, large amounts of blood may be lost in a short period of time. Flowing blood that is darker in color is most likely coming from a *vein.* Even if the bleeding is slow, it may be life threatening if the patient has been bleeding for a long period of time.
   Look at the amount of blood that has been lost on the ground, in clothing, and in the hair. Your concern is for the total amount of blood that has been lost, not just how fast or slow the bleeding is.

Assessment of circulation may be altered slightly when you immediately see profuse bleeding. In this case, attempt to control the bleeding as soon as it is discovered. Do what you can to control it, but never neglect the patient's airway and breathing status.

After initial assessment you will continue with a rapid trauma survey or focused exam depending on the mechanism of injury and the results of the initial assessment of the injured. The trauma patient is classified as either having no significant mechanism of injury (probably not causing a serious injury) or having a significant mechanism of injury (probably causing a serious injury).

- To assess a trauma patient with no significant mechanism of injury, begin by performing a focused assessment on the area that the patient tells you is injured
- To detect and care for serious injuries in a patient with a significant mechanism of injury, perform a rapid trauma assessment looking for obvious injuries

*Table 1. Significant mechanism of injury for adults and children*

| **Signs of significant mechanisms of injury for an adult include:** | **Significant mechanisms of injury for a child include:** |
|---|---|
| • Ejection from a vehicle<br>• Death of one or more passengers in a motor-vehicle crash<br>• Falls greater than 15 feet<br>• Rollover vehicle collision<br>• High-speed vehicle collision<br>• Vehicle-pedestrian collision<br>• Motorcycle crash<br>• Unresponsiveness or altered mental status<br>• Penetrations of the head, neck, chest, or abdomen | • Falls of more than 10 feet<br>• Bicycle collision<br>• Medium-speed vehicle collision |

RAPID TRAUMA SURVEY

It is a quick method (should take no more than two to three minutes), to identify hidden and obvious injuries in a trauma victim. The goal is to identify and treat immediate threats to life that may not have been obvious during an initial assessment.

Rapid trauma survey is quick and systematic (from head to toe) exam of these body sections: Head and Neck, Chest, Abdomen, Pelvis, Extremities and Back :

- **Head**: DCAP-BLS-TIC (Deformity, Contusions, Abrasions, Puncture/Penetration - Burns, Tenderness, Laceration, Swelling - Trauma, Instability, Crepitus)
- **Neck**: DCAP-BLS-TIC, Tracheal Deviation, Neck veins
- **Chest**: DCAP-BLS-TIC, Look (equal rise and fall), Listen (auscultate) and Feel (crepitus, subcutaneous emphysema, fractures, flail segment), Percuss, Heart Sounds

- **Abdomen**: DCAP-BLS, Penetrating wounds, Flaccid, rigid, pain, tender
- **Pelvis**: DCAP-BLS-TIC
- **Extremities:** crepitus, pain, exposed bone ends
- **Back**: Examine posterior during roll and placement on backboard- DCAP-BLS-TIC

Finally take initial vital signs (blood pressure, pulse and breathing rate) and collect details of the history according to the form designated by the acronym SAMPLE

- The first set of ***vital signs*** is called *baseline vital signs*. Compare all other vital sign readings to the baseline vital signs. This comparison helps determine if the patient is stable or unstable, improving or growing worse, and benefiting or not benefiting from care procedures.
  For an adult, a continuous pulse rate of less than 60 beats per minute or above 100 beats per minute is considered abnormal. Likewise, a respiratory rate above 26 breaths per minute or below 10 breaths per minute is considered serious. You should be concerned about these vital signs because they indicate unstable situations that could become life threatening, and the patient could worsen quickly.

- ***SAMPLE history*** is a mnemonic acronym to remember key questions for a person's assessment. It is used for alert people, but often much of this information can also be obtained from the family or friend of an unresponsive person. The parts of the mnemonic are:

  1. **S**igns and Symptoms
  2. **A**llergies
  3. **M**edications
  4. **P**ast medical history, injuries, illnesses
  5. **L**ast meal/intake
  6. **E**vents leading up to the injury and/or illness

If the patient has impaired consciousness make a brief neurological examination (perform this exam now if there is altered mental status; otherwise, perform it during the detailed secondary survey).

This mini neurological assessment is made to establish:

- Level of consciousness, using GCS.

*Table 2. Glasgow coma scale.*

| Glasgow Coma Scale | | | | | | |
|---|---|---|---|---|---|---|
| | **1** | **2** | **3** | **4** | **5** | **6** |
| **Eye** | Does not open eyes | Opens eyes in response to painful stimuli | Opens eyes in response to voice | Opens eyes spontaneously | N/A | N/A |
| **Verbal** | Makes no sounds | Incomprehensible sounds | Utters inappropriate words | Confused, disoriented | Oriented, converses normally | N/A |
| **Motor** | Makes no movements | Extension to painful stimuli (decerebrate response) | Abnormal flexion to painful stimuli (decorticate response) | Flexion / Withdrawal to painful stimuli | Localizes painful stimuli | Obeys commands |

*The scale is composed of three tests: eye, verbal and motor responses. The three values separately as well as their sum are considered. The lowest possible GCS (the sum) is 3 (deep coma or death), while the highest is 15 (fully awake person). Generally, brain injury is classified as Severe, with GCS < 8-9, Moderate, GCS 8 or 9–12 (controversial), Minor, GCS ≥ 13.*

- Pupils: size, symmetry and reaction.

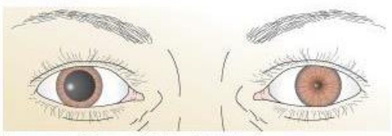



*Figure 3 . Pupil abnormalities in head trauma (eg one pupil may be dilated and the other may be constricted or pupil reaction may be sluggish to light)*

- Any lateralising signs or signs of cerebral herniation (unconscious, dilated pupil(s), hypertension, bradycardia, posturing)?

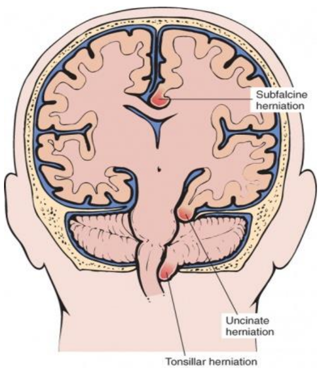



*Figure 4. Brain herniation - it occurs when something inside the skull produces pressure that moves brain tissues. This is most often the result of brain swelling from a head injury. Herniation compresses brain tissue and thus damages it.*

FOCUSED EXAM

When your trauma patient has no significant mechanism of injury, the steps of further assessment are appropriately simplified. Instead of examining the patient from head to toe (rapid trauma survey), focus your assessment on just the areas that the patient tells you are painful or that you suspect may be injured because of the mechanism of injury. The assessment includes a physical exam, a baseline set of vital signs, and a patient SAMPLE history.

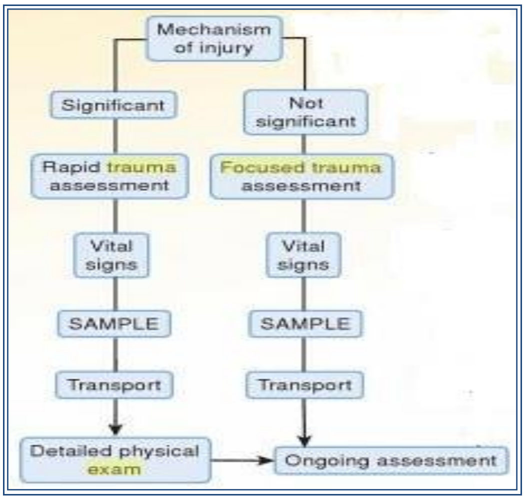


*Figure 5. The mechanism of injury and further examination of the injured*

This is used for patients with a medical complaint who are conscious, able to adequately relate their chief complaint to you, and have no life-threatening conditions.

## LOAD AND GO SITUATION

In some situations, the nature or severity of a patient's illness or injury may exceed the EMS personnel's ability to effectively manage the patient. Lifesaving treatments may need to be initiated within a short time frame (minutes to hours) if the patient is to have any chance for survival. These critically ill or injured patients require immediate transport for appropriate, specialized lifesaving techniques and therapies available in a hospital setting.

Life-threatening conditions for load and go situations may be identified during initial assessement or during rapid trauma survey/focused exam of primary survey, or at any point during the response.

*Table 3. Life-threatening conditions for load and go situations*

| | |
|---|---|
| Initial assessement | Decreased LOC or altered mental status |
| | Abnormal respirations |
| | Abnormal circulation (shock or uncontrolled hemorrhage) |
| Rapid Trauma Survey | Abnormal Chest Exam |
| | Tender, distended abdomen |
| | Pelvic instability |
| | Bilateral femur fractures |
| | Significant mechanism of injury or poor general impression |

***LOC** - *level of* consciousness

## SECONDARY SURVEY

When the primary survey is completed, resuscitation efforts are well established, and the vital signs are normalizing, the secondary survey can begin.

It includes 2 components :

1. Repeated initial assessment, and
2. Detailed exam


*Figure 6 . ITLS secondary survey*

The detailed exam includes a detailed history, repeat vital signs and thorough but efficient physical examination.

In detailed, head to toe physical exam, you will examine the same body areas that you examined during your rapid assessment but you will look more closely at each area to search for findings of lesser priority than life threats and/or signs of injury that have worsened.

You should look for abnormalities. This can take the form of asymmetry, deformity, bruising, point tenderness, minor bleeding and medic alert bracelets or necklaces.

The injured should be reviewed in the following order:

- **Head** - Check the scalp for cuts, bruises, swellings, and other signs of injury. Examine the skull for deformities, depressions, and other signs of injury. Inspect the eyelids/eyes for impaled objects or other injury. Determine pupil size, equality, and reactions to light. Note the colour of the inner surface of the eyelids. Look for blood, clear fluids, or bloody fluids in the nose and ears. Examine the mouth for airway obstructions, blood, and any odd odors.

- **Neck** - Examine the patient for point tenderness or deformity of the cervical spine. Any tenderness or deformity should be an indication of a possible spine injury. If the patient's C-spine has not been immobilized immobilize now prior to moving on with the rest of the exam. Check to see if the patient is a neck breather, check for tracheal deviation
- **Chest** - Examine the chest for cuts, bruises, penetrations, and impaled objects. Check for fractures. Note chest movements a look for equal expansion.
- **Abdomen** - Examine the abdomen for cuts bruises, penetrations, and impaled objects. Feel the abdomen for tenderness. Gently press on the abdomen with the palm side of the fingers, noting any areas that are rigid, swollen, or painful. Note if the pain is in one spot or generalized. Check by quadrants and document any problems in a specific quadrant.
- **Lower Back** - Feel for point tenderness, deformity, and other signs of injury
- **Pelvis** - Feel the pelvis for injuries and possible fractures. After checking the lower back, slide your hands from the small of the back to the lateral wings of the pelvis. Press in and down at the same time noting the presence of pain and/ or deformity
- **Genital Region** - Look for wetness caused by incontinence or bleeding or impaled objects. In male patients check for priapism (persistent erection of the penis). This is an important indication of spinal injury
- **Lower Extremities** - Examine for deformities, swellings, bleedings, discolorations, bone protrusions and obvious fractures. Check for a distal pulse. The most useful is the posterior tibial pulse which is felt behind the medial ankle. If a patient is wearing boots and has indications of a crush injury, do not remove them. Check the feet for motor function and sensation.
- **Upper Extremities** - Examine for deformities, swellings, bleedings, discolorations, bone protrusions and obvious fractures. Check for the radial pulse (wrist). In children check for capillary refill. Check for motor function and strength.

Do not delay transport to perform a detailed physical exam; it is only performed while enroute to the hospital or while waiting for transport to arrive.

The secondary survey plays a crucial role in avoiding missed injuries.

*Table 4. Commonly missed injuries*

| |
|---|
| • Blunt abdominal trauma: Hollow viscus injury, pancreatoduodenal injuries, diaphragmatic rupture |
| • Penetrating abdominal trauma: Rectal and ureteral injuries<br>• Thoracic trauma: Aortic injuries, pericardial tamponade, esophageal perforation |
| • Extremity trauma: Fractures (especially in distal extremities), vascular disruption, compartment syndrome |

## ONGOING EXAM

The ongoing assessment will be performed on all patients while the patient is being transported to the hospital. It is designed to reassess the patient for changes that may require new intervention. You will also evaluate the effectiveness of earlier interventions, and reassess earlier significant findings.

You should be prepared to modify treatment as appropriate and begin new treatment on the basis of your findings during the ongoing assessment.

Seriously ill or injured patients should be reassessed every five minutes. A good rule to follow is that by the time you finish a reassessment from start to finish, it is time to start over with the beginning of the next reassessment. Patients who are not seriously ill or injured should be reassessed every 15 minutes.

*Table 5. Ongoing exam*

| ITLS Ongoing Exam |
|---|
| **Subjective Changes**- “How do you feel? |
| **Reassess Mental Status** - LOC, Pupils, GCS |
| **Reassess ABCs** - Patency, Vital Signs, Color, Skin Condition, Temperature, JVD, Tracheal Deviation, Breath Sounds, Heart Tones |
| **Reassess Abdomen** - Development of Tenderness, Distention, Rigidity |
| **Check Each Identified Injury** - Change in Status, PMS |
| **Check Interventions** - Patency, Position, Flow Rate, Security |

***PMS** - pulse, motor, and sensory*

# 6. SHOCK

Nenad Karanović, MD, PhD

Shock is a clinical syndrome whose principal characteristic is the inability to maintain adequate blood flow (perfusion) to tissues and organs with the consequential lack of oxygen, which, if continued, leads to severe disorders of organ function with lethal outcome. This is a condition in which the circulation is not able to supply sufficient oxygen for the tissue demands, resulting in cellular dysfunction. The result is a so-called cellular disoxia (disorder between the delivery of oxygen and its consumption. This is an explanation for the condition between anoxia - scarcity of oxygen and hypoxia - reduced oxygen concentration). Some clinical syndromes suggest the disorder at the level of microcirculation.

**Pathophysiology:**

Shock is an acute clinical condition, immediate circulatory failure, which is a result of one or up to four different mechanisms.

The first cause is a decrease in venous return of blood due to circulating volume loss (caused

by external or internal reasons).
Another cause is failing of the heart as a pump, which occurs due to loss of contractile function (resulting from ischemia, infarction, myopathies or myocarditis) or the occurrence of serious arrhythmias (ventricular tachycardia or severe disorders in the vascular system of the heart).
The third cause is the occurrence of obstruction (due to pulmonary embolism, pneumothorax or cardiac tamponade).
The fourth cause is the loss of vascular tone that results in poor distribution of circulating blood (sepsis, anaphylaxis, spinal injury).
There are a number of shock classifications, however, none is complete. For the simplicity reasons, a common classification into four basic types is applied nowadays; cardiogenic, distributive, obstructive and hypovolemic shock. Cardiogenic shock is caused by cardiac function failure. Distribution shock is caused by the widening of vascular structures due to various reasons and consequent "reduction" of circulating fluid. Obstructive shock is the result of impediments to the circulation of the blood, for example; pulmonary embolism. Hypovolemic shock arises from the reduction of circulating volume, mostly due to exsanguination.
There are various causes of shock situations involving injuries, external and internal bleeding, various diseases and organ systems failure, allergies, etc.
The forms of the above mentioned types of shock are often intertwined, so patients admitted for treatment of one form of shock can develop other forms. For example, the patient that was hospitalized due to hypovolemic or cardiogenic shock may occasionally develop septic shock.

**Epidemiology**: Approximately one third of patients admitted to the intensive care unit (ICU) are in a state of circulatory shock.
According to recent studies the septic shock is one of the most common in the ICU admissions with approximately 62% of prevalence. Next are cardiogenic and hypovolemic shock with 17% to 16%. The mortality of septic shock is between 40-50%, however, in some studies, it reaches up to 80%. The mortality rate of cardiogenic shock is projected to be 59.4%.

**Shock diagnosis**: Shock is diagnosed based on a combination of clinical, hemodynamic and biochemical signs.

**Clinical presentation**

1. Blood pressure is lowered (systolic pressure less than 95 mmHg or 40 mmHg of the value before deterioration), however hypotension is not always present.
2. Reduction of diuresis up to a standstill
3. Progressive increase in anaerobic metabolism indicators (lactate) in arterial blood
4. Consciousness disorder: from anxiety, through lethargy to coma.
5. Cardiac arrhythmia: in the early stages of shock heart rate is rapid while in the final stages of shock, especially in the case of severe hypovolemic shock, heart rate is bradycardic. Other arrhythmias also often appear.
6. Peripheral cyanosis

7. Rapid breathing (tachypnea) with shortness of breath (tachydyspnoea)
8. Sweating: skin moist and cold

**Treatment**: is based on solving the etiology of shock and supporting the organ systems. Etiological treatment is of great importance in the treatment of shock. Therefore in the case of, for example, septic shock antibiotics and antifungals should be administered along with other treatment measures. Bleeding must be stopped in hypovolemic shock along with compensation of circulating volume. Causes must be addressed in cases of cardiogenic shock , most commonly acute myocardial infarction, myocarditis and heart failure caused by various diseases.

**Hemodynamic support** is based on compensation and maintenance of circulating blood volume and maintaining the cardiovascular function with vasoactive drugs and inotropes (drugs that press or expand blood vessels and improve contractility of the heart). However, a strict attention should be paid to fluid intake in cardiogenic shock. Crystalloid solution are most frequently administered, then the colloids and blood derivatives. It should be noted that all drugs used in resuscitation also are applicable during the shock states.

**Respiratory support**: Delivery of oxygen is decreased in these patients, so the oxygen must be administered in high flow through a mask or nasal cannula. However, it is often necessary to use a machine-controlled ventilation using special devices with different modes of ventilation.

Other measures of treatment are based on the maintenance of homeostasis [(from the Greek. Homoios - similar, same; Stasis - state) feature of an organism to maintain its internal conditions stable and relatively constant] of the organism using different drugs and procedures. This includes measures to prevent various complications.

**Anaphylactic shock**

Is the most severe form of acute allergic reactions. It most frequently occurs very quickly, within a few minutes after exposure to an allergen, but it can also be delayed for several hours. It is caused by different drugs, medical devices (radiological contrasts, latex). However it can be caused by food, different insects and solutions used in the household. Mortality remains high even with measures of treatment and according to some data reaches up to 5%.

**Clinical presentation**
Cardiocirculatory system: hypotension and tachycardia.
Cutaneous manifestations (including mucosal): urticaria, generalized edema, localized Quincke's edema, itching.
Respiratory system: laryngospasm, bronchospasm, difficult and rapid breathing
Gastrointestinal system: vomiting, diarrhea

Nervous system: disorder and loss of consciousness
It is important to emphasize that the clinical presentation does not have to develop fully and that sometimes it is difficult to assess in the early stages whether the symptoms are caused by an allergic reaction or other events.

**Treatment**

1. Ensure patency of the airway (chin lift, deflection of the head - head tilt, oropharynegea, endotracheal tube)
2. Adrenaline 0.5-1 ml im or in severe cases 0.2 ml iv (not recommended for those who have no experience in the intravenous administration of these types of drugs) repeated if necessary every 10-15 minutes
3. Oxygen at high concentration
4. Administration of fluids, usually with crystalloid solution.
5. Antihistaminic drugs
6. Aminophilin if there is bronchospasm
7. Corticosteroids, however, they act with a delay
8. Support to the cardiocirculatory system. Vasoactive drugs, if necessary, inotropes.

# 7. HEART AND RESPIRATORY FAILURE

Mladen Carev, M.D. Ph.D.

## *1. HEART FAILURE*

### *1.1. INTRODUCTION*

*Heart failure* is a clinical syndrome that occurs due to changes in the function of the heart as a pump, and is typically manifested by characteristic clinical signs and symptoms. These signs and symptoms arise because cardiac function does not meet the metabolic needs of the organism. Because of the increase in retrograde pressure there is a congestion of organs, and consequently the fluid accumulates retrogradely ("backward") from one or both ventricles. Therefore, it is sometimes called congestive heart failure (congestion means the accumulation of blood in a part of the body).

Heart failure may be divided into *acute* and *chronic*. Acute heart failure has a sudden onset (e.g. after acute myocardial infarction). Chronic heart failure develops more slowly and is commonly seen in patients with dilated cardiomyopathy (cardiomyopathies are myocardial diseases with structural and functional changes in the heart muscle). There is also a classification into *systolic* and *diastolic* heart failure. In systolic failure the problem is in poor ventricular contractility i.e. in reduced ejection fraction (percentage of blood that is squeezed out of the heart with each contraction - normal is 50-75%). On the contrary, in diastolic heart failure there is a problem with filling and relaxation of the ventricles, while the contractility is normal, at least initially. It is sometimes referred to as heart failure with preserved ejection fraction (HFNEF)

When we talk about heart failure, we usually think of dysfunction and failure of the ventricles.

### *1.1.1 .LEFT VENTRICULAR FAILURE*

It occurs most frequently in coronary artery disease, uncontrolled hypertension, critical aortic stenosis and massive mitral and aortic regurgitation. Left ventricular failure usually causes dyspnea (shortness of breath) due to the increase in pulmonary blood pressure, and pulmonary congestion, as well as general malaise due to low cardiac output. In severe cases dyspnea might occur at night during sleep. Other possible clinical signs are increased heart (tachycardia) and respiratory rate (tachypnea).

