Ministry of Higher Education and Scientific Research

College of Health and Medical Technology

Anaesthesia Techniques Department

Teaching package for anaesthesia techniques

Subject: anesthesia techniques, 3rd stage.

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LECTURE ONE (1)

Preoperative assessment and premedication

The pre-operative assessment is an opportunity to identify co-morbidities that may lead to patient complications due to anesthesia or surgical procedure, during the operative or post-operative periods**.** Patients scheduled for elective procedures will generally attend a pre-operative assessment 2-4 weeks before the date of their surgery.

Premedication is using of medications in order to prepare the patient for anesthesia and to help provide optimal conditions for surgery. Specific needs will depend on the individual patient and procedure.

Goals of preoperative assessment:

1) Doctor-patient relationship
2) Plan of Anesthetic Technique
3) Screen for and manage co-morbid disease.
4) To assess and minimize risks of anesthesia.
5) To identify need for specialized techniques.
6) To identify need for advanced post-op care.
7) Preoperative Preparation.
8) Perioperative risk determination.
9) Reduce patient anxiety.
10) To obtain informed consent.

Minimum preoperative visit components (according to ASA):

1) Medical, anesthesia and medication history.
2) Appropriate physical examination.
3) Review of diagnostic data (ECG, labs, x-rays).
4) Assessment of ASA physical status.
5) Formulation and discussion of anesthesia plan.

Note// ASA = American Society for Anesthesiologists.

The ASA physical classification:

ASA1: normal healthy patient.

ASA2: Mild systemic disease - no impact on daily life.

ASA3: Severe systemic disease - significant impact on daily life.

ASA4: Severe systemic disease that is a constant threat to life.

ASA5: Moribund, not expected to survive without the operation.

ASA6: Declared brain-dead patient - organ donor.

E: Emergency surgery.

ASA Classification	Definition	Examples
ASA I	A normal healthy patient	Healthy, non-smoking, no or minimal alcohol use
ASA II	A patient with mild systemic disease	Mild diseases only without substantive functional limitations. Current smoker, social alcohol drinker, pregnancy, obesity (30<BMI<40), well-controlled DM/HTN, mild lung disease
ASA III	A patient with severe systemic disease	Substantive functional limitations; One or more moderate to severe diseases. Poorly controlled DM or HTN, COPD, morbid obesity (BMI ≥40), active hepatitis, alcohol dependence or abuse, implanted pacemaker, moderate reduction of ejection fraction, ESRD undergoing regularly scheduled dialysis, history (>3 months) of MI, CVA, TIA, or CAD/stents.
ASA IV	A patient with severe systemic disease that is a constant threat to life	Recent (<3 months) MI, CVA, TIA or CAD/stents, ongoing cardiac ischemia or severe valve dysfunction, severe reduction of ejection fraction, shock, sepsis, DIC, ARD or ESRD not undergoing regularly scheduled dialysis
ASA V	A moribund patient who is not expected to survive without the operation	Ruptured abdominal/thoracic aneurysm, massive trauma, intracranial bleed with mass effect, ischemic bowel in the face of significant cardiac pathology or multiple organ/system dysfunction
ASA VI	A declared brain-dead patient whose organs are being removed for donor purposes	

History

1) Medical problems (current & past).

- DM, HTN,COPD,CAD,thyroid disorder..
- Regular medications
- Previous surgeries; date:
- 5.Family anesthesia history:
- Problems with anesthesia in family
- type of anesthesia:
- (Pseudocholinesterase deficiency and malignant hyperpyrexia)

2) Previous anesthesia & related problems.

- Allergy to drugs
- PONV
- Anesthesia awareness
- Difficult intubation
- Delayed emergence

3) Allergies and drug intolerances.
4) Medications, alcohol & tobacco.
5) Review of systems (include snoring and fatigue).
6) Exercise tolerance and physical activity level.

Physical examination

1) Airway.
2) Heart and lungs.
3) Vital signs including O_2 saturation (

- Blood pressure, Resting pulse, rate, rhythm,
- Respiration, rate, depth, and pattern at rest,
- Body temperature

4) Height and weight (BMI).
5) Other Specific examinations depending on the individual patient and procedure.

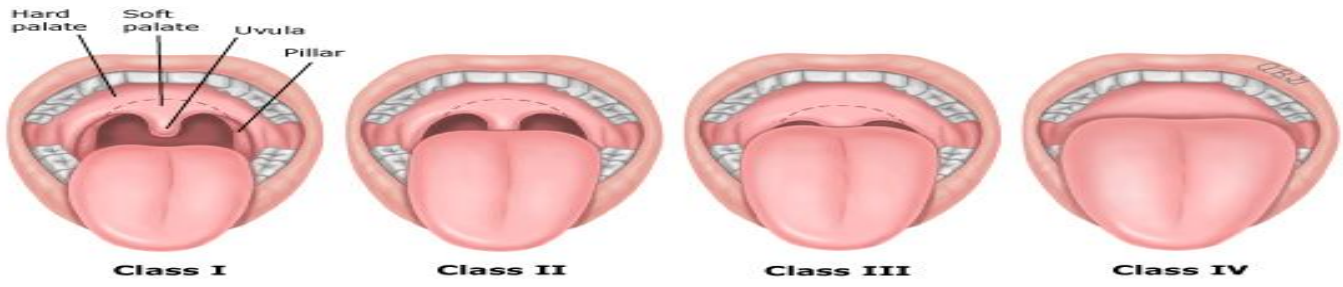
Airway Assessment

Predictors of difficult intubation

- Mallampati classification
- ULBT (upper lip bite test)
- Inter-incisors gap (IID)
- Thyromental distance (TMD)
- Forward movement of mandible
- Document loose or chipped teeth
- Tracheal deviation
- Movement of the Neck

Modified Mallampati score:

Used to predict the ease of endotracheal intubation, the score is assessed by asking the patient, in a sitting posture, to open his or her mouth and to protrude the tongue as much as possible.

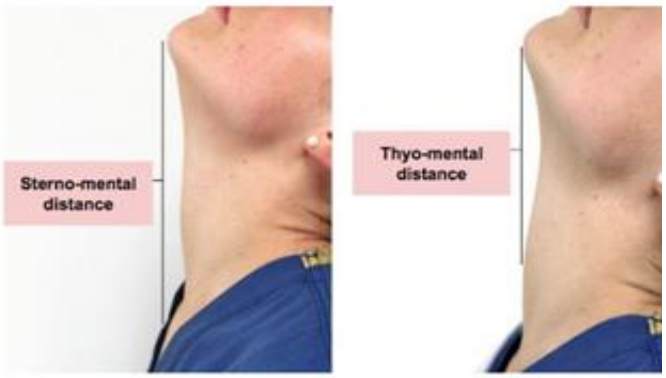


- **Class** I: Soft palate, uvula, fauces, pillars visible.
- **Class** II: Soft palate, major part of uvula, fauces visible.
- **Class** III: Soft palate, base of uvula visible.
- **Class** IV: Only hard palate visible.

A high Mallampati score (class 3 or 4) is associated with more difficult intubation as well as a higher incidence of sleep apnea.

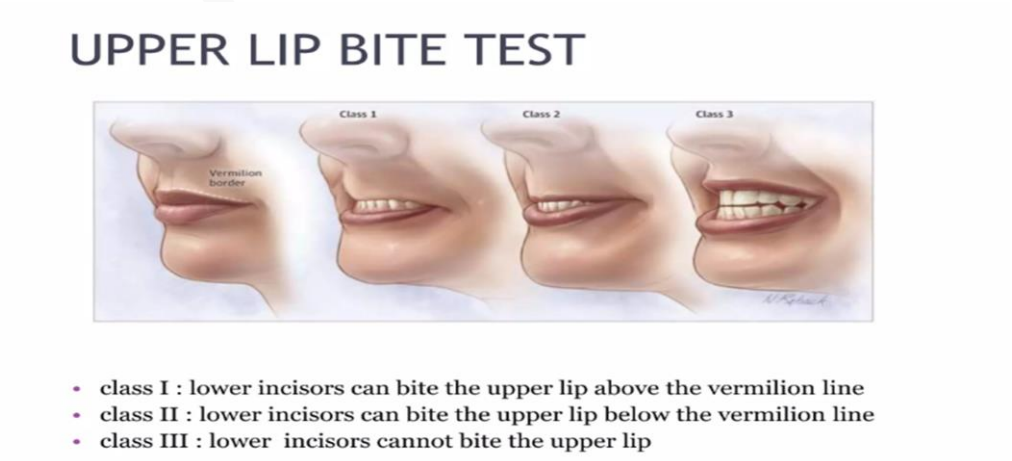
Thyromental distance (TMD)

Distance from the thyroid cartilage to the mental prominence when the neck is extended fully. Should be 7 cm

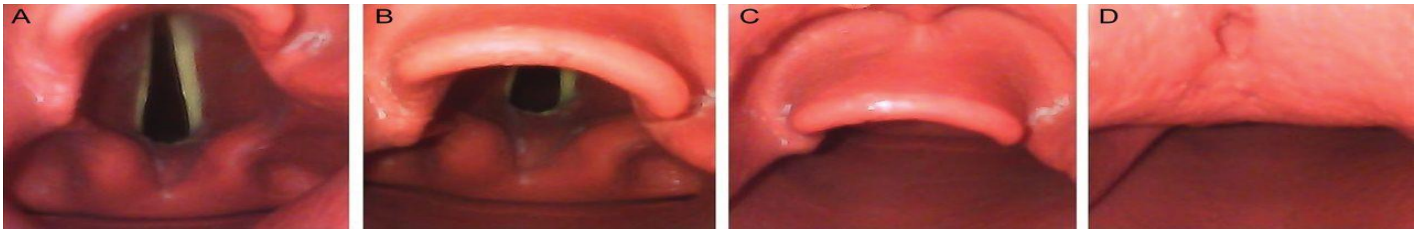


Sternomental distance (SMD)

Distance from the upper border of the manubrium sterni to the tip of the chin, with the mouth closed and the head fully extended. Should be > 12.5 cm



Laryngoscopy: Cormack and Lehane



Also Look for:

Body: obese? If female: large pendulous breast?

Neck anatomy: short? thick? webbed?

Mouth: limitations (opening)?

Teeth? (number & health) Enlarged tongue? (hypothyroidism, acromegaly & obesity)

Mandible (+TMJ): micrognathia,receding mandible (ask patient to sublux their lower incisor beyond upper incisor) Maxilla: protruding? (buck teeth) |

Face: beard? Facial trauma? |

Nose: nasal passage patency, Head size: Children (ex. hydrocephalus or rickets) | Adults (ex. acromegaly)

Cardiovascular system:

- Dysrhythmias
- Atrial fibrillation
- Heart failure
- Heart murmur
- Valvular heart disease
- Blood pressure is best measured at the end of the examination

Respiratory system

- cyanosis
- pattern of ventilation
- respiratory rate RR
- Dyspnoea
- Wheeziness
- signs of collapse
- consolidation and effusion

Pulmonary disease

Smoking

• Increased carboxyhemoglobin levels.

• Decrease ciliary function.

• Increase sputum production.

• Nicotine adverse effects on cardiovascular system.

• Preoperative advices:

❖ 2 days cessation can decreases nicotinic effect, improve mucus clearance and decrease carboxyhemoglobin levels

❖ 4-8 weeks of cessation are believed to be needed for postoperative complication reduction

Asthma

• Obtain information about irritating factors, severity and current disease status.

• Frequents use of bronchodilators, recurrent hospitalization and requirements for systemic steroids are all indicators of severe disease.

• Those who received more than a (burst and taper) of steroids in the previous 6 months should be considered for stress dose perioperatively.

Respiratory Tract Infection

• Patients presenting on the day of surgery with symptoms and signs of a lower respiratory tract infection should be treated appropriately and postponed to such time that they are symptom free.

• Viral upper respiratory tract infection can cause bronchial reactivity which may persist for 3-4 weeks.

• Unless surgery is urgent, such patients should be postponed for 4 weeks to minimize the risk of postoperative respiratory infection

INVESTIGATIONS

Basic Investigations

- **CBC: Hb,TC/DC,**
- **ABO Rh typing**
- **RBS**
- **Urea, Creatinine, Na+ ,K+**
- **PT/INR**
- **Urine RME**

. CXR

- **ECG**

Serology

Other (if needed)

Echocardiography

TFT

HbA1c

BT/CT, aPTT

- **LFT**

PFT

Ingested material	Minimum fasting hours
Clear liquid (water, clear tea, black coffee, fruit juice without pulp)	2
Breast milk	4
Formula milk, non huma milk, light meal	6
Regular or heavy meal	8

Prolonged fasting should be avoided as this is associated with dehydration, increased postoperative nausea and vomiting, electrolyte imbalance and patient distress.

Optimal fasting hours decreases volume and acidity of stomach contents and reduce aspiration and regurgitation risk.

Premedication:

Is the administration of medication before the induction of anesthesia.

The patient should enter the operation room:

- Free of apprehension
- Sedated
- Arousable
- Cooperative

and these characteristic can be achieved by the premedication drugs

The goal of the premedication was to:

- Relief anxiety
- Sedation
- Amnesia
- Analgesia
- Drying of the airways
- Preventing the autonomic reflex response
- Reduction in the gastric fluid volume and acidity
- Antiemetic activity
- Reduction of anesthesia requirement
- Facilitate smooth induction of anesthesia
- Prophylaxis against allergic reaction

The pharmacological premedication should be given either before surgery for 1- 2 hours (can be taken with small amount of water orally ; less than 30 ml) or at night before the operation

Anxiolytic:

These drugs used to decrease the anxiety of the patients and make him sedative and calm with amnesia (unable to remember)

- They act predominantly on GABA receptors
- Minimum cardiac and respiratory depression
- Do not produce nausea and vomiting
- Lack the analgesic effect
- Cross the placenta and may cause neonatal depression
- The main drugs that used are the benzodiazepines ; diazepam , midazolam , lorazepam ,alprazolam.

