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# BASICS OF ANESTHESIOLOGY AND REANIMATOLOGY



Minsk BSMU 2016

МИНИСТЕРСТВО ЗДРАВООХРАНЕНИЯ РЕСПУБЛИКИ БЕЛАРУСЬ
БЕЛОРУССКИЙ ГОСУДАРСТВЕННЫЙ МЕДИЦИНСКИЙ УНИВЕРСИТЕТ
КАФЕДРА АНЕСТЕЗИОЛОГИИ И РЕАНИМАТОЛОГИИ

**Р. Е. РЖЕУТСКАЯ**

# ОСНОВЫ АНЕСТЕЗИОЛОГИИ И РЕАНИМАТОЛОГИИ

# BASICS OF ANESTHESIOLOGY AND REANIMATOLOGY

Рекомендовано Учебно-методическим объединением
по высшему медицинскому, фармацевтическому образованию
Республики Беларусь в качестве учебно-методического пособия
для студентов учреждений высшего образования, обучающихся
на английском языке по специальности 1-79 01 01 «Лечебное дело»

Минск БГМУ 2016

УДК 616-089.5-036.882(811.111)-054.6(075.8)
ББК 54.5:53.5 (81.2 Англ-923)
Р48

Р е ц е н з е н т ы: канд. мед. наук, доц. каф. анестезиологии и реаниматологии с курсом ФПК и ПК Витебского государственного ордена Дружбы народов медицинского университета Е. В. Никитина; канд. мед. наук, доц. каф. анестезиологии и реаниматологии Гродненского государственного медицинского университета К. М. Бушма

**Ржеутская, Р. Е.**

Р48 Основы анестезиологии и реаниматологии = Basics of anesthesiology and reanimatology : учеб.-метод. пособие / Р. Е. Ржеутская. – Минск : БГМУ, 2016. – 68 с.

ISBN 978-985-567-459-8.

Содержит краткую информацию по основным направлениям современной анестезиологии и реаниматологии в пределах учебной программы.

Предназначено для студентов 4–6-го курсов, обучающихся на английском языке.

**УДК 616-089.5-036.882(811.111)-054.6(075.8)**
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Учебное издание

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# ОСНОВЫ АНЕСТЕЗИОЛОГИИ И РЕАНИМАТОЛОГИИ

# BASICS OF ANESTHESIOLOGY AND REANIMATOLOGY

Учебно-методическое пособие

На английском языке

Ответственный за выпуск О. Т. Прасмыцкий
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Компьютерная верстка Н. М. Федорцовой

Подписано в печать 20.04.16. Формат 60×84/16. Бумага писчая «Снегурочка».
Ризография. Гарнитура «Times».
Усл. печ. л. 3,95. Уч.-изд. л. 4,1. Тираж 60 экз. Заказ 246.

Издатель и полиграфическое исполнение: учреждение образования
«Белорусский государственный медицинский университет».
Свидетельство о государственной регистрации издателя, изготовителя,
распространителя печатных изданий № 1/187 от 18.02.2014.
Ул. Ленинградская, 6, 220006, Минск.

**ISBN 978-985-567-459-8**

# 1. BASIC CONCEPTS OF ANESTHESIOLOGY AND REANIMATOLOGY

**Anesthesiology and Reanimatology** is the discipline, the main aspect of which is studying and development of anesthetic methods and mechanisms, methods of the vital functions recovery.

An important common point in Anesthesiology and Reanimatology is a single principle of correction and maintenance of disturbed vital functions.

The common methods and practices used in Anesthesiology and Reanimatology are as follows: tracheal intubation, vessels canulation, mechanical ventilation (MV), infusion therapy, parenteral nutrition, etc.).

The main **objective of Anesthesiology** is to protect the patient from the surgical trauma and to create the best conditions for the work of a surgeon.

The main **goal of Reanimatology** is to treat patients in a terminal condition (resuscitation), and those with severe violations of the vital functions (intensive care).

# 2. ORGANISATION OF ANESTHESIOLOGY AND REANIMATOLOGY SERVICE

## 2.1. INTENSIVE CARE UNIT

Intensive or critical care medicine refers to the medical specialty which focuses on the management of critically ill patients.

A critical illness is a general state which may arise from various medical pathologies (e. g. trauma, infection, acute coronary syndrome, stroke etc.) and leads to the impairment of vital (consciousness, circulation or respiration) or single organ functions (e. g. kidney or liver function).

Furthermore, intensive care includes care of patients after major surgery or observation of patients with the potentially rapid development of a critical illness.

**Definition**

**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.

The common methods and practices used in the intensive care are as follows: tracheal intubation, vessels cannulation, mechanical ventilation (MV), infusion therapy, hemodialysis and extracorporeal liver support, parenteral nutrition, etc.

The definition also includes intensive monitoring (clinical, technical and laboratory) and general care of the patient.

In Belarus Intensive Care Units (ICUs) are small, specialized wards, which usually have 6, 12 or 18 beds (in other countries something close to these numbers), wider staff, and special medical equipment (patient monitors, suction machines, mechanical ventilators, infusion and syringe pumps). Within the hospital, at least 1–2 % of beds are to be assigned for the critically ill.

Levels of hygiene must be kept very high, so it is obviously to use alcohol hand rub before and after entering an ICU. Basic hygiene routines including hand washing before and after contact with patients and application of disposable gloves should be rigorously followed to reduce nosocomial infections.

Dispensers are usually found at the entrance to the ICU and by every bed space. The number of people around a bed will usually be limited for the safety of the patient.

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.

Many of these patients have a good prognosis if they receive adequate critical care for a limited period of time. Indeed, many ICUs have begun as postoperative units.

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.

### 2.2. CRITERIA FOR ICU ADMISSION AND DISCHARGE

Abnormal vital signs (heart rate, respiratory rate, systolic blood pressure, conscious level, body temperature, oxygen saturation) have been shown to predict mortality in a low-income setting.

Limits or "triggers" can be defined for each vital sign and any patient with one or more observations falling beyond these triggers is categorised as critically ill (table 1).

An ICU should have well defined admission criteria. These criteria depend on the facilities and expertise available but should be based on the hospital's triage systems. The goal is to admit the patients to the ICU who could most benefit from the critical care, i. e. those who have life threatening conditions and a reasonable chance of recovery.

*Table 1*

**Single parameter score**

| | Adult normal range | Critical illness triggers |
|---|---|---|
| Heart rate | 50–90 | < 40 $min^{-1}$<br>> 130 $min^{-1}$ |
| Respiratory rate | 10–20 | < 8 $min^{-1}$<br>> 30 $min^{-1}$ |
| Systolic BP | 100–140 | < 90 mm Hg |
| Conscious level | Awake, alert | Any sudden deterioration responds only to pain or unresponsive |
| Temperature | 36–37.5 °C | > 39 °C |
| Oxygen saturation | ≥ 95 % | < 90 % |

**Indications for ICU hospitalization include the following**:

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.

**N.B.** ICU beds are expensive and limited, that's why it is really important to understand that only **treatable** conditions are supposed to be admitted to an ICU.

Equally important are **discharge criteria**. Those patients who have sufficiently improved and no longer require critical care, or those who are judged to be too severely ill to benefit from the available care should be discharged from the ICU to free up beds for other critically ill patients.

## 3. MONITORING IN ICU AND DURING ANESTHESIA

In the intensive care unit (ICU), monitoring a patient's physiological parameters is an important part of their overall care package. Monitoring alerts you to any deterioration in a patient's condition and also helps asses their response to treatment.

Patient monitoring includes the following:

a) ***clinical*** (patient's skin color, chest movement, sweating, lacrimation, reactions of the pupil);

b) ***technical***:

- *cardiovascular*:

– heart rate;

– electrocardiogram;
– noninvasive arterial pressure;
– pulse oximetry;
• *respiratory*:
– respiratory rate;
– end-tidal carbon dioxide concentration;
– inspired oxygen.

Current Canadian guidelines to the Practice of Anaesthesia and patient monitoring are:

1. Presence of an anaesthetist. "The only indispensable monitor is the presence at all times, of an appropriately trained and experienced physician".

2. A completed preanaesthetic checklist. (Current history and physical documented, appropriate laboratory investigations reviewed, pre-anaesthesia evaluation completed, ASA classification recorded, and policy observed if it is an elective procedure).

3. An anaesthetic record. Every patient receiving general anaesthesia, major regional anaesthesia, or monitored intravenous conscious sedation, should have their HR and BP measured at least every 5 minutes. The time, dose, and route of all drugs and fluids should be charted.

4. Oxygenation, ventilation, circulation, and temperature are continually evaluated both clinically and quantitatively. (continually is defined as "repeated regularly").

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

The inspired oxygen concentration ($FiO_2$ 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.

**III. Circulation.** Circulation is monitored clinically by using one or more of the following methods: checking of the pulse, auscultation of heart sounds, arterial pressure monitoring, doppler pulse monitoring, or oximetry.

Quantitative evaluation of circulation includes an audible electrocardiogram (ECG) signal, and arterial blood pressure measurements at least every 5 minutes.

There are three most commonly used electronic monitoring systems — electrocardiogram (ECG), pulse oximetry ($SaO_2$) and capnography.

While these monitoring systems are important and useful, it should be remembered that they are always an addition to, rather than a replacement for,

good clinical monitoring of heart rate, blood pressure, capillary refill time, respiratory rate, neurological status and urine output.

The benefits include:

• **Additional clinical information**. ECG, $SaO_2$ and $CO_2$ monitoring give very useful information about the patient's cardiorespiratory function. This information is continuous and in "real time" and so is especially useful in critically ill patients.

• **Non-invasive**. These monitors are non-invasive, and so are well tolerated.

• **Early warning system**. The monitor's alarm systems can be adjusted to detect deviation of parameters from acceptable levels, thus providing a prompt warning of any change in the patient's condition.

Careful attention to the trends of these deviations will alert you to early signs of clinical deterioration.

### 3.1. PULSE OXIMETRY

**Pulse oximetry ($SpO_2$)** — method of oxygenation assessment, used in all clinical settings when hypoxemia may occur: in operating rooms, intensive care units, postanesthesia care units, emergency departments and ambulances, endoscopy suites, sleep laboratories, and cardiac catheterization laboratories, delivery suites.

The principle of pulse oximetry is the absorption of two different infrared light wavelengths (oxyhaemoglobin and desoxyhaemoglobin).

Partial pressure of oxygen dissolved in arterial blood is termed $PaO_2$. Percent saturation of oxygen bound to hemoglobin in arterial blood is termed $SaO_2$. The most important information available from this monitor is the arterial blood oxygen saturation ($SaO_2$), which is given as a percentage.

When measured by a pulse oximeter, $SaO_2$ is termed $SpO_2$. In a normal person breathing air, a value of 96–100 % is normal. Note that in smokers and those with chronic lung disease the value is likely to be slightly lower, around 92–95 %.

The relationship between saturation and $PaO_2$ is described by the oxyhaemoglobin dissociation curve. Pulse oximetry saturation ($SpO_2$) ~ 90 % is a critical threshold. Below this level a small fall in $PaO_2$ produces a sharp fall in $SpO_2$. $SpO_2$ ~ 90 % corresponds to $PaO_2$ 60 mm Hg, $SpO_2$ ~ 80 % corresponds to $PaO_2$ 50 mm Hg.

In general, if a good signal is received, this indicates that perfusion to the patient's extremities is good. This also has specific role where the perfusion of a limb is at risk, for example following trauma or vascular surgery.

A weak or absent signal, should alert you to assess the patients perfusion and blood pressure. Be aware that the signal will transiently disappear if the blood pressure cuff inflates on that arm.

The $SpO_2$ only gives us only the part of the picture regarding oxygen delivery to the tissues, as this is also defined by the haemoglobin level and the cardiac output.

There are **no contraindications** for the use of pulse oximetry, provided that the data obtained are evaluated in the context of the patient's clinical circumstances. It is generally safe to use pulse oximetry for the monitoring of all patients.

**Complications** include ischemic pressure necrosis, mechanical injury.

### 3.2. CAPNOGRAPHY ($CO_2$) MONITORING

Carbon dioxide can then be detected in the patient's expired breath. The measurement of expired carbon dioxide is useful because it can provide insight into important life-sustaining processes, including metabolic, circulatory, and respiratory activity.

Capnography is the standard of care for monitoring the adequacy of ventilation in patients receiving general anesthesia. It is also used to monitor ventilation during procedures performed while the patient is under moderate or deep sedation in endoscopy suites, sleep laboratories, and cardiac catheterization laboratories.

Capnography is increasingly being used to monitor patients receiving mechanical ventilation and to monitor patients for hypercapnia or hypocapnia in intensive care units, postanesthesia care units, emergency departments and ambulances, and to aid in the assessment and treatment of patients in case of cardiac arrest.

Although pulse oximetry is useful in the assessment of oxygenation, capnography provides more direct information on the ventilatory status of a patient.

The monitor will give you a number in mmHg, kPa or percentage, with the percentage very close numerically to the kPa value, since atmospheric pressure is 101 kPa. As a guide, 4–6 kPa or 35–45 mm Hg are normal values in healthy non-smokers. Hypercapnia and hypercarbia refer to a greater-than-normal $PaCO_2$ in blood. Hypocapnia and hypocarbia refer to a lower-than-normal $PaCO_2$.

**Loss of the capnographic waveform** may occur in several circumstances. These include apnea, the disconnection of a ventilator circuit, the occlusion or dislodgment of an endotracheal tube, or the occlusion or disconnection of the sampling catheter.

**Decreases in $ETCO_2$** can result from hyperventilation, pulmonary embolism, cardiac arrest, sudden hypotension, hypovolemia, hypothermia, leaks in the sampling system, or a partial airway obstruction.

**Increases in $ETCO_2$** may be caused by hypoventilation, rising body temperature, bronchospasm, adrenergic discharge, release of a tourniquet on an arm or leg, or ventilation of a previously unventilated lung.

There are no contraindications for the use of capnography, provided that the data obtained are evaluated in the context of the patient's clinical circumstances. It is generally safe to use capnography for the monitoring of all patients.

## 3.3. HAEMODYNAMIC MONITORING

### 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.

It is recomended to use ECG and pulse oximetry together. In case of broad complex tachycardia the loss of the pulse oximetry waveform indicates pulseless ventricular tachycardia (VT), a medical emergency.

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.

**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:

$$MAP = DBP + 1/3(SBP – DBP).$$

***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.

**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.

**Advantages of IBP monitoring:**

• Continuous “beat-to-beat” blood pressure monitoring is useful in patients who are likely to display sudden changes in blood pressure (e. g. vascular surgery), in whom close control of blood pressure is required (e. g. head injured patients),or in patients receiving drugs to maintain the blood pressure (e. g. patients receiving inotropes such as adrenaline).

• The technique allows accurate blood pressure readings at low pressures, for example in shocked patients.

• The trauma of repeated cuff inflations is avoided in patients who are likely to need close blood pressure monitoring for a long period of time e. g. ICU patients.

