EXAMINATION QUESTIONS IN ANESTHESIOLOGY AND REANIMATOLOGY FOR 4th YEAR STUDENTS OF MEDICAL FACULTY

40.Main principles and ways of Basic Life Support (BLS).

Basic Life Support (BLS) is a temporary delivery of oxygen to vital tissues is accomplished by providing effective airway management, ventilation, and artificial circulation, with or without supportive equipment.

Basic Life Support includes the following steps:

– A — Airway opening;

– B — Breathing;

– C — Circulation.

Airway opening. Obstruction of the hypopharynx by the base of the tongue is the most common cause of airway obstruction in the unconscious persons. The unsupported tongue falls against the posterior pharyngeal wall, obstructing the airway.

Hypopharyngeal obstruction by the base of the tongue can occur regardless of patient's position.

Airway opening "Head tilt — chin lift" maneuver. The cardinal principle of opening the airway is anterior displacement of the mandible and elevation of the tongue from the posterior pharyngeal wall. This may be accomplished by chin lift, neck lift, jaw thrust, or head hyperextension (so-called "triple method of Safar").

Airway management. The jaw thrust without head tilt and hyperextension is the preferred method for opening the airway in a patient with a cervical spine injury

The so-called "Safar triple method" to provide straight open airway includes:

– tilting the victim's head (do not overtilt, the position is supposed to be as if one is "scenting the morning air";

– lifting the victim's mandible;

– opening the victim's mouth.

***Breathing types*:**

– Mouth-to-Mouth;

– Mouth-to-Nose.

How to provide breathing:

- Pinch closed the soft part of the nose, using the index finger and thumb of your hand on the forehead.
- Allow the mouth to open, but maintain the chin lift.
- Take a normal breath and place your lips around his mouth, making sure that you have a good seal.
- Blow steadily into his mouth while watching for his chest to rise.
- Taking about 1 s as in normal breathing.
- Tidal volume of 400–500 ml.
- Take your mouth away from the victim and watch for his chest to fall as air comes out.

Successful resuscitation depends on rescucitator ensuring adequate ventilation with every breath by using criteria such as observing the patient's chest rise and fall, feeling the patient's lung compliance during lung inflation, and hearing and feeling the air escape during ventilation.

The volume of air, required for each inflation to be been quoted as
800–1200 ml, with each breath taking 1–1.5 s. It has been shown recently that
a tidal volume of 400–500 ml is sufficient to provide adequate ventilation in adults BLS because carbon dioxide delivery during cardiac arrest is very low.

***Circulation*.** Checking the carotid pulse (or any other pulse) is an inaccurate method to confirm the presence or absence of circulation

Circulation (Chest compression). Start chest compression as follows:

– Place the heel of one hand in the centre of the victim's chest.

– Place the heel of your other hand on the top of the first hand.

– Interlock the fingers of your hands and ensure that pressure is not applied over the victim's ribs.

– Do not apply any pressure over the upper abdomen or the bottom end of
the bony sternum.

Chest compression:

- position yourself vertically above the victim's chest and press down on
the sternum at least 5 cm (but not exceeding 6 cm);
- after each compression, release all the pressure on the chest without losing contact between your hands and the sternum; repeat at a rate of at least 100 min^{-1} (but not more than 120 min^{-1});
- minimize interruptions in chest compression in order to ensure the victim receives at least 60 compressions each minute;
- do not rely on feeling the carotid or other pulse as a sign of effective arterial flow during chest compressions.

Note: The Compression-ventilation ratio is 30:2!

Combine rescue breathing and compression: after 30 compressions tilt
the head, lift the chin and give two effective breaths; return your hands immediately to the correct position on the sternum and give another
30 compressions, continuing compressions and breaths with a ratio of 30:2.

Recovery position. There are several variations of the recovery position, each with its own advantages. No single position is perfect for all victims.

The position should be stable, near to a true lateral position with the head dependent, and with no pressure on the chest to impair breathing.

To complete the recovery position keep the head tilted to open the airway and the face down to allow fluids to go out.

BLS consists of the following steps (sequence of actions)

1. Make sure that you, the victim and any bystanders are safe.

2. Check the victim for a response. Gently shake his shoulders and ask loudly: “Are you all right?”

3a. If he responds:

– leave him in the position in which you found him, provided there is no further danger;

– try to find out what is wrong with him and get help if needed;

– reassess him regularly.

3b. If he does not respond:

• shout for help:

– turn the victim onto his back and then open the airway using head tilt and chin lift;

– place your hand on his forehead and gently tilt his head back;

– with your fingertips under the point of the victim’s chin, lift the chin to open the airway.

4. Keeping the airway open, look, listen and feel for breathing:

– look for chest movement;

– listen at the victim’s mouth to breath sounds;

– feel for air on your cheek;

– check if breathing is normal, not normal or absent.

In the first few minutes after cardiac arrest, a victim may be barely breathing, or taking infrequent, slow and noisy gasps. Do not confuse this with normal breathing. Look, listen and feel for no more than 10 s to determine whether
the victim is breathing normally. If you have any doubt whether breathing is normal, act as if it is not normal.

5a. If he is breathing normally:

– turn him into the recovery position;

– send or go for help — call 112 or local emergency number for an ambulance;

– continue to control breathing.

5b. If the breathing is not normal or absent:

• send someone for help and someone to find and bring an AED if available;

or if you are on your own, use your mobile phone to alert the ambulance service — leave the victim only when there is no other option;

• start chest compression as follows:

– kneel by the side of the victim;

– place the heel of one hand in the centre of the victim's chest (which is
the lower half of the victim's breastbone (sternum));

– place the heel of your other hand on top of the first hand;

– interlock the fingers of your hands and ensure that pressure is not applied over the victim's ribs. Keep your arms straight. Do not apply any pressure over
the upper abdomen or the bottom end of the sternum;

– position yourself vertically above the victim's chest and press down on
the sternum at least 5 cm (but not exceeding 6 cm);

– after each compression, release all the pressure on the chest without losing contact between your hands and the sternum; repeat at a rate of at least 100 min^{-1} (but not exceeding 120 min^{-1});

– compression and release should take equal amounts of time.

6a. Combine chest compression with rescue breaths:

• After 30 compressions open the airway again using head tilt and chin lift.

• Pinch the soft part of the nose closed, using the index finger and thumb of your hand on the forehead.

• Allow the mouth to open, but maintain chin lift.

• Take a normal breath and place your lips around his mouth, making sure that you have a good seal.

• Blow steadily into the mouth while watching for the chest to rise, taking about 1 s as in normal breathing; this is an effective rescue breath.

• Maintaining head tilt and chin lift, take your mouth away from the victim and watch for the chest to fall as air comes out.

• Take another normal breath and blow into the victim's mouth once more to achieve a total of two effective rescue breaths. The two breaths should not take more than 5 s in all. Then return your hands without delay to the correct position on the sternum and give a further 30 chest compressions.

• Continue with chest compressions and rescue breaths in a ratio of 30:2.

• Stop to recheck the victim only if he starts to wake up: to move, opens eyes and to breathe normally. Otherwise, do not interrupt resuscitation.

If your initial rescue breath does not make the chest rise as in normal breathing, then before your next attempt:

– look into the victim's mouth and remove any obstruction;

– recheck that there is adequate head tilt and chin lift;

– do not attempt more than two breaths each time before returning to chest compressions.

Do not interrupt resuscitation until:

– professional help arrives and takes over;

– the victim starts to wake up: to move, open eyes and to breathe normally;

– you become exhausted.

If there is more than one rescuer present, another rescuer should take over delivering ***CPR every 2 min*** to prevent fatigue.

41.Airway management. The Safar triple method.

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42.Artificial lung ventilation with the application of the elementary methods.

How to provide breathing:

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- Allow the mouth to open, but maintain the chin lift.
- Take a normal breath and place your lips around his mouth, making sure that you have a good seal.
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800–1200 ml, with each breath taking 1–1.5 s. It has been shown recently that
a tidal volume of 400–500 ml is sufficient to provide adequate ventilation in adults BLS because carbon dioxide delivery during cardiac arrest is very low.

43.Cardiac massage. Methods, complications. Indicators of resuscitation efficiency.

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– Place the heel of your other hand on the top of the first hand.

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– Do not apply any pressure over the upper abdomen or the bottom end of
the bony sternum.

Chest compression:

- position yourself vertically above the victim's chest and press down on
the sternum at least 5 cm (but not exceeding 6 cm);

- after each compression, release all the pressure on the chest without losing contact between your hands and the sternum; repeat at a rate of at least 100 min^{-1} (but not more than 120 min^{-1});
- minimize interruptions in chest compression in order to ensure the victim receives at least 60 compressions each minute;
- do not rely on feeling the carotid or other pulse as a sign of effective arterial flow during chest compressions.

Note: The Compression-ventilation ratio is 30:2!

44.Main principles of Advanced Life Support (ALS).

Advanced Cardiac Life Support (ACLS)

– D — Drugs and Fluids;

– E — ECG control;

– F — Fibrillation treatment.

Airway and ventilation. Tracheal intubation provides the most reliable airway, but should be attempted only if the healthcare provider is properly trained and has regular, ongoing experience with the technique.

After intubation, confirm correct tube position and secure it adequately. Ventilate the lungs at 10 breaths min^{-1}; do not hyperventilate the patient. Once
the patient's trachea has been intubated, continue chest compressions, at a rate of 100 min^{-1} without pausing during ventilation.

In the absence of personnel skilled in tracheal intubation, a supraglottic airway device is an acceptable alternative. Once a supraglottic airway device has been inserted, attempt to deliver continuous chest compressions, uninterrupted during ventilation.

Additional devices (adjuncts) to basic airway techniques:

– Oropharyngeal airways;

– Nasopharyngeal airways;

– Facemask;

– Tracheal tube;

– Laryngeal mask airway (LMA);

– Combitube;

– I-gel.

45.Drugs used during the treatment of cardiac arrest. Routes for drug delivery.

Drugs used during the treatment of cardiac arrest:

- Adrenaline (epinephrine).
- Anti-arrhythmics:

– Amiodarone;

– Lidocaine;

– Magnesium.

- Other drugs:

– Atropine;

– Calcium;

– Buffers.

Routes for drug delivery. Establish **intravenous access** if this has not already been achieved. Peripheral venous cannulation is quicker, easier to perform and safer than central venous cannulation. Drugs injected peripherally must be followed by a flush of at least 20 ml of fluid. If intravenous access is difficult or impossible, consider the IO route.

Intraosseous injection of drugs achieves adequate plasma concentrations in a time comparable with injection through a central venous catheter. The recent availability of mechanical IO devices has increased the ease of performing this technique.

Unpredictable plasma concentrations are achieved when drugs are given via a tracheal tube, and the optimal tracheal dose of most drugs is unknown, thus, the tracheal route for drug delivery is no longer recommended.

Intravenous fluids. Hypovolaemia is a potentially reversible cause of cardiac arrest. Infuse fluids rapidly if hypovolaemia is suspected.

Use intravenous fluid to flush peripherally injected drugs into the central circulation. Infuse fluids rapidly if hypovolaemia is suspected.

Use 0.9 % sodium chloride or Hartmann's solution.

Avoid dextrose, which is redistributed away from the intravascular space rapidly and causes hyperglycaemia

46.Methods of cardiac defibrillation. Indications, contraindications.

Delivery of defibrillation:

- Give one shock (360 J monophasic or 150–200 J biphasic).
- Without rhythm reassessment or feeling for a pulse, resume CPR (CV ratio 30:2) immediately after the shock, starting with chest compressions.
- Continue CPR for 2 min, then pause briefly to assess the rhythm; if still VF/VT persists, give a second shock (360 J monophasic or 150–360 J biphasic).

47.Criteria for CPR cessation. Clinical signs of 'brain death'.

The **purpose of cardiopulmonary resuscitation (CPR)** during cardiac arrest is restoration of blood circulation and breathing.

CPR Components are the following:

– Basic Life Support (BLS);

– Advanced Cardiac Life Support (ACLS);

– Post-resuscitation Intensive Care (PIC).

Some of the signs of brain death include:

- The pupils don't respond to light.
- The person shows no reaction to pain.
- The eyes don't blink when the eye surface is touched (corneal reflex).
- The eyes don't move when the head is moved (oculocephalic reflex).
- The eyes don't move when ice water is poured into the ear (oculo-vestibular reflex).
- There is no gagging reflex when the back of the throat is touched.
- The person doesn't breathe when the ventilator is switched off.
- An electroencephalogram test shows no brain activity at all.

48.Post-cardiac arrest syndrome: pathologic physiology, intensive care. Complications, their prevention and treatment.

Post-cardiac arrest syndrome is a unique and complex combination of pathophysiological processes, which include: 1) post-cardiac arrest brain injury;
2) post-cardiac arrest myocardial dysfunction; 3) systemic ischemia/reperfusion response.

This state is often complicated by a fourth component: the unresolved pathological process that caused cardiac arrest.

Pathophysiology of Post-Cardiac Arrest Syndrome. The high mortality rate of patients who initially achieve ROSC after cardiac arrest can be attributed to a unique pathophysiological process that involves multiple organs.

The 4 key components of post-cardiac arrest syndrome are: 1) post-cardiac arrest brain injury; 2) post-cardiac arrest myocardial dysfunction; 3) systemic ischemia/reperfusion response; 4) persistent precipitating pathology.

Clinical manifestations of post-cardiac arrest brain injury include coma, seizures, myoclonus, various degrees of neurocognitive dysfunction (ranging from memory deficits to persistent vegetative state), and brain death.

Monitoring. Post-cardiac arrest patients generally require intensive care monitoring. This can be divided into 3 categories: general intensive care monitoring, more advanced hemodynamic monitoring, and cerebral monitoring.

Early hemodynamic optimization or early goal-directed therapy is an algorithmic approach to restoring and maintaining the balance between systemic oxygen delivery and demands.

The goals in these studies have included a central venous pressure of 8 to 12 mm Hg, MAP of 65 to 90 mm Hg, $ScvO_2$ 70 %, hematocrit 30 % or hemoglobin 8 g/dL, lactate 2 mmol/L, urine output 0.5 $mL \cdot kg^{-1} \cdot h^{-1}$, and oxygen delivery index 600 $mL \cdot min^{-1} \cdot m^{-2}$.

The primary therapeutic tools are intravenous fluids, inotropes, vasopressors, and blood transfusion.

The balance between systemic oxygen delivery and consumption can be monitored indirectly with mixed venous oxygen saturation (SvO_2) or $ScvO_2$.
The optimal $ScvO_2$ goal for post-cardiac arrest patients has not been defined by prospective clinical trials, and the value of continuous $ScvO_2$ monitoring remains to be demonstrated.

Lactate concentrations are elevated early after ROSC because of the total-body ischemia of cardiac arrest. This limits the usefulness of a single measurement during early hemodynamic optimization.

On the basis of the limited available evidence, reasonable goals for post-cardiac arrest syndrome include an MAP of 65 to 100 mm Hg (taking into consideration the patient's normal blood pressure, cause of arrest, and severity of any myocardial dysfunction), central venous pressure of 8 to 12 mm Hg, $ScvO_2$ — 70 %, urine output — 1 $mL \cdot kg^{-1} \cdot h^{-1}$, and a normal or decreasing serum or blood lactate level.

Hemodynamic instability is common after cardiac arrest and manifests as dysrhythmias, hypotension, and low cardiac index. Underlying mechanisms include intravascular volume depletion, impaired vasoregulation, and myocardial dysfunction.

Dysrhythmias can be treated by maintenance of normal electrolyte concentrations and use of standard drug and electrical therapies.

The first-line intervention for hypotension is to optimize right-heart filling pressures by use of intravenous fluids.

Inotropes and vasopressors should be considered if hemodynamic goals are not achieved despite optimized preload.

CAD is present in the majority of out-of-hospital cardiac arrest patients, and acute myocardial infarction is the most common cause of sudden cardiac death.

Other causes of out-of-hospital cardiac arrest include pulmonary embolism, sepsis, hypoxemia, hypovolemia, hypokalemia, hyperkalemia, metabolic disorders, accidental hypothermia, tension pneumothorax, cardiac tamponade, toxins, intoxication, and cerebrovascular catastrophes.

Hyperventilation should be avoided in the post-cardiac arrest.

Therapeutic hypothermia should be part of a standardized treatment strategy for comatose survivors of cardiac arrest.

49.Phases of the postoperative period. Post-operative intensive care.

In terms of its duration, the postoperative period (POP) encompasses
the period starting from the end of the operation up to the patient's complete recovery or his/her recognition being disabled.

The stages of the postoperative period are identified as follows:

– **early postoperative period** (from the end of surgery till the moment of
the patient discharge from hospital);

– **late postoperative period** (from the moment of the patient discharge till the time of his complete recovery fully or his recognition of being disabled).

Both surgery and anesthesia bring about particular pathological changes in the body, which are the manifestations of the body's response to the surgical trauma. This induces defensive mechanisms to eliminate the consequences of
the trauma and therefore restore homeostasis. The surgery affects the balance in the rate of metabolic (anabolic and catabolic) reactions rather than launches new metabolic events.

The postoperative state is divided into the four phases:

– catabolic phase;

– resolution phase;

– anabolic phase;

– the body weight gain phase.

The catabolic phase lasts from three to seven days. The period tends to be more acute when the serious changes in the body are caused by the severity of
the principal condition or the extent and hazards of the surgery itself.
The catabolic stage may also be prolonged and worsened if postoperative complications develop. These include the following:

– bleeding;

– infection;

– hypovolaemia;

– fluid, electrolyte, and acid-base balance disorders.

The improper management (e.g. inadequate analgesia or nutritional support, parenteral nutritional support, pulmonary hypoventilation) is known to contribute to prolonged catabolic phase as well.

The catabolic phase is a defensive body reaction aimed at enhancing
the resistance of the body by prompt transfer of the energetic and plastic materials to the vital organs. It is characterised by specific neuroendocrine reactions: activating the sympathetic nervous system and adrenals, hypothalamus and pituitary, intensive synthesis with subsequent release of catecholamines, glucocorticoids, aldosterone and ACTH into the blood. This results in an increase in the amount of glycogen in the circulation, accompanied by a fall in insulin blood levels.
Furthermore, intensive synthesis of angiotensin and renin occurs.

The neurohumoral disturbances alter the vascular tone to cause vascular spasm and defects in microcirculation and tissue perfusion, which, in its turn, leads to hypoxia, metabolic acidosis, electrolyte imbalance, fluid redistribution,
an increase in blood viscosity and blood cellular stasis. This further affects
the extent of disturbance in the tissue redox processes that take place in
the acceleration of anaerobic glycolysis as a result of tissue hypoxia.
The myocardium, liver and kidneys are therefore the first to be affected.

The catabolic phase also involves accelerated protein break-down which is manifested by the decrease in muscle and connective tissue protein, and,
the depletion in enzymes. The proteolysis in the liver and digestive tract is
the fastest to occur, while that in the striated muscles takes significantly longer time to complete.

The resolution phase commonly lasts from **4 to 6 days** and is a gradual transition from the catabolic phase to anabolic one. This period is characterised by the reduction in the overactivity of the sympathetic nervous system and adrenals, including slowing down catabolism which becomes evident as a decrease
in nitrogen urinary excretion to as low as 5–8 g/24 hours (as compared with
15–20 g/24 hours in the catabolic phase).

The positive nitrogen balance (the amount of nitrogen excreted is less than the given amount) suggests improvement in protein metabolism. In this period, potassium urinary excretion decreases as the electrolyte starts accumulating to take part in the synthesis of protein and glycogen. The fluid and electrolyte balance is restoring.