Analgesic:

They are groups of drugs that produces pain relief , the main group that used in premedication is the opioid analgesics which contains morphine , pethidine and fentanyl

- Can be given parentally
- Produce sedation
- Control elevated blood pressure during endotracheal intubation
- Fentanyl is preferred due to its rapid onset and short duration of action

Fentanyl:

- Potent analgesic 100 times more than morphine
- Metabolized in the liver and excreted in the urine
- Produce respiratory depression so use in caution with COPD
- Cause less nausea and vomiting
- Can be reversed by naloxone
- Dose 1-5 microgram / kilogram i.v

3. Anti-autonomic:

They contain either anticholinergic or beta1blocker

The anticholinergic drugs that used in the premedication are ; atropine ,hyoscine and Glycopyrrolate .

Atropine has vagal inhibition (tachycardia), CNS stimulation , Antisialagogues (decrease salivation) .

The dose of atropine is 0.3 – 0.4 mg iv

Hyoscine had less vagal inhibition effect with more effect on salivation but produce sedation and amnesia so should be avoided in elderly

Glycopyrrolate

- Has no central action, because it does not cross the blood brain barrier.
- Had longer duration and less tachycardia
- They produce dry mouth
- They produce mydriasis (dilation of eye pupil)

Atropine may cause central anticholinergic syndrome (restlessness, agitation, somnolence, convulsion) and this can be reversed by physostigmine

4. Antiemetic:

They are drugs used to decrease the incidence of nausea and vomiting , the most commonly used are :

- Ondansetron and Metoclopramide (plasil)
- The most common used antiemetic drug
- Dose 0.15 to 0.3 mg /kg and last for 12 hour
- Used in emergency anesthesia
- Act centrally as dopaminergic antagonist on the vomiting center in the medulla
- Act peripherally by increasing the rate of gastric emptying and increasing in the gut peristalsis
- May produce oculogyric (extrapyramidal) side effect

Chronic drug used by patients:

Drugs to be continued till the day of operation:

- antihypertensives except ARBs & ACE inhibitors (stop 24-36 hrs. preoperatively)
- Diuretics
- cardiac medications (beta blockers.digoxin, calcium channel blockers)
- Antidepressants
- Anxiolytics
- Thyroid medications
- Steroids & Statins
- psychiatric medications, birth control pills, eye drops
- heart burn & reflux medications
- asthma medications

Medicines with special attention

- aspirin :reversal of platelet inhibition within 3 days of stopping

do not discontinue in patients with drug eluting coronary stents until 12months of dual antiplatelet therapy completed

bare metal stents :continue for 1 month

- Thienopyridines (clopidogrel (Plavix),ticlopidine)

reversal of platelet inhibition in 7 days for clopidogrel ,14 days for Ticlopidine

for cataract sx:no need to stop

for stents,same as aspirin

- Oral hypoglycemics: discontinue on day of sx
- Diuretics: discontinue on day of sx except thiazides taken as antihypertensive
- sildenafil: discontinue 24hrs before sx
- COX 2 inhibitors: continue on day of sx unless surgeon is concerned about bone healing
- NSAIDs: discontinue 48hrs before day of Sx
- Warfarin: discontinue 4days before day of sx
- Mono amine oxidase inhibitors: continue medication and adjust anaesthesia plan accordingly.

LECTURE TWO (2)

General anesthesia

Definition: is a reversible state of hypnosis, analgesia, amnesia, muscle relaxation with good physiological homeostasis.

Components of general anesthesia:

1. **Sedation** and reduced anxiety
2. **Lack** of **awareness** and **amnesia**
3. Skeletal muscle **relaxation**
4. **Suppression** of undesirable **reflexes**
5. **Analgesia**

Note: Because **no single agent** provides all desirable properties, several categories of drugs are combined to produce optimal anesthesia.

STEPS of ANESTHESIA

General anesthesia has three main strategies:

1- Induction 2- Maintenance 3- Recovery

Induction

General anesthesia in adults is normally induced with an IV agent like propofol, producing unconsciousness in 30 to 40 seconds. Additional inhalation and/or IV drugs may be given to produce the desired depth of anesthesia

Maintenance

After administering the anesthetic, vital signs and response to stimuli are monitored continuously to balance the amount of drug inhaled and/or infused with the depth of anesthesia. Maintenance is commonly provided with volatile anesthetics, which offer

good control over the depth of anesthesia. Opioids such as fentanyl are used for analgesia along with inhalation agents, because the latter are not good analgesics.

Recovery

Postoperatively, the anesthetic admixture is withdrawn, and the patient is monitored for return of consciousness. For most anesthetic agents, recovery is the reverse of induction.

Stages of anesthesia

The depth of anesthesia has **four sequential stages** characterized by increasing CNS depression as the anesthetic accumulates in the brain.

Stage I: Analgesia: Loss of pain. (loss of eyelid reflex)

Stage II: Excitement: The patient displays delirium and possibly combative behavior. Potentially dangerous responses can occur during this stage including vomiting, laryngospasm, HTN, tachycardia, and uncontrolled movement

Stage III: Surgical anesthesia :
There is gradual loss of muscle tone and reflexes as the CNS is further depressed. Regular respiration and relaxation of skeletal muscles with eventual loss of spontaneous movement occur. This is the ideal stage for surgery.

Stage IV: Medullary paralysis (Overdose)

Severe depression of the **respiratory** and **vasomotor** centers occurs, Onset of apnea, dilated and nonreactive pupils, and hypotension to complete circulatory failure. Ventilation and/or circulation must be supported to prevent death.

INDUCTION METHODS

• Most anaesthetic inductions are performed using **intravenous** or **inhalational** ('gas') induction; each has advantages and disadvantages as below:

Intravenous	Inhalational
Advantage 1- Rapid onset 2- Patient comfort 3- Depression of pharyngeal reflexes allows early insertion of LMA 4- Airway protection in rapid-sequence induction	**Advantages** 1- Does not require IV access (Useful for patient with needle-phobia) 2- useful in pediatrics 3- Respiration is maintained 4- Upper esophageal sphincter tone maintained

Intravenous	Inhalational
Disadvantage 1- Venous access required 2- Risk of hypotension 3- Apnea common 4-Loss of airway control 5- Anaphylaxis	**Disadvantage** 1- Slow process 2- Potential excitement phase 3- Irritant and unpleasant, may induce coughing 4- Pollution 5- May cause a rise in ICP/IOP

LECTURE THREE (3)

Intravenous Anaesthesia

• General anaesthesia may be produced by many drugs which depress the CNS, including sedatives, tranquillizers and hypnotic agents.

• Only a few drugs are suitable for use routinely to produce anaesthesia after intravenous (i.v.) injection.

Properties of Intravenous Anesthesia

• Developed **later** than **inhalational** anaesthesia.

• used commonly to induce anaesthesia

• induction is usually **smoother** and more **rapid** than most of the inhalational agents.

• preferred now because it is **faster** with **less** risk of **excitement** or laryngospasm

• may also be used for maintenance, either alone or in combination with nitrous oxide

• they may be administered as repeated bolus doses or by continuous i.v. infusion.

• Used for sedation during regional anaesthesia

• used for sedation in the intensive care unit (ICU)

• used for treatment of status epilepticus.

• Used as the sole drug for short procedures (deep sedation)

From induction to wake up-bolus of IV induction drug

On entering the blood stream, a percentage of the drug **binds** to the **plasma proteins**, with the rest remaining unbound or '**free**'. The degree of protein binding will depend upon the physical characteristics of the drug.

The drug is carried in the venous blood to the right side of the heart, through the pulmonary circulation, and via the left side of the heart into the systemic circulation.

The majority of the cardiac output (**70**%) passes to the **brain**, **liver** and **kidney** (often referred to as '**vessel rich organs**'); thus a high proportion of the initial bolus is delivered to the cerebral circulation. The drug then passes along a concentration gradient from the blood into the brain.

❖ **The rate of this transfer is dependent on a number of factors**:

- Arterial concentration of the unbound **free** drug
- **Lipid solubility** of the drug
- Degree of **ionization**.

❖ **Unbound**, **lipid soluble**, **unionized** molecules cross the blood brain barrier the **quickest**.

❖ Once the drug has penetrated the CNS tissue, it exerts its effects. Like most anaesthetic drugs, the exact mode of action of the intravenous drugs is unknown. It is thought that each drug acts at a specific receptor – **GABAA**, **NMDA** and **acetylcholine** receptors.

❖ Following the initial flooding of the CNS and other vessel rich tissues with **non-ionized** molecules, the drug starts to diffuse into other tissues that do not have such a rich blood supply. This secondary tissue uptake, predominantly by **skeletal muscle**, causes the plasma concentration to fall, allowing drug to diffuse out of the CNS down the resulting reverse concentration gradient. It is this initial **redistribution** of drug into other tissues that leads to the rapid **wake up** seen after a single dose of an induction drug.

❖ **Metabolism** and **plasma clearance** have a much **less important** role following a **single bolus**, **but** are **more important** following **infusions** and **repeat doses** of a drug.

❖ The **fat**, with its poor blood supply (**vessel poor tissues**), makes little contribution to the **early redistribution** of free drug following a bolus. However, following repeat doses or infusions, equilibration with adipose tissue forms a drug reservoir, often leading to a delayed wake up.

In state of reduced cardiac output

In circumstances when **cardiac output** is **reduced**, for example after major blood loss, the body compensates by diverting an increased proportion of the cardiac output to the cerebral circulation. This preservation of cerebral blood flow in these situations is paramount.

Thus a greater proportion of any given drug will enter the cerebral circulation. As a result, the dose of induction drug must always be reduced. Furthermore, as global cardiac output is reduced, the time taken for an induction drug to reach the brain and exert its effect is prolonged. **The slow titration of a reduced dose of drug is the key to a safe induction in these patients.**

Properties of The Ideal Intravenous Anaesthetic Agent

1. **Physical properties**
   - Water soluble & stable in solution
   - Stable on exposure to light
   - Long shelf life
   - No pain on intravenous injection
   - Painful when injected into an artery
   - Non-irritant when injected subcutaneously
   - Low incidence of thrombophlebitis
   - Cheap

2. **Pharmacokinetic properties**
   - **o** Rapid onset in one arm-brain circulation time
   - **o** Rapid redistribution to vessel rich tissue
   - **o** Rapid clearance and metabolism
   - **o** No active metabolites.

Pharmacodynamics properties

- **o** High therapeutic ratio (ratio of toxic dose : minimally effective dose)
- **o** Minimal cardiovascular and respiratory effects
- **o** No histamine release/hypersensitivity reactions
- **o** No emetic effects
- **o** No involuntary movements
- **o** No emergence nightmares
- **o** No hang over effect
- **o** No adrenocortical suppression
- **o** Safe to use in porphyria.

BENZODIAZEPINES:

The mechanism of action was on the **GABAA** receptor. Flumazenil is a specific benzodiazepine–receptor antagonist that effectively reverses most of the central nervous system effects of benzodiazepines

Pharmacokinetics

Absorption: Benzodiazepines are commonly administered orally, IM, and IV to provide sedation or, less commonly, to induce general anesthesia.

Diazepam (**Valium**) was well absorbed from the gastrointestinal tract.

IM and IV injections of **diazepam** are **painful** while midazolam has no pain.

Distribution: valium (diazepam) is **lipid soluble** and readily penetrates into the blood, Redistribution is fairly rapid for the benzodiazepines while midazolam is **water soluble**.

Biotransformation: diazepam depends on the liver for biotransformation (metabolism) into water-soluble end products. The **metabolites** of diazepam are pharmacologically **active**. It has long elimination half-life for diazepam (**30** h).

Excretion The metabolites of benzodiazepine are excreted chiefly in the urine.

• **Intestinal - hepatic circulation** produces a secondary peak in diazepam.

Effects on Organ Systems (pharmacodynamics)

A. Cardiovascular The diazepam had **minimal** cardiovascular depressant effects, it decreases blood pressure and sometimes increase heart rate.

B. Respiratory: diazepam **depresses** the respiratory system. apnea may occur after induction; even small intravenous doses of diazepam have resulted in respiratory arrest.

C. Cerebral: diazepam reduces cerebral oxygen consumption, cerebral blood flow, and intracranial pressure. It **prevents** and **controls** seizures, produce amnesia, mild muscle-relaxation, anti-anxiety, and sedation

BARBITURATES: Sodium thiopental (thiopentone)

Barbiturates depress the reticular activating system (**RAS**) in the **brainstem**, which controls multiple vital functions. Their primary mechanism of action is believed to be through binding to (GABA) receptor. Barbiturates potentiate the action of GABA

• Thiopental's great lipid solubility and high nonionized fraction (60%) account for rapid brain uptake (within 30 s)

Pharmacokinetics

• **Absorption**: thiopental was frequently administered intravenously for induction of general anesthesia in adults and children (prior to the introduction of propofol). Rectal thiopental has been used for induction in children

• **Distribution** The **duration of sleep** doses of the thiopental is determined by **redistribution**, not by metabolism or elimination. Redistribution to the peripheral compartment specifically, the **muscle** group lowers plasma and brain concentration within **20–30 min.**

• Patients typically lose consciousness within **30 s** and awaken within **20 min**.

• Induction dose of thiopental will **depend on** body **weight** and **age**.