### *1.1.2. RIGHT VENTRICULAR FAILURE*

Right-sided heart failure is very frequent with left ventricular failure, i.e. when the right ventricle pumps blood versus increased resistance (pulmonary hypertension). It is also seen with pulmonary embolism, right ventricular myocardial infarction, tricuspid regurgitation and stenosis of the pulmonary valve. Right ventricular failure causes fluid accumulation in the periphery (usually symmetrical ankle edema), due to the increase in systemic venous pressure. Hepatic dysfunction is a common feature. Edema may be extended all over the body, and this condition is termed anasarca. There could also be an enlarged liver, accumulation of fluid in the abdomen (ascites), and jugular veins distension.

### *1.1.3. BIVENTRICULAR FAILURE*

This term describes the failure of both ventricles, relatively often seen especially in the advanced stages of heart disease. Pleural effusions (fluid accumulation in the space between the lung and chest wall) can be a clinical sign of the biventricular failure, since the pleural veins drain both in the systemic and pulmonary venous circulation.

## 1.2. FUNCTIONAL CLASSIFICATION OF HEART FAILURE, METABOLIC EQUIVALENT OF TASK.

The New York Heart Association (NYHA) Functional Classification provides a simple way of classifying the extent of heart failure. It places patients in one of four categories based on how much they are limited during physical activity (*Table 1*). The patients in clinical categories NYHA III and NYHA IV require special clinical attention and care. Regarding this issue we often use abbreviation MET (Metabolic Equivalent of Task). MET is an attempt to compare energy consumption during various activities among patients of different weights. MET is a physiological measure that expresses the energy requirements to perform a variety of physical activities, and is most frequently defined as the ratio between energy consumed during specific activities and basal energy consumption; thereby 1 MET is considered as a metabolic consumption at rest during a quiet sitting and this reference value is by convention 1 kcal/kg/ h. MET values could range from 0.9 (sleep) to 23 (fast running) (*Table 2*).

Table 1. NYHA Functional Classification of Heart Failure

| NYHA CLASS | DEFINITION | LIMITATIONS | LINKAGE TO MET |
|---|---|---|---|
| I | Cardiac disease, but no symptoms and no limitation in ordinary physical activity, e.g. no shortness of breath when walking, climbing stairs etc. | None | Any activity requiring ≤7 MET can be performed |
| II | Mild symptoms (mild shortness of breath and/or angina) and slight limitation during ordinary activity. | Mild | Any activity requiring ≤5 MET can be performed |
| III | Marked limitation in activity due to symptoms, even during less-than-ordinary activity, e.g. walking short distances (20–100 m).<br>Comfortable only at rest. | Moderate | Any activity requiring ≤2 MET can be performed |
| IV | Severe limitations. Experiences symptoms even while at rest. Mostly bedbound patients. | Maximal | Any activity requiring ≤2 MET can not be performed |

Table 2. The association of certain activities with metabolic equivalent (MET)

| **PHYSICAL ACTIVITY** | **MET** |
|---|---|
| **Light intensity activities** | **<3** |
| Sleeping | 0.9 |
| Watching TV | 1 |
| Writing, desk work, typing | 1.8 |
| Work – standing (cooking, washing dishes) | 2-2.5 |
| Walking (4 km/h pace) | 2.9 |
| **Moderate intensity activities** | **3-6** |
| Cleaning, washing windows, vacuuming | 3-3.5 |
| Walking (5.5 km/h pace) | 3.6 |
| Bicycling, <16 km/h, leisure, to work or for pleasure | 4 |
| Badminton | 4.5 |
| Sexual activity | 5.8 |
| **Vigorous physical activity** | **>6** |
| Jogging | 7 |
| Heavy exercising (pushups, situps, pullups) | 8 |
| Rope jumping | 10 |
| Fast running (22.5 km/h) | 23 |

## 1.3. CLINICAL SYMPTOMS AND SIGNS OF HEART FAILURE

The most common clinical symptoms that may suggest heart failure are: dyspnea (shortness of breath), orthopnea, paroxysmal nocturnal dyspnea, fatigue, and some cerebral symptoms (confusion, difficulty in concentration and memory).

*Dyspnea* is considered to be a subjective unpleasant feeling of shortness of breath (sometimes described as "air hunger"), occurring in the initial stages only on exertion and later at rest.

Orthopnea is dyspnoea in a patient lying flat and is a sign of the advanced heart failure. The mechanism is increased venous return of blood to the heart in supine position. Consequently, patients sleep with more and pillows under head, aiming at reducing venous return and pulmonary congestion. The number of set pillows might be considered proportional to the severity of heart failure.

*Paroxysmal nocturnal dyspnea* is a very unpleasant sensation that awakens patients from sleep. It has a similar mechanism as orthopnea; it is caused by a gradual accumulation of alveolar fluid during sleep. Patients may sit on the edge of the bed or open the windows trying to relieve the symptoms. Fatigue and cerebral symptoms occur because of decreased flow through the muscles, and the brain.

Objective clinical signs may include the *Cheyne-Stokes respiration* (cyclic breathing, i.e. the intervals of rapid and deep breathing followed by intervals of complete cessation of breathing), *engorged neck veins*, *rales* found with auscultation of the lungs, together with the possible occurrence of *pink*, *frothy sputum* (*acute pulmonary edema*). Furthermore there is a *peripheral edema*, usually on the ankles, but also in the sacral area. It is possible to encounter also *pleural effusion* (somewhat more to the right), and aforementioned *hepatomegaly* and *ascites*. *Jaundice* is the result of the liver damage due to congestion. Other possible clinical signs are c*old*, *cyanotic limbs* and *oliguria*.

For a better understanding of this issue the Framingham criteria for the diagnosis of heart failure will be presented here (http://www.medicalcriteria.com/criteria/framingham.htm). According to them, the simultaneous presence of at least two major criteria or 1 major with two minor criteria is required for the diagnosis of heart failure.

Major criteria are: paroxysmal nocturnal dyspnea, neck vein distention, rales, radiographic cardiomegaly (increasing heart size on chest radiography), acute pulmonary edema, S3 gallop (third heart sound), increased central venous pressure (>16 cm $H_2O$ at right atrium), hepatojugular reflux, weight loss >4.5 kg in 5 days in response to treatment

Minor criteria are: bilateral ankle edema, nocturnal cough, dyspnea on ordinary exertion, hepatomegaly, pleural effusion, decrease in vital capacity by one third from maximum recorded, tachycardia (heart rate>120 beats/min.). Minor criteria are acceptable only if they can not be attributed to another medical condition (such as pulmonary hypertension, chronic lung disease, cirrhosis, ascites, or the nephrotic syndrome). The Framingham Heart Study criteria are 100% sensitive and 78% specific for identifying persons with definite congestive heart failure.

## 1.4. CARDIOGENIC AND CARDIOCOMPRESSIVE SHOCK

Cardiogenic shock is, by definition, the loss of proper functioning of the heart pump (decreased cardiac output), associated with subsequent decreased blood flow to the vital organs. Cardiocompressive (or obstructive) shock is a condition with low cardiac output due to external compression of large veins and heart cavities, which reduces their normal filling and emptying.

### *1.4.1. CARDIOGENIC SHOCK*

Clinical symptoms of cardiogenic shock are reduced urinary output, mental disturbances, cold limbs, engorged neck veins and hypotension with evidence of systemic (peripheral edema, hepatomegaly) and pulmonary venous congestion (pulmonary edema - fluid accumulation in the lungs). One should not forget rales during lung auscultation, as well as the third heart sound and heart murmurs during heart auscultation.

The most common causes of cardiogenic shock are:

- Non-mechanical causes
  - Acute myocardial infarction (pay attention to chest pain with typical expansion!!!)
  - Low cardiac output syndrome (cardiomyopathy, cardiac surgery)
  - Right ventricle myocardial infarction,
  - Terminal cardiomyopathy
- Mechanical causes
  - rupture of the septum or free wall,
  - Severe mitral insufficiency,
  - Severe aortic insufficiency,
  - Papillary muscle rupture,
  - Critical aortic stenosis
- Arrhythmias
  - Bradycardia (and bradyarrhythmias) - slow (and irregular) rhythm
  - Tachycardia (and tachyarrhythmias) - fast (and irregular) rhythm

Treatment of cardiogenic shock consists in the optimizing blood (diuretics usually required - stimulate urination), and if necessary endotracheal intubation (especially with pulmonary edema), and inotropes (the special category of drugs enhancing myocardial contractility) - dobutamine, epinephrine, milrinone, levosimendan. Sometimes the intraortic balloon pump (IABP) and emergency cardiac surgery will be required.

### *1.4.2. CARDIOCOMPRESSIVE SHOCK – PERICARDIAL TAMPONADE, TENSION PNEUMOTHORAX*

Cardiocompressive shock is actually a type of obstructive shock (it includes also pulmonary embolism described below). The clinical picture is quite the same as in cardiogenic shock, except that the hypotension is usually accompanied by tachycardia.

A common cause of this shock is *pericardial tamponade*, i.e. the accumulation of fluid in the pericardial sac. This accumulation of fluid prevents the normal filling of the heart during diastole, increases pressure in the great veins, and decreases the stroke volume and cardiac output, with a resulting hypotension and increased heart rate as a compensatory response.

It is possible after trauma in the case of coronary artery laceration, which then bleeds into the pericardial sac, but it is also seen in some chronic diseases (uremia, malignancy). In pericardial tamponade the classic Beck's triad of *hypotension* (due to a decrease in stroke volume), *distension of the jugular veins* (due to the impaired venous return) and *muffled heart sounds* (due to accumulation of fluid in the pericardium) is usually described. Other signs of tamponade are *pulsus paradoxus* (decrease of arterial blood pressure of at least 10 mmHg during inspiration), *low voltage QRS complex* on the ECG, as well as *general symptoms of shock* (tachycardia, dyspnea, decreased level of consciousness). It should be noted that all of these signs are not always present. It is vital to recognize this condition (clinical picture, echocardiography), and act in an urgent manner (pericardiocentesis, cardiac surgery).

*Tension pneumothorax* is the most severe form of pneumothorax (accumulation of air in the pleural space). It is dangerous because the air on inspiration enters the pleural space, while on the exhalation it does not exit the pleural space. With every breath the amount of air increases rapidly, thereby increasing intrathoracic pressure. Lung tissue collapses, and is shifted towards the hilus, also moving the heart and major blood vessels to the healthy side. Symptoms and signs are rapidly developing and, if not promptly intervened, death occurs in 10-15 minutes. It is most commonly seen after the rupture of emphysematous bullae, caverns, lung abscess or cyst, perforation of the esophagus, after penetrating chest trauma, as a complication of ribs fractures, tracheostomy and mechanical ventilation. There are following symptoms: dyspnea, absent breathing sound during auscultation on the affected side, hyperventilation, hypoxia, sweating and tachycardia, decrease in blood pressure, using accessory respiratory muscles, cough, and fear of death. This condition needs to be quickly identified, because the only effective treatment is emergency needle puncture or chest drainage.

## 1.5. PULMONARY EMBOLISM

It is, by definition, complete or partial obstruction of the pulmonary arterial circulation. It is a very dangerous condition that can lead to sudden death, and acute right heart failure (obstructive shock, cor pulmonale). In most cases the thrombus is coming from deep leg or pelvis veins; this condition is also known as venous thromboembolism (VTE). Less frequently pulmonary embolism may be caused by fatty embolism particles, air or amniotic fluid.

The clinical symptoms are abrupt, the most common being dyspnea and chest pain which is intensified by breathing. Clinical signs may be absent, but the most frequent are tachypnea (rapid breathing), tachycardia, fever and signs of right ventricular dysfunction (engorged neck veins). There could be also coughing of blood (hemoptysis). Patients are usually hypoxemic (lack of oxygen in arterial blood). The cyanosis of the lips and fingers could also be found. The most severe cases are called massive pulmonary embolism, and they are accompanied by hypotension and bradycardia, followed usually by fatal outcome.

Diagnosis is based on clinical symptoms, certain laboratory tests (blood gases, D-dimer) and radiological examination (MSCT angiography of the pulmonary artery). Treatment is based on anticoagulants - heparin, warfarin, and a fibrinolytic in the most severe cases.

Rarely the cardiac surgery intervention for removal of the clot from the pulmonary artery is required.

## 2. RESPIRATORY FAILURE

### 2.1. INTRODUCTORY REMARKS, DEFINITION

The main role of breathing is to maintain normal partial pressures of oxygen and carbon dioxide, as well as pH in arterial blood. Therefore, respiratory failure, by definition, is inadequate gas exchange by the respiratory system, i.e. $PaO_2$ and $PaCO_2$ can not be maintained in the normal range. Respiratory failure can also be defined as a clinical syndrome in which the respiratory system fails in one or both of its essential functions of gas exchange: oxygenation and/or elimination of $CO_2$. It can be caused not only by a lung disease, but also by a heart disease, respiratory muscles weakness, chest deformities, and the loss of central control of breathing (events in the brain).

### 2.2. CLASSIFICATION

Respiratory failure is classically divided into two main types (type I and type II), even though in recent years four types of respiratory failure have been increasingly cited in the literature.

#### *2.2.1. TYPE I RESPIRATORY FAILURE*

Type I is also called *hypoxemic respiratory failure*; the main determinant is, of course, hypoxemia ($PaO_2$ <60 mm Hg) with a normal (or even low) $PaCO_2$. It is the most common disorder, and occurs due to the collapse of the alveoli or their filling with fluid. There is also a V/Q ratio disorder (V = minute alveolar ventilation, Q = cardiac output passing through the pulmonary artery, the normal ratio is about 0.8-1). If alveolar ventilation is insufficient (or even zero as in lung atelectasis), there is a rapid decline in V/Q ratio, so-called "shunt" (i.e. blood passing through the lungs is not oxygenated).

Clinical examples of type I respiratory failures include: cardiac or non-cardiac pulmonary edema, pneumonia, atelectasis, aspiration of gastric contents, pulmonary fibrosis.

The clinical signs of hypoxemia in the type I respiratory failure include: anxiety, tachycardia, tachypnea, circulatory instability, sweating, disorders of consciousness, convulsions, and cyanosis (blue skin and mucosa caused by increased concentration > 50 g/L of reduced hemoglobin)

With regard to type I respiratory failure it is necessary to mention the clinical conditions ALI and ARDS. Abbreviation ALI stands for the Acute Lung Injury and indicates less severe form of ARDS and usually precedes ARDS (Acute Respiratory Distress Syndrome), which is a life threatening condition, requiring admission to the ICU and mechanical ventilation. It is a clinical syndrome of acute onset, manifested by tachypnea, hypoxemia and severe loss of pulmonary compliance, and is caused by various medical conditions. There are diffuse alveolar infiltrates on chest X-ray not associated with heart failure. ARDS is, in fact, a very serious reaction to various forms of direct and indirect lung injury (*Table 3*). ARDS is very common in severe sepsis, and one of the principal components of MODS (multiple organ dysunction syndrome), having a high mortality rate of about 40%.

Table 3. The most frequent causes of acute respiratory distress syndrome

| | **DIRECT LUNG INJURY** | **INDIRECT LUNG INJURY** |
|---|---|---|
| **FREQUENT CAUSES** | • **Pneumonia**<br>• **Aspiration of gastric contents** | • **Sepsis**<br>• **Polytrauma associated with shock and massive transfusion** |
| **LESS COMMON CAUSES** | • Pulmonary contusion<br>• Fat embolism<br>• Drowning<br>• Inhalation injury<br>•Reperfusion injury after pulmonary embolism | • Cardiopulmonary «bypass»<br>• Overdosage of drugs<br>• Acute pancreatitis<br>• Allogenic blood transfusion |

*2.2.2. TYPE II RESPIRATORY FAILURE*

Type II is also called *hypercapnic respiratory failure*, and results in an increase of $CO_2$ in arterial blood (normal values are 35-45 mmHg). The causes of type II respiratory failure are drug overdose, neuromuscular diseases, chest abnormalities, chronic obstructive pulmonary disease. It occurs due to inadequate ventilation, while oxygenation is preserved.

Clinical signs are disturbances of consciousness (ranging from somnolence to coma - the so-called CO2 narcosis), pulmonary hypertension, tachycardia, arrhythmias, tremor, headache.

*2.2.3. OTHER TYPES OF RESPIRATORY FAILURE*

During surgery under general anesthesia there is an increased tendency toward lung atelectasis. It is a principal determinant of *type III respiratory failure* related to anesthesia and surgery (also called *perioperative respiratory failure*). It is manifested clinically either as type I or type II respiratory failure. The clinical outcome in this type of respiratory failure can be improved by the correct method of anesthesia, the correct positioning of patients, effective postoperative analgesia, physical therapy, and a reduction of intra-abdominal pressure.

*Type IV respiratory failure* refers to a group of *patients in shock*, endotracheally intubated and mechanically ventilated in the resuscitation process. The goal of mechanical ventilation here is to improve gas exchange and reduce the load (and oxygen consumption) of respiratory muscles.

*2.2.4. RESPIRATORY FAILURE - SUMMARY*

The overview of the types of respiratory failure along with the proposed mechanisms and clinical examples is shown in Table 4.

Table 4. The overview of the types of respiratory failure

| **TYPE** | **I** | **II** | **III** | **IV** |
|---|---|---|---|---|
| **Name** | Acute hypoxemic failure | Ventilatory failure | Perioperative failure | Shock |
| **Mechanism** | V/Q ratio disorder | Inadequate ventilation | Atelectasis | ⇓perfusion of respiratory muscles |
| **Clinical example** | •Cardiogenic pulmonary edema<br>•ARDS<br>•pneumonia | •ALS<br>•Status asthmaticus<br>•Opioid overdose | •Postoperative respiratory failure | •Sepsis<br>•Hypovolemia<br>•Cardiogenic shock |

ARDS = Acute respiratory distress syndrome, ALS = amyotrophic lateral sclerosis

## 2.3. BRIEF OVERVIEW OF RESPIRATORY FAILURE TREATMNET

The principal objective of respiratory failure treatment is to find and treat the cause, as well as to provide useful supporting measures:

- increasing the concentration of inhaled oxygen - FiO2,
- establishing an artificial airway,
- mechanical ventilation.

Giving higher concentrations of oxygen is necessary in most cases. In some cases alveolar ventilation can significantly be improved by establishing adequate and effective airway, as well as by aspiration of content from airway (sputum, blood, secretions), and endotracheal intubation. Mechanical ventilation shall be required in most seriously ill patients.

### *2.3.1. OXYGEN ADMINISTRATION*

In many cases of respiratory failure the oxygen in concentrations greater than atmospheric ($FiO_2$ = 0.21 or 21%) should be delivered to patients breathing spontaneously. This can be achieved in several ways:

a) through nasal catheter (cannula) - oxygen flow of 1-6 l/min, resulting in a $FiO_2$ 0.24 to 0.44 (any increase in flow rate of 1 liter/minute causes increase in $FiO_2$ of about 0.04)

b) by a simple mask - flow rates 6 l/min and more required, resulting in a $FiO_2$ 0.5-0.6

c) through a mask with a reservoir ("non-rebreathing") - this mask has an attached reservoir for oxygen and special valves that prevent the entry of atmospheric air. With a flow of 6-10 l/min, and good adhesion to the face of the patient, $FiO_2$ of 1 (100%) may be achieved in some spontaneously breathing patients

d) through a Venturi mask – these bring very precise oxygen concentration for a particular patient. Oxygen at high flow rates enters through a narrow opening which has the side

entrance for atmospheric air. By changing the oxygen flow and the size of inlet for atmospheric air one could achieve precise $FiO_2$ of 0.24, 0.28, 0.31, 0.35, 0.40 and 0.50 (each different concentration is being launched with a mask and connector in a different color).
e) A patient who is intubated and mechanically ventilated gets a very precise inhaled oxygen concentration, by manipulating the $FiO_2$ settings of the mechanical ventilator; it can vary from 0.21 to 1.0.

### *2.3.2. ENDOTRACHEAL INTUBATION*

Endotracheal intubation is the procedure in which a hollow tube is put into the trachea used using a laryngoscope. It is a "gold standard" for securing the airway and has a great advantage over all other methods. If the cuff of the endotracheal tube is properly inflated, there is an absolute protection against aspiration of the gastric contents into the lungs. Besides, there is no loss of gas during ventilation of patients. Depending on the way of passage the tube may be orotracheal (on the way to the trachea the tube passes through the oral cavity) and nasotracheal (on the way to the trachea the tube passes through the nasal cavity). It is worth mentioning that an awake patient usually can not withstand inserted endotracheal tube without pain and/or coughing; therefore the patients who are intubated are usually given sedatives and/or analgesics.

Indications for endotracheal intubation may be physiological and clinical. Physiological indications are: 1) Hypoxemia after administration of $O_2$, 2) $PaCO_2 > 55$ mmHg + pH <7.25, 3) vital capacity <15 ml/kg without neuromuscular disease. Clinical indications are: 1) changes in the level of consciousness with inability to protect airway, 2) respiratory failure with hemodynamic instability, 3) airway obstruction, 4) a large amount of secretions, which can not be expectorated by the patient

Moreover, almost all patients who need to be mechanically ventilated, must first be endotracheally intubated (it is possible to mechanically ventilate only a very small number of patients without endotracheal intubation; the so-called noninvasive ventilation).

