Maternal anatomical and physiological changes

About pregnancy

Duration 280 days . 40 weeks . 9 months

1st Trimester : First 3 months

2nd Trimester : Second 3 months

3rd Trimester : Third 3 months

First signs of pregnancy:

• Nausea and vomiting (morning sickness) (till 12th weeks)
• Breast tenderness and enlargement
• Frequent urination
• Weakness and fatigue
• Changes in eating habit

**Complaints in later phase**

• Difficulty in sleeping

• Faint when lying on the back (vena cava syndrome)

• Frequent urination

• Fetal movement (sometimes painful)

• Constipation (sometimes requires laxatives)

• Tachypnea

**Anatomical and physiological changes during pregnancy:**

During pregnancy, anatomical and physiological changes occur due to:

a) increased metabolic needs to provide nourishment to the developing embryo

b) to prepare the body for childbirth
The changes begin to occur early in the first trimester, peaking at the term or labour

1- Weight gain: The average weight gain in pregnancy is 24 pounds; (fetus, extra-embryonic structures, uterus and the breasts, accumulated fluid, and fat deposits.)

2- Fat deposits in special places: – Abdomen – Breasts – Hip

3- Bones: – Volume of Calcium in the bones is decreasing (extra Ca into diet

4-Joints: – The sacroiliac and symphysis pubis are widened and rendered movable

Bones and joints changes) are believed to be caused by the action of hormone relaxin.

Extra weight and hormones (relaxin, E2) resulting in a special body shape, special curves in the spinal column! (Special type of walking!) – Abdominal lordosis ,(Frequent lumbar pain!) – Thoracic kyphosis

**Physiological maternal changes**

Once a woman conceives and becomes pregnant, several physiological changes begin to occur in her body.

The following major physiological maternal changes occur during pregnancy:
1- The **uterine size** increases from 50g in non-pregnant females to about one kg. The vagina enlarges and the introitus opens more widely.

2- The **breasts** also become almost double in size. Primary areola deepens in color – Secondary areola develops—lighter

3- The **basal metabolic rate** (BMR) also increases by 15% during the latter half of pregnancy

**Digestive and Urinary System Changes**

Nausea and vomiting, sometimes triggered by an increased sensitivity to odors, are common during the first few weeks to months of pregnancy. This phenomenon is often referred to as “morning sickness,” although the nausea may persist all day. The nausea is thought to be due to increased circulation of pregnancy-related hormones (estrogen and progesterone).

Decreased intestinal peristalsis may also contribute to nausea. By about week 12 of pregnancy, nausea typically disappear

A common gastrointestinal complaint during the later stages of pregnancy is gastric reflux, or heartburn, which results from the upward, pressure of the growing uterus on the stomach.

decreased peristalsis may thought to be responsible for pregnancy-related constipation as pregnancy progresses.
The downward pressure of the uterus also compresses the urinary bladder, leading to frequent urination. The problem is exacerbated by increased urine production. In addition, the maternal urinary system processes both maternal and fetal wastes, further increasing the total volume of urine.

**Circulatory System Changes**

There is an increase in stroke volume by about 90% that leads to an increase in left ventricular muscle mass.

There is a 10-20% increase in the maternal heart rate. Blood pressure decreases during the first and second trimester and returns to normal levels during the third trimester.

Blood volume increases substantially during pregnancy, so that by childbirth, it exceeds by 30 percent, or approximately 1–2 liters.

The greater blood volume helps to manage the demands of fetal nourishment and fetal waste removal. In conjunction with increased blood volume, the pulse also rise moderately during pregnancy.

The **cardiac output** of the mother increases by 40% because the heart must supply additional blood to the placenta.
As the fetus grows, the uterus compresses underlying pelvic blood vessels, reducing venous return from the legs and pelvic region. As a result, many pregnant women develop varicose veins or hemorrhoids.

**Hematological changes in pregnancy**

The maternal blood volume increases to 30% above normal during pregnancy.

The level of hemoglobin and **hematocrit** and red blood cell count decrease

Due to the above changes, pregnancy is associated with increase in iron requirement for hemoglobin synthesis and fetal development. Similarly, there is a need for increased folate and vitamin $\text{B}_{12}$.

Pregnancy is referred to as a **hypercoagulable**. This is thought to prevent blood loss during parturition.

**Respiratory changes in pregnancy**

During the second half of pregnancy, the respiratory minute volume (volume of gas inhaled or exhaled by the lungs per minute) increases by 50 percent to compensate for the oxygen demands of the fetus and the increased maternal metabolic rate.
The growing uterus exerts upward pressure on the diaphragm, decreasing the volume of each inspiration and potentially causing shortness of breath, or dyspnea. During the last several weeks of pregnancy, the pelvis becomes more elastic, and the fetus descends lower in a process called **lightening**. This typically reduce dyspnea.

**Renal changes in pregnancy:**

Increase in the renal blood flow. Thus, the glomerular filtration rate (GFR) increases by 50% to excrete out excessive water and salts retained in the body due to hormonal changes occurring during pregnancy. This increase in blood flow causes the kidneys to enlarge by 1-1.5 cm.

**Endocrine changes**

There is an increased level of all thyroid hormones; thus, pregnancy is considered a hyperthyroid state.

increase in aldosterone secretion

The pituitary gland enlarges during pregnancy

Pregnancy is a diabetogenic state associated with an increase in maternal glucose to supply the fetus and accompanying insulin resistance.
Skeletal and bone changes

Pregnancy is associated with reversible bone loss because of increased bone turnover to supply the fetus with calcium.

Skin Changes

The dermis stretches extensively to accommodate the growing uterus, breast tissue, and fat deposits on the thighs and hips. Torn connective tissue beneath the dermis can cause striae gravidarum (stretch marks) on the abdomen, which appear as red or purple marks during pregnancy.

An increase in melanocyte-stimulating hormone, in conjunction with estrogens, darkens the areolae and creates a line of pigment from the umbilicus to the pubis called the linea nigra. Melanin production during pregnancy may also darken or discolor skin on the face to create a chloasma, or “mask of pregnancy.”
The linea nigra, a dark medial line running from the umbilicus to the pubis, forms during pregnancy and persists for a few weeks following childbirth.
Striae gravidarum
Children are not small adults. Pediatric patients include the following groups:

1- Neonates – a baby of one month of age
2- Infants – a child of up to 12 months of age
3- Child – 1 to 12 years
4- Adolescent – 13 to 16 years
Children are different from adults in various aspects and the differences between Pediatric and adult anaesthetic practice are reduced as the patients become older.

Body weight
Airway
Drug behavior
Fluid requirements
Blood loss
Thermo-regulation
Respiratory system
Cardio-vascular system
Renal system etc.,

The differences between Pediatric and adult anaesthetic practice are reduced as the patients become older.

**Respiratory System:**
1- The airway is funnel shaped & narrowest at level of cricoid cartilage
2– Epithelium loosely bound to underlying tissue – Trauma to airway results in edema – 1 mm of edema narrow baby’s airway by 60%
3-Neonates – Obligatory nose breathers – Narrow nasal passage and easily blocked by secretions
A leak around endotracheal tube is preferred • Prevents post extubation edema and stridor
4- They have a large head, short neck and a prominent occiput. The tongue is relatively large.

5- The larynx is high and anterior. The epiglottis is long, stiff and U-shaped. It flops posteriorly. The ‘sniffing position will not help bag mask ventilation or to visualize the glottis. The head needs to be in a neutral position.

6- Narrowest at cricoid rather than vocal cords • Tube may be small enough to pass through cords but not cricoid • Larynx is funnel shaped, so secretions accumulate in retropharyngeal space

7- Neonates & infants have limited respiratory reserve
8- Ventilation mainly diaphragmatic • Bulky abdominal organs limits movement

neonates and infants generally need IPPV during anaesthesia and would benefit from a higher respiratory rate and the use of PEEP.
Cardiovascular System:

In neonates: –Myocardium less contractile –Limits the size of stroke volume –Cardiac output therefore rate dependent

Vagal sympathetic tone high, therefore they are more prone for bradycardia –Prophylactically atropine is given IV 0.01 mg/kg –Diluted atropine ready

Normal Blood Volumes

Age Blood Volume

New-born 85 –90 ml/kg

6 weeks to 2 yrs. 85 ml/kg
2 yrs. to puberty 80 ml/kg

**Renal System**

Renal blood flow and glomerular filtration are low in the first 2 years of life due to high renal vascular resistance. Tubular function is immature until 8 months, so infants are unable to excrete a large sodium load.

**Dehydration is poorly tolerated due to:**

1- Premature infants have increased insensible losses as that have a large surface area relative to weight.

2- There is a larger proportion of extra cellular fluid in children (40% body weight as compared to 20% in the adult).

Urine output 1-2 ml/kg/hr.

Conclusion: – Newborn kidneys has limited capacity to compensate for Volume EXCESS or Volume DEPLETION

**Hepatic System:**

- Immature liver function with decreased function of hepatic enzymes • Barbiturates & opioids have a longer duration of action due to slower metabolism

- Glucose Metabolism: Hypoglycaemia is common in stressed neonate. Glucose level should be monitored regularly • Glycogen stores are located in the liver & myocardium
**Hematology:**

At birth 70-90% of hemoglobin = HbF – < 3 months, levels ↓ 5% & HbA  95%

Hb in newborn = 18-20g/dL

The vitamin K dependent clotting factors (II, VII, IX, X) and platelet function are deficient in the first few months. Vitamin K is given at birth to prevent hemorrhagic disease of the newborn.

Transfusion recommended when 15% of the circulating volume has been lost.

**Temperature control :**

Newborns have a higher ratio of surface area to volume than adults. - newborns produce heat slowly and loose quickly – Large surface area to weight ratio – Minimal subcutaneous fat – Poorly developed shivering/sweating/vasoconstriction – Monitoring temperature is must –

Temperature control • Heat loss during anesthesia due to: –

Conduction/Convection/Evaporation •

Optimal ambient temp to prevent heat loss: –

Premature infant: 34°C

Neonates: 32°C

**Effect of hypothermia:** – Causes respiratory depression – Acidosis – Decreased cardiac output – Increases duration of action of drugs – Decrease platelet function – Increases risk of infection
Central Nervous System:
Blood brain barrier (BBB) poorly formed – Drugs (barbiturates, opioids, antibiotics, bilirubin cross BBB cause prolong & variable duration of action

Psychology

Infants less than 6 months of age are not usually upset by separation from their parents and will more readily accept a stranger.

Children up to 4 years of age are upset by the separation from their parents and the unfamiliar people and surroundings.

School age children are more upset by the surgical procedure.

What makes Pediatric Anesthesia different?

HYPOXIA is the most common cause of pediatric perioperative cardiac arrest. – Infants turn blue fast. upper airway obstruction during anesthesia (particularly at induction and emergence) Laryngospasm is common • Infants and young children are not small adults. "one size fits all" does not apply.

Pediatric Vital Sign Normal Ranges

Remember:

1-The patient's normal range should always be taken into consideration.
2-Heart rate, BP & respiratory rate are expected to increase during times of fever or stress.

3-Respiratory rate on infants should be counted for a full 60 seconds.

4-In a clinically decompensating child, the blood pressure will be the last to change. Just because your pediatric patient's BP is normal, don't assume that your patient is "stable".

5-Bradycardia in children is an ominous sign, usually a result of hypoxia. Act quickly, as this child is extremely critical.

**Pre-Operative Visit:**

Evaluate: – Medical conditions of the child – The needs of planned surgical procedure
drugs calculated according to weight

Investigations - Hb: Large expected blood loss, premature infant, systemic disorder, congenital heart disease

Electrolytes: Renal or metabolic disease, IV Fluid, dehydration •

Discuss regarding post op pain management: – Suppository medications

**Fasting:**

Solids 6 Hours

Formula milk 4 hours

Breast milk 3-4 hours

Clear fluids 2 hours
Premedication:

Sedations Analgesics Midazolam Paracetamol Chloral hydrate Codeine Ketamine Fentanyl

Endotracheal tubes: Tube Size: ID (mm) =

\[
\text{Age (years)} / 4 + 4.5 \quad (\text{Age} > 6 \text{ years})
\]

Length: For oral = \( \text{Age}/2 + 12 \text{ cm} \)

For nasal = \( \text{Age}/2 + 15 \text{ cm} \)

The ideal position for the tip of the tube is mid-trachea Auscultation on both sides to make sure both lungs are ventilated

Uncuffed tubes used till 8 years to prevent post extubation edema

Endotracheal tubes The rule of thumb is tube should be as large as the small finger of child

Laryngoscope: Miller Laryngoscope

Breathing Circuits • For children less than 20 kgs = Jackson Ree’s modification of Ayre’s T piece is used •Minimal dead space •Minimal resistance •Light weight •Fresh gas flow = 3 times the minute volume

Breathing circuit for older children (circle system)
Geriatric Anatomical and Physiological changes

Most of the world countries have accepted the chronological age of 65 and more as a definition of geriatric patients (Three Groups):

1. Elderly ------ Age 65 to 74
2. Aged -------- Age 75 to 84
3. Very Old ---- Age 85 and more

All cells experience changes with aging. They become larger and are less able to divide and multiply.

**common signs and symptoms of aging include:**

1. Increased susceptibility to infection.
2- Greater risk of heat stroke or hypothermia.

3- Slight decrease in height as the bones of our spines get thinner and lose some height.

3- Bones break more easily.

4- Joint changes, ranging from minor stiffness to severe arthritis.

5- Stooped posture

**Physiological changes:**

A number of physiological changes occur as we grow older. Some of the common changes are the result of diseases.

**Height**

The height falls after the age of 50. The primary factors contributing to this reduction in height include □ compression of vertebrae □ changes in posture, □ and increased curvature of the hips and knees.

By the time of age 80, most of loose an average height of about 2 inches

**Nose and ears** continue to grow in length.

**Male and female shapes** become more similar with aging.

**Male weight** tend to increase till late 50 and then decline.

**Female weight** tend to increase till 60 and then decline.
Total body water in male: young 60% to elderly 54%.

Total body water in female: young 52% to elderly 46%.