• **Reduced** induction doses are required for **elderly patients** primarily due to **slower redistribution.**

Sodium thiopental (thiopentone)

Presentation:

• Supplied as a pale **yellow powder**.

• Vials commonly contain sodium thiopental with 6% sodium carbonate.

• Reconstituted with water to yields a **2.5%** solution (25mg.ml-1) with a **pH** of **10.8**.

• The alkaline solution is bacteriostatic and safe to keep for **48** hours, but make it incompatible with many basic drugs.

Main action:

• **Hypnotic and anticonvulsant**

Pharmaceutical:

• A **dose** (**3–6** mg kg-1) of thiopentone produces a smooth onset of hypnosis within **30** seconds of intravenous injection.

• Recovery after a single dose is **rapid** due to **redistribution** and there is a low incidence of restlessness and nausea and vomiting.

• Thiopentone is **65-85%** protein bound in plasma. Metabolism is slow and occurs in the liver. Excretion of metabolites occurs mainly in the urine.

• Repeated doses or infusions of thiopental leading to an **accumulation** of the active drug and **delayed recovery**.

• **Biotransformation**: thiopental is principally metabolized by the liver via hepatic oxidation to **inactive** water-soluble metabolites.

• **Excretion**: the metabolites are excreted by urine

• **Effects on Organ Systems**

A. Cardiovascular: Intravenous bolus induction doses of barbiturates cause a decrease in blood pressure and an increase in heart rate.

Patients with poorly controlled hypertension are particularly prone to wide swings in blood pressure during anesthesia induction. The cardiovascular effects of barbiturates depend on **rate of administration, dose, volume status, baseline autonomic tone, and preexisting cardiovascular disease**.

B. Respiratory: thiopental causes:

• Depress respiratory center.

• Apnea may occur after induction dose.

• Tidal volume and respiratory rate are decreased

• Thiopental incompletely depress airway reflex responses to laryngoscopy and intubation (much less than propofol) and airway instrumentation may lead to bronchospasm (in asthmatic patients) or laryngospasm in lightly anesthetized patients.

• Bronchospasm may occur

Clinical use:

- induction of anaesthesia
- Treatment of status epilepticus
- Brain protection from the effects of hypoxia after stroke and head injury.

Effects on Organ Systems

C. Cerebral:

• Thiopental decrease cerebral blood flow, cerebral blood volume, and intracranial pressure.

• It also decrease cerebral oxygen consumption

• Some patients relate a taste sensation of garlic, onions, or pizza

• Thiopental do not impair the perception of pain. In fact, they sometimes appear to lower the pain threshold (increase the pain sensation)

• It controls seizures.

Other

• Thiopental may precipitate acute intermittent porphyria in susceptible individuals.

• Anaphylactic allergic reactions are rare.

• In particular, the usual recommended dose may need to be considerably reduced in **elderly**, **debilitated** and **hypovolemic** patients.

Ketamine

Chemical:

• Ketamine is a derivative of phencyclidine

Presentation:

• Ketamine is prepared in a slightly acidic colourless solution (pH 3.5–5.5) containing 10,50 or 100mg.ml 1.

Main action:

Dissociative anaesthesia.

Pharmaceutical:

- Ketamine is unique amongst induction drugs in that it can be administered IV, IM, orally, nasally, rectally, subcutaneously and the preservative free solution epidurally.
- For induction of anaesthesia a **dose** of 1–2 mg.kg-1 can be given IV, or 3–5 mg.kg-1 IM. plasma levels are usually achieved within 10–15 min after intramuscular injection.
- The onset of action is slower than other induction drugs
- Ketamine is metabolized in the liver, and excreted in the urine.

Ketamine has multiple effects throughout the central nervous system,

- Inhibition of reflexes in the spinal cord
- Excitation in selected areas of the brain.

ketamine functionally "**dissociates**" the brain from the awareness sensation.

Clinically, this state of **dissociative anesthesia** may cause the patient to appear conscious (eg, eye opening, swallowing, muscle contracture) but unable to process or respond to sensory input.

Ketamine has been demonstrated to be an N -methyl- d-aspartate (**NMDA**) receptor antagonist.

Pharmacokinetics

A. Absorption

Ketamine has been administered orally, nasally, rectally, subcutaneously, and epidurally, but in usual clinical practice it is given intravenously or intramuscularly plasma levels are usually achieved within 10–15 min after intramuscular injection.

B. Distribution

Ketamine is more lipid soluble and less protein bound than thiopental. So rapid brain uptake and subsequent redistribution was found in ketamine (the distribution half-life is 10–15 min). Awakening is due to redistribution from brain to peripheral compartments.

C. Biotransformation

Ketamine is metabolized in the liver to several metabolites, one of which (nor ketamine) retains anesthetic activity.

D. Excretion: End products of ketamine are excreted renally.

Effects on Organ Systems

A. **Cardiovascular**

• ketamine increases arterial blood pressure, heart rate, and cardiac output. These cardiovascular effects are due to central stimulation of the sympathetic nervous system and inhibition of the reuptake of norepinephrine.

• Increases in pulmonary artery pressure and myocardial work.

• ketamine's indirect stimulatory effects on the heart may be beneficial to patients with acute shock.

B. **Respiratory**

• Ventilatory drive is minimally affected by induction doses of ketamine

• It may produce apnea.

• Potent bronchodilator, making it a good induction agent for asthmatic patients

• Upper airway reflexes remain largely intact

C. **Cerebral**

• Ketamine **increases** cerebral oxygen consumption cerebral blood flow, and intracranial pressure.

• Undesirable psychotomimetic side effects (eg, disturbing dreams and delirium)during

recovery

• It had analgesic effect

• Ketamine comes closest to being a "complete" anesthetic as it induces analgesia, amnesia, and unconsciousness.

D. Gastrointestinal:

• Postoperative nausea and vomiting are common.

• Increase in salivation (can be reduced by premedication with anti-muscarinic drug such as Glycopyrrolate or atropine)

PROPOFOL

- Propofol action is on **GABA** receptor.
- Propofol actions are **not** reversed **flumazenil**
- it is **not water soluble** but it is available for intravenous administration as an oil-in-water emulsion containing soybean oil, glycerol, and egg lecithin.
- A history of egg allergy **does not** necessarily contraindicate the use of propofol because most egg allergies involve a reaction to egg white (egg albumin), whereas egg lecithin is extracted from egg yolk.
- Intravenous bolus dose (1–2.5 mg kg-1) for induction of anesthesia.
- It causes **pain during injection** that can be decreased by prior injection of lidocaine.
- Propofol formulations can support the **growth of bacteria**, so sterile technique must be observed in preparation and handling and it should be administered **within 6 h of opening of ampule**
- Postoperative nausea and vomiting appear to be extremely uncommon

Pharmacokinetics

A. Absorption

Propofol is available only for intravenous administration for the **induction of general** anesthesia and for **moderate** to **deep sedation**.

B. Distribution

• Propofol has a rapid onset of action(approximately 30 seconds). Awakening from a single bolus dose is also rapid due to a very short rapid distribution half-life (2–8 min).

• Recovery from propofol is more rapid than recovery from thiopental and ketamine

• Propofol should be titrated against the response of the patient until the **clinical signs** show the onset of anaesthesia. The **best endpoint is loss of verbal contact with the patient**

C. Biotransformation

• It has extrahepatic metabolism.

• Conjugation in the liver results in inactive metabolites

• The pharmacokinetics of propofol do not appear to be affected by obesity, cirrhosis, or kidney failure.

• **Use of propofol**

infusion for long-term sedation of children who are critically ill or young adult neurosurgical patients has been associated with sporadic cases of lipemia, metabolic acidosis, and death, the so-termed propofol infusion syndrome.

D. Excretion: Propofol is excreted in the urine by the kidney

Effects on Organ Systems

A. Cardiovascular: the major cardiovascular effect of propofol is

• decrease in arterial blood pressure (**Propofol** causes the **most marked decrease** in blood pressure of all the induction drugs.)

• Decrease in cardiac contractility.

- Factors associated with **propofol- induced hypotension** include **large doses**, **rapid injection**, & **old age.**
- Myocardial oxygen consumption and coronary blood flow usually decrease comparably

B. Respiratory

- Propofol is a profound respiratory depressant that usually causes apnea following an induction dose.
- it depresses the normal response to hypercarbia.
- It induced depression of upper airway reflexes exceeds that of thiopental, allowing intubation, endoscopy, or laryngeal mask placement in the absence of neuromuscular blockade.
- Although propofol can cause histamine release, induction with propofol is accompanied by a lesser incidence of wheezing in both asthmatic and non-asthmatic patients compared with barbiturates or Etomidate.

Effects on Organ Systems

C. Cerebral

- Propofol **decreases** cerebral blood flow, cerebral blood volume and intracranial and intraocular pressure.
- propofol and thiopental probably provide a similar degree of **cerebral protection**.
- Unique to propofol are its antipruritic properties.
- Its antiemetic effects provide yet another reason for it to be a preferred drug for outpatient anesthesia.
- Propofol has anticonvulsant properties, has been used successfully to terminate status epilepticus, and may safely be administered to epileptic patients.
- Induction is occasionally accompanied by excitatory phenomena such as muscle twitching, spontaneous movement, opisthotonus, or hiccupping.

• Tolerance does not develop after long-term propofol infusions.

• Propofol is often combined with remifentanil, or ketamine for TIVA.

Etomidate

Chemical:

• Etomidate is an imidazole ester.

Presentation:

• It is usually presented as a **lipid emulsion** or as a clear solution containing propylene glycol at a concentration of 2mg.ml-1.the pH of aqueous solution is 8.1

Main action:

• Hypnotic

Pharmaceutical:

• The standard induction dose is 0.3mg.kg-1

• The recovery is rapid due to **redistribution** to muscle and fat.

• It is rapidly metabolized by hepatic and plasma esterases

• Excretion is predominantly **urinary**

Clinical use:

• induction of anaesthesia

• is an ultrashort-acting, non-barbiturate hypnotic intravenous anesthetic agent.

• Etomidate does not have any analgesic properties.

• It is administered only by intravenous route.

• Etomidate has a favorable hemodynamic profile on induction, with minimal blood pressure depression, making **it ideal for shock trauma, hypovolemic patients, or patients with significant cardiovascular disease.** Etomidate has been approved for use during induction.

Pharmacokinetics: The onset of action: 30 to 60 seconds, Peak effect: 1 minute

• **Metabolism**: Metabolism is primarily hepatic by ester hydrolysis to inactive metabolites.

• **Excretion**: 75% of the administered dose is excreted in the urine on the first day after injection. Another route of excretion is bile.

• Like most intravenous anesthetics, etomidate is highly protein-bound (77%). Thus, it can achieve a higher concentration in the brain in low albumin states since it will be less bound to albumin, and more free-drug would be available in the brain.

Adverse Effects

Transient inhibition of adrenal steroid synthesis is considered etomidate's most significant adverse effect.

• Etomidate is no longer administered by continuous infusion because of the risks of sustained suppression of endogenous cortisol and aldosterone production.

• The most common adverse reaction associated with etomidate use is transient intravenous **pain on injection**. The pain appears less frequent when larger, more proximal arm veins are used, or IV lidocaine is given before an etomidate bolus.

• Transient skeletal muscle movements or myoclonus were observed in about 32% of the patients.

Postoperative nausea and vomiting with etomidate are comparable to the general frequency of PONV. The incidence of PONV was higher when etomidate was used for both induction and maintenance of anesthesia in short procedures such as dilation and curettage or when analgesia was insufficient

Cardiovascular Effects

• Etomidate has minimal effects on the cardiovascular system and is the major reason for choosing this drug as an induction agent.

• Etomidate does **not release histamine**. However, etomidate by itself, even in large doses, produces relatively light anesthesia for laryngoscopy, and marked increases in

heart rate and blood pressure may be recorded when etomidate is solely used for induction.

Respiratory Effects

- Ventilation is not significantly affected. Induction doses do not result in apnea unless opioids have also been administered.
- The most distinctive effect on the respiratory system is a slight rise in arterial carbon dioxide tension (PaCO2).

Central Nervous System Effects

- Etomidate decreases cerebral metabolic rate, cerebral blood flow, and intracranial pressure.
- Because of minimal cardiovascular effects, cerebral perfusion pressure is well maintained.
- Etomidate lacks analgesic properties

LECTURE FOUR (4)

Inhalational Anaesthetic Agents

Definition: An inhalational anesthetic is a chemical compound possessing general anesthetic properties that can be delivered via inhalation.

- Inhalational Anesthesia refers to the **delivery of anesthetics gases or vapors to the respiratory system to produce anesthesia.**
- The main use of inhalational was to **maintain anesthesia**, some of them can be used as **induction**
- They are administered through a **face mask**, **laryngeal mask airway** or **tracheal tube** connected to an anesthetic vaporizer and an anesthetic delivery system.
- The famous demonstration of an **ether** anaesthetic by William Morton in 1846.
- The dose of inhalational anesthetic was mentioned as MAC

Classification

1. Gases

Nitrous oxide and Xenon

2. Volatile agent

A-fluorinated ethers (isoflurane, sevoflurane and desflurane)

B-halogenated hydrocarbon (halothane)

Mechanisms of Action

• The exact mechanism of action for inhaled anesthetics remains mostly unknown. they work within the central nervous system by **augmenting** signals to (GABA receptors) while **depressing** neurotransmission pathways of **acetylcholine** to muscarinic and nicotinic receptors

• The **two main theories** for inhalational anesthetic action focus on direct interaction with two components of the cell membrane.