Tracheostomy is an opening in the neck resulting from tracheotomy, a procedure which approaches the trachea from the anterior aspect of the neck, either percutaneously, either surgically (hence it is called percutaneous and surgical tracheostomy). High-quality airway could be inserted through tracheostomy (tracheal cannula); it is possible for patient to breathe spontaneously with tracheal cannula as well as to be mechanically ventilated. Tracheostomy is usually performed after several days of endotracheal intubation in patients who can not be weaned from the mechanical ventilation.

Supraglottic airways (tip positioned above the entrance to the larynx and trachea) include laryngeal mask airway (LMA) and perilaryngeal airways (Cobra, i-Gel). They are nowadays often used in general anesthesia for elective patients and sometimes in resuscitation, but only as a temporary measure, since they do not prevent against very dangerous aspiration of gastric contents.

### *2.3.3. MECHANICAL VENTILATION*

Mechanical ventilation is a method which helps or replaces spontaneous breathing. It is the most common way to support vital functions in the operating room and ICU. The main indication is, of course, respiratory failure. Other indications are: cardiopulmonary resuscitation, severe sepsis, increased intracranial pressure, and postoperative period in certain patients; those with high fluid requirements and unstable vital signs, polytrauma patients with unstable fractures, patients with "open" abdomen, closed head injury, and severe burns.

The goal of mechanical ventilation is to achieve and maintain an adequate pulmonary gas exchange, thereby reducing the risk of lung injury and the work of breathing, as well as improving patient comfort.

Today, the mechanical ventilators deliver ventilation to patients using intermittent positive pressure relative to atmospheric (IPPV - intermittent positive pressure ventilation).

The mechanical ventilatory support can be a full and partial one. With full support, also called controlled mechanical ventilation (CMV), the whole respiratory effect is done by the machine, and respiration can be controlled in volume or pressure mode (physician adjusts the machine for the desired tidal volume or peak inspiratory pressure level for a particular patient). In partial support mode, there is an increasing proportion of patient’s breathing. Special sensors sense the patient's respiratory effort and the machine helps with synchronization and pressure support. Partial support modes include SIMV (synchronized intermittent mandatory ventilation) mode, BiPAP (Bi-level Positive Airway Pressure) mode, and finally the CPAP (Continuous Positive Airway Pressure) mode, in which the patient begins each breath and the machine provides only pressure support. These modes are used during gradual weaning from the mechanical ventilation.

The mechanical ventilation is an indispensable method of modern medical practice and saves lives of many critically ill patients. However, it may also have its complications. Hemodynamic instability is possible in some cases, since the mechanical ventilation creates a positive pressure in the chest, leading to a reduction in venous return and cardiac output. Furthermore, patient’s lungs might be exposed to possible barotrauma (hyperdistension of alveoli due to high inflating pressures), and volutrauma (hyperdistension of alveoli due to high inflating volumes), leading to alveolar rupture, pneumothorax and subcutaneous emphysema in the most severe cases. Moreover, patients on mechanical ventilation may have a high incidence of nosocomial infections, so called VAP (Ventilator-associated pneumonia).

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# 8. DISTURBANCES OF CONSCIOUSNESS

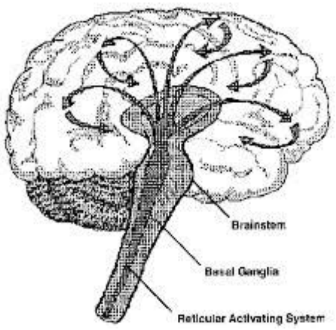
Branka Polić, M.D.

An alert patient has a normal state of arousal. An alteration in arousal represents an acute, life threatening emergency, requiring prompt intervention for preservation of life and brain function.

Degrees of decreased consciousness level are: somnolence, sopor, coma. Somnolence is a clinical state of deeper sleepiness but the patient can wake up on call or stimuli. Sopor is a clinical state in which patients have impaired responsiveness to external stimulation. Patient may frown or move the shoulder and released incomprehensible sounds. Coma is defined as "unarousable unresponsiveness".

**Etiologies** - The ascending reticular activating system (ARAS) is a network of neurons originating in the tegmentum of the upper pons and midbrain, believed to be integral to inducing and maintaining alertness. These neurons project to structures in the diencephalon, including the thalamus and hypothalamus, and from there to the cerebral cortex (piture 1).

Picture 1 ARAS




Alterations in alertness can be produced by focal lesions within the upper brainstem by directly damaging the ARAS. Injury to the cerebral hemispheres can also produce coma, but in this case, the involvement is necessarily bilateral and diffuse, or if unilateral, large enough to exert remote effects on the contralateral hemisphere or brainstem. Most cases of sopor and coma presenting to an emergency department are due to: trauma, cerebrovascular disease, hypoxia, intoxications, infections, metabolic derangements, postictal state after an epileptic seizure, hypothermia, drugs, intracerebral bleeding and tumors. These conditions impair oxygen or substrate delivery, which in turn alters cerebral metabolism or interferes with neuronal excitability and/or synaptic function.

Neurological examination — The neurological examination in a comatose patient is necessarily brief and is directed at determining whether the pathology is structural or due to metabolic dysfunction (including drug effects and infection). The examiner assesses:

- Level of consciousness
- Motor responses
- Brainstem reflexes: pupillary light, extraocular, and corneal reflexes

Important findings are abnormal reflexes that indicate dysfunction in specific regions of the brainstem, or a consistent asymmetry between right- and left-sided responses.

**Level of consciousness** — Arousability is assessed by noise (e.g. shouting in the ear) and somatosensory stimulation. Pressing on the supraorbital nerve (medial aspect of the supraorbital ridge) or the angle of the jaw, or squeezing the trapezius, may have a higher yield than the more commonly used sternal rub. Important responses include vocalization, eye opening, and limb movement. Rapid assessment of consciousness level includes AVPU scale: **A**-**A**lert; **V**- responds to **V**oice; **P**-responds only to **P**ain; **U**- **U**nresponsive to all stimuli. The Glasgow coma scale (GCS) demonstrates a hierarchy of responses which reflect the severity of the coma. The GCS is useful as an index of the depth of impaired consciousness and for prognosis (table 1).

Table 1

| **Glasgow coma scale** | **Score** |
|---|---|
| **Eye opening** | |
| Spontaneous | **4** |
| Response to verbal command | **3** |
| Response to pain | **2** |
| No eye opening | **1** |
| **Best verbal response** | |
| Oriented | **5** |
| Confused | **4** |
| Inappropriate words | **3** |
| Incomprehensible sounds | **2** |
| No verbal response | **1** |
| **Beast motor response** | |
| Obeys commands | **6** |

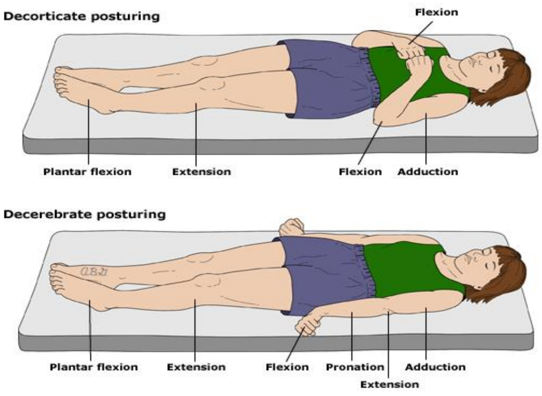
| | |
|---|---|
| Localizing response to pain | **5** |
| Withdrawal response to pain | **4** |
| Flexion to pain | **3** |
| Extension to pain | **2** |
| No motor response | **1** |
| **Total** | |

**Motor examination** — It is important to assess muscle tone, as well as spontaneous and elicited movements and reflexes. Asymmetries of these often indicate a hemiplegia of the non-moving side, implying a lesion affecting the opposite cerebral hemisphere or upper brainstem.

Purposeful movements include crossing the midline, approaching the stimulus, pushing the examiner's hand away or actively withdrawing from the stimulus. In addition to decreased spontaneous or purposeful movement, acute structural disease usually produces decreased muscle tone or flaccidity. Abnormal postures include:

- ●Decorticate posturing consists of upper-extremity adduction and flexion at the elbows, wrists, and fingers, together with lower-extremity extension, which includes extension and adduction at the hip, extension at the knee, and plantar flexion and inversion at the ankle. This occurs with dysfunction at the cerebral cortical level or below.
- ●Decerebrate posturing consists of upper-extremity extension, adduction, and pronation together with lower-extremity extension (picture 2) and implies dysfunction below the red nucleus, allowing the vestibulospinal tract to predominate.

Picture 2 Decorticate/decerebrate postures




**Cranial nerves** — The fundi should be carefully inspected. The most important cranial nerve reflexes with respect to coma are: pupillary, corneal, and the vestibuloocular reflex.

**Pupils** — The pupillary light reflex is tested in each eye individually to evaluate direct and consensual responses. Disruption of the pupillary light reflex in comatose patients usually occurs because of either:

- ●Downward herniation of mesial temporal structures from an expanding supratentorial mass and/or a lateral shift in the supratentorial compartment with stretching of the oculomotor nerve against the clivus or
- ●Primary brainstem lesions

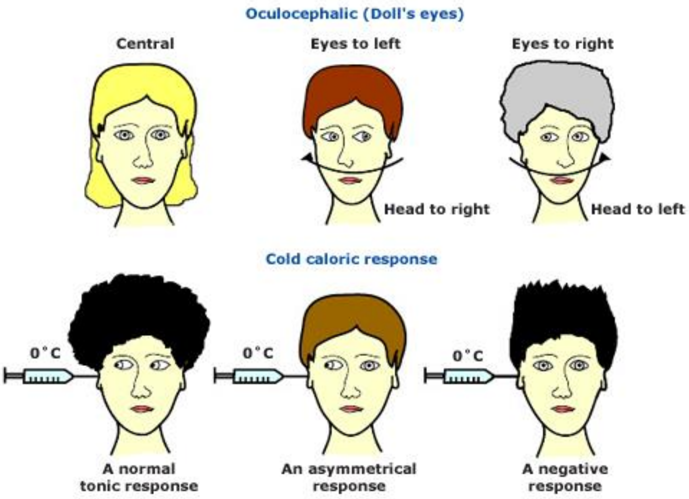
In either of these, the third cranial nerves or their nuclei in the midbrain are injured, producing a unilateral or bilateral oculomotor palsy. When unilateral, the ipsilateral pupil is dilated and unreactive directly and consensually, but the contralateral pupil reacts to light shone in either eye (picture 3). When bilateral, there is neither a direct nor consensual response, the pupils are symmetrically enlarged, and the eyes are deviated outward.

Picture 3 Left pupil is dilated and unreactive


**Eye movements** — Horizontal eye movements can be tested with two vestibuloocular reflexes:

- ●In the oculocephalic maneuver (or doll's eyes), the head is abruptly rotated from one side to the other in the horizontal plane. When the oculocephalic reflex is present (positive doll's eyes), the eyes do not turn with the head, but in the opposite direction, as if the patient is maintaining visual fixation on a single point in space. **The cervical spine must be cleared of fracture in any patient with suspected head trauma before this is performed**. This reflex is usually suppressed (and therefore not tested) in conscious patients (figure 3).
- ●Caloric testing of the oculovestibular reflex provides a stronger stimulus for reflex eye movements. In this test, the head or upper torso is inclined 30 degrees up from the horizontal. After inspecting the ears for obstruction from wax or a perforated drum, at least 50 mL of ice water is injected into the ear canal using a syringe with a small catheter attached. This stimulus has the same effect on the horizontal semicircular canal as sustained turning of the head in the opposite direction, and results in sustained deviation of both eyes toward the ear being stimulated. Five minutes should elapse before testing the other side (picture 4).

Picture 4 Oculocephalic and caloric response



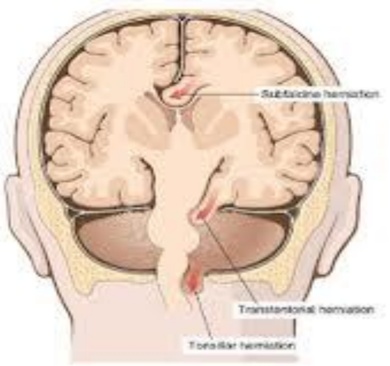

A cold caloric response is also present in conscious people, producing not only deviation of the eyes toward the stimulated ear, but also nystagmus (with the fast phase away from the irrigated side), severe vertigo, nausea, and vomiting. If nystagmus occurs, the patient is awake and not truly in coma.

**Corneal reflex** — The corneal reflex is tested by gently touching the edge of the cornea with a rolled tissue or cotton swab and observing the responsive blink. The reflex can be suppressed acutely contralateral to a large, acute cerebral lesion, and also with intrinsic brainstem lesions. Loss of the corneal reflex is also an index of the depth of metabolic or toxic coma.

**Herniation syndromes** — Transtentorial herniation can occur with expanding mass lesions. The initial impairment of consciousness with supratentorial mass lesions usually relates to lateral rather than downward displacement. Other signs of increased intracranial pressure, papilledema, and Cushing's triad (hypertension, bradycardia, irregular respiration) may be observed in this setting.

Two variants are recognized: a central herniation and an uncal herniation syndrome. In the latter, more laterally directed compressive forces lead to asymmetric herniation of the temporal uncus. An ipsilateral third cranial nerve palsy (pupillary dilation, downward and outward eye deviation) can occur prior to diencephalic signs as the nerve is displaced and stretched over the clivus. Loss of reactivity of the contralateral pupil usually reflects midbrain damage. Hemiplegia due to compression of the cortical spinal tract in the midbrain often follows immediately. The syndrome then follows the sequence of central herniation (picture 5).

Picture 5 Herniation of the brain




DIAGNOSIS — The goal of diagnostic testing in a patient in coma is to identify treatable conditions (infection, metabolic abnormalities, seizures, intoxications/overdose, surgical lesions). Because neurologic recovery is often reliant on early treatment, testing must proceed rapidly in concert with the clinical evaluation. Investigations almost always include laboratory testing, lumbar puncture, electroencephalography (EEG) and neuroimaging (CT, MRI). Testing should be prioritized according to the clinical presentation.

**Laboratory tests** — Screening laboratory tests for patients presenting in coma of uncertain cause include:

- ●Complete blood count
- ●Serum glucose, electrolytes, urea, creatinine, liver function tests, lactate, amonia and osmolarity
- ●Arterial blood gas
- ●Prothrombin and partial thromboplastin time
- ●Drug screen (usually done on urine and serum), including ethyl alcohol, opiates, benzodiazepines, barbiturates, salicylates, cocaine, amphetamines, ethylene glycol, and methanol

MANAGEMENT — In the emergency department, basic care should be done in concert with the clinical and laboratory investigations. The primacy of ABCs (airway, breathing, and circulation) applies to cases of coma. Vital signs should be taken, an initial Glasgow Coma Scale score (GCS) established, and a set of arterial blood gases, along with the other blood and urine tests sent to the laboratory. If a herniation syndrome is evident clinically or appears imminent based on computed tomography (CT) findings, urgent treatment is recommended. This includes administration of mannitol, a 20-30˚ head-up position with the head in-line, mild hyperventilation and 3% (hypertonic) saline. Definitive therapy depends on establishing the precise diagnosis. In presence of fever and infection antibiotics or/and antiviral drugs are given. Monitoring of the course of the patient and looking for improvement, worsening, and complications follow, along with establishing a prognosis.

PROGNOSIS — Coma is a transitional state which means that patients either recover or evolve into brain death or a persistent vegetative or minimally conscious state. The prognosis depends on the underlying etiology, as well as the severity of the insult and other premorbid factors, including age.

# 9. BASIC LIFE SUPPORT IN CHILDREN

Branka Polić, M.D.

Basic life support (BLS) involves a systematic approach to initial patient assessment, the initiation of cardiopulmonary resuscitation (CPR) and activation of emergency medical services. Key components of effective CPR include adequate ventilation and chest compressions. BLS can be performed by trained lay persons, as well as by healthcare providers. Cardiopulmonary arrest among infants and children is typically caused by hypoxia and acidosis as the result of respiratory and/or circulatory failure. This is in contrast to adults, for whom the most common cause of cardiac arrest is ischemic cardiovascular disease.

**THE 2010 INTERNATIONAL RESUSCITATION GUIDELINES** — The American Heart Association (AHA) and the International Liaison Committee on Resuscitation (ILCOR) published updated guidelines for pediatric basic life support (BLS) in 2010. For the purposes of these guidelines, a newborn is defined as from birth to one month, an infant is younger than one year of age, and a child is from one year to the puberty. The guidelines are designed to be simple, practical, and effective.

**BASIC LIFE SUPPORT SEQUENCE**

**Initial approach** – **SSS** approach.

**S**AFETY: Approach with care and free from danger. The rescuer must ensure that the scene is safe for them and the victim. The rescuer does not become a second victim and the child is removed from danger.

**S**TIMULATE: Gently apply a stimulus. Holding the head and shaking the arm, ask the child: Are you all right? The child makes a sound or opens the eyes or is unresponsive (picture 1).

Picture 1


Stimulating the child

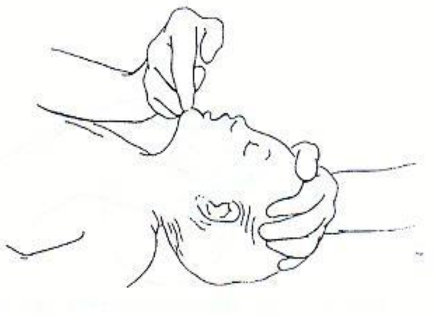
**S**HOUT: Shout for help if the child is unresponsive.

**Initiate CPR**—The actions that constitute cardiopulmonary resuscitation (CPR) are opening the airway, providing ventilations (rescue breaths), and performing chest compression. The chin-lift and/or jaw-thrust maneuvers should be performed to open the airway in an unresponsive child. High quality CPR focuses on the effective delivery of chest compressions and avoidance of excessive ventilation.

**AIRWAY OPENING MANOEUVRES**

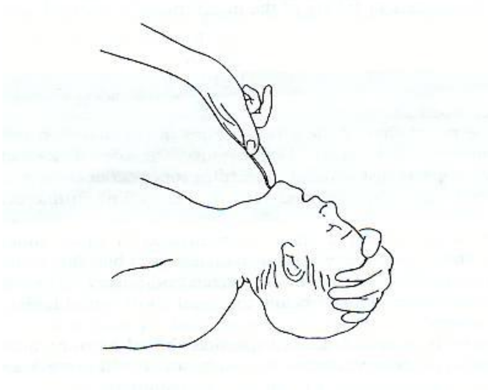
**Head-tilt-chin lift**—In this technique, one hand may be placed on the child's forehead to gently tilt the head back, and the fingers of other hand are placed under the mandible, which is gently lifted upward to move the chin anteriorly. The thumb of the same hand lightly depresses the lower lip to open the mouth. During the chin lift procedure, care must be taken to avoid closing the mouth, pushing on the soft tissues under the chin, or hyperextending the neck since these actions contribute to airway obstruction. The position of the head in infant is neutral (picture 2) and in children is sniffing (picture 3).

Picture 2



Infant – neutral position of the head

Picture 3

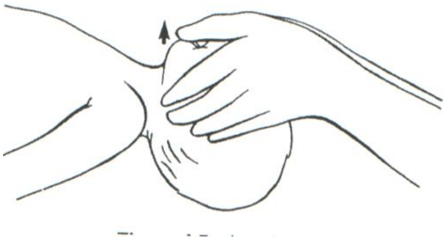


Children – sniffing position of the head

The head-tilt-chin-lift maneuver should **not** be used in children who are suspected of having head or neck injuries.

**Jaw thrust** — The jaw thrust is the preferred method for opening the airway when trauma is suspected, in which case, cervical spine immobilization should also be maintained. The jaw thrust maneuver is performed by grasping the angles of the lower jaw with one hand on each side, and moving the mandible forward so that the lower central incisors are anterior to the upper central incisors (picture 4).

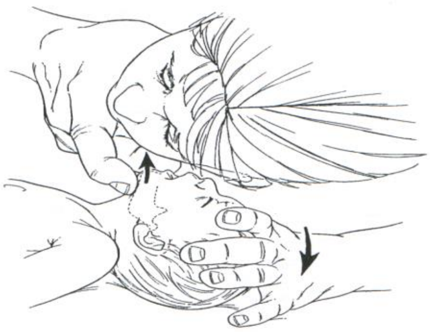
Picture 4



Jaw thrust

**The patency of airway** should be assessed by placing a face above the child's with ear over the nose, the cheek over the mouth and the eyes looking along the line of the chest for up to 10 sec. **LOOK** for chest movements; **LISTEN** for breath sounds and **FEEL** for exhaled breath (picture 5).

Picture 5



Look, listen, feel

**VENTILATION** — If the child is not breathing, give 5 rescue breaths. Ventilation can be provided with mouth-to-mouth, mouth-to-nose, or with mouth-to-mouth and nose. Each rescue breath should be delivered over one to two seconds. The volume of each breath should be sufficient to see the chest wall rise. For an INFANT rescuer seals a mouth around the victim's mouth and nose (picture 6). For a CHILD rescuer seals a mouth around the victim's mouth with nose pinched closed using the thumb and index fingers of the hand that is maintaining the head tilt (picture 7).