• Intravascular volume status can be estimated from the shape of the arterial pressure trace, either by eye or by waveform analysis by a specific device e. g. a pulse contour analysis system.

• IBP measurement allows accurate assessment of blood pressure in certain patients not suitable for non-invasive blood pressure monitoring, e. g. patients with gross peripheral oedema in ICU or morbidly obese patients.

• The indwelling arterial cannula is convenient for repeated arterial blood sampling, for instance for arterial blood gases. This is not usually the sole reason for insertion of an indwelling arterial catheter.

All arterial lines should be clearly labelled and the tubing colour coded (usually with a red stripe) to avoid confusion. Drugs should never be administered via the arterial line.

**Central Venous Pressure**

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.

**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 ($SvO_2$).

**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).

**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**.

### 3.4. NEUROLOGICAL MONITORING

**Conscious level.** Reduced conscious level is a common finding in critically ill patients. The simple AVPU scale of conscious level (A = Awake; V = responds to Voice; P = responds to Pain; U = Unresponsive) allows conscious level to be objectively measured and documented, and deterioration identified.

The **Glasgow Coma Scale** (table 2) is good for predicting prognosis, but may be overly complex for triage.

*Table 2*

**Glasgow Coma Scale**

| Score | Eyes open | Best motor response | Best verbal response |
|---|---|---|---|
| 6 | – | Obeys commands | – |
| 5 | – | Localises pain | Orientated |
| 4 | Spontaneously | Flexion withdrawal | Confused |
| 3 | To speech | Decerebrate flexion | Inappropriate words |
| 2 | To pain | Decerebrate extension | Incomprehensible sounds |
| 1 | Never | No response | Silent |

It is the only system used universally in ICUs, though limitations exist in mechanically ventilated, sedated patients. It can be used for prognostication and is also frequently used for therapeutic decision making, e. g. elective ventilation in patients presenting with a GCS < 8.

**EEG monitoring.** The EEG reflects changes in cortical electrical function. This, in turn, is dependent on cerebral perfusion and oxygenation. EEG monitoring can be useful to assess epilepti form activity as well as cerebral well-being in patients who are sedated and paralysed.

***Bispectral Index (BIS) Monitor.*** BIS is a statistical index derived from the EEG and expressed as a score between 0 and 100. Scores below 50 have been reliably associated with anaesthesia-induced unconsciousness.

Assessment in the critically ill patient may be complicated by various confounding factors such as septic encephalopathy, head trauma and hypoperfusion.

**Anesthetic depth monitoring.** Because of the **Minimum Alveolar Concentration** (MAC) limitations, monitors measuring some parameters correlate with anesthetic depth have been introduced into clinical practice. These monitors convert spontaneous electroencephalogram (EEG) waveforms into a single value that correlates with anesthetic depth for some general anesthetics.

BIS measurements can help clinicians formulate the precise type and optimal dosages of anesthetic or sedative medication for each patient. It is widely used in the operating room to help:

– Regulate anesthetic drug use.

– Decrease the incidence of post operative side-effects such as nausea and vomiting.

– Reduce length of stay in the recovery room.

– Prevent intraoperative awakness.

BIS monitoring can also be useful during outpatient surgery. In the ICU, it has been shown to reduce recall of unpleasant experiences and provide objective sedation assessment during:

– mechanical ventilation;

– neuromuscular blockade;

– barbiturate coma;

– bedside procedures.

Well managed sedation levels in the ICU can also aid in ventilator weaning.

**Monitoring of the Neuromuscular Function.** In order to quantify the depth of neuromuscular blockade, electomyography is commonly employed during anaesthesia. This involves the application of two electrodes over an easily accessed peripheral nerve.

The electrodes are attached to a nerve stimulator, which applies an electrical impulse to the nerve. By attaching a strain gauge to the muscle being stimulated, the muscle response to stimulation may be observed or measured. Ulnar nerve stimulation results in the contraction of the abductor pollicis muscle and a twitch in the thumb.

Common methods of stimulation of the nerve include a single twitch stimulus, four twitch stimuli (each separated by 112 seconds and referred to as a train-of-four stimulus (TOF)) or a continuous stimulus referred to as a tetanus stimulation.

When all twitches disappear 90 % of all receptors are occupied. For procedures requiring muscle relaxation attempts are made to maintain one twitch present with a TOF stimulus. Reversal of a neuromuscular block will be easily accomplished if all four twitches of the TOF are present, and will be difficult or impossible if one or no twitches are observed.

### 3.5. TEMPERATURE MONITORING

A temperature monitor must be readily available to continuously measure temperature. Temperature monitoring is mandatory if its changes are anticipated or suspected.

Remember that all monitors are only as good as the person using them — think about what you are measuring, set your alarms appropriately and always use them in conjunction with clinical examination.

## 4. GENERAL ANESTHESIA (GA)

**Anesthesia** the term derived from the Greek αν-, an-, "without"; and αἴ σθησις, aisthēsis, "sensation". It is a reversible suppression of all types of sensitivity including tactile, pain, etc.

There are the following types of Anesthesia:

– General Anesthesia;

– Local Anesthesia;

– Sedation.

**General anesthesia** is a reversible drug-induced (anesthetics) depression of the CNS to prevent all perception of sensation during a procedure or surgery (absence of consciousness, suppression of all types of sensitivity including tactile, pain, reflex activity, etc.).

**Stages of General Anaesthesia.** There are four stages in GA performed with the application of diethyl eather as defined by Guedel in 1937 (fig. 1).

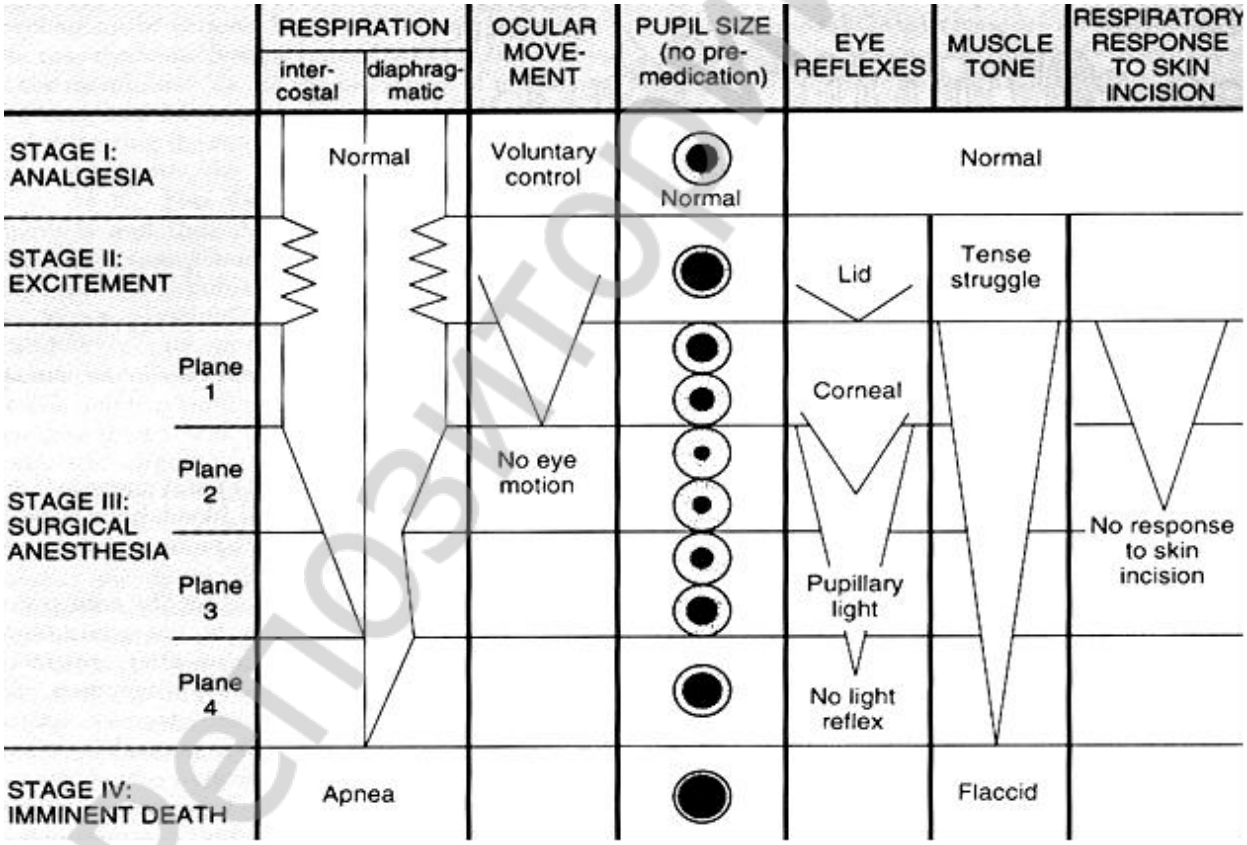


*Fig. 1.* Stages of general anesthesia, described by A. Guedel in 1937

***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.

**Components of modern general anesthesia** include the following:

– *Hypnosis*, or loss of consciousness;
– *Amnesia*, or loss of the procedure memory;
– *Analgesia*, or pain relief;
– *Muscle relaxation*;
– *Suppression of reflex activity*.

According to the American Society of Anesthesiologists, 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.

**Periods of general anesthesia** include the following:

– Induction of anesthesia;
– Maintenance of anesthesia;
– Recovery (after anesthesia).

**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.

## 4.1. INHALATION ANESTHESIA

**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.

**Minimum Alveolar Concentration (MAC).** Regardless of solubility and boiling point, each agent has its own potency value, called the MAC (the Minimum Alveolar Concentration). This is the concentration at equilibrium required to prevent a reflex response to a skin incision in 50 % of patients.

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.

**Inhaled Anesthetics.** The popularity of inhaled anesthetics to achieve general anesthesia is based on their ease of administration (via inhalation) and the ability to monitor their effects (clinical signs and end-tidal concentrations).

**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.

**Clinical Overview of Current Inhaled Anesthetics**

***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).

***Desflurane*.** Desflurane is a completely fluorinated methyl ethyl ether that differs from isoflurane only by replacement of a chlorine with a fluorine atom.

Compared with isoflurane, fluorination of desflurane results in low tissue and blood solubility (similar to nitrous oxide), greater stability (near-absent metabolism to trifluoroacetate), loss of potency, and a high vapor pressure (decreased intermolecular attraction). A heated and pressurized vaporizer requiring electrical power is necessary to deliver desflurane.

*Disadvantages of desflurane* include its pungency (smell; it cannot be administered by face mask to an awake patient), transient sympathetic nervous system stimulation when $F_I$ is abruptly increased, and degradation to carbon monoxide when exposed to dry carbon dioxide absorbents (more than isoflurane).

***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 (it resembles desflurane), 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.

***Xenon.*** This inert gas has many characteristics of an "ideal" inhaled anesthetic (blood gas partition coefficient of 0.14, provides some analgesia, nonpungent, does not produce myocardial depression).

The principal disadvantages of xenon are its expense (difficult to obtain) and high minimum alveolar concentration (MAC) (71 %).

***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 $B_{12}$, 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.

**Types of General Anesthesia Depending on the Mode of V entilation:**

– Spontaneous breathing (using Face mask, Laryngeal mask Airway (LMA);

– Controlled ventilation (using Endotracheal tube (ETT), LMA).

**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.

**Pressure Regulators.** Pressure regulators are used in anaesthetic machines for three purposes:

– to reduce the high pressure of gas in a cylinder to a safe working level;

– to prevent equipment damage on the anaesthetic machine, e. g. flow control valves;

– as the contents of the cylinder are used, the pressure within the cylinder decreases and the regulating mechanism maintains a constant outlet pressure.

**Flowmeters.** Once the pressure has been reduced to a safe level, each kind of gas must pass through flow-control valves and is measured by flowmeters before mixing with other gases, entering the active vaporizer, and exiting the machine's common gas outlet.

Gas lines proximal to flow valves are considered to be in the high-pressure circuit whereas those between the flow valves and the common gas outlet are considered part of the low-pressure circuit of the machine. A dual taper design can allow a single flowmeter to read both high and low flows.

Flowmeters are calibrated for specific gases, as the flow rate across a constriction depends on the gas's viscosity at low laminar flows and its density at high turbulent flows.

Some flowmeters have two glass tubes, one for low flows and another for high flows; the two tubes are in series and are still controlled by one valve.

**Vaporizers.** Anesthesia vaporizers create a saturated vapor concentration of the anesthetic and then dilute it to clinically useful concentrations. The ideal vaporizer construction material has high specific heat and thermal conductivity.

Contemporary vaporizers for halothane, isoflurane, enflurane, and sevoflurane are variable-bypass, concentration-calibrated, and temperature-compensated vaporizers. The volume of gas leaving a vaporizer is greater than that entering it because of the addition of anesthetic vapor.

All modern vaporizers are agent specific, capable of delivering a constant concentration of agent regardless of temperature changes or flow through the vaporizer.

Anesthesia circuits are classified as open, closed, semiopen, semiclosed, as rebreathing (no $CO_2$ absorption) or nonrebreathing (including a $CO_2$ absorber; e. g., circle system). In all circuits, the higher the fresh gas flow, the closer inspired gas composition approaches fresh gas.

**Heat and moisture exchanger.** A heat and moisture exchanger (HME) functions as an "artificial nose" is attached between the tracheal tube and the right-angle connector of the breathing circuit.

**Carbon dioxide absorber.** Rebreathing alveolar gas conserves heat and humidity. However, the $CO_2$ in exhaled gas must be eliminated to prevent hypercapnia. $CO_2$ chemically combines with water to form carbonic acid. $CO_2$ absorbents (e. g., soda lime or barium hydroxide lime) contain hydroxide salts, capable of neutralizing carbonic acid.

Reaction end products include heat (the heat of neutralization), water, and calcium carbonate. Soda lime is the more common absorbent.

All modern anesthesia machines are equipped with a ventilator. Ventilators generate gas flow by creating a pressure gradient between the proximal airway and the alveoli. Anesthesia ventilators are traditionally pneumatic and of "bag-in-a-bottle" or "double-circuit" design.

The breathing bellows is the interface between the patient circuit and the driving gas circuit. In traditional ventilators a standing bellows design is preferred as it makes a leak in the breathing system more obvious. The bellows descend on inspiration and ascend on expiration.

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.

**Waste-gas scavenging systems.** Waste-gas scavengers dispose gases that have been vented from the breathing circuit by the APL (adjustable pressure-limiting) valve and ventilator spill valve. Pollution of the operating room environment with anesthetic gases may pose a health hazard to surgical personnel.

Reduction to these trace levels is possible only with properly functioning waste-gas scavenging systems. The outlet of the scavenging system may be a direct line to the outside via a ventilation duct beyond any point of recirculation (passive scavenging) or a connection to the hospital's vacuum system (active scavenging).

**Inhalation anesthesia.** Has only 1 advantage: easy to manage.

Disadvantages include:

– slow induction;
– problems particularly during stage 2 (Excitement);
– airway obstruction, bronchospasm;
– laryngeal spasm, hiccups;
– environmental pollution.