✓ **Lipid theory (Meyer–Overton relationship):**Meyer and Overton showed a close relationship between the lipid solubility of the inhalational agent and its potency of anaesthetic activity. They noticed that there was a straight line relationship between log minimum alveolar concentration (MAC; i.e. potency) and the lipid solubility; the more lipid soluble the agent (represented by a **higher** log oil/gas partition coefficient), the greater the **potency**.

✓ **Protein site of action theory:** Throughout the CNS, there are many excitatory and inhibitory ligand-gated ion channels. There is increasing evidence that anaesthetic agents act by inhibiting excitatory (serotonergic, neuronal nicotinic and N-methyl-D-aspartate (**NMDA**)) channels and activating inhibitory channels (γ-aminobutyric acid A (**GABAA**) and glycine).

Pharmacokinetic of inhalational anesthetics

The forward movement of inhalational agent is determined by a **series of partial pressure gradients**, beginning at the vaporizer of the anesthetic machine, continuing in the breathing circuit, the alveolar tree, blood, and then tissue.

• The principal objective of that movement is to achieve equal partial pressures on both sides of each single barrier.

• The alveolar partial pressure governs the partial pressure of the anesthetic in all body tissues; they all will ultimately equal the alveolar partial pressure of the gas.

• After a short period of equilibration the alveolar partial pressure of the gas equals the brain partial pressure.

So there was an **uptake , ventilation and concentration** that effect the induction rate and awaking time

• **Partial pressure** is the **ratio** of the amount of substance in one phase to the amount in another phase

• Recovery from anesthesia depends on lowering the concentration of anesthetic in brain tissue.

• Anesthetics can be eliminated by biotransformation, transcutaneous loss, or exhalation.

• Biotransformation usually accounts for a minimal increase in the rate of decline of alveolar partial pressure.

• Diffusion of anesthetic through the skin is insignificant.

So The most important route for elimination of inhalation anesthetics is the alveolus.

AGENTS IN COMMON CLINICAL USE

In Western countries, it is customary to use one of the **four** modern volatile anaesthetic agents –**isoflurane**, **desflurane**, **sevoflurane** or **halothane**–vaporized in a mixture of nitrous oxide in oxygen or air and oxygen.

The use of halothane has declined because of medicolegal pressure relating to the very rare occurrence of hepatotoxicity.

The use of **sevoflurane** has increased rapidly, particularly in paediatric anaesthesia because of its superior quality as an inhalational induction agent.

Desflurane produces rapid recovery from anaesthesia, but it is very **irritant** to the airway and is therefore not used as an inhalational induction agent.

Characteristic of IDEAL inhalational:

•Non-flammable, non-explosive at room temperature

• Stable in light.

• Liquid and vaporizable at room temperature

• Stable at room temperature, with a long shelf life

• Stable with soda lime, as well as plastics and metals

• Environmentally friendly - no ozone depletion

• Cheap and easy to manufacture

• Non toxic

• Rapid induction and rapid recovery

• Safe with no toxic side effect

*There was no ideal inhalation till now

Common Undesirable effects of the volatile agents

1.They all depress respiration

2.Reduce uterine tone

3.Trigger MH

4.All increase cerebral blood flow and ICP.

Feature	Halothane	Isoflurane	Enflurane	Desflurane	Sevoflurane
Thymol required	+	–	–	–	–
Decomposed by light	+	–	–	–	–
Approximate cost/100 ml (£)	4–5	15–20	10–13	15–20	45–50
Irritant to breathe	–	+	+/–	++	–
Cardiac output	↓↓	↓	↓↓↓	↓	↓
SVR	(↓)	↓↓	↓	↓↓	↓↓
Heart rate	↓	↑	↑↑	↑	↑↓
Sensitivity to catecholamines	+++	+	++	–	–
Metabolised (%)	20	0.2	2	0.02	3–5
Others	Halothane hepatitis	Coronary steal	Epileptiform EEG	Direct metering vaporiser required	Decomposed by soda lime/baralyme

POTENCY

The potency of an inhalational anaesthetic agent can be measured by its (Minimum Alveolar Concentration- **MAC**)

MAC :- (Minimal alveolar concentration)

• The minimum alveolar concentration (**MAC**) of an inhaled anesthetic is the One MAC is **defined as:** The minimum alveolar concentration of an inhaled anesthetic is the alveolar concentration that prevents movement in 50% of patients in response to a standardized stimulus (eg, surgical incision). e.g : Halothane MAC 0.75%

• **'The MAC is a useful measure because it mirrors brain partial pressure, allows comparisons of potency between the inhaled agents**,

• The **MAC** values for different anesthetics are roughly additive. For example, a mixture of 0.5 MAC of nitrous oxide (53%) and 0.5 MAC of halothane (0.37%) produces the same likelihood that movement in response to surgical incision will be suppressed as 1.0 MAC of isoflurane (**1.7**%) or 1.0 MAC of any other single agent.

MAC is reduced by:

• Nitrous oxide
• Hypothyroid/myxedema
• Hypocapnia
• Hypothermia-decrease is roughly linear
• Hyponatraemia
• Increasing age
• Hypoxaemia
• Hypotension
• Anemia
• Pregnancy
• CNS depressant drugs
• Other drugs: lithium, lidocaine, magnesium, Acute alcohol abuse

MAC is increased by:

• Hyperthermia
• Hypernatraemia
• Sympatho-adrenal stimulation
• Chronic alcohol abuse
• Chronic opioid abuse
• Increases in ambient pressure
• Hypercapnia
• Decreasing age
• Thyrotoxicosis

HINT: Sex, Weight and Duration of anesthesia does not affect MAC Nitrous oxide MAC105% , Halothane (Fluothane) 0.75% ,Isoflurane 1.2% ,Desflurane 6.0% ,Sevoflurane 2.0 %

NITROUS OXIDE (N2O)

N2O is a relatively insoluble agent with a **low blood–gas partition coefficient** And has a **high MAC** (105) and is widely used in combination with other inhaled anaesthetic agents or with O2 as **entonox**.

Physical proprieties:

- It is inorganic anesthetic gas
- Slightly sweet-smelling gas
- Non-flammable but supports combustion
- Breaking down to O2 and nitrogen at high temperatures.
- Supplied as a **liquid/gas** in French blue cylinders
- Ice often forms on the cylinder during use
- It is colorless and essentially odorless
- It is stored as a **liquid** in **blue** cylinders with a gauge pressure of 51bar at 20ºC.
- The gauge pressure does not give an indication of cylinder content until all the remaining N2O is in the gaseous phase.
- It is more diffusible than oxygen or nitrogen

Induction and recovery:

Nitrous oxide is

• Fast onset and recovery;

• strongly analgesic but weakly anaesthetic.

• It is widely used in obstetric practice to relieve pain during childbirth and in minor surgical procedures.

• Useful analgesic for dental extraction

• Both induction and recovery from anaesthesia are extremely rapid.

• It's previously known as (laughing gas)

• It rapidly enters enclosed air-containing spaces more rapidly than oxygen or nitrogen can leave. These spaces include the **cuff** of an endotracheal tube, the **bowel**, **pneumothorax** ,the **middle ear**, **nasal sinuses** and the eye and air **emboli**, and nitrous oxide will increase their volume

- It may cause postoperative temporary hypoxia (**diffusion hypoxia**).

Effects of N2O

Respiratory

· Non-irritant. Depresses respiration slightly.

• May cause diffusion hypoxia at the end of surgery.

Cardiovascular

· Mild direct myocardial depression which is offset by an increase in sympathetic activity via its central effects.(Little effect on heart rate and BP usually)

Central nervous system

· Increases cerebral metabolism, cerebral blood flow and ICP slightly.

Toxicity

· It interferes with DNA synthesis even after relatively brief exposure.

Others:

• Post operative nausea and vomiting

• Does not affect hepatic or renal function, nor uterine or skeletal muscle tone.

• Prolonged use may cause bone marrow depression, megaloblastic anaemia and peripheral neuropathy.

• Generally considered as being safe during pregnancy

Xenon

• It Is a noble gas with an anaesthetic effect.

• Xenon is a nearly ideal anaesthetic agent.

• At a concentration of 70% mixed with 30% oxygen it induces general anaesthesia without side effects.

• It cannot be synthetized and is isolated from air, which contains 0.0000087% xenon.

• inert and odourless gas

- very fast onset and offset of anaesthesia (low blood:gas partition coefficient)
- Expensive
- It is not metabolized and is excreted unchanged via the lungs.
- It is non-toxic, not flammable,
- non-irritant to the airway.
- Xenon has analgesic properties
- Muscle relaxation at higher concentrations.

- **minimal cardiovascular effects**

• (small decrease in heart rate only)

• minimal respiratory effects (slows respiratory rate slightly, but increase tidal volume to compensate).

• It does not cause malignant hyperthermia.

HALOTHANE

• Halothane is a highly soluble agent with a **high** blood–gas partition coefficient and its **MAC** is (0.75)

Physical Properties

• Colorless liquid, Pleasant smell

• Halothane is a halogenated alkane, Supplied in liquid form with thymol 0.01%

• Halothane is unstable when exposed to light so thymol preservative and amber-colored bottles retard spontaneous oxidative decomposition.

• it was nonflammable and non-explosive

• It corrodes certain metals and dissolves into rubber.

• Its use rapidly spread because of its greater potency, ease of use, non-irritability and non-inflammability

• Risks of arrhythmias and liver damage on repeated administration (halothane hepatitis)

• Halothane is the least expensive volatile anesthetic.

Effects:

Respiratory

• Depress Minute ventilation largely due to decreased tidal volume

• The normal response to hypoxia and hypercarbia are blunted.

• It has a sweet non-irritant odour and may be used for gaseous induction.

• Halothane also bronchodilator and is useful in asthmatic patients.

• Non-irritant. Pharyngeal, laryngeal and cough reflexes are abolished early

- Respiratory depressant, with increased respiratory rate and reduced tidal volume.
- inhibition of secretions.
- **Cardiovascular**
- Myocardial depression and bradycardia.
- Hypotension is common. (reduce sys. Vascular resistance)
- Myocardial O2 demand decreases.
- Arrhythmias are common, e.g. Bradycardia, ectopic
- Sensitizes the myocardium to catecholamines, e.g. Endogenous or injected adrenaline.
- **Central nervous system**
- Smooth rapid induction, with rapid recovery.
- Anticonvulsant action.
- Increases cerebral blood flow but reduces intraocular pressure.
- **Others**
- Dose-dependent uterine relaxation.
- Nausea/vomiting is uncommon.
- May precipitate Malignant Hyperthermia.
- Up to 20% is metabolized in the liver.
- Repeat administration after recent use may result in hepatitis.
- **Toxicity**

· Hepatic damage (halothane hepatitis)

· Factors include: multiple exposures, obesity, middle age and female sex.

· Mortality is around 50-75%.

· Halothane should be avoided:

✓ If it has been given in the previous 3 months
✓ If there is a past history of adverse reaction to halothane
✓ If there is preexisting liver disease.

Contraindications

· Severe hypovolemia

· Malignant hyperthermia

· Intracranial hypertension

· Halothane hepatitis

Isoflurane

Physical proprieties:

• Colorless liquid

• Pungent odor

• MAC 1.20

• Non-flammable, non-corrosive.

• With no additive.

• Relatively insoluble and has a **low** blood– gas partition coefficient.

• Is **widely used for** maintenance of anaesthesia and treatment of severe asthma in patients requiring mechanical ventilation in ICU.

Effects

Central nervous system

• Smooth, rapid induction, but speed of uptake is limited by respiratory irritation.

• Recovery is slower than with sevoflurane and desflurane.

• Anticonvulsant properties

• Reduces Cerebral Metabolic Rate of O2.

• Increases cerebral blood flow and ICP.

• Decreases intraocular pressure.

• Has poor analgesic properties.

Respiratory

•Irritant; more likely to cause coughing and laryngospasm(gaseous induction is not recommended)

• Respiratory depressant(more than halothane), with increased rate and decreased tidal volume.

• Causes bronchodilation.

Cardiovascular

• Reduce SVR.

• Myocardial depression is less than with halothane

• Vasodilatation and hypotension commonly occur

• Compensatory tachycardia is common

• Myocardial O2 demand decreases, but tachycardia may reduce myocardial O2 supply.

• It may cause (coronary steal) whereby normally responsive coronary arterioles are dilated and divert blood away from areas supplied by diseased and unresponsive vessels, resulting in ischaemia

Other:

• Dose-dependent uterine relaxation.

• Nausea/vomiting is uncommon.

• Skeletal muscle relaxation

• May precipitate MH.

• Widely used in neurosurgery

Contraindications

· Severe hypovolemia

· Malignant hyperthermia

· Intracranial hypertension

SEVOFLURANE

It was originally used as an inhalational agent in **Japan** and is now widely used in the world, particularly in paediatric practice. Sevoflurane is relatively insoluble in blood and has a low blood–gas partition coefficient.

Physical proprieties:

• Colorless liquid

• Pleasant smelling

• MAC is (2)

• Non-flammable, non-corrosive, stable at ambient temperatures

• Supplied in liquid form with no additive.

• Interacts with soda lime to produce compounds A

• **Non-pungent, low solubility- excellent for inhalation induction**

• **muscle relaxation (enough for pediatrics intubation)**

• **potentiates NMBA.**

• The degradation of sevoflurane by soda lime and baralyme is results in the formation of Compound A which may cause renal tubular necrosis

- **Effects**

Respiratory

• Well-tolerated

• Minimal airway irritation.

• Respiratory depressant, with increased rate and decreased tidal volume.

• Causes bronchodilatation.