As far as the autonomous nervous and endocrine systems are concerned,
the parasympathetic activity predominates and blood growth hormone (GH) level is rising, as are those of insulin and androgen.

During **the resolution phase**, the increased waste of energy and plastic materials (protein, fat, carbohydrates) slows down. When this eventually fades away, the active synthesis of protein and glycogen starts with subsequent production of fat, which rises as the intensity of catabolism falls. The persistent predominance of anabolism over catabolism is a sign of the postoperative period transfer to the anabolic phase.

The resolution phase starts from **the 3rd to 7th days** after the surgery if the postoperative period is uneventful (i.e. without complications).

The signs suggestive of the resolution stage onset are generally as follows:

– absence of pain;

– normalization of body temperature;

– resumption of appetite.

In addition, patients become active, and their body functions restore (the skin colour returns to normal; breathing becomes deep and its rate reduces; the heart rate returns to the preoperative one; peristaltic bowel sounds and flatus passage resume).

The anabolic phase is characterized by an increase in the synthesis of protein, glycogen, and fat, depleted during the operation and the postoperative catabolic period.

Furthermore, the parasympathetic nervous system tends to be overactive. Similarly, the secretion of anabolic hormones (GH and androgens) increases to allow the protein synthesis. GH, for instance, is responsible for the transport of amino acids from the intercellular space to the cells, while androgens promote the synthesis of proteins in the liver, kidneys, and myocardium directly.

Clinically, the anabolic phase is, in fact, the period of recovery, restoration of the impaired functions of the cardiac, respiratory, excretory, digestive and nervous systems. During this phase, the patient's general condition improves, appetite increases, the heart beat and rate return to normal as do the blood pressure levels, and the digestive functions (food passage, intestinal absorption and spontaneous bowel movements) are restored.

The anabolic phase usually lasts for **2–5 weeks**, which is dependent on the extent of the surgery, the patient's preoperative state and the severity and duration of the catabolic phase.

Phase of body weight gain (3–6 months). This phase of the postoperative period ends with an increase in weight, which occurs after 3–4 weeks and continues till full recovery, which sometimes can take several months.

Postoperative intensive care

Immediately after the operation the patient is transferred either to the ward or to the Intensive Care Unit (ICU) which are arranged to monitor the patients and, if needed, to provide them with emergency and intensive care.

Patients may be admitted to the ICU after surgery, either electively or after unexpected peri-operative complications.

Indicatons for elective ICU admission:

– airway monitoring: e.g. major oral, head and neck surgery;

– respiratory monitoring: e.g. cardiothoracic surgery, upper abdominal surgery, prolonged anesthesia, previous respiratory disease;

– cardiovascular monitoring: e.g. cardiac surgery, vascular surgery, major abdominal surgery, prolonged anesthesia, previous cardiovascular disease;

– neurological monitoring: e.g. neurosurgery, cardiac surgery with
a circulatory arrest;

– elective ventilation: e.g. cardiac surgery, major abdominal surgery, prolonged anesthesia, previous respiratory disease;

– patients may be admitted to the ICU after surgery, either electively or after unexpected peri-operative complications.

50.Importance of a painful syndrome in the mechanism of functional disorders development in the postoperative period. Pain Management.

Management of pain. The degree of tissue damage is related to
the magnitude of the pain stimulus. The site of injury is also important; thoracic and upper abdominal injury is more painful than injury elsewhere. However,
the perception of pain is dependent on other factors, e.g. simultaneous sensory input, personality, cultural background and previous experiences of pain.

***Systemic analgesia*:**

– Opioid analgesics form the mainstay of analgesic drug treatment in intensive care.

– Small, frequent IV doses or a continuous infusion provide the most stable blood levels. Since the degree of analgesia is dependent on blood levels it is important that they are maintained.

– Higher doses are required to treat rather than prevent pain.

– The dose of drug required for a particular individual depends on his perception of pain and tolerance built up after previous analgesic use.

– The use of non-opioid drugs allows the doctors to reduce the dose of opioid drugs or even avoid the opioids. This includes paracetamol and non-steroidals, ketamine and α2-agonists such as clonidine and dexmedetomidine.

***Regional analgesia*:**

– Regional techniques reduce respiratory depression but require experience to ensure safety procedures.

– Epidural analgesia may be achieved with local anaesthetic agents or opioids.

– Opioids allow to avoid vasodilatation and hypotension associated with local anaesthetic agents but do not produce as profound analgesia.

– The combination of opioid and local anaesthetic is synergistic.

– Intravenous opioids should be avoided or close monitoring should continue for 24 h after cessation of epidural opioids due to the potential for late respiratory failure.

– Local anesthetic agents may be used to block superficial nerves, e.g. intercostal nerve block with 3–5 ml 0.5 % bupivacaine plus adrenaline.

Non-pharmacological techniques. Adequate explanation, positioning and physical techniques may all reduce drug requirements.

51.The basic forms of acid-base balance disorders, pathophysiology. Clinical manifestations, correction principles.

The answer of this question is so extended. Should read all first and answer is based of understanding of the topic.

Acid base calculation. Rules and practical application

An overview of the six sequential steps involved is outlined. A check of pH, pCO_2 & HCO_3 against the Henderson-Hasselbalch equation is usually difficult without a calculator. However, a quick check of the logical consistency of
the results is often possible. For example, pH must be less then 7.4 if PCO_2 is high & HCO_3 is low. It is preferable to review the result print-out from the machine.

The six steps of systematic acid-base evaluation:

1. **pH**: assess the net deviation of pH from normal.

2. **Pattern**: check the pattern of bicarbonate & pCO_2 results.

3. **Clues**: check for additional clues in other investigations.

4. **Compensation**: assess the appropriateness of the compensatory response.

5. **Formulation**: bring the information together and make the acid base diagnosis.

6. **Confimation**: consider if any additional tests to check or support
the diagnosis are necessary or available & revise the diagnosis if necessary.

The **first step** is to look at the arterial pH (table 2). A net acidaemia means that acidosis must be present. A net alkalaemia means that alkalosis must be present. A normal pH gives 2 possibilities: no acid-base disorder or a mixed disorder with alkalosis compensating acidosis.

The **next step** is to determine whether any disorder is of the respiratory or metabolic type by reviewing the pattern and magnitude of the bicarbonate and pCO_2 results. If the disorder is minor (i.e. only one primary disorder present) then the acid-base disorder is diagnosed at this step. But the real problem is difficult to define, so a mixed disorder must always be checked. This is an important part of steps 2, 3 and 4.

Step 3 involves reviewing of other results looking for specific evidence of particular disorders.

The 4th step is to assess acid-base compensation. The approach discussed here involves the application of six rules. Much of the emphasis is made here to pick the presence of a second acid-base disorder.

Step 5: this stage is reached when overall acid-base assessment can be made.

Step 6: Sometimes the diagnosis suggests additional tests that can be used to confirm the diagnosis or at least allows doctors to make a more precise diagnosis (eg. measurement of the blood salicylate level in a child. In case it is high it can confirm a clinical suspicion of a salicylate overingestion). If a diagnosis of renal tubular acidosis is suspected then further specific tests should be done to specify further diagnosis.

The method of acid-base disorders assessment uses a set of six rules which are used primarily to check the magnitude of the patient's compensatory response. The rules should always be kept in mind — with practice this is not difficult.

A full assessment of blood-gas results must be based on a clinical assessment of each patient and understanding of the pathophysiology of the clinical conditions underlying the acid-base disorder. Do not interpret the blood-gas results as
an intellectual exercise in itself. It is only one part of the overall assessment and management process.

Diagnosing a "metabolic acidosis", for example, by itself, is often of little clinical use. What is really required is a more specific diagnosis of the metabolic acidosis cause (e.g. diabetic ketoacidosis, acute renal failure, lactic acidosis) to initiate the appropriate management. The acid-base analysis must be interpreted and managed in the context of the overall clinical picture.

Metabolic acidosis

Metabolic acidosis is a common component of a critical illness. Evaluation of this component can aid in making diagnosis, assessing the severity (and
the likely outcome) and allow the clinician to determine whether current treatment is adequate.

Metabolic acidosis (a low pH in the tissue) exists when there is an excess level of fixed or exogenous acids in the body. Fixed acids include hydrochloric acid, sulphuric acid, phosphoric acid, ketoacids and lactic acid. Examples of exogenous acids are salicylate and methanol.

Metabolic acidosis is accompanied by a drop in plasma bicarbonate concentration (relative to the bicarbonate concentration present prior to the onset of the acidosis). This drop in bicarbonate can either be caused by bicarbonate loss or by the presence of extra acid.

When evaluating a critically ill patient with a metabolic acidosis it is necessary to determine the type of acidosis to identify the cause of acidosis.

To classify metabolic acidosis it is useful to calculate the anion gap and, if present, the size of the osmolar gap.

The role of the anion gap

The anion gap is defined as the concentration difference between the major measured cations (positively charged ions) and anions (negatively charged ions) within the plasma (normally from 12 to 18 mmol/l)

Anionic proteins, phosphate, sulphate and low levels of organic acids, which are not measured, account for the difference (i.e. the "gap"). When examining
the cause of a metabolic acidosis it is useful to calculate the anion gap.

Anion gap = $[Na^+ + K^+] - [HCO_3^- + Cl^-] = 15(\pm 3)$ mmol/L^{-1}.

A normal anion gap implies that acidosis occurs due to primary bicarbonate loss:

• Plasma bicarbonate is low (the hallmark of acidosis) and chloride concentration is raised.

• This bicarbonate loss may be:

– gastrointestinal (diarrhoea, fistula);

– renal (renal tubular acidosis, drug effect).

• Also occurs with rapid intravenous infusion of normal saline (excess chloride) or intravenous nutrition rich in cationic amino acids (e.g. arginine).

An increased anion gap implies that fixed acids are being retained or an abnormal organic acid is present.

• Plasma bicarbonate is low and chloride concentration is normal.

• Fixed acids may be retained in:

– uraemia;

– ketoacidosis (diabetic, alcoholic);

– lactic acidosis.

• If fixed acids are normal, exogenous acids should be considered:

– salicylate (aspirin) poisoning;

– methanol poisoning;

– ethylene glycol poisoning.

Compensation for metabolic acidosis

When treating critically ill patients with metabolic acidosis, it is important to consider the adequacy of their ventilatory response to acidosis when deciding on treatment priorities. Buffering provides the main means of accommodating
a metabolic acidosis.

As buffering capacity is exceeded, acidaemia develops. Once this rise in hydrogen ion concentration has reached the CSF, it is detected by chemoreceptors and compensation occurs by reducing carbon dioxide levels through hyperventilation (first described by Kussmaul).

Detection of low pH in CSF rather than blood explains the delay in this compensation; rapid onset acidosis (for example during convulsions) tends not to stimulate respiration in spite of a low blood pH. Even though respiratory compensation occurs relatively quickly, it can take up to twelve hours to reach maximal capacity.

Rule for a Metabolic Acidosis: the expected pCO2 (in mmHg) is calculated from the following formula:

Expected pCO_2 = 1.5 × $[HCO_3]$ + 8 (range: +/– 2)

Comments:

– maximal compensation may take 12–24 hours to reach;

– the limit of compensation is a $PaCO_2$ of about 10 mmHg,

– hypoxia can increase the amount of peripheral chemoreceptor stimulation.

If the $PaCO_2$ is higher, then the compensation is at a very early stage or
the patient has a superimposed respiratory acidosis. If this is the case then earlier intervention with respiratory support is indicated.

Metabolic Acidosis Correction. The treatment for a metabolic acidosis is judged largely on clinical grounds. Bicarbonate therapy is justified when metabolic acidosis accompanies difficulty in resuscitating an individual or in maintaining cardiovascular stability.

A typical dose of bicarbonate might be 1 mEq per kilogram of the body weight followed by a repeated blood gas analysis. The effect of a bicarbonate dose can be anticipated by calculating the

dose required for complete correction.
A lesser dose has a proportionately less effect.

Dose (mEq) = 0.3 × Wt (kg) × BE (mEq/L)

Respiratory Acidosis

The decision to ventilate a patient to reduce the $PaCO_2$ is a clinical decision and is based on exhaustion, prognosis, prospect of improvement from concurrent therapy, and in part on the $PaCO_2$ level. Once the decision is made, the $PaCO_2$ helps calculate the appropriate correction.

The Rule for Acute Respiratory Acidosis. The $[HCO_3]$ will increase by 1 mmol/l for every 10 mmHg elevation in pCO_2 above 40 mmHg.

Expected $[HCO_3]$ = 24 + {(Actual pCO_2 – 40) / 10}

Comment: the increase in CO_2 shifts the equilibrium between CO_2 and HCO3 to result in an acute increase in HCO_3. This is a simple physicochemical event and occurs almost immediately.

The Rule for Chronic Respiratory Acidosis. The $[HCO_3]$ will increase by 4 mmol/l for every 10 mmHg elevation in pCO_2 above 40 mmHg.

Expected $[HCO_3]$ = 24 + 4 {(Actual pCO_2 – 40)/10}

Comment: with chronic acidosis, the kidneys respond by retaining HCO_3, that is, renal compensation occurs. This takes a few days to reach its maximal value.

Respiratory Alkalosis

The Rule for Acute Respiratory Alkalosis. The $[HCO_3]$ will decrease by 2 mmol/l for every 10 mmHg decrease in pCO_2 below 40 mmHg.

Expected [HCO3] = 24 – 2 {(40 – Actual pCO_2)/10}

Comment: in practice, this acute physicochemical change rarely results in a $[HCO_3]$ of less than about 18 mmol/l. (After all there is a limit to how low pCO_2 can fall as negative values are not possible!) So a $[HCO_3]$ of less than 18 mmol/l indicates a coexisting metabolic acidosis.

The Rule for a Chronic Respiratory Alkalosis. The $[HCO_3]$ will decrease by 5 mmol/l for every 10 mmHg decrease in pCO_2 below 40 mmHg.

Expected $[HCO_3]$ = 24 – 5 {(40 – Actual pCO_2) / 10} (range: +/– 2)

Comments: it takes 2 to 3 days to reach maximal renal compensation;
the limit of compensation is a [HCO3] of about 12 to 15 mmol/l.

Metabolic Alkalosis

A supranormal arterial blood pH with a base excess > 2 mmol/l caused either by loss of (non-carbonic) acid or gain of base. As the kidney is usually efficient at excreting large quantities of bicarbonate, persistence of a metabolic alkalosis usually depends on either chronic renal failure or a diminished extracellular fluid volume with severe depletion of K^+.

The patient is usually asymptomatic though, in case of spontaneous breathing he will hypoventilate. A metabolic alkalosis will cause a left shift of
the oxyhaemoglobin curve, reducing oxygen availability to the tissues.

Causes. Loss of total body fluid, Na^+, Cl^+, K^+ usually due to:

– diuretics;

– large nasogastric aspirates, vomiting;

– secondary hyperaldosteronism with potassium depletion;

– use of haemofiltration replacement fluid containing excess buffer (e.g. lactate);

– renal compensation for chronic hypercapnia. This can develop within
1–2 weeks. Although more apparent when the patient hyperventilates, or is hyperventilated to normocapnia, an overcompensated metabolic alkalosis can occasionally be seen in the chronic state (i.e. a raised pH in an otherwise stable long term hypercapnic patient);

– excess administration of bicarbonate;

– excess administration of citrate (large blood transfusion);

– drugs, including laxative abuse, corticosteroids;

– Rarely, Cushing's, Conn's, Bartter's syndrome.

Management:

1. Replacement of fluid, sodium, chloride (i.e. give 0.9 % saline) and potassium losses are often sufficient to restore acid-base balance.

2. With distal renal causes related to hyperaldosteronism, addition of spironolactone (or potassium canrenoate) can be considered.

3. Active treatment is rarely necessary. If so, give ammonium chloride 5 g tds PO. Hydrochloric acid has been used on occasion of severe metabolic alkalosis (pH > 7.7). It should be given via a central vein in a concentration of 1 mmol HCl per ml water at a rate not exceeding 1 mmol/kg/h.

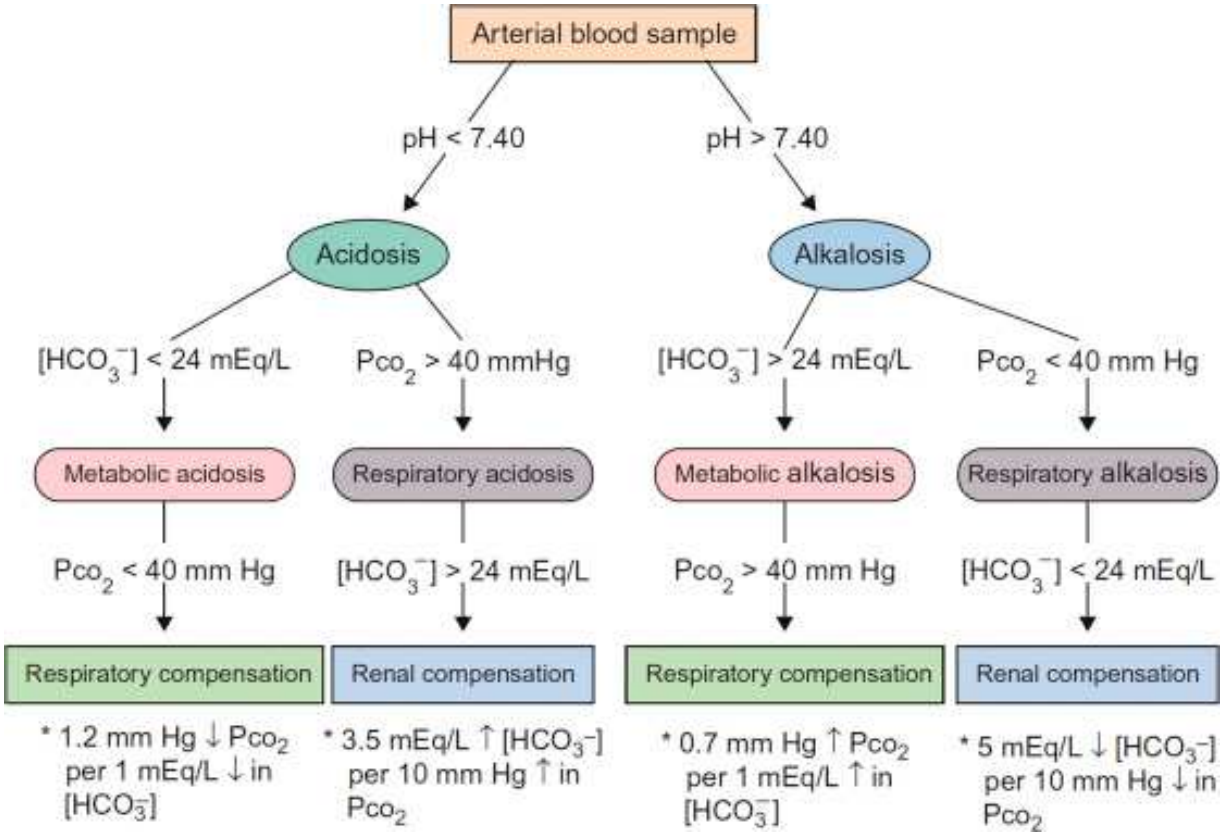
4. Compensation for a long-standing respiratory acidosis, followed by correction of that acidosis, e.g. with mechanical ventilation, will lead to
an uncompensated metabolic alkalosis. To correct it, treatment with acetazolamide can be considered. Mechanical "hypoventilation", i.e. maintaining hypercapnia, can also be considered.