### 4.2. NONINHALATION ANESTHESIA

**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.

**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.

**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.

**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.)

**Flumazenil** acts as a competitive antagonist in the presence of benzodiazepine agonist compounds.

In general, 45 to 90 minutes of antagonism can be expected after IV administration of 1 to 3 mg of flumazenil. (Sustained effects require repeated doses or continuous infusion.)

**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.

**Etomidate. Application:** Induction in patients with cardiovascular problems.

**Advantages:**

– Rapid induction;
– Ultra-short effect (5 min);
– No cardiovascular depression;
– Minimal respiratory depression.

**Disadvantages:**

– Pain while making the injection;
– Involuntary muscular movement;
– Nausea and vomiting;
– Hiccups;
– Non analgetic.

**Clinical Use of Intravenous Anesthetics**

**Use of Intravenous Anesthetics as Induction Agents.** Propofol has become the IV drug of choice for outpatients undergoing ambulatory surgery.

Clinical use of midazolam (often combined with other injected drugs), etomidate (cardiac stability), and ketamine (hypovolemic patients) is restricted to specific situations in which their unique pharmacologic profiles offer advantages over other available IV anesthetics.

The availability of IV drugs with more rapid onset and shorter recovery profiles, as well as easier-to-use infusion delivery systems, has facilitated the maintenance of anesthesia with continuous infusion of IV drugs (total IV anesthetic techniques [TIVA]), producing an anesthetic state that compares favorably with the volatile anesthetics.

None of the currently available IV drugs can provide a satisfactory level of anesthesia without producing prolonged recovery and undesirable side effects; therefore, combinations of drugs must be administered to achieve hypnosis, amnesia, analgesia, and hemodynamic stability.

***Narcotic Agonists and Antagonists.*** Opium is derived from the dried juice of the poppy plant, which contains over twenty plant alkaloids, including morphine and codeine. An opiate refers to any preparation from or derivative of opium.

A narcotic refers to any substance that produces both analgesia and stupor, and includes both opium alkaloid derivatives and synthetic analgesic compounds.

5 commonly used intraoperative anaesthetic narcotics include: morphine, meperidine, fentanyl, sufentanil and alfentanil. Naloxone is a pure narcotic antagonist.

**Site of Action.** 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 in the table 3.

*Table 3*

**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 |

Features common to all narcotics include a dose related depression of respiration, sensorium, and pain perception. They are rapidly distributed through the body following intravenous injection.

**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.

Fentanyl, sufentanil, and alfentanil are the most common narcotic agents used during induction and maintenance of anaesthesia. This is due to their rapid onset, and predictable duration of action.

**Morphine** may be used in the perioperative period to provide long lasting analgesia. It should be administered slowly at a rate not exceeding 5 mg per minute to avoid excessive histamine release.

**Meperidine** is less commonly used for induction and maintenance of anaesthesia because of its negative ionotropic activity.

**Fentanyl** is much more potent than morphine, and because of its high lipid solubility, it has a rapid onset effect.

Fentanyl's short duration of action is due to its redistribution from the CNS to other tissue sites in the body.

**Sufentanil** is the most potent narcotic that is in clinical use today. It has a much smaller volume of distribution than fentanyl, and is ideally suited for intravenous infusion techniques during longer procedures.

Infusion rates of 0.1 to 0.5 mcg/kg/hr are appropriate for anaesthesia with an inhalational anaesthetic agent (balanced anaesthesia).

**Alfentanil** has a rapid onset and rapid recovery and is ideally suited for short procedures requiring intense analgesia.

An intravenous loading dose of **15–30** mcg/l/kg i. v. may be followed with an intravenous infusion rate of **0.25** to **1.5** mcg/kg/min to maintain analgesic plasma levels.

**Remifentanil** is the newest addition to our clinically available opioids. It is classified as an ultra-short acting opioid agonist, and has both a rapid onset and peak effect.

Adverse side effects such as hypotension, bradycardia, muscle rigidity and respiratory depression or arrest, may be more pronounced with remifentanil compared to other opioids because of it's rapid onset of action.

**Remifentanil** should only be administered by personnel specifically trained in the use of anesthestic drugs, and recognition and management of their adverse effects.

Immediate measures including the ability to establish and maintain a patent airway and institute controlled ventilationand cardio-respiratory resuscitation must be available when administering remifentanil.

Infusion doses of **0.1–2** ug/kg/min with supplemental bolus doses of **0.5–1** ug/kg are recommended during general anesthesia with **66 %** nitrous oxide in healthy adults.

***Narcotic Antagonists***

**Naloxone** (Narcan@) is a pure narcotic antagonist, which competes with opioids at the mu, delta, kappa and sigma receptors.

**Naloxone** is supplied in ampules of **0.02** mg/ml, **0.4** mg/ml, and **1** mg/ml. The **0.4** mg/ml and **1** mg/ml ampules should be diluted with saline to provide a concentration of **0.04** to **0.05** mg/ml to ease administration.

**Naloxone** reaches its peak effect within **1–2** minutes of intravenous administration, and has a duration of **30** to **60** minutes. Perioperative surgical patients, with evidence of excessive sedation or respiratory depression secondary to opioids, may be given small incremental doses of 40 mcg naloxone.

There are several advantages of Intravenous anesthesia including:

– easy administration;
– rapid induction;
– short acting;
– rapid recovery.

**Disadvantages:**

– overdosage is not easily corrected;
– no antagonists or antidotes;
– prolonged after effects (hangover).

**Neuromuscular blocking agents** (table 4) are divided into two classes: depolarizing and nondepolarizing. This division reflects distinct differences in the mechanism of action, response to peripheral nerve stimulation, and block reversal.

*Table 4*

**Neuromuscular blocking agents**

| **Muscle Relaxants** | |
|---|---|
| **depolarizing** | **nondepolarizing** |
| Short-acting<br>Succinylcholine | Short-acting<br>Mivacurium |
| | Intermediate-acting |
| | Atracurium<br>Cisatracurium |
| | Vecuronium<br>Rocuronium<br>Long-acting |
| | Doxacurium<br>Pancuronium<br>Pipecuronium |

Depolarizing muscle relaxants act as ACh receptor agonists, while nondepolarizing muscle relaxants function as competitive antagonists.

It is important to realize that muscle relaxation does **not ensure** unconsciousness, amnesia, or analgesia.

**Suxamethonium** (Succinylcholine) is predominantly used during emergency tracheal intubation, but the resultant rise in serum potassium must be expected which makes it inappropriate for use in cases of renal failure. Excessive potassium release also occurs after 48 hrs in extensive burns and spinal cord injury.

**Pancuronium** is long acting, but it may cause tachycardia and accumulates in renal failure.

**Vecuronium** is an analogue of the aminosteroid pancuronium, but causes minimal cardiovascular side effects. It is suitable for intubation and infusion.

**Atracurium** is a benzylisoquinolinium and is metabolised by ester hydrolysis and Hoffman (spontaneous) elimination. Its metabolites are inactive and it doesn't accumulate in renal or hepatic dysfunction. Histamine release occasionally occurs with boluses, but recovery occurs predictably within one hour, regardless of duration of infusion. The intubating dose is 0.5 $mg/kg^{-1}$, infusion — 4–12 $mcg/kg^{-1}/min^{-1}$.

***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.

## 4.3. SEDATION

Sedation is an essential component of the management of intensive care patients. It is required to relieve the discomfort and anxiety caused by procedures such as tracheal intubation, ventilation, suction and physiotherapy. It can also minimise agitation yet maximise rest and appropriate sleep.

Analgesia is an almost universal requirement for the intensive care patient. Adequate sedation and analgesia ameliorates the metabolic response to surgery and trauma.

Too much or too little sedation and analgesia can cause increased morbidity, for example over sedation can cause hypotension, prolonged recovery time, delayed weaning, gut ileus, DVT, nausea and immunosuppression; under sedation can cause hypertension, tachycardia, increased oxygen consumption, myocardial ischaemia, atelectasis, tracheal tube intolerance and infection.

Sedation in the ICU varies widely from producing complete unconsciousness and paralysis to being nursed awake yet comfortable. There are many components to the ideal regimen but key elements include recognition of pain, anxiolysis, amnesia, sleep and muscle relaxation.

**Levels of Sedation**

***Minimal sedation*** provides a drug induced state of Anxiolysis, during which a patient responds normally to verbal commands.

***Moderate sedation*** (Conscious Sedation or Analgesia), creates a drug induced depression of consciousness during which a patient responds purposefully to verbal commands. No interventions to maintain the patient airway are required.

***Deep sedation*** (Analgesia) is a drug induced depression of consciousness during which the patient cannot be easily aroused. He responds purposefully to repeated or painful stimulation. The ability to maintain ventilation function may be impaired.

Procedures, particularly invasive or painful, may require deep sedation.

***Conscious Sedation*.** Is used in diagnostic procedures, including minor surgery endoscopy, colonoscopy, transesophageal echocardiograms.

Effects of conscious sedation:

- Beneficial effects:

– Relaxation, comfort;
– Patient's ability to cooperate;
– Reversible amnesia;
– Rapid onset, quick recovery.

- Potential adverse effects:

– Respiratory depression or arrest;
– Hypotension.

Medications for Sedation (table 5).

*Table 5*

**Medications for Sedation**

| Medication | Dose | Comments |
|---|---|---|
| ***Sedatives*** | | |
| Chloral Hydrate | 50–100 mg/kg p. o. or p. r. | Up to 1 gram/single dose.<br>Max dose = 2 grams |
| Diazepam | 0.1 mg/kg IV slowly (over 3 min)<br>0.15–0.3 mg/kg p. o. | |
| Droperidol | 0.02–0.05 mg/kg IV slowly (over 3 min) | Onset: 3–10 min. Peak: 30 min.<br>Duration: 2–4 hrs |
| Midazolam | 0.05 mg/kg slowly (over 3 min)<br>0.1–0.3 mg/kg IM<br>0.5–0.7 mg/kg p. o. | |
| ***Narcotics (not in infants less than 3 mos.)*** | | |
| Meperidine | 1 mg/kg IM, SQ or IV (slowly) | |
| Morphine | 0.1 mg/kg IM, SQ or IV (slowly) | |
| Butorphanol | 0.01–0.02 mg/kg IV (slowly) | |
| Fentanyl | 1–3 mcg/kg (0.001–0.003 mg/kg) | Diminished sensitivity to $CO_2$ stimulation may persist longer than depression of resp. rate |
| ***Antagonists*** | | |
| Naloxone (for narcotics) | 0.01–0.10 mg/kg IV to desired effect | Brief duration of action (30 to 45 min) |
| Physostigmine (for anticholinergic syndrome) | 0.015–0.025 mg/kg to desired effect | Watch for cholinergic side effects (bradycardia, emesis, cramping, salivation) |
| Flumazenil (for benzodiazepines) | 0.1–0.2 mg/kg (partial antagonism)<br>0.4–1.0 mg (complete antagonism) | Benzodiazepine with drawl-induced seizures: residual sedation/hypoventilation |

## 5. LOCAL ANESTHESIA

**Local and regional anaesthesia** plays an important role in modern anaesthetic management. This form of anaesthesia may be used as an alternative to general anaesthesia, or may be used in combination with general anaesthesia in the hope of reducing the severity of the perioperative surgical stress response.

While numerous local anaesthetics (LA) are available for use by clinicians, this chapter will focus on three commonly used LA'S; Lidocaine, bupivicaine, and chlorprocaine. These three agents illustrate differences in the classification, potency and duration of local anaesthetics. Knowledge of these agents provides a basis for understanding other local anaesthetics which may be used in future practice.

**Local Anesthesia** is a reversible elimination of painful sensation from a specific part of the body without loss of consciousness

**The types of Local Anesthesia** are the following:

– Topical anesthesia;

– Infiltration anesthesia;

– Regional anesthesia.

**Topical anesthesia** — agents are applied to mucosal surface membranes (eye, nose, throat, etc.) to cause local loss of sensation (in case of cocaine and benzocaine use).

Topical anesthesia is applied in:

– ophthalmology;

– otorhinolaryngology;

– laryngoscopy;

– bronchoscopy;

– urethroscopy, etc.

**Infiltration anesthesia** is local anesthesia achieved by deposition of anesthetic solution into a superficial area.

**Regional Anesthesia** is the production of the area insensibility by interrupting the sensory nerve conductivity from that region of the body.

**Intravenous Regional Anesthesia.** *Bier's local anesthesia* is a regional anesthesia achieved by an intravenous injection, used for surgical procedures on the arm below the elbow or the leg below the knee; (performed in a bloodless field maintained by a pneumatic tourniquet that also prevents the anesthetic from entering the systemic circulation).

Types of Regional Anesthesia include the following: central neuroaxial blockade (spinal, epidural, combined spinal/epidural) and peripheral neural block (plexus, nerve).

*Peripheral neural block.* Peripheral neural blockade is induced by drug injection next to a nerve bundle. This deadens a large area served by the target nerve and reduces amount of drugs required in case of repeated direct infiltration of the same region.

*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.

*Epidural anesthesia* is a drug injection into epidural space outside the dura where the drug acts on nerve trunks after diffusing through the dura.

This added diffusion barrier slows down the onset of anesthesia. More drugs are required than with the spinal route potentially increasing the risk of systemic toxicity if drug accumulates in the circulation.

**Caudal anesthesia** is a regional anesthesia produced by injection of a local anesthetic into the *caudal or sacral canal*.

Caudal epidural anesthesia is one of the most commonly used regional techniques in pediatric patients. It may also be used in anorectal surgery in adults. The caudal space is the sacral portion of the epidural space.

Caudal anesthesia involves needle and/or catheter penetration of the sacrococcygeal ligament covering the sacral hiatus that is created by the unfused S4 and S5 laminae.

**Local Anaesthetics.** Over the next two clinical years you are likely to encounter and use local anaesthetics in many clinical settings. Minor procedures in

the emergency room, topical application for eye examination, and local infiltration for diagnostic and therapeutic procedures, are a few common uses of local anaesthetic agents.

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).

Esters are hydrolysed in the blood by plasma cholinesterase with the formation of paraaminobenzoic acid, a metabolite to which some patients are allergic.

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, etidocaine, and ropivacaine) have a half-life of a few hours.

Bupivicaine has the ability to produce a **differential nerve blockade** when used in dilute concentrations. This form of blockade is especially advantageous in obstetrical patients requiring pain management for labour and delivery. Such a block can provide excellent pain control (sensory nerve block), yet it still allows the patient to move and push during labour (minimal motor nerve blockade).

Hence, bupivicaine's ability to provide a differential nerve blockade together with its relatively long duration of action makes it a common choice for epidural pain management in the obstetrical patient.

Lidocaine provides a faster onset (about 10 minutes) but shorter duration (about 1–2 hours), as compared with bupivicaine, which has an onset time of up to 30 minutes and a duration of 2 or more hours.