Cardiovascular

• Heart rate and contractility are unchanged, but a fall in SVR leads to a reduction in blood pressure.

• Vasodilatation and hypotension may occur

• Myocardial O2 demand decreases.

• Arrhythmias uncommon

• Central nervous system

• Smooth, extremely rapid induction and recovery.

• Early postoperative analgesia may be required as emergence

• Is so rapid.

• Increases the risk of emergence agitation

• Anticonvulsant properties.

• Reduces cerebral metabolic rate of O2

• Decreases intraocular pressure.

• Has poor analgesic properties

• **Other**

• Dose-dependent uterine relaxation.

• Nausea/vomiting occurs.

• Skeletal muscle relaxation

• May precipitate MH.

• Tracheal intubation may be performed easily with spontaneous Respiration.

• Considered the agent of choice for inhalational induction in pediatrics because of its rapid and smooth induction characteristics.

• Has also been used for the difficult airway, including airway obstruction.

DESFLURANE

Its **low blood: gas partition coefficient** results in fast onset and offset of action. These properties make it ideal for long procedures where rapid wake-up is important to assess the patient.

Physical proprieties:

• Introduced in the UK in 1994

• a colorless liquid with slightly **pungent vapor**

• boiling point: 23°C, MAC: 5%–7% in adults; 7.2%–10.7% in children

• non-flammable, non-corrosive

• supplied in liquid form with no additive

• may react with dry soda lime to produce carbon monoxide

• Desflurane uses specific electrically powered vaporizer (Tec 6) due to its low boiling point.

Effects

Respiratory

1) Causes airway irritation; not recommended for induction of anesthesia because respiratory complications (e.g. laryngospasm, breath-holding, cough, apnea) are common and may be severe.
2) Respiratory depressant, with increased rate and decreased tidal volume.

Cardiovascular

1) Vasodilatation and hypotension may occur, similar to isoflurane, may cause tachycardia and hypertension via sympathetic stimulation, especially if high concentrations are introduced rapidly.
2) Myocardial ischemia may occur if sympathetic stimulation is excessive, but has cardioprotective effects in patients undergoing cardiac surgery.
3) Arrhythmia as uncommon, as for isoflurane, little myocardial sensitization to catecholamines.
4) Renal and hepatic blood flow generally preserved.

Effects

Central nervous system

1) Rapid induction (although limited by its irritant properties) and recovery.
2) May increase cerebral blood flow, although the response of cerebral vessels to CO2 is preserved.
3) ICP may increase due to imbalance between the production and absorption of CSF.
4) Reduces CMRO2 as for isoflurane.
5) Has poor analgesic properties.

Others

• 0.02% of Desflurane undergoes metabolism by liver

• Dose-dependent uterine relaxation (although less than isoflurane and sevoflurane).

• Skeletal muscle relaxation; non-depolarising neuromuscular blockade may be potentiated

Contraindications

· Severe hypovolemia

· Malignant hyperthermia

· Intracranial hypertension

Advantages and disadvantages of the modern volatile anaesthetic agents

	Isoflurane	Sevoflurane	Desflurane
Advantages	• Low cost; approximately 70% cheaper than sevoflurane • Bronchodilator • No significant toxic metabolites • Non-arrhythmogenic	• Good for inhalational induction • Non-irritant to airways • Faster onset/offset than isoflurane • Non-arrhythmogenic	• Fast onset/recovery from anaesthesia • Minimal, non-toxic metabolites
Disadvantages	• Concerns about coronary steal syndrome • Irritant to airways	• Expensive • Metabolised to toxic metabolites (not thought to be of clinical concern) • Formation of potentially toxic compounds on interaction with soda lime/ baralyme (not thought to be of clinical concern)	• Expensive • Irritant to airways • Causes tachycardia at higher concentrations • Heated/pressurised vaporiser required for delivery
All volatile agents: • may trigger malignant hyperpyrexia in susceptible individuals; • have a higher incidence of PONV compared with total intravenous anaesthesia; • have a possible association with postoperative cognitive dysfunction (see Chapter 31); • are environmental greenhouse gases; and • cause dose-dependent CVS/RS depression.			

LECTURE FIVE (5)

Pediatric Anesthesia

Part2

Introduction

It is often said that **paediatric** patients are '**not simply small adults**'. The truth is that from the premature neonate to the near-adult adolescent, children are very diverse.

Pediatric patients involve the fallowing age groups: -

1.**Neonates** (0–1 months). Up to 44 weeks post conception (includes premature neonates)

2.infants (1–12 months). From 44 weeks post conception – 1 year

3.toddlers (12–24 months).

4.young children (2–12 years of age).

Pediatric anesthesia has differing anesthetic requirements. **physiological**, **anatomic**, and **pharmacological** characteristics of each group.

Indeed, **infants** are at much greater risk of anesthetic morbidity and mortality than older children; **risk** is generally inversely proportional to **age**.

Estimation of weight

It is essential that every child is weighed prior to anaesthesia. This allows correct calculation of drug doses and selection of anaesthetic equipment. Weight can also be

estimated from the age of the child from standard growth charts, from the length of the child, or using this formula:

Age of child	Formula to estimate weight in kg
0-12 months	(0.5 x age in months) +4
1-5 years	(2x age in years) +8
6-12 years	(3x age in years) +7

Anatomical & Physiological Differences

1-Respiratory System Differences

The major anatomical differences affecting **airway management** in neonates and infants are:

· Relatively **large head** and prominent occiput

· **Small mandible**

· Relatively **large tongue**

· **Short neck**

• **narrower nasal passages**, an anterior and cephalad larynx, a longer epiglottis, and a shorter trachea. These anatomic features make neonates and young infants **obligate nasal breathers** until about **5** months of age

· **Soft tracheal cartilages**, easily compressed.

These differences predispose to **airway obstruction**, particularly if the child's head is placed on a pillow, or the soft tissues on the floor of the mouth are compressed, or the head is hyperextended. **Ideally**, maintain the child's head in a **neutral** position, or **slightly** extended.

Anatomical differences affecting the larynx include:

- ❖ A high, anterior position of the larynx (level of **C3-4** in infants compared to **C5-6** in **adults**)
- ❖ A **long**, U-shaped **epiglottis**
- ❖ The narrowest part of the airway is at the **cricoid cartilage** (below the vocal cords).
  - o The narrowest part of the airway in adults is at the **vocal cords**.
  - o Adeno-tonsillar hypertrophy is common in children 3 – 8 years of age.
  - o Airway obstruction may develop after induction of anaesthesia; an oropharyngeal may help to maintain a patent airway.
  - o Take care when passing nasopharyngeal, nasotracheal and nasogastric tubes in these children.

Children aged 5-13 years may have loose teeth; take note of loose teeth at your pre-assessment visit

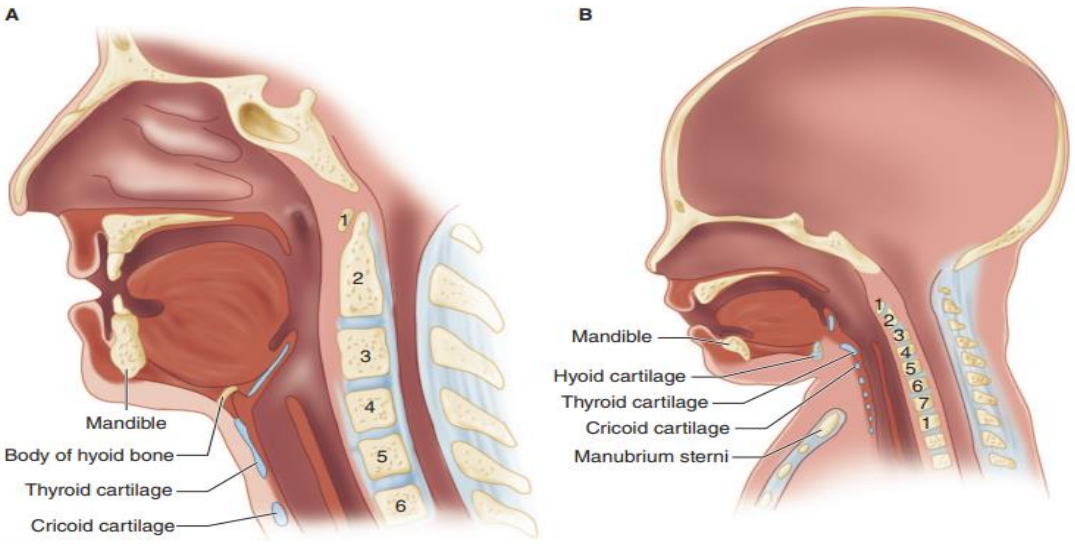


FIGURE 42–1 Sagittal section of the adult (**A**) and infant (**B**) airway. (Reproduced with permission from Snell RS, Katz J. *Clinical Anatomy for Anesthesiologists*. New York, NY: Appleton & Lange; 1988.)

The major physiological differences in respiratory system

- ❖ Faster respiratory rate
- ❖ Lower lung compliance
- ❖ Greater chest wall compliance
- ❖ Lower functional residual capacity
- ❖ high metabolic rate and oxygen consumption

Note: children **tidal volume** is relatively **fixed** (5-7 ml.kg-1), and the infant can **only** increase minute ventilation by increasing **respiratory rate**.

❖ **Apnoeas** are particularly **common** in **premature** and **ex-premature infants**, so monitor

all babies for apneas after surgery; until they are 60 weeks post conception.

If a mechanical ventilator is used, select the appropriate tidal volume and respiratory rate for age – **pressure control ventilation is preferred**

2-Cardiovascular considerations

❖ Residual fetal circulation

❖ **Noncompliant left ventricle** : so increase in **cardiac output** is achieved through

an increase in **heart rate** (Heart-rate-dependent cardiac output)

❖ Faster heart rate: It is important to **avoid bradycardia**. This should be treated rapidly if it occur; the most **common cause** is **hypoxia**

Lower blood pressure

Age-related changes in vital signs.

Age	Respiratory Rate	Heart Rate	Arterial Blood Pressure	
			Systolic	*Diastolic*
Neonate	40	140	65	40
12 months	30	120	95	65
3 years	25	100	100	70
12 years	20	80	110	60

❖ Activation of the **parasympathetic** nervous system by anesthetic **overdose**, or **hypoxia** can quickly trigger **bradycardia** and profound reductions in **cardiac output**.

❖ **Bradycardia** that can lead to **hypotension**, **asystole**, and intraoperative **death**.

❖ The **immature heart** is more sensitive to depression by **volatile anesthetics** and to **opioid-induced** bradycardia.

❖ The main causes of neonatal bradycardia and cardiac arrest during anesthesia are: -

✓ **Respiratory causes:** - airway obstruction, bronchospasm, inadequate O2 delivery.

✓ **Pharmacology causes:** -inhalation agents, succinylcholine, anticholinesterase.

✓ **Metabolic causes**: -hypothermia, anemia, hypoglycemia.

✓ **Children are more** susceptible than adults to **cardiac arrhythmias, hyperkalemia, masseter spasm, and malignant hyperthermia associated with succinylcholine.**

✓ When a child experiences cardiac arrest following administration of **succinylcholine**, immediate treatment for **hyperkalemia** should be instituted

3. Metabolism & Temperature Regulation Differences.

Neonates promote greater **heat loss to the environment** and liable to hypothermia **Because: -**

1. Thin skin.
2. Low fat content.
3. Greater surface area relative to weight.
4. Inadequately warmed operating rooms (cold theater)
5. Prolonged wound exposure.
6. Administration of room temperature intravenous or irrigation fluid.
7. Dry anesthetic gases
8. Effects of anesthetic agents on temperature regulation center.

Even mild degrees of hypothermia can cause perioperative problems which including: -

• Delayed awakening from anesthesia.

• Cardiac irritability and arrest.

• Respiratory depression.

• Altered responses to anesthetics and Neuromuscular blockers, and other agents.

4-Renal & Gastrointestinal Function Differences

- The **total body water** is about **80**% of body weight at birth, gradually **decreasing** with **age**. fluid loss is more critical problem to them.
- **Immature kidney function** increases the importance of meticulous attention to fluid administration in the early days of life
- **Neonates also have a relatively increased incidence of gastroesophageal reflux.**
- **The immature liver conjugates drugs and other molecules less readily early in life.**

5-Glucose Homeostasis Differences

▪Neonates have relatively **reduced glycogen stores**, predisposing them to **hypoglycemia**.

6-Pharmacological Differences: The main difference is prolonging the clinical duration of action of drugs such as **thiopental** and **fentanyl**. this Because: -

1. **larger** pediatric intravascular and extracellular fluid **compartments** compare with adult. Neonates and infants have a proportionately greater total water content than adults (50–60%).

2.Immaturity of hepatic **biotransformation** pathways,

3.Decreased **protein** for drug binding.

4.Smaller **muscle mass** in neonates **prolongs** or delaying redistribution of some drugs such as thiopental and fentanyl.

Volatile anaesthetic

- Neonates are more sensitive to volatile agents than older children
- The minimum alveolar concentration (**MAC**) values are decreased in neonates but increased in infants and children compared to adults.

Sedatives and hypnotics

Children are particularly sensitive to sedative and hypnotic drugs such as **barbiturates** and **benzodiazepines** due to the **Immature hepatic biotransformation** and Decreased
protein binding so these drugs should be used with caution, in weight appropriate doses, titrated according to effect.

Muscle relaxants

Neonates and **infants** are **more sensitive** to non-depolarizing neuromuscular blocking drugs **because** Immature neuromuscular junction.