Picture 6

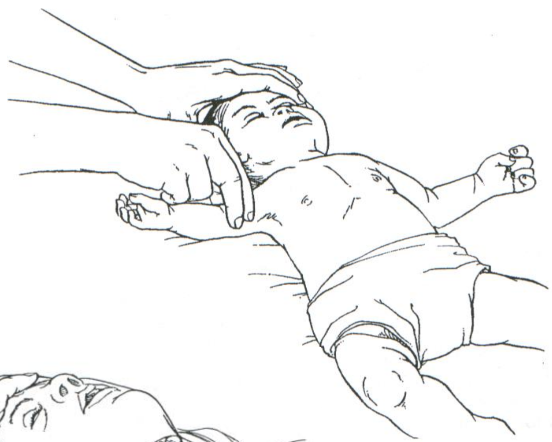
INFANT - mouth-to-mouth and nose ventilation

Picture 7

CHILDREN - mouth-to-mouth ventilation

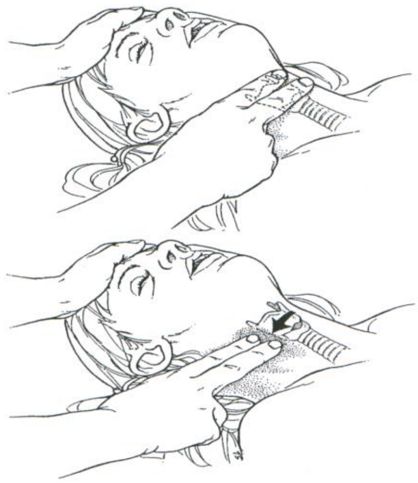
**CIRCULATION --** Inadequacy of the circulation is recognized by the absence of signs of life, the absence of a central pulse for up to 10 seconds or the presence of a pulse at an insufficient rate. Even experienced health professionals can find it difficult to be certain that the pulse is absent within 10 seconds. Therefore, the absence of 'signs of life' is the primary indication to start chest compressions. Signs of life include: movement, coughing or normal breathing**.** In infants the brachial artery in the medial aspect of the antecubital fossa or the femoral artery in the groin can be palpated (picture 8). In the children the carotid artery in the neck, lateral to the trachea, can be palpated (picture 9).

Picture 8



INFANTS- palpating the femoral and

Picture 9



Children - palpating the carotid artery

If the pulse is absent for up to 10 seconds or is inadequate - less than 60 beats per minute - with signs of poor perfusion then cardiac compression is required. Signs of poor perfusion include pallor, lack of responsiveness and poor muscle tone.

**CHEST COMPRESSIONS**—The 2010 international resuscitation guidelines emphasize the importance of hard, fast chest compression, with full chest recoil and minimal interruptions. Evidence in adults and animals suggest that these are the essential elements for effective chest compressions.

Chest compressions should be performed over the lower half of the sternum. Compression of the xiphoid process can cause trauma to the liver, spleen, or stomach, and must be avoided. The chest should be depressed at least one-third of its anterior-posterior diameter with each compression, approximately 4 cm in most infants and 5 cm in most children. The optimum rate of compressions is approximately 100-120 per minute. Each compression and decompression phase should be of equal duration. The sternum should return briefly to its normal position at the end of each compression, allowing the chest to recoil fully. A smooth compression-decompression rhythm, with minimal interruption, should be developed.

**Infants** — Chest compressions for infants (younger than one year) may be performed with either two fingers or with the two-thumb encircling hands technique.

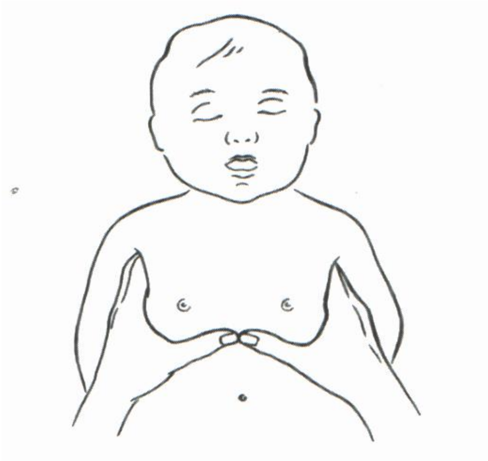
**Two-finger** — This technique is recommended by the American Heart Association when there is a single rescuer. Compressions are performed with index and middle fingers, placed on the lower half of sternum, just below the nipples. Because of the infant's large occiput, slight neck extension and the placement of a hand or rolled towel beneath the upper thorax and shoulders may be necessary to ensure that the work of compression is focused on the heart (picture 10).

Picture 10

Chest compression – two-finger technique

**Two-thumb encircling hands** — The two thumb-encircling hands technique is suggested when there are two rescuers. The thorax is encircled with both hands and cardiac compressions are performed with the thumbs. The thumbs compress over the lower half of the sternum, just below the nipples, avoiding the xiphoid process, while the fingers are spread around the thorax (picture 11).

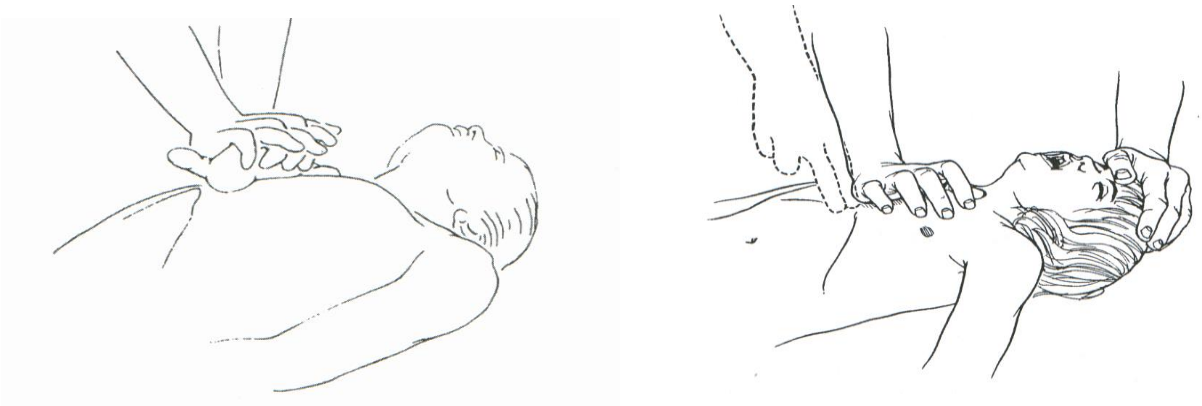
Picture 11



Two-thumb encircling hands technique

**Children** — For children (from one year until the puberty), compressions should be performed over the lower half of the sternum with either the heel of one hand or with two hands, as for adult victims (picture 12).

Picture 12



Compression with one or two hands

**COMPRESSION TO VENTILATION RATIO** — Every effort should be made to avoid excessive ventilation and to limit interruptions of chest compressions to less than ten seconds. For health care providers, two ventilations should be delivered at the end of every 15th compression. For lay persons two ventilations should be delivered at the end of every 30th compression.

Once the trachea is intubated, ventilation and compression can be performed independently. Ventilations are given at a rate of 8 to 10 per minute. Compressions are delivered at a rate of 100-120 per minute without pauses.

Experimental work has shown that coronary perfusion pressure increases if sequences of compression are prolonged rather than curtailed. Do not interrupt chest compressions for more than 10 seconds except for defibrillation or/and for emergency call. Compressions can be recommenced at the end of inspiration and may augment exhalation.

**Conventional versus compression-only CPR** — In some cases if rescuer is unwilling/unable to ventilate, use compressions-only CPR.

After **1 minute of CPR** the rescuer must call emergency services. In the case of a baby or a small child the rescuer will be able to take the victim to a telephone and continue CPR on the way.

## BLS

S.S.S.

⇓

Airway opening maneuvers

⇓

Look, listen, feel

⇓

5 rescue breaths

⇓

Check for signs of life

Check pulse (*Take no more than 10 seconds)*

⇓

CPR

15 (30) chest compressions : 2 ventilations

*(1 minute)*

⇓

Call emergency services

# 10. NEWBORN RESUSCITATION

**Branka Polić, M.D.**

The successful transition from intrauterine to extrauterine life is dependent upon significant physiological changes that occur at birth. In almost all neonates (90 percent), these changes are successfully completed at delivery without requiring any special assistance. However, about 10 percent of neonates will need some intervention, and 1 percent will require extensive resuscitative measures at birth.

The 2010 AHA/AAP/ILCOR guidelines recommend the following approach.

- Initial steps (provide warmth, clear **A**irway if necessary, dry, and stimulate)
- **B**reathing (ventilation)
- **C**hest compressions
- **D**rugs administration, such as epinephrine and/or volume expansion

A time allocation of 30 seconds is given to apply the resuscitative procedure, evaluate, and decide whether to proceed to the next intervention. Monitoring of oxygen saturation by using pulse oximetry should be performed in neonates who are apneic, gasping, have labored breathing, have persistent cyanosis, or have a heart rate less than 100 beats per minute (bpm). No further resuscitative actions are required if the baby responds with adequate spontaneous respirations and a heart rate above 100 beats per minute.

**Provide warmth**

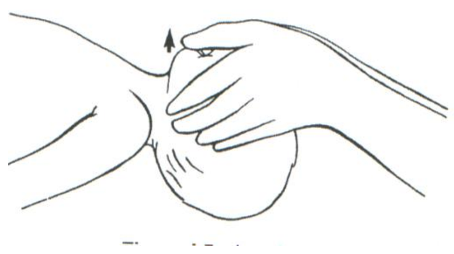
To minimize heat loss, the delivered baby is first placed in a warmed towel or blanket and then under a prewarmed radiant heat source, where he/she is dried with another warmed towel or blanket. The servo–controlled temperature of the warmer is set to maintain the neonate's temperature at 36.5ºC, which is monitored by a temperature skin probe placed upon the neonate's abdomen.

**Airway** — The baby is positioned to open the airway by placing on his/her back on a flat radiant warmer bed with the neck in a neutral position; the neck should not be hyperextended or flexed. The proper position aligns the posterior pharynx, larynx, and trachea, and facilitates air entry. A rolled blanket or towel may be placed under the baby's shoulder to slightly extend the neck to maintain an open airway (picture 1). Another method is jaw thrust (picture 2).

Picture 1

Neutral position of a head

Picture 2



Jaw thrust

Suctioning immediately after birth is reserved only for babies with obvious obstruction due to secretions or depressed vital signs and who require positive pressure ventilation. Once the neonate has been correctly positioned, the mouth and nose should be suctioned either with a bulb syringe or mechanical suction device. The mouth is suctioned first and then the nares to decrease the risk for aspiration. In the presence of meconium-stained amniotic fluid and non-vigorous babies it is necessary to perform endotracheal suctioning.

**Supplemental oxygen -** Resuscitation should be initiated with blended oxygen. If blended oxygen is not available, room air should be used.

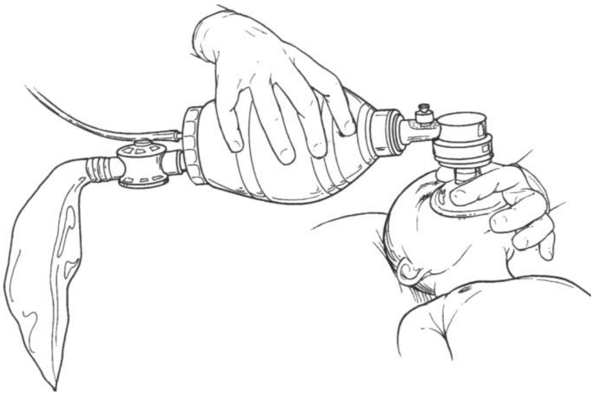
- The oxygen concentration should be adjusted to achieve targeted $SpO_2$ levels, which are monitored by pulse oximetry.
- If the heart rate is below 60 bpm after 90 seconds of resuscitation, the oxygen concentration should be increased to 100 percent until recovery of a normal heart rate.

**Breathing** – If the baby is not breathing, give five initial breaths and look for chest movements.

The initial administered breaths often require pressures of 30 to 40 cm $H_2O$ to inflate the lungs of the newly-born term neonate. In most preterm neonates, an initial inflation pressure of 20 to 25 cm $H_2O$ is usually adequate. Adequacy of ventilation is demonstrated by improvement in heart rate. Chest wall movement should be assessed if heart rate does not improve. The neonate should be ventilated at a rate of 40 to 60 times per minute to achieve a heart rate >100 bpm.

Ventilation is achieved with a bag and mask and addition of oxygen if it is necessary. An airtight seal between the rim of the mask and the face is essential to achieve the positive pressure required to inflate the lungs. An appropriately sized mask is selected and positioned to cover the chin, mouth, and nose, but not the eyes of the infant. The mask is held on the face by positioning the hand of the clinician so that the little, ring, and middle fingers are spread over the mandible in the configuration of the letter "E" and the thumb and index are placed over the mask in the shape of the letter "C". The ring and fifth fingers lift the chin forward to maintain a patent airway. An airtight seal is formed by using light downward pressure on the rim of the mask and gently squeezing the mandible up towards the mask. With the other hand squeeze the bag in order to achieve ventilation (picture 3).

Picture 3



Bag-mask ventilation

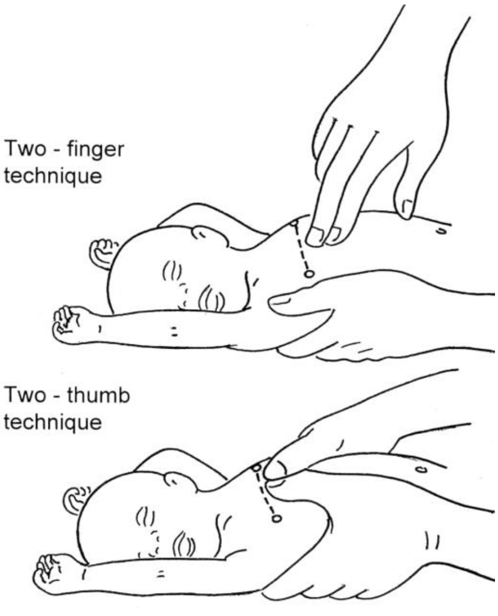
**Chest compressions** — Chest compressions are initiated if the neonate's heart rate remains <60 beats per minute despite adequate ventilation for 30 seconds. In newborns heart rate is not to be palpated but is auscultated with stethoscope at the apex of the heart.

Chest compression applies pressure to the lower half of the sternum, visualized as an imaginary line just below the nipples. Two methods are used to deliver neonatal chest compressions.

●Thumb technique – In this method, both hands encircle the neonate's chest with the thumbs on the sternum and the fingers under the neonate. This is the preferred method.

●Two-finger technique – In this method, the tips of the first two fingers, or the index and middle finger, are placed in a perpendicular position over the sternum (picture 4).

Picture 4




Two chest compression techniques

In both methods, pressure is applied to the chest wall sufficiently to depress the sternum about one-third of the anteroposterior diameter of the chest, and then pressure is released to allow the heart to refill. Care should be taken to avoid applying pressure directly over the xiphoid, as this may cause hepatic injury. The thumb technique is recommended in neonates because it generates higher systolic and coronary perfusion pressure.

Chest compressions must always be accompanied by positive pressure ventilation. During neonatal resuscitation, the chest compression rate is 90 per minute accompanied by 30 ventilations per minute, with one ventilation interposed after every third compression. Thus, the ventilation rate is reduced from the 40 to 60 breaths per minute used in the absence of chest compression to 30 breaths in the presence of chest compression.

After 30 seconds of chest compression and positive pressure ventilation (PPV), reassessment of the neonate's heart rate, color, and respiratory rate should determine whether further interventions are required (intubation or administration of medications).

**Drugs** — Drugs are rarely required in neonatal resuscitation. Delivering adequate ventilation is the most important resuscitative step because the most common cause of bradycardia is inadequate lung inflation or profound hypoxemia. If the heart rate remains <60 beats per minute despite adequate ventilation and chest compressions, administration of epinephrine is indicated.

**Vascular access** — Medications need to be given intravenously. The quickest means of obtaining intravenous access in the newborn is cannulation of the umbilical vein. This is accomplished by aseptically inserting a catheter into the umbilical vein to a depth of four to five cm until there is free flow of blood.

**Epinephrine** — A guidelines recommend intravenously administered epinephrine at a dose of 0.01 to 0.03 mg/kg (0.1 to 0.3 mL/kg of a 1:10,000 solution [concentration 0.1mg/mL]). Higher doses of epinephrine have not been shown to be more effective. Epinephrine may be repeated every three to five minutes if the heart rate remains <60 beats per min.

**Volume expansion** — Hypovolemia may be suspected if there is ante- or intrapartum hemorrhage. The guidelines recommend a 10 mL/kg bolus of normal saline given to correct hypovolemia. This dose can be repeated if necessary based upon the response to the initial bolus. If hemorrhage is present, transfusion of O Rh-negative blood is needed.

**Sodium bicarbonate** — If sodium bicarbonate is used, it should be given only after adequate ventilation and circulation have been established. If it is used, the usual dose is 1 or 2 mEq/kg, given at a rate no faster than 1 mEq/kg per minute.

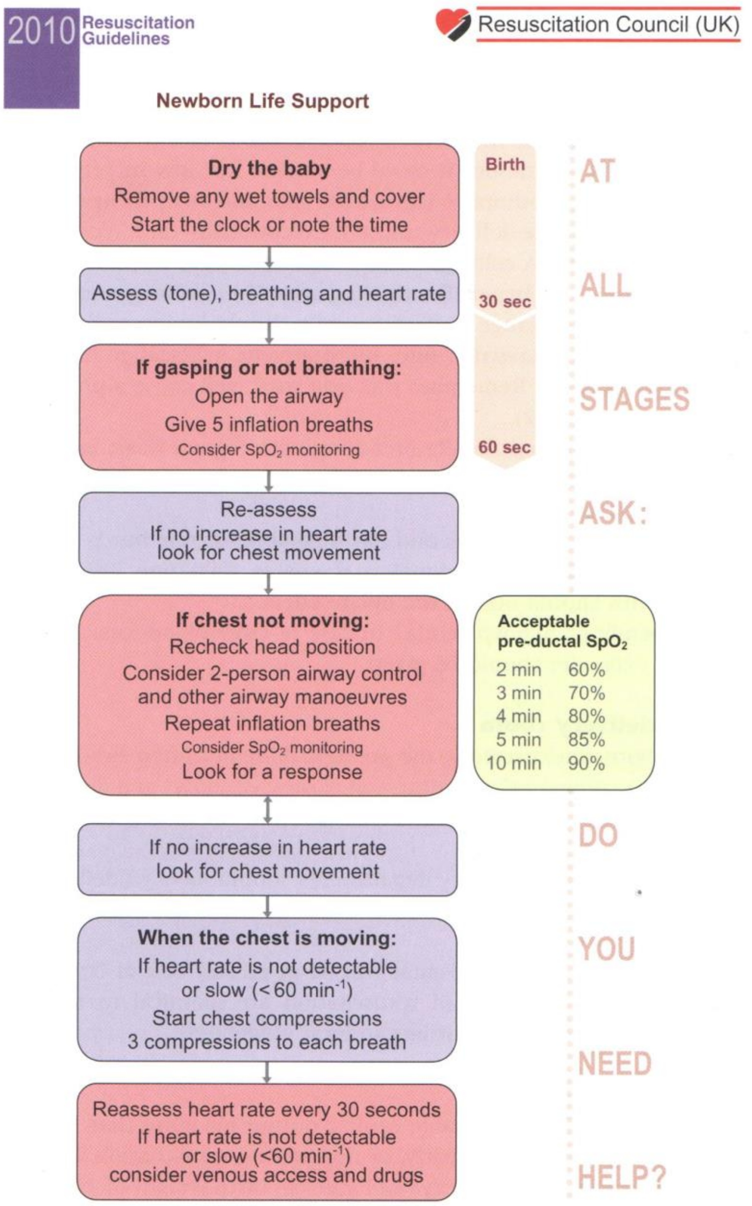



**Figure I.8** Newborn resuscitation algorithm. HR, heart rate. (Reproduced with kind permission from the Resuscitation Council (UK))

# 11. ADVANCED LIFE SUPPORT IN ADULTS

**Mihajlo Lojpur, M.D., Ph.D.**

## INTRODUCTION

**What is cardiac arrest and what are its consequences?**

**Cardiac arrest** (also known as cardiopulmonary arrest or circulatory arrest) is the cessation of normal circulation of the blood due to failure of the heart to contract effectively.

Cardiac arrest or circulatory arrest occurs when the heart is not beating (asystolia, pulseless electrical activity - PEA), but also when it beats too fast (pulseless ventricular tachycardia – VT) or in a asynchronised manner (ventricular fibrillation - VF).

Brain injury is likely if cardiac arrest goes untreated for more than five minutes. The brain accounts for about 2% of body weight but consumes 20% of the oxygen from the blood. That is why the brain is most sensitive to the cessation of oxygen supply.
For the best chance of survival and neurological recovery, immediate and decisive treatment is imperative. The treatment for cardiac arrest is cardiopulmonary resuscitation (CPR) to provide circulatory support, followed by defibrillation, if a shockable rhythm (VF and VT) is present.