Cardiovascular system:

Heart disease is the leading cause of death in elderly. The size of heart increases in old age. But the amount of blood in the chamber decreases because the heart wall thickening. The maximum heart rate decreases.

Heart: – Cardiac output Decrease 1% per year after 30 years of age (at 80 year age CO is half that of a 20 year old person)

Blood Pressure – BP increase 1 mm of hg every year after 50 years as a normal consequence of aging. Systolic will increase and Diastolic remains unchanged or increase. (around 50% are Hypertensive in geriatric age group)

• Arteriosclerosis and Coronary Artery Disease and Loss of elasticity Loss of SA node cells causing slowed conduction

• Coronary artery Disease
• Congestive Heart failure
• Risk of arrhythmias
• Diminished peripheral pulse and cold extremities

• Increased blood pressure
• Postural Hypotension

Anaesthesia Implication

Hypotension and Bradycardia should be kept in mind during induction

For emergency anesthesia BP up to 180/110 mm of Hg should be allowed

• Heart Rate up to 50 at rest is allowed for induction

• Minor ECG changes are not threatening for anesthesia induction
Use of Beta blockers and Anti platelets in pre-operative period gives more cardio stability in old heart
Remember old heart cannot compensate decrease CO

**Respiratory system:**

Reduced gas exchange
Decrease respiratory muscle strength and elasticity
Stiffer chest wall, Anteroposterior diameter increase
Ventilation perfusion mismatch
Functional capacity declines
Decrease cough reflex and airway ciliary action • Frequent airway collapse • Reduced Compliance
Snoring and Sleep apnea common • Higher chances of aspiration
Increased risk of infection and bronchospasm with airway obstruction

**Anesthetic Implications:**

Advice to stop smoking at least 2 weeks before planned surgery and anesthesia
Proper Antibiotic & Anti-aspiration prophylaxis
Educate older people for deep breathing and coughing reflex preoperatively
Oxygen therapy in Pre-Intra- Post anesthesia period • Avoid or reduce doses of Opioids
Gastrointestinal system:

Reduced GI secretions. Reduced GI motility. Fall of teeth is an obvious sign of aging. Delayed gastric emptying.

Liver: Reduced blood flow Altered clearance of some drugs Decreased weight of liver Liver metabolizes less efficiently

Drug metabolism is slow in old age group

As we grow old, the small intestines absorb less calcium and vitamin D. Therefore, we need more calcium to prevent bone mineral loss and osteoporosis in later life.

Reduced peristalsis of the colon can increase risk for constipation.

There is decreased salivation, so there is difficulty in swallowing food.

Anesthetic Implications:
Correct Fluid, Electrolytes
Increased risk of gastric aspiration (so give protein pump inhibitor)
Avoid NSAID because it induce ulcers
Keep in mind about constipation & complain of constant abdominal disturbance Post-Op
Decrease metabolism of anesthesia drugs and risk of adverse drug reactions because of liver changes
**Urinary system:**

**Kidneys**: Gradual decrease in volume and weight of kidneys with aging -- Renal blood flow decrease, GFR decrease – Decrease in total glomeruli leading to age related decrease in creatinine clearance (no change in serum creatinine with advance age) – Age related increase in blood urea nitrogen

**Bladder**: Urinary incontinence found in almost 20% population more than 65 years -- Capacity of bladder decrease & late sensation leading to overflow incontinence

**Prostate**: Enlargement of prostate in 90% male more than 65 years age, but only 10% have symptomatic hyperplasia require surgery

**Anesthesia Implication**

Age related Renal changes interferes with the excretion of anesthesia drugs • Because of bladder and prostatic changes urinary catheterization is prime importance in major anesthesia and surgery • Renal insufficiency, dehydration and renal failure common in elderly, so prompt actions to be taken • Geriatric patients allowed clear fluid at least two hours before anesthesia

**Skin and Musculoskeletal System Changes**: One of the most common physical changes that people associate with aging is □ wrinkling □ Pigment alteration □ Thinning of the skin
The skin becomes less able to retain fluids and is more easily dry and cracked. As a result, both the thickness and elasticity of skin decrease.

Skin: Atrophy around face, neck, chest and extensor surface of limbs
Skin loses its elasticity resulting wrinkling
Decreased sensitivity to pain and pressure
Skeletal -- Degenerative Joint Diseases causing disability -- Pain response is severe -- 30%

Muscle mass reduced leading to decrease peripheral metabolism of drugs,

Osteoarthritis and Osteoporosis

**Anesthesia Implication**: 
Consider difficult IPPR and Intubation
Body temperature to be cared during anesthesia period. Avoid excessive cold temperature in OT and preferably cover geriatric patient fully. • Avoid pressure ulcers and padding of pressure points
Handle all geriatric patients carefully to avoid fractures and excessive manipulation during different surgical position
Pre-operative transfer of geriatric patient from ward to OT is always in presence of medical attendant (in wheel chair or in supine position)

**Nervous system Changes**

Neurologic Changes • Weight of brain decrease • Loss of brain cells • Blood flow to brain decrease • State of confusion • Interference with
Thinking, Reading Interpreting, Remembering • Sense of smell, Vision and hearing diminish

Problems in physiological regulation of Hypotension and temperature

**Anesthesia implication**

Difficulty in Communication, Cooperation & Coordination

Old patients take more time to recover from GA especially if they were disoriented preoperatively

Old Patient experience varying degrees of delirium • Sensitive to centrally acting anticholinergic agents • The % of delirium is less with regional anesthesia, provided there is no additional sedation • Dose requirements for local, general & inhalation anesthetics are reduced

**Temperature Regulation Changes**:

Elderly are prone to hypothermia because of :

1- Lower body metabolism
2- Vasodilatation of skin blood vessels
3- Decrease thermo genesis capability leading to – Shivering – Increase metabolic demand – Slow drug metabolism – Increase risk of myocardial ischemia

**Anesthesia Implication**:

Hypothermia should be avoided • Shivering will increase oxygen demands • To prevent heat loss - Use warm solutions - Use warm Blankets - Keep OT temperature warm
Immune System Changes

Increases risk of getting sick. An autoimmune disorder may develop. Healing is also slowed in older persons. Increase in the risk of cancer.
Effects Of Anesthesia On Respiratory Function

Normal Function Of Respiratory System:

1) Ventilation; The movement of air in and out of the lungs.
2) Gases exchange; The transfer of oxygen in to the blood and removal of carbon dioxide from blood.

General anesthesia has number of effect on both these function. The passage of gases into the lungs may be impaired by Obstruction of airway.
General anesthesia can have profound effects on pulmonary function. These effects are due to:

1- Direct effects of anesthetic drugs on the respiratory system

2- Altered body position

3- Intubation

4- Mechanical ventilation,

5- Use of neuromuscular blocking agents and other drugs.

6- Alterations in the cardiovascular system caused by general anesthesia can also affect pulmonary function by changing the amount and distribution of pulmonary blood flow.

It is difficult to generalize the effects of anesthesia on pulmonary function because of differences between anesthetics and the routes of administration employed, differences in the body positions and ventilatory modes used in various surgical procedures and differences in the age and cardiopulmonary status.

General anesthesia may have major effects on the mechanics of the lung and the chest wall, the volume and distribution of alveolar ventilation, the cardiac output and the distribution of pulmonary blood flow, the
matching of ventilation and perfusion, and the control of breathing. General anesthesia may also affect the airways, and the transport and diffusion of oxygen and carbon dioxide.

Effects Of Anesthesia On Respiratory Mechanics

General anesthesia has significant effects on the mechanics of the respiratory system. It alters the functional residual capacity (FRC), respiratory muscle function, the shape and motion of the lungs and chest wall, and it may affect the diameter of the airways.

Effects on the FRC

the FRC is determined by the balance between the inward recoil of the lungs and the outward recoil of the chest wall when the respiratory muscles are relaxed. When a normal conscious person changes from the upright to the supine position, the FRC decreases by about one-third. For a typical 70-kg person that would represent a decrease of about 1 l, from an FRC of 3 l ml/kg in the upright position to an FRC of 2 0 ml/kg in the supine position., this decrease in FRC that occurs when a person changes from a standing or upright position to the supine position is apparently a result of a decreased effect of gravity pulling downward on the
diaphragm in the supine position. This decreases the outward elastic recoil of the chest wall and decreases the lung volume.

**Effect Of Anesthesia On Ventilation:**

**Loss of airway patency**

1- In GA with or without use of NMBA, relaxation of pharyngeal muscles and posterior displacement of tongue occur causing airway problems.

2- Management of secretion is lost, as a result saliva and mucous can obstruct the oropharynx.

**Laryngospasm And Bronchospasm**

- Loss of cough reflexes allow secretion (reflux gastric content) on to the vocal cord causing laryngospasm and bronchospasm and ultimately infection.

These effect result in airway obstruction causing hypoxia and hypercapnia.

**Reduce Ventilation**

All anesthetic drugs except ketamine, ether and nitrous oxide cause reduction in ventilatory minute volume. This is because of either reduction in RR(opioids) or reduction in TV(volatile agent) or both (propofol). Thus ventilation decrease and paco2 increase.

This will cause:

1) Vasodilatation
2) Tachycardia
3) Arrhythmias
4) Hypertension

**Effect Of Anesthesia On Gases Exchange:**

**Change in FRC:**

*Functional residual capacity (FRC)* is the volume of air present in the lungs at the end of passive expiration. At FRC, is the opposing elastic recoil forces of the lung

the FRC is determined by the balance between the inward recoil of the lungs and the outward recoil of the chest wall when the respiratory muscles are relaxed. When a normal conscious person changes from the upright to the supine position, the FRC decreases by about one-third

*Residual volume (RV):* is the volume of air remaining in the lungs after maximum forceful expiration. In other words, it is the volume of air that cannot be expelled, thus causing the alveoli to remain open at all times

\[
\text{FRC} = \text{ERV} + \text{RV} \ (3000\,\text{ml})
\]

ERV; (expiratory reserve volume) Volume that can be forcefully expired after normal expiration.

RV; Residual volume is the volume of air remaining in the lungs after maximum forceful expiration. In other words, it is the volume of air that cannot be expelled.
In GA, the FRC decreases causing hypoxia

**Effect of Anesthetic Agents:**

<table>
<thead>
<tr>
<th>IV induction agent</th>
<th>Positive effect</th>
<th>Negative effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>Propofol</td>
<td>Laryngeal relaxation cause bronchodilation and LMA insertion</td>
<td>Respiratory depression</td>
</tr>
<tr>
<td>Ketamine</td>
<td>Less respiratory depression and reduction in bronchial smooth muscle</td>
<td>Increase mucous production</td>
</tr>
<tr>
<td>Etomidate</td>
<td></td>
<td>Coughing and apnea</td>
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</tbody>
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MANAGEMENT

A) Preparation:
1- Positioning patients at 45(angle) help to reduce the fall in FRC
2- Pre oxygenation increase the time for intubation
3- Anticholinergic drugs ( atropine )reduce saliva

B) Intra operative management
1) positive end expiratory pressure prevent hypoxia
2) Maintain adequate perfusion pressure with fluids and vasopressors to reduce V/Q mismatch
3) focus on capnograph and pulse oximeter
Endotracheal Tube (Et Tube)

Endotracheal intubation is the placement of a flexible plastic tube into the trachea to maintain an open airway.

Endotracheal tubes are curved tubes. These tubes were previously made up of latex (Indian rubber) and those still available, nowadays, plastic tubes made of poly vinyl chloride (PVC) are preferred because of following advantages:

1- Disposable (less chances of infection)
2- Less allergic (latex allergy is fairly common)

3- Transparent (easy visualization of blockage ETT due to blood, pus, secretions

**Components of ET**

It has the following components:

1- **Proximal End** – 15mm adapter (connector) which fits to ventilator or ambu bag

2- **Central Portion** –

1. A vocal cord guide (black line) which should be placed at the level of the opening of the vocal cords so that the tip of the ET tube is positioned above the bifurcation if the trachea.

2. A radio-opaque marker which is essential for accurate visualization of the position of the ET tube within the trachea by means of an X-ray

3. The distance indicator (marked in centimeters) which facilitates placement of ET tube.

4. A cuff - in case of cuff ET tube

**Distal End** – has Murphy’s eye (opening in the lateral wall) which prevents complete blockage of ET tube incase the distal end is impacted with secretion, blood, etc.
TYPES:

ET tubes can be: - cuffed or uncuffed tube

Cuffed ET tubes are used in children > 8 years• The cuff when inflated maintains the ET tube in proper position and prevents aspiration of contents from GI tract into respiratory tract also it provide air tide anesthesia.

In children < 8 years-- uncuffed ET tubes are used because the narrow subglottic area performs the function of a cuff and prevents the ET tube from slipping. The cuff is either:
a) High volume Low pressure cuff

b) Low volume High pressure cuff

**SIZE:**

1) 0-1 yrs. 2.5 to 3.5 mm (plain)

2) 1-3 yrs. 4. to 5 mm

3) 4-6 yrs. 5 to 6 mm

4) 6-10 yrs. 6 to 7 mm (cuffed)
5) adult female. 7 to 8 mm

6) adult male. 8 to 9 mm

The size of the tube can be determined by – internal diameter of ETT (mm) = age in years /4 + 4

Roughly the diameter of the child’s little finger is the same as that required for the ETT.

**Depth of endotracheal tube:**

Mid trachea or below vocal cord ~ 2 cms

Adult Male = 23 cms

Female = 21 cms

Children Oral endotracheal tube = (Age/2) + 12 (cm)

Nasal endotracheal tube = (Age/2) + 15 (cm)

**Indication of ET:**

Endotracheal intubation is the placement of a special tube in trachea

1- To secure airway

2- to supply oxygen

3- general Anesthesia

4- Cardio pulmonary resuscitation

5- ventilatory therapy in ICU
Complication:

1-Mechanical trauma to tongue, teeth, palate, pharynx & larynx during intubation procedure

2- Stimulation of posterior pharyngeal wall leading to coughing, vomiting or vasovagal episode with resultant hypoxia, bradycardia.