The Rule for a Metabolic Alkalosis. The expected pCO_2 (in mmHg) is calculated according to the following formula:

Expected pCO_2 = 0.7 [HCO_3] + 20 (range: +/– 5)

Comment: the variation in pCO_2 predicted by this equation is relatively large.

Remember that only primary processes are called acidosis or alkalosis.
The compensatory processes are just that — compensation. Phrases such as "secondary respiratory alkalosis" should not be used.



52.The main types of water and electrolyte balance disorders, pathophysiology. Clinical signs, intensive therapy.

The answer of this question is so extended. Should read all first and answer is based of understanding of the topic.

WATER AND ELECTROLYTE BALANCE DISORDERS

Disorders of Na^+ and K^+ homeostasis are very common problems, encountered in clinical practice on an almost daily basis. They are frequently mismanaged due to poor understanding of Na^+ and K^+ metabolism.

Surgical patients are frequently affected by electrolyte imbalance. They are often sedated or not allowed to eat and drink, and hence have intravenous fluid infusions prescribed for extended periods.

Fluid and electolyte physiology

Fluids and electrolytes are present in a number of "compartments" in the body, according to their chemical composition.

Plasma is the fluid component of blood surrounding the red cells, intracellular fluid (ICF) is the fluid within the body's cells, and interstitial fluid (ISF) is the fluid found between the cells, outside blood vessels.

The intracellular and extracellular compartments are separated from one another by the plasma membrane of the cells. The extracellular compartments (interstitial/plasma/lymph) are separated by a layer of endothelial cells surrounded by a basement membrane; the capillaries.

To cross from the plasma to the cells or vice versa, substances must either cross both membranes of the endothelial cells or travel between the cells and then cross the basement membrane.

Capillaries act rather like a leaky hosepipe; although the bulk of the fluid flows along the pipe, the pressure forces some fluid out of the walls. The balance of hydrostatic and osmotic forces causing movement out and into the capillaries is known as Starling forces.

Water is present in plasma, ISF and ICF and passes freely between compartments under the influence of osmotic pressure gradients. The ISF and plasma together make up the extra cellular fluid (ECF).

Water accounts for 60 % of adult body weight (total body water (TBW) = 42 litres for a 70 kg adult).

TBW = 0.6 × Body Weight

Two thirds of this is ICF (28 litres) and one third is ECF (14 litres). The ECF can then be further subdivided into ISF (three quarters — 10.5 litres) and plasma (one quarter — 3.5 litres)

The ECF contains most of the sodium in the body, with equal sodium concentrations in the ISF and plasma. Sodium and water can pass freely through capillary membranes whilst albumin (the most important oncotically active constituent of the ECF)does not.

Albumin is unequally distributed in the intravascular and interstitial compartments (normal concentrations of 40 g/l^{-1} and 10 g/l^{-1} respectively) and is excluded from the intracellular compartment. This distribution helps to retain fluid within the plasma due to the osmotic effect of albumin.

Fluid replacement. Fluid replacement should address daily maintenance requirements and additional losses. Maintenance fluid, for patients who are unable take fluid enterally, should provide at least the minimal requirements of water, sodium and potassium. Remember that water and electrolyte requirements may increase in certain disease processes such as diarrhoea and vomiting.

Types of intravenous fluid

Crystalloids are substances which contain relatively small molecules that dissociate into ions to form true solutions, and are therefore capable of passing through a semi-permeable membrane. Commonly used crystalloids include 0.9 % saline, glucose and Hartmann's (Ringer's lactate) solution.

On intravenous infusion, 0.9 % saline and Hartman's solution rapidly distribute into the entire ECF, leaving 1/4 of the infused volume in the IVS, i.e. 250 ml of a 1000 ml fluid bolus.

5 % glucose (the optical isomer 'dextrose' is now rarely found) loses all of its glucose at first pass through the liver and skeletal muscles.

The remaining water is distributed evenly throughout the entire TBW, leaving only 1/12 of the original volume in the intravascular space (IVS) (i.e. only 83 ml of a 1000 ml fluid bolus).

Colloids contain larger molecules that are dispersed throughout a solvent, i.e. they do not dissolve to form solutions. They cannot pass through semi-permeable membranes and consequently tend to remain in the IVS.

Gelatins (e.g. gelofusine, haemaccel). These colloids are polysaccharides, derived from gelatin, which are in turn derived from collagen and have an average molecular weight of 35,000 Daltons. (A Dalton or "atomic mass unit" is a unit of mass, equal to 1/12 the mass of carbon 12, which is assigned a mass of 12.)

Their half-life in the IVS is approximately 3 hours and they are renally excreted. Anaphylactic reactions have been reported with an incidence of 1 in 13,000. Gelatin

solutions may interfere with platelet function and coagulation via a reduction in levels of the von Willebrand factor.

Dextrans (e.g. dextran 40, dextran 70). A dextran is a polysaccharide, derived from sucrose by the action of the bacterium Leuconostoc mesenteriodes. Dextran 40 has a molecular weight of 40,000 Daltons and dextran 70 a molecular weight of 70,000 Daltons.

The intravascular half-life increases with a molecule size, and ranges from 15 minutes to several days. The smaller molecular weight dextrans are predominantly excreted unchanged by the kidney (accounting for up to 70 % of Dextran 40), while the larger molecular weight dextrans are retained in the circulation for several days. Dextrans have an incidence of anaphylaxis of 1 in 4,500.

Dextrans are rarely used for fluid resuscitation, but they have a role in thrombo-embolic prophylaxis via volume expansion, reduction in viscosity, and lowering platelet and erythrocyte aggregation. Side effects include renal failure (due to tubular obstruction), interference with cross matching and coagulopathy.

Starches (e.g. hydroxyethyl starch (HES), Hetastarch). These synthetic colloids are of a similar structure as glycogen, consisting of glucose chain of molecules (> 90 % amylopectin).

They have molecules with a large range of molecular weights, with the smaller molecules (approximately 50,000 Daltons) excreted by the kidney and the larger molecules slowly broken down by alpha-amylase hydrolysis of glycosidic bonds, yielding molecules small enough for renal clearance.

Anaphylactic reactions are rare with an incidence of 1 in 16,000. Slight prolongation of coagulation may occur after large infusions, and pruritis has also been reported. Total dose should not exceed 20 ml/kg^{-1} (1500 ml/day^{-1} for an average male).

Human albumin solutions are derived from human plasma by fractionation, and then they are heat sterilized to reduce the risk of infective transmission. They are presented as either 4.5 % (40–50 g/l^{-1}) or 20 % (150–200 g/l^{-1}) solutions in 0.9 % saline.

Traditionally albumin solutions were used as colloid in patients who were hypoalbuminaemic or in cases where high albumin loss was anticipated (e.g. burns), and also in the resuscitation of children.

Distribution of sodium and potassium

The distribution of Na^+ and K^+ can be thought of as opposite — where one is found abundantly, the other is at low concentration. Sodium is the most prevalent cation in the ECF, with a normal level of about 140 mmol/l^{-1}, but with a typical intracellular concentration of around 10 mmol/l^{-1}.

In contrast, potassium is the most prevalent cation in the ICF, with a concentration around 150 mmol/l^{-1}. The intracellular space is the largest fluid compartment in the body, and this makes it the most abundant cation overall. Only about 1 % of the total body K^+ is found in the plasma with the levels kept between 3.5 and 4.5 mmol/l^{-1}.

The cell membrane acts as the barrier between the potassium-rich ICF and the sodium-rich ECF. While it allows free passage of water and non-polar, hydrophobic molecules, it is impermeable to large molecules or charged particles. Hence Na^+ and K^+ can only cross where specific carrier proteins allow them to do so.

In vivo, the membrane remains relatively impermeable to both Na^+ and K^+. Excitable cells can change their permeability to allow the influx and efflux of ions that constitute an action potential.

At rest, large concentration gradients for Na^+ and K^+ are maintained by the action of Na^+/K^+-ATPase, a transmembrane protein which pumps out 3 Na^+ for each 2 K^+ it pumps in. This also maintains the net negative resting membrane potential since it involves a net transfer of one positive charge out of the cell on each cycle.

Although the Na^+/K^+-ATPase maintains the concentration gradients across the cell membrane, other mechanisms are in overall control of total body Na^+ and K^+ levels.

Sodium homeostasis

The volume of circulating plasma is vitally important to the body, since an adequate plasma amount is required for normal tissue perfusion. The plasma volume is proportional to the ECF volume, and since Na^+ is the major cation of the ECF, total body Na^+ content is proportional to ECF volume.

In healthy individuals, the kidney strives to achieve Na^+ balance — that is, to have Na^+ excretion equal to Na^+ ingestion. Long-term control of blood pressure is achieved by the excretion or retention of Na^+ (and hence plasma volume) in the kidney.

The vast majority (99–99.5 %) of Na^+ filtered by the kidney is reabsorbed in the proximal tubule and the loop of Henle. This reabsorption seems to be largely fixed, even in sodium overload. There is much greater control over the 0.5 % of filtered Na^+ reabsorbed in the distal tubule and collecting ducts.

It is this proportionately a tiny amount, that allows the body to either retain sodium and water or excrete them when necessary. Various hormones influence this balance of retention and excretion.

Potassium homeostasis

Small increases in the serum potassium concentration can quickly become life-threatening. The kidneys cannot excrete potassium quickly enough to contain surges due to oral potassium loads, and hence intracellular buffering plays an important role in homeostasis.

As the kidneys excrete the excess of potassium and its serum concentration falls, K^+ is released again from the cells. In the normal state 90 % of daily potassium intake is excreted via the kidneys and the rest via the colon. About 90 % of the filtered potassium load is reabsorbed by the start of the distal tubule, and this figure is largely constant through a wide range of potassium intake.
The overall urinary excretion of K^+ is therefore controlled by the distal tubule and collecting ducts.

Water and electrolyte requirements for adults:

– water — 30–40 ml/kg^{-1};

– Na^+ — 1.0–2.0 $mmol/kg^{-1}$;

– K^+ — 0.7–1.0 $mmol/kg^{-1}$;

– Ca_2^+ — 0.1 $mmol/kg^{-1}$;

– Mg_2^+ — 0.1 $mmol/kg^{-1}$;

– Cl^- — 1.0–2.0 $mmol/kg^{-1}$.

Routine intraoperative administration

The goals of intraoperative fluid administration are to maintain adequate oxygen delivery, normal electrolyte concentrations and normoglycemia.

The total fluid requirement is composed of compensatory intravascular volume expansion (CVE), deficit replacement, maintenance fluids, restoration of losses and substitution of fluid redistribution (3rd space fluids):

$$\text{Rate} = \text{CVE} + \text{deficit} + \text{maintenance} + \text{loss} + \text{third space}$$

Deficits. The fluid deficit is equal to the maintenance fluid requirement multiplied by the hours since the last intake plus unreplaced preoperative external and third space losses. When hypovolemia is present, it is necessary to infuse sufficient fluid to restore MAP, heart rate, and filling pressures to neally normal values prior to induction.

Losses. External losses (e.g., blood, ascites) should be replaced to maintain normal blood volume and normal composition of the ECFV. Blood loss is replaced initially with either 3 ml of balanced salt solution or 0.9 percent NaCl for each milliliter of blood loss.

For each milliliter of blood loss, 1 ml of colloid solution should be used, when crystalloid replacement provides only transient improvement of filling pressures, arterial blood pressure and heart rate despite full calculated infusion rates. Packed RBC infusions are used roughly 1 ml for each 2 ml of the lost blood plus either crystalloid or colloid as described above.

Evaporation from exposed viscera is entirely water, but the electrolyte is left behind, leading to a need of free water. The amount evaporated is directly proportional to temperature, exposed surface area and inversely proportional to relative humidity.

Redistribution. Redistribution, or so-called the 3rd-space losses, occurs primarily due to tissue edema and transcellular fluid displacement. Functionally this fluid is not available to the vascular space. Colloid enters the injured tissue at a more rapid rate than normally, but at a slower rate than electrolyte.

For example, bowel wall edema is lessened by utilizing colloid containing fluids compared with crystalloid fluids. The composition of the third-space losses is the equivalent to the ECFV electrolyte concentration plus a smaller amount of protein. Therefore, balanced salt solution is the most appropriate replacement fluid.

The redistributed volume correlates roughly with the degree of tissue manipulation. Intra-abdominal procedures with small incisions (e.g., hysterectomy) may require an additional 2 ml/kg/h, while a major bowel resection will require an additional 4 to 6 ml/kg/h.

Dehydration. Dehydration is the pathological loss of fluids (and usually accompanying electrolytes). It may be the primary issue, or merely a manifestation of some other processes.

History. In addition to the usual history, pay particular attention to "ins and outs". Determine what fluids the patient has, see if there is a history of poor intake, or if the patient has been drinking excessive amounts of fluids. Ask which fluids and food the patient has been consuming.

On the output side, get a quantitative sense of what fluids have been lost. Has the patient been vomiting, had diarrhoea, lost blood, had an NG tube present to remove gastric secretions, sweated excessively? How frequently and how much did the patient urinate? Place all this information in a time frame.

Causes of hyponatraemia

Osmolality and osmolarity differ according to whether the number of osmotically active particles is dissolved in a kilogram or a liter of solvent respectively.

Osmolality is measured in the laboratory with an osmometer that either assesses the depression of a sample's freezing point or the depression of its vapour pressure. It is preferablly to use the former as any volatile alcohols in the sample will evaporate as the sample is heated and the results from this method use will be inaccurate. Osmolarity can be calculated using various formulae including
the following:

Calculated osmolarity = 2 [Na^+] + urea + glucose

The overall osmolarity of all three compartments is identical to about
280–300 mOsmol/l.

Consideration of the osmotic state of the patient is essential in the evaluation of hyponatraemia:

Normal osmolarity — pseudohyponatraemia:

– Due to a measurement error which can result when the solid phase of plasma (that due to lipid and protein) is increased.

– Typically caused by hypertriglyceridaemia or paraproteinaemia.

High osmolarity — translocational hyponatraemia:

– Occurs when an osmotically active solute that cannot cross the cell membrane is present in the plasma.

– Most solutes such as urea or ethanol can enter the cells, and cause hypertonicity without cell dehydration.

– However, in case of the insulinopenic diabetic patient, glucose cannot enter cells and hence water is displaced across the cell membrane, dehydrating the cells and 'diluting' the sodium in the serum.

– This is also the cause of hyponatraemia seen in the TURP syndrome, in which glycine is inadvertently infused to achieve the same effect.

Low osmolarity — true hyponatraemia. True hyponatraemia is always a hypo-osmolar condition. The next stage is to consider the volume status of the patient:

Hypovolaemic hyponatraemia:

– Loss of both sodium and water, but proportionately more sodium.

– Caused by solute and water losses from either a renal or gastrointestinal source.

– Usually these patients are consuming water or hypotonic fluid, although not in quantities sufficient to restore normovolaemia.

– An estimation of the urinary sodium level can be helpful: a level below 30 $mmol/l^{-1}$ suggests an extrarenal cause, while a level above 30 $mmol/l^{-1}$ suggests a primary renal problem.

Euvolaemic hyponatraemia:

– The most common form seen in hospitalized patients.

– May have a slight increase or decrease in volume, but it is not clinically evident, and they do not have oedema.

– The most common cause is the inappropriate administration of hypotonic fluid.

– The syndrome of inappropriate ADH secretion (SIADH) also causes euvolaemic hyponatraemia; in order to make this diagnosis one must first exclude renal, pituitary, adrenal or thyroid dysfunction, and the patient must not be taking diuretics.

Hypervolaemic hyponatraemia:

– Characterised by both sodium and water retention, with proportionately more water.

– Therefore has an increased amount of total body sodium.

– Causes are all characterised by disordered water excretion, and are usually easy to diagnose.

Effects and treatment of hyponatraemia. The normal range of serum sodium is usually quoted as being approx. 135–145 mmol/l^{-1}; however, levels between 125 mmol/l^{-1} and 150 mmol/l^{-1} are often asymptomatic.

Outside this range there is an increasing frequency of nausea, lethargy, weakness and confusion, and levels above 160 mmol/l^{-1} or below 110 mmol/l^{-1} are strongly associated with seizures, coma and death. As serum sodium and osmolarity fall, water tends to enter the cells causing them to swell. Clinically this is the most important for the brain.

Several factors put patients at increased risk of complications of hyponatraemia or its treatment:

– Postoperative patients, premenopausal women, elderly women taking thiazides, children, and patients who are hypoxaemic are all at increased risk of acute hyponatraemic cerebral oedema.

– Malnourished patients, alcoholics, those with burns or hypokalaemia are all at increased risk of osmotic myelinolysis due to overly rapid correction of hyponatraemia.

A recent review of the literature has pointed out that there is as yet no consensus on the optimum treatment of dysnatraemia. However, all authorities stress the importance of distinguishing between hyponatraemia that has developed acutely (usually it takes less than 48 hours) and chronic hyponatraemia. This is because of important differences in the management between the two groups.

Most authors suggest that hyponatraemia that has developed acutely (for instance, in the immediate postoperative period) can be safely treated with rapid correction. Rapid correction should only be undertaken in patients who are symptomatic, and the aim of treatment is to correct the level until the symptoms resolve.

Some sources have suggested that correction by up to 2 mmol/l^{-1}/h^{-1} is safe in the initial treatment of acute hyponatraemic states. Correction to a serum Na^{+} of > 135 mmol/l^{-1}

may be safe in this situation, but it is not necessary to correct rapidly once the symptoms have resolved.

Methods of rapid correction might include the administration of furosemide and/or hypertonic saline; however management should be by a specialist in an appropriate setting, with hourly monitoring of serum Na^+ levels.

Determine the Sodium Deficit

Na deficit (mEq) = (135 – Na (current) × Bode weight (kg) ×0.6

This formula is based on the assumption that the desired [Na^+] is 135 mEq/L, and that total body water is about 60 % of body weight (although this varies with age).

The treatment of chronic hyponatraemia is also determined by the presence or absence of symptoms. In the presence of symptoms, a rapid correction of up to 10 $mmol/l^{-1}$ may be permissible.

Following this, however, the rate of reversal should be limited to 1.5 $mmol/l^{-1}/h^{-1}$ and to no more than 8 $mmol/l^{-1}$ over 24 hrs. Some sources suggest that a rate of 12 $mmol/l^{-1}$ in 24 hrs is safe.

Fluid restriction is the mainstay of treatment in these patients, who need to have regular neurological assessment and rechecking of serum electrolytes at least every 12 hours.

In the long-term, treatment is aimed at identifying and dealing with the underlying cause. Future advances in the shape of selective ADH (AVP) antagonists (so-called aquaretics) contribute to the improvement in the long-term management of chronic hyponatraemia.

In all cases, hypovolaemia, if present, must be corrected first with 0.9 % saline. This removes the ADH response that is accentuating the sodium/water imbalance.

In hypervolaemic patients the treatment is aimed at fluid and salt restriction and application of loop diuretics. Aquaretics may also be useful for these patients. While evidence is lacking that chronic hyponatraemia is associated with worse surgical outcomes, asymptomatic hyponatraemia should be regarded as a relative contraindication to elective surgery.

Causes of hypernatraemia

Hypernatraemia is either caused by excessive salt intake, or (much more frequently) inadequate water intake. As with hyponatraemia, consideration of the volume status of the patient is essential.

Hypovolaemic hypernatraemia:

– Loss of both sodium and water, but relatively more water.

– An estimation of the urinary sodium level can be helpful: a level below 30 $mmol/l^{-1}$ suggests an extrarenal cause, while a level above 30 $mmol/l^{-1}$ suggests a primary renal problem.