**What are the signs of cardiac arrest?**

Cardiac arrest is an abrupt cessation of pump function of the heart, as evidenced by the absence of a palpable pulse.
Arrested blood circulation prevents oxygen delivery to the body. Due to inadequate cerebral perfusion, the patient will be unconscious and will have stopped breathing
Therefore, the three signs of cardiac arrest are **unconsciousness**, **cessation of breathing** and **absent pulse**!

**Diagnosis of cardiac arrest**

The main diagnostic criterion of cardiac arrest is lack of circulation, however there are a number of ways of determining this.
A cardiac arrest is usually diagnosed clinically by the **absence of a pulse**. In many cases lack of carotid pulse is the gold standard for diagnosing cardiac arrest, but lack of a pulse (particularly in the peripheral pulses) may be a result of other conditions (e.g. shock), or simply an error on the part of the rescuer.
Studies have shown that rescuers often make a mistake when checking the carotid pulse in an emergency, whether they are healthcare professionals or lay persons. In the face of this evidence, the current recommendation is that cardiac arrest should be diagnosed in all casualties who are unconscious and not breathing normally!

**Causes of cardiac arrest**

Approximately 60–70% of cardiac arrest is **related to cardiac disease**. Among adults, ischemic heart disease is the predominant cause of arrest. No less than 30% of them at autopsy show signs of recent myocardial infarction.
A number of other cardiac abnormalities can increase the risk of cardiac arrest including cardiomyopathy, cardiac rhythm disturbances, hypertensive heart disease, congestive heart failure.
Cardiac arrest is **unrelated to heart problems** in 35% of cases. The most common non-cardiac causes are trauma, non-trauma related bleeding (such as gastrointestinal bleeding, aortic rupture, and intracranial hemorrhage), overdose, drowning and pulmonary embolism.
**Remember -** In infants and children, the most common cause of cardiac arrest is respiratory arrest!

**Treatment of cardiac arrest (cardiopulmonary resuscitation)**

Cardiopulmonary resuscitation (CPR) is a procedure to support and maintain breathing and circulation for someone who has stopped breathing (**respiratory arrest**) and/or whose heart has stopped (**cardiac arrest**).

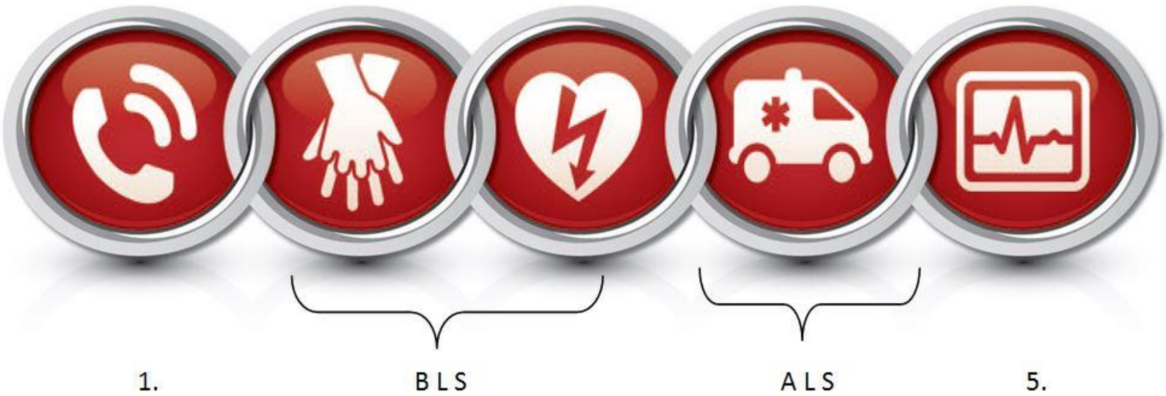
It is performed to restore and maintain breathing and circulation and to provide oxygen and blood flow to the heart, brain, and other vital organs

CPR from didactic reasons can be divided into Basic life support (BLS) and Advanced life support (ALS):

1. **Basic life support** consists of essential non-invasive life-saving procedures including compressions of the chest, automated external defibrillation, rescue breathing and basic airway management. It generally does not include the use of drugs or invasive skills.
2. **Advanced life support** consists of invasive life-saving procedures including the placement of advanced airway adjuncts, intravenous infusions, manual defibrillation, electrocardiogram interpretation, and much more.

ALS and BLS are both life supporting mechanisms but one is just basic and the other one is advanced. However they are inextricably linked. Namely, successful resuscitation following cardiac arrest requires an integrated set of coordinated actions represented by the links in the so-called **Chain of Survival**. The links include the following:

1. Immediate **recognition of cardiac arrest and activation of** the emergency response system
2. Early **CPR with an emphasis on chest compressions**
3. Rapid **defibrillation**
4. Effective **advanced life support**
5. Integrated **post– cardiac arrest care**




As you can see, two important and inseparable links of this chain are BLS and ALS!

ALS actually impacts three key links in the chain of survival - intervention that:

- prevent cardiac arrest,
- treat cardiac arrest, and
- improve outcomes of patients who achieve return of spontaneous circulation (ROSC) after cardiac arrest.

**Preventing cardiac arrest**

Cardiopulmonary resuscitation is actually applied in the three phases of cardiac arrest, in :

- pre-cardiopulmonary arrest (pre-arrest) phase,

- cardiopulmonary arrest (arrest) phase, and
- post-cardiopulmonary arrest (post-arrest) phase.

In the pre-arrest phase, actual or potentially life-threatening health problems occur. These problems require "complex assessment, high intensity therapies and interventions, and continuous nursing vigilance" Why? Because the effectiveness of cardiopulmonary resuscitation (CPR) remains questionable. Survival-to-discharge rates vary widely, averaging 15%. With positive outcomes following cardiac arrest so unlikely, a great deal of effort has been spent in finding effective strategies to prevent new cardiac arrest.

ALS interventions aimed at preventing cardiac arrest include airway management, ventilation support, antiarrhythmic treatment and application of other therapeutic measures in patients with various dysrhythmias, acute coronary syndromes, acute pulmonary edema, hypotension, shock, stroke and related conditions

**ADVANCED LIFE SUPPORT**

Advanced life support consists of:

- placement of advanced airway adjuncts,
- manual defibrillation and other electrical therapy,
- giving medications and intravenous infusion,
- electrocardiogram interpretation,
- and much more.

**Basic notions**

At the beginning it should be noted that the majority of cardiac arrest events occur out of hospital. For cardiac arrest that occurs out of hospital, the emphasis would be on BLS.

For cardiac arrest that occurs in hospital, the emphasis would be on ALS. However, prior to the implementation of ALS measures, for all patients who collapse in the hospital there is a need to ensure the following:

- Immediate recognition of cardiac arrest
- A system of calling for help within ward areas, ambulatory clinics and public areas of the hospital
- Staff who are immediately available to perform chest compressions and ventilation (with devices such as the bag-valve mask or pocket masks)
- Resuscitation equipment, including defibrillator and other devices and drugs to be brought to the patient in the shortest possible time.

CPR is performed if the adult is unresponsive and not breathing or not breathing normally. For healthcare providers “Look, listen, and feel” was removed from the sequence. Therefore, breathing is briefly checked as part of a check for cardiac arrest

The CPR sequence begins with compressions (C-A-B sequence). After the first set of chest compressions (C), the airway is opened (A), and the rescuer delivers 2 breaths (B). Continue with chest compressions and rescue breaths in a ratio of 30:2.

**Chest compressions**

Compress the chest at a rate of 100-120 per minute. You have to remember that the number of chest compressions delivered per minute during CPR is an important determinant of ROSC (***Return of spontaneous circulation)*** and survival with good neurologic function.

Each time compressions are resumed, place your hands without delay ‘in the centre of the chest’. Pay attention to achieving the full compression depth of 5-6 cm (for an adult). Compressions create blood flow primarily by increasing intrathoracic pressure (thoracic pump) and directly compressing the heart (cardiac pump).
Allow the chest to recoil completely after each compression.Take approximately the same amount of time for compression and relaxation.
Minimise interruptions in chest compression. Provision of adequate chest compressions requires an emphasis not only on an adequate compression rate but also on minimizing interruptions to this critical component of CPR.
Do not rely on a palpable carotid or femoral pulse as a gauge of effective arterial flow.
Even when performed correctly, conventional, manual, standard cardiopulmonary resuscitation provides only 10 – 20% of normal blood flow to the heart, and 20 – 30% of normal blood flow to the brain. This is partially due to the fact that conventional CPR is inherently inefficient for two reasons:

- Filling of the heart (preload) is dependent upon the chest wall’s ability to passively recoil. Inadequate chest wall recoil can occur if the chest is not very compliant, rescuer is tired and begins to “rest” on the chest or ribs are broken.
- With an open airway, as the chest wall recoils, air is drawn in and wipes out the vacuum (negative intrathoracic pressure) that is responsible for creating preload.

The **CardioPump®** permits the rescuer to actively re-expand the chest during the decompression phase of cardiopulmonary resuscitation (CPR). Active compression - decompression CPR (ACD CPR) enhances the intrathoracic vacuum (negative pressure) during chest wall recoil, resulting in more blood being returned to the heart (preload). Enhanced preload leads to increased cardiac output on the subsequent chest compression.
The **ResQPOD®** impedance threshold device (ITD) enhances circulation during basic or advanced life support CPR. Attached to a face mask or other airway adjunct, the ResQPOD selectively prevents air from re-entering the lungs during chest wall recoil. This enhances the vacuum needed to pull blood back into the heart. As a result, more blood is circulated to vital organs until the heart can be restarted.


**CardioPump®** **ResQPOD®**

Both devices significantly increase the blood flow caused by compressions, and this consequently increases cerebral and coronary perfusion pressure. In this way, each device increases the survival rate of about 50%, in combination, survival increased by another 6-9%.
Given the difficulty of performing consistent CPR compressions, technology has turned to automation. There are two types of automated compressors available:

- LUCAS, a new gas-driven CPR device providing automatic chest compression and active decompression, and
- AUTOPULSE, mechanically actuated and battery driven CPR device which provides both direct compression and semi-circumferential thoracic compression.

Both devices operate in 30:2 and continuous compression mode.

**LUCAS**

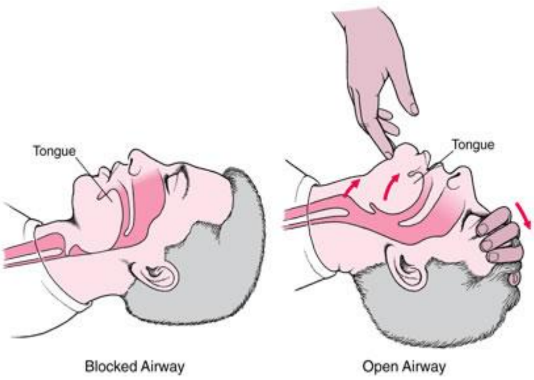

**AutoPulse**

**Airway**

The simplest way of ensuring an open airway in an unconscious patient is to use a head tilt / chin lift technique :

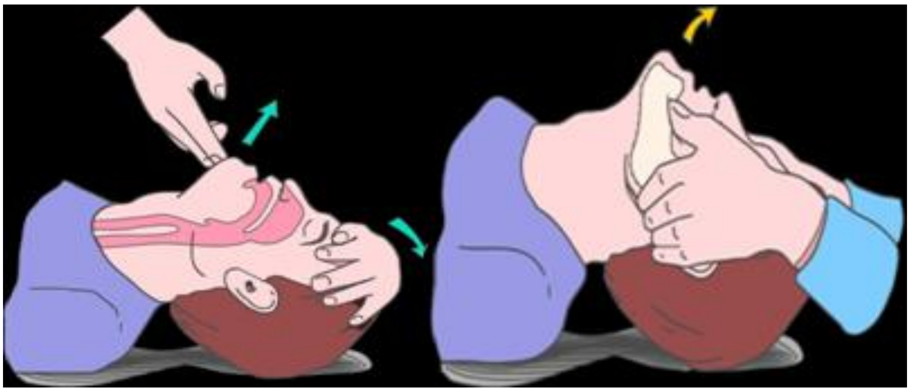
- **Head tilt** - One hand is placed over victim's forehead and firm, backward pressure is applied with palm to tilt the head back

- **Chin lift** - Place the fingers of the other hand under the bony part of the chin. Lift the chin forward and support the jaw, helping to tilt the head back. The fingers must not press deeply in the soft tissues under the chin, as this might obstruct the airway!




**Head tilt / chin lift**

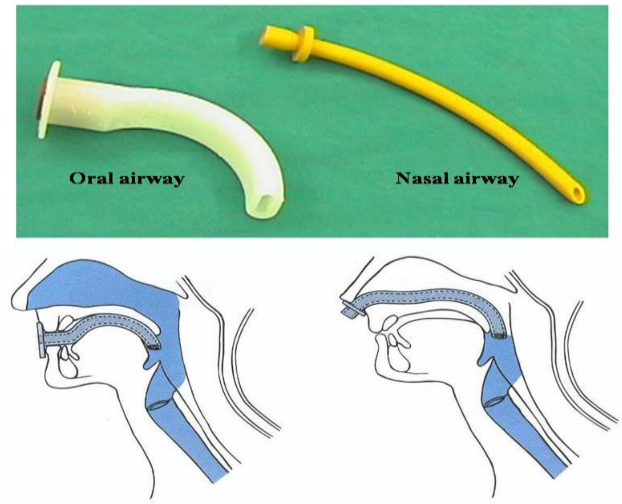
The **jaw thrust technique** is not recommended for lay rescuers because it is difficult to learn and perform. Therefore, the lay rescuer should open the airway using a head-tilt, chin-lift manoeuvre for both injured and non-injured victims. Health professionals must be familiar with this technique and must apply it when indicated!



**Head tilt /chin lift** **Jaw thrust**

**Oropharyngeal airways** prevent the tongue from occluding the upper airway. It may be used in unresponsive patients (those with no cough or gag reflex), or during bag-mask ventilation.

**Nasopharyngeal airways** may be used if a clenched jaw prevents insertion of the oral airway. It has been known to cause nose bleeding in up to 30% of instances and is to be used with caution in the presence of cranio-facial injury.




**Oropharyngeal and nasopharyngeal airway**

Advanced airways are :

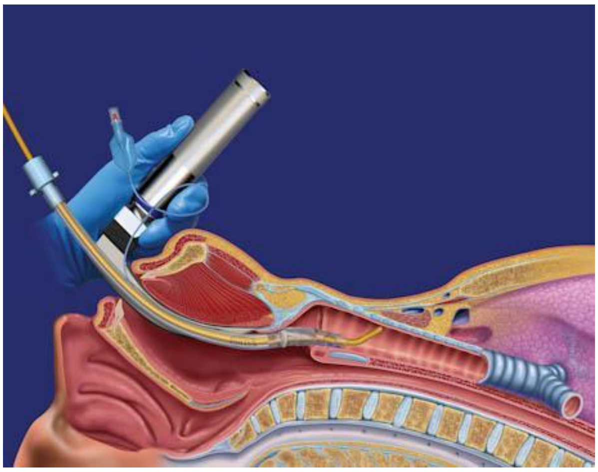
- Endotracheal tube (ETT) and
- Supraglottic airways - a device with an orifice for air situated above the glottis (laryngeal mask, laryngeal tube, combi tube, i-gel,..)

It must be remembered that placement of an advanced airway may be associated with a number of potential risks. This would be less likely with use of supraglottic airways. There is, therefore, a need for frequent training and retraining in these procedures to ensure currency of skills.

**ET intubation** remains the gold standard for airway management. Namely, ETT placement has many benefits such as allowing a definitive patent airway, suction of secretions, reliable oxygen delivery and protect from aspiration of gastric contents. It would be indicated when there is inability to ventilate adequately with a bag-valve mask resuscitator or if the patient is in coma or in cardiac arrest (absence of protective reflexes)

Endotracheal intubation by an unskilled provider can result in the following:

- Trauma to the oropharynx with bleeding
- Long interruptions to compressions / ventilations with their adverse impact on outcomes.
- Hypoxemia for prolonged periods and the resulting cerebral hypoxia
- Failure to recognize misplacement / displacement of the tube which has been recognized in as many as 6 – 25% of instances



**Endotracheal intubation**

**Supraglottic airways** such as the LMA (laryngeal mask airway), Combitube or the Laryngeal tube are alternative procedures that may be used especially by prehospital care providers or by physicians if direct airway control is desired for short periods.

Laryngeal mask

I-gel

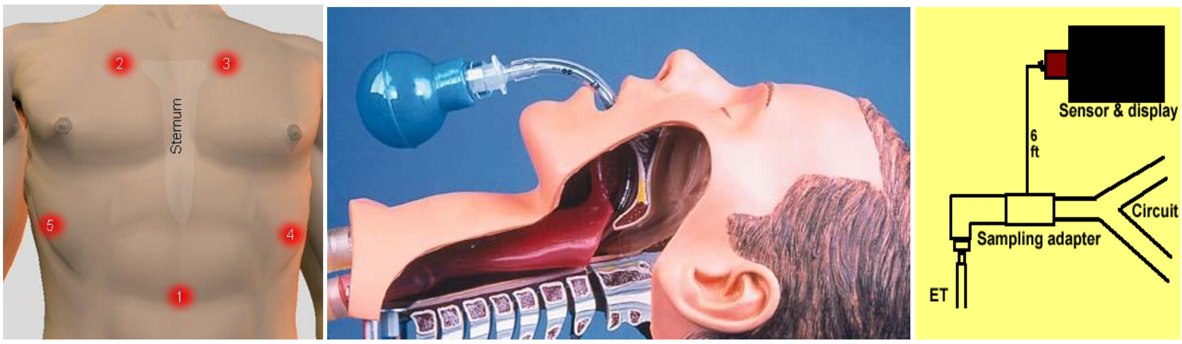
Laryngeal tube

Its main advantage is that direct visualization of the glottis is not required and training and skills maintenance are easier. A main disadvantage of these devices are theirs inability to protect against pulmonary aspiration and regurgitation of gastric contents.

Once the advanced airway is in position and secured, correct placement should be confirmed by :

1. Bilateral chest expansion
2. 5-point auscultation
3. Esophageal Detector Devices

4. Continuous $ETCO_2$ monitoring
5. Chest X ray



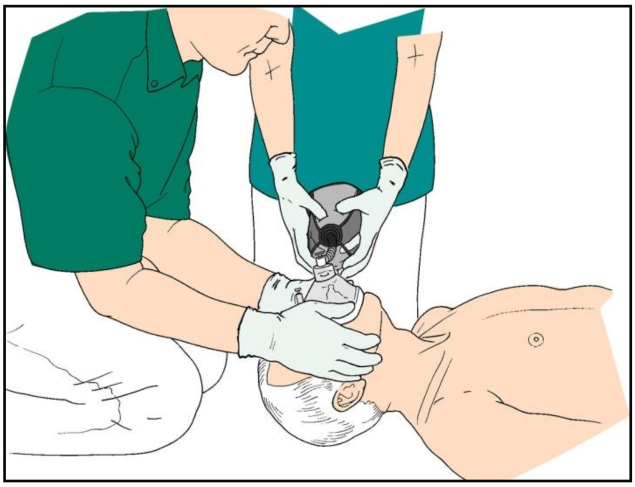

Methods of test for correct placement:
auscultation, application of esophageal detector devices or continuous $ETCO_2$ monitoring

**Breathing**

Basic ventilation skills (mouth-to-mouth ventilation or mouth-to-barrier device including using a pocket mask) are rarely used in ALS as opposed to positive pressure ventilation.

Positive-pressure ventilation (it means that airway pressure is applied at the patient's airway through an endotracheal or tracheostomy tube) has been a mainstay of ALS. However, positive pressure ventilation, especially when associated with high tidal volumes and ventilation rates can potentially lead to increased intra-thoracic pressures, decreased venous return and low cardiac output. For these reasons it is recommended to limit rate of ventilation to 8 -10 breaths per minute and tidal volume to 400 - 600 ml, for adults.

**Bag-Mask ventilation** (BMV) is the most common method of providing positive-pressure ventilation. Both the oropharyngeal airway and the nasopharyngeal airway may be used as adjuncts to improve effectiveness of patient ventilation. The oropharyngeal airway may only be used on the unconscious patient because it can stimulate gagging and vomiting in a conscious patient. The nasopharyngeal airway may be used on the unconscious patient or on the semiconscious patient and is also indicated if a patient has massive trauma around the mouth or wiring of the jaws.
If bag-mask ventilation is being used, it is recommended that the user should compress the bag by about one-third which would be sufficient to produce a chest rise over 1 second. If given in excess and at higher rates, BMV can result in gastric inflation, regurgitation, aspiration and chest infection.



**Bag-mask ventilation**

In both out-of-hospital and in-hospital settings, **automatic transport ventilators** (ATVs) can be useful for ventilation of adult patients in cardiac arrest who have an advanced airway in place. During prolonged resuscitative efforts the use of an ATV (pneumatically powered and time- or pressure-cycled) may allow the EMS team to perform other tasks while providing adequate ventilation and oxygenation.