3- Tube in oesophagus

4- Right intubation: the tube is in the right main bronchus

5--Prolonged intubation may cause pressure necrosis of laryngeal structures leading to persistent hoarseness (hence tracheostomy) is indicated in patients requiring long-term mechanical ventilation)

6- Pneumothorax.

Equipment’s used for Endotracheal intubation

1. Different sizes of ETT
2. Laryngoscope
3. Magill’s forceps (different sizes)
4. Syringe to inflate cuff
5. Suction
6. Stethoscope
difficult intubation

An intubation is called difficult if a normally trained anesthesiologist needs more than 3 attempts or more than 10 min for a successful endotracheal intubation

causes difficult intubation:

1- patients with a small mouth opening, and protruding upper teeth,
2- stiff neck,
3- engorgement of the tongue
4- patients with an unstable cervical spine. and cervical joint rigidity in elderly patients
5- low Mallampati score : if the soft palate cannot be visualized (Mallampati classification)
6- narrow Interincisor gap (< 4.5 cm)

Handling: (management):

General handling of difficult intubation, use of special material including:

1- confirmation of the endotracheal position by (CO2 Et, SaO2,)
2- fiberoptic bronchoscopy,
2-video laryngoscopy: direct visualization of the trans laryngeal position of the tube

3-The laryngeal mask airway,

4-Jet ventilation: refers to delivery of oxygen via high pressure

   a- trans tracheal jet ventilation

   b- needle jet insufflation?

Needle cricothyrotomy or percutaneous jet ventilation (PCJV) can truly be a lifesaving procedure. It is a fast, effective way of providing oxygen to a patient with an obstructed airway who does not respond to more conventional means of opening the airway.

5-Awake intubation
With better preoperative evaluation and clear guidelines and training for difficult intubation, anaesthetic morbidity and mortality can be reduced.

If a difficult airway is known or suspected:
1-Ensure that a skilled individual is present or immediately available to assist with airway management when feasible.
2-Inform the patient or responsible person of the special risks and procedures pertaining to management of the difficult airway.
3-Properly position the patient,
4-administer supplemental oxygen before initiating management of the difficult airway and continue to deliver supplemental oxygen whenever feasible throughout the process of difficult airway management, including extubation.
5-Ensure that, at a minimum, monitoring according to the ASA Standards
Patient positioning in anesthesia

Patient positioning is vital to a safe and effective surgical procedure. Proper patient positioning depends on the type and length of procedure, anesthesia access to the patient, devices required and other factors.

All members of the surgical team play a significant role in the process and share responsibility for establishing and maintaining the correct patient positions.

aim of optimal positioning during anesthesia

1 – No interference with respiration
2 – No interference with circulation
3– No pressure on peripheral nerves  
4– Minimal skin pressure  
5– Accessibility to operative site  
6 – Accessibility for anesthetic administration  
7 – No musculoskeletal discomfort  

Frequently, the patient can assist in positioning prior to induction of anesthesia. However, under general anesthesia, the operating room team must carefully move and position each patient.

**Risk factors associate with patient position:**

1- advanced age  
2- nutritional status  
3- respiratory disorder  
4- circulatory disease  
4- obese patient  
5- chronic immobility  
6- nature of surgery  

**Pulmonary Concerns**

1- Any position which limits movements of abdomen, chest wall or diaphragm increase atelectasis  
2- Change from standing to supine - decrease FRC due to cephalic displacement of the diaphragm
Surgical Positions

Four basic surgical positions include:

1– Supine 2– Lateral
3– Prone 4– Lithotomy

Variations include:

– Trendelenburg  – Reverse Trendelenburg
– Fowler’s/semi fowler  – Beach chair position
– kidney position  – Position for robotic surgeries

Supine position
The supine is the most common position used in the operating room. Typically, the head is rested on a foam pillow, keeping the neck in a neutral position. The patient’s arms are either tucked at their side or abducted to less than 90 degrees on padded arm boards.
Fowler's Position

Fowler's position, also known as sitting position, is typically used for neurosurgery and shoulder surgeries.

The beach chair position is often used for nasal surgeries, abdominoplasty, and breast reduction surgeries. When positioning a patient in Fowler's position, the surgical staff should minimize the degree of the patient's head elevation as much as possible and always maintain the head in a neutral position.

Prone Position

In Prone position, the patient is face-down with their head in a neutral position without excessive flexion, extension or rotation. A face positioner is used when the patient's head is in midline. Prone position is often used for spine and neck surgeries, neurosurgery, colorectal surgeries, vascular surgeries, and tendon repairs.
Lithotomy Position

In Lithotomy position, the patient can be placed in either a boot-style leg holder or stirrup-style position. Modifications to this type of position include low, standard, high. This position is typically used for gynecology, colorectal, urology, perineal, or pelvis procedures. The risks in a Lithotomy position include fractures, nerve injuries, hip dislocation, muscle injuries, pressure injuries, and diminished lung capacity.
**Lateral Position:**

A patient may be positioned in Lateral position during back, colorectal, kidney, and hip surgeries. It's also commonly used during thoracic and ENT surgeries, and neurosurgery. Some variations on this position include Lateral Kidney, Lateral Chest and Lateral Jackknife positions. In Lateral position, the patient may be placed on either their left or right side depending on the side of the surgical site. A pillow or head positioner should be placed under the patient's head with the depended ear assessed after positioning. a safety restraint should be secured across the patient's hips.

**Trendelenburg Position:**

Trendelenburg position is typically used for lower abdominal, colorectal, gynecology, and genitourinary surgeries, cardio-version, and central venous catheter placement. In this type of position, the patient's arms should be tucked at their sides, and the patient must be secured to avoid sliding on the surgical table. The Trendelenburg position should be avoided for extremely obese patients.
Risks to a patient while in this position include diminished lung capacity, diminished tidal volume and pulmonary compliance, venous pooling toward the patient's head, and sliding

**Reverse Trendelenburg Position:**

Reverse Trendelenburg position is typically used for laparoscopic, gallbladder, stomach, prostrate, gynecology, Risks to a patient in this type of position include deep vein thrombosis, sliding
Operative Nursing Roles:
1- Ensure patient is fully anaesthetized before positioning
2- Never reposition without anesthetist supervision
3- Table fitting must be placed without obstruction to incision site
4- All fitting and attachments must be secure completely
5- Applying diathermy plate

Position during induction of anesthesia:
supine position • head extended • neck flexed • The aim is to visualize oral, pharyngeal and tracheal spaces
possible complications – Trauma to lips and teeth, Jaw dislocations, laryngeal or vocal cords injury, epistaxis and trauma to pharyngeal wall

Positioning of elderly patient:
Elderly patient usually has fragile skin surfaces • arthritic joints • limited range of motion and sometime paralysis •
1- lifting the patient rather than sliding •
2- avoid of adhesive tape for strapping •
3- adequate padding for bony prominences •
4- allow patient to positioning before anaesthetized

Positioning of Pediatric patient:
think of ‘appropriate size’ • right size for bed and attachments
never overextended limbs or keep in one position for longer periods.

due to small size, children are prone to and has greater risk.

**Surgery:**

is a procedure that involves cutting of a patient's tissues or closure of a previously sustained wound. (Or) Surgery is defined as the treatment of injuries or disorders of the body by incision or manipulation, especially with instruments.

**Types Of Surgery and classification**

Surgical procedures are classified according to

A) Urgency   B).Risk   C).Purpose

A) **Surgery Based On Urgency**

1.Elective surgery

2.Urgent surgery

3.Emergency surgery

**Elective Surgery**

It is a procedure that is pre-planned and based on patients choice and availability of scheduling for the patient, surgeon and the facility. • Delay of surgery has no ill effects.
Examples: Hernia repair, Cataract extraction, Tonsillectomy, Hip prosthesis

**Urgent Surgery**
Must be done within a reasonably short time to preserve health. Usually done within 24 – 48 hours • Examples: Removal of gall bladder, Amputation, Appendectomy

**Emergency Surgery**
Must be done immediately to preserve life, a body part or function. Examples: Control of haemorrhage, Repair of trauma, perforated ulcers, intestinal obstruction.

**B) Surgery Based On Degree Of Risk**

1. **Major surgery**
   - requires hospitalization and specialized care, is usually has a higher degree of risk, involves major body organs or life threatening situations, and has a greater risk for postoperative complications.
   - Examples: Open Cholecystectomy, Nephrectomy, Hysterectomy, Radical mastectomy, Laparotomy
2- **Minor surgery**: is usually brief, carries a low risk and results in few complications. • Minor surgeries are mostly elective. • Examples: Teeth extraction, Cataract extraction

**C) Surgeries Based On Purpose**

1- **Diagnostic**: Surgeries to make or confirm a diagnosis • Examples biopsy, Bronchoscopy, Endoscopy

2- **Ablative**: Surgeries to remove a diseased body part. • Examples: Appendectomy, Amputation

3- **Palliative**: Surgeries to relieve or reduce intensity of an illness. It is not curative. • Examples: Colostomy, Nerve root resection

4- **Reconstructive**: Surgeries to restore function to traumatized or malfunctioning tissue or to improve self-concept. • Examples: Scar revision, Plastic surgery, Internal fixation of a fracture, Breast reconstruction

5. **Transplantation**: Surgeries to replace organs or structures that are diseased or malfunctioning. • Examples: Kidney, liver, heart transplantation.

6. **Constructive**: Surgeries to restore functions in congenital anomalies. • Examples: Cleft lip Repair, Closure of Atrial Septal Defect
Hypoxia during anesthesia

**Hypoxia**: reduction of oxygen supply at the tissue level, which is not measured directly by a laboratory value

**Hypoxemia**: a condition where arterial oxygen tension or partial pressure of oxygen (PaO2) is below normal (normal value is between 80 and 100 mmHg)

**Dysoxia**: The condition where the energy produced as a result of nutrient metabolism is limited by the availability of oxygen. Dysoxia can be the result of an inadequate supply of O2, which results in tissue
hypoxia, Or caused by a defect in oxygen utilization in the mitochondria, which is called cytopathic hypoxia.

Hypoxemia is considered as one of the most serious risks for patients during anesthesia and surgical care. Pulse oximetry has become an essential component of operating room to detect, treat, and reduce the degree of intraoperative hypoxemia.

hypoxemia is the most common cause of tissue hypoxia.

**Hypoxia and brain function:**

![SpO₂ (%)](chart.jpg)
The brain requires a significant amount of oxygen to help it metabolize glucose and power the function of neurons. In fact, although the brain only makes up 2% of your body mass, it uses up to 20% of your regular oxygen supply!

When brain cells don’t get enough oxygen, they begin to die off in as little as a minute. After five minutes, there is risk of brain death and lasting brain damage, even if the oxygen supply is restored.

**Classification of hypoxia:**

1- **Hypoxemic hypoxia:** Insufficient oxygen reaching the blood  
2- **circulatory hypoxia:** Decreased blood flow to the tissues  
3- **Anaemic hypoxia:** Decreased oxygen carrying capacity of blood  
4- **Histotoxic hypoxia:** Impaired utilization of oxygen by the tissues

**What are the requirements for adequate oxygen transport?**

1- Fraction of oxygen in inspired air (FiO2 )  
2- Partial pressure of oxygen in inspired air ( PiO2 )  
3- Alveolar ventilation (VA)  
4- The relation of ventilation to perfusion in the lungs (VA/Q)  
5- cardiac output and blood volume  
6- Hb content  
7- Arterial blood pH, body temperature
Preoperative hypoxia

Etiology of hypoxia before anesthesia?

1- Pre-existing cardiopulmonary disease: Already low PaO2
2- Trauma: May involve ventilatory and circulatory systems • May cause atelectasis and shunting increased by immobilization and pain medication.
3- Old age: • PaO2(mmHg) after age of 60, the range is from 70 to 80 mm Hg.
4- Obesity
5- Severe anemia
6- Drug administration

Intra Operative Hypoxemia

Hypoxia is common during surgery, In fact, some studies have revealed that even in highly advanced surgical settings, as many as one in 15 patients experience hypoxia for at least two consecutive minutes during surgery and one in 64 patients experience it for five or more minutes

General anesthesia and mechanical ventilation will impair the pulmonary function, even in normal individuals, and result in decreased oxygenation in the post-anesthesia period.
Causes of Intra Operative Hypoxemia: Classified into two:

A) Problems with oxygen delivery system:

1- at the level of central oxygen supplies: Gas leak, Inadequate pressure at central supply, Decreased oxygen level at the tank

2- At the level of pipeline distributing system: Leak, Connecting wrong hose to Oxygen yoke

3- At the level of oxygen cylinders attached to anaesthesia machine: Empty cylinders, Substitution of non-oxygen cylinder at the yoke, Insufficiently opened cylinder

4- At the level of anaesthesia machine: Incorrect setting of flow meter, Crack in the oxygen flow meter tubes, Leak in machine

5- At the level of anaesthesia Ventilator: Low tidal volume, Low respiratory rate, Inadequate minute volume, Disconnection of tubing

6- At the level of the anaesthesia circuit: Disconnection

7- At the level of Endotracheal tube
   a- Esophageal intubation
   b- Endobronchial intubation
   c- Accidental extubation
   d- Kinking of the endotracheal tube

B) Problems with patient

1- Hypoventilation: a- due to drug induced respiratory depression.

   b- due to inadequate IPPV
2- **Reduced functional residual capacity:** Induction of GA will cause reduction in FRC by 15-20%. This will be more in patients with preexisting lung disease, obese patients. The reduction FRC is continued in post-operative period. The reduction in FRC may be restored normal by application of PEEP.