– These patients are either not able to take in adequate fluid to replace their losses, or are prevented from doing so.

Euvolaemic hypernatraemia:

– Occurs when body water losses are partially replaced.

– May be due to a lack of available water, or due to a blunting of the normal thirst response seen in the extremes of age.

Hypervolaemic hypernatraemia:

– Seen where sodium retention is not matched by increased fluid intake.

– More uncommon than the other two types of hypernatraemia.

Treatment of hypernatraemia. Firstly any volume deficit should be corrected with 0.9 % saline until the hypovolaemia, as measured by orthostatic hypotension, improves.

The water deficit is calculated as:

$$\textbf{Water deficit} = \textbf{TBW} \times (\textbf{Na current} - \textbf{Na desired}) / \textbf{Na current}$$

The cause of fluid loss should also be investigated and treated. The total body water deficit can be calculated based on the serum sodium and the assumption that 60 % of the body is water — this deficit should then be corrected with 5 % dextrose, with half given in the first 12–24 hours, and the rest over the next 24–36 hours.

In case of hypervolaemic hypernatraemia, the removal of excess sodium is the aim, and loop diuretics or dialysis may achieve this if the patient has renal dysfunction.

Causes of hypokalaemia

Hypokalaemia is caused by a shift of potassium into cells, or more commonly by a total body potassium deficit. Occasionally the two situations may co-exist.

Intracellular potassium shifting:

– Excess insulin (exogenous or endogenous).

– β-adrenoceptor agonists (such as endogenous catecholamines or exogenous salbutamol).

– Theophylline toxicity.

– Acute rise in plasma pH.

Total body potassium deficit:

– May result from either decreased intake or increased losses.

– Diet must be severely deficient in K^+ over a long period in order to reach a position of clinical hypokalaemia; hence seen most commonly in alcoholics.

– Excessive losses may be either renal or extrarenal.

Renal causes include:

– Diuretics.

– Mineralocorticoid excess.

– Glucocorticoid excess.

– Renal tubular acidosis Type I and II.

– Diabetic ketoacidosis — glucose causes an osmotic diuresis, washing out potassium.

– Vomiting — which is not caused by a loss of K^+ in the vomit, it is rather caused by a loss of H^+ and water leading to metabolic alkalosis and increased aldosterone.

– Ureterosigmoidostomy.

– Rare inherited conditions such as Bartter's and Gitelman's Syndromes.

Extrarenal causes include:

– Inadequate intake.

– Excessive perspiration.

– Chronic diarrhoea.

– Gastrointestinal fistulae.

Effects of hypokalaemia. The effects of hypokalaemia depend upon the serum level. A normal value of 3.5–4.5 mmol/l^{-1} is generally accepted, but levels of 3.0–3.5 mmol/l^{-1} are usually asymptomatic. Below 3.0 mmol/l^{-1} general symptoms of weakness, lassitude and constipation are common.

Below 2.5 mmol/l^{-1} muscle necrosis has been described (probably due to an inability to increase blood flow during exercise), and below 2.0 mmol/l^{-1} an ascending paralysis may be seen, eventually leading to respiratory compromise.

Patients without underlying cardiac disease are unlikely to suffer myocardial effects, even at levels below 3.0 mmol/l^{-1}. However, those with ischaemic heart disease, heart failure or left ventricular dysfunction are at risk of arrhythmias with only mild or moderate hypokalaemia. Initially U-waves are seen on the ECG, with gradual sagging of the ST segment and flattening of the T-wave.

Slight widening of the QRS complex and PR elongation may be seen, and there is a predisposition to both ventricular and supraventricular ectopic rhythms, especially in a patient taking digoxin.

Renal effects of hypokalaemia include metabolic acidosis, increased ammoniagenesis and numerous structural changes in the kidney if the condition persists.

As with sodium, the rapidity of the change in K^+ level has a large influence on the severity of the symptoms.

Treatment of hypokalaemia. Once intracellular K^+ shifts have been excluded (theophylline toxicity, hyperinsulinaemia) the treatment of hypokalaemia is aimed at replacement of potassium. Ideally, this should be oral supplementation, but if the shifts

are severe the initial replacement is best given intravenously, through a central vein within a critical care facility.

Careless administration of intravenous potassium is the commonest cause of hyperkalaemia in hospitalized patients, so appropriate consideration should be given to this decision. In any case, the rate of administration should not exceed 20 $mmol/h^{-1}$, and the patient should have continuous cardiac monitoring.

In the absence of factors causing potassium shifting into cells, the serum potassium is a good guide to the total body potassium deficit.

A fall from 3.5 to 3.0 $mmol/l^{-1}$ suggests a deficit in the order of 5 % (around 175 mmol); a decline from 3.0 to 2.0 $mmol/l^{-1}$ suggests a further 200–400 mmol deficit.

Magnesium deficiency is very commonly associated with hypokalaemia and levels should be checked and magnesium replaced if appropriate.

Prophylactic administration of potassium to postoperative patients at risk of cardiac abnormalities is common practice. There is evidence that minor elevation of K^+ (within the normal range) can reduce the incidence of electrocardiac abnormalities such as U-waves, bifid T-waves and signs of digitalis toxicity.

Additionally, potassium supplementation may benefit patients with abnormal repolarisation in the context of congestive cardiac failure.

However, the practice of artificially augmenting the potassium level to abolish single ventricular ectopic beats or as a routine treatment for all postoperative patients is no longer considered best practice.

Causes of hyperkalaemia

Hyperkalaemia may be due to either an overall increase in total body potassium, or an acute shift of potassium from the intracellular to the extracellular compartment.

Extracellular potassium shifts occur in case of:

– Acidosis — H^+ is taken into the cell in exchange for K^+.

– Insulin deficiency, with hyperglycaemia — note that this is often found coexistent with a profound total body potassium deficit.

– Digitalis toxicity — due to inhibition of the Na^+/K^+-ATPase.

– β-blockers — typically cause only a mild elevation in K^+.

– Exercise — potassium efflux from skeletal muscle as a result of muscular contraction

– Suxamethonium administration.

Fasciculations lead to an efflux of potassium from the skeletal muscle, similar to the effect of exercise but more pronounced and more acute. A single 100 mg dose may cause serum potassium to rise by up to 1.0 mmol/l^{-1}. If the patient already has elevated serum potassium, this may be enough to cause a fatal arrhythmia.

In patients with a denervated muscle, the usual mechanisms keeping the acetylcholine receptors in the synaptic cleft are disturbed, and they spread out to cover the whole of the muscle fibre (extrajunctional receptors). Suxamethonium administration is contraindicated in these patients as it causes a much bigger potassium efflux and often leads to dangerous hyperkalaemia.

Excessive potassium input occurs in case of:

– Cellular lysis, as seen in haemolysis, rhabdomyolysis or tumour lysis syndrome.

– Inappropriate prescription of K^+-containing IV fluids or supplements is a very important cause in hospitalized patients.

Impaired renal excretion occurs in case of:

– Decreased GFR — renal failure is the commonest cause of hyperkalaemia.

– Mineralocorticoid insufficiency — this may be due to primary adrenal failure, hyporeninaemic hypoaldosteronism (Renal tubular acidosis type IV), or due to drugs similar to ACE inhibitors, Angiotensin-II receptor antagonists or spironolactone.

– Potassium sparing diuretics.

Primary renal insults (such as interstitial nephritis) causing decreased potassium excretion in the distal tubules and collecting ducts.

Pseudohyperkalaemia:

– A common cause of spuriously elevated potassium levels.

– The most common causes are in vitro haemolysis, or leaving the tourniquet on for an extended period prior to blood sampling.

– It is also seen in patients with highly elevated white cell or platelet counts, due to secretion of potassium from these cells prior to laboratory analysis.

Effects of hyperkalaemia. The most important effects of hyperkalaemia are on the heart. Levels below 6.0 mmol/l^{-1} rarely cause any clinical symptoms.
As the serum K^+ level increases, ECG changes are noted: firstly peaking of the T waves, then broadening of the P-waves and QRS-complex when the level is > 7.0 mmol/l^{-1}.

Finally the ECG takes on a sinusoidal pattern, which is a precursor to cardiac arrest. Terminal ECG changes may develop very quickly, and even with mildly elevated potassium levels any sign of ECG involvement should prompt immediate treatment.

As with other electrolyte disturbances, the speed of hyperkalaemia onset is very important. A relatively small increase, if it occurs over a short time, can precipitate a fatal arrhythmia where a much higher level may be tolerated (for instance, in the insidious onset of renal failure) if it has developed over a longer period.

Other sequelae of hyperkalaemia include paraesthesiae, weakness, paralysis, a decreased renal production of ammonia, an increased renal retention of H^+ and a subsequent metabolic acidosis, natriuresis, and elevated levels of aldosterone and insulin.

Treatment of hyperkalaemia. The various therapies commonly used to reduce the K^+ level acutely may be divided into two groups: those that seek to transiently move the potassium to the intracellular compartment, and those that seek to remove an overall surplus of potassium from the body.

While the former group may be used in the vast majority of hyperkalaemic patients, not all hyperkalaemic patients have the excessed total body potassium.

The classic example is an acidotic patient with diabetic ketoacidosis, who has elevated serum potassium due to cellular impermeability in the absence of insulin, but who is often profoundly depleted in potassium levels overall.

Such patients require emergency management to lower the high potassium levels, but as treatment commences and their cells rapidly become permeable to potassium. Caution must be taken to avoid them developing a rebound hypokalaemia.

The therapies most often suggested for acute potassium lowering are infusions of glucose and insulin, β_2-adrenoceptor agonists, either nebulised or inhaled, and IV sodium bicarbonate.

Of these, glucose-insulin and beta-agonists both seem to be effective, and a combination seems more effective than either being used in isolation.

The same review investigated two methods for removing excess potassium from the system: K^+-absorbing styramine resins, and dialysis. Of these, the evidence was that resins were not effective at 4hrs after administration, but longer-term studies have not been done. Dialysis was effective at decreasing total body potassium over the same period.

In addition, the administration of calcium (as either calcium gluconate or calcium chloride) is recommended as a means of rapidly reversing the repolarisation abnormalities seen in severe hyperkalaemia. Furthermore, it must always be remembered that a cornerstone of treatment is to diagnose the underlying cause of hyperkalaemia and take steps to reverse this.

53. Parenteral nutrition. Purposes, indications, types, intravenous nutrients. Control of parenteral nutrition.

Parenteral feeding is the intravenous administration of nutrients. This may be supplemental to oral or tube feeding, or it may provide the only the source of nutrition as total parenteral nutrition (TPN).

The only absolute indication for parenteral nutrition (PN) is gastrointestinal failure. All efforts to improve tolerance of enteral feeding such as use of pro-kinetic agents and/or a post-pyloric feeding tube should be used before starting PN.

Patients receiving less than 25 % of their predicted needs are at the increased risk of sepsis and those who are intolerant of enteral nutrition, despite all attempts to improve this, should be considered for parenteral supplementation.

During acute illness, the aim is to provide energy as close as possible to estimated or measured energy expenditure in order to decrease the negative energy balance. In the absence of indirect calorimetry, ICU patients should receive 25 $kcal/kg^{-1}/day^{-1}$ increasing to target levels over the next 2–3 days.

PN can be given as separate components but is more commonly given as a sterile emulsion of water, protein, lipid, carbohydrate, electrolytes, vitamins and trace elements according to the recommendations discussed earlier regarding nutritional requirements.

Standard formulations require thorough mixing before infusion.
The electrolyte concentration can be altered for each patient and additional trace elements and vitamins may be added.

Protein is given as amino acids and includes essential amino acids. It should also ideally include most of the non-essential amino acids.

Critical illness results in a relative deficiency of glutamine. In a number of small studies IV glutamine improves survival and infection rates in patients on PN, particularly with traumas and burns. Glutamine supplementation is likely to be beneficial for patients receiving TPN for more than 10 days.

Lipid is commonly given as Intralipid®; it is emulsion made of soya with chylomicron-sized particles. It provides a source of essential fatty acids, (linolenic acid, an omega-3 fatty acid and linoleic acid, an omega-6 fatty acid) and is
a vehicle to deliver fat-soluble vitamins.

Because lipid preparations are expensive, it is possible to give parenteral nutrition with low levels of lipid thus giving 6 % of total energy requirement as lipid is enough to avoid essential fatty acid deficiency.

If no parenteral lipid is given, vegetable oil should be massaged into
the patient's limbs once a day; lipid is absorbed through the skin and may prevent or delay essential fatty acid deficiency, although requirements in critical illnesses may be too high for this to be sufficient.

Watch for signs of deficiency: dry, scaly skin, with or without hair loss, and abnormal liver function tests. Most vegetable oils can be used (safflower, corn, soya, groundnut or sunflower) but not palm oil, as it contains virtually no linolenic acid. Fat-soluble vitamins will need to be given separately.

Carbohydrate is given as glucose. The minimal amount of carbohydrate required is about 2 g/kg^{-1} glucose per day. It should provide approx. 60 % of non-protein calories.

Electrolytes and micronutrients. Critically ill patients are prone to fluid and sodium overload, and renal dysfunction is frequent. The exact electrolyte requirement needs to be determined by close plasma electrolyte monitoring and should not be a fixed element of parenteral nutrition prescription.

Patients with sepsis may have large amount of vitamin A losses in their urine, burn patients lose selenium, zinc and copper via their exudates and trauma patients lose selenium and zinc through their drains.

Selenium impairs the role of glutathione peroxidase as a free radical scavenger and selenium supplementation may be helpful in general ICU patients.

54.Parametres of central haemodynamics. Shock. Classification of shock, principles of diagnostics.

Shock is a condition of inadequate tissue perfusion or insufficient delivery of oxygenated blood and nutrients. Shock is a progressive disorder that, if uncorrected, leads to death.

Shock can be classified into 4 major types. Further there are the parameters and examples to differentiate between the types of shock:

Hypovolemic — associated with decreased preload.

Cardiogenic — results from heart pump dysfunction (more commonly left-sided) causing a decrease in cardiac output in the setting of increased preload.

Obstructive — secondary to obstruction of the cardiac flow or filling.

Distributive — associated with significant vasodilation in the setting of relative hypovolemia and decreased SVR. The classic description of distributive shock includes an elevated cardiac output.

Parameters to evaluate shock. The usual parameters of BP, heart rate, and urine output, which have been traditionally used as indicators of global perfusion since the 1960s, have given way to a new definition of shock, based on the demand and supply of oxygen at the tissue level; thus, measures of regional perfusion have replaced global vital signs. Normal BP does not necessarily mean normal perfusion, as adequate pressure does not equate to adequate cardiac output (CO).

Cardiac output (CO) is the product of heart rate (HR) and stroke volume (SV): CO = HR × SV.

Stroke volume is determined by three main factors: preload, afterload and contractility.

55.Pathophysiology, principles of diagnostics and intensive therapy of Hypovolemic shock.

Hypovolemic shock refers to a medical or surgical condition in which rapid fluid loss results in multiple organ failure due to inadequate perfusion. Most often, hypovolemic shock is secondary to rapid blood loss (hemorrhagic shock).

The human body responds to acute haemorrhage by activating 4 major physiologic systems: the hematologic, cardiovascular, renal, and neuroendocrine systems.

The hematologic system responds to an acute severe blood loss by activating the coagulation cascade and contracting the bleeding vessels.

The cardiovascular system initially responds to hypovolemic shock by increasing the heart rate, myocardial contractility, and constricting peripheral blood vessels. This response occurs secondary to an increased release of norepinephrine and decreased baseline vagal tone (regulated by the baroreceptors in the carotid arch, aortic arch, left atrium, and pulmonary vessels).
The cardiovascular system also responds by redistributing blood to the brain, heart, and kidneys and away from skin, muscle, and GI tract.

The renal system responds to hemorrhagic shock by stimulating an increase in renin secretion from the juxtaglomerular apparatus.

The neuroendocrine system responds to hemorrhagic shock by causing an increase in circulating antidiuretic hormone (ADH). ADH indirectly leads to an increased reabsorption of water and salt (NaCl) by the distal tubule, collecting ducts, and the loop of Henle.

The atraumatic patient with hypovolemic shock requires ultrasonographic examination in the ED if an abdominal aortic aneurysm is suspected. If GI bleeding is suspected, a nasogastric tube should be placed, and gastric lavage should be performed. Endoscopy can be performed (usually after the patient has been admitted) to further delineate the source of bleeding. If abdominal injury is suspected, diagnostic peritoneal lavage may be performed in unstable patients, or CT scanning in stable patients. If long-bone fractures are suspected, radiographs should be obtained.

***Management of haemorrhagic shock*:**

– Remember the ABC of resuscitation.

– Check and correct any problems with the airway and breathing.

– Give oxygen in a high inspired concentration by face mask. Intubate patients who are unconscious.

– Control external haemorrhage by elevating the limb and by direct firm pressure with a clean pad over the bleeding site.

– Insert a large cannula (14 gauge) into a suitable vein, use two when shock is worse than class one. When it is difficult to find veins, cannulate the external jugular or femoral vein or perform a cut down at the ankle or antecubital fossa.

– In small children the intra-osseus route has been used with success. Do not use leg veins when intra-abdominal haemorrhage is suspected, or cannulate veins in an injured arm or shoulder.

– Once IV access is obtained, initial fluid resuscitation is performed with an isotonic crystalloid, such as lactated Ringer solution or normal saline. An initial bolus of 1–2 L is given in an adult (20 mL/kg in a pediatric patient), and the patient's response is assessed.

56.Pathophysiology, principles of diagnostics and intensive therapy of Cardiogenic shock.

Cardiogenic shock is characterized by a decreased pumping ability of the heart that causes inadequate perfusion to the tissues. It most commonly occurs in association with, and as a direct result of, acute ischemic damage to the myocardium. The most common initiating event in cardiogenic shock is acute myocardial

infarction (AMI). Dead myocardium does not contract, and once more than 40 % of the myocardium is involved, cardiogenic shock may result.

Most patients with cardiogenic shock have an AMI and, therefore, with the constellation of symptoms of acute cardiac ischemia (e.g. chest pain, shortness of breath, diaphoresis, nausea and vomiting). Patients experiencing cardiogenic shock may also have pulmonary oedema and presyncopal or syncopal symptoms.

physical examination often reveals that the patient is in the middle of an AMI. Patients are in frank distress, are profoundly diaphoretic, and have severe shortness of breath and chest pain.

– Clinical assessment begins with paying attention to the ABCs and vital signs.

– Breathing may be laboured, with audible coarse crackles or wheezing.

– Patients with marked tachycardia, cool and clammy extremities, poor peripheral pulses, and varying degrees of end-organ dysfunction (e.g., decreased mental function and urinary output).

– Neck examination may reveal jugular venous distention, which may be prominent. This finding is evidence of right ventricular failure.

– Careful cardiac examination may reveal mechanical causes of cardiogenic shock.

Lab studies. In most cases, the usual workup includes tests, which usually are assessed in cases of suspected cardiac ischemia:

– cardiac enzymes (e.g., creatine kinase, troponin, myoglobin);

– electrolytes;

– coagulation profile (e.g., prothrombin time, activated partial thromboplastin time);

– an ABG test may be useful to evaluate acid-base balance, because acidosis can have a particularly deleterious effect on the myocardial function.

Other tests. An ECG is helpful if it reveals an acute injury pattern consistent with an AMI. A normal ECG, however, does not rule out the possibility. ECGs are often most helpful when they can be compared with previous tracings.

Emergency department care:

– Treatment begins with the ABC assessment.