High concentrations of LA in the blood may result in a spectrum of symptoms reflecting an initial excitation of the CNS followed by CNS, respiratory, and cardiovascular depression. In severe cases, this may be observed as a sudden loss of consciousness, respiratory arrest, or cardiovascular arrest.

When the clinician administers a large dose of a local anaesthetic, the patient should be closely monitored by maintaining verbal contact, as well as by continuous ECG monitoring, pulse oximetry, and blood pressure readings.

**Complications of Local Anesthesia:**

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

**Systemic Toxicity of LAs depends on accumulation in blood!**

**Dose** injected or applied, **rate of absorption** from injection or topical site and **metabolism** — all this determine the systemic drug concentration. High circulating levels of LAs can cause toxicity to the brain and cardiovascular system.

Management of CNS, respiratory, and cardiovascular toxicity begins with the **ABCs** (airway, breathing, and circulation) followed by the ACLS recommendations for resuscitation. The only deviation in the algorithm is the avoidance of the use of Class Ia and Ib antiarrhythmia such as procainamide, quinidine, and Iidocaine.

**Physiology of Spinal Anaesthesia.** There are three classes of nerves: motor, sensory and autonomic. The motor conduct impulses for muscles to contract; in case they are blocked, muscle paralysis results. Sensory nerves transmit sensations such as touch and pain to the spinal cord and from there to the brain.

Autonomic nerves control the calibre of blood vessels, heart rate, gut contraction and other functions not under conscious control.

Generally, autonomic and pain fibres are blocked first and motor fibres last. This has several important consequences. For example, vasodilation and a drop in blood pressure may occur when the autonomic fibres are blocked.

The patient should be well hydrated before the local anaesthetic is injected and should have an intravenous infusion in place so that further fluids or vasoconstrictors can be given if hypotension occurs.

***Anatomy.*** 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.

Remember the structures that the needle will pierce before reaching the CSF.

*The skin*. It is wise to inject a small bleb of local anaesthetic into the skin before inserting the spinal needle.

1. Subcutaneous fat. This, of course, is of variable thickness. Identifying the intervertebral spaces is far easier in thin patients.

2. The supraspinous ligament which joins the tips of the spinous processes together.

3. The interspinous ligament which is a thin flat band of ligament running between the spinous processes.

The ligamentum flavum is quite thick, up to about 1 cm in the middle and is mostly composed of elastic tissue. It runs vertically from lamina to lamina. When the needle is within the ligaments it will feel gripped and a distinct “give” can often be felt as it passes through and into the epidural space.

4. The epidural space contains fat and blood vessels. If blood comes out of the spinal needle instead of CSF when the stylet is removed, it is likely that an epidural vein has been punctured. The needle should simply be advanced a little further.

5. The dura. After feeling a “give” as the needle passes through the ligamentum flavum, a similar sensation may be felt when the needle is advanced a short distance further and pierces the dural sac.

6. Subarachnoid space. This contains the spinal cord and nerve roots surrounded by CSF. An injection of local anaesthetic will mix with the CSF and rapidly block the nerve roots with which it comes in contact.

***Local Anaesthetics for Spinal Anaesthesia.*** Local anaesthetic agents are either heavier (hyperbaric), lighter (hypobaric), or have the same specific gravity (isobaric) as the CSF.

Hyperbaric solutions tend to spread below the level of the injection, while isobaric solutions are not influenced in this way. It is easier to predict the spread of spinal anaesthesia when using a hyperbaric agent.

Isobaric preparations may be made hyperbaric by the addition of dextrose. Hypobaric agents are not generally available.

Bupivacaine (Marcaine). 0.5 % hyperbaric (heavy) bupivacaine is the best agent to use if it is available. 0.5 % plain bupivacaine is also popular. Bupivacaine lasts longer than most other spinal anaesthetics: usually 2–3 hours.

Lignocaine (Lidocaine/Xylocaine). Best results are obtained with 5 % hyperbaric (heavy) lignocaine which lasts 45–90 minutes. 2 % lignocaine can also be used but it has a much shorter duration of action. Cinchocaine (Nupercaine, Dibucaine, Percaine, Sovcaine) is a 0.5 % hyperbaric (heavy) solution similar to bupivacaine.

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.

***Positioning of the Patient for Lumbar Puncture***. Lumbar puncture is most easily performed when there is maximum flexion of the lumbar spine.

Spinal anesthesia is usually induced with the patient in the lateral decubitus or the sitting position. The lateral decubitus position has the advantage of being more comfortable for the patient.

***Preparation for Lumbar Puncture.*** Assemble the necessary equipment on a sterile surface. It will include: a spinal needle. The ideal would be 24–25 gauges with a pencil point tip to minimize the risk of the patient developing a post-spinal headache.

– An introducer, if using a fine gauge needle as they are thin and flexible and therefore difficult to direct accurately.

– A standard 19 gauge (white) disposable needle is suitable for use as an introducer.

– A 5 ml syringe for the spinal anaesthetic solution.

– A 2 ml syringe for local anaesthetic to be used for skin infiltration.

– A selection of needles for drawing up the local anaesthetic solutions and for infiltrating the skin.

– A sticking plaster to cover the puncture site.

– The local anaesthetic to be injected intrathecally should be in a single use ampoule.

**Performing the Spinal Injection.** The L4–5 interspace is the site most commonly used for spinal anesthesia. The spinal cord ends at the level L1–2 interspace in adults. Therefore, lumbar puncture performed at L2 and below is virtually free of risk of injuring the spinal cord.

The anesthesiologist should choose the inerspace below L2, which appears to afford the easiest access to the subarachnoid space.

***Sequence of actions*:**

– Draw up the local anaesthetic to be injected intrathecally into the 5 ml syringe, from the ampoule opened by your assistant.

Read the label. Draw up the exact amount you intend to use, ensuring that your needle does not touch the outside of the ampoule (which is unsterile).

– Draw up the local anaesthetic to be used for skin infiltration into the 2 ml syringe. Read the label.

– Clean the patient's back with the swabs and antiseptic ensuring that unsterile skin is not touched by your gloves. Swab radially outwards from the proposed injection site.

– Define a suitable interspinous space. You may have to press hard to feel the spinous processes in an obese patient.

– Raise an intradermal wheal of local anaesthetic with a disposable 25 gauge needle at the proposed puncture site.

Insert the introducer if using a 24–25 gauge needle. Ideally it should be advanced into the interspinous ligament but care should be exercised in thin patients that an inadvertent dural puncture does not occur.

– Insert the spinal needle (through the introducer, if applicable). Ensure that the stylet is in place so that the tip of the needle does not become blocked by a tiny particle of tissue or clot.

It is angled slightly cephalad (towards the head) and slowly advanced. An increased resistance will be felt as the needle enters the ligamentum flavum, followed by a loss of resistance as the epidural space is entered.

Another loss of resistance may be felt as the dura is pierced and CSF should flow from the needle when the stylet is removed.

If no CSF appears, replace the stylet and advance the needle a little further and try again.

When the injection is complete, withdraw the spinal needle, introducer and syringe as one and apply a sticking plaster to the puncture site.

It is essential to monitor the respiration, pulse and blood pressure closely. The blood pressure can fall following induction of spinal anaesthesia, particularly in the elderly and those who have not been adequately preloaded with fluid.

Warning signs of falling blood pressure include pallor, sweating or complaining of nausea or feeling generally unwell.

Nerve blockade of the sympathetic nervous system results in vasodilation which decreases venous return, stroke volume and cardiac output. If this produces hypotension, it is first treated with fluid boluses and, if a sympathomimetic drug such as ephedrine is required.

Sympathetic blockade of the cardioaccelerator fibres at levels T1 to T4 can result in vagal effects causing bradycardia, or even asystole.

Bradycardia is quite common during spinal anaesthesia particularly if the surgeon is manipulating the bowel or uterus. If the patient feels well, and

the blood pressure is maintained, then it is not necessary to give atropine. If, however, the heart rate drops below 50 beats per minute or there is hypotension, then atropine 300–600 mcg should be given intravenously.

It is generally considered good practice for all patients undergoing surgery under spinal anaesthesia to be given supplemental oxygen by face mask at a rate of 2–4 litres/minute, especially if sedation has also been given.

Increase the speed of the intravenous infusion to maximum until the blood pressure is restored to acceptable levels and, if the pulse is slow, give atropine intravenously.

Vasoconstrictors should be given immediately if the hypotension is severe, and to patients not responding to fluid therapy.

The chance of a dural puncture headache varies with the size and type of a needle used, direction of the bevel, and age of the patient. A 25- or 26-gauge needle should be employed in young patients.

**Epidural anesthesia.** The epidural space extends from the base of scull 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.

**Regional Anaesthesia.** *Indications*: for the most procedures, performed below the neck.

*Contra-indications to Regional Anaesthesia*. These include:

– **Inadequate resuscitative drugs** and equipment. No regional anaesthetic technique should be attempted if drugs and equipment for resuscitation are not immediately to hand.

– ***Regional Anaesthesia should be avoided in patients with disturbed vital functions!***

– **Clotting disorders.** If bleeding occurs into the epidural space because an epidural vein has been punctured by the spinal needle, a haematoma could be formed thus compressing the spinal cord. Patients with a low platelet count or receiving anticoagulant drugs such as heparin or warfarin are at risk.

– **Hypovolaemia** due to different causes e. g. bleeding, dehydration caused by vomiting, diarrhoea or bowel obstruction. Patients must be adequately rehydrated or resuscitated before performing spinal anaesthesia or they will become very hypotensive.

– **Sepsis** on the back near the site of lumbar puncture.

– **Septicaemia.** Due to the presence of infection in the blood there is a possiblity of meningitis development in such patients if a haematoma forms at the site of lumbar puncture and becomes infected.

– **Patient refusal**.

– **Uncooperative patients**. Although spinal anaesthesia is suitable for children, their cooperation is necessary and this must be carefully assessed at the pre-operative visit. Likewise, mentally handicapped patients and those with psychiatric problems need careful pre-operative assessment.

– **Anatomical deformities** of the patient's back. This is a relative contraindication, as it will probably only serve to make the dural puncture more difficult.

– **Neurological disease**. The advantages and disadvantages of spinal anaesthesia in the presence of neurological disease need careful assessment. Any worsening of the disease postoperatively may be blamed erroneously on the spinal anaesthetic.

Raised intracranial pressure, however, is an absolute contra-indication as a dural puncture may cause dislocation of the brain stem.

**The Advantages of Regional Anesthesia.**

*Cost.* The cost associated with regional anesthesia is minimal.

*Respiratory system*. Regional anesthesia produces few adverse effects on the respiratory system in case high blocks are avoided.

*Airway*. As control of the airway is not compromised, there is a reduced risk of airway obstruction or the aspiration of gastric contents.

*Muscle relaxation.* Spinal anaesthesia provides excellent muscle relaxation for lower abdominal and lower limb surgery.

*Bleeding*. Blood loss during operation is less than in case of the same operation done under general anesthesia. It is occurs due to decreased blood pressure and heart rate, and improved venous drainage which results in less oozing. The average inraoperative blood loss was 22–50 % lower during total hip

replacements performed under regional anesthesia, as compared to similar procedures performed under general anesthesia.

*Visceral tone.* Normal gut function rapidly returns after surgery.

*Coagulation*. Post-operative deep vein thromboses and pulmonary emboli are less common after spinal anesthesia.

**Disadvantages of Regional Anestesia.**

*Hypotension* may occur with higher blocks and the anaesthetist must know how to manage this situation.

*Some patients are not psychologically suited to be awake*, even if sedated, during an operation. They should be identified during the preoperative assessment.

Even if a long-acting local anaesthetic is used, spinal anesthesia is not suitable for *surgery lasting longer than approximately 2 hours.*

There is a theoretical *risk of introducing infection* into the subarachnoid space and causing meningitis.

*A postural headache* may occur postoperatively.

## 6. PREPARATION FOR ANESTHESIA. ANESTHETIC TECHNIQUE CHOICE

**Preoperative assessment.** Surgical procedures and administration of anesthesia are associated with a complex stress response proportional to the magnitude of injury, total operating time, amount of intraoperative blood loss and degree of postoperative pain.

The adverse metabolic and hemodynamic effects of this stress response can present many problems in the perioperative period. Decreasing the stress response to surgery and trauma is the key factor in improving outcome and lowering the length of hospital stay as well as the total cost of patients care.

The process of preoperative assessment provides the anaesthetist with the information about the patient and the proposed surgery. This allows the anaesthetist to plan various aspects of perioperative management. Some aspects must be conveyed to staff in the operating theatre suite in advance.

Examples include the planned use of invasive monitoring, issues related to patient positioning, and any special needs the patient might have, such as an interpreter. If senior anaesthetic assistance is needed, this should be arranged in advance, and organization of appropriate postoperative care should also be initiated.

The overall ***aims of preoperative assessment*** should include the following:

– Confirm that the surgery proposed is realistic when comparing the likely benefit to the patient with the possible risks involved.

– Anticipate potential problems and ensure that adequate facilities and appropriately trained staff are available to provide satisfactory perioperative care.

– Ensure that the patient is prepared correctly for the operation, improving where feasible any existing factors which may increase the risk of an adverse outcome.

– Provide appropriate information to the patient and obtain consent for the planned anaesthetic technique.

– Prescribe premedication and/or other specific prophylactic measures if required.

Traditionally, surgery was classified as being either elective or emergency.

– *Elective*: operation at a time to suit both patient and surgeon; for example hip replacement, varicose veins.

– *Scheduled*: an early operation but not immediately lifesaving; operation usually within 3 weeks, for example, surgery of malignancy.

– *Urgent*: operation as soon as possible after resuscitation and within 24 h, for example intestinal, major fractures.

– *Emergency*: immediate life-saving operation, resuscitation simultaneous with surgical treatment; operation usually within 1 h, for example major trauma with uncontrolled haemorrhage, extradural haematoma.

**Operative severity:**

– *minor* (LA procedures, uncomplicated hernia repair or varicse vein operation);

– *moderate* (appendectomy, cholecystectomy, mastectomy, TURP);

– *major* (laparotomy, bowel resection major amputations);

– *major* + (any aortic procedures, AP resection oesophagectomy).

**Presenting patient's condition and concurrent medical history.** Direct questions should be asked about the following items of specific relevance to anaesthesia. The indication for surgery determines its urgency and thus influences aspects of anaesthetic management.

There are many surgical conditions which have systemic effects and these must be sought and quantified, e. g. bowel cancer may be associated with malnourishment, anaemia, and electrolyte imbalance. The presence of coexisting medical disease must also be identified, together with the assessment of the extent of any associated limitations to normal activity.

The most relevant tend to be related to cardiovascular and respiratory diseases because of their potential effect on perioperative management.

**Anaesthetic history.** Details of the administration and outcome of previous anaesthetic exposure should be documented, especially if problems were encountered. Some symptoms such as sore throat, headache, or postoperative nausea may not seem of great significance to the anaesthetist but may form the basis of considerable preoperative anxiety for the patient.