3- **Increased airway resistance:** Due to following factors
   a. Reduction in FRC
   b. Decrease in caliber of airways
   d. Anaesthesia apparatus
   e. Laryngospasm
   f. Obstruction of ETT

4- **Atelectasis:** It is a condition of alveolar collapse. Atelectasis occur due to airways secretions, and prolonged procedures. PEEP may be useful in such situation

5- **Diffusion defect:** Even adequate oxygen is supplied to the alveoli, defect at alveolar level which prevents its absorption in to blood. This is due to:
   a. Thickened alveolar membrane
   b. Inflammation
   c. Edema
   d. Fibrosis or loss of alveolar surface area (Eg: sarcoidosis Emphysema)
6- Poor oxygen delivery to tissues: Due to following
   a. Systemic hypo perfusion
   b. Embolus
   c. Sepsis
   d. Local problem: cold limb, Reynaud phenomenon, sickle cell disease

7-Increase oxygen demand: Malignant hyperpyrexia. Shivering and Sepsis

Postoperative hypoxia:

1-Diffusion hypoxia: The solubilities of nitrogen and nitrous oxide differ sufficiently to reduce PAo2 when nitrous oxide anesthesia is stopped and the patient allowed to breathe room air. • Nitrous oxide comes out of solution and enters the alveoli more rapidly than nitrogen which cause washing of the alveoli from O2 and CO2.

2- Prolonged effect of muscle relaxants

3. Pain: pain may restrict the patient from taking a normal breathing especially after thoracic and upper abdominal operation

4. Pain relieving drugs: mainly morphine • result in an increased incidence of respiratory complications. • high doses which depress ventilation • morphine may reduce the reflex activity.
Diagnosis of hypoxia during operation:
During early days of anaesthesia, defective oxygenation of the patient was identified by cyanosis & dark blood in the surgical field. Cyanosis usually observed when Hb saturation is 85%. Cyanosis may not be apparent in the presence of anaemia or peripheral vasoconstriction.
Several monitors are used now to detect hypoxemia.

1- Pulse oximeter is most commonly used

2- Other monitors:
   a) oxygen analyzer
   b) ABG
   c) Airway pressure monitor
   d) Capnography

Management of intraoperative hypoxia:
1. Expose the chest, & all airway connections
2. Give 100%O2 (FiO2=1.0)
3. Hand ventilation
4. Confirm ETT position (auscultation, endobronchial, obstruction)
5. Check the ventilator pattern is correct
6. Decreased FRC – hyperventilate gently with PEEP, Absorption Atelectasis – decrease FiO2 and remove secretions
7. Increased airway resistance - deepen anaesthesia, salbutamol nebulization, volatile anaesthetics, inject Aminophylline infusion
8. Hypovolemia – IVF, Blood. Increased O2 demand: give 100% O2

**Prevention of intraoperative hypoxia:**

1- Anaesthesia machine checkup should carried out properly before every anaesthetic procedure.
2- Use machine with O2 pressure failure alarm
3- Hypoxic guard
4- O2 flow meter tubes placed down stream
5- Check valve to prevent flow of gases from the machine to cylinder or pipeline
Hypoxia in special condition

**In Children:** Neonates & infants are prone for more rapid desaturation (hypoxia) due to:

1- Smaller diameter of airways.

2- Increased oxygen consumption.

3- Difference in airway anatomy – difficult intubation, mask ventilation.
In pregnancy: hypoxia in pregnancy is due to:

1- FRC reduced by 20%, oxygen reserve decreased •
2- Oxygen consumption increased by 20% •
3- Difficult intubation, difficult ventilation.
4- Incidence of aspiration worsen the situation •

Preoxygenation is must, rapid sequence induction is preferred using sellick’s maneuver (Cricoid pressure to occlude the upper end of the oesophagus, Sellick’s maneuver, may be used to decrease the risk of pulmonary aspiration of gastric contents during intubation for rapid induction of anaesthesia.

In elderly: They are more prone for hemoglobin desaturation due to:

1- Reduced compliance, increased residual volume, loss of vital capacity 2- Impaired efficiency of gas exchange, •
3- Compromised cardiovascular system •
4- Prolonged drug effect seen after sedatives, narcotics & muscle relaxants

In obese patients: due to:
1- Difficult mask ventilation,
2- difficult laryngoscopy, difficult intubation •
3- Decreased lung volumes & capacities.
4- FRC is further reduced in supine position •
5- More sensitive to depressant effects of hypnotics & opioids

**one lung ventilation:**
Hypoxemia occurs in almost all cases during one lung ventilation
This is due to V/Q mismatch, because the non-dependent lung is
not ventilated but continues to get perfused.

**Measures to be taken to maintain oxygenation during OLV**
1. Two lung ventilation as long as possible
2. High FiO2=100 %
3. Begin OLV with VT=10ml/kg
4. Adjust RR so that PaCO2 =40mm of Hg
5. Monitor oxygenation & ventilation continuously
6. Non-dependent lung-- CPAP
7. Dependent lung-- PEEP
8. Intermittent two lung ventilation
9. Clamp pulmonary artery as soon as possible

The V/Q ratio is the amount of air that reaches your alveoli
divided by the amount of blood flow in the capillaries in your lungs.
When your lungs are functioning properly, 4 liters of air enter your respiratory tract while 5 liters of blood go through your capillaries every minute. for a V/Q ratio of 0.8. A number that’s higher or lower is called a V/Q mismatch

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**Post-operative hypoxia**

Post-operative hypoxia is caused by several factors:

**I- General—patient’s factors**

a) preoperative problems such as:

b) Extreme obesity

c) Geriatric age

d) Pre-existing cardiopulmonary disease

**2- Diminished inspired oxygen**

a) Lack of oxygen supplementation devices

b) Excessive suctioning

c) Diffusion hypoxia after nitrous oxide

**3- Impaired ventilatory efficiency**
a) Obstructed upper airway due to tongue fall, large uvula, laryngeal spasm, vocal cord palsy,
b) Obstructed lower airway due to: Secretions, Bronchospasm, Atelectasis
c) Muscular weakness
d) Pharmacological causes: Residual effects of respiratory depressant drugs such as anaesthetic agents, narcotic analgesics, neuromuscular blocking agents.

4-The other factors that cause Post-operative hypoxia are:

a) Surgical factors such as binders or type of incision
b) Postoperative pain
c) Excessive analgesics medication postoperatively

**Signs And Symptoms Of Hypoxia**

Hypoxia symptoms vary depending on the severity, underlying cause and what parts of your body are affected. When your oxygen is low, you might feel like you can’t breathe or think properly. Some hypoxia symptoms include:

- Restlessness.
- Headache.
- Confusion.
- Anxiety.
- Rapid heart rate (tachycardia).
- Rapid breathing (tachypnea).
- Difficulty breathing or shortness of breath (dyspnea).

Severe hypoxia can cause additional symptoms:

- Slow heart rate (bradycardia).
- Extreme restlessness.
- Bluish skin (cyanosis).
Symptoms of Hypoxia

- Restlessness.
- Headache.
- Confusion.
- Rapid Heart Rate (tachycardia).
- Rapid Breathing (tachypnea).
- Anxiety.
- Difficulty breathing (dyspnea).

Symptoms of Severe Hypoxia
Treatment:

1- Treatment of the cause

2- Oxygen administration. Supplemental oxygen is administered at atmospheric pressure of 760 mmHg by a nasal cannula or simple oxygen facemask connected to an oxygen reservoir.

If a patient requires oxygen therapy, choose an oxygen delivery system based on your patient’s requirements. Oxygen is initially started at a low concentration (2 L/min) using nasal prongs. Then the flow is titrated up to maintain oxygen saturation of 92% or greater.

Diffusion hypoxia (Fink’s effect):

It occurs at the end of G.A when N2O :O2 is switched off and patient allowed to breathe air. N2O is 31 times more soluble than nitrogen. For every one molecule of nitrogen entering into blood from alveoli, 31 molecules of N2O enters into alveoli from blood. The alveolar oxygen is diluted and hypoxemia results. This is more common during first 5-10 minutes of recovery. Administration of 100% O2 is essential to overcome this situation.
Hypercapnia and Hypocapnia

For most patients receiving general or regional anesthesia, the arterial carbon dioxide tension (Paco₂) should be maintained within normal physiologic limits (35-45 mm Hg). Alterations in homeostasis may lead to hypercarbia or hypocarbia.

Hypocarbia (Hypocapnia)

Hypocarbia, also known as hypocapnia, is a decrease in alveolar and blood carbon dioxide (CO₂) levels below the normal reference range of 35 mmHg.

CO₂ is a metabolic product of the many cellular processes within the body involved in the processing of lipids, carbohydrates, and proteins.
The primary organ systems responsible for regulating CO₂ homeostasis are the pulmonary system and the renal system. Additionally, CO₂ is regulated through the CO₂/HCO₃ pH buffering system.

**Respiratory Alkalosis**

Hypocapnia often leads to a condition called **respiratory alkalosis**;

Respiratory alkalosis occurs when the CO₂ level of the blood drops below normal and the pH of the blood becomes too high.

This happens because your body naturally tries to produce more CO₂ when your levels are low. The body does this by joining bicarbonate (HCO₃⁻) and hydrogen ions to create more CO₂ molecules. The hydrogen ions determine blood pH; by using up hydrogen ions to create CO₂, the pH of the blood increases and becomes basic.

Respiratory alkalosis is when people have both hypocapnia (low blood CO₂) and alkalosis (high blood pH).

**Presentation**

Hypocarbia, or hypocapnia, occurs when levels of CO₂ in the blood become abnormally low (Paco₂ <35 mm Hg). Hypocarbia is confirmed by arterial blood gas analysis. Hypocarbia, especially if only transient, is usually well tolerated by patients.

Hyperventilation, leading to hypocarbia, is often used to decrease intracranial pressure in neurosurgical patients.
Causes of hypocarbia:

A. Increased Carbon Dioxide Elimination

1. Hyperventilation
   
   a) Excessive minute ventilation in mechanically ventilated patients and in spontaneously ventilating patients in
      
      Response to metabolic acidosis
      
      Response to pain
      
      Pregnancy

2. Decreased dead space ventilation

3. Decreased CO$_2$ rebreathing

B. Decreased Pulmonary Perfusion

1. Decreased cardiac output
   
   o Hypovolemia
   
   o Hypotension
   
   o Cardiac arrest

2. Pulmonary embolism

C. Decreased Carbon Dioxide Production

1. Hypothermia

2. Deep anesthesia
3. Hypothyroidism
4. Decreased metabolism

D. Airway/Equipment Problems

1. Esophageal intubation
2. Accidental extubation or circuit disconnection
3. Dilution with circuit gases

Effects Of Hypocapnea:

1. Effects on Respiration: Respiratory centers are depressed, leading to decreased rate and force of respiration.

2. Effects on Blood: The pH of blood increases, leading to respiratory alkalosis. Calcium concentration decreases. It causes tetany?, which is characterized by neuromuscular hyper-excitability and carpopedal spasm.

Carpopedal spasm. : a spasmodic contraction of the muscles of the hands, feet, and especially the wrists and ankles (as that occurring in alkalosis and tetany)

3. Effects on Central Nervous System: Dizziness, mental confusion, muscular twitching and loss of consciousness are the common features of hypocapnia.
Management

1. Assess oxygenation status
2. Obtain arterial blood gas to confirm capnography results
3. Since the most common cause of hypocarbia during surgery is iatrogenic hyperventilation, the first step in management should focus on decreasing minute ventilation
4. Assess and restore circulation if the problem involves decreased cardiac output

Hypercarbia

Presentation

Hypercarbia, or hypercapnia, occurs when levels of CO\textsubscript{2} in the blood become abnormally high (Paco\textsubscript{2} $>$ 45 mm Hg). Hypercarbia is confirmed by arterial blood gas analysis.

In the awake or sedated patient, signs and symptoms include dyspnea, sweating, muscle tremors, flushed skin, headache, lethargy, and confusion. Spontaneously breathing patients develop tachypnea while mechanically ventilated patients may over breathe the ventilator.

In patients breathing room air or low inspired oxygen concentrations, severe hypercarbia leads to severe hypoxemia.
Causes of Hypercapnia:

conditions when Hypercapnia occurs:

1- Breathing the air containing excess carbon dioxide content.

2- In case of Asphyxia. (Asphyxia is the condition characterized by combination of hypoxia and Hypercapnia, due to obstruction of air passage)

3- Hypoventilation

4- Lung disease

5- Diminished consciousness

Effects Of hypercapnia:

1. Effects on Respiration: During hypercapnia, the respiratory centers are stimulated excessively. It leads to dyspnea.

2. Effects on Blood: The pH of blood reduces and blood becomes acidic.

3. Effects on Cardiovascular System: Hypercapnia is associated with tachycardia and increased blood pressure.

4. Effects on Central Nervous System: During hypercapnia, the nervous system is also affected, resulting in headache, depression and laziness.
Hypercapnia during anesthesia:

Causes:

Hypercapnia, or high PaCO2, can occur during anesthetic and post-anesthetic care. In the operating room, hypercapnia is typically detected with Capnography.

When assessing a patient with known or suspected hypercapnia, one should assess for a number of potential causes:

1- Hypoventilation Inadequate ventilation is the most common cause of hypercapnia. In mechanically ventilated patients this is typically iatrogenic and the result of inadequate tidal volumes and/or respiratory rate contributing to a low minute ventilation. In spontaneously breathing patients, this is typically due to drug-induced depression of the ventilatory response to CO2. Common causative agents are opioids, benzodiazepines, other sedative hypnotics (i.e. propofol), and halogenated inhalational agents.