– Once the ABCs are managed, an early focus of treatment should be reversed to the underlying cause and in most cases means revascularization.
If a catheterization laboratory is readily available, percutaneous transluminal coronary angioplasty with or without stent placement may be performed to achieve a better outcome. If no

catheterization laboratory is available, the next best option is thrombolytic therapy, if no contraindications are present.

– Supportive care and the prevention of further ischemia have various implications in these patients. They require oxygen, intravenous fluids, and close cardiac and hemodynamic monitoring. Providing high-flow oxygen, decreasing myocardial oxygen consumption, and increasing perfusion of the ischemic myocardium may reduce ischemia. Extreme heart rates should be avoided because they may increase myocardial oxygen consumption and infarct size thus impairing the pumping ability of the heart.

– Pharmacologic interventions may be advisable, depending on the circumstances.

– Nitrates and/or morphine are advised for the management of pain; however, they must be used with caution because such patients are in shock, and excessive use of either of these agents can produce profound hypotension.

– Dopamine may provide vasopressor support. With higher doses, it has the disadvantage of increasing heart rate and myocardial oxygen consumption.

– Dobutamine, amrinone, or milrinone may provide inotropic support.

57.Pathophysiology, principles of diagnostics and intensive therapy of Anaphylactic shock.

Anaphylaxis is an acute systemic reaction caused by the release of mediators from mast cells and basophils. More than one organ system should be involved for the reaction to be considered anaphylaxis. The most common organ systems involved include the cutaneous, respiratory, cardiovascular, and gastrointestinal systems.

Patients often initially describe a sense of impending doom, accompanied by pruritus and flushing. This can evolve rapidly into the following symptoms, broken down by organ system:

– cutaneous/ocular — urticaria, angioedema, conjunctival pruritus, and swelling;

– respiratory — nasal congestion, rhinorrhea, throat tightness, shortness of breath, cough, hoarseness;

– cardiovascular — dizziness, weakness, syncope, chest pain, palpitations;

– gastrointestinal — nausea, vomiting, diarrhoea, bloating, cramps;

– neurologic — headache (rare, except in exercise-induced anaphylaxis) and seizure (very rare).

Symptoms usually begin within 5–30 minutes from the time the antigen is injected. If the antigen is ingested, symptoms usually occur within 2 hours, although they often occur much faster, as with severe food allergy. In rare cases, symptoms can be delayed in onset for several hours.

The first priority should be to assess the patient's respiratory and cardiac status. Respiratory:

– Severe angioedema of the tongue and lips may obstruct airflow.

– Laryngeal edema may manifest as stridor or severe air hunger.

– Loss of voice may also be observed.

– Bronchospasm, airway edema, and mucus hypersecretion may manifest as wheezing.

– Hypoxia can cause altered mental status.

Cardiovascular:

– Tachycardia is present in one fourth of patients, usually as a compensatory measure for intravascular volume loss.

– Bradycardia is more suggestive of a vasovagal reaction, although it has been observed in true anaphylaxis.

– Hypotension (and resultant loss of consciousness) may be observed secondary to capillary leak, vasodilation, and hypoxic myocardial depression.

– Cardiovascular collapse with shock can occur immediately, without any other findings.

Cutaneous:

– Urticaria (hives) can occur anywhere on the body, often localizing on
the palms, soles, and inner thighs. The lesions are erythematous, raised, highly pruritic, and of variable size.

– Angioedema is also commonly observed. These lesions involve the deeper dermal layers of skin and are usually nonpruritic and nonpitting. Common areas of involvement are larynx, lips, eyelids, hands, feet, and genitals.

– Isolated whole-body erythematous flushing is also occasionally observed.

– Gastrointestinal: Vomiting, diarrhoea, and abdominal distention are observed frequently.

Medical care. Anaphylaxis is a medical emergency requiring immediate recognition and intervention. Basic equipment and medication should be readily available in the physician's office.

– For the initial assessment, check the airway closely and secure as needed. Assess the level of consciousness and obtain blood pressure, pulse, and oximetry values.

– Place the patient in the supine position, and begin supplemental oxygen.

– Remove the source of the antigen if possible (e.g., stinger after bee sting).

– A tourniquet applied to the extremity with the antigen source can retard antigen exposure to the systemic circulation. Release the tourniquet every
5 minutes, and do not leave it in place for longer than 30 minutes.

58.Principles of diagnostics and intensive therapy of pulmonary edema.

Pulmonary oedema is an emergency state caused by filtration of fluid out of the pulmonary capillaries into the interstitial space (interstitial oedema), and eventually in the alveolar spaces (alveolar oedema).

Diagnostics:

Increased pulmonary capillary pressure is caused by any type of left ventricular failure (acute myocardial infarction or chronic heart failure) and by mitral valve stenosis. A pressure above 20 mmHg causes interstitial oedema, and as the pressure rises above 4 kPa, alveolar oedema develops.

The patient is severely dyspnoeic, with tachypnoea, tachycardia, and coughing up a frothy pink sputum containing red cells. There is basal crepitation by auscultation and often whistling bronchi.

Initially, the non-affected alveoli are overventilated and $PACO_2$ is low. Hypercapnia is a late complication when the gas exchange is severely compromised.

Therapy key points:

– Primarily, it is important to find the cause of pulmonary oedema, such as left cardiac failure, and correct the disorder.

– Patients with chronic cardiac failure have reduced contractility, which improved by positive inotropic agents such as digoxin.

– Patients with lung oedema must sit down in bed and calm down. This reduces venous return and cardiac output and the effective filtration pressure.

– Breathing of the air enriched with oxygen reduces hypoxia and dilatates the lung vessels. The filtration pressure is reduced.

– Effective diuretics increase the excretion of Na^+ and water via the kidneys. The loss of fluid also implies oedema fluid.

– Positive pressure breathing is thought to minimise the difference between the central and the peripheral venous pressure, so the venous return and cardiac output are reduced. The blockade of lung capillary bloodflow in the overpressure-phase, and the fear of the patient (increases cardiac output) do not make this treatment the best of choice.

First-line actions:

– oxygen and intubation as needed;

– nitroglycerine;

– furosemide IV 0.5–1.0 mg/kg;

– morphine IV 2 to 4 mg;

– if the patient's BP is adequate, sitting upright position;

– monitor O_2 saturation with pulse oximeter.

59.Hypertensive crisis. Pathogenesis, clinical manifestations, diagnosis and intensive therapy.

Signs and symptoms of a hypertensive crisis that may be life-threatening may include:

- Severe chest pain
- Severe headache, accompanied by confusion and blurred vision
- Nausea and vomiting
- Severe anxiety
- Shortness of breath
- Seizures
- Unresponsiveness

Certain tests will be performed to monitor blood pressure and assess organ damage, including:

- Regular monitoring of blood pressure
- Eye exam to look for swelling and bleeding
- Blood and urine testing

Treatment:

ACE inhibitors

Beta blockers

Calcium channel blockers

Diuretics

60.Intensive therapy in case of complicated myocardial infarction.

61.Pulmonary embolism. Pathogenesis, clinical manifestations, diagnostics and intensive therapy.

Pulmonary embolism (PE) is an extremely common and highly lethal condition and a leading cause of death in all age groups. Deep vein thrombosis (DVT) and PE are much more common than usually realized. Most patients with DVT develop PE and the majority of cases are unrecognized clinically.

Pathophysiology: pulmonary thromboembolism is not a disease. Rather, it is an often fatal complication of underlying venous thrombosis. Under normal conditions, microthrombi (tiny aggregates of red cells, platelets, and fibrin) are formed and lysed continually within the venous circulatory system.

Under pathological conditions, microthrombi may escape the normal fibrinolytic system to grow and propagate. PE occurs when these propagating clots break loose and embolize to block pulmonary blood vessels.

History. PE is so common and so lethal that the diagnosis should be sought actively in every patient with any chest symptoms that cannot be proven to have another cause.

Symptoms that should provoke a suspicion of PE must include chest pain, chest wall tenderness, back pain, shoulder pain, upper abdominal pain, syncope, hemoptysis, shortness of breath, painful respiration, new onset of wheezing, any new cardiac arrhythmia, or any other unexplained symptom referable to
the thorax.

The classic triad of signs and symptoms of PE (hemoptysis, dyspnea, chest pain) are neither sensitive nor specific. They occur in fewer than 20 % of patients with the diagnosis of PE, and most patients with such symptoms are found to have some aetiology other than PE.

Patients with PE often present with primary or isolated complaints of seizure, syncope, abdominal pain, high fever, productive cough, new onset of reactive airway disease ("adult-onset asthma"), or hiccoughs. They may also have new-onset atrial fibrillation, disseminated intravascular coagulation.

Physical. Massive PE causes hypotension due to acute cor pulmonale, but
the physical examination findings early in submassive PE may be completely normal. Initially, abnormal physical findings are absent in most patients with PE.

After 24–72 hours, loss of pulmonary surfactant often causes atelectasis and alveolar infiltrates that are indistinguishable from pneumonia on clinical examination and by x-ray. New wheezing may be appreciated. If pleural lung surfaces are affected, pulmonary rub may be heard.

Emergency department care. Fibrinolytic therapy has been the standard of care for all patients with massive or unstable PE since the 1970s. Unless overwhelming contraindications are evident, a rapidly acting fibrinolytic agent should be administered immediately to every patient who has suffered any degree of hypotension or is significantly hypoxemic from PE.

Heparin reduces the mortality rate of PE because it slows or prevents clot progression and reduces the risk of further embolism. Heparin does nothing to dissolve clot that has developed already, but it is still the single most important treatment that can be provided, because the greatest contribution to the mortality rate is the ongoing embolization of new thrombi.

Oxygen should be administered to every patient with suspected PE, even when the arterial PO_2 is perfectly normal, because increased alveolar oxygen may help to promote pulmonary vascular dilatation.

General management of pulmonary embolism:

1. Give oxygen with FIO_2 0.6–1. 1.0 to maintain $SaO_2 \geq 90–95$ %.

2. Place a patient in a flat position; improvement often follows increased venous return.

3. Provide fluid replasement to optimise right heart filling.

4. Give epinephrine infusion if circulation still compromised.

5. Provide mechanical ventilation if the patient is tired or cannot maintain adequate oxygenation. Gas exchange may worsen due to loss of preferential shunting and decrease in cardiac output.

6. Provide anti-coagulation

7. Consider pulmonary embolectomy.

62.Definition, types and pathophysiology of Acute Respiratory Failure (ARF).

Respiratory failure is a syndrome in which the respiratory system fails in one or both of its gas exchange functions: oxygenation and carbon dioxide elimination in the normal work of breathing.

In practice, respiratory failure is defined as a PaO_2 value of less than 60 mm Hg while breathing air or a $PaCO_2$ of more than 50 or less than 30 mm Hg. Respiratory failure may be acute or chronic.

The problems are as follows: central — a problem of respiratory drive, peripheral — a problem of the respiratory pump, large airway — a problem of gas transfer, or alveolar — a problem of gas exchange.

Types of Respiratory Failure:

– ***Hypoxemic respiratory failure (type I)*** is characterized by a PaO_2 of less than 60 mm Hg with a normal or low (less than 30 mm Hg) $PaCO_2$.

– ***Hypercapnic respiratory failure (type II)*** is characterized by a $PaCO_2$ of more than 50 mm Hg. Actually the process of CO_2 elimination is called ventilation.

Pathophysiological mechanisms of Hypoxemic respiratory failure (type I) include the following:

– Ventilation/Perfusion Mismatch;

– Diffusion abnormality.

63.Principles of ARF treatment.

ARF emergency therapy includes:

– airway opening;

– oxygen therapy;

– normalization of sputum drainage;

– mechanical ventilation/noninvasive ventilation.

Airway opening. The so-called "**Safar triple method**" to provide straight open airway includes:

– ***tilting the victim's head*** (do not overtilt, the position is supposed to be as if one is "scenting the morning air";

– ***lifting the victim's mandible***;

– ***opening the victim's mouth***.

Additional devices (adjuncts) to basic airway techniques:

– oropharyngeal airways;

– nasopharyngeal airways;

– tracheal tube;

– laryngeal mask airway (LMA);

– conicotomy, trachestomy.

64.Mechanical Ventilation (MV). Methods of mechanical ventilation. Indications and technique. Complications, their prevention and treatment.

Clinical criteria for mechanical ventilation:

– apnea or bradypnea less than 10;

– tachypnoea more than 40;

– respiratory distress with altered mentation;

– coma;

– clinically apparent increased work of breathing;

– obtundation and need for airway protection;

– controlled hyperventilation (e.g. in head injury);

– severe circulatory shock.

Laboratory criteria for mechanical ventilation:

– PaO_2 < 55 mm Hg;

$PaCO_2$ > 50 mm Hg ;

– PH < 7.32.

Pulmonary function tests for mechanical ventilation:

– vital capacity < 10 mL/kg;

Negative inspiratory force < 25 cm H_2O;

– FEV1 < 10 mL/kg.

Indications:

– hypoxaemia requiring high FIO_2;

– optimisation of pressure-volume curve in severe respiratory failure;

– hypoxaemia secondary to left heart failure;

– improvement of cardiac output in left heart failure;

– reduced work of breathing during weaning in patients with high PEEP;

– neurogenic pulmonary oedema.

Types of support. Most ventilators can be set to apply the delivered tidal volume in control or support mode.

***Control mode*.** In control mode, the ventilator delivers the present tidal volume once it is triggered regardless of patient effort. If the patient is apneic or possesses limited respiratory drive, control mode can assure delivery of appropriate minute ventilation.

***Support mode*.** In support mode, the ventilator provides inspiratory assistance through the use of an assist pressure. The ventilator detects inspiration and supplies an assist pressure during inspiration; it terminates the assist pressure upon detecting the onset of the expiratory phase. Support mode requires adequate respiratory drive.

Two basic types of ventilators:

1. ***Pressure cycled ventilators*** deliver gas into the lungs until a prescribed pressure is reached, when inspiratory flow stops and, after a short pause, expiration occurs by passive recoil.

This has the advantage of reducing the peak airway pressures without impairing cardiac performance in situations such as ARDS. However, if
the airway pressures increase or compliance decreases the tidal volume falls, so patients need to be monitored closely to avoid hypoventilation.

2. **Volume cycled ventilators** deliver a preset tidal volume into the lungs over a predetermined inspiratory time (usually ~30 % of the breathing cycle), hold the breath in the lungs (for ~10 % of the cycle), and then allow passive expiration as the lungs recoil.

65.Methods of oxygen therapy. Indications and technique.

Giving patients supplemental O_2 to breathe is one of the most common therapeutic manoeuvres. The risks of O_2 therapy are small, and O_2 should be given as an emergency procedure to all patients in respiratory distress. Simultaneously, PaO_2 and $PaCO_2$ should be measured, so that subsequent O_2 therapy can be applied rationally to raise PaO_2, and to prevent or reverse severe tissue hypoxia.

Hudson-type masks do not give precise FIO_2 and should only be used when hypoxemia is not a major concern. But Hudson-type masks do allow delivery of humidified gas.

Masks fitted with a Venturi valve deliver a reasonably accurate FIO2 (0.24, 0.28, 0.35, 0.40, 0.60) except for the patients with very high inspiratory flow rates.

A tight-fitting anaesthetic mask and reservoir bag allows 100 % oxygen to be delivered.

In case of O_2 therapy application there are essentially two risks: O_2 toxicity and CO_2 narcosis. Parenchymal lung damage from oxygen occurs with FiO_2 > 60 % for more than 48 hours without intermittent periods of breathing air.

Methods of sputum drainage normalization include:

– intravenous rehydration therapy;

– medication inhalation;

– vibration percussion, vacuum massage;

– postural drainage;

– cough stimulation and imitation;

– sputum aspiration;

– tracheobronchial lavage/bronchoscopy.

66.Noninvasive Positive Pressure Ventilation (NIPPV). Indications and technique. Complications, their prevention and treatment.

Many patients with ventilatory failure can be successfully treated with noninvasive positive pressure ventilation (NIPPV). NIPPV improves gas exchange, reduces the work of breathing, and relieves dyspnoea. Patients who most likely benefit include those with acute hypercapnic exacerbations of chronic obstructive pulmonary disease (COPD) or with hypercapnic forms of acute cardiogenic pulmonary edema. In selected patients with acute hypoxemic nonhypercapnic respiratory failure, NIPPV may obviate the need for endotracheal intubation. Selection may require exclusion of patients with hemodynamic instability, central neurologic dysfunction, or an inability to protect the upper airway.

Clinical criteria for mechanical ventilation:

– apnea or bradypnea less than 10;

– tachypnoea more than 40;

– respiratory distress with altered mentation;

– coma;

– clinically apparent increased work of breathing;

– obtundation and need for airway protection;

– controlled hyperventilation (e.g. in head injury);

– severe circulatory shock.

Laboratory criteria for mechanical ventilation:

– PaO_2 < 55 mm Hg;

$PaCO_2$ > 50 mm Hg ;

– PH < 7.32.

Pulmonary function tests for mechanical ventilation:

– vital capacity < 10 mL/kg;

Negative inspiratory force < 25 cm H_2O;

– FEV1 < 10 mL/kg.

67.Indications for tracheostomy and conicotomy. Complications. Specific care of patients with tracheotomy tubes.

68.Intensive therapy of severe Community-Acquired Pneumonia (CAP).

Treatment of CAP. General:

– *antimicrobials (empirical antibiotics).* Antibiotic therapy should cover community-acquired pathogens. Antibiotics should be administered intravenously and only stepped down to oral administration in patients with a good clinical response;

– oxygen;

– mechanical ventilation;

– fluid to correct dehydration and provide maintenance requirements;

– organ support.

69.Resuscitation and intensive therapy of Foreign-Body Airway Obstruction (FBAO).

Patients with complete airway obstruction require immediate medical attention and typically are aphonic and unable to breathe. Patients who are coughing, gagging, and vocalizing have partial obstruction.

- Use of the Heimlich maneuver has improved the mortality rate of patients with complete airway obstruction, but use of it in patients with partial obstruction may produce complete obstruction.
- Most patients who arrive at the hospital are beyond the acute stage and are not in respiratory distress.
- After a complete history and physical examination are completed and radiographic studies are performed, a decision is made in regard to the need for surgical intervention.
- In most cases, antibiotics and steroids are not administered initially.

70.Intensive therapy of Severe Life-Threatening Asthma.

Severe life-threatening asthma.

First-line therapy:

– oxygen 4 L/min O_2 (SpO_2 > 90 %);

– inhaled B2 agonists such as albuterol or salbutamol;

– infusion therapy;

– systemic corticosteroids;

– methylxanthines (aminophylline);

– ipratropium bromide;

– magnesium sulphate;

– epinephrine or terbutaline.

Indications to tracheal intubation for life-threatening asthma:

– violation of consciousness;

– progression of ARF, despite adequate therapy;

– severe cardiac arrhythmia;

– progressive acidosis (pH < 7.2);

– progressive hypoxemia;

– hypercapnia;

– respiratory depression, respiratory muscle fatigue.

When the patient is sitting upright, using accessory muscles in neck and chest to breathe he is at risk of sudden respiratory failure.

When the patient is in the state of somnolence, confusion, or exhausted — it may indicate that he has impending respiratory arrest.

71.Intensive therapy of Pulmonary Oedema.

First-line actions:

– oxygen and intubation as needed;

– nitroglycerine;

– furosemide IV 0.5–1.0 mg/kg;

– morphine IV 2 to 4 mg;

– if the patient's BP is adequate, sitting upright position;

– monitor O_2 saturation with pulse oximeter.