Previous anaesthetic records should be examined if available, as more serious problems such as difficulty with tracheal intubation should have been documented.

**Family history.** There are several hereditary conditions which influence planned anaesthetic management, such as malignant hyperthermia, cholinesterase abnormalities, porphyria, some haemoglobinopathies and dystrophia myotonica.

**Drug history.** A complete history of concurrent medication must be documented carefully. Many drugs interact with agents or techniques used during

anaesthesia, but problems may occur if drugs are withdrawn suddenly during the perioperative period.

**History of allergy.** A history of allergy to specific substances must be sought, whether it is a drug, food or adhesives, and the exact nature of the symptoms and signs should be elicited in order to distinguish true allergy from some other predictable adverse reactions.

**Alcohol.** Patients may present with acute intoxication from alcohol or signs of chronic consumption. The latter are mainly non-specific features of secondary organ damage such as cardiomyopathy, pancreatitis and gastritis.

**Physical examination.** Full physical examination should be performed on every patient admitted for surgery and the findings documented in the medical notes. It might be argued that this is unnecessary in young healthy patients undergoing short or minor procedures.

**Urine analysis.** This should be performed for every patient. It is inexpensive and may occasionally reveal undiagnosed diabetes mellitus or the presence of urinary tract infections. Positive results should be confirmed by further evidence of pathology.

**Full blood count.** This provides information about the haemoglobin concentration, white blood cell count and platelet count, together with details of red cell morphology. Haemoglobin concentration tends to be of greatest interest to the anaesthetist.

**Blood chemistry.** The measurements available include the serum concentrations of urea, creatinine and electrolytes, blood glucose concentration and liver function tests. There are specific conditions in which knowledge of preoperative values is important (e. g. diuretic therapy, chronic alcohol abuse).

Beyond these situations, the value of preoperative screening is less clear, and detection of an unexpected abnormality seldom alters anaesthetic management.

Blood sugar measurement is required in patients receiving corticosteroid drugs and in those who have diabetes mellitus or vascular diseases; a fasting sample is usually required.

**Coagulation studies.** Coagulation tests (PTTK and INR) are required in patients who give a history of bleeding disorders, in patients receiving anticoagulant therapy and in those with liver disease.

Assessment of platelet function is worth considering in patients with potential inherited or acquired disturbances, especially if a regional anaesthetic technique is being proposed; however, this involves measurement of the bleeding time or thromboelastography.

**EGG.** A 12-lead electrocardiogram can demonstrate many acute or longstanding pathological conditions affecting the heart, particularly changes in rhythm or the occurrence of myocardial ischaemia or infarction.

It has some value as a preoperative baseline in patients with known or potential cardiovascular disease, although in the resting state the trace may appear normal despite the presence of clinically significant coronary artery disease.

More extensive investigations are available in many departments to supplement the 12-lead EGG and these are discussed elsewhere.

**Chest X-ray.** This investigation should be reserved for an older population (e. g. over 60 years of age) and patients with a clear indication. It probably has little value as a preoperative baseline because postoperative abnormalities are treated predominantly on the basis of their clinical relevance.

**Other X-rays.** Cervical spine X-rays should be considered in any patient in whom there is a possibility of vertebral instability, e. g. in the presence of rheumatoid arthritis. Thoracic inlet X-rays are required in patients with thyroid enlargement.

**Pulmonary function tests.** Peak expiratory flow rate, forced vital capacity (FVC) and forced expiratory volume in 1 s (FEVi) should be measured in all patients with dyspnoea at rest and in patients scheduled for elective thoracotomy arterial blood gas analysis is required. This information is a useful supplement to spirometry values.

In patients with progressive disease, these investigations serve as a useful reference for future admissions.

**Assessment of the operative risk**

Specific factors affect the operative risk:

– age;

– systemic organ dysfunctions CV diseases RESP diseases GIT disease hematological conditions

– obesity;

– DM;

– drugs;

– delayed wound healing;

– risk of thromboembolism;

– immune competence;

– operative severity and operating surgeon.

In 1940, the American Society of Anesthesiologists (ASA) established a Committee to develop a “tool” to collect and tabulate statistical data that would be used to predict the operative risk.

The Committee was unable to develop such a predictive tool, but instead focused on classifying the patient’s physical status, which led the ASA to adopt a five-category physical status classification system for preoperative patient assessment. A sixth category was later added to address the brain-dead organ donor (table 6).

After the patient’s history, examination and relevant investigations have been collected the anaesthetist must answer two questions:

– Is the patient in the best physical condition for anaesthesia and surgery?

– Are the anticipated benefits of surgery greater than the combined risks of undergoing anaesthesia and surgery, taking into account any concurrent disease?

Having taken a full clinical history, performed a physical examination and reviewed the relevant investigations, the anaesthetist should decide if further

measures are required to prepare the patient satisfactorily before proceeding to anaesthesia and surgery.

*Table 6*

**Preoperative Physical Status Classification of Patients According to the American Society of Anesthesiologists**

| Class | Definition |
|---|---|
| P1 | A normal healthy patient |
| P2 | A patient with mild systemic disease (no functional limitations) |
| P3 | A patient with severe systemic disease (some functional limitations) |
| P4 | A patient with severe systemic disease that is a constant threat to life (functionality incapacitated) |
| P5 | A moribund patient who is not expected to survive without the operation |
| P6 | A brain-dead patient whose organs are being removed for donor purposes |
| E | If the procedure is an emergency, the physical status is followed by "E" (for example, "2E") |

**Preparation for anesthesia.** ***Premedication*** is a medicament preparation, immediately prior to anesthesia.

It may be *non-specific* (general) — benzodiazepine with or without small dose of opioid, anticholinergic) and *specific* (oxygen, antibiotics, steroids, antihistamines, $H_2$ blockers, beta blockers, calcium channel blockers, nitroglycerine, bronchodilators, antacids, desmopressin, insulin, etc.)

Premedication refers to the administration of drugs 1–2 h prior the induction of anaesthesia. It is not a routine part of preoperative preparation, but the need for premedication must be considered after all relevant factors have been identified.

The objectives of premedication are to:

– avoid anxiety and fear;
– reduce secretions;
– enhance the hypnotic effect of general anaesthetic agents;
– reduce postoperative nausea and vomiting;
– produce amnesia;
– reduce the volume and increase the pH of gastric contents;
– weaken vagal reflexes;
– weaken sympathoadrenal responses.

**Drugs used for premedication.** Some of the objectives listed above may be achieved by administration of drugs at induction or during maintenance of anaesthesia.

*Benzodiazepines*. The benzodiazepines possess several properties which are useful for premedication, including anxiolysis, sedation and amnesia. The extent of such effects depends on individual drugs.

*Diazepam* was the first drug of this group to be commonly used.

*Lorazepam* (1–5 mg) produces a greater degree of amnesia than the other drugs in this group.

Benzodiazepines produce anxiolysis in doses that do not produce excessive sedation, and this is advantageous if respiratory function is compromised;

however, great caution should be exercised in these patients because depression of ventilation may be caused even by small doses.

**Opioid analgesics.** It is necessary to prescribe opioid analgesic drugs for premedication only when patients are in pain preoperatively. The opioids cause sedation, but are not good anxiolytic agents.

For premedication It is desirable to administer opioids intravenously at or after induction of anaesthesia than intramuscularly.

There are several important side-effects of the opioids:

– Depression of ventilation and delayed resumption of spontaneous ventilation at the end of anaesthesia in which a muscle relaxant has been used.

– Nausea and vomiting, produced by stimulation of the chemoreceptor trigger zone in the medulla, are extremely common. Opioids should always be used in combination with an antiemetic agent.

– Morphine causes spasm of the sphincter of Oddi and this may result in right upper quadrant pain in patients presented for surgery on the biliary tract.

**Butyrophenones.** Of the two butyrophenones, haloperidol and droperidol, only the latter enjoys popularity in anaesthetic practice. This drug possesses neuroleptic effects (which may be manifested as withdrawal and seclusion), a-blocking effects and antiemetic effects.

Occasionally, droperidol may produce dose-dependent dysphoric reactions and extrapyramidal side-effects. Butyrophenones are long-acting drugs and may delay recovery from anaesthesia, particularly in elderly patients.

**Phenothiazines.** These have been regarded as useful agents for premedication because they produce the following effects:

– central antiemetic action;
– sedation;
– anxiolysis;
– $H_2$-receptor antagonism;
– a-adrenergic antagonism;
– anticholinergic properties;
– potentiation of opioid analgesia.

**Disadvantages** include extrapyramidal side-effects, synergism with opioids which may delay postoperative recovery, and potentiation of the hypotensive effects of anaesthetic agents.

**Anticholinergic agents.** The three anticholinergic agents used commonly in anaesthesia are atropine, hyoscine and glycopyrronium. Anticholinergic drugs are used clinically to produce the following effects:

– *Antisialagogue effects.* These drugs block secretions when irritant anaesthetic gases are used and reduce excessive secretions and bradycardia associated with succinylcholine when it is given either as bolus or as an infusion.

– *Sedative and amnesic effects.* In combination with morphine, hyoscine produces powerful sedative and amnesic effects.

– *Prevention of reflex bradycardia.* Anticholinergics are given for both prophylaxis and treatment of bradycardia. Atropine is used commonly as

premedication in ophthalmic surgery to block the oculocardiac reflex in patients undergoing squint surgery and is used also for small children to reduce the bradycardia which may occur in association with halothane anaesthesia.

**Side-effects of anticholinergic drugs** include the following:

– CNS toxicity;

– *reduction in lower oesophageal sphincter tone*;

– *tachycardia*;

– *mydriasis and cycloplegia*;

– *pyrexia*.

**Pulmonary aspiration** of gastric contents is associated with significant morbidity and mortality. Factors predisposing to regurgitation and pulmonary aspiration include:

– inadequate anaesthesia;

– pregnancy;

– obesity;

– difficult airway;

– emergency surgery;

– full stomach;

– altered gastrointestinal motility.

In 1999, the ASA made the following recommendations on preoperative fasting in elective, healthy patients (table 7).

*Table 7*

| Ingested material | Minimum fasting period |
|---|---|
| Clear liquids | 2 h |
| Light meal | 6–8 h |
| Infant formula milk | 4–6 h |
| Non-human milk | 6 h |
| Breast milk | 4 h |

**Prophylaxis of venous thromboembolism.** Reasons for an increased risk of venous thromboembolism at the time of an operation include:

– Hypercoagulability caused by surgery or other factors (cancer, hormone therapy).

– Stasis of blood in the venous plexuses of the leg during anaesthesia.

– Further stasis due to reduced mobility after the operation.

– Damage to veins at the time of surgery.

– Interference with venous return (pregnancy, pelvic surgery, pneumoperitoneum during laparoscopic surgery).

– Dehydration.

– Conditions causing poor cardiac output.

**Risk factors for venous thromboembolism.** Patients can be divided into three categories of risk, low, medium, or high, dependent upon the type of operation, patient factors, and associated diseases.

***Duration and type of operation*:**

– Operations lasting less than 30 min are considered minor (low risk) and more than 30 min major (higher risk).

– Particularly high-risk procedures include major joint replacements (hip and knee) and surgery to the abdomen and pelvis.

***Patient factors*:**

– previous history of DVT or PE;

– thrombophilia;

– pregnancy, puerperium, oestrogen therapy (contraceptive pill, HRT);

– age over 40 (risk increases with age);

– obesity and immobility;

– varicose veins (in abdominal and pelvic surgery, but no evidence of increased risk for varicose vein surgery).

***Associated disease*:**

– malignancy (especially metastatic, or in abdomen/pelvis);

– trauma (especially spinal cord injury and lower limb fractures);

– heart failure, recent myocardial infarction;

– systemic infection;

– lower limb paralysis (e. g. after stroke);

– haematological diseases (polycythaemia, leukaemia, paraproteinaemia);

– other diseases, including nephrotic syndrome and inflammatory bowel disease.

For example, a fit patient over 40 having minor surgery is low risk, whilst a fit patient under 40 having major abdominal surgery is moderate risk; and an elderly patient having pelvic surgery for cancer is high risk for thromboembolism.

**Methods of prophylaxis.** The need for the prophylaxis depends on the operative risk (low, moderate, high risk). Protocols include chemical prophylaxis (s. c heparin (low & high molecular (LMWH)), oral anticoagulants, dextran 70) and mechanical methods (compression stocking, intermittent pneumatic compression).

Every hospital should have a policy detailing local practice. General measures which seem logical include:

– avoidance of prolonged immobility (encourage early mobilization);

– avoidance of dehydration.

Subcutaneous heparin reduces the incidence of DVT and fatal PE by about two-thirds. Traditionally, unfractionated (ordinary) heparin has been used, but there are advantages to the newer low-molecular-weight heparins.

The small risk of bleeding in case of epidural analgesia can be minimized by giving LMWH on the evening before surgery, so that 12 h or more have elapsed before the epidural is inserted (LMWH plasma half-life is 4 h).

**Suitable anesthetic technique choice.** There are several factors that determine whether a local, regional or general anaesthetic technique is the most appropriate. Examples of surgical, patient and anaesthesia factors to consider when planning a patient's anaesthetic are shown in table 8.

*Table 8*

**Surgical, patient and anaesthesia factors**

| Surgical factors | Patient factors | Anaesthesia factors |
|---|---|---|
| Type and site of surgery<br>Duration of surgery<br>Anticipated postoperative course (day-case or in-patient)<br>Requirement for muscle relaxation<br>"Shared airway" between anaesthetist and surgeon<br>Likelihood of major blood loss<br>Surgeon's preference | Age<br>Comorbidities<br>Obesity<br>Anatomical considerations<br>Patient's preference | Anticipated difficult intubation<br>Equipment available<br>Family history (e. g. malignant hyperpyrexia)<br>Fasting status, oesophageal reflux etc<br>Anaesthetist's preference or experience |

All patients should be told of common complications associated with the proposed anaesthetic technique (e. g. succinylcholine pains, postdural puncture headache). All patients should be told what they may experience in the perioperative period, including temporary numbness and weakness in the postoperative period if a local or regional technique is to be used.

If a technique of a sensitive nature (e. g. insertion of an analgesic suppository) is to be used during anaesthesia, the patient should be informed.

Patients should be informed of any increased risk related to their preoperative condition (e. g. damage to loose or crowned teeth, or cardiac complications in the presence of severe coronary artery disease).

All patients should be given the opportunity to ask questions; specific questions relating to anaesthesia must be answered honestly; if the questions relate to surgery, then the anaesthetist should ensure that a surgeon speaks to the patient before anaesthesia is induced.

A summary of the matters discussed, the risks explained and the techniques agreed should be documented on the anaesthetic record.

## 7. CARDIO-PULMONARY RESUSCITATION (CPR)

**Terminal conditions.** Terminal conditions are the worst outcome of a disease, characterized by vital signs fading according to the pathological processes development irrespective of the causes.

Terminal conditions include the following:

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

– **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.