One of automatic transport ventilator,  very easy to use and quite safe

**Electrical therapy**

Electrical therapy in CPR consist of:

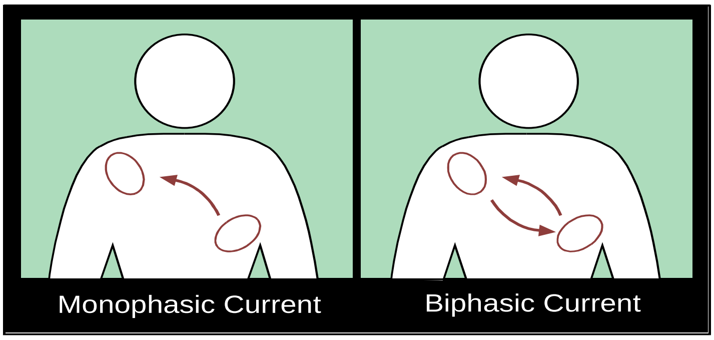
1. defibrillation with automated external defibrillators (AEDs) or manual defibrillators,
2. synchronized cardioversion, and
3. pacing.

**Defibrillation** is a process in which an electronic device, called a defibrillator, gives an electric shock to the heart. This causes the whole heart to stop all activity. The heart's normal pacemaker (SA node) then can try and restart normal beating. This helps reestablish normal contraction rhythms in a heart having dangerous arrhythmia or in cardiac arrest.

There are two types of defibrillators by method of delivery of electric shock, monophasic and biphasic:

- Monophasic defibrillators deliver current of one polarity
- Biphasic defibrillators, deliver current that alternates the direction, completing one cycle in approximately 10 milliseconds

Biphasic technology are used in almost all AEDs and manual defibrillators sold today.




ALS defibrillators, used by healthcare professionals in hospitals and ambulances, allow professionals to monitor the patient rhythm and manually intervene if it is determined that a shock is required. It can be used with either paddles or electrodes, though the trend today is to use the defibrillation electrode as it is much safer for the rescuer and delivers the shock more uniformly and effectively.


Manual defibrillator

Beyond the ability to deliver a shock, ALS defibrillators are often outfitted with a number of parameters to aid rescuers. Most in-hospital ALS units have an *external pacing* capability to allow external pacing of bradycardias. Many also offer $SPO_2$, a means to monitor the oxygenation level of the patient via an external sensor. Other options available on ALS defibrillators include $EtCO_2$ to monitor carbon dioxide levels.

All emergency personnel should be trained and allowed to use a properly maintained defibrillator if they're likely to respond to cardiac arrest victims. This includes all first-responding emergency personnel, both hospital and non-hospital.

**How to apply manual defibrillator for defibrillation :**

| |
|---|
| 1. Turn defibrillator on.<br>2. Apply defibrillator paddles/pads according to manufacturer specifications.<br>3. Charge defibrillator to energy level specified in appropriate protocol or according to manufacturer specifications.<br>4. Check rhythm.<br>5. Defibrillate patient if indicated.<br>6. Recheck pulse and rhythm.<br>7. If VF or pulseless VT persist, provide additional defibrillation per protocol |
| The recommended initial energy dose for defibrillation in adults is :<br>o **For monophasic defibrillation** - 360 joules per shock<br>o **For biphasic defibrillation** - 150 joules with consideration for escalating higher energy defibrillation up to a maximum of 360 joules, if needed. |

To make early defibrillation possible, a defibrillator must be immediately available to emergency personnel responding to a cardiac arrest. Thus, all emergency ambulances and other emergency vehicles that respond to or transport heart patients should have a defibrillator.

Modern defibrillators can be operated in both manual and semiautomatic (AED-similar) modes. Use of the manual mode enables chest compressions to be continued during charging, thereby minimizing the preshock pause. A shorter preshock pause and lower total hands-off ratio increased vital organ perfusion and the probability of ROSC.

Synchronized cardioversion is shock delivery that is synchronized with the QRS complex. This synchronization avoids shock delivery during the relative refractory portion of the cardiac cycle, when a shock could produce VF.

- Synchronized cardioversion is recommended to treat :
    - supraventricular tachycardia due to reentry,
    - atrial fibrillation,
    - atrial flutter,
    - atrial tachycardia, and
    - monomorphic VT with pulses.

| **How to apply manual defibrillator for synchronized cardioversion :** |
|---|
| 1. Consider IV sedation per protocol.<br>2. Turn synchronizer switch "on". Assure QRS complex is marked.<br>3. Apply defibrillator paddles/pads according to manufacturer specifications.<br>4. Charge defibrillator to energy level specified in appropriate protocol or according to manufacturer specifications.<br>5. Check rhythm.<br>6. Cardiovert patient.<br>7. Recheck pulse and rhythm<br>8. If rhythm does not convert, re-cardiovert according to the appropriate protocol.<br>9. If ventricular fibrillation occurs, deactivate synchronized mode and defibrillate. |

| The recommended initial biphasic energy dose for synchronized cardioversion of adult is : |
|---|
| ○ for atrial fibrillation 120 to 200 J<br>○ for atrial flutter and other supraventricular tachycardias 50 J to 100 J<br>○ for monomorphic VT (regular form and rate) with a pulse 100 J. |

**Pacing** is not recommended for patients in asystolic cardiac arrest and may delay or interrupt the delivery of chest compressions. It is indicated in symptomatic bradycardia, in patients who do not respond to atropine (or second-line drugs).

| How to apply transcutaneous pacing |
|---|
| 1. Monitor EKG.<br>2. Consider sedation per protocol.<br>3. Apply Pacing Electrodes (anterior - posterior preferred).<br>– One pad to left anterior chest and one pad beneath left scapula or per manufacturer specifications.<br>– Pads may also go on antero-lateral (lead II) position<br>4. Assure adequate amplitude of QRS complexes<br>5. Set external pacemaker rate to 70 beath per minute<br>6. Rapidly dial up at increments of 10-20 MA until capture occurs.<br>– Use only minimal power current (MA) needed for mechanical capture.<br>7. Assure adequate electrical/mechanical capture.<br>– **Electrical:** Visible pacer spike immediately followed by wide QRS and T-wave.<br>– **Mechanical:** improved pulses, awareness (LOC), BP<br>8. If mechanical capture is not obtained, contact medical control or perform CPR if appropriate. |

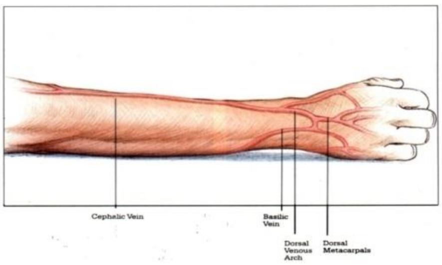
**Drugs**

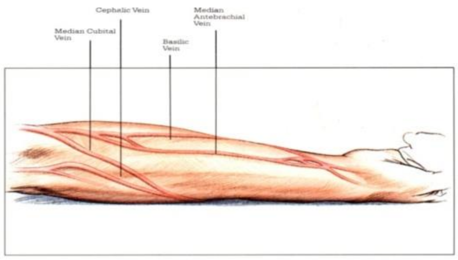
Drug therapy is not the primary form of management of cardiac arrest. The use of drugs in cardiac arrest patients is an adjunct to the earlier components of care.

The special features concerning use of pharmacological agents during cardiac arrest management are as follows:

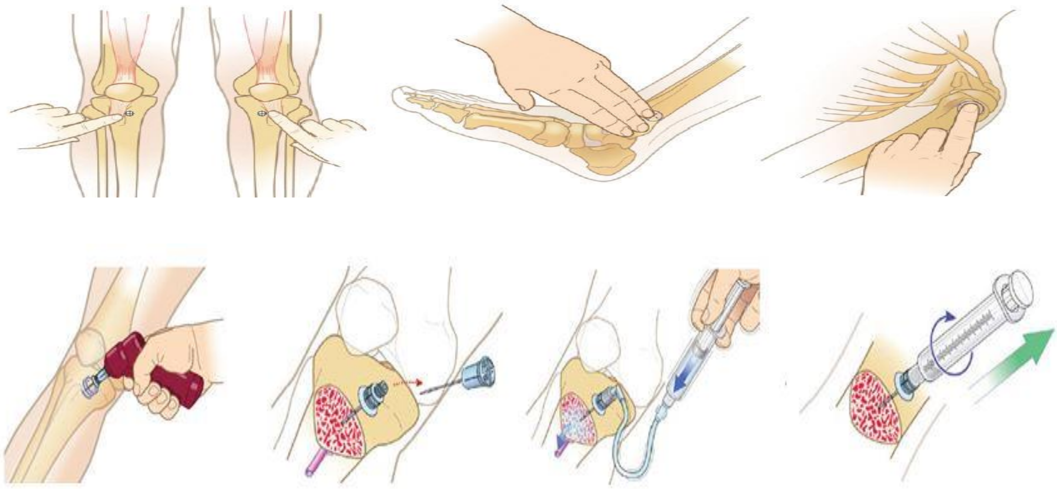
A. Routes of drug delivery:

   a. **Peripheral large caliber veins**, especially the antecubital and external jugular veins will be the commonest route to be used.



b. **Intra-osseus (IO) cannulation** using IO needles can allow drug delivery to the non-collapsible venous plexuses in the bone marrow. This may be resorted to if intravenous access is not easily available.



c. **Central venous lines** either through the subclavian or internal jugular veins shorten medication access to the central circulation. In addition, the presence of a central line allows measurement of central venous pressure to guide fluid resuscitation. Central lines, however, cannot be used for rapid infusion of fluids. Also, the insertion of a central line is also likely to result in interruptions to chest compressions.

B. Circulation time during CPR is prolonged.

a. Drugs need to reach the central circulation from the peripheral vasculature to exert their effects. This requires at least 30 to 60 seconds of good quality chest compressions.

b. Therefore, after each drug administration flush the line with 10 – 20 ml of normal saline and continue CPR for at least 30-60 sec before next shock is given, if needed.

C. Common resuscitation drugs to remember: Adrenaline, Amiodarone, Lignocaine, Adenosine, Dopamine, Magnesium sulphate,...

| Drug | Dose | Indications | Timing of administration | Other |
|---|---|---|---|---|
| **Adrenaline** | 1 mg IV (0.01 mg/Kg) | ► Given immediately in **non-shockable** rhythm.<br>► Given after the **3rd** shock in **shockable** rhythm (VT/VF). | Repeated every 4 minutes (every other cycle).<br><br>***"Once adrenaline ALWAYS adrenaline***". | Given as a vasopressor for its α-adrenergic effect. Not as an inotrope. |

| | | | | |
|---|---|---|---|---|
| **Amiodarone** | 300 mg IV **bolus** (5 mg/Kg) | ► Given after the **3rd** shock in **shockable** rhythm. | ► A further dose of 150 mg if VT/VF persists. | If amiodarone is not available **Lidocaine** can be used instead. |
| **Lidocaine** | 100 mg IV (1-1.5 mg/Kg) | ► Given after the **3rd** shock in **shockable** rhythm (IF amiodarone is unavailable). | ► A further dose of 50 mg can be given if necessary. | Total dose must not exceed 3 mg/Kg during the 1st hour. |
| **Magnesium** | 2 g IV | ► VT.<br>► Torsade de pointes.<br>► Digoxin toxicity with hypomagnesemia | ► May be repeated after 10-15 minutes. | |
| **Sodium Bicarbonate** | 50 mmol IV | ► Routine use is not recommended.<br>► Hyperkalemia.<br>► Overdose of TCA. | ► May be repeated according to ABG. | Do NOT give calcium solutions and $NaHCO_3$ simultaneously by the same route. |

**H's and T's of ACLS**

The H's and T's of ALS is a mnemonic used to help recall the major contributing factors to pulseless arrest including PEA, Asystole, Ventricular Fibrillation, and Ventricular Tachycardia. These H's and T's will most commonly be associated with PEA, but they will help direct your search for underlying causes to any of arrhythmias associated with ALS.

*The H's include:*

1. **Hypovolemia** or the loss of fluid volume in the circulatory system can be a major contributing cause to cardiac arrest. Looking for obvious blood loss in the patient with pusleless arrest is the first step in determining if the arrest is related to hypovolemia. After CPR, the most import intervention is obtaining intravenous access/IO access. A fluid challenge or fluid bolus may also help determine if the arrest is related to hypovolemia.
2. **Hypoxia** or deprivation of adequate oxygen supply can be a significant contributing cause to cardiac arrest. You must ensure that the patient's airway is open, and that the patient has chest rise and fall and bilateral breath sounds with ventilation. Also ensure that your oxygen source is connected properly.
3. **Hydrogen ion (acidosis)** - To determine if the patient is in respiratory acidosis, an arterial blood gas evaluation must be performed. Prevent respiratory acidosis by providing adequate ventilation. Prevent metabolic acidosis by giving the patient sodium bicarbonate.
4. **Hyper-/hypokalemia** - Both a high potassium level and a low potassium level can contribute to cardiac arrest.

The major sign of hyperkalemia or high serum potassium is taller and peaked T-waves. Also, a widening of the QRS-wave may be seen. This can be treated in a number of ways which include sodium bicarbonate (IV), glucose+insulin, calcium chloride (IV), Kayexalate, dialysis, and possibly albuterol. All of these will help reduce serum potassium levels.

The major signs of hypokalemia or low serum potassium are flattened T-waves, prominent U-waves, and possibly a widened QRS complex. Treatment of hypokalemia involves rapid but controlled infusion of potassium. Giving IV potassium has risks. Always follow the appropriate infusion standards. Never give undiluted intravenous potassium.

5. **Hypothermia** - If a patient has been exposed to the cold, warming measures should be taken. The hypothermic patient may be unresponsive to drug therapy and electrical therapy (defibrillation or pacing). Core temperature should be raised above 30°C (86 F) as soon as possible.

*The T's include:*

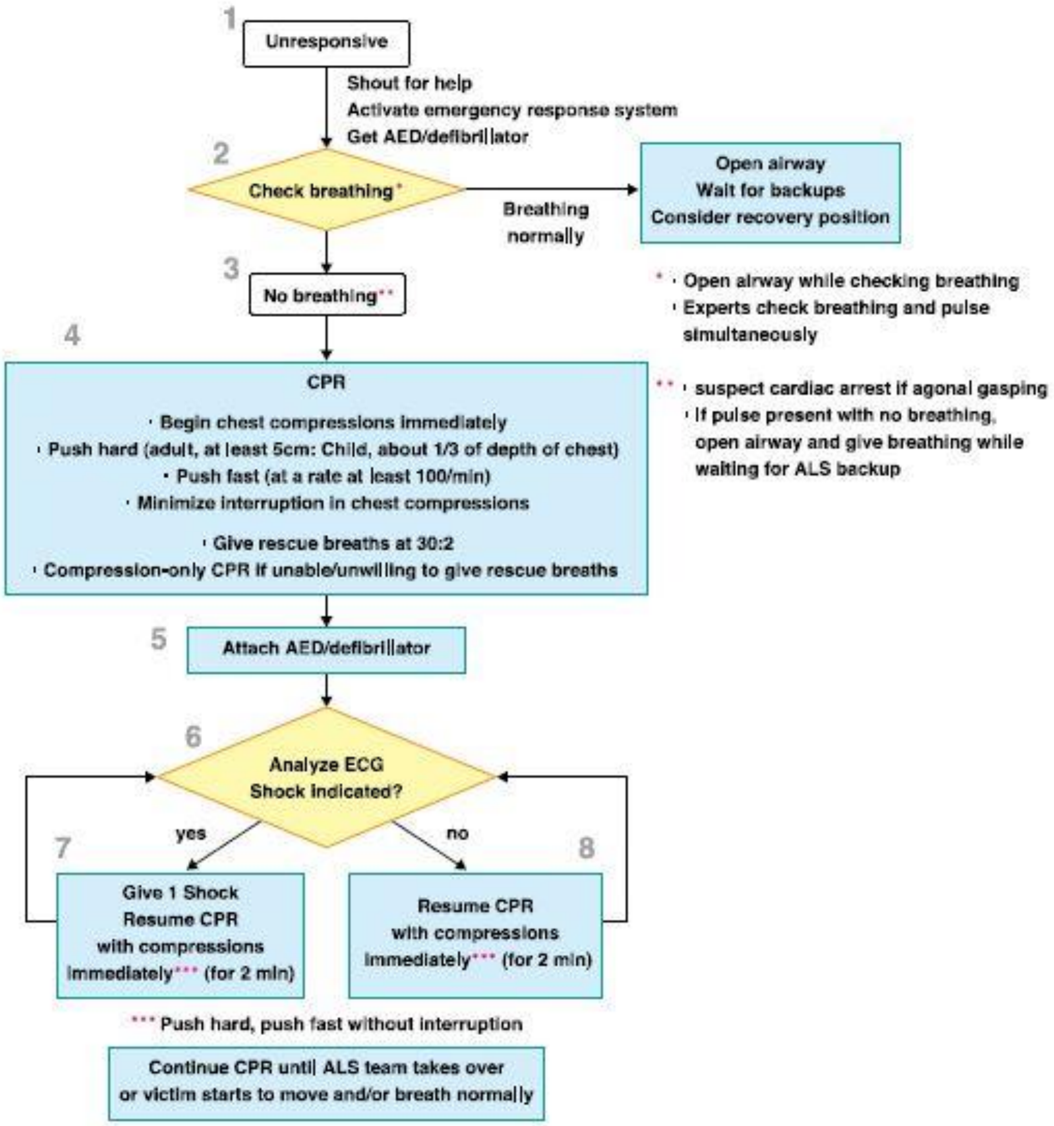
1. **Toxins** - Accidental overdose of a number of different kinds of medications can cause pulseless arrest. Some of the most common include: tricyclics, digoxin, betablockers, and calcium channel blockers). Street drugs and other chemicals can precipitate pulseless arrest. Cocaine is the most common street drug that increases incidence of pulseless arrest. ECG signs of toxicity include prolongation of the QT interval. Physical signs include bradycardia, pupil symptoms, and other neurological changes. Support of circulation while an antidote or reversing agent is obtained is of primary importance. Poison control can be utilized to obtain information about toxins and reversing agents.
2. **Tamponade** - Cardiac tamponade is an emergency condition in which fluid accumulates in the pericardium (sac in which the heart is enclosed). The buildup of fluid results in ineffective pumping of the blood which can lead to pulseless arrest. ECG symptoms include narrow QRS complex and rapid heart rate. Physical signs include jugular vein distention (JVD), no pulse or difficulty palpating a pulse, and muffled heart sounds due to fluid inside the pericardium. The recommended treatment for cardiac tamponade is pericardiocentesis.
3. **Tension pneumothorax** occurs when air is allowed to enter the plural space and is prevented from escaping naturally. This leads to a build up of tension that causes shifts in the intrathroacic structure that can rapidly lead to cardiovascular collapse and death. ECG signs include narrow QRS complexes and slow heart rate. Physical signs include JVD, tracheal deviation, unequal breath sounds, difficulty with ventilation, and no pulse felt with CPR. Treatment of tension pneumothorax is needle decompression.
4. **Thrombosis (heart: acute, massive MI)** - Coronary thrombosis is an occlusion or blockage of blood flow within a coronary artery caused by blood that has clotted within the vessel. The clotted blood causes an acute myocardial infarction which destroys heart muscle and can lead to sudden death depending on the location of the blockage. ECG signs during PEA indicating coronary thrombosis include ST-segment changes, T-wave inversions, and/or Q waves. Physical signs include: elevated cardiac markers on lab test. Treatments for coronary thrombosis before cardiac arrest include use of fibrinolytic

therapy, or PCI (percutaneous coronary intervention). The most common PCI procedure is coronary angioplasty with or without stent placement.