2- Rebreathing When under general anesthesia, faulty breathing circuits or inadequate fresh gas flow in some circuit types, can lead to an increase in the inspired CO2 and consequently and increase in the expired CO2. Exhausted absorbent agents (soda lime) and faulty expiratory check valves are the most common causes in modern anesthesia machines.
3- Increased CO2 Production In several physiologic/pathologic states, the body can produce excessive carbon dioxide, resulting in hypercapnia under anesthesia.

a) Fever results in a hypermetabolic state and increased CO2 production.

b) Systemic absorption during laparoscopic procedures using CO2 of insufflation.

c) Malignant hyperthermia

4- Increased Dead Space While all patients have a degree of anatomic dead space (25-30%) can result in hypercarbia due to excessive minute ventilation being delivered to areas of the lung not actively participating in gas exchange. This is most commonly seen in patients with obstructive lung diseases.

Management

1- Ensure adequate oxygenation

2- Ensure adequate ventilation

3- Check FiO2

4- Blood gases to confirm Capnography

5- Consider secondary causes, especially those requiring specific treatment (eg. MH)

6- Treat complications of hypercapnia
Controlled Hypotensive Anaesthesia

**Definition:**
It is a state of induced controlled hypotension during anaesthesia to reduce bleeding and improve the surgical field adjusted to the patient’s age, pre-operative blood pressure and past medical history. First used by Cushing in 1917.

**Advantages:**
1. Decreases blood loss during surgery
2. Decreases operative time
3. Provides bloodless operative field
4-Avoid the needed for intraoperative blood transfusion which may associated with many complications.

**Principle:**

1- Reduction in systolic blood pressure to 80 – 90 mmHg.
2- Decrease in MAP to 50 – 60 mmHg in normotensive patients.
3- Reduction in MAP by 30% of the baseline values.

**Mean arterial pressure (MAP):** is the average arterial pressure throughout one cardiac cycle, (systole, and diastole). MAP is influenced by cardiac output and systemic vascular resistance,

**What is normal MAP (mean arterial pressure)?**

In general, most people need a MAP of at least 60 mm or greater to ensure enough blood flow to vital organs, such as the heart, brain, and kidneys. Doctors usually consider anything **between 70 and 100 mm Hg** to be normal

**What is MAP formula?**

To calculate a mean arterial pressure, double the diastolic blood pressure and add the sum to the systolic blood pressure. Then divide by 3. For example, if a patient's blood pressure is 83 mm Hg/50 mm Hg, his MAP would be 61 mm Hg. Here are the steps for this calculation:

$$MAP = \frac{SBP + 2(DBP)}{3}$$
Vital Organ Physiology:

Controlled hypotension rarely results in damage because organ blood flow is normally well maintained. Three main organs whose proper functioning is vital which auto-regulate their blood pressures include: Brain, Kidney and Heart

Cerebral Circulation: perfusion of the cerebral circulation is the critical factor that limits MAP reduction. Auto regulation – MAP range of 50-150mmHg

There are various Factors Under Control Of Anesthetists To Maintain MAP. These factors include:

1- paCO2 (partial pressure of Arterial CO2) : increase in PaCO2 there is an increase in cerebral blood flow

2- paO2 High O2 mainly in hyperbaric range can lead to cerebral damage and thus the brain compensates by Vasoconstriction. If O2 below normal then Vasodilation

3- Volatile anaesthetic : Volatile anesthetics attenuate or abolish the auto regulation of cerebral blood flow in a dose dependent manner

Coronary Circulation:
Coronary blood flow is dependent upon the aortic diastolic blood pressure and the coronary vascular resistance.
Renal Blood Flow

Renal blood flow is controlled in two ways: Extrinsic autonomic and hormonal mechanisms and Intrinsic autoregulation.

Aim of hypotensive anesthesia:

The aim of Hypotensive anaesthesia is to reduce blood loss and provide a “dry” operating field. Hence, the degree of hypotension should be individualized.

The hypotension should be considered satisfactory when bleeding appears to be minimal and organ perfusion adequate. Inducing hypotension to a MAP of 30% below a patient’s usual MAP, with a minimum of 50mmHg in young patients and 80mmHg in the elderly is clinically acceptable.

Conditions that limit the use of hypotensive anesthesia;

A-Patient Limitations:

1- Cardiac disease
2- Diabetes mellitus
3- Anaemia, haemoglobinopathies, polycythemia
4- Hepatic disease
5- Ischemic cerebrovascular disease
6- Renal disease
7- Respiratory insufficiency
8- Severe systemic hypertension
9- Intolerance to drugs available to produce hypotension

**B-Anesthetist Limitations:**

1- Lack of understanding of the technique.

2- Lack of technical experience.

3- Inability to monitor the patient adequately.

**Indications of hypotensive anesthesia:**

1. Expected major blood loss
2. Orthopedic surgeries: scoliosis, revision hip surgery.
3. Major vessel surgery.
5. Microsurgery: Plastic, Middle ear,

**Technique:**

MAP = Cardiac Output x Systemic Vascular Resistance The key equation in the provision of hypotensive anaesthesia .

MAP can be manipulated by reducing either SVR or Cardiac output or both.

**MAP = Cardiac Output X Systemic Vascular Resistance = (Stroke volume x Heart rate) x SVR**

Methods To Reduce Cardiac Output
Methods To Reduce Peripheral Vascular Resistance

1-Arterial bleed directly proportional to MAP – beta blockers reduce Stroke volume and Alpha blockers reduce Peripheral vascular resistance.
2-Capillary bleed by local adrenaline
3- hyperventilation to reduce arterial and venous PaCO2
4-Venous Tone reduced by intravenous nitrates,
5- positioning

Methods To Reduce Cardiac Output And Reduce Peripheral Vascular Resistance:

1-Inhalational anesthetics: has negative inotropic effect and vasodilation. Isoflurane is ideal • Halothane is OK but think of bradycardia and myocardial depression.

2-Nitrates drugs Which includes :
   A. Sodium nitroprusside (SNP).
   B. Nitroglycerin (GTN).
both arteriolar and venous vasodilator
Sodium nitroprusside is a potent and reliable antihypertensive. It is usually diluted to a concentration of 100 mcg/mL and administered as a continuous intravenous infusion (0.5–10 mcg/kg/min). and the use of mechanical infusion pumps.
3-Beta adrenergic antagonist
4-Calcium channel blocker
How do you induce hypotension?

Sodium nitroprusside, nitroglycerin, or β-blockers are given intravenously, possibly with continuous positive airway pressure and deep inhalation anesthesia, to initiate and maintain induced hypotension.

New Techniques to induce hypotension

Use the natural hypotensive effects of anaesthetic drugs with regard to ideal hypotensive agent:
- Remifentanil (0.05-2 μg/kg/min)
- Propofol (2-3 mg/kg)
- Sevoflurane (2-2.5 %)
- Clonidine IV (α2 agonist)

Mechanical Maneuvers’ To Potentiate The Action Of Hypotensive Agents

1-Positioning: Position of the patient is critical to ensure success of the controlled hypotensive technique.

Elevation of the site of operation allows easy venous drainage from the site of surgery. This is critical to ensure a bloodless field. Change in blood pressure is at a rate of 0.77 mmHg per cm change in vertical height from the heart.

Hypotensive patients are often placed in the Trendelenburg position while resuscitative efforts, such as establishing intravenous access and administering fluids, are initiated.
The Trendelenburg position was thought to increase venous return and thereby increase cardiac output

2. **Positive airway pressure**: An attractive adjunct to hypotensive anaesthesia is the use of positive pressure ventilation:
   1. with high tidal volumes,
   2. prolonged inspiratory times and
   3. raising positive end expiratory pressure.

**Anaesthetic management**

**Preoperative management**
Good knowledge by the anesthetist.
Proper patient evaluation and selection.
HB of 10 g/dl.
Arterial blood gas analysis sampling.
Good level of anxiolytics, analgesics.
Vagolytic drugs (atropine) should be avoided.

**Intraoperative management:**
1- Stress free induction. (smooth induction)
2- Enough peripheral venous access.
3-Monitoring:
   - HR, NIBP, SPO2, ETCO2
   - Invasive blood pressure.
   - ECG V5 lead
Central venous pressure.
Urine output. and Temperature monitoring

**Fluid therapy**:
Proper fluid therapy is essential during hypotensive anaesthesia. Preoperative fluid status must be assessed and corrected. Blood loss must be replaced with an equal amount of colloid or three to four times the amount of crystalloid. If the blood loss exceeds a predetermined level (eg. 20-25% of the patient’s total blood volume), a blood transfusion is warranted.

**Postoperative management**:
Be aware about Rebound hypertension and Reactionary hemorrhage.
Alcohol And Anesthesia

Alcohol use disorder (AUD) is defined as the repetitive, long-term ingestion of alcohol in ways that impair psychosocial functioning and health.

**Effect of Alcoholism:**

**Vitamin deficiencies:** Alcohol abuse is the leading cause of thiamine (vitamin B1) deficiency.

**Metabolic abnormalities:** Acidosis--- Up to 25% of patients with an alcohol use disorder will have metabolic acidosis on admission.
Magnesium: due to poor dietary intake and increased urinary and faecal losses.

Phosphate: Hypophosphatemia arises as a result of increased renal excretion.

Alcoholic liver disease: alcoholic fatty liver, alcoholic hepatitis or alcohol-related cirrhosis.

Pancreatitis: Alcohol is the major causative factor of acute pancreatitis in about 32% of cases.

Immune dysfunction: Patients with AUD have a three to five-fold increased postoperative infection rate compared to non-alcoholic patients.

Alcoholic cardiac dysfunction: Chronic alcohol ingestion leads to alcoholic cardiomyopathy. Increased risk of stroke and hypertension

Pre-operative assessment:

- The period of abstinence (stop drinking alcohol) in the preoperative period decreases postoperative morbidity.

- Extensive history.

- Full physical examination, with special attention to cardiac and respiratory systems
**Preoperative investigation:**

1- CXR and ECG

2- Electrolyte and biochemical profile

3- Full blood count,

4- INR (international normalized ratio). An INR test measures the time for the blood to clot. It is also known as prothrombin time or PT. It is used to monitor blood-thinning medicines. In healthy people an INR of 1.1 or below is considered normal. An INR range of 2.0 to 3.0 is generally an effective therapeutic range for people taking warfarin.

**Peri-operative period**

- Altered induction agent dose.
- Rapid sequence intubation if acute intoxication.
- Intraoperative - Lower MAC of inhaled agents in acute intoxication.
- Careful opioid administration.
- Muscle relaxants with organ independent metabolism.

**Postoperative**

1- Risk alcohol withdrawal syndrome.

2- Choose analgesia carefully.
Anaesthetic drugs

- Propofol - Decreased dosing requirement in acute intoxication and increased dosing requirement in chronic alcoholism.
- Thiopentone - Decreased dosing requirement in acute intoxication.
- Etomidate - No evidence of altered doses.
- Neuromuscular Blocking agents - Altered pharmacokinetics with increased volume of distribution and decreased binding proteins in alcoholic liver disease.
- Inhalation anesthetics - Decreased MAC in acute intoxication.
- Decreased clearance of halothane in alcoholic liver dysfunction
- Opioids - Decrease metabolism of morphine, Pethidine and fentanyl in chronic alcoholism and risk of accumulation with repeated doses.

**Alcohol withdrawal syndrome (AWS):**

It is important for anesthesiologist to know the symptoms, clinical signs and management of alcohol withdrawal symptoms. It is one cause of post-operative delirium.

**Signs and symptoms of AWS:**

If you stop drinking alcohol suddenly, it can cause withdrawal symptoms such as, nausea and vomiting, anxiety, insomnia, agitation,
fever, extreme sweating and seizures. You may experience other symptoms such as high blood pressure, fast heart rate, or confusion. You can be at risk for other complications such as bleeding, infections, and heart problems.

You can give medications to prevent and reduce symptoms of alcohol withdrawal, but only if you let them know how much you drink every day!

Alcohol withdrawal can begin four to 12 hours after your last drink and can last up to 24 hours. The more you drink daily, the higher your chance of experiencing alcohol withdrawal.

**How does alcohol affect Anaesthesia?**

General anesthesia involves powerful drugs that suppress functions such as breathing, heart rate, and blood circulation. Alcohol affects the same body systems. All these substances must be metabolized in the liver, which may not be able to keep up. In addition, both anesthesia and alcohol can cause nausea and vomiting and increase the risk of aspiration.

**Do heavy drinkers need more anesthesia?**

These findings suggest that the doses of propofol required to induce anesthesia in chronic alcoholic patients are more than in patients who drink socially.
How long should you not drink alcohol before surgery?

If you drink alcohol, do not consume it for at least 48 hours before surgery.

Alcohol use, especially the amount of alcohol you drink daily, can affect your surgery and recovery. Decreasing your use of alcohol or stopping altogether before surgery will help speed up your recovery and reduce your risk of developing complications post-operatively. It is very important to let your healthcare team know how much you drink daily so that we can better plan your care.

Side effects of alcohol use during surgery

1- Interferes with anesthesia. You may require higher doses during surgery.
2- Interferes with specific .This means you could require more or less for the medication to work.
3- Increased surgical recovery time requiring longer hospital stay.
4- Delayed wound healing and surgical site infections.
5- Increase in bleeding episodes requiring transfusion post-operatively.
6- Increased risk of infection after surgery, such as pneumonia, urinary tract infection, and sepsis.
Anemia:  

Anemia is defined as a reduction of the hemoglobin concentration or red blood cell (RBC) volum below the range of in healthy persons.

Normal hemoglobin and hematocrit (packed red cell volum) vary with age and sex.

Causes of anemia

1- Iron deficiency anemia. This most common type of anemia is caused by a reduction of iron in the body.

2- Vitamin deficiency anemia.
3- Anemia of inflammation.

4- Aplastic anemia.

5- Anemia’s associated with bone marrow disease.

6- Hemolytic anemia’s.

7- Sickle cell anemia.

**Pathophysiology:**

Tissue oxygen delivery is determined by the oxygen content of the blood and the cardiac output (CO)

The major proportion of oxygen contained within the blood is bound to haemoglobin. thus anaemia results in a significant reduction in oxygen delivery

Anemia is a common blood disorder of perioperative patients. The primary physiologic consequence of severe anemia is inadequate tissue oxygen delivery, which may lead to tissue hypoxia, biochemical imbalances, organ dysfunction, and ultimately organ damage. Mismanagement of the anemic surgical patient can adversely affect perioperative outcomes.