**Causes of cardiac arrest**:

– Hypoxemia;

– Acid-base disturbances;

– Violation of potassium, calcium, and magnesuium content in the body;
– Hypovolemia;
– Adverse drug effects;
– Pericardial tamponade, tension pneumotorax.

The organism vital signs fading are characterized by the general laws of sample pathological processes development irrespective of the causes.

The main initiation factor in development of terminal states is the hypoxia and acidosis which results in vital organs function distress, invoking metabolic disturbances.

Actually the difference between clinical and biological death is the ability of brain cortex to fulfill its function after the resuscitation. It is less tolerable part of human organism towards terminal hypoxemia and acidosis.

We have only 5–7 minutes to regenerate vital activity of an organism with complex methods of resuscitation and artificial circulation.

Functions of brain cortex are less resistant to a hypoxia and time of cortex neuron vitality conservation in case of clinical death is limited to 3–5 minutes. The main goal of resuscitation is regeneration of the CNS function.

**Signs and symptoms of clinical death:**

– loss of consciousness (measurable brain activity stops within 20 to 40 seconds);
– respiratory arrest;
– lack of carotid pulse (the gold standard to diagnose cardiac arrest).

At the onset of clinical death, consciousness is lost within several seconds. Measurable brain activity stops within 20 to 40 seconds. Irregular gasping may occur during this early time period, and is sometimes mistaken by rescuers as a sign when CPR is not necessary.

During clinical death, all tissues and organs in the body steadily accumulate ischemic injury.

Stopped blood circulation has historically been difficult to reverse. The absence of blood circulation and vital functions related to blood circulation was considered to be the definition of death.

In the middle of the 20th century it often became possible to reverse cardiac arrest through cardiopulmonary resuscitation (CPR), defibrillation, epinephrine injection, and other treatments to restore normal heartbeat and circulation.

Instead of death, cardiac arrest came to be called “clinical death”, meaning the clinical appearance of death. Clinical death is now seen as a medical condition that precedes death rather than actually being dead.

**Limits of reversal.** Most tissues and organs of the body can survive clinical death for considerable periods. Blood circulation can be stopped in the entire body below the heart for at least 30 minutes, with injury to the spinal cord being a limiting factor.

Detached limbs may be successfully reattached after 6 hours of no blood circulation at warm temperatures. Bone, tendon, and skin can survive as long as 8 to 12 hours.

The brain, however, accumulates ischemic injury faster than any other organ. Without special treatment after circulation is restarted, full recovery of the brain after more than 5 minutes of clinical death at normal body temperature is rare.

Usually brain damage or later brain death results after longer intervals of clinical death even if the heart is restarted and blood circulation is successfully restored. Brain injury is therefore the limiting factor for recovery from clinical death.

Although loss of function is almost immediate, there is no specific duration of clinical death at which the non-functioning brain clearly dies. The most vulnerable cells in the brain, CA1 neurons of the hippocampus, are fatally injured by as little as 10 minutes without oxygen.

Brain failure after clinical death is now known to be due to a complex series of processes that occur after blood circulation is restored, especially processes that interfere with blood circulation during the recovery period.

Cardiac arrest is synonymous to clinical death. If clinical death occurs unexpectedly, it will be treated as a medical emergency. CPR must be initiated.

**Heart rhythms associated with cardiac arrest** are:

• ***asystolia*** (absence of myocardium electrical and mechanical activity);

• ***fibrillation and paroxismal ventricular tachicardia*** (cessation of circulation because of ineffective response to the pathological electrical activity);

• ***pulseless electrical activity*** (absence of mechanical response to normal or idioventricular electrical effect) including:

– pseudo-electromechanical dissociation (pseudo-EMD);

– idioventricular rhythms:

✓ ventricular escape rhythms;

✓ postdefibrillation idioventricular rhythms;

✓ bradyasystolic rhythms.

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).

**Chain of survival.** The concept of the "chain of survival" means that optimum results can be achieved only with the four elements of: early access, early CPR, early defibrillation and early advanced cardiac life support.

A rapid response is a crucial part of emergency life support, called the "chain of survival". It involves:

– *early recognition* of the emergency and call for an ambulance;

– *early CPR* — to save time before paramedics arrive;

– *early defibrillation* — to restart the heart in case of severe rhythm disturbances;

– *early post-resuscitation care* — early hospital treatment increases the patient's chance of recovery.

**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)** (fig. 2).

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

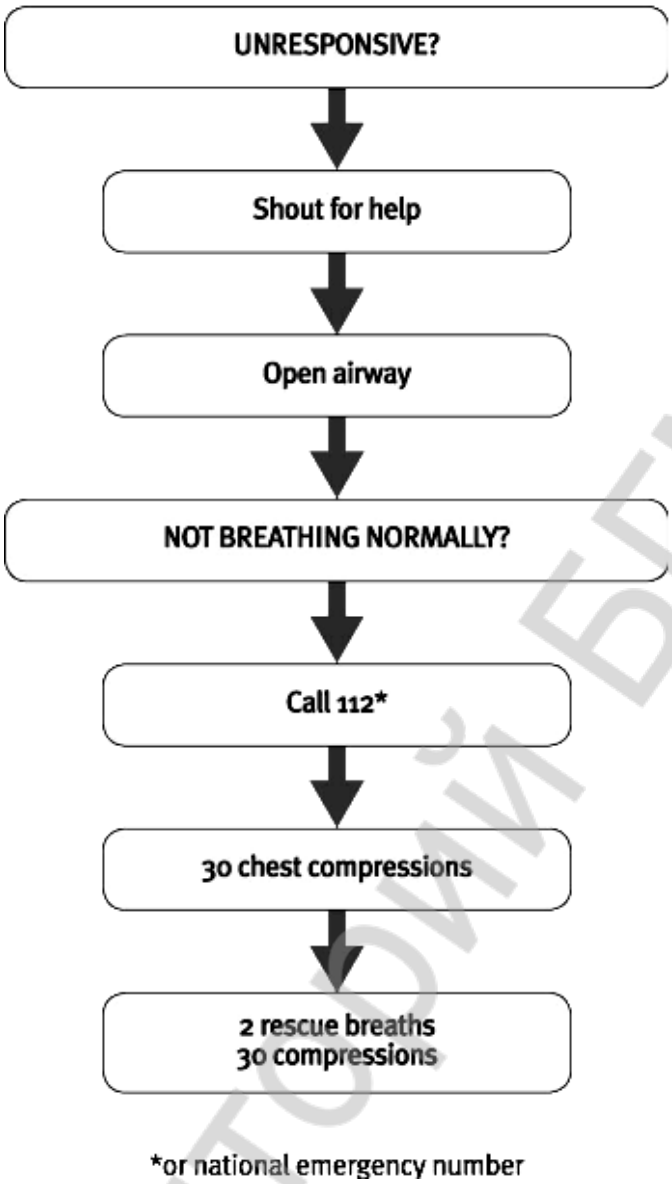


*Fig. 2.* Basic life support algorithm

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.

**Paediatric Basic Life Support. Sequence of actions.** Rescuers who have been taught adult BLS and have no specific knowledge of paediatric resuscitation (fig. 3) may use the adult sequence, as outcome is worse if they do nothing.

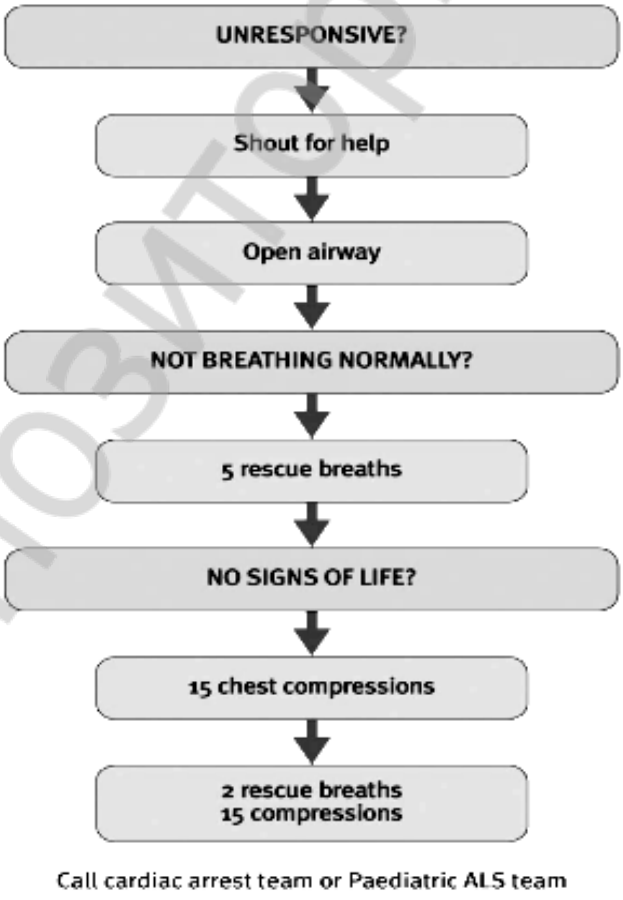


*Fig. 3*. Paediatric basic life support algorithm for those with a duty to respond

Non-specialists who wish to learn paediatric resuscitation because they have responsibility for children (e. g., teachers, school nurses, lifeguards), should be taught that it is preferable to modify adult BLS and perform five initial breaths followed by approximately one minute of CPR before they go for help (see adult BLS guideline).

**Foreign-body airway obstruction (FBAO)** is an uncommon but potentially treatable cause of accidental death. As most choking events are associated with eating, they are commonly witnessed. Thus, there is often the opportunity for early intervention while the victim is still responsive.

1. If the victim shows signs of mild airway obstruction (fig. 4): encourage continued coughing but do nothing else.

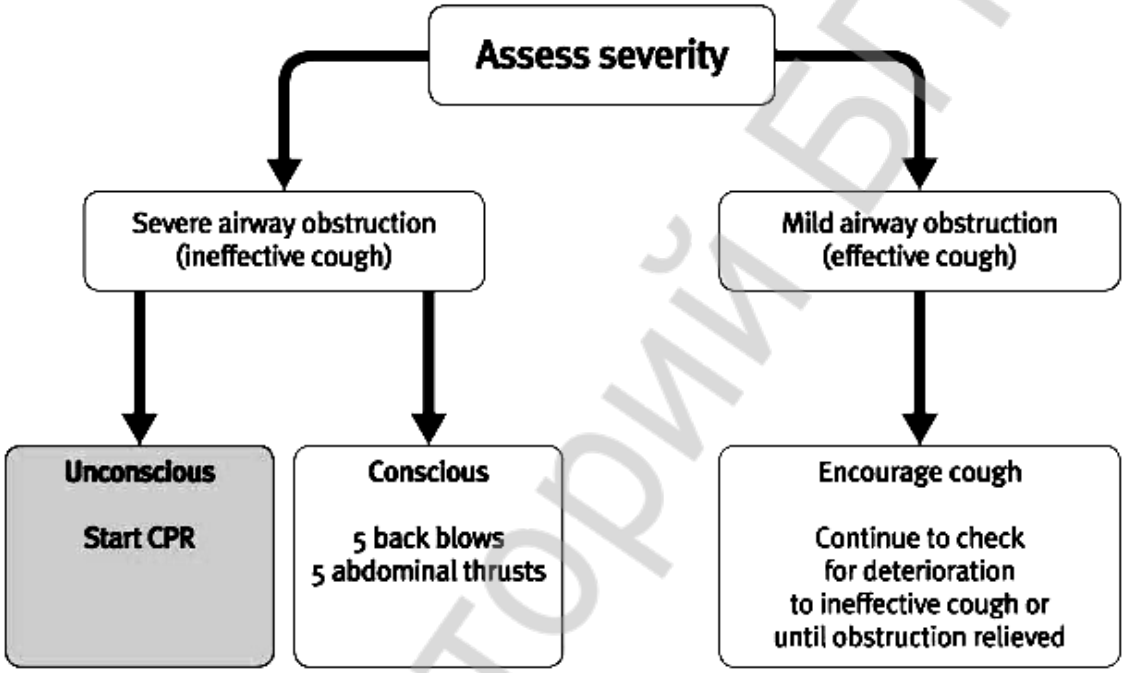


*Fig. 4*. Foreign-body airway obstruction (FBAO)

2. If the victim shows signs of severe airway obstruction and is conscious:

• Apply five back blows in the following way:

– stand to the side and slightly behind the victim;

– support the chest with one hand and lean the victim well forwards so that when the obstructing object is dislodged it comes out of the mouth rather than goes further down the airway;

– give five sharp blows between the shoulder blades with the heel of your other hand.

• If five back blows fail to relieve the airway obstruction, give five abdominal thrusts as follows:

– stand behind the victim and put both arms round the upper part of the abdomen;

– lean the victim forwards;

– clench your fist and place it between the umbilicus (navel) and the ribcage;

– grasp this hand with your other hand and pull sharply inwards and upwards;
– repeat five times.

• If the obstruction is still not relieved, continue alternating five back blows with five abdominal thrusts.

3. If the victim at any time becomes unconscious:
– support the victim carefully to the ground;
– immediately activate the ambulance service;
– begin CPR with chest compressions.

**Complications of BLS:**
– Laceration of the liver (*do not press down on the xiphoid process!)*
– Delayed rupture of the spleen.
– Regurgitation followed by aspiration of gastric contents.
– Costochondral separation and fractured ribs.
– Pneumothorax secondary to rib fracture.

**Advanced Cardiac Life Support (ACLS)** (fig. 5):
– 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.

**Drugs used during the treatment of cardiac arrest:**
• Adrenaline (epinephrine).
• Anti-arrhythmics:
– Amiodarone;
– Lidocaine;
– Magnesium.

• Other drugs:
– Atropine;
– Calcium;
– Buffers.