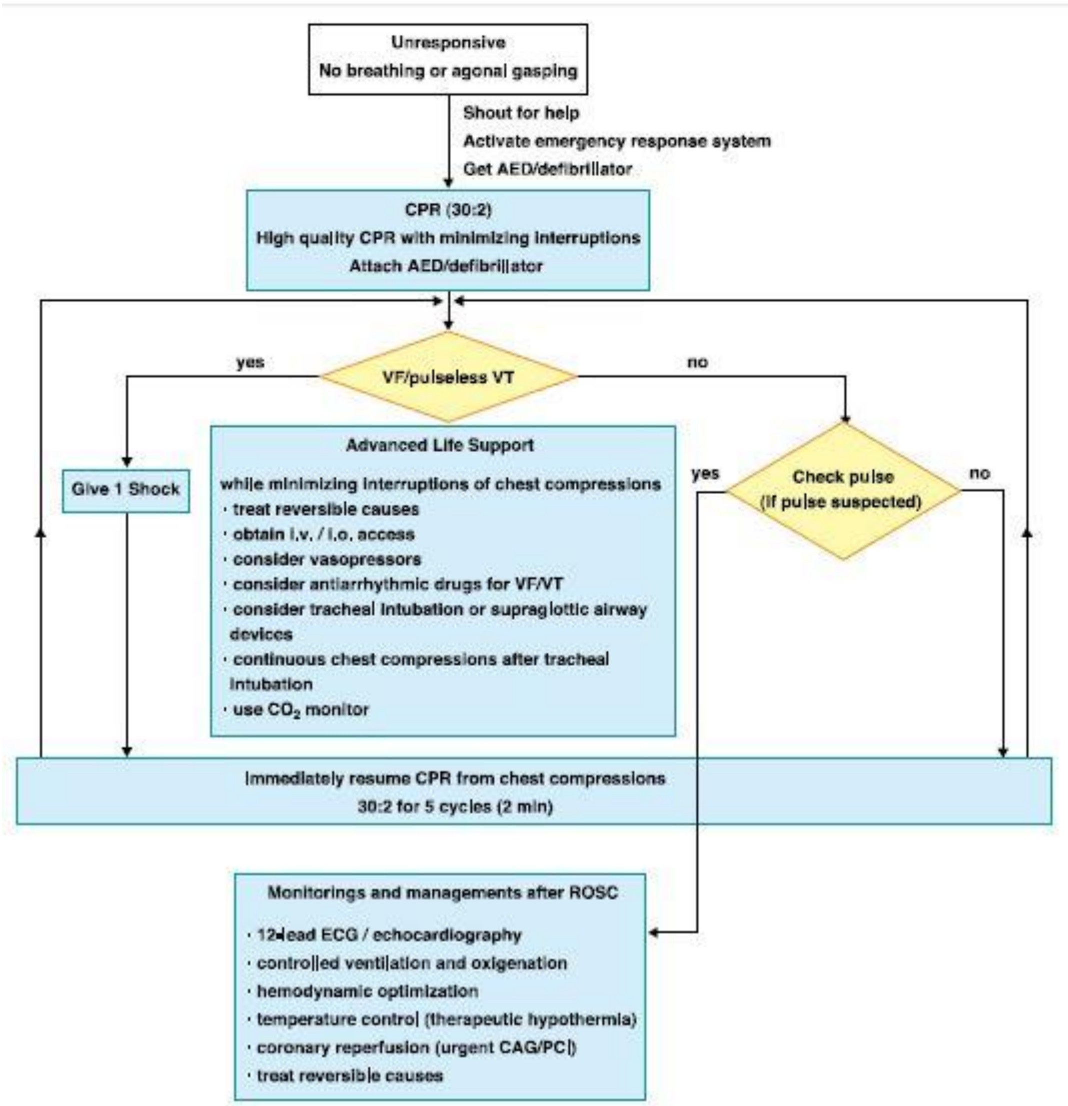
5. **Thrombosis (lungs: massive pulmonary embolism)** - Pulmonary thrombus or pulmonary embolism (PE) is a blockage of the main artery of the lung which can rapidly lead to respiratory collapse and sudden death. ECG signs of PE include narrow QRS Complex and rapid heart rate. Physical signs include no pulse felt with CPR. distended neck veins, positive d-dimer test, prior positive test for DVT or PE. Treatment includes surgical intervention (pulmonary thrombectomy) and fibrinolytic therapy.

**Cardiac arrest algorithm**

When a lay rescuer gives CPR, they should follow the BLS algorithm. But healthcare providers who would treat a cardiac arrest patient in hospital or as their duty are recommended to folow the BLS algorithm for healthcare providers as a preface of ALS algorithm. These algorithms are ideal when rescuer can use the device and drugs if needed.




**Basic life support for healthcare providers**




**Advanced life support algorithm**

*** ALS algorithm includes the managements during cardiac arrest and after return of spontaneous circulation (ROSC).**

**POST–CARDIAC ARREST CARE**

It should be noted that return of spontaneous circulation (ROSC) is just the first step toward the goal of complete recovery from cardiac arrest. Namely, after ROSC, many patients remain disabled or die. Brain injury, heart dysfunction, systemic inflammation and the underlying disease that caused the cardiac arrest all contribute to the morbidity and high death rate of patients after ROSC. That's why resuscitation DOES NOT STOP after ROSC.

The aims of post–cardiac arrest care are to:

1. Continue respiratory support - It is reasonable to titrate inspired oxygen to achieve a $SaO_2$ of 94-98%. Rutine hyperventilation may be detrimental (result in cerebral vasoconstrictions) and should be avoided. Ventilation to normocarbia (eg. $PaCO_2$ 35 to 40 mm Hg) is appropriate.

2. Optimize blood pressure and maintain adequate organ perfusion, particuraly cerebral and coronary perfusion - It is resonable to titrate IV fluid, vasopressors and/or inotropes, to achieve systolic pressure greather than 100 mm Hg or MAP ≥ 65 mm Hg .

3. Treat and prevent cardiac arrhythmias - No studies specifically and directly addressed the prophylactic use of antiarrhythmic therapy after resuscitation from cardiac arrest. It may be reasonable to continue an infusion of an antiarrhythmic drug that successfully restored stable rhythm during resuscitation (eg. amiodarone 0,6 mg/kg/hr or lignocaine 2-4 mg/min for 12-24 hours).

4. Determine and treat the cause of the arrest - If not already undertaken, management should be directed toward the treatment of underlying causes that have been identified.

5. Identify and treat acute coronary syndromes (ACS) - A 12-lead ECG should be obtained as soon as possible after ROSC to determine whether acute ST elevation is present. If there is evidence of coronary occlusion, consider immediate revascularisation by thrombolysis or percutaneous coronary intervention. If the facilities are available, primary PCI is the preferred technique for revascularisation.

6. Control body temperature - Treat any hyperthermia occurring in the first 72 h after cardiac arrest with antipyretics or active cooling.

7. Treat seizures - Prolonged seizure activity may cause cerebral injury, and should be treated promptly and effectively.

8. Control blood glucose - Hyperglycemia after cardiac arrest is associated with a poor neurologic outcome. Recent studies indicate that post cardiac arrest patients may be treated optimally with a target range for blood glucose concentration of up to 10 mmol/L. Ovoid hypoglycemia!

9. Reduce or prevent brain injury with therapeutic hypothermia - For protection of the brain (and other organs), hypothermia is a helpful therapeutic approach in patients who remain comatose after ROSC. Unconscious adult patients with spontaneous circulation after out-of-hospital VF cardiac arrest should be cooled to 32–34°C. Cooling should be started as soon as possible and continued for at least 12–24 h. Rewarm slowly (0.25–0.50 C/h).

10. Prevent multiple organ dysfunction - This includes avoiding excessive ventilation and hyperoxia.

Finally it should be noted that systematic post–cardiac arrest care has significant potential to reduce **early mortality** caused by hemodynamic instability, and **later morbidity and mortality** from multiorgan failure and brain injury, and can improve the likelihood of patient survival with good quality of life.

# 12. MEDICAL CARE OF THE INJURED PATIENT

**Mihajlo Lojpur, M.D., Ph.D.**

The evaluation and management of the trauma patient, by necessity, begins with the primary survey - a brief and focused physical diagnostic sequence. During this interval, any major life-threatening injuries are systematically identified, and immediate lifesaving interventions are performed.

The secondary survey represents a brief trauma-related history and complete physical examination.

**The primary survey** is often described by the mnemonic ABCDE (Figure 1.). **A** refers to the evaluation and management of the airway while maintaining control of a vulnerable cervical spine. **B** depicts the assessment of breathing. **C** relates to evaluation of circulation, with the immediate control of active hemorrhage. **D** describes disability - a rapid neurological status assessment. **E** denotes exposure of the patient with environmental control - a prescription to remove all clothing, turn the patient to expose the posterior torso and any other regions where external stigmata of injury may be evident, as well as to cover the patient with some barrier against heat loss.

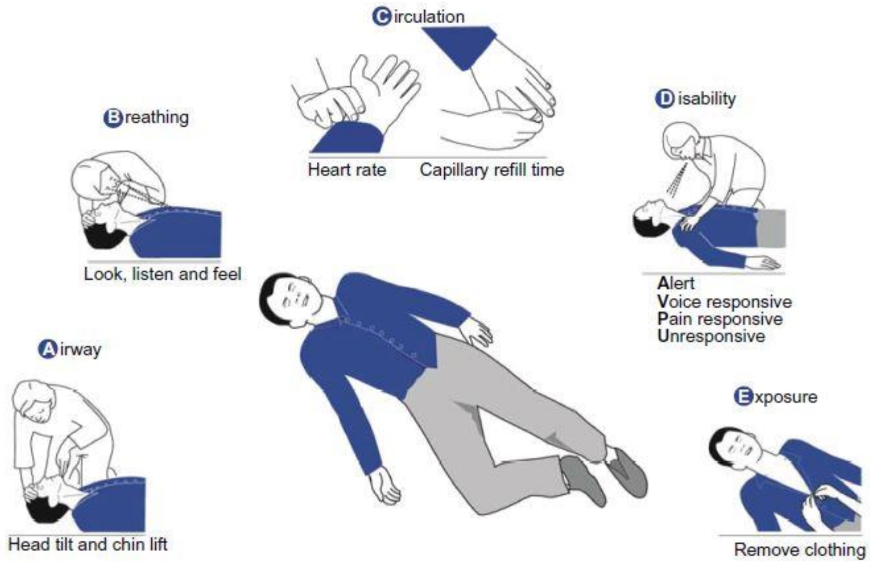



*Figure 1. The ABCDE approach without the use of equipment*

In each area of concern, the examination is simplified and rapid. Any immediately life-threatening issue must be addressed expeditiously. Although the components of the primary survey may be assessed almost simultaneously by a coordinated trauma team, lifesaving interventions are best prioritized with airway considerations preceding those of breathing, circulation, and so on. Moreover, the primary survey should be performed every time there is a decline in patient status, no matter when in the temporal course of patient management.

The airway in a neurologically intact patient is not difficult to assess. The injured patient who is alert and able to speak normally is maintaining a patent airway. However, this must be

carefully monitored as facial fractures with associated bleeding or edema, emesis, or foreign bodies can eventually compromise airway patency.
In acutely injured patients, particularly those sustaining blunt trauma, the cervical spine is unlikely to be ‘‘cleared’’ with respect to occult injury, and airway management must take into consideration the potential for a bony or ligamentous cervical spine injury.
Initial airway management is performed with a jaw thrust maneuver with inline cervical spine immobilization and administration of supplemental oxygen. The commonly taught head tilt, chin lift maneuver is contraindicated unless cervical spine injury has been ruled out.

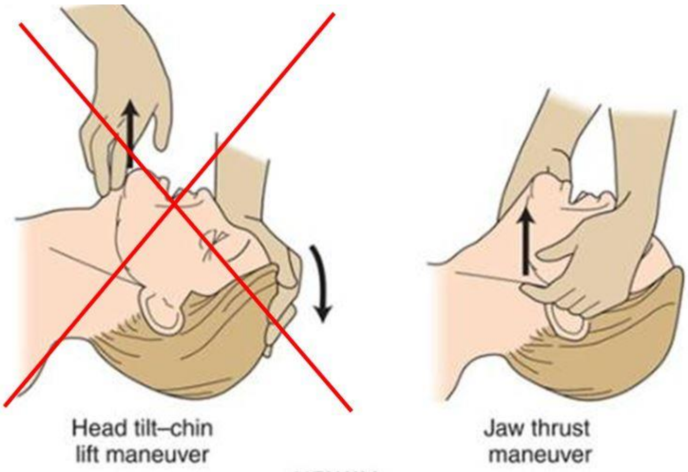



Figure 2. *Airway opening in suspected cervical spine injury*
Bag valve ventilation should be performed until a definitive airway can be obtained. The Sellick maneuver involves applying cricoid pressure that subsequently compresses the esophagus. It should be performed whenever a self-inflating resuscitation bag is used to prevent gastric insufflation and aspiration at the time of intubation.

*Figure 3. Bag valve ventilation and Sellick maneuver*

As a general rule, for patients with severe lower facial or neck trauma, tracheal intubation is required in the presence of the following conditions:

- severe brain injury,
- documented or highly suspected thermal inhalation injury,

- severe pulmonary contusion with hypoxemia or ventilatory insufficiency, high cervical spine injuries, or any injury resulting in pulmonary failure.

Furthermore, ongoing large-volume resuscitation can cause a decline in respiratory status due to airway or pulmonary edema that may necessitate emergent airway control.

Tracheal intubation via the orotracheal route is completed while inline stabilization of the neck is maintained by an assistant. This makes intubation more technically difficult, and the assistance of an experienced provider is recommended.

*Figure 4. In line stabilisation of the neck for ET intubation*

As few patients have been fasting prior to the traumatic incident, rapid sequence intubation, employing cricoid pressure, is the preferred method to prevent the potential aspiration of gastric contents. A suction device should always be readily available when managing the airway to facilitate the expeditious clearing of oral secretions or refluxed gastric contents.

Assessment of breathing begins with evaluation of the patient’s thorax and neck. Observe for deviation of the trachea, equal and normal expansion of the chest, abnormal chest wall motion, or the use of accessory respiratory muscles. Listen to the patient’s speech for labored effort. Auscultate the chest, listening for the presence of equal bilateral breath sounds. Palpate the chest to determine if there is tenderness, crepitus, or areas of abnormal chest wall movement, including flail segments.

Hemodynamic instability associated with tracheal deviation and loss of breath sounds on the side away from deviation is suggestive of a tension pneumothorax and merits immediate thoracic decompression. Jugular venous distension will also be present if the patient is not severely hypovolemic.

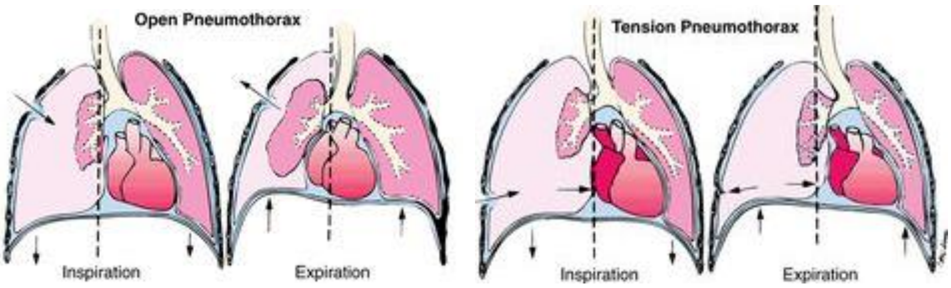



*Figure 5. Open and tension pneumothorax*

Needle decompression of the thorax with a 4 - 5 cm 14 or 16 gauge catheter, placed in the second intercostal space in the midclavicular line, should be performed as a temporizing measure, followed by a tube thoracostomy. Tube thoracostomy can be performed immediately instead of needle decompression if the equipment is readily available and the procedure can be performed rapidly.

*Figure 6. Needle decompresision and tube thoracostomy*

Diminished anterior breath sounds in the supine patient with ipsilateral crepitus, but without hemodynamic compromise, is suggestive of a simple pneumothorax. This will often also require a tube thoracostomy that can be accomplished using an 8 or 10 French drain.

Administration of high concentrations of oxygen via a face mask is a standard component of the treatment of the trauma patient until the full scope of injury is known and resuscitation is complete. In the intubated patient, adequate ventilation should also be assured, generally targeting a $PaCO_2$ in the range of 35 to 40 mm Hg.

Circulation is assessed, initially, simply by observing the patient. If he or she is alert and oriented, their central nervous system is receiving adequate perfusion.
Observe skin color (skin color is often utilized as an indicator of perfusion but may be an unreliable end point for dark-skinned individuals) and feel the temperature of the skin. Is the skin warm and dry, or cool and diaphoretic?
Capillary refill, often judged at the thenar eminence or great toe, should be less than 2 seconds.

Figure 7. Capillary refill test

Palpate the radial pulse. Is it strong and regular, or weak and rapid? As a general guideline, a palpable radial pulse indicates a systolic pressure of at least 80 mm Hg. A femoral pulse indicates a systolic pressure of at least70 mm Hg. Finally, if only the carotid pulse is palpable, the systolic pressure is $\sim$ 60 mm Hg.

These observations give a rapid, albeit approximate, estimate of circulatory adequacy but do not rule out shock. Heart rate and blood pressure are more objective findings; however, ‘‘normal’’ vital signs do not always exclude hypoperfusion.

The severity of hemorrhage can be graded on a 1 - 4 scale on the physical signs ;

- Class I hemorrhage is characteristically associated with up to 15% blood volume loss and is manifested by no change in vital signs or minimal elevation of the heart rate. The pulse usually remains below 100 beats per minute (bpm).
- Class II hemorrhage is often considered the first level of shock, manifesting as a tachycardia, >100 bpm, and narrowing of the pulse pressure. Tachypnea is also seen in this stage. The systolic blood pressure (SBP) changes minimally up to ~ 30% blood volume loss.
- Class III hemorrhage, with 30 to 40% blood loss, is associated with the first significant drop in SBP (<100 mm Hg) and the heart rate generally exceeds 120 bpm. Oliguria, restlessness, and changes in skin perfusion are seen in this stage.
- Class IV hemorrhage (greater than 40% blood volume loss, often exceeding 2000 mL in adults) manifests more profound hypotension, a heart rate greater than 140 bpm, and lethargy.

*Table 1. Classification of hemorrhage*

| | **Class** | | | |
|---|---|---|---|---|
| **Parameter** | I | II | III | IV |
| **Blood loss (ml)** | <750 | 750–1500 | 1500–2000 | >2000 |
| **Blood loss (%)** | <15% | 15–30% | 30–40% | >40% |
| **Pulse rate (beats/min)** | <100 | >100 | >120 | >140 |
| **Blood pressure** | Normal | Decreased | Decreased | Decreased |
| **Respiratory rate (breaths/min)** | 14–20 | 20–30 | 30–40 | >35 |
| **Urine output (ml/hour)** | >30 | 20–30 | 5–15 | Negligible |
| **CNS symptoms** | Normal | Anxious | Confused | Lethargic |

*CNS = central nervous system

If profound shock is determined at this juncture of the primary survey, all other considerations become subordinate, and an immediate search to address the source of blood loss must be conducted.

The torso is able to sequester significant volumes of blood, and attention must focus upon potential injuries within the thorax, abdomen, and pelvis. In this scenario, all investigations and interventions should proceed in the trauma resuscitation bay, the operating room, or the interventional radiology (IR) suite.

Within the thorax, the clinical entities of tension pneumothorax, massive hemothorax, open pneumothorax, and cardiac tamponade must be ruled out or treated immediately. A combination of physical examination and plain anteroposterior (AP) chest radiographs often

lead to a diagnosis; however, bedside ultrasonography (i.e., focused assessment with sonography for trauma [FAST] exam) is increasingly finding utility.
Tube thoracostomy, pericardiocentesis, or possibly thoracotomy may be indicated to treat or control the etiology of the hypotension.
Long-bone fractures can also cause significant blood loss and, in the thigh in particular, do not achieve tamponade prior to significant blood loss. Reduction of these fractures helps to stem ongoing hemorrhage.
A traumatically amputated extremity or deep laceration extending to large blood vessels can result in fatal hemorrhage. However, direct pressure can often control these injuries until more definitive management can be undertaken. Placement of commercially available tourniquets to control major arterial hemorrhage has been validated in recent combat casualty care and is again considered an acceptable practice to initially control hemorrhage.Blind placement of hemostats on bleeding vessels should not be undertaken, due to the high risk of iatrogenic nerve injury leading to loss of function in the extremity.
A scalp injury can be the source of a tremendous amount of blood loss. Frequently, a scalp injury is initially covered with a dressing; however, topical application of gauze is usually not adequate to control scalp bleeding. Figure-of-eight suturing, approximation with skin staples, or the placement of Raney clips (that compress the edge of the scalp) may be required as temporizing measures to control bleeding vessels.

During the evaluation of circulation, the adequacy of vascular access, if previously established in the prehospital setting, should be reevaluated, and additional intravenous lines should be obtained as needed. The preferred method of vascular access utilizes large-bore (14 to 16 gauge) intravenous catheters that are ideally placed in the antecubital fossa. Central venous access is time-consuming, requires greater skill in the hypovolemic patient, and is fraught with potential complications. Furthermore, the length and diameter of central lines restrict the volume of resuscitative fluid that can be administered per unit time. Introducer sheath central venous catheters are a notable exception to this rule; however, their short length makes dislodgement more common. Systems for intraosseous infusion as well as venous cutdowns are employed when percutaneous access repeatedly fails.
Once large-bore intravenous access has been obtained, intravenous fluid resuscitation should be initiated with a bolus of 1 to 2 L of warm normal saline or lactated Ringer's solution. Hypertonic saline is controversial, although it may be initially valuable in patients eventually requiring massive transfusion.
It is important that hemostatic interventions (dressings, wound packing, tourniquets, and so on) are reevaluated as assessment and definitive management progresses. Rebleeding can occur with resuscitation, return of normal SBP, and loss of vasospasm.
The goals of the circulatory evaluation are to identify shock, limit ongoing hemorrhage, restore intravascular volume, and maintain oxygen delivery to end organs.

Following the assessment of circulation, a disability examination focusing on the neurological status of the patient is initiated. The three components of a Glasgow coma scale (GCS) score are determined: eye opening, verbal capacity, and motor score (Table 2).