**Diagnosis:**

Both hematocrit (HCT) level and hemoglobin (Hb) concentration measurements reflect the body's RCM (Red Cell Mass)
The HCT level, defined as the fractional volume of sampled blood that erythrocytes occupy, is an indirect measurement of the body's RCM.

The HCT is a simple, commonly used test to indirectly assess the severity of anemia as well as estimate whole-blood viscosity, oxygen-carrying capacity, and RCM.

Hb is the major carrier transporting oxygen, carbon dioxide, Hb concentration is a directly measured value that is commonly used

**Management**

Treatment of anemia should be based on the physiology and etiology of anemia. Maintenance of normovolemia and cardiac output (CO) are necessary.

While transfusion of allogenic red blood cells is a rapid means of correcting anaemia, but transfusion itself is associated with increased perioperative mortality and morbidity.

Treatment of preoperative anaemia should be directed at correcting the underlying cause.

correct anaemia before surgery reduce perioperative transfusion rates, postoperative infectious and ischemic complications and length of hospital stay.
Iron deficiency anaemia. Iron can be replaced by the oral or parental route. Oral iron is prescribed as a 100–200 mg daily dose. I.V. iron is more effective

Vitamin deficiency anaemia: folate and vitamin B12 deficiencies should be corrected when identified in an Anaemic patient

Renal anaemia and anaemia of chronic disease: human erythropoietin is an established therapy for patients suffering from renal anaemia and anaemia related to chronic disease.

- Despite efforts to identify and correct preoperative anaemia, some patients will still present for surgery with low haemoglobin, particularly if they require urgent intervention. In these circumstances, steps should be taken to minimize blood loss, optimize tolerance to anaemia, and if necessary transfuse red cells. They should be monitored carefully for postoperative bleeding and complications such as ischemia

**Anesthetic management for anemic patient:**

**Preoperative**

1- Detection of anemia
   - History: (bleeding or co-morbid disease)
   - Investigations: - CBC, coagulation & iron studies where indicated

2- Management of preexisting anemia
3- If possible, delay surgery to optimize the condition
- Iron
- Vitamin B12/folate
- Erythropoietin stimulating agents
- Refer the patient to hematology clinic

**Intraoperative**

1-Optimize hemostasis
- Stop antithrombotic medications preoperatively where appropriate
- Topical hemostatic
- Maintain normothermia

2-Minimize blood loss
- Minimally invasive techniques
- The use of Tourniquet if possible

3-Maintain blood volume & hemoglobin concentration
- Replace losses by IV fluid (crystalloid, colloid)
  - avoid large volume crystalloid infusion
- Blood transfusion
  - Consider threshold of 90 g/L if:
    - significant ongoing bleeding

4-Preop autologous donation (usually discouraged) or
- Acute normovolemic hemodilution
Postoperative

- Prevention of new-onset (hospital-acquired) anemia (or exacerbation of existing anemia)
  - Monitor closely for postoperative bleeding

Sickle Cell Anaemia:

Goals of the perioperative management of patients with sickle cell disease are focused on clinical measures to avoid precipitating a vaso-occlusive crisis and include:

1- avoiding hypoxia

2- hypothermia, and

3- dehydration.

4-standard or exchange transfusions for sickle cell patients with the goal of reducing Hb S concentration to less than 30% to 40% can be helpful to reduce the incidence of a perioperative vaso-occlusive crisis.
Why is anemia bad for surgery?

Very severe anemia may cause organ damage and death. Patients who have anemia around the time of surgery have more complications from surgery including:

1- a higher risk of infection,
2- longer need for breathing assistance with a machine,
3- higher death rates than people who do not have anemia after surgery

Can an anemic person have surgery?

Don't proceed with elective surgery in patients with anemia until properly diagnosed and treated. Anemia is common, presenting in approximately 1/3rd of patients undergoing elective surgery

How is anemia treated before surgery?

Can anemia be corrected prior to surgery? In most situations, anemia can be corrected with intervention preoperatively. Interventions, including B12, IV Iron infusions, and growth factors can be employed to achieve a safe hemoglobin count prior to surgery
Red Cell Transfusion Guidelines

Recently updated guidelines from the American Society of Anesthesiology recommend transfusion if hemoglobin level is less than 6 g/dL and that transfusion is rarely necessary when the level is more than 10 g/dL.

What is the lowest hemoglobin level before death?

People also sometimes want to know how low can hemoglobin go before causing death. In general, a hemoglobin less than 6.5 gm/dL is considered life-threatening.
Renal impairment and anaesthesia

Function of kidney:

1- Excretory Function : excretion of waste product, drug, and toxin

2- Homeostatic Function;
   - Regulation of the body’s fluid balance
   - Regulation of the body’s Acid-Base
   - Regulation of electrolyte balance

3- Endocrine Function:
   - Renin secretion ---- which regulates blood pressure
Erythropoietin---- stimulates the production of RBC in the bone marrow
Prostaglandins---- various systemic effects
The kidneys are situated on the posterior abdominal wall, with the
diaphragm and 11\textsuperscript{th} and 12\textsuperscript{th} ribs posteriorly. They are about 10cm long,
5cm wide and 3cm thick

**Renal impairment**: can be either acute or chronic.

**Acute Kidney Injury (AKI)**:
Acute renal failure is characterized by the sudden and often reversible
deterioration of renal functions over a period of hours to few days or
weeks, resulting in failure of the kidneys to excrete nitrogenous waste
products and to maintain fluid, electrolytes and acid-base homeostasis.

**Chronic Renal Failure**  Chronic Kidney Disease (CKD) refers to an
irreversible and progressive deterioration in renal function which
develops over months to years. It causes a multi-systemic dysfunction.

**Aetiology of CKD**
- Hypertension
- Diabetes Mellitus
- Glomerulonephritis, SLE, HIV, Sickle Cell Disease
**Surgery:**

Operations on the kidney include pyeloplasty (to overcome obstruction at the pelviureteric junction) excision of cysts, open nephrostomy, pyelolithotomy, nephrolithotomy and partial or total nephrectomy.

Renal stones may be broken into small pieces using lithotripsy.

Open operations are usually performed in the full lateral position with either padding or table positioning ("breaking" the table in the middle) used to raise the loin and improve surgical access. The lumbar incision is the most common approach.

A thoracoabdominal incision using any intercostals space from the eight to the eleventh gives excellent exposure for large renal masses.

A trans abdominal approach may also be used with the patient supine.

**Investigation:**

1. Urinalysis (GUE): is inexpensive and informative laboratory tests. Haematuria and the presence of casts, bacteria, white cells may be found on microscopy. Urinary specific gravity is an index of renal tubular function. The ability to excrete concentrated urine (specific gravity $>1.030$) indicates good tubular function,

   Proteinuria indicates glomerular damage.

   Glycosuria usually indicates the presence of diabetes mellitus.
2- A full blood count may reveal anaemia either due to excessive hematuria, or because of reduced production of erythropoietin by the failing kidney.

3- Plasma creatinine and urea concentrations provide good information about general kidney function. Creatinine clearance can also be used to measure glomerular filtration rate (GFR). clearance decreases with age.

4- If impaired renal function is suspected, serum electrolyte concentrations should be measured.

5- In severe renal failure, determination of arterial blood gases may reveal a metabolic acidosis due to impaired acid excretion by the kidney.

6- Other tests such as chest X-ray and ECG may be needed depending on the patient’s symptoms, and on any other co-morbidities.

7- All patients undergoing open or laparoscopic renal surgery should have blood group and cross matching because of the risk of haemorrhage intra-operatively

**Pre-Operative Management**

The patient’s condition should be optimized as far as possible prior to surgery.
1- Hypertension should be well controlled with appropriate medication. Any urinary tract infection should be treated.

2- Anemia should be corrected. For elective surgery, preoperative iron or erythropoietin therapy may be used to increase haemoglobin levels.

3- Patients with severe renal failure may have fluid and electrolyte disturbances. These should be corrected and dialysis may be used.

4- Diabetes mellitus is a common cause of renal problems, and an appropriate plan should be made for the management of such patients in the perioperative period.

5- Premedication may be used as necessary, and antacid prophylaxis should be considered in those with chronic renal failure.

**Pre Medication:**
Reduced doses of an opioid or Benzodiazepine, and H2 blocker

Aspiration prophylaxis: Metoclopramide -10 mg for accelerating gastric emptying, prevent vomiting, ↓risk of aspiration,

Antihypertensive agents should be continued until the time of surgery.

**Monitoring:**
All routine monitoring – ECG, NIBP, SpO₂, EtCO₂, • Monitoring urinary output and intravascular volume (desirable urinary output: 0.5 ml/kg/hr.) • Intra-arterial, central venous, pulmonary artery monitoring are often indicated.
Induction: The dose of induction agent should be carefully considered. Many patients will need a reduced dose. In Hypovolemia there is a diversion of blood to essential organs and across the blood brain barrier, therefore effects of induction agents may be exacerbated. Patients are at increased risk of aspiration: rapid-sequence induction with cricoid pressure should be done.

Effects of drugs in patients with reduced renal function

Some drugs are eliminated unchanged in the urine. In particular non-depolarizing muscle relaxants are largely excreted by the kidneys. However, when maintenance doses are used, these should be smaller than for patients with normal renal function and the interval between doses should be increased. Exceptions to this are atracurium and cisatracurium which are broken down by enzymatic hydrolysis (Hofmann elimination) to inactive products, and so are not dependent on renal excretion.

Succinylcholine is metabolized by pseudocholinesterase, and although levels of this enzyme are reduced in uremia, succinylcholine administration cause a rise in serum potassium, which may be dangerous in patients with severe renal impairment who already have an elevated potassium level.
Other drugs which are largely excreted unchanged in the urine include atropine and Glycopyrrolate, however a single dose will not cause clinical difficulties.

Drugs which are extensively bound to albumin, such as many induction agents, will be affected by the reduction in albumin levels in uremic patients. This results in an increase in the free fraction of the drug, and a reduction in the dose required to produce anaesthesia.

Inhaled anaesthetic agents are preferred for the maintenance of anaesthesia because their excretion is via the respiratory system, and so impaired kidney function will not alter the response to these agents.

Opioids are extensively metabolized in the liver, and therefore unaltered by renal disease. However, morphine and Pethidine, both have active metabolites which are excreted by the kidney and may accumulate in renal failure. Doses of these two drugs should therefore be reduced or limited.

**Post-Operative**:

1- Monitoring of fluid overload or Hypovolemia •
2- Monitoring of urea and electrolytes,
3- ECG monitoring for detecting cardiac dysrhythmias.
4- Continue oxygen supplementation in post-operative period
5- Analgesia with regional analgesia
6- Carefully titrated opioids, ↑CNS depression, respiratory depression – give naloxone.
7- postoperative cardiac assessment should be performed and continued for 3-5 days with daily ECGs and screening of cardiac enzyme levels to detect and treat possible perioperative MI.

**Local Anaesthesia** : LA infiltration of the surgical field by the surgeon is the most physiologically stable of the anaesthetic techniques, and is therefore used in patients with severe co-morbidity.

**Regional Anaesthesia** : This offers many advantages over other techniques, including intraoperative hemodynamic stability and good postoperative analgesia. Adequacy of coagulation should be considered and the presence of uremic neuropathies excluded before regional anesthesia is performed in these patients.

Regional anesthesia should be avoided if there is any coagulopathy or thrombocytopenia, or recent hemodialysis with anticoagulation
Anesthesia and hypertension

Peri-operative Hypertension: It means Hypertension occurring in the pre-operative, intra-operative or post-operative period.

Hypertension is most common medical reason for postponing surgery. Peri-operative hypertensive are very common

The risk of chronic hypertension on perioperative is determined primarily by the presence of organ damage, that is, coronary artery disease, stroke, heart failure, and renal failure, all of which are known to affect perioperative morbidity and mortality.

Effect of anesthesia on blood pressure regulation:

Blood pressure is a compromise between cardiac output and systemic vascular tone. Blood pressure regulation depends therefore on heart rate (HR), left ventricle stroke volume (LVSV) and vascular resistance. The sympathetic nervous system is the main regulatory system, which is
affected by anesthesia. Fortunately, the backup systems, renin angiotensin system and vasopressin, can compensate the sympathetic nervous system impairment

Laryngoscopy & intubation are known causes of hypertension (Sympathetic activation)

**Classification of Hypertension**

Stages of hypertension described in the National Institute for Health and Care Excellence (NICE) guidelines:

<table>
<thead>
<tr>
<th>Stage</th>
<th>Clinic blood pressure (mmHg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>140/90–159/99</td>
</tr>
<tr>
<td>2</td>
<td>160/100–179/119</td>
</tr>
<tr>
<td>3</td>
<td>Systolic &gt;180 or diastolic &gt;120</td>
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Most antihypertensive drugs should be continued to the day of surgery and restart as soon as possible (when the patient will be able to swallow). Only agents that affect the RAS (Renin-Angiotensin) should be cancelled, e.g., ACEIs

**Preoperative assessment:**

When assessing patients for anaesthesia who have elevated blood pressure, a number of questions must be answered.

1. Is the patient known to be hypertensive on a previous occasion?
2. Are they on antihypertensive medication?

3. Does the patient have a treatable cause for their hypertension?

4. Does the patient have “white coat” hypertension?

5. Does the blood pressure control need alteration before surgery?

    Untreated, severe hypertension can lead to serious post-operative complications for example myocardial infarction, cerebral haemorrhage, infarction and renal failure.

    When assessing a patient for anaesthesia, ask about related illnesses such as ischemic heart disease, renal failure and cerebrovascular disease. This can assess the extent of hypertensive end organ damage, and therefore risk for anaesthesia.

    Patients with hypertension are often asymptomatic, and preoperative assessment with routine blood pressure measurement, is often the first manifestation of any potential problem.