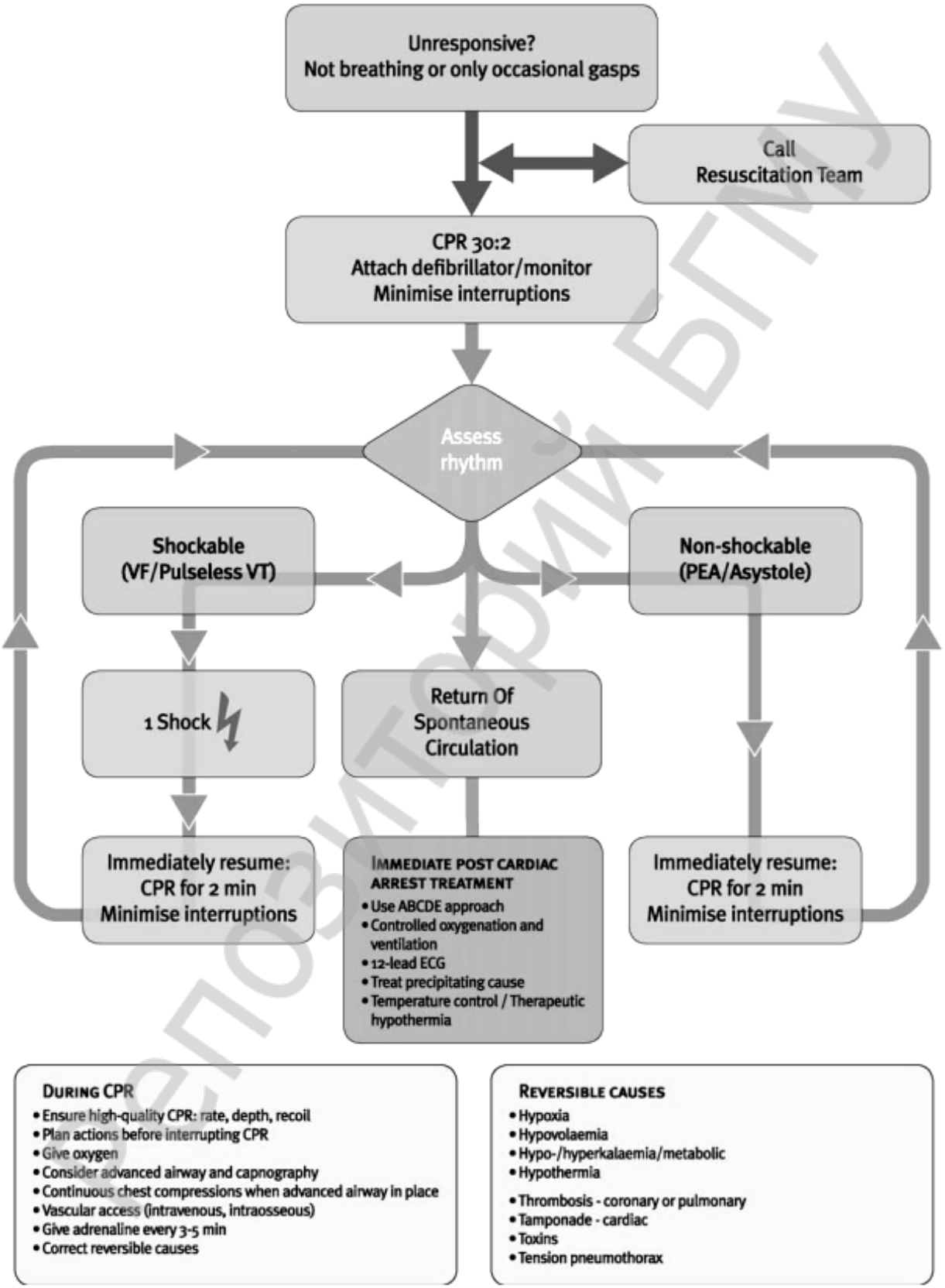


*Fig. 5*. ALS cardiac arrest algorithm

**Adrenaline** (**Epinephrine).** Adrenaline is the first drug used in cardiac arrest of any cause: it is included in the ALS algorithm to be used every 3–5 min of CPR (alternate cycles).

– [alpha]-adrenergic + [beta]-adrenergic effect;

– increase coronary and cerebral perfusion pressure during CPR;

– 1 mg dose of epinephrine IV/IO every 3 to 5 minutes;

– may be given by the endotracheal route at a dose of 2 to 2.5 mg.

On the basis of expert consensus, for VF/VT give adrenaline after the third shock once chest compressions have resumed, and then repeat every 3–5 min during cardiac arrest (alternate cycles). Do not interrupt CPR to give drugs.

**Amiodarone** is indicated in:

– refractory VF/VT;

– haemodynamically stable ventricular tachycardia (VT);

– resistant tachyarrhythmias.

Dose: consider an initial intravenous dose of 300 mg amiodarone, diluted in 5 % dextrose (or other suitable solvent) to a volume of 20 ml, if VF/VT persists *after the third shock*. Give a further dose of 150 mg if VF/VT persists.

**Lidocaine** is indicated in refractory VF/VT (when amiodarone is unavailable) but do not give lidocaine if amiodarone has been given already.

Dose:

• Consider an initial dose of 100 mg (1–1.5 mg/kg$^{-1}$) of lidocaine for VF/pulseless VT *refractory to three shocks*.

• Give an additional bolus of 50 mg if necessary.

The total dose should not exceed 3 mg/kg during the first hour.

**Magnesium sulphate** is indicated in ventricular or supraventricular tachycardia associated with:

– hypomagnesaemia;

– torsades de pointes;

– digoxin toxicity.

Dose: give an initial intravenous dose of 2 g (4 ml (8 mmol)) of 50 % magnesium sulphate) peripherally over 1–2 min; it may be repeated after 10–15 min.

**Atropine** is indicated in sinus, atrial, or nodal bradycardia when the haemodynamic condition of the patient is unstable.

*Atropines routine use for asystole or PEA is no longer recommended.*

**Calcium.** Give calcium during resuscitation only when indicated specifically, (that is) i. e. in pulseless electrical activity caused by:

– hyperkalaemia;

– hypocalcaemia;

– overdose of calcium channel-blocking drugs.

The initial dose of 10 ml 10 % calcium chloride (6.8 mmol $Ca^{2+}$) may be repeated if necessary.

**Sodium bicarbonate.** Routine administration of sodium bicarbonate during cardiac arrest and CPR or after ROSC is not recommended.

Consider sodium bicarbonate for:

– life-threatening hyperkalaemia;

– cardiac arrest associated with hyperkalaemia;

– tricyclic overdose.

Dose: give 50 mmol (50 ml of 8.4 % solution) of sodium bicarbonate intravenously.

**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.

**Non-shockable rhythms (PEA and asystole).** Pulseless electrical activity (PEA) is defined as cardiac arrest in the presence of electrical activity that would normally be associated with a palpable pulse. PEA is often caused by reversible conditions, and can be treated if those conditions are identified and corrected.

Survival following cardiac arrest with asystole or PEA is unlikely unless a reversible cause can be found and treated effectively.

If the initial monitored rhythm is PEA or asystole, start CPR 30:2 and give adrenaline 1 mg as soon as venous access is achieved. If asystole is displayed, check without stopping CPR, that the leads are attached correctly. Once an advanced airway has been sited, continue chest compressions without pausing during ventilation.

After 2 min of CPR, recheck the rhythm. If asystole is present, resume CPR immediately. If organized rhythm is present, attempt to palpate pulse. If no pulse is present (or if there is any doubt about presence of pulse), continue CPR.

Give adrenaline 1 mg (IV/IO) every alternate CPR cycle (i. e. about every 3–5 min) once vascular access is obtained. If a pulse is present, begin post-resuscitation care. If signs of life return during CPR, check the rhythm and attempt to palpate a pulse.

During the treatment of asystole or PEA, following a 2-min cycle of CPR, if the rhythm has changed to VF, follow the algorithm for shockable rhythms. Otherwise, continue CPR and give adrenaline every 3–5 min following the failure to detect a palpable pulse with the pulse check.

If VF is identified on the monitor midway through a 2-min cycle of CPR, complete the cycle of CPR before formal rhythm and shock delivery if appropriate — this strategy will minimize interruptions in chest compressions.

**Shockable rhythms (ventricular fibrillation/pulseless ventricular tachycardia).** The first monitored rhythm is VF/VT in approximately 25 % of cardiac arrests, both in- or out-of-hospital. VF/VT will also occur at some stage during resuscitation in about 25 % of cardiac arrests with an initial documented rhythm of asystole or PEA.

Having confirmed cardiac arrest, summon help (including the request for a defibrillator) and start CPR, beginning with chest compressions, with a CV ratio of 30:2. When the defibrillator arrives, continue chest compressions while applying the paddles or self-adhesive pads. Identify the rhythm and treat according to the ALS algorithm.

If VF/VT is confirmed, charge the defibrillator while another rescuer continues chest compressions. Once the defibrillator is charged, pause the chest compressions, quickly ensure that all rescuers are clear of the patient and then give one shock (360 J monophasic or 150–200 J biphasic).

Minimise the delay between stopping chest compressions and delivery of the shock (the preshock pause); even 5–10 s delay will reduce the chances of the shock being successful.

Without reassessing the rhythm or feeling for a pulse, resume CPR (CV ratio 30:2) immediately after the shock, starting with chest compressions.

Even if the defibrillation attempt is successful in restoring a perfusing rhythm, it takes time until the post-shock circulation is established and it is very rare for a pulse to be palpable immediately after defibrillation. Furthermore, the delay in trying to palpate a pulse will further compromise the myocardium if a perfusing rhythm has not been restored.

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

If IV/IO access has been obtained, give adrenaline 1 mg and amiodarone 300 mg once compressions have resumed. If ROSC has not been achieved with this 3rd shock the adrenaline will improve myocardial blood flow and may increase the chance of successful defibrillation with the next shock. In animal studies, peak plasma concentrations of adrenaline occur at about 90 s after a peripheral injection.

If ROSC has been achieved after the 3rd shock it is possible that the bolus dose of adrenaline will cause tachycardia and hypertension and precipitate recurrence of VF. However, naturally occurring adrenaline plasma concentrations are high immediately after ROSC, and any additional harm caused by exogenous adrenaline has not been studied.

**Electrical therapies: automated external defibrillators, defibrillation, cardioversion and pacing.**

**Automated external defibrillators** (AEDs) are safe and effective when used by either laypeople or healthcare professionals (in- or out-of-hospital). Use of an AED by a layperson makes it possible to defibrillate many minutes before professional help arrives.

AEDs are defibrillator machines designed to be operated by untrained members of the public. They are sometimes available in public places such as in offices, shopping centres, railway stations and on aeroplanes.

AEDs analyze a casualty's heart rhythm through pads connected to the chest area, and deliver an electric shock if VF is detected. They give spoken instructions and are easy for people without advanced training to use.

As soon as the AED arrives (fig. 6):

– switch on the AED and attach the electrode pads to the victim's bare chest (Place the first electrode pad in the midaxillary line just below the armpit. Place the second electrode just below the right collarbone (clavicle));

– if more than one rescuer is present, CPR should be continued while electrode pads are being attached to the chest;

– follow the spoken/visual directions immediately;

– ensure that nobody is touching the victim while the AED is analysing the rhythm.

If a shock is indicated:

– ensure that nobody is touching the victim;

– push shock button as directed (fully automatic AEDs will deliver the shock automatically);

– immediately restart CPR 30:2 ;

– continue as directed by the voice/visual prompts.

If no shock is indicated:

– immediately iniciate CPR, using a ratio of 30 compressions to 2 rescue breaths;

– continue as directed by the voice/visual prompts.

Continue to follow the AED prompts until:

– professional help arrives and takes over;

– the victim starts to wake up: moves, opens eyes and breathes normally;

– you become exhausted.

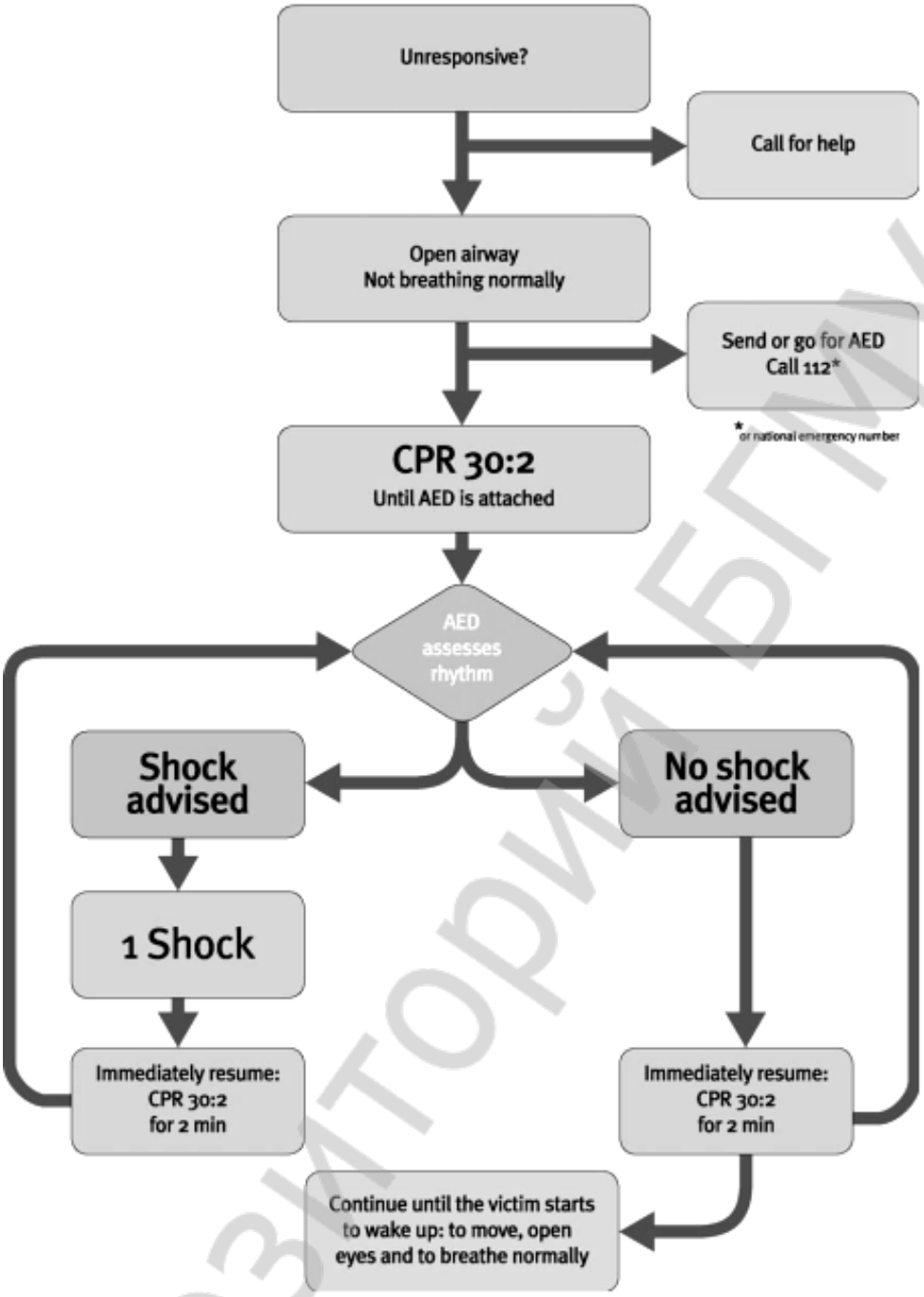


*Fig. 6.* AED algorithm

**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).

**Cardioversion.** If electrical cardioversion is used to convert atrial or ventricular tachyarrhythmias, the shock must be synchronized to occur with the R wave of the electrocardiogram rather than with the T wave: VF can be induced if a shock is delivered during the relative refractory portion of the cardiac cycle.

**Pacing.** Consider pacing in patients with symptomatic bradycardia refractory to anti-cholinergic drugs or other second line therapy Immediate pacing is indicated especially when the block is at or below the His-Purkinje level. If transthoracic pacing is ineffective, consider transvenous pacing.