*Table 2. Glasgow coma scale (GCS)*

| Glasgow Coma Scale | | | | | | |
|---|---|---|---|---|---|---|
| | **1** | **2** | **3** | **4** | **5** | **6** |
| **Eye** | Does not open eyes | Opens eyes in response to painful stimuli | Opens eyes in response to voice | Opens eyes spontaneously | N/A | N/A |
| **Verbal** | Makes no sounds | Incomprehensible sounds | Utters inappropriate words | Confused, disoriented | Oriented, converses normally | N/A |
| **Motor** | Makes no movements | Extension to painful stimuli (decerebrate response) | Abnormal flexion to painful stimuli (decorticate response) | Flexion / Withdrawal to painful stimuli | Localizes painful stimuli | Obeys commands |

Normality is indicated by a score of 15. Scores between 13 and 15 indicate a mild traumatic brain injury (TBI). Moderate TBI is represented by scores between 9 and 13. A comatose patient has a score of 8 and below. This indicates the presence of a severe TBI and the need to secure a definitive airway if not previously done for other indications. The lowest GCS possible is 3.
A second component of the neurological survey is the pupillary examination, noting relative size and reactivity. Both the GCS score and eye exam are determined with this aspect of the primary survey and are repeated as needed to follow the neurological status of the patient.

Once the traditional primary survey is complete, a series of adjunctive interventions are initiated. One of these is the FAST. This constitutes an abbreviated sonographic examination of the abdomen, including the subxiphoid region, the left- and right-upper quadrants, and the pelvis. The specific goal of the FAST examination is to rapidly evaluate the torso for evidence of hemorrhage. The FAST exam often begins with an examination of the pericardium to assess for intrapericardial fluid (blood) and early evidence of impending tamponade. The three areas of the abdomen most likely to accumulate intra-abdominal fluid are then evaluated. Sagittal views of Morrison's pouch (hepatorenal fossa) and the splenorenal recess are acquired. The third region is the pelvis, which is examined via a suprapubic transverse view. The sensitivity of this study depends on operator skill, patient body habitus, and severity of hemorrhage. The FAST exam requires at least 200 mL of intraperitoneal fluid to be present so that it can be visualized by ultrasound. As can be anticipated, the likelihood of a positive FAST is increased in patients with hemodynamic abnormality.

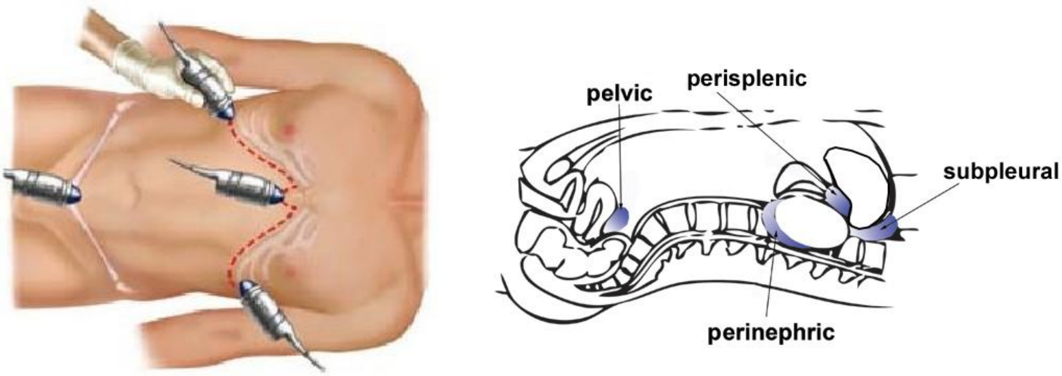



*Figure 8. FAST - focused assessment with sonography for trauma exam*

Immediately following the primary survey, a supine AP chest X-ray is obtained and evaluated. Some centers also routinely obtain AP pelvis films and cross-table lateral cervical spine films to screen for injuries within these regions. The utility of obtaining these studies in hemodynamically normal patients, who will undergo a computed tomography (CT) scan to further evaluate for injuries, is under debate.

Once the primary survey has been completed and immediately life-sustaining interventions have been accomplished, the secondary survey should be initiated. If the patient is unstable, the secondary survey should be abbreviated or eliminated altogether.

**The secondary survey** includes a brief history and systematic head-to-toe evaluation of the patient: the trauma-related history and physical examination. The ‘‘SAMPLE’’ history is obtained to determine signs and symptoms, allergies, mediations, past medical history, last oral intake, and events surrounding theinjury.

The entire patient is inspected and palpated in the course of the secondary survey. The head is examined for cranial deformities, crepitus, and tenderness as well as scalp lacerations. Pupillary function, first assessed in the D part of the primary survey, is again evaluated. Examination for eye injury includes an abbreviated visual acuity examination as well as gross inspection and evaluation of extraocular muscle function. Facial deformity, tenderness, and crepitus may indicate a facial fracture. Similarly, epistaxis, nasal septal deviation, or hematoma may be consistent with a nasal fracture. Cerebrospinal fluid discharge from the ears or nose, tympanic membrane rupture or hematoma, mastoid (Battle sign) and periorbital ecchymoses (Raccoon eyes) are consistent with a basilar skull fracture. Dental trauma, tenderness at the temporomandibular joint, and abnormal alignment of bite (malocclusion) may indicate a jaw fracture. The presence of bilateral mandibular fractures requires special attention as it may be associated with airway compromise due to loss of lingual support.

When inspecting the neck, inline cervical spine immobilization is maintained and the cervical spine is examined for tenderness and step-offs that could suggest a significant cervical spine injury. The position of the trachea is again inspected, and the cervical skin is palpated for crepitus. An evaluation for jugular venous distension is repeated, as well as inspection of wounds and hematomas that could indicate an underlying injury.

The chest is again inspected as was done during the primary survey, however, in more detail with respect to the clavicles, ribs, and shoulders.
The abdomen is inspected and palpated. Distension, tenderness, and muscular guarding or rigidity indicate developing peritonitis and may independently indicate the requirement for an exploratory laparotomy.
Ecchymoses and abrasions across the abdomen, as would be produced by a high-riding lap belt (‘‘seatbelt sign’’) with tenderness to deep palpation should heighten suspicion for hollow visceral injury. The pelvis is inspected by palpating and applying gentle pressure to the pubis, followed by simultaneous gentle pressure to both iliac wings, at the anterior superior iliac spines, assessing for pain and abnormal mobility. Pain and instability in the conscious patient suggest a pelvic fracture. These diagnostic maneuvers should not be vigorous or repetitive to avoid iatrogenic hemorrhage.
The urethral meatus is inspected for blood. The genitalia and perineum are inspected for hematomas or ecchymoses. A digital rectal examination evaluates for gross blood, rectal tone, and the position of the prostate.
If no blood is found at the urethral meatus and the prostate is not displaced superiorly, a Foley catheter is routinely placed in the severely injured patient. However, if blood is present at the meatus, a perineal ecchymosis is noted, or a Foley catheter does not pass with ease, a retrograde urethrogram is indicated. This will identify a urethral injury that may preclude blind insertion of a catheter.
If a severe pelvic fracture is suspected on physical examination or identified by plain film, a bedsheet wrap or commercially available pelvic binder should be placed and tightened to reduce pubic diastasis. The binder should be placed at or just above the greater trochanters. This reduces the volume of the pelvis, approximates fractures to prevent displacement and pain, reduces the risk of further vascular injury, and helps to tamponade the pelvic venous plexus.

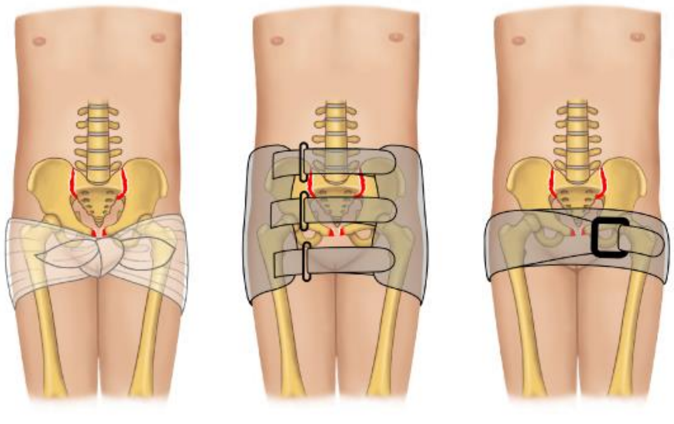


*Figure 9. Pelvis binders*

The thoracic, lumbar, and sacral spines are further inspected and palpated. The extremities are similarly examined and ranged to evaluate for injury. Distal pulses are palpated and suspected fractures are splinted.

Once the initial evaluation has been completed, to the level of detail appropriate for the patient's hemodynamic stability, a decision point has been reached. In the stable patient, further ancillary studies are routinely undertaken.
Laboratory analyses are routinely obtained including complete blood count, chemistry panel, coagulation panel, arterial blood gas, liver function tests, urinalysis, and toxicology screening. The advent of point-of-care testing renders the results of some of these laboratories available in time to assist in the acute decision making. If the patient's status allows, further detailed physical examination of injuries, plain radiographs of suspected fractures, and a 12-lead electrocardiogram, in patients with a cardiac history or risk factors, are completed.

A CT scan is an important adjunct to the physical examination and plain radiographs in the assessment and triage of the multiply injured trauma patient. It should be emphasized that movement to the CT suite is reserved for the hemodynamically stable patient, as access to the patient and resources allowing intervention for sudden destabilization are temporarily compromised.
An unenhanced CT scan of the head is obtained in patients with significant loss of consciousness, with a GCS <15, or with significant cranial trauma on physical examination. A facial CT scan is employed in a patient with evidence of facial trauma, based upon the secondary survey. A CT scan of the cervical spine is obtained in a patient with cervical spine tenderness to palpation, a depressed mental status, or significant distracting injury. Torso scans, including the chest, abdomen, and pelvis, are obtained to rule out great vessel, pulmonary, solid organ, hollow viscus, and bony injuries as dictated by mechanism and physical signs of trauma. Many busy trauma centers have adopted the routine use of combining multiple CT examinations (''pan-scanning'') in an effort to avoid missed injuries.
Most trauma patients who require the care of an interventional radiologist have undergone a primary and secondary survey. Through physical examination and imaging studies, injuries requiring the care of an interventional radiologist may be identified early in this initial evaluation. These include potentially devastating injuries, such as splenic and hepatic lacerations, great vessel disruptions, and pelvic fractures, all of which can lead to profound hemorrhage. These are usually heralded by active intravenous contrast extravasation, aneurysmal dilatation, or vascular dissection on screening CT studies. The interventional radiologist may find him- or herself involved in the management of these patients within minutes of injury identification, even while the trauma patient remains hemodynamically abnormal. Moreover, because of the need for emergent intervention to control hemorrhage, the interventional radiologist is often consulted in the care of critically injured trauma patients while the initial workup is incomplete.

Changes in patient status mandate expedient reevaluation, regardless of physical location. This rapid reassessment, initiated with a repetition of the primary survey, facilitates the identification of missed injuries or the failure of current management strategies to control problems that have already been identified.

The hemodynamically abnormal patient, who does not respond to initial resuscitation, will usually be taken emergently to the operating room to control hemorrhage. These patients will

likely undergo exploration of the body cavity or cavities with the highest probability of harboring the site of exsanguination.
This assessment is based on clinical suspicion, derived from the mechanism of injury, the superficial injury pattern, the FAST examination, AP chest and pelvis pelvic radiographs, the output of thoracostomy tubes, and, if performed, diagnostic peritoneal lavage. Though these patients may have been taken to the operating room, their ongoing hemorrhage may still require the assistance of the interventional radiologist because of the injury location and issues of inaccessibility.
Liver lacerations and hemorrhage from pelvic fractures are infamously difficult to control even with packing and damage control techniques. The patient who has had an emergent abdominal exploration or thoracotomy may be brought from the operating room directly to the IR suite for angiography and embolization based solely on the findings in the trauma room and in the operating room. However, if operating room support is adequate in terms of fluoroscopy, angiographic equipment, and technical training and, if patient status demands, the interventional radiologist can also perform selective embolization in the operating room.
This may be needed in the patient whose total trauma burden remains unknown and frequent reevaluation is requisite. Active resuscitation is usually proceeding for such patients. A hybrid operating room with fixed fluoroscopic equipment would be the ideal setting for a trauma laparotomy, which is complemented by an endovascular intervention. Alternatively, the use of a portable fluoroscopy unit (C-arm) may be the only means to accomplish the endovascular intervention.

The trauma patient who initially presents with hemodynamic instability and responds well to initial volume resuscitation but subsequently manifests recurrent hypotension (‘‘transient responder’’) is a patient who has persistent bleeding and may benefit significantly from interventional radiologic techniques. This patient is often temporarily stable enough to obtain CT scans that can demonstrate the injuries requiring intervention. The specific injuries are better defined, allowing angiography to be optimally targeted.

Blood product utilization is an area of trauma management where a widely accepted algorithm has not yet emerged. Generally, early trauma resuscitation is initiated with readily available crystalloid intravenous fluids: normal saline or lactated Ringer’s solution. If shock persists after 2 to 3 L of crystalloid resuscitation in the emergency department, packed red blood cell (PRBC) transfusions are initiated.
These are usually not type matched, in the face of time urgency, and are either type specific or, most often, O- for females and males, respectively. Traditionally, 4 to 6 units PRBC are transfused prior to the initiation of fresh frozen plasma, platelet, and cryoprecipitate units.
The optimal end point for intravascular resuscitation is also often obscure. Some have argued that permissive hypotensive resuscitation, with goals of SBP as low as 70 mm Hg, may be sufficient; however, this paradigm has only been applied to penetrating trauma. Generally, the goal of an SBP of at least 90 mm Hg is the goal of intravenous fluid and blood product resuscitation.
In the patient with persistent hemorrhage, with an ongoing transfusion requirement, the appropriate mix of PRBC, plasma, platelets, and cryoprecipitate is unknown.

Targeting correction of laboratory values such as partial thromboplastin time, prothrombin time, and international normalized ratio (INR), serum fibrinogen level, and platelet count and function is difficult due to the time delay between obtaining the specimen, processing, and reporting of the laboratory study. Point of care testing such as thromboelastography is not universally available and standardized.

Current literature supports increasing the relative quantity of plasma transfused with respect to each unit of PRBC. Combat casualty care experience, recently gleaned from the Iraq war, demonstrates a higher survival rate in patients transfused with plasma : PRBC ratio of 1:1 in distinction from those transfused with ratios of 1:2. However, a randomized prospective study has yet to be completed in the civilian trauma population where blunt trauma is the norm.

Despite the absence of consensus regarding hemostatic resuscitation, other traditional clinical targets remain less controversial. These include the directive to transfuse with blood products until massive bleeding has been definitively controlled. Resuscitation goals of mean arterial pressures approximating 65 mm Hg, heart rate <100 bpm, arterial oxygen saturation >94%, central venous pressure of 8 to 12 mm Hg, improved base deficit or lactate to near normal, mixed venous oxygenation saturation >70%, and urine output exceeding 0.5 mL/kg/h are commonly accepted.

Goals and corrections for coagulopathy, either demonstrated visually at the time of surgery or by laboratory values, have consensus. Examples include fresh frozen plasma for an INR >1.5, cryoprecipitate to maintain fibrinogen >100, and platelets to maintain the platelet count >50,000 *m*L.

Special consideration must be made to the patient’s temperature. Hypothermia is associated with worsened patient outcome and is identified as part of the deadly triad along with coagulopathy and acidosis.

Once the physical examination of the patient is complete, the patient should be covered with warm sheets and blankets. Optimal care would include warming blankets, warmed intravenous fluids and blood products, as well as heated ventilator circuits. This emphasis on warming is of the utmost importance while the patient is in the operating room where exposure and evaporation contribute to heat loss.

## 13. IMMOBILIZATION AND TRANSPORT DEVICES FOR TRAUMA PATIENTS

**Radmila Majhen-Ujević, M.D.**

RIGID CERVICAL COLLAR alone does not provide adequate immobilization; it simply aids in supporting the neck and promotes a lack of movement. It limits flexion by about 90% and limits extension, lateral bending and rotation by about 50%. It is an important adjunct to immobilization but must be used with manual stabilization or mechanical immobilization provided by a suitable spine-immobilization in field.

The unique primary purpose of a cervical collar is to protect the cervical spine from compression. Prehospital methods of immobilization (using a vest, shortboard or a long backboard device) still allow some slight movement of the patient and the spine because these devices only fasten externally to the patient and the skin and muscle tissue move slightly on the skeletal frame even when the patient is extremely well immobilized. Most rescue situations involve some movement of the patient and spine when extricating, carrying and loading the patient. This type of movements also occurs when an ambulance accelerates and decelerates in normal driving conditions.
An effective cervical collar sits on the chest, posterior thoracic spine and clavicle, and trapezius muscles, where the tissue movement is at a minimum. This still allows movement at C6, C7 and T1 but prevents compressions of these vertebrae. It must be the correct size for the patient and must be applied properly. It is not supposed to be comfortable but must not be too tight to cause breathing difficulties or to raise intracranial pressure.
It should be applied after bringing the patient's head into a neutral in-line position. If the head cannot be returned to a neutral in-line position (pain or resistance), collar cannot be applied and neck immobilization should be provided by using improvised devices – rolled blankets, sheets, towels….

Summary:
Rigid cervical collars:
1. Do not adequately immobilize by their use alone
2. Must be properly sized for each patient
3. Must not inhibit a patient's ability to open the mouth or the care provider's ability to open the patient's mouth if vomiting occurs
4. Should not obstruct or hinder ventilation in any way

LONG SPINAL BACKBOARD - The traditional spinal immobilisation device is simply a flat board which can have an attached dedicated triple immobilisation system. Most devices cover up to 2 m and come in various widths and thicknesses. It is versatile and can be used for rapid take downs of standing or sitting casualties as well as prone, supine or irregular casualties. Long backboards are commonly used to attempt to provide rigid spinal immobilization among EMS trauma patients. Appropriate patients to be immobilized with a backboard may include those with:
- Blunt trauma and altered level of consciousness;
- Spinal pain or tenderness;
- Neurologic complaint (e.g., numbness or motor weakness)
- Anatomic deformity of the spine
- High energy mechanism of injury and:
- Drug or alcohol intoxication
- Inability to communicate
- Distracting injury.

Patients for whom immobilization on a backboard is not necessary include those with all of the following:

- Normal level of consciousness (GCS 15);
- No spine tenderness or anatomic abnormality;
- No neurologic findings or complaints;
- No distracting injury;
- No intoxication.

The long backboard can induce pain, patient agitation, and respiratory compromise. Further, the backboard can decrease tissue perfusion at pressure points, leading to the development of pressure ulcers. Low grade ulcers can appear in as few as two hours and even healthy persons complain on pain after 30 minutes.  Utilization of backboards for spinal immobilization during transport should be judicious, so that potential benefits outweigh risks. Patients with penetrating trauma to the head, neck or torso and no evidence of spinal injury should not be immobilized on a backboard.  Spinal precautions can be maintained by application of a rigid cervical collar and securing the patient firmly to the EMS stretcher, and may be most appropriate for:

- Patients who are found to be ambulatory at the scene;
- Patients who must be transported for a protracted time, particularly prior to

interfacility transfer; or

- Patients for whom a backboard is not otherwise indicated.

Two major adjustments in the previous methods are necessary when immobilizing a small child to a long board.

• Due to the relatively large size of the child's head, padding is needed under the torso to elevate it and maintain the spine in neutral alignment. The padding must extend form the lumbar area to the top of the shoulders, and to the right and left edges of the board. A folded blanket usually works well.

• Small children are usually narrower than an adult sized board. Blanket rolls can be placed between the child's sides and the sides of the board to prevent lateral movement. Pediatric immobilization devices take these differences into account, and are preferable.

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Patients should be removed from backboards as soon as practical in an emergency department.

SPLIT DEVICES are popular in prehospital circles as they can avoid a log roll in supine casualties or help extricate from a vehicle.These include the aluminium orthopaedic scoop stretcher and the Ferno 65 XL (modern scoop). However, it is designed as a transfer device and should not be carried for any distance. Those who are not supine will still need to be rolled. [

VACUUM MATTRESS: Perhaps the gold standard device in true spinal cord injury, the vacuum mattress provides an individually moulded cocoon for the casualty through a double bagged polystyrene ball system which becomes rigid when the air is removed. They come in a range of sizes and widths and avoid the problems of local pressure areas as the force is evenly spread out along the whole body. However, if they puncture, the valve fails or the pump is lost, then they become of little value. Hence they should always be used with a backup device available. They are becoming more and more popular with the ambulance service as the device of choice, especially at pregnant women and at transport that lasts longer than 30 minutes.

KENDRICK EXTRICATION DEVICE (KED)

It is an immobilization vest and it is used when spinal stabilization is indicated for a sitting trauma patient without life-threatening conditions, usually in a vehicle extrication. Typically used in conjunction with a cervical collar, the KED is a semi-rigid brace that secures the head, neck and torso in an anatomically neutral position.

It is important that manual –in –line stabilisation is always performed before applying other devices.The techniques we use to put the patients on transport devices are log-roll (LR), lift-slide (LS), lateral trauma position (LTP)

Some other issues on immobilization and transport:

- Pregnant women in left lateral position with an angle till 30 degrees
- Suspect traumatic brain injury with head elevated till 30 degrees
- Anti-shock position with elevated legs
- If the child has no life-threatening injuries it may be better to transport the child in their child safety seat
- Torso and legs are always fastened before head
- Removal of the helmet is necessary to assess the patient and to move the head into neutral alignment
- Log- roll should be avoided in suspected pelvis and two long bones injury

Literature:

1. McSwain NE Jr, ed. Prehospital Trauma Life Support. 7. ed. St. Louis:Mosby JEMS Elsevier;2011