72.Intensive therapy of Acute Respiratory Distress Syndrome (ARDS).

Management — supportive measures. There are no established treatments for ARDS, but treating the underlying condition (for example eradicating infection with antibiotics or surgery) and providing support for each system are paramount.

Management of patients with ARDS:

– search and treat the underlying cause;

– provide supportive therapy;

– ventilatory management (ventilate at low tidal volume, apply generous PEEP);

– rescue therapies (maintain a low hydrostatic pressure in the lungs (avoid fluid overload));

– non-ventilatory management (the prone position in severe cases, consider steroids in persistent ARDS).

73.Intensive therapy of aspiration syndrome.

74.Hyperbaric oxygenation therapy (HBOT). Indications and contraindications.

Hyperbaric oxygenation therapy

The word "hyperbaric" is from the Greek root "hyper" meaning "*over, above*" and "baro" meaning "*weight*". Therefore, hyperbaric is "above
the (normal) weight" of the atmosphere.

Hyperbaric Oxygenation Therapy (HBOT) is the treatment method of using hyper-pressurized oxygen. The principles of such treatment are based on physical laws, which regulate gas dissolution in body fluids and gas propagation in tissues.

Oxygen in blood is chemically linked with hemoglobin (19.1 volume percents), as well as dissolved in plasma (0.3 volume percents). Normally it is
the hemoglobin of red blood cells that delivers oxygen to tissues, while dissolved fraction plays only a regulatory function in this process.

Diseases, associated with impairment of oxygen delivery to organs and tissues, lead to hypoxia (oxygen deprivation). Vital organs (head, brain, kidneys, liver) are extremely sensitive to the lack of oxygen and are not able to function normally under oxygen deficit conditions.

Hypoxia can have various causes, such as disruption of vascular permeability, damage of blood-supplying organs (atherosclerosis, inflammation, edema, etc.), low hemoglobin contents and many other reasons, related to respiratory pathology, cardiac abnormalities, etc.

Such conditions can be treated not only with medication, but also with oxygenation therapy. However, under normal atmospheric pressure even pure oxygen respiration cannot solve the

hypoxia problem at the level of cells and tissues. The sole solution is to increase the contents of oxygen, transferred by blood.

HBOT sessions are conducted in a special pressure chamber (hyperbaric apparatus), where high oxygen pressure is created under hermetic conditions. Modern hyperbaric apparatuses, manufactured by Russian and international producers, provide wellness and comfort during treatment sessions. The patient can either seat or lie in a free pose, inhaling the flow of curing oxygen. He or she can even sleep during the session.

Every patient passes thorough pre-HOT medical check with laboratory tests, diagnosis and is prescribed an individual treatment plan, which may include various forms of therapy apart from HOT, if necessary.

Duration and number of HOT sessions is determined individually, depending on the diagnosis and indications. Usually the whole treatment program takes from 5 to 12 sessions, depending on particular pathology, of 45–60 minutes each.

The patient's condition is constantly monitored by his/her attending physician. Usually patients feel good during HOT sessions. Constant control guarantees absence of any advers effects.

In case of Hyperbaric Oxygen Therapy the patient's entire body is exposed to pure oxygen at above-atmospheric pressures.

Hyperbaric Oxygen Therapy speeds healing in patients with problem wounds, decreases swelling in the tissues and contributes to the acceptance of skin grafts. HBO benefits patients by:

– increasing oxygen levels in the blood stream;

– helping new blood vessels form in injured tissue;

– helping the body more effectively fight infection and kill bacteria;

– helping reverse the toxic effects of poisons, such as carbon monoxide;

– decreasing the size of air bubbles and allow more blood and oxygen to reach your tissues.

75.Definition of coma, classification by etiology and severity, pathophysiology.

Definition. *Coma* (from the Greek *komas*, or deep sleep) is a state of unresponsiveness in which the patient is incapable of arousing to external or internal stimuli (lack of alertness). **Coma is a clinical syndrome characterized mainly by the decreased or absent consciousness and protective reflexes**. The degree of coma can vary from lighter stages with observed changes in autonomic function or brief moaning to strong stimulation, to the deepest stage with absence of any brain stem responses (e.g., pupillary and corneal reflexes), cyclic autonomic activity, and motor tone.

Causes of altered mental status include the following (**AEIOU TIPS**):

A: Alcohol, other toxins, drugs.

E: Endocrine, electrolytes.

I: Insulin (diabetes).

O: Oxygen, opiates.

U: Uremia (renal, including hypertension).

T: Trauma, temperature.

I: Infection.

P: Psychiatric, porphyria.

S: Subarachnoid hemorrhage, space-occupying lesion.

Assess the the Glasgow Coma Scale (GCS). The GCS is the globally accepted method of quantifying and recording the neurological status of the head-injured patient. It is also useful in determining any improvement or deterioration in neurological function and facilitates accurate communication between health professionals. The scale is made up of three sections, with a minimum score of 3 and a maximum of 15. The components of GCS are:

***Eye opening*:**

– spontaneously — 4;

– to speech — 3;

– to pain — 2;

– none — 1.

***Verbal response*:**

– orientated — 5;

– confused — 4;

– inappropriate — 3;

– incomprehensible sounds — 2;

– none — 1.

***Motor response*:**

– obeys commands (for movement) — 6;

– purposeful movement to painful stimuli (“localises”) — 5;

– withdrawal from painful stimuli — 4;

– abnormal (spastic) flexion, decorticate posture — 3;

– extensor (rigid) response, decerebrate posture — 2;

– none — 1.

76.Intensive therapy and monitoring of patients in coma.

Coma: immediate management

Priorities:

– Stabilize the patient (airway, breathing, circulation). Give oxygen (**Give thiamine, dextrose, naloxone, or flumazenil. Check BM stix)**

– Consider giving thiamine, dextrose, naloxone, or flumazenil.

– Examine the patient. Is there meningism? Establish Glasgow Coma Scale score. Is there evidence of brainstem failure? Are there focal or lateralizing signs?

– Plan for further investigations.

– Observe for signs of deterioration and attempt to reverse them.

Monitoring progress:

– This requires regular observations of vital signs and a neurological state (including GCS score).

– An important cause of deterioration in structural brain lesions is brain shift leading to herniation syndromes.

– Other reasons for deterioration are electrolyte or metabolic changes, hypovolaemia, or fluid overload. Plasma electrolytes and fluid balance need to be regularly assessed to avoid such problems.

Prognosis. In coma due to head injury, prognosis is clearly related to GCS score. Patients scoring 8 or less have a very poor prognosis. In non-traumatic coma, GCS alone is not a very good predictor. Patients with drug intoxications may have very low scores on admission but, in general, have good outcomes. Assessment of prognosis in non-traumatic coma is aided by simple features of the examination. For example, if after 24 hours it is still not possible to elicit pupillary responses, corneal reflexes, and oculovestibular response, survival is extremely unlikely.

77.General Toxicology. Classification of poisonings. Stages of poisoning. Intensive therapy at different stages of poisoning.

Toxicology is the study of the effects of poisons.

The most common classification of intoxication is:

I. Accidental intoxication:

- Occupational.

- Domestic:

a) as a result of self-treatment;

b) alcohol or drug intoxication;

c) medication overdose.

- Medical mistakes.

II. Deliberate intoxication:

- Criminal:

a) homicide;

b) as a way of bringing in a helpless state.

- Suicide attempt.

Classification according to the toxicity of substances:

1. *Extremely toxic* (chemical weapons, senile acid compounds);

2. *Highly toxic* (methanol, dichloretan);

3. *Moderately toxic* (benzol, phenol, herbicides);

4. *Low toxic* (some herbicides and insecticides).

Classification according to the system-organ tropism and clinical manifestations:

1. ***Heart poisons*** (arrhythmias, myocarditis): glycosides, tricyclic antidepressants, barium and potassium compounds;

2. ***Neural toxins*** (psychoses, seizures, coma): drugs, hypnotics, CO, alcohol and its substitutes);

3. ***Liver toxins*** (hepatopathy): toxic fungi, phenols, aldehydes;

4. ***Blood toxins*** (hemolysis, methemoglobinemia): anilines, nitrites;

5. ***Lung toxins*** (edema, fibrosis): NO-substances, phosgene;

6. ***Renal toxins*** (nephropathy, ARF): ethylenglycole, heavy metal compounds;

7. ***Gastrointestinal intoxication*** (gastroenteritis): acid and alkaline substances, heavy metals.

1. Toxicogenic stage;

2. Somatogenic stage.

Toxicogenic stage is defined as a period of time, when the toxin is in the organism in an amount that can cause a specific effect (exotoxic shock, coma). This stage includes resorbtion period (maximal concentration in the blood) and elimination (complete withdrawal of toxin).

Somatogenic stage is defined as a period of time after the elimination or degradation of toxin with consequences of toxic injury of the organs and systems (acute renal and hepatic failure, pneumonia, sepsis).

GENERAL PRINCIPLES OF INTENSIVE THERAPY IN TOXICOLOGY

Treatment principles include the following strategies:

- reduce absorption,
- increase elimination,
- provide general supportive measures,
- use of specific antidotes.

78.General principles of acute poisoning treatment.

Treatment principles include the following strategies:

- reduce absorption,
- increase elimination,
- provide general supportive measures,
- use of specific antidotes.

79.Specific features of acute alcohol/ its substitutes intoxication treatment.

Alcohol is rapidly absorbed from the gut, and therefore gut decontamination is unlikely to be of benefit. Intravenous thiamine (e.g. Pabrinex®) should be given to chronic alcohol abusers to protect against the onset of Wernicke's encephalopathy. This should be achieved before administration of glucose to treat hypoglycemia. Hypoglycemia should be treated as quickly as possible with oral glucose if the patient is awake, or otherwise with intravenous 5% or 10% glucose.

If facilities allow, hemodialysis should be considered in case the blood concentration is greater than 5g.L-1, if arterial pH is <7.0, or if the patient's condition deteriorates in spite of maximal supportive measures.

80.Specific features of carbon monoxide poisoning treatment.

Treatment guidelines:

• Remove from exposure.

• Give oxygen in as high concentration as possible to reduce the half-life of caboxyhemoglobin and hence improve oxygen delivery to the tissues. Pulse oximetry is unreliable in carbon monoxide poisoning, as it overestimates oxygen saturation.

• Metabolic acidosis generally improves with oxygen therapy. However, if acidosis persists or has a severe form it can be corrected with sodium bicarbonate.

• If a patient has been exposed to carbon monoxide due to a house fire consider the possibility of concurrent cyanide poisoning and treat accordingly.

• Treat raised intracranial pressure conventionally.

• Use of hyperbaric oxygen should be discussed with the national/ regional poisons unit.

81.Specific features of mushroom poisoning treatment.

Treatment of Mushroom poisoning includes the following:

- Intensive care of acute intoxication according to the general principles;
- Treatment of gastrointestinal intoxication stage;
- Treatment of toxic hepatitis;
- Infusion therapy to perform rehydration, detoxication and parenteral nutrition;
- In severe cases (ARF, AHF) methods of efferent therapy - hemodialysis, plasmapheresis, are performed.

82.Specific features of drug (sedatives) poisoning treatment.

83.Specific features of narcotic poisoning treatment.

Airway and breathing; significant CNS and respiratory depression are the most common life-threatening developments. It is reversal with antidote therapy. Patients with noncardiogenic pulmonary edema may require oxygen and BiPAP, CPAP, or mechanical ventilation with PEEP.

GI decontamination may be considered after oral overdose. Syrup of ipecac is contraindicated. Gastric lavage is not routine. Naloxone, a pure opioid antagonist, is the antidote most frequently used to reverse opioid toxicity. Naloxone has a rapid onset of action. IV, SC, and IM routes can be used, as well as an ET tube. Naloxone is indicated for patients with opioid intoxication who have significant CNS or respiratory depression. The duration of naloxone effect is 1 to 2 hours.

Naloxone administration can precipitate acute withdrawal in chronic opioid users. When naloxone is used in this population, the dose should be started very low and slowly titrated to clinical response without withdrawal. Clinical response to naloxone administration is not pathognomonic for opioid intoxication. Other intoxications may improve with naloxone therapy as well, including valproic acid, clonidine, tramadol, captopril, and ethanol.

84.Specific features of organic phosphorus agent poisoning treatment.

Treatment guidelines:

• Avoid self contamination – wear protective clothing.

• Prevent further absorption by removing source, including soiled clothing.

• Wash patients with soap and water.

• Consider gastric lavage if ingestion occured within 1 hour.

• If intubation is required avoid suxamethonium because of prolonged effects.

• Give atropine (2mg for adults, 0.02mg/kg for children) IV every 10-30 minutes until adequate atropinisation is achieved. Continuous atropine infusions can be used in doses of 0.02-0.8mg/kg/h, titrated to effect.

• The maximum dose of atropine is required on day 1 and decreases over the next few days. When the patient improves the dose should be slowly reduced over the next 24 hours. Rebound toxicity may occur due to organophosphates being lipid soluble.

• Oximes (pralidoxime, obidoxime) reactivate phosphorylated acetylcholinesterase before deactivation occurs, and are clinically used to reverse neuromuscular blockade (atropine has no useful effect on the neuromuscular junction). The World Health Organization recommended the following dosing regime: 30mg.kg-1 pralidoxime chloride bolus followed by 8mg.kg-1.h-1 infusion. Although the evidence base for this is limited, oxime application is still recommended for patients with moderate to severe organophosphorus poisoning.

• Benzodiazepines should be given to reduce agitation and control convulsions.

85.Specific features of cauterant agent poisoning treatment.

86.Intensive care in case of snake and insect bites.

Management of snake bite

The management of snake envenomation is controversial. It can be divided into first aid and prehospital care, specific antivenom therapy and supportive therapy.

First aid and prehospital care

Reassurance and immobilization of the affected limb, with prompt transfer to a hospital are of prime importance. The application of a 'constriction band' to delay absorption and venom spread has been advocated during transit to hospital for bites to a limb.

A firm, but not tight, ligature may be applied just above the bite. The tension is correct if one finger can pass between the limb and the bandage. This will impede lymphatic drainage, but not arterial or deep venous flow. It should preferably not be released until the administration of antisnake venom. If the limb becomes oedematous the band should be advanced proximally. However, the band should not be left in place for too long, due to the risk of venous thrombo-embolism and distal ischemia. An increase in local envenomation has also been reported subsequent to release of the band.

Venous or arterial tourniquets are contraindicated.

The site of the bite should be cleaned and covered with a handkerchief or dressing. Incision and mechanical suction of the bite (intended to open the puncture wound so that suction can be more effective) may be beneficial when performed by a health care worker within a few minutes of the bite, in a victim who is more than 30 to 60 minutes from hospital.

The incision should be parallel to the axis of the extremity and should be only approximately 6 mm long and 3mm deep. Cross cuts or multiple cuts should be avoided.

Mechanical suction (e.g. the 'extractor' device found in a Sawyer first aid kit) is preferable to mouth suction, in order to avoid wound contamination with oral flora and to prevent possible envenomation of the rescuer through breaks in their oral mucosa. Suction should be maintained for about 30-60 minutes for maximal benefit, but due care should be taken as laceration of nerves, tendons and vessels has been reported following suction by untrained rescuers.

Application of cooling measures such as ice packs or cryotherapy, at the site of bite, were initially advocated, but have not been proven to be effective and this practice is not now recommended.

Antitetanus toxoid should always be given following snakebite. There is controversy about the use of drugs as a part of first aid care. It has been suggested that NSAIDS may be beneficial to relieve local pain but may precipitate bleeding, especially if the venom is vasculotoxic.

Paracetamol and/or codeine may be useful, however, there are no clear-cut recommendations for the application sedatives.

If the snake has been killed, it should be taken to hospital, or it should be left alone, since attempts to find or kill it may result in further bites. The snake, even if judged to be dead, should be handled very carefully, since decapitated heads can bite for up to one hour.

Patient assessment

Evaluation should begin with the assessment of the airway, breathing and circulatory status. Oxygen should be administered to every envenomed patient and a large bore intravenous line with normal saline or Ringer's lactate established in the unbitten limb.

Cardiac monitoring and pulse oximetry, if available, is indicated. Attempts should be made to determine whether a venomous snake has actually bitten the patient, and the severity of envenomation should be assessed.

During the initial evaluation, several locations on the bitten extremity (at the bite site and at least two sites more proximal) should be marked and the circumferences should be measured every 15 minutes until swelling is no longer progressing and every 1-4 hours thereafter. The extremity should be placed in a well-padded splint for at least 24 hours.

Laboratory investigations

Although laboratory tests are of little value in the diagnosis of snake envenomation, nevertheless they are useful for monitoring the patient's state and deciding about specific interventions and prognosis. They should include a full blood count, electrolytes, glucose, creatinine, serum amylase, creatinine phosphokinase (CPK), prothrombin time (PT), partial thromboplastin time (PTT), fibrinogen and fibrin degradation products (FDPs).

Commonly hyperkalemia and hypoxemia with respiratory acidosis may be noted, particularly with neuroparalysis. Urine examination may reveal hematuria, proteinuria, hemoglobinuria or myoglobinuria. Arterial blood gases and urine examination should be repeated at frequent intervals during the acute phase to assess progressive systemic toxicity.

Blood changes include anemia, leukocytosis (raised white cell count) and thrombocytopenia (low platelet count). The peripheral blood film may show evidence of hemolysis, especially in viperine bites. Clotting time and prothrombin time may be prolonged and a low fibrinogen may be present. Blood should be typed and cross matched on the first blood drawn from the patient, as both direct venom and antivenom effects can interfere with later cross matching. Some specialized centers can identify the species of snake involved.

Non specific ECG changes such as bradycardia and atrioventricular block with ST and T segment changes may be seen. Recently electroencephalogram (EEG) changes have also been reported in many patients of snake envenomation. They may manifest within hours of bite without any clinical features suggestive of encephalopathy.

Antivenom therapy

Antisnake venoms (ASV) are prepared by immunizing horses with venom from poisonous snakes, extracting serum and purifying it. The WHO has designated the Liverpool School of Tropical Medicine as the international collaborating center for antivenom production and testing. Antivenoms may be species specific (monovalent) or effective against several species (polyvalent)

Supportive therapy. The patient should be moved to an appropriate area of the hospital - ICU will be required for severe envenomation. Fasciotomy should be undertaken in patients with the compartment syndrome and debridement should be performed for necrotic tissue.

Coagulopathy should be corrected with fresh frozen plasma and platelets. Blood transfusion should be given to replace blood loss from hemolysis and bleeding.

Ventilatory support and hemodialysis may be necessary for pulmonary and renal complications, due to severe envenomation.

Corticosteroids are of no proven value and in fact may interfere with the effect of ASV. However, corticosteroids may be used for hypersensitivity reactions to ASV.

Prophylactic antibiotics are of no proven value. If infection occurs, broad spectrum antibiotics, such as ciprofloxacin and clindamycin, should be used.

Intravenous immunoglobin therapy has also been used for envenomation and it may improve coagulopathy, but has no effect on neurotoxicity. Certain reports on the evaluation of intravenous immunoglobin suggest that it may reduce the need for repeated antivenom therapy for envenomations associated with coagulopathy.

A compound (2-hydroxy 4-methoxy benzoic acid) isolated and purified from anatamul (Hemidesmus indicus), an Indian herb, has also been observed to have potent anti-inflammatory, antipyretic and antioxidant properties, especially against Russell's viper venom. Analgesia should be given - opioids may be required.