**Potentially reversible causes.** Potential causes or aggravating factors for which specific treatment exists must be considered during any cardiac arrest. For ease of memory, these are divided into two groups of four based upon their initial letter: either H or T:

– Hypoxia;
– Hypovolaemia;
– Hypo/hyperkalaemia;
– Hypothermia;
– Thrombosis (coronary or pulmonary);
– Tamponade (cardiac);
– Toxins;
– Tension pneumothorax.

## 8. POST-CARDIAC ARREST SYNDROME

**Resumption of spontaneous circulation (ROSC)** after prolonged, complete, whole-body ischemia is an unnatural pathophysiological state created by successful cardiopulmonary resuscitation (CPR).

In the early 1970s, Dr Vladimir Negovsky discovered that the pathology caused by complete whole-body ischemia and reperfusion was unique as it had a clearly definable cause, time course, and constellation of pathological processes.

Negovsky named this state “postresuscitation disease”. Although appropriate at the time, the term “resuscitation” is now used more broadly to include treatment of various shock states in which circulation has not ceased. Moreover, the term “postresuscitation” implies that the act of resuscitation has ended.

**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 (fig. 7).

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.

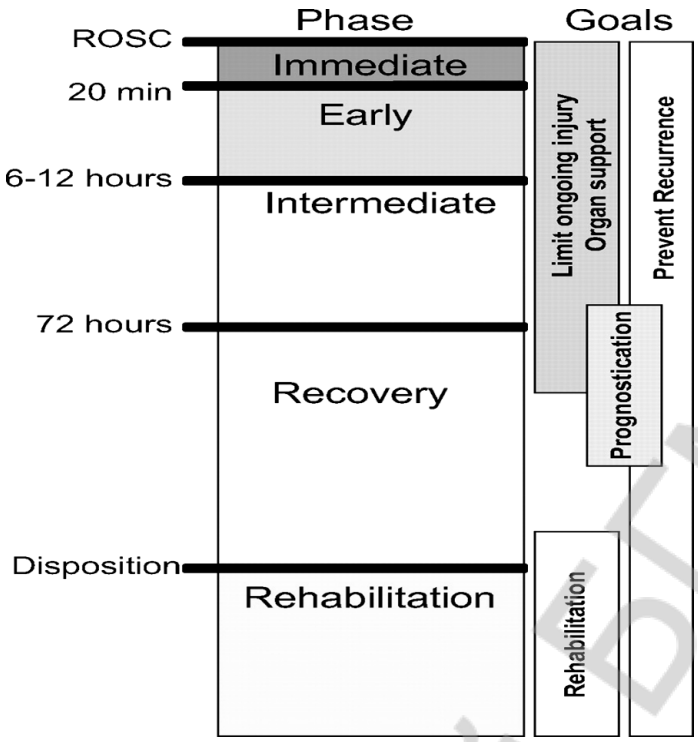


*Fig. 7.* Phases of post-cardiac arrest syndrome

**Post-Cardiac arrest myocardial dysfunction** also contributes to the low survival rate after in- and out-of-hospital cardiac arrest.

Immediately after ROSC, heart rate and blood pressure are extremely variable.

It is important to recognize that normal or elevated heart rate and blood pressure immediately after ROSC can be caused by a transient increase in local and circulating catecholamine concentrations.

Cardiac index values reached their nadir at 8 hours after resuscitation, improved substantially by 24 hours, and almost uniformly returned to normal by 72 hours in patients who survived out-of-hospital cardiac arrest.

The whole-body ischemia/reperfusion of cardiac arrest with associated oxygen debt causes generalized activation of immunologic and coagulation pathways, which increases the risk of multiple organ failure and infection. This condition has many features in common with sepsis.

Clinical manifestations of systemic ischemic-reperfusion response include intravascular volume depletion, impaired vasoregulation, impaired oxygen delivery and utilization, and increased susceptibility to infection. In most cases, these pathologies are both responsive to therapy and reversible.

**Persistent Precipitating Pathology.** The pathophysiology of post-cardiac arrest syndrome is commonly complicated by persisting acute pathology that caused or contributed to the cardiac arrest itself.

Diagnosis and management of persistent precipitating pathologies such as acute coronary syndrome (ACS), pulmonary diseases, hemorrhage, sepsis, and various toxidromes can complicate and be complicated by the simultaneous pathophysiology of the post-cardiac arrest syndrome.

Other precipitating causes of cardiac arrest may require specific treatment during the post-cardiac arrest period. For example, drug overdose and intoxication may be treated with specific antidotes, and environmental causes such as hypothermia may require active temperature control.

Specific treatment of these underlying disturbances must then be coordinated with specific support for post-cardiac arrest neurological and cardiovascular dysfunction.

**General Measures.** The general management of post-cardiac arrest patients (fig. 8) should follow the standards of care for most critically ill patients in the ICU setting. This statement focuses on the components of care that specifically impact the post-cardiac arrest syndrome.

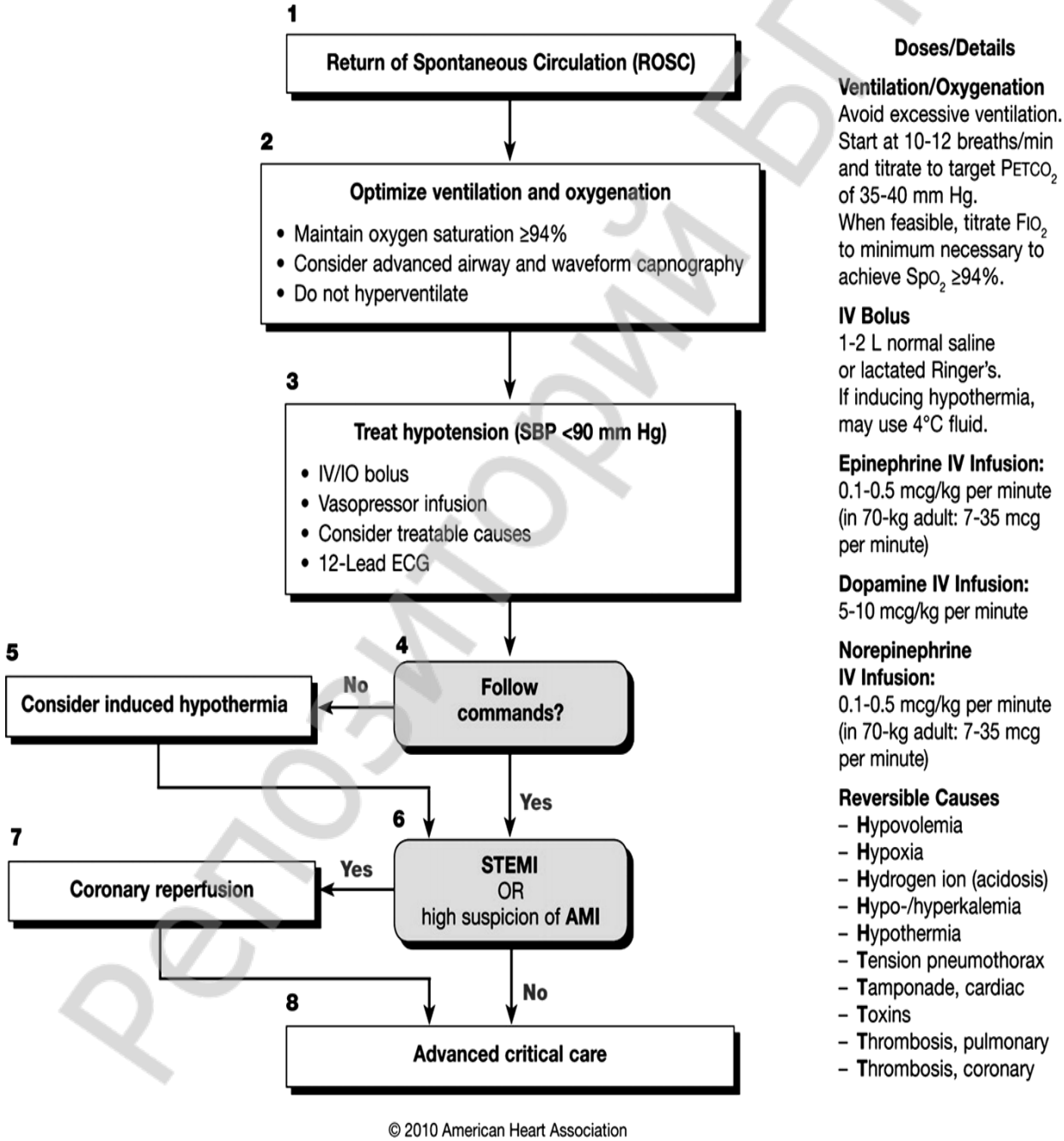


*Fig. 8*. General management of post-cardiac arrest patients

**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.

**Sedation and Neuromuscular Blockade.** If patients do not show adequate signs of awakening within the first 5 to 10 minutes after ROSC, tracheal intubation (if not already achieved), mechanical ventilation, and sedation will be required. Adequate sedation will reduce oxygen consumption, which is further reduced with therapeutic hypothermia.

Use of published sedation scales for monitoring these patients (e. g. the Richmond or Ramsay Scales) may be helpful. Both opioids (analgesia) and hypnotics (e. g. propofol or benzodiazepines) should be used. During therapeutic hypothermia, optimal sedation can prevent shivering and achieve target temperature earlier.

If shivering occurs despite deep sedation, neuromuscular-blocking drugs (as an intravenous bolus or infusion) should be used with close monitoring of sedation and neurological signs, such as seizures.

The duration of sedation and ventilation may be influenced by the use of therapeutic hypothermia.

In summary, critically ill post-cardiac arrest patients will require sedation for mechanical ventilation and therapeutic hypothermia. Use of sedation scales for monitoring may be helpful.

Adequate sedation is particularly important for prevention of shivering during induction of therapeutic hypothermia,maintenance, and rewarming.

Seizures, myoclonus, or both occur in 5 % to 15 % of adult patients who achieve ROSC and 10 % to 40 % of those who remain comatose. Seizures increase cerebral metabolism by up to 3-fold. No studies directly address the use of prophylactic anticonvulsant drugs after cardiac arrest in adults.

In summary, prolonged seizures may cause cerebral injury and should be treated promptly and effectively with benzodiazepines, phenytoin, sodium valproate, propofol, or a barbiturate.

**Glucose Control.** Tight control of blood glucose (4.4 to 6.1 mmol/L or 80 to 110 mg/dL) with insulin reduced hospital mortality rates in critically ill adults in a surgical ICU and appeared to protect the central and peripheral nervous system.

In summary, evidence to recommend any pharmacological neuroprotective strategies to reduce brain injury in post-cardiac arrest patients is inadequate.

Although relative adrenal insufficiency may exist after ROSC, no evidence is available that treatment with steroids improves long-term outcomes. Therefore, routine use of steroids after cardiac arrest is not recommended.

For patients with underlying coronary disease, an implantable cardioverter-defibrillator is strongly recommended if myocardial ischemia was not identified as the single trigger of sudden cardiac death or if it cannot be treated by coronary revascularization.

**Post-Cardiac Arrest Prognostication.** With the brain's heightened susceptibility to global ischemia, the majority of cardiac arrest patients who are

resuscitated successfully have impaired consciousness, and some remain in a vegetative state.

The need for protracted high-intensity care of neurologically devastated survivors presents an immense burden to healthcare systems, patients' families, and society in general.

**Brain Death.** The practice of diagnosing death varies between countries. Set definitions and criteria allow this concept to be applied for the purposes of withdrawal of critical care, when it is deemed to be futile.

It is particularly important when futility of treatment is discussed or when patients are considered for organ donation.

**Death of the brain** equates to the death of an individual and should involve an irreversible inability to breathe and an irreversible lack of capacity for consciousness.

The brain is particularly susceptible to injury. It has a high metabolic requirement, comprising 20 % of the body's oxygen consumption and receiving 15 % of the total cardiac output.

Swelling occurs in the injured brain, with the effects of swelling exacerbated by the brain's location in the fixed volume skull.

**Diagnosis of brainstem death.** Today, both the legal and medical communities use "brain death" as a legal definition of death. Using brain-death criteria, the medical community can declare a person legally dead even if life support equipment keeps the body's metabolic processes working.

**Preconditions for brainstem death testing:**

1. There must be an identifiable pathology causing irremediable brain damage. This may be intra- or extracranial.

2. The patient must be deeply unconscious:

a) Hypothermia must be excluded as the cause of unconsciousness and the patient's core temperature should be over 34 °C.

b) There should be no evidence that the patient's state is due to depressant drugs. This refers to narcotics, hypnotics and tranquillizers, as well as neuromuscular blocking drugs. A careful drug history is required, whilst drug levels and antagonists may need to be used.

c) Potentially reversible circulatory, metabolic and endocrine disturbances must have been excluded as the cause of the continuing unconsciousness. Some of these disturbances may occur as a result of the condition, rather than the cause, and these do not preclude the diagnosis of brainstem death.

3. The patient must be apnoeic, needing mechanical ventilation. This condition must not be secondary to the effect of sedative drugs or neuromuscular blockade.

This may require testing with a nerve stimulator to show intact neuromuscular transmission.

Two sets of tests should be performed to remove the risk of observer error. The two doctors may perform the tests together or separately and, although no

defined time interval has to elapse between the tests, it should be of sufficient duration to reassure the patient's next-of-kin.

The time of death is recorded when the first test indicates brain death.

**The tests:**

1. Pupils must be fixed in diameter and not responsive to incident light. (Cranial nerves II, III).

2. There must be no corneal reflex (avoid damaging the cornea). (Cranial nerves V, VII).

3. Vestibulo-ocular reflexes are absent. No eye movements occur following the slow injection of at least 50 ml ice cold water over one minute, into each external auditory meatus. Note that the normal reflex is deviation of the eyes away from the side of the stimulus.

Access to the tympanic membrane should be confirmed by otoscopy. Injury or pathology may prevent this test being performed on both sides — this does not invalidate the test. (Cranial nerves VIII, III).

4. No motor responses in the cranial nerve distribution should occur as a result of stimulation of any somatic area. No limb movement should occur in response to supra-orbital pressure. (Cranial nerves V, VII).

5. No gag reflex should occur in response to posterior pharyngeal wall stimulation with a spatula (Cranial nerve IX).

6. No cough or other reflex should occur in response to bronchial stimulation by a suction catheter being passed down the endotracheal tube (Cranial nerve X).

7. No respiratory movements should occur in response to disconnection from the ventilator ("apnoea test"). Hypoxia should be prevented by preoxygenation and insufflation of oxygen through a tracheal catheter.

This tests the stimulation of respiration by arterial carbon dioxide tension which should be allowed to rise to 6.65 kPa — confirmed by arterial blood gases.

**Organ donation.** A local transplant coordinator should be contacted early once the potential for organ donation is recognized. In case brainstem death has been established, the priority becomes preserving and optimizing the potential transplantable organs.

Respiratory support should be continued, maintaining normal blood gas parameters, but minimizing the harmful effects of positive pressure ventilation (e. g. avoidance of excessive positive end-expiratory pressure and excessive $FiO_2$).

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