A normal **loading dose** is given but **subsequent** doses should be reduced

LECTURE SIX (6)

Pediatric Anesthesia

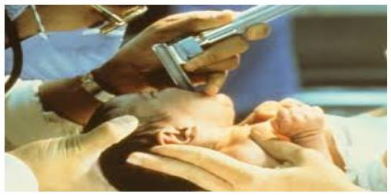
Part2

Anaesthesia management

- **Preoperative Preparation**
- All children should be visited preoperatively by the anaesthetist responsible for caring for them in the perioperative period.
- There is an increased incidence of airway problems during anaesthesia
- children are more at risk of laryngeal spasm, breath-holding and bronchospasm
- in the postoperative period the chance of post-intubation croup is increased.
- It is extremely important that the child is weighed before arrival in theatre, because body weight is the simplest and most reliable guide to drug dosage.
- Veins suitable for insertion of a cannula should be identified.
- Morbidity and mortality caused by aspiration of gastric contents are extremely rare in children undergoing elective surgery.
- Prolonged periods of starvation in children, especially the very young infant, are harmful.
- These children, who have a rapid turnover of fluids and a high metabolic rate, are at risk of developing hypoglycaemia and hypovolaemia.
- Solids (including breast and formula milk) should not be given for at least 6 h before the anticipated start of induction.

- In the emergency setting, e.g. the child who has sustained trauma shortly after ingesting food, it is probably best (if possible) to wait 4 h before inducing anaesthesia. Clearly, in this situation risk–benefit judgements have to be made.
- If it is surgically possible to wait 4 h, an i.v. infusion of a glucose-containing solution such as 5% dextrose with 0.9% NaCl, must be commenced and, if necessary, appropriate fluid resuscitation undertaken.
- **Intravenous Induction**
- The same induction sequence can be used as in adults: a rapid-acting barbiturate (eg, thiopental, 3 mg/kg in neonates, 5–6 mg/kg in infants and children) or propofol (2–3 mg/kg) followed by a non-depolarizing muscle relaxant (eg, rocuronium, cisatracurium, atracurium, mivacurium, or succinylcholine).
- Atropine should be given intravenously prior to succinylcholine.
- It is important that children are accompanied into the anaesthetic room by someone with whom they are familiar.
- The appropriate monitoring should be applied as soon as possible after the start of anaesthesia.
- When inhalational induction is planned, clear, scented plastic masks are much more acceptable to small children than the traditional **Rendell–Baker rubber masks**.
- Clear masks allow respiration and the presence of vomitus to be observed.
- An alternative to using a mask is cupping the hands over the face of the child while holding the T-piece, It is important to ensure that the flow of fresh gas is directed away from the child's eyes because anaesthetic gases may be irritant.
- When using a face mask, it is important that the soft tissue behind the chin is not pushed backwards by the fingers, thereby obstructing the airway. **The anaesthetist's fingers should rest only on the mandible.**






- **Airway Management**
- The Jackson–Rees modification of the Ayre's T-piece is the breathing system used traditionally for children under **20 kg** in weight.
- It has been designed to be **lightweight** with a **minimal** apparatus dead space. The apparatus may be used for both spontaneous and **controlled** ventilation
- The **open-ended** reservoir bag is used for manually controlled ventilation. This mode of ventilation is especially useful in the neonate and infant.
- Laryngeal mask airway (**LMA**) should be used only when it is planned that the child is to breathe spontaneously during surgery. It follows that **it is <u>unwise</u>** to use the device when neuromuscular blocking drugs are used.
- It is **mandatory** to intubate the trachea during **artificial** ventilation.
- Neonates with a tracheal tube must undergo artificial ventilation in order to reduce the work of breathing.
- Infants have a head which is large and a neck which is short relative to the size of the body. Instead of placing a pillow under the head, **it is usually necessary to place a small pad or pillow under the torso.**
- **Tracheal intubation**

For children over 1 year:

- Appropriate tube internal diameter (ID) can be approximately estimated by the formula: age / 4 + 4.

- Appropriate tube length in cm. can be approximately estimated by the formula: age / 2 + 12 oral (+15 for nasal).

In infants:

- Appropriate tube ID sizes for preterm: <1500 g = 2.5 mm, 1500-3000 g = 3 mm, over 3000 g = 3.5 mm.
- Oral length in cm. is given by the formula (6 + weight in kg).

Laryngeal mask airway (LMA):

They are useful in short procedures with spontaneous ventilation. They have less resistance than endotracheal tubes and are of considerable use for insertion of fiberoptic bronchoscopes. Approximate sizes are:

• 1 for less than 6.5 kg.

• 2 for 6.5-20 kg.

• 2.5 for 20-30 kg.

• 3 for 30 kg and above.

A size 1.5 is also available. The armored versions have reduced risk of kinking and are longer and narrower. The use of the size 1 has not been widespread because of concerns about secure insertion, increased dead space and atelectasis. Although it has been used in neonatal resuscitation, it is not yet recommended for controlled ventilation in small children because of the risk of ventilator impairment from gastric distension.

- If too large a tube is selected, the tracheal mucosa is damaged and the child may develop **post-intubation croup**; if it is **too small**, excessive leak makes effective positive pressure ventilation impossible
- Generally, cuffed tubes are used only in children above the age of 8 years.
- In the case of an awake intubation in a neonate or young infant, adequate preoxygenation may help prevent hypoxemia.

- A **prominent occiput** tends to place the head in a **flexed position** prior to intubation. This is easily corrected by slightly elevating the shoulders with towels. **Straight** laryngoscope blades aid intubation of the anterior larynx in neonates, infants, and young children.
- Mucosal trauma from trying to force a tube through the cricoid cartilage can cause postoperative edema, stridor, croup, and airway obstruction.
- **Inhalational Induction**

- Most children do not arrive in the operating room with an intravenous line in place.
- Modern potent volatile anesthetics can render small children unconscious within minutes. This is usually easier in children who have been **sedated** prior to entering the operating room and who are sleepy enough to be anesthetized without ever knowing what has happened (**steal induction**).
- **Inhalational Induction**
- Equipment appropriate for age and size should be selected.

Typically, the child is coaxed into breathing an odorless mixture of nitrous oxide (70%) and oxygen (30%). Sevoflurane is added to the anesthetic gas mixture in 0.5% increments every **three** to **five** breaths.

- **Sevoflurane** consider the agent of choice for inhalation induction. Single breath induction technique with sevoflurane (7–8% sevoflurane in 60% nitrous oxide) can be used to speed up induction.
- After an adequate depth of anesthesia has been achieved, an intravenous line can be started and a muscle relaxant administered.

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Maintenance

- **Ventilation** is usually controlled during anesthesia of neonates and infants.
- During spontaneous ventilation, even the low resistance of a circle system can become a significant obstacle for a sick neonate to overcome.
- For patients weighing **less than 10 kg, it's** preferred to use the **Mapleson D** circuit or the **Bain system** because of their low resistance and light weight.
- The circle system can be safely used in patients of all ages if ventilation is controlled.
- Anesthesia can be maintained in pediatric patients with the same agents as in adults.

Maintenance

- **Isoflurane** Can be use following a **sevoflurane** induction to help **reduce** the likelihood of postoperative **delirium or agitation on emergence**.
- If **sevoflurane** is continued for maintenance, administration of an **opioid** (eg, fentanyl 1–1.5 mg/kg) 15–20 min before the end of the procedure can reduce the incidence of emergence delirium and agitation.

- Although the MAC is higher in children than in adults, neonates may be particularly susceptible to the cardiodepressant effects of general anesthetics. Nondepolarizing muscle relaxants are often required for optimal surgical conditions.

Perioperative Fluid Requirements

- Fluid therapy can be divided into **maintenance**, **deficit**, and **replacement** requirements.

MAINTENANCE FLUID REQUIREMENTS

Maintenance requirements for pediatric patients can be determined by the **4:2:1 rule**: 4 mL/kg/h for the first 10 kg of weight, 2 mL/kg/h for the second 10 kg, and 1 mL/kg/h for each remaining kilogram.

- The ideal maintenance solution is **0.18% saline** in **4% dextrose** with added **potassium chloride** (**KCl**) 20mmol.l-1 if required)
- Neonates require 3–5 mg/kg/min of a glucose infusion to maintain euglycemia; premature neonates require 5–6 mg/kg/min.

DEFICITS

- In addition to a maintenance infusion, any preoperative fluid deficits must be replaced. Calculated as (**maintenance fluid x starvation hours**) For example, if a **5-kg** infant has not received oral or intravenous fluids for **4 h** prior to surgery, a deficit of 80 mL has accrued (5 kg x 4 mL/kg/h x 4 h).
- Preoperative fluid deficits are typically administered with hourly maintenance requirements in aliquots of **50%** in the **first hour** and **25**% in the **second** and **third** hours. In the example above, a total of 60 mL would be given in the first hour (**80/2** + **20**) and **40** mL in the second and third hours (80/4 + 20).
- **Preoperative fluid deficits are usually replaced with a balanced salt solution (eg, lactated Ringer's injection) or ½ normal saline.**

REPLACEMENT REQUIREMENTS

- Replacement can be subdivided into blood loss and third-space loss.

Blood Loss

- **Blood** loss is typically replaced with non-glucose-containing crystalloid (eg, 3 mL of lactated Ringer's injection for each milliliter of blood lost) or colloid solutions (eg, 1 mL of 5% albumin for each milliliter of blood lost).

LECTURE SEVEN (7)

Anaesthesia for geriatric surgery

Introduction

The incidence of **perioperative complications** is much **higher** in these patients due to **reduced functional reserve** and a **high incidence of co-morbidity**.

but these complications **can be minimized** by:

- **careful** preoperative assessment, a **meticulous** anaesthetic technique and **good** postoperative care.

❖ **Age related physiological changes**

Ageing is a process where progressive cell loss occurs.

- The concept of "**functional reserve**" is derived from the difference between the **basal** level of organ function **at rest** and the maximum level of organ function that can be achieved in response to **increased demand**, for example during exercise or in response to surgical stress.
- **Functional reserve is often reduced in elderly patients**, and is thought to be a **major** factor in the increased morbidity and mortality of the elderly population.

Alterations in Organ Function

Reduction in **cardiovascular**, **pulmonary**, **renal** and **central nervous system** function may be the most important determinants of outcome from surgical procedures under general or regional anaesthesia.

❖ **Cardiovascular system**

- **Ischaemic heart disease** is common in affluent societies. The net effect on the heart is reduced cardiac output.

- In contrast, **valvular heart disease** secondary to **rheumatic fever** is more commonly seen in developing countries.
- Over **50**% of patients will have mitral valve disease. Aortic lesions are less common.
- The reduced cardiac output in heart disease compromises blood flow to the kidneys and brain, and therefore both the kidneys and brain are prone to perioperative ischaemia.
- The physiological response to **cardiovascular stressors** (such as hypovolaemia) may be **blunted** due to reduced baroreceptor sensitivity and autonomic function.
- This lack of compensation may be significant if the patient is taking medication such as **betablockers** or **ACE inhibitors**.
- **Atrial fibrillation** (**AF**) in the elderly population is common. The fast ventricular rate in AF leads to reduced cardiac output. Preoperatively, a patient in AF should ideally be cardioverted, or controlled to <100/minute.
- **Respiratory system**

Pulmonary elasticity, **lung and chest wall compliance**, **total lung capacity (TLC)** will decrease.

- Although **functional residual capacity** (**FRC**) is unchanged, closing capacity (**CC**) rises progressively with age, and may become greater than the FRC - this occurs in the supine position at 44 years of age and in the upright position at 66 years.
- The end result of these changes is airways collapse, VQ mismatch and hypoxaemia.
- The efficiency of gas exchange is reduced, and as a result **PaO2** decreases with age although **PaCO2** remains constant.
- **Atelectasis**, **pulmonary embolism** and **chest infections** are all more common in elderly patients, particularly following **abdominal** or **thoracic** surgery.
- **Early mobilisation** and good **analgesia** following abdominal surgery help **reduce** lung atelectasis and collapse.

❖ **Renal system**

▪ **Glomerular filtration** is reduced. **Clearance** of renally excreted drugs is reduced, and **fluid balance** is more critical as responses to both fluid loading and dehydration are impaired.

▪ Renal function may deteriorate rapidly in hypovolaemic patients. Close monitoring of hourly urine output after major surgery should be routine.

❖ **Nervous system**

▪ An age-related decline in central nervous system (CNS) function is common. As a result, confusion is more common, both pre and post-operatively.

❖ **Endocrine**

The incidence of **diabetes** is increased in the elderly. Diabetics frequently have cardiovascular, renal, neurological and visual impairment, and require control of blood glucose levels during the perioperative period.

❖ **Pharmacology**

▪ **Pharmacokinetics** may be altered, with **reduced hepatic and renal blood flow and a reduction in total body water**. **Plasma proteins** are often reduced, resulting in reduced protein binding of drugs and metabolites, thereby increasing free drug levels and possible toxic effects.

▪ **Pharmacodynamics** may also be altered, with increased sensitivity to many agents, especially CNS depressants.

▪ Minimum alveolar concentration (**MAC**) decreases steadily with age.

▪ Long-term medication should usually be continued throughout the hospital stay.

❖ **Musculoskeletal**

Arthritis usually affect the elderly. This may limit exercise tolerance and makes it difficult to assess fitness.

▪ **Osteoporosis** and **ligament laxity** makes epidurals and spinals technically difficult; in addition, the patient is prone to fractures or dislocation of joints (including the

cervical spine) while anaesthetised. Care should be taken with patient movement and intra-operative positioning. Vulnerable pressure points should be well padded.

PREOPERATIVE PREPARATION

- **Assessment**

✓ **A full history** and thorough **clinical assessment** is required.