    A series of blood pressure measurements, taken with the correct size cuff.

    A number of preoperative blood pressure readings may be taken over 2 – 3 hours with the patient resting. Frequently blood pressure reduces over this time, indicating underlying anxiety as a likely cause.

    Those patients with “white coat” hypertension have not been shown to be at higher risk from anaesthesia than controls and therefore surgery should not be delayed. Elderly patients with systolic blood pressures below 180 mmHg should also be considered for surgery, particularly if there is little evidence of end organ damage, as these values are considered in the normal range for elderly patients due to normal physiological change.
Investigation

1- ECG
2- electrolyte measurement.
3- chest radiograph should be performed,
4- If significant cardiac disease is suspected an exercise tolerance test.

Patients with uncontrolled hypertension requiring urgent surgery may benefit from regional anaesthetic techniques to avoid the risks of general anaesthesia

Conduct Of Anaesthesia

General principles

Cardiovascular stability is important during anaesthesia. Hypertensive patients are at risk of greater swings of blood pressure than the normal population and it has been shown that blood pressure liability can be associated with increased cardiovascular morbidity and mortality postoperatively, particularly in patients with severe uncontrolled hypertension. Optimization of such patients with investigation and drug treatment can improve long term outcome and prevent such complications.

Patients who have hypertension require a higher blood pressure for adequate organ perfusion than normotensive patients – this is particularly in the elderly. Avoidance of hypotension may prevent complications of under-perfusion.

Induction
Give a small dose of intravenous sedation (midazolam 1 – 3mg).

Abrupt and marked reductions in blood pressure may occur on induction of anaesthesia, and large increases during stimulating procedures such as laryngoscopy, intubation and pain. It is possible to augment induction with other pharmacological agents. Short acting opioids such as fentanyl 1mcg/kg or alfentanil 10mcg/kg given in conjunction with induction agents will diminish stimulatory effects.

Intraoperative blood pressure monitoring may be non-invasive or invasive. Standard non-invasive blood pressure monitoring is mandatory for anaesthesia, with a minimal interval of five minutes. High risk patients undergoing major surgery can, in addition, have intra-arterial monitoring to improve control and allow early pharmacological intervention. These measures are used in conjunction with continuous monitoring of the ECG and oxygen saturation.

**Maintenance**

Patients with underlying organ dysfunction need careful consideration of anesthetic agents. For example underlying renal failure, will alter metabolism and excretion of drugs. Patient with recent myocardial infarction may necessitate surgical delay.

Effective opioid analgesia, regional anaesthetic techniques and avoiding hypoxia, hypercarbia and light anaesthesia all aid to reduce episodes of hypertension intraoperatively. Vasopressor agents and intravenous fluids may be required if hypotension results from cardiac depression.

Spinals or epidurals may increase intraoperative hypotension, particularly in
hypertensive patients who are dehydrated or those receiving vasodilator drugs.

**Recovery**

Monitoring should include ECG, blood pressure and SpO₂. Coughing during recovery from anaesthesia may produce hypertension, and preoperative lignocaine spray on the endotracheal tube or deep extubation may improve this. Patients may develop hypertension due to pain, bladder distension or anxiety. It is also important to remember some patients will be confused and disorientated after anaesthesia and this may worsen blood pressure values.

After reversible causes have been treated (pain, urine retention) treatment of continuing hypertension may be required.

**Antihypertensive drugs and anaesthesia**

Elective surgical cases are usually given their regular antihypertensive medications on the morning of surgery.

Remember that anesthetic agents also cause vasodilation and cardiac depression, and the effects may be cumulative. Analgesic agents will reduce anaesthetic requirement and allow a smoother induction. Anaesthetic agents such as ketamine should be avoided due to their cardiovascular stimulatory properties.

**Drug action and side effects**

- Beta blockers improve perioperative morbidity and mortality in patients with ischemic heart disease,
• Beta blockers are avoided in patients with asthma, and may also prevent changes in the patient’s physiology to adverse events such as tachycardia which may be due to pain, reduced cardiac output, awareness, anaphylaxis

• ACE inhibitors will usually be continued prior to anesthesia, but cessation should be considered if marked blood loss is anticipated or regional techniques such as epidurals are carried out, as the cumulative hypotensive effects may be detrimental. Renal function should be carefully monitored in those patients taking ACE inhibitors, particularly in the perioperative period.

• Non-steroidal anti-inflammatories should be used with caution in patients taking ACE or A2 inhibitors.

**Intraoperative hypertension** may have many causes, reversible causes should be excluded and anaesthesia deepened with analgesics or anaesthetic agents before using antihypertensive in the acute setting. Labetalol,( a combined alpha and beta blocker with a short onset time) may be titrated intravenously in patients who require supplementary treatment

**Postoperative care**

Patients must be followed up after anaesthesia to ensure post-operative complications are minimized and treated appropriately if they occur. It is important to ensure patients continue their antihypertensive after surgery and alternative routes of administration are considered for those patients who are nil by mouth. Patients with severe hypertension must be closely monitored
High risk patients should also be given supplementary oxygen to prevent hypoxia and therefore reduce ischemic episodes after anaesthesia.
Liver disease and anesthesia

The largest organ in the body is the liver.

Surgery and anesthesia affect hepatic function primarily due to their effect on hepatic blood flow and as a result of the medications or anesthetic technique

**Functions of the liver**: which include

1. metabolism of carbohydrates and fats
2. protein synthesis and metabolism
3. drug metabolism
4. Digestion: excretion of bilirubin.

**Important facts**
albumin can be decreased with liver disease

colloid osmotic pressure will be reduced

fewer binding sites for drugs and the unbound,(active portion of protein-bound drugs) will be increased example Thiopental.

**Causes of liver disease**

**Chronic**

The most common causes of chronic liver disease are viral hepatitis (hepatitis B and C), autoimmune disease, and alcoholic liver disease. other known factors are cholestatic conditions (primary biliary cirrhosis and sclerosing cholangitis), drugs, toxins, and metabolic disease

**Acute**

drugs (paracetamol) • viral hepatitis • idiopathic

**Extra hepatic manifestations of liver disease**

**Gastrointestinal**

Portal hypertension is associated with the development of a collateral venous circulation, ascites, and splenomegaly. The presence of gastric and esophageal varices can result in catastrophic gastrointestinal haemorrhage.

ascites can raise intra-abdominal pressure with adverse effects on respiratory and renal function. Gastric emptying is also delayed and patients are therefore at increased risk of acid aspiration syndrome, necessitating protection with H2 antagonists and cricoid pressure.

**Cardiovascular system**

The circulation in patients with advanced liver disease is characteristically hyper dynamic with a high cardiac output and low systemic vascular
Patients with advanced liver disease may have risk factors for coronary artery disease. These may become apparent during anaesthesia or surgery.

**Respiratory**

The presence of pleural effusions restricts alveolar ventilation, reduces FRC, and predisposes to atelectasis and hypoxia. Associated gastro-oesophageal reflux disease, and massive ascites may increase the risk of aspiration of gastric contents.

**Haematological**

Anaemia may be present secondary to chronic blood loss from the gastrointestinal tract, hypersplenism-induced haemolysis, chronic illness, and malnutrition. Reduced capacity to synthesize clotting factors results in coagulopathy, particularly affecting the vitamin K-dependent factors II, VII, IX, and X,

**Renal and metabolic**

Secondary hyperaldosteronism leads to water retention and hypernatremia resulting in the formation of ascites and peripheral edema. Loop diuretics used to treat the ascites and oedema can cause relative hypovolaemia and hypokalaemia. Conversely, the aldosterone antagonist spironolactone can cause hyperkalemia. Vasodilatation associated with general anesthesia may result in renal hypoperfusion and the development of pre-renal failure.

Depletion of hepatic and muscle glycogen stores may result in perioperative hypoglycemia. Muscle wasting is common due to impaired protein synthesis and malnutrition.

**Central nervous system**

The development of hepatic encephalopathy in patients with chronic liver disease can be precipitated by infection, gastrointestinal haemorrhage,
electrolyte or acid-base disturbance, sedative drugs, hypoglycaemia, hypoxia, hypotension, or excessive dietary intake of protein. (reflect cerebral oedema with raised intracranial pressure

Relevant pharmacology

I.V. anesthetic agents

The dose of thiopental should be reduced because a reduction in plasma proteins results in an increased unbound fraction of drug

Sensitivity to the sedative and cardiorespiratory depressant effects of propofol is increased; hence the dose should be reduced.

Etomidate may be used safely but offers little advantage over thiopental. Chronic alcohol use may increase anesthetic requirements, but all i.v. agents should be used with great care.

Neuromuscular blocking drugs

The metabolism of succinylcholine may be slowed because of reduced pseudo cholinesterase concentrations.

There is an apparent resistance to non-depolarizing neuromuscular blockers (NMBs) in patients with liver disease, which may be due to an increased volume of distribution or to altered protein binding. Vecuronium and rocuronium, have a prolonged elimination phase in severe liver disease.

Atracurium and cisatracurium are suitable NMBs as they do not rely on hepatic excretion

Opioids

Elimination of morphine is delayed in cirrhotic. Morphine is perhaps best avoided in patients with liver failure as it may precipitate hepatic encephalopathy.
Fentanyl, given in low doses, is suitable for intraoperative use as it does not have an active metabolite and is renally excreted.

Remifentanil is ideally suited to intraoperative use as it is metabolized by tissue and red cell esterases.

**Volatile anesthetics**

All volatile anesthetics reduce cardiac output and mean arterial pressure and thereby reduce liver blood flow.

Isoflurane, Sevoflurane, and Desflurane undergo minimal hepatic metabolism and can be regarded as safe. Desflurane is probably the ideal volatile agent, being the least metabolized and providing the quickest emergence from anesthesia.

**Other drugs**

Tramadol - said to be safe with mild hepatic impairment

Paracetamol if severe liver failure – should be avoid

**Preoperative evaluation and optimization**

Preoperative evaluation of patients with liver disease should focus on the extent of liver dysfunction and effects on other organ systems. Viral hepatitis presents a risk to operating theatre personnel and a diagnosis of hepatitis B or C should be ascertained. Patients with hepatitis of unknown aetiology should be considered infectious.

**History and examination**

Patients with compensated liver disease may be asymptomatic or have only vague symptoms such as malaise, weight loss, or dyspepsia. Physical signs may be absent or non-specific. A full physical examination should be performed with particular reference to the presence of muscle wasting, spider naevi, pleural effusions, ascites, splenomegaly, and level of
encephalopathy

Preoperative evaluation • History (dyspnea, bleeding, presence of esophageal varices, encephalopathy) • Physical examination (orientation, saturation, pleural effusion, pulse, blood pressure, ascites, bruising, malnutrition)

Investigations

1- Full blood count will detect anaemia, thrombocytopenia, or raised white cell count if infection is present.

2- Prothrombin time (PT) is a useful indicator of hepatocellular function and is used as a prognostic indicator in acute liver failure and after surgery in patients with chronic liver disease. Where possible, vitamin K should be administered for several days before operation.

3- renal function (creatinine, urea, GFR)

4- liver function is best assessed by PT, but albumin levels are also useful.

5- Cardiac investigations should include ECG and also echocardiography

6- Chest X-ray or ultrasound may be useful for demonstrating pleural effusions in need of drainage before operation.

7- Lung function tests to detect any restrictive or obstructive pulmonary disease.

Conduct of anaesthesia

Elective surgery should only be considered in patients who have well-compensated chronic liver disease. For patients needing emergency surgery, urgent optimization of the patient is mandatory and should include attention to intra-vascular volume status, coagulation function, and also
neurological assessment and screening for infection.

Sedative premedication should be avoided as it may precipitate encephalopathy; however, premedication with an H$_2$ receptor antagonist such as ranitidine is advisable. The goals of intraoperative management should be maintenance of adequate hepatic blood flow and oxygen delivery

**Intraoperative considerations**

All patients should receive standard monitoring but for major surgery, invasive monitoring of both arterial and central venous pressure is recommended

1- ECG,

2- pulse oximetry,

3- end-tidal CO$_2$,

4- peripheral nerve stimulator,

5- temperature, urine output

6- invasive BP • large-bore i.v. access • CVC, consider PA-catheter •

**Anesthetic complications due to severe liver disease:**

1- aspiration (ascites, delayed gastric emptying)

2- hypoxemia (ascites, pleural effusion)

3- hypotension (hypovolemia)

4- bleeding (oesophageal varices, coagulopathy) •

5- hypoglycemia

The choice of drugs for anaesthesia induction and maintenance is less important than the care with which they are used. A suggested technique is
i.v. induction of anaesthesia using propofol and Remifentanil, in most cases using a modified rapid sequence induction with cricoid pressure and rocuronium 1 mg/ kg followed by maintenance with oxygen/air/Desflurane and Remifentanil infusion.

Target-controlled infusion of propofol is an alternative to inhalation anaesthesia and is useful for gastrointestinal endoscopic and radiological procedures. Atracurium is preferred for maintenance of neuromuscular block.

Antibiotic prophylaxis is required before surgery.

Large-bore i.v. access is mandatory. All fluids should be administered via a fluid warming device.

**Postoperative pain management**

I.V. patient-controlled analgesia using fentanyl (or occasionally morphine). Regional analgesia may be very useful in reducing the need for systemic analgesia.

NSAIDs are not recommended because of the risk of gastrointestinal haemorrhage.

Acetaminophen (paracetamol) is better to be avoided.

**Note:**

An INR test measures the time for the blood to clot. It is also known as prothrombin time, or PT. It is used to monitor blood-thinning medicines, which are also known as anticoagulants. The INR, or international normalized ratio, can also be used to check if you have a blood clotting problem.

What is a normal INR?
In healthy people an INR of 1.1 or below is considered normal. An INR range of 2.0 to 3.0 is generally an effective therapeutic range for people taking warfarin for disorders such as atrial fibrillation or a blood clot in the leg or lung

What happens when INR is high?