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Source: Belarusian State Medical University website, Anesthesiology Department

BSMU 2019

ANESTHESIOLOGY AND REANIMATOLOGY FOR 4th YEAR STUDENTS OF MEDICAL FACULTY

1. Anesthesiology and Reanimatology **:** concept, the purposes, problems:

   The **purpose** is to study the basic concepts of Anesthesiology and Reanimatology, organisation of ICU, methods of the patient's condition objective control, applied in Anesthesiology and Intensive Care

2. History of Anesthesiology and Reanimatology.

Herbal remedies;

• Alcohol;

• An extract from the mandrake fruit;

• The use of preparations similar to opium in surgery is recorded in the Ebers Papyrus.

In ancient Russia "Pagan Anesthesiology" was used.

Such methods as skin limb vessels coating with ice, squeezing the carotid arteries to lose of consciousness, etc. were used.

an English mathematician(joseph Priestley)who discovered nitrous oxide, nitric oxide, ammonia, hydrogen chloride and oxygen.

• In1775 published his research in Experiments and Observations on Different Kinds of Air.

• his technique for dental extraction under nitrous oxide general anesthesia, ended in failure when the patient cried out in pain in the middle of the operation

William Morton became the first to publicly demonstrate the use of diethyl ether as a general anesthetic , known today as the Ether Dome.

•In 1847 - was the first who use chloroform

as a general anesthetic for labor pain relief.

•John Snow (1813-1858)

In 1847 he published the first book on general anesthesia

"On the Inhalation of Ether"

In 1858 he published his second book " On Chloroform and Other Anesthetics"

•Anrep discovered the local

anesthetic effect of cocaine, and developed the methods of the Topical and Infiltration local anesthesia.

•In 1898 August Bier performed Spinal anesthesia using cocaine
for the first time.
In 1908 he described intravenous regional anesthesia for the first time.
• In 1891 Dr.Friedrich Maass performed the first documented chest compression in humans.
• The first successful (open) human defibrillation with recovery of the patient was performed by Claude Beck in 1947.
Peter Safar wrote the book "ABC of resuscitation" in 1957;
• he created the first guidelines for community-wide emergency medical services;
• he founded the International Resuscitation Research Centere ;
• he established the United States' first intensive care unit in 1958;
• he was nominated three times for the Nobel Prize in medicine.

3. Organisation of Intensive Care Units (ICU).

4. Methods of the patient's condition objective control, applied in anesthesiology and intensive care.(3-4)

*Intensive care is a complex of medical actions aimed at restoration of the vital functions of the patients in critical conditions (when one or more vital functions disturbed to such an extend that without their artificial compensation a body cannot survive.

*ICUs receive patients from other hospital wards, including patients directly after surgery or from surgical wards, accident and emergency departments. Postoperative patients can leave a theatre in a critical state, due to the effects of the surgery and anaesthesia.

*Hospitals without an ICU should have a recovery room, where the patients can be cared for directly after the operation, and critical care or observation beds on the general ward.

*An ICU needs the presence of well trained and experienced ICU personnel 24-hours a day, 7-days a week. An ideal ICU team consists of nurses, specially trained in intensive care medicine, one or more intensivists (physicians, specialized in providing intensive care medicine) and a variable number of nurse assistants, technicians and cleaners.

*ICU personnel provide intensive care according to the National Recommendations in diagnosis and treatment of critically ill patients.

*One of the most important drugs required in the ICU is oxygen. Oxygen can be stored and supplied in various ways. Oxygen concentrators provide 90–100 % oxygen but rely on a constant electricity supply and usually do not provide oxygen flows higher than 4–6 L/min–1.

*While patient monitors measuring ECG, respiratory rate, arterial blood pressure and oxygen saturation should be available at each bed, suction machines and mechanical ventilators can be used specifically for patients in need of these devices.

*Mechanical ventilation is the major invasive intervention offered in the ICU. Patients who are unable to breathe spontaneously require some form of ventilatory support.

*A ventilator is an artificial breathing machine which moves oxygen-enriched air in and out of lungs. Being helped to breathe with the help of a ventilator, means, the patient usually needs to be sedated.

*If patients need help breathing through a ventilator, they are not be able to swallow normally, so a feeding tube can be placed in their nose, through

*the throat, and down into the stomach, and small intestines to provide nutrition, containing all the nutrients that they need, in the right amounts, including protein, carbohydrates, vitamins, minerals and fats.

*If the digestive system is not working properly, nutritional support can be provided directly into veins. A nasogastric tube, sometimes referred to as a NG tube, can be placed through the nose and down into the stomach. If necessary, it can be used to remove solid food, or liquids, from the stomach.

*Tubes inserted intravenously are used to provide a steady supply of essential fluids, vitamins, nutrients, and medication, directly into bloodstream. A tube inserted into the main veins is known as a “central line”.

*Infusion and syringe pumps allow drugs (e. g. catecholamines) and fluids to be administered at exact rates and dosage but, in the clinical practice of resource-poor settings, may well be replaced by mechanical drop regulators or close clinical surveillance by a nurse.

*ICU beds are a very expensive and a limited resource because they provide specialized monitoring equipment, a high degree of medical assistance (in Belarus usually one

doctor per six beds), and constant access to highly trained nurses (in Belarus usually one nurse per three beds).

*Some ICUs are attached to units treating specific conditions, such as heart, kidney, liver, breathing, circulation, or nervous disorders. Others specialize in the care of babies (neonatal), children (pediatrics), or deal with severe injury or trauma.

*Intensive care medicine is an integrative medical specialty, requiring close cooperation with several other medical disciplines and technical services (e. g. laboratory services, blood bank etc.) in the hospital.

*Therefore, to assure adequate and efficient care of critically ill patients, other medical departments and hospital services need to be prepared and trained to manage the needs of critically ill patients.

*Similarly, a basic set of essential disposable materials, drugs and laboratory tests need to be available to adequate and safe care for critically ill patients.

*The patient's observations, received treatments and fluid balances should be regularly documented. This enables early recognition of the deteriorating patient, monitors the success of the care and reduces errors in drugs prescription and dispensing. Documentation can also be useful for quality control.

5. Hemodynamic monitoring used in Anesthesiology and Intensive Care.

1)Electrocardiogram (ECG) monitoring: ECG monitoring in ICU usually involves display of one lead — lead 2 — and measures the electrical activity of the heart along its long axis from right to left . Three electrodes are required for this — one on the right shoulder (usually red), one on the left shoulder (usually yellow) and one more placed on the left side of the chest (usually green). Lead 2 is sensitive in detection of most arrhythmias .Using capnography , pulse oximetry and ECG monitoring can be an invaluable addition to treating a patient in the ICU setting, increasing safety and optimising treatment.

2)BP Measurements: is the simplest method of blood pressure determination estimating the systolic blood pressure by palpating the return of the arterial pulse as an occluding BP cuff is deflated.Other methods include auscultation of the Kortokoff

sounds with cuff deflation. This allows us to make both systolic (SBP) and diastolic (DBP) pressure measurements. The mean arterial pressure (MAP) can be estimated as:

MAP = DBP + 1/3(SBP – DBP).

3) Automated non-invasive BP measurements : are routinely performed intraoperatively every 3 to 5 minutes during general anaesthesia using

a microprocessor-controlled oscillotonometer such as a Dinamape.

4) Invasive (intra-arterial) blood pressure (IBP) monitoring : is a commonly used technique in the Intensive Care Unit (ICU) or in the operating theatre.

The technique involves the insertion of a catheter into a suitable artery and then displaying the measured pressure wave on a monitor.

5) A central venous pressure (CVP) catheter : provides an estimate of the right atrial and right ventricular pressures. The CVP reflects the patients blood volume, venous tone, and right ventricular performance.Serial measurements are much more useful than a single value. The HR, BP, and CVP response to a volume infusion (100–500 ml of fluid) is a very useful test of right ventricular performance.CVP monitoring is useful in patients undergoing procedures associated with large fluid volume shifts. Shock states, massive trauma, significant cardiopulmonary disease or the need for vasoactive medications are other indications for using a CVP catheter.

6) Pulmonary Artery Wedge Pressure (PCWP) : Unlike a CVP catheter that lies in the superior vena cava, the pulmonary artery catheter (PAC) passes through the right atrium and right ventricle and rests in a branch of one of the pulmonary arteries. Inflation of a plastic cuff at the tip of the catheter allows occlusion of the proximal pulmonary artery and measurement of the distal pressure.This distal (back) pressure is referred to as the pulmonary artery wedge pressure (PCWP) and reflects the left atrial filling pressure.Thermodilution calculations of cardiac output are performed by injecting a fixed volume of cool fluid into the right atrial port and measuring the temperature change over time from a thermistor probe at the distal tip of the PA catheter. A sample of blood taken from the distal tip of the PA catheter can be analyzed to determine the mixed venous oxygen saturation (SvO2).

7) Cardiac Output Monitoring : Cardiac Output (CO) informs us of global blood flow and therefore oxygen delivery (the product of cardiac output and blood oxygen content), but does not describe delivery of oxygen to each organ, whose function must be assessed individually.Cardiac output is the volume of blood ejected from each of the ventricles of the heart per minute, and is therefore the product of stroke volume and heart rate. The unit of cardiac output is L/min–1.

Methods of CO monitoring include the following : Non-invasive methods : Doppler ultrasound ,Echocardiography ,Transoesophageal echocardiography ,Thoracic bioimpedance.

Invasive methods: Pulse contour analysis , Dilution methods (Thermodilution pulse contour monitoring — PiCCO plus).

8) Urine Output Monitoring : Measurement of urine output is performed during prolonged surgery to ensure maintenance of adequate circulating volume where there is likely to be major blood loss, where diuretics are used and in all critically ill patients.Urine output needs to be measured at least hourly, aiming for a flow of approximately 1 ml/kg/h. Failure to produce urine indicates that urine blood flow is inadequate, as well as the flow to the other vital organs (heart and brain). Catheterization also eliminates bladder distention or incontinence.Oliguria is defined as a urine output < 0.5 ml/kg/h.

6. Respiratory and functional monitoring used in Anesthesiology and Intensive Care.

*respiratory:

– respiratory rate;

– end-tidal carbon dioxide concentration;

– inspired oxygen.

I. Oxygenation. Oxygenation is monitored clinically by providing adequate illumination of the patient’s colour and by pulse oximetry.

The inspired oxygen concentration (FiO2 is quantitatively monitored during all types of general anesthesia using an oxygen analyzer. Each analyzer is equipped with an audible low oxygen concentration alarm.

II. Ventilation. Ventilation is monitored clinically by verification of a correctly positioned endotracheal tube as well as by observing chest excursions, reservoir bag displacement, and breath sounds over both lung fields. Ventilation is quantitatively monitored using end tidal carbon dioxide analysis as well as an audible disconnection alarm on all mechanically ventilated patients.The measurement of expired gas volumes and the ability to perform arterial blood gas analysis are useful adjuncts in assessing the adequacy of both oxygenation and ventilation.

7. Indications for hospitalisation to the ICU.

1.Preoperative treatment in case of severe homeostasis disturbances (e. g. water-electrolyte, protein etc.).

2.Treatment after major surgery.

3.Acute circulatory failure including all types of shock.

4.Acute respiratory failure.

5.Acute renal failure.

6.Acute hepatic failure.

7.Acute CNS disturbances (severe head or spine trauma, coma, psychosis).

8.Acute metabolic disturbances (e. g. diabetic comas).

9.Acute coagulation disturbances (DIC-syndrome).

10. Severe infection (sepsis).

11. Multiple trauma.

8. Preoperative patients' physical status assessment. Anesthetic technique choice

9. Preparation of patients for elective and emergency surgery and anesthesia

10. The objectives of premedication. Drugs for premedication.

11.Common and special components of the General Anesthesia (GA).

–Hypnosis, or loss of consciousness;

– Amnesia, or loss of the procedure memory;

– Analgesia, or pain relief;

– Muscle relaxation;

– Suppression of reflex activity.

the patient is unconscious in general anesthesia and has no awareness or other sensations while, in addition the patient is carefully monitored, controlled and treated by the anesthesiologist.

12.Types of modern GA.

Types of general anesthesia are the following:

– Inhalation anesthesia;

– Noninhalation anesthesia (usually intravenous anesthesia);

– Balanced anesthesia.

Agents used for general anesthesia may be either gases or volatile liquids that are vaporized and inhaled with oxygen, or drugs delivered intravenously.

A combination of inhaled anesthetic gases and intravenous drugs are usually delivered during general anesthesia; this practice is called balanced anesthesia and is used because it takes advantage of the beneficial effects of each anesthetic agent to reach surgical anesthesia.

13.Stages and clinical signs of GA.

There are four stages in GA performed with the application of diethyl eather as defined by Guedel in 1937

•Stage One: Analgesia. The patient experiences analgesia or a loss of pain sensation but remains conscious and can communicate.

•Stage Two: Excitement. The patient may experience delirium or become violent. Blood pressure rises and becomes irregular, and breathing rate increases. This stage is typically bypassed by administering a barbiturate, (such as sodium pentothal), prior to the anesthesia.

•Stage Three: Surgical Anesthesia. During this stage, the skeletal muscles relax, and the patient's breathing becomes regular. Eye movements slow, then stop, and surgery can begin. There are 4 planes of the stage 3:

– Plane 1 ~ eyeball movements;

– Plane 2 ~ negative corneal reflex — at this stage surgery can begin;

– Plane 3 ~ moderately dilated pupillary size;

– Plane 4 ~ diaphragm breathing.

Plane 3 and 4 are the stages of overdosage.

•Stage Four: Medullary Paralysis. This stage occurs if the respiratory centers in the medulla oblongata of the brain that control breathing and other vital functions cease to function. Death can result if the patient cannot be revived quickly. This stage should never be reached. Careful control of the amounts of anesthetics prevent this event.Recovery occurs in the reversed order.

14. 1) Inhalation General Anaesthesia .2) Concept of the Minimum Alveolar Concentration (MAC). 3) General properties of inhalation anesthetics. *

15. Methods, indications, contraindications to application of inhalation GA. Complications, their prevention and treatment.(14-15)

1)Inhalation anesthesia is produced by the inhalation of vapors of a volatile liquids or anesthetic gases.

Inhaled anesthetics are compounds that enter the body through the lungs and then carried by the blood to the body tissues.

Modern Inhaled Anesthetics are Vapors (or volatile liquids) such as:

– Halothane, Enflurane , Isoflurane , Desflurane , Sevoflurane And Gassess ,Nitrous Oxide , Xenon.

Induction and recovery from anesthesia depend on the rate of partial pressure changes in the brain.These drugs are small lipid-soluble molecules crossing the alveolar membrane easily. Movement into and out of the blood is based on the partial pressure gradient.

2) MAC is the alveolar concentration of an anesthetic at 1 atmosphere that prevents movement in 50 % of patients in response to a noxious stimulus (e. g. surgical incision).

Minimum Alveolar Concentration: The MAC is a measure of the potency of an anesthetic. A low MAC means high potency. An anesthetic's potency is correlated with its lipophilicity (i. e. that is in case of low MAC the anesthetic is very lipophilic).

Dose response curve is steep — 99 % of patients are immobile in case of 1.3 MAC. MACs of two different agents are summarized (i. e. 0.5 MAC anesthetic A + 0.5 MAC anesthetic B corresponds to the effectiveness of 1.0 MAC of A or B).

MAC is age-dependent: The highest is in infants; drops to about half by the age of 80.

Analgesia begins at about 0.3 MAC; while Amnesia at about 0.5 MAC.

Thus the potency of different agents can be compared with the amount need to produce the desirable effect.

An agent with a low MAC, is a potent one because only a small amount is required to produce anaesthesia. A high MAC means the agent is weak because a large amount of it is required to produce anaesthesia.

3) Unique Features of Inhaled Anesthetics : – Speed, Gas State, and Route of Administration. The inhaled anesthetics are among the most rapidly acting drugs in existence, and when administering .a general anesthetic, this speed provides a margin of safety and also means efficiency.

– Technically, nitrous oxide and xenon are the only true gases; the other inhaled anesthetics are vapors of volatile liquids (for simplicity, all of them are referred to as gases).

– A unique advantage of anesthetic gases is the ability to deliver them to

the bloodstream via the patient's lungs.

16.Clinical Pharmacology of Halothane.

It can be used to start or maintain anaesthesia ,One of its benefits is that it does not increase the production of saliva, which can be particularly useful in those who are difficult to intubate .It is given by inhalation . Side effects include an irregular heartbeat, decreased effort to breathe, and liver problems. Like all volatile anesthetics, it should not be used in people with a personal or family history of malignant hyperthermia.

17.Clinical Pharmacology of Isoflurane .

Isoflurane is a halogenated methyl ethyl ether that has a high degree of stability and has become the "gold standard" anesthetic since its introduction in the 1970s. Coronary vasodilation is a characteristic of isoflurane, and in patients with coronary artery disease, there has been concern that coronary steal could occur (rare occurrences).

18.Clinical Pharmacology of Sevoflurane.

Sevoflurane is completely fluorinated methyl isopropyl ether with a vapor pressure similar to that of isoflurane. It can be used in a conventional vaporizer. Compared with isoflurane, sevoflurane is less soluble in blood and tissues , is less potent, and lacks coronary artery vasodilating properties. Sevoflurane has minimal odor and pungency (it is useful for mask induction of anesthesia) and is a potent bronchodilator. Similar to enflurane, the metabolism of sevoflurane results in fluoride, but unlike enflurane, this has not been associated with renal concentrating defects. Unlike other volatile anesthetics, sevoflurane is not metabolized to trifluroacetate but rather to hexafluoroisopropanol, which does not stimulate formation of antibodies and immune-mediated hepatitis. Sevoflurane does not decompose to carbon monoxide or to dry carbon dioxide absorbents but rather is degraded to a vinyl halide (compound A), which is a dose-dependent nephrotoxin in rats. Renal injury has not been shown to occur in patients, even when fresh gas flows are 1 L/min or less.

19. Clinical Pharmacology of Nitrous Oxide.

Nitrous oxide is a sweet-smelling, nonflammable gas of low potency and limited blood and tissue solubility that is most often administered as an adjuvant in combination with other volatile anesthetics or opioids.

Controversy surrounding the use of nitrous oxide is related to its unclear role in postoperative nausea and vomiting, potential toxicity related to inactivation of vitamin B12, effects on embryonic development, and adverse effects related to its absorption into air-filled cavities and bubbles (Compliant spaces such as a pneumothorax expand, and noncompliant spaces such as the middle ear experience increased pressure.)

Inhalation of 75 % nitrous oxide may expand a pneumothorax to double its size in 10 minutes . Accumulation of nitrous oxide in the middle ear may diminish hearing after surgery.

20.Equipment for inhalation anaesthesia. The scheme of an anesthetic machine.

Functional schem of an anesthesia machine/workstation. In its most basic form, the anesthesia machine receives medical gases from a gas supply; controls the flow of desired gases reducing their pressure, when necessary, to a safe level; vaporizes volatile anesthetics into the final gas mixture; and delivers the gases to a breathing circuit that is connected to the patient's airway.

The anaesthetic machine comprises:

– a means of supplying gases either from attached cylinders or from piped medical supplies via appropriate unions on the machine;

– tools of measuring gases flow rate;

– apparatus for vaporizing volatile anaesthetic agents;

– breathing systems and a ventilator for vapours and gases delivery from

the machine to the patient;

– apparatus for scavenging anaesthetic gases withdrawal to minimize environmental pollution.

21.Types of breathing circuits. Accessory Kit and devices.