✓ An **ECG** is required for all patients.

✓ A **chest X-ray** should be arranged for patients with known malignancy or possible tuberculosis, and for anyone with symptomatic cardiovascular or respiratory disease.

✓ Note the level of cognitive function.

✓ Assessment of **exercise tolerance** and functional ability is important.

✓ The baseline functioning of the patient should be well documented. If a decreased

functional reserve is detected, a high- care or intensive care facility may be appropriate post-operatively.

✓ The American Society of Anaesthesiologists (**ASA**) score should be recorded - it remains a good predictor of outcome in the elderly

Resuscitation/optimisation pre-operatively

✓ Dehydration is common.

✓ Preoptimisation enhance the oxygen delivery to the tissues during the perioperative period, by using fluid therapy, oxygen and possibly inotropic agents.

- **PERIOPERATIVE CARE**

In general the full range of anaesthetic drugs and techniques used for young fit adults may be used in elderly patients, within the limitations of their physiology. Modification of the techniques, and particularly drug doses, may be required.

- **Induction of anaesthesia**

Arm-brain circulation time is **increased**, and induction agent **dose requirements** are **reduced**.

✓ Titrate drugs slowly against effect, and inject into a running intravenous **infusion**.

✓ Thiopentone or propofol are both useful but should be given slowly to avoid overdose.

✓ An induction dose of propofol may result in hypotension and require a vasopressor.

✓ Avoid **ketamine** in the presence of cardiac disease as the tachycardia and hypertension that may result can increase myocardial oxygen consumption and precipitate ischaemia. However, ketamine's hallucinogenic effects are not as marked in the elderly, and that it remains a very safe and effective analgesic, anaesthetic and sedative.

Maintenance of anaesthesia

- **Maintenance of anaesthesia** with **inhalational agents** is a suitable technique for elderly patients.
- Halothane has the advantage of being non-irritant to the upper airway and respiratory tract, although it sensitises the myocardium to catecholamines and so may predispose to tachyarrhythmias.
- **Fluid management**

Careful peri-operative fluid balance is mandatory in the elderly. Always consider measuring the **CVP** with large fluid shifts. Excess fluids in an elderly patient, can cause **pulmonary oedema**. Conversely, dehydration in the elderly can precipitate **renal failure.**

POSTOPERATIVE CARE

Oxygen therapy

- It is good practice to prescribe post-operative oxygen therapy for all elderly patients, and especially following abdominal or thoracic surgery.
- **Nasal cannulae** are often better tolerated than **facemasks**.

High dependency care

High dependency care or **intensive care** facilities may improve the long-term outcome of elderly patients, especially those undergoing urgent or emergency surgery.

Analgesia

Consider prescribing a regular simple analgesic such as paracetamol, and use **NSAID's** with caution; the **complications** of **NSAIDs**, including renal impairment and peptic ulceration, are more prevalent in older patients.

Regional techniques or an IV opioid infusion (with appropriate close supervision) may be the most appropriate method of pain relief.

LECTURE EIGHT (8)

Anesthesia for Gynecology and Obstetric

Specific consideration

- Many patients are apprehensive, even for relatively minor surgery.
- PONV is a particular problem. With high-risk patients, use appropriate techniques; avoid N2O, and give prophylactic antiemetic.
- Pelvic surgery is associated with DVT—ensures that adequate prophylactic measures have been taken.
- Prophylactic antibiotics reduce post-operative wound infection rates for certain operations—check your hospital protocol.
- Vagal stimulation may occur during cervical dilatation, traction on the pelvic organs or the mesentery, or during laparoscopic procedures.
- Take care during patient positioning: Patients are often moved up or down the table, when airway devices can be dislodged and disconnections can occur. Pre-existing back or joint pain may be worsened in the lithotomy position, and, if the legs are supported in stirrups, there is a potential for common peroneal nerve injury.
- It may be reasonable to ask the gynecologist to administer analgesic drugs rectally during anesthesia—ensure that you have the patient's permission to do so.
- During laparotomies, ensure that patients are kept warm.
- During major gynecological surgery, considerable blood loss may occur, and surgery may be prolonged.
- Many gynecological operations formerly done through an open approach (e.g., hysterectomy, tubal pregnancy repair) are now done primarily using laparoscopic techniques.

Specific gynecological operations

Hysteroscopy:

Telescopic equipment enables the gynecologist to inspect the uterine cavity and explore it with accuracy for diagnosis and treatment of intrauterine disease. Before hysteroscopy the cervix is dilated, and this carries the risk of stimulating an autonomic response. The anesthetic for a hysteroscopy includes induction and maintenance with a selection of drugs relevant today – case surgery (e.g., propofol for the passage of a laryngeal mask airway, and either total intravenous or inhalational anesthesia). **Complications** include uterine perforation and bleeding, so facilities to manage these risks should be available. Depending on the type of anesthetic (e.g., opioid) used, prophylactic antiemetic may be indicated.

Laparoscopic surgeries:

Many gynecological operations may be performed laparoscopically, such as; female sterilization, ovarian cystectomy, emergency surgery for ectopic pregnancy, and vaginal hysterectomy. Most of these procedures require longer operating and anesthetic times than open versions of the same procedures, but are less painful after surgery, require a shorter duration of stay in hospital and lead to earlier return to normal activities. A pneumoperitoneum is created, most commonly by insufflating the peritoneal cavity with carbon dioxide

Physiological changes due to pneumoperitoneum:

1) Increased intra – abdominal pressure, reduced both chest wall and lung compliance and also functional residual capacity. These effects are more marked for patients undergoing surgery in the lithotomy posture.

2) Hypoventilation and increased intrapulmonary shunt.

3) Decreased venous return and cardiac output.

4) Increased the ventilation/ perfusion ratio and alveolar dead space.

5) Bradycardia is a common occurrence after peritoneal insufflations, and occasionally asystole occurs.

Hysterectomy and myomectomy:

Hysterectomy

A. Abdominal hysterectomy:

Procedure removal of uterus through abdominal incision (may also include ovaries as bilateral salpingo-oophorectomy**)**

Time 1hr, often longer

Pain +++

Position: supine, head-down

Blood loss 250–500mL,

Practical techniques GA, ETT, IPPV.

B-Vaginal hysterectomy:

Procedure removal of the uterus through the vagina

Time 50min

Pain ++

Position: Lithotomy

Blood loss Variable, usually <500mL

Practical techniques GA or regional: LMA, SV, caudal. Spinal

A myomectomy is indicated for the removal of symptomatic fibroids and can be associated with major blood loss, bleeding can be also a problem during hysterectomy. Prophylaxis for postoperative nausea and vomiting is considered part of the anesthetic technique.

Obstetric Anaesthesia

Physiological Changes During Pregnancy

Parturients undergo remarkable changes during pregnancy, labor, and the immediate postpartum period that can directly affect anesthetic techniques. Introduction of a good anesthetic management depend on understanding of **physiological** and **pharmacological changes** occurs during pregnancy

Respiratory Effects

- **The combination of decreased FRC and increased oxygen consumption promotes rapid oxygen desaturation during periods of apnea.**
- **Preoxygenation** (denitrogenation) prior to induction of general anesthesia is therefore mandatory to avoid hypoxemia in pregnant patients.
- **Capillary engorgement** of the respiratory mucosa during pregnancy predisposes the upper airways to trauma, bleeding, and obstruction.
- Gentle laryngoscopy and smaller endotracheal tubes (6–6.5 mm) should be employed during general anesthesia.

Respiratory	
Oxygen consumption	+20 to 50%
FRC	−20%
Minute ventilation	+50%
Tidal volume	+40%
Respiratory rate	+15%
Pao_2	+10%
$Paco_2$	−15%
HCO_3	−15%

Cardiovascular Effects

❖ **Cardiac output** and **blood volume increase** to meet accelerated maternal and fetal metabolic demands.

❖ An increase (**55**%) in **plasma volume** in excess of an increase in **red cell** mass (**45**%) produces **dilutional anemia** and **reduces blood viscosity**.

❖ At term, **blood volume** has increased by **1000–1500 mL** in most women, allowing them to easily tolerate the blood loss associated with delivery; total blood volume reaches **90** mL/kg.

❖ Average blood loss during **vaginal** delivery is 400–500 mL, compared with 800–1000 mL for a **cesarean** section.

❖ Approximately **5**% of women at term develop the supine hypotension syndrome (aortocaval compression), which is characterized by hypotension, pallor, sweating, or nausea and vomiting.

❖ This because complete or near-complete occlusion of the inferior vena cava by the gravid uterus. And can readily produce fetal asphyxia. Turning the patient on her side restores venous return from the lower body and corrects the hypotension in such instances. This maneuver doing by placing a wedge (>15°) under the right hip.

Hematological Effects

❖ Pregnancy is associated with a hypercoagulable state that may be beneficial in limiting blood loss at delivery.

❖ Fibrinogen and concentrations of factors VII, VIII, IX, X, and XII all increase.

Gastrointestinal Effects

Gastroesophageal reflux and esophagitis are common during pregnancy. This factor places the parturient at **high risk for regurgitation and pulmonary aspiration.**

Hepatic Effects

A 25–30% decrease in **serum pseudocholinesterase** activity is also present at term but rarely produces significant prolongation of succinylcholine's action

Summary of anatomical and physiological changes of pregnancy:

1) Increased basal metabolic rate (hypoxia occurs faster).
2) Enlarging abdominal mass and decreased sphincter tone (regurgitation, aspiration).
3) Reduced functional residual capacity (reduced oxygen reserve on preoxygenation).
4) Altered air way anatomy (failure to intubation).
5) Increased blood flow to uterus/placenta (potential for hemorrhage).
6) Enlarged uterus (uterine atony and bleeding; aortocaval occlusion/supine hypotension)

Challenges in obstetric procedures:

A) Aortocaval occlusion (supine hypotension) syndrome: It is also called aortocaval compression syndrome, it is the compression of the enlarging uterus on the major vessel in the abdomen when a pregnant woman lies in the supine position.
It is characterized by pallor, tachycardia, sweating, nausea, hypotension and dizziness. It is dangerous and may be fatal.
B) Aspiration of stomach contents:
Regurgitation (passive) and vomiting (active) may result in aspiration of liquids or solids. **Preparation for anesthesia begins with**:
1) A policy of nil by mouth for at least 4 hours, if not longer – prior use of opioids can delay stomach emptying.
2) Reduction in gastric acid production (e.g. oral ranitidine 150 mg twice daily started the night before anesthesia, or I.V ranitidine 50 mg slowly 2 hours before anesthesia).

3) Neutralization of any acid produced (e.g. clear alkaline solution such as 0.3 mmol/ L sodium citrate 30 ml given just before anesthesia).

4) Increasing lower esophageal sphincter tone. (E.g. metoclopramide 10mg I.V).

C) Failed intubation:

Pregnancy is a contributing factor to failed intubation because it results in an increase in breast size, which impedes laryngoscopy, an increase in soft tissue mass around the airway making it more difficult to visualize the larynx, and an increase in metabolic rate and decrease in the reservoir of oxygen in the functional residual capacity after preoxygenation, thus increasing the risk of hypoxia if the lungs are not ventilated.

At caesarean section, if intubation fails the mother is awakened and a regional nerve block is considered.

Regardless of the time of last oral intake, all patients are considered to have a full stomach and to be at risk for pulmonary aspiration.

The supine position should be avoided unless a left uterine displacement device (>15° wedge) is placed under the right hip.

Anesthesia for Labor & Vaginal Delivery

- **Meperidine** (Pethidine), a commonly used opioid, can be given in doses of 10–25 mg intravenously or Intravenous fentanyl, 25–100 mcg/h, has also been used for labor.
- **Morphine** is **not** used because it appears to cause greater respiratory depression in the fetus than meperidine
- Low-dose intravenous **ketamine** is a powerful analgesic. In doses of 10–15 mg intravenously, good analgesia can be obtained in 2–5 min without loss of consciousness.

Regional Anesthetic Techniques

❖ **Epidural** or intrathecal techniques (**spinal**), alone or in combination, are currently the most popular methods of pain relief during labor and delivery. They can provide excellent analgesia while allowing the mother to be awake and cooperative during labor.

Anesthesia for Caesarean section

Regional anesthesia for Caesarean section was initially driven by maternal preference. It was subsequently found that regional anesthesia is also safer than GA.

- **Advantages of regional anesthesia**

• Both mother and partner can be present at the delivery

• Minimal risk of aspiration and lower risk of anaphylaxis.

• The neonate is more alert, which promotes early bonding and breastfeeding.

• Fewer drugs are administered, with less 'hangover' than after GA.

• Better post-operative analgesia and earlier mobilization.

Spinal anesthesia

Is the most commonly used technique for elective Caesarean sections.

It is rapid in onset, produces a dense block, and, with intrathecal opioids, can produce long-acting post-operative analgesia. However, hypotension is much commoner than with epidural anesthesia

Technique:

✓ History/examination/explanation and consent.

✓ Ensure that antacid prophylaxis has been given.

✓ Establish 16G or larger IV access.

✓ Start crystalloid co-load.

✓ Position the patient:

A sitting position usually makes finding the midline easier, which may be helpful with obese patients, and may be associated with a faster onset, although the height of block is less predictable. A lateral position is associated with a slower onset of block, particularly if a full lateral position is maintained until the block has fully developed.

✓ Perform spinal anesthetic at L3/4 interspace, using a 25G or smaller pencilpoint needle. After injection of the solution, move the woman to a supine position with a left lateral tilt or wedge. If supine hypotension occurs, increase the tilt, or, if severe, temporarily move the woman to a full lateral position.