The higher your PT or INR, the longer your blood takes to clot. An elevated PT or INR means your blood is taking longer to clot than your healthcare provider believes is healthy for you. When your PT or INR is too high, you have an increased risk of bleeding
Massive blood transfusion

Massive transfusion or large volume transfusion is defined as the replacement of blood loss equivalent to or greater than the patient’s total blood volume in less than 24 hours: (70 mL/kg in adults, 80-90 mL/kg in children or infants). Morbidity and mortality tend to be high among such patients, not because of the large volumes infused, but because of the initial trauma and the tissue and organ damage secondary to haemorrhage and hypovolaemia.

**Massive transfusion occurs during severe bleeding due to**

1. Trauma
2. Ruptured aortic aneurysm
3. Surgery
4. Obstetrics complications.
The goals to the management of massive transfusion include:

1- early recognition of blood loss

2- maintenance of tissue perfusion and oxygenation by restoration of blood volume and hemoglobin (Hb)

3- arrest of bleeding including with early surgical

4- use of blood component therapy to manage coagulopathy.

Indications

The primary indication for massive transfusion is any situation resulting in acute blood loss and hemodynamic instability. due to trauma, obstetrical catastrophes, surgery, and gastrointestinal bleeding.

Contraindications

There are no absolute contraindications for massive transfusion.

Equipment

Access is necessary to deliver intravenous blood products adequately. Blood products can be delivered through peripheral intravenous (IV) catheters, central IV catheters, or through intraosseous (IO) catheters.

In most patients receiving a massive transfusion, we would like to give blood products quickly. Therefore, catheters larger in diameter and shorter in length will give us higher flow rates and are more desirable. Large-bore IV catheters (14 to 18
(g), central IV catheters, and intraosseous should be inserted into the patient as necessary.

Other equipment such as high-speed transfusion devices and blood warming devices should also be collected.

Send frequent laboratory testing for hemoglobin levels, ABGs, coagulation, electrolyte, lactate, and thromboelastogram (TEGs).

There should also be monitors available for continuous reassessments of temperature, pulse oximetry, blood pressure, and heart rate.

**Preparation**

preparing for a massive transfusion should ensure that the patient is hooked to a monitor and there is adequate intravenous access to deliver the blood products when they become available. Notifying the blood bank will also help avoid delays in getting further deliveries of blood products if necessary.

**Complications**

Potential complications of massive transfusion include metabolic alkalosis, hypocalcaemia, hypothermia, and hyperkalemia. Non-fatal complications have been seen in more than 50% of patients when more than 5 units of blood products are transfused.

1- Metabolic alkalosis and hypocalcemia result from sodium citrate and citric acid that is added to blood products in storage to prevent coagulation. Each unit of blood can generate a total of 23 mEq of bicarbonate as citrate is metabolized. This can result in a metabolic alkalosis if the kidneys are unable to excrete the excess
bicarbonate. Citrate also binds ionized calcium, which can lead to significant free hypocalcemia.

2- Hypothermia can also result from the infusion of blood products. Blood products are stored at 4 C. Rapid infusion of cold blood can lead to lower core body temperatures

3- Hyperkalemia is also a possible complication as potassium can increase in blood during long-term storage. It is typically only seen when blood products have been stored for long periods and are infused through central access at high speeds

4- depletion of fibrinogen and coagulation factors: Plasma undergoes progressive loss of coagulation factors during storage, particularly factors V and VIII, unless stored at -25 degree Celsius or colder

5- Depletion Of Platelets Platelet function is rapidly lost during the storage of whole blood and there is virtually no platelet function after 24 hrs.

6- Disseminated Intravascular Coagulation(DIC): DIC is the abnormal activation of the coagulation and fibrinolytic systems, resulting in the consumption of coagulation factors and platelets

7- Microaggregates: White cells and platelets can aggregate together in stored whole blood, forming microaggregates. During transfusion, particularly a massive transfusion, these microaggregates cause embolism to the lung and their presence has been implicated in the development of Adult Respiratory Distress Syndrome(ARDS).

8- Adverse Effects Of Blood Transfusion: Acute transfusion reactions occur during or shortly after(within 24 hrs.) the transfusion.
Local anesthetics (LA)

LA are agents which produce reversible block of nerve conduction without any structural damage to the neuron and without any loss of consciousness. • local anesthetics are used to block all sensation in the part supplied by the nerve.

Classification :

A- classification according to chemistry

1- Ester Type :- eg. Cocaine, Tetracaine, Procaine, Benzocaine Etc.
2- Amide Type :- eg. Lidocaine, Bupivacaine, Prilocaine, Dibucaine etc.

B- according to duration of action:

a- short duration of action procaine
b- medium duration of action cocaine, Lidocaine, mepivacaine, Prilocaine
c- long duration of action Tetracaine, bupivacaine, ropivacaine

C-classification according to clinical uses

1- surface anesthesia
2- infiltration anesthesia
3- field block anesthesia \(\rightarrow\) lignocaine \(\rightarrow\) procaine \(\rightarrow\) bupivacaine \(\rightarrow\) Tetracaine \(\rightarrow\) cocaine \(\rightarrow\) benzocaine
4- nerve block anesthesia \(\rightarrow\) procaine \(\rightarrow\) lignocaine \(\rightarrow\) bupivacaine \(\rightarrow\) Tetracaine \(\rightarrow\) ropivacaine
5- spinal anesthesia \(\rightarrow\) lignocaine \(\rightarrow\) Tetracaine \(\rightarrow\) bupivacaine
6- epidural anesthesia \(\rightarrow\) Lignocaine \(\rightarrow\)

Mechanism Of Action :
Sensory information passes along nerve fibers via electrical impulse, or action potential. When nerve is at rest, the interior has a negative charge. An action
potential is generated by the influx of Na ions into the interior of the nerve, giving it a positive charge. Depolarization is the nerve fiber is returned to its resting potential by efflux of k ions - repolarization.

The action potential is then generated along the axon by successive depolarization & repolarization of adjacent regions.

The primary mechanism of action of local anesthetics is blockade of voltage-gated sodium channels. This reduces the permeability of cell membrane to Na ions, so action potential is not generated.

**Pharmacokinetics:**

**Absorption:** Systemic absorption of injected local anesthetic from the site of administration is determined by several factors, including dosage, site of injection, drug-tissue binding, local blood flow. Application of a local anesthetic to a highly vascular area such as the tracheal mucosa or the tissue surrounding intercostal nerves results in more rapid absorption.

**Metabolism And Excretion:**

The local anesthetics are converted in the liver (amide type) or in plasma (ester type) to more water-soluble metabolites and then excreted in the urine. Ester type LA is metabolized by pseudocholinesterase and amide type by hepatic microsomal enzymes and enzyme amidase.

**Side Effects** of LA:

1. Cardiovascular: depression of heart, bradycardia, hypotension, cardiac arrhythmias etc.
2. CNS: rapid absorption produce restlessness, tremor, convulsions.
3. Anaphylactic reaction - common with ester type. -causes asthma, dermatitis, skin rash etc.
4. Corneal change - very rarely reversible corneal change may occur.
Commonly used drug with LA:
Adrenaline – to prolong the effect of LA and to reduce the toxicity by reducing absorption from local area.

**Example of Local anesthetic drugs**

**Tetracaine**: Topical anaesthetic [0.5%] in ophthalmic practice. Onset of action is 30 minutes. Side effects – stinging sensation, drug allergy.

**Cocaine**: First local anaesthetic obtained from leaves of plant Coca. It is no more in use nowadays because of its corneal toxicity, addicting nature etc.

**Procaine**: First synthetic local anaesthetic. • Used as a small area infiltration and spinal anaesthetic. • It is poorly absorbed from mucous membrane - so no topical use. • 2% injection is the usual preparation.

**Lignocaine**: Most commonly used LA. In ophthalmology 4% [topical] and 2% [infiltration] solutions are commonly used. It has quick onset of action and high degree of penetration. The drug is recommended for topical, nerve block, infiltration and epidural injection and for dental analgesia. It may cause drowsiness.

**Bupivacaine**: Onset of action is slow. • A 0.75% solution produce anaesthesia for 8-12 hours.
Aspiration During Anesthesia (Mendelsohn’s syndrome)

Mendelsohn's syndrome, named in 1946 for American obstetrician Lester Mendelson. It is a form of chemical pneumonitis or aspiration pneumonitis caused by aspiration of stomach contents (principally gastric acid) during anaesthesia into the airway below the level of the true vocal cords.

**Presentation:**

Mendelson's syndrome is characterized by a bronchopulmonary reaction following aspiration of gastric contents during general anaesthesia due to abolition of the laryngeal reflexes.

The main clinical features are signs of general hypoxia, two to five hours after anaesthesia. Such features may include cyanosis, dyspnea, fever, pulmonary wheeze, crepitation, rhonchi, and tachycardia with a low blood pressure. Decreased arterial oxygen tension is also likely to be evident. Pulmonary edema can cause sudden death or death may occur later from pulmonary complications.

**Risk factors**

Patients with a high risk should have a rapid sequence induction. **High risk factors includes:**

1. Emergency surgical procedure
2. Light anaesthesia
3. Gastrointestinal reflux
4. Obesity
5. Opioid medication
6. Neurological disease, impaired conscious level, or sedation
7. Lithotomy position
8. Difficult intubation/airway
9. Hiatal hernia

**Prevention:**

A. **Preoperative fasting**

B. **Reducing gastric acidity:** Histamine (H-2) antagonists and proton pump inhibitors (PPIs)

1. An oral H2 antagonist (ranitidine 150-300mg PO) must be given night before and 1-2 hours before anaesthesia and a PPI, (omeprazole 40mg before the night and 2hr preop.)

2. Metoclopramide has a prokinetic effect promoting gastric emptying 10mg/PO 15min before surgery

C. **Rapid Sequence Induction (RSI) and adequate depth of anaesthesia** is important to avoid coughing, laryngospasm and vomiting

Cricoid pressure (sellick): Aim is to compress the esophagus between the cricoid ring cartilage and the sixth cervical vertebral body thus preventing reflux of gastric contents

D. **Nasogastric tube placement**

E. **Airway device:** A cuffed ETT is considered the gold standard device used for airway protection. Alternative supraglottic devices include the classic laryngeal airway (LMA)
Pheochromocytomas:

are endocrine tumors that originate from the adrenal medulla. These tumors secrete endogenous catecholamine’s that cause significant hemodynamic effects. If left untreated, these tumors can cause chronic, permanent damage to organs and possibly lead to death.

**symptoms** include headache, heart palpitations and diaphoresis (excessive sweating)

Symptoms are managed with alpha-adrenergic blockers until surgical intervention can be performed. Surgery is the only curative treatment.

**What is the best test for pheochromocytoma?**

Plasma free metanephrines constitute the best test for excluding or confirming pheochromocytoma and should be the test of first choice for diagnosis of the tumor. A negative test result excludes pheochromocytoma

Increased sympathetic activation during induction of anesthesia, tracheal intubation, and surgical incision, and increase catecholamine release during manipulation of the tumor can precipitate severe hemodynamic instability. Intraoperative complications include malignant hypertension, arrhythmia, myocardial infarction, pulmonary edema, and stroke.

**preoperative preparation:**

All patients with pheochromocytoma should receive appropriate preoperative medical management to block the effects of released catecholamine. The main goals of preoperative preparation:
1) normalize blood pressure, heart rate and functions of other organs
2) restore volume depletion
3) prevent patient from surgery induced catecholamine storms

Careful preoperative preparation with α- and β-blockers and oral intravascular fluid expansion have been shown to decrease the incidence and severity of perioperative hemodynamic instability

The most important consideration is to always block the alpha-adrenergic receptors before you block the beta adrenergic receptors. Failure to do this will result in extensive vasoconstriction, leading to life-threatening hypertension or heart failure.

After removal of the tumor, routine check-ups should be performed to prevent reoccurrence

1) α adrenoceptor antagonists:
   a) phenoxybenzamine (- initial dose 10 mg bd/day )
   b) prazosin - should be given before sleeping as patient may develop severe postural hypotension

2) β adrenoceptor antagonists - used after adequate pretreatment with α adrenoceptor antagonists to avoid hypertensive crisis from unopposed α adrenoceptor overstimulation - inhibition of β2 adrenoceptor mediated vasodilatation leaving α adrenoceptor mediated vasoconstriction unopposed

atenolol: 12.5 -25 mg 2-3 times/day; metoprolol: 25- 50 mg 3-4 times/day;
propranolol
Preoperative tests

Preoperative tests should include 24-h urine catecholamine levels and cardiac evaluation with an electrocardiogram and an echocardiogram.

During anesthesia:

In addition to standard intraoperative monitors, all patients should have an arterial line placed prior to induction of anesthesia.

During the induction of anesthesia, a deep plane of anesthesia and paralysis should be achieved prior to tracheal intubation, to avoid excessive sympathetic stimulation.

Potent arterial and venous vasodilators should be readily available to treat hypertensive crises during anesthesia. Arrhythmias should be treated promptly with β-blockers and antiarrhythmic agents. Vasopressors are usually needed to treat hypotension after tumor resection.

Maintenance of general anesthesia is achieved with a volatile anesthetic, with the careful titration of narcotics. Isoflurane lowers peripheral vascular resistance and blood pressure and can be used.

The goal of fluid management is to achieve euvoemia. Fluid management should be guided by dynamic parameters of fluid responsiveness.

All patients should be recovered in an intensive care unit. Elderly patients are at higher risk for postoperative complications and prolonged hypotension after tumor resection.
Medications contraindicated in pheochromocytoma:

1- Drugs that increase sympathetic tone such as ketamine, ephedrine, pancuronium,
2- Metoclopramide should not be used in patients with pheochromocytoma
3- Histamine provoking drugs such as morphine and Atracurium should also be avoided
4- halothane contraindicated in pheochromocytoma. Halothane can induce arrhythmia potential
5- Desflurane (sympathetic stimulation) are not preferred in pheochromocytoma