Anesthesia circuits are classified as open, closed, semiopen, semiclosed, as rebreathing (no CO2 absorption) or nonrebreathing (including a CO2 absorber; e. g., circle system). In all circuits, the higher the fresh gas flow, the closer inspired gas composition approaches fresh gas.

22. General Anesthesia with spontaneous breathing (using Face mask, Laryngeal Mask Airway (LMA). Indications , contraindications to application. Complications, their prevention and treatment.

23.General Anesthesia with Controlled Ventilation (with an endotracheal tube). Indications, contraindications to application. Complications, their prevention and treatment.

During controlled ventilation (the most basic mode of all ventilators),

the next breath always occurs after a preset time interval. Thus tidal volume and rate are fixed in volume-controlled ventilation, while peak inspiratory pressure is fixed in pressure-controlled ventilation.

24.Noninhalation General Anesthesia. Indications, contraindications. Complications, their prevention and treatment.

Intravenous Anesthetics. Commonly administered intravenous general anesthetics include the following: Barbiturates; Benzodiazepines; Opioids; Propofol; Ketamine; Etomidate.

Despite thiopental's proven clinical usefulness, it has been supplanted by

a variety of drugs (midazolam, ketamine, etomidate, propofol) from different groups.

25.Clinical Pharmacology of Barbiturates.*

Barbiturates. Thiopental and thiamylal are thiobarbiturates with similar potency (adult induction dose, 3–5 mg/kg IV) and pharmacologic profile. Methohexital is an oxybarbiturate with a greater potency (adult induction dose, 1.5 mg/kg IV) than the

thiobarbiturates and is associated with a high incidence of myoclonic-like muscle tremors and other signs of excitatory activity (e. g., hiccoughing). Barbiturates cause dose-dependent depression of ventilation.Cardiovascular effects of barbiturates include decreases in blood pressure, decreased venous return because of peripheral pooling and direct myocardial depression and a compensatory increase in heart rate.

Hypotension is exaggerated in the presence of hypovolemia.

26. Clinical Pharmacology of Benzodiazepines.*

Benzodiazepines. The benzodiazepines of primary interest to anesthesiologists are diazepam, lorazepam, and midazolam and the antagonist flumazenil.These drugs are primarily used as preoperative medication and adjuvant drugs because of their anxiolytic, sedative, and amnestic properties.Diazepam and lorazepam are insoluble in water, and their formula contains propylene glycol, a tissue irritant that causes pain on injection and venous irritation.Midazolam is a water-soluble benzodiazepine that produces minimal irritation after IV or intramuscular (IM) injection. When exposed to physiologic pH, an intramolecular rearrangement occurs that changes the physiocochemical properties of midazolam so that it becomes more lipid soluble.Diazepam is metabolized to active metabolites, which may prolong its residual sedative effects. Lorazepam is directly conjugated to glucuronic acid to form pharmacologically inactive metabolites. The primary metabolite of midazolam (1-hydroxy-methylmidazolam) has some CNS depressant activity.The context-sensitive half-times for diazepam and lorazepam are very long, so only midazolam should be used for continuous infusions. Similar to the other sedative-hypnotic drugs, the benzodiazepines are potent anticonvulsants commonly used to treat status epilepticus. Benzodiazepines produce dose-dependent depression of ventilation that is enhanced in patients with chronic respiratory disease, and synergistic depressant effects occur when benzodiazepines are coadministered with opioids. Both midazolam and diazepam produce decreases in systemic vascular resistance and systemic blood pressure (accentuated with hypovolemia) when large doses are administered for induction of anesthesia, but a ceiling effect appears to exist above which little further change in arterial pressure occurs. In contrast to all other sedative-hypnotic drugs, there is a specific antagonist for benzodiazepines. (Flumazenil has a high affinity for CNS benzodiazepine receptors but possesses minimal intrinsic activity.)

27.Clinical Pharmacology of Opioids.

Opioid receptors are predominately located in the brain stem, spinal cord, and gastrointestinal tract. Narcotics exert their analgesic action by interacting with opioid

receptors in the brainstem (amygdala, corpus striatum, periaqueductal gray matter, and medulla), and in the substantia gelatinosa in the spinal cord.

Three classes of opioid receptors are primarily involved with mediation of the analgesic and anaesthetic properties of narcotics. The effects of mu (p), kappa (K) and sigma (a) receptors stimulation are summarized

Classes of opioid receptors

p (Mu) receptor :Analgesia, respiratory depression, euphoria, physical dependence

K (Kappa) receptor :Analgesia, sedation, respiratory depression, miosis

a (Sigma) receptor :Dysphoria, hallucinations, tachypnea, tachycardia

CNS :Opioids produce both sedation and interfere with the sensory perception of painful stimuli. Stimulation of the chemoreceptor trigger zone by narcotics may result in nausea and emesis.

RESP : Narcotics result in a depression of the respiratory rate and minute ventilation accompanied by an increase in the tidal volume.

CVS: Opioids have little to no myocardial depressant effects even when administered in high doses.Narcotics decrease systemic vascular resistance (SVR) by either decreasing sympathetic outflow or, in the case of morphine and meperidine, by direct release of histamine.

Synthetic opioids, such as fentanyl and its related congeners, are less likely to release histamine. Opioids produce bradycardia by stimulating the vagal nucleus in the brainstem.

28.Clinical Pharmacology of Propofol.

Propofol. As an alkylphenol compound, this drug is virtually insoluble in water, requiring its preparation in an egg-lecithin emulsion as 1 % (10 mg/mL) solution. Propofol is rapidly cleared from the central compartment by hepatic metabolism and the context-sensitive half-time for continuous IV infusions (≤ 8 hours) is less than 40 minutes. Emergence and awakening are prompt and complete even after prolonged infusions. Hepatic metabolism is prompt to inactive water-soluble metabolites eliminated by the kidneys. The induction dose in adults is 1.5 to 2.5 mg/kg IV, and the recommended IV infusion rate is from 100 to 200 µg/kg/min for hypnosis and from 25 to 75 µg/kg/min

for sedation. Induction of anesthesia with propofol is occasionally accompanied by excitatory motor activity (nonepileptic myoclonia). This drug is an anticonvulsant. The duration of seizure activity after electroconvulsive therapy is shorter with propofol than methohexital and is effective in terminating status epilepticus. Propofol produces dose-dependent depression of ventilation. Apnea occurs in 25 % to 35 % of patients after induction of anesthesia. Bronchodilatation may occur in patients with chronic obstructive pulmonary disease. Propofol produces greater cardiovascular depressant effects than thiopental, reflecting decreased systemic vascular resistance (arterial and venous dilation) and direct myocardial depressant effects. Propofol does not trigger malignant hyperthermia and may be considered the induction drug of choice in patients who are susceptible to malignant hyperthermia.

29. Clinical Pharmacology of Ketamine.

Ketamine is an acrylcyclohexilamine structurally related to phencyclidine.The commercially available preparation is a racemic mixture, although the S+ isomer possesses more potent anesthetic and analgesic properties, reflecting its fourfold greater affinity at the binding sites on the NMDA receptor. Ketamine produces dose-dependent CNS depression, leading to a so-called “dissociative anesthetic state” characterized by profound analgesia and amnesia. Low-dose ketamine (75–200 μg/kg/min IV) produces opioid-sparing effects when administered as an adjuvant during general anesthesia.Induction of anesthesia can be accomplished with 1 to 2 mg/kg IV (4–8 mg/kg IM), producing an effect that lasts for 10 to 20 minutes, although recovery to full orientation may require an additional 60 to 90 minutes. Subanesthetic doses of ketamine (0.1–0.5 mg/kg IV) produce analgesic effects. A low-dose infusion of ketamine (4 μg /kg/min IV) is equivalent to morphine (2 mg/hr IV) for production of postoperative analgesia .Ketamine can activate epileptogenic foci in patients with known seizure disorders but otherwise appears to possess anticonvulsant activity. Ketamine is often recommended for induction of anesthesia in patients with asthma because of its ability to produce bronchodilation. Depression of ventilation is minimal in clinically relevant doses. Increased oral secretions may contribute to the development of laryngospasm. Ketamine has prominent cardiovascular stimulating effects (increased blood pressure, heart rate, pulmonary artery pressure) most likely because of direct stimulation of the sympathetic nervous system. This is possibly undesirable in patients with coronary artery disease.

30.Clinical application of neuromuscular blocking agents. Indications, contraindications. Complications, their prevention and treatment.

Neuromuscular blocking agents have been inadvertently administered to patients who were not receiving proper ventilator assistance. Because the respiratory muscles were paralyzed, some patients have died or sustained serious, permanent injuries.

Indication :Neuromuscular blocking agents are used in critical illness to reduce metabolic demands and prevent ventilator asynchrony in patients refractory to sedation and anxiolysis.

Complication :the development of myopathy and paresis has been increasingly recognized after prolonged use of NMB drugs in the ICU . Pathophysiologic changes in the nerve, muscle, or neuromuscular junction may also play a role in the development of some cases of prolonged weakness or myopathy after discontinuation of NMB drugs.

Prevention: Limit access. When possible, have anesthesia personnel bring neuromuscular blocking agents to the scene when a patient must be intubated, or dispense neuromuscular blocking agents from the pharmacy as prescribed for patients. Allow floor stock of these agents only in the OR, ED, and critical care units where patients can be properly ventilated and monitored.

31.Balanced General Anesthesia, types and methods.

Balanced general anesthesia with controlled ventilation is a type of anesthesia that uses a combination of drugs, each in an amount sufficient to produce its major or desired effect to the optimum degree and keep its undesirable or unnecessary effects to a minimum.

Advantages:

– airway;

– reduced toxicity;

– adequate gas exchange.

Disadvantages: Requirement of sophisticated equipment and highly-professional staff.

32.Periods of General Anesthesia.

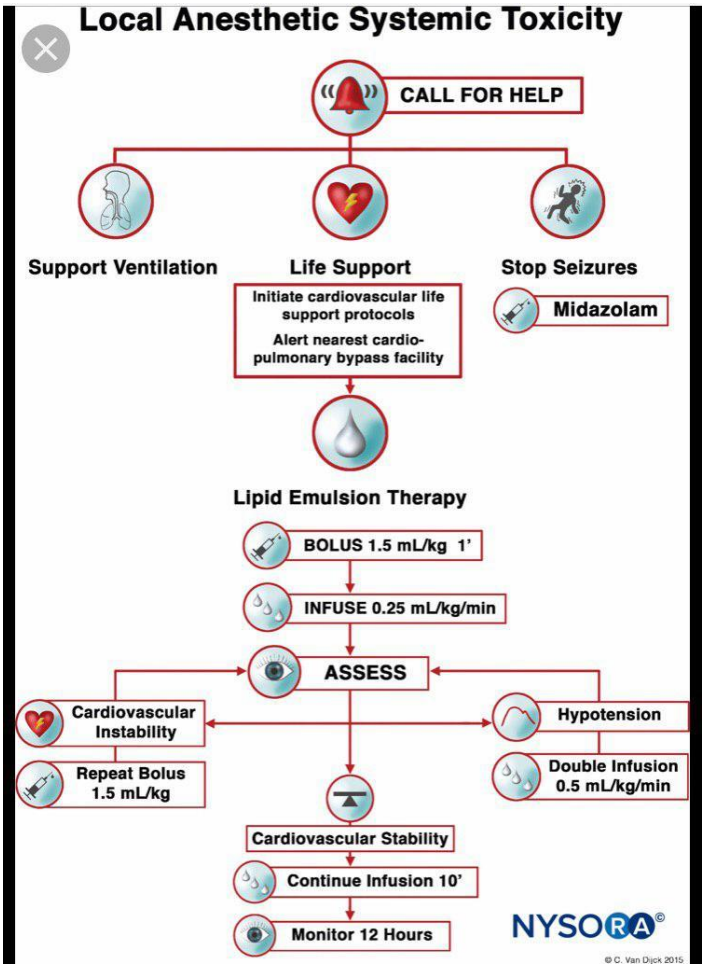
33.Types of Local Aanesthesia. Indications, contraindications.

34.Complications of Local Anesthesia, their prevention and treatment.

Complication: 1.Systemic toxicity of local anesthetics 2.Allergic reactions. 3.Mechanical damage of anatomic structures and nerve trunk. 4.Infectious complications.

If the signs and symptoms develop during administration of the local anesthetic, stop the injection immediately and prepare to treat the reaction. Ensure adequate oxygenation, whether by face mask or by intubation.

Attention to impending respiratory arrest, significant hypotension, dysrhythmias ,and seizures takes precedence .



35.Clinical Pharmacology of local anesthetics.

Local anaesthetics are drugs that reversibly block impulse conduction in nerve fibers. The molecular structure of most local anaesthetics consists of an aromatic group linked to a hydrophilic amine by either an amide link (amino amides) or an ester link (amino esters).

Unlike ester local anaesthetics, amides are metabolized in the liver and are rarely associated with allergic reactions.

Esters (procaine, cocaine, chloroprocaine, tetracaine) have a very short half-life (a few minutes), and have limited clinical use secondary to their toxicity and potential for allergic reactions.

Amides (lidocaine, mepivacaine, bupivacaine, etidoc

36.Epidural anesthesia. Indications, contraindications. Complications, their prevention and treatment.

Epidural anesthesia. The epidural space extends from the base of skull to the sacrococcygeal membrane. Posteriorly it is bounded by the ligamentum flavum, the anterior surfaces of the laminae, and the articular process. Anteriorly it is bounded by the posterior longitudinal ligament covering the vertebral bodies and intervertebral disks.local anesthetic placed in the epidural space acts directly on the spinal nerve roots located in the lateral part of the space. The onset of block is slower than with spinal anesthesia and the intensity of the sensory and motor block is decreased.The epidural space is most easily entered in the lumbar region. The patient should be positioned with the lumbar spine in maximal flexion so that the intervertebral spaces are maximally opened. This can be done in the lateral or the sitting position. In the lateral position, the patient's knees are flexed as high as possible in front of the abdomen, and the head is bent onto the chest. Epidural anesthesia must be performed utilizing an aseptic technique. The iliac crest is observed or palpated, and the L3–L4 interspace identified. The space that appears to offer the easiest access to the epidural space is closed for needle insertion. Subcutaneous infiltration may be employed to decrease the pain associated with insertion of the epidural needle.

Epidural anaesthesia typically requires 5–10 times more of LA that would be used for spinal anaesthesia. Local anaesthetics that contact the nerves directly, as in spinal anaesthesia, produce a very rapid and intense nerve block. By contrast, epidural anaesthesia has a slower onset because the nerves are, in a sense insulated, and it

produces a less intensive block. Continuous infusions of local anaesthetics and opioids into the epidural space can be used intraoperatively and continued postoperatively.

37.Spinal anesthesia. Indications, contraindications. Complications, their prevention and treatment.

Spinal anesthesia is a drug injection into lumbar subdural space below the termination of the spinal cord.

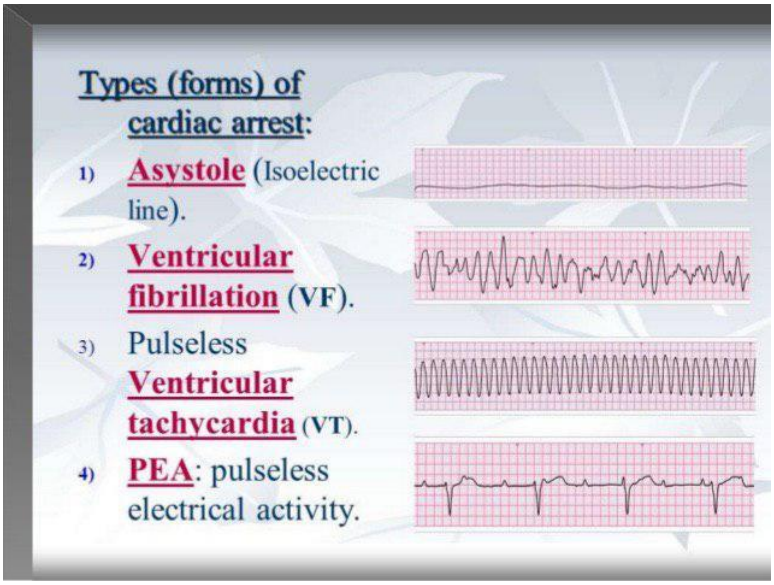
Drug diffuses within the dura to effect on nerve trunks coming to or from the spinal cord. The spinal cord usually ends at the level of L2 in adults and L3 in children. Dural puncture above these levels is associated with a slight risk of damaging the spinal cord and is better to avoid. An important landmark to remember is that a line joining the top of the iliac crests is at L4 to L4/5. All patients with spinal anaesthesia must have a large intravenous cannula inserted and be given intravenous fluids immediately before the spinal. The volume of fluid given will vary with the age of the patient and the extent of the proposed block.

38.Types of terminal conditions: death agony, clinical death. Pathologic physiology and clinical signs.

1) Death agony (agony of death means inconscious state of a person who is about to die);

2) Clinical death (is the medical term for cessation of blood circulation and breathing). Sudden cardiac arrest is responsible for more than 60 % of adult deaths from coronary heart disease.

39.Types of cardiac arrest, signs and diagnosis.



Cardiac arrest is a sudden loss of blood flow resulting from the failure of the heart to effectively pump. Signs include loss of consciousness and abnormal or absent breathing. Some individuals may experience chest pain, shortness of breath, or nausea before cardiac arrest. If not treated within minutes, it typically leads to death.

Diagnosis: Cardiac arrest is synonymous with clinical death. 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, as lack of a pulse (particularly in the peripheral pulses) may result from other conditions (shock), or simply an error on the part of the rescuer.

40.Main principles and ways of Basic Life Support (BLS).

Basic Life Support (BLS) is a temporary delivery of oxygen to vital tissues is accomplished by providing effective airway management, ventilation, and artificial circulation, with or without supportive equipment.

Basic Life Support includes the following steps:

A — Airway opening : Obstruction of the hypopharynx by the base of the tongue is the most common cause of airway obstruction in the unconscious persons. The unsupported tongue falls against the posterior pharyngeal wall, obstructing the airway.

“Safar triple method” to provide straight open airway includes: 1– tilting the victim’s head (do not overtilt , the position is supposed to be as if one is “scenting the morning air”; 2– lifting the victim’s mandible; 3– opening the victim’s mouth.

B — Breathing :

Breathing types : 1)Mouth-to-Mouth 2)Mouth-to-Nose.

How to provide breathing:Pinch closed the soft part of the nose, using the index finger and thumb of your hand on the forehead.Allow the mouth to open, but maintain the chin lift.

Take a normal breath and place your lips around his mouth, making sure that you have a good seal.Blow steadily into his mouth while watching for his chest to rise.

Take your mouth away from the victim and watch for his chest to fall as air comes out.

Successful resuscitation depends on rescucitator ensuring adequate ventilation with every breath by using criteria such as observing the patient's chest rise and fall, feeling the patient's lung compliance during lung inflation, and hearing and feeling the air escape during ventilation.

The volume of air, required for each inflation to be been quoted as

800–1200 ml, with each breath taking 1–1.5 s. It has been shown recently that a tidal volume of 400–500 ml is sufficient to provide adequate ventilation in adults BLS because carbon dioxide delivery during cardiac arrest is very low.

C — Circulation : Checking the carotid pulse (or any other pulse) confirm the presence or absence of circulation.

Circulation (Chest compression). Start chest compression as follows:

– Place the heel of one hand in the centre of the victim's chest.

– Place the heel of your other hand on the top of the first hand.

– Interlock the fingers of your hands and ensure that pressure is not applied over the victim's ribs.

– Do not apply any pressure over the upper abdomen or the bottom end of

the bony sternum.