General anesthesia

Elective GA is now uncommon, limiting opportunities for training. The majority of complications relate to the airway. Failed intubation is much more frequent in obstetric than non-obstetric anesthesia. All obstetric theatres should have equipment to help with the difficult airway, and all obstetric anesthetists should be familiar with a failed intubation drill.

Indications for GA include:

• Maternal request.

• Urgent surgery (in experienced hands and with a team that is familiar with rapid regional anesthesia, a spinal or epidural top-up can be performed almost as rapidly as a GA).

• Regional anesthesia contraindicated (e.g. coagulopathy, maternal hypovolemia).

• Failed regional anesthesia.

• Additional surgery planned at the same time as a Caesarean section.

Technique:

History and examination. In particular, assess the maternal airway—mouth opening, Mallampati score, thyromental distance, neck mobility

• Antacid prophylaxis

• Start appropriate monitoring.

• Position supine with a left lateral tilt or wedge.

• Preoxygenate for 3–5min or, in an emergency, with 4–8 VC breaths with a high flow through the circuit. Ensure a seal with the face mask

• Perform RSI with an adequate dose of induction agent (e.g. 5–7mg/ kg of thiopental

• A 7.0mm ETT is adequate for ventilation and may make intubation easier.

• Propofol has also been used for Caesarean section, without any major reported complications, although, at present, thiopental is still the most commonly used agent in the UK.

• Ventilate with 50% O2 in N2O. If severe fetal distress is suspected, then 75% O2 or higher may be appropriate. Maintain ETCO2 at 4.0–4.5kPa (30–34 mmHg).

• Use 'overpressure' of the inhalational agent to rapidly increase the end-tidal concentration of the anesthetic agent to at least 0.75 MAC (e.g. 2% isoflurane for 5min, then reduce to 1.5% for a further 5min).

• At delivery: Give 2–5IU of oxytocin IV bolus. If tachycardia must be avoided, then an IV infusion of 30–50IU of oxytocin in 500mL of crystalloid, infused over 4hr, is effective

Administer opioid (e.g. 10–15mg of morphine ± 100 micrograms of fentanyl), IV paracetamol, and IV diclofenac (unless contraindicated)

• If a woman has eaten shortly before surgery, consider passing a large-bore orogastric tube to empty the stomach before extubation.

• Extubate awake. Be aware that extubation is a high-risk time.

• Give additional IV analgesia, as required

Recovery:

Be aware that recovery units are potentially dangerous places for mothers after GAs, particularly if the recovery is staffed by midwives who may be less familiar with airway care. The same standard of recovery staff should be available to women on labour wards as in a normal theatre recovery unit.

LECTURE NINE (9)

Anesthesia of Orthopedic surgery

Common Anaesthetic Considerations

The common Anaesthetic considerations for Orthopedic surgery may be related to:

1.Trauma association: some of orthopedic patients presence with other injuries due to trauma, and carry risks of **emergency surgery** (e.g. **aspiration of gastric contents**, **internal bleeding).**

2.Musculoskeletal disease, some patients have musculoskeletal disease need **Orthopaedic surgery** e.g. **rheumatoid arthritis RA**, **connective tissue diseases, muscular abnormalities.**

Patients with rheumatoid arthritis (RA) specifically require orthopedic surgery need special attention. because RA multisystem disease need special considerations include: -

A. **Pulmonary system e.g. pulmonary fibrosis**
B. **Cardiac system: - coronary artery disease. Myocarditis**
C. **Musculoskeletal systems: - atlentooccipital subluxation**
D. **Hematological system: -anemia, platelet dysfunction**
E. **Endocrine system adrenocortical impairment**

▪ **Airway management** in patients has **RA** can be challenging in these patients **because** RA involvement of the **cervical spine and temporomandibular joints results in limited neck range of motion and mouth opening.** Also Patients with RA on **chronic steroid therapy** may require perioperative steroid replacement.

3 - MH (malignant hyperthermia) There is a higher than normal incidence of **MH** susceptibility in young patients with musculoskeletal abnormalities .
4 – Risk of congenital malformations: may be accompanied by other system involvement, in **Orthopaedic surgery** and other injury e.g. cardiac lesions.

5 – Hyperkalemia: - there is a risk of massive **hyperkalemia** following **Suxamethonium** if neurological or muscle lesions are present. **Hyperkalemia may lead to cardiac arrest.**

6 - Tourniquets use

✓ Use of a pneumatic tourniquet on an extremity creates a bloodless field and decrease blood loss during surgery.

✓ The pressure in the arterial tourniquet should, in all cases, exceed arterial pressure. For the lower limb, this pressure is typically 300 mmHg (or 150 mmHg above systolic arterial pressure) and for the upper limb, 250 mmHg (or 100 mmHg above systolic arterial pressure)

✓ The maximum period of safe ischemia is not known precisely. Lasting damage is unlikely if a tourniquet time of 90 for upper limb and 120 minutes for lower limb is not exceeded.

✓Tourniquet on more than one limb should never be deflated (or inflated) simultaneously.

✓ **tourniquets Inflation can produce potential clinical problems including: -**

A- Hemodynamic changes: - this is because of a rapid shift of blood volume into the central circulation. This is well tolerated in normal patients but in patients with noncompliant ventricles and diastolic dysfunction may be disaster.

B- Tourniquets pain, Tourniquet pain start gradually beginning approximately **1 h** after cuff inflation and becomes so severe over time which presented by Signs of

progressive sympathetic activation include marked hypertension, tachycardia, and diaphoresis.

C- Arterial Thromboembolism, and pulmonary embolism. Tourniquet-by induced ischemia of a lower extremity may lead to the development of deep venous thrombosis.

D- Muscle dysfunction: - Prolonged inflation **(>2 h**) routinely leads to transient muscle dysfunction from ischemia and may produce rhabdomyolysis or permanent peripheral nerve damage. Tourniquet inflation has also been associated with increases in body temperature in pediatric patients undergoing lower extremity surgery.

7- bone cement: **Cement implantation syndrome** due to Systemic absorption of residual methylmethacrylate monomer can produce vasodilation and trigger platelet aggregation, microthrombus formation in the lungs, and cardiovascular instability.

The clinical manifestations of bone cement implantation syndrome include

A- Hypoxia (increased pulmonary shunt),

B- Hypotension,

C- Arrhythmias (including heart block and sinus arrest),

D- Pulmonary hypertension (increased pulmonary vascular resistance), and decreased cardiac output.

Treatment strategies for this complication include

A- Increasing inspired oxygen concentration prior to cementing,

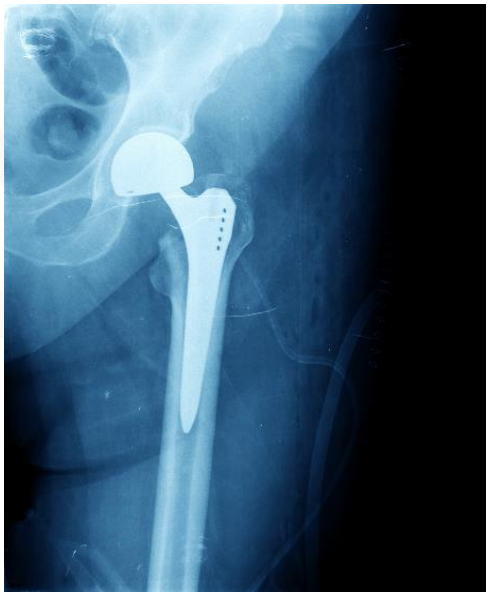
B- Maintain euvolemia,

C- Creating a vent hole in the distal femur to relieve intramedullary pressure,

D- Using a femoral component that does not require cement.

8.DVT (deep venous thrombosis) and PE(pulmonary embolism) are common, especially after hip surgery; and can cause morbidity and mortality following orthopedic operations on the pelvis and lower extremities. Risk **factors of DVT and PE include**

A. **Obesity,**
B. **Age greater than 60 years,**
C. **Procedures lasting more than 30 min,**
D. **Use of a tourniquet,**
E. **Lower extremity fracture.**
F. **Immobilization for more than 4 days.**



Positioning

Orthopedic surgery often requires the use of unusual positions, some of which carry risks of nerve damage, soft tissue ischemia, electrical and thermal injury and joint pain. Care must be taken in protecting areas at risk of injury. Forceful movement of the patient by the surgeon is often inevitable during orthopedic surgery. When such movement occurs, it is advisable to re-check the patient's position ensuring that soft tissues, nerves, eyes, airway connections and venous access are safe.

Although some procedures may be performed under regional anesthesia alone, long operations may result in significant discomfort related to posture. Some positions adopted during orthopedic surgery are associated with venous air embolism, which occurs when large veins are open to air, particularly, when venous pressure is low. These postures include the **lateral position for hip surgery**, the **sitting position** for shoulder surgery and the **prone position** for spinal surgery.

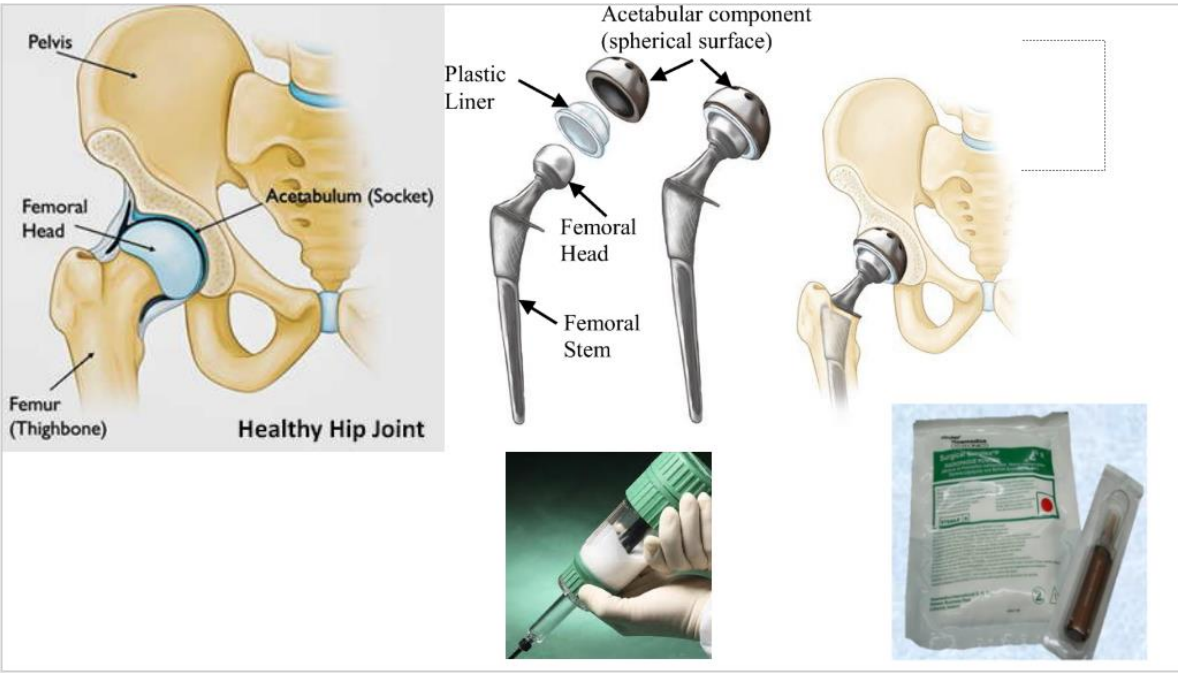
ANAESTHESIA FOR HIP REPLACEMENT

- Hip replacement can be performed under general, spinal or epidural anaesthesia, and a combination of techniques is often used.
- The advantages of regional techniques include:

· Reduced blood loss, reducing the need for transfusion
· Avoids effects of general anaesthesia on pulmonary function
· Avoid intubation
· Good early postoperative analgesia
· Reduced incidence of postoperative venous thrombosis and pulmonary embolism

- The advantages of general anaesthesia include:

· Easier for patients that cannot tolerate lying flat
· Safer in patients with fixed output states like aortic stenosis, where maintenance of normal sinus rhythm, heart rate and intravascular volume is critical.
· Patient preference



Spinal anaesthesia

- A simple **THR** is particularly **amenable** to spinal anaesthesia and this can be supplemented with **sedation** or **general anesthesia**.
- **Target-Controlled-Infusion (TCI) propofol** is useful sedation for the lateral position, using facemask supplemental oxygen. Intermittent doses of **midazolam**, also can be used.
- For the **supine position** in a patient who wishes to be asleep during surgery, consider an **LMA** with a **light GA** to maintain the airway.
- The addition of **intrathecal opioid** helps cover the longer duration of surgery necessary for a more complex primary hip replacement.
- It is a **suitable technique** for up to **3 hours** of surgery. Alternatively, or for longer cases, a combined **spinal/epidural** technique can be used.

- **GA** (rather than sedation) may be combined with an epidural for any **complex primary operation** because of the prolonged surgical time. An **LMA**, or **endotracheal** tube and **IPPV**, may be considered.
- **Intraoperative**
- Inserting a urinary catheter will help to monitor fluid balance.
- Aim to **maintain blood pressure** at an adequate level based on preoperative readings. In **elderly** patients with **vascular disease** **hypotension** should be treated immediately.
- Intra-operative **antibiotic** prophylaxis will be required.
- Ensure **adequate** IV loading prior to cementing of femoral component.
- **Hypotension** can occur on **pressurisation** of the cement into the femur, **usually due to vasodilatation and direct myocardial depression from the monomer.**
- The **transient hypotension** does not correlate with the level of monomer in the circulation, but with deficit in blood volume.

Postoperative

- The **surgeon** usually **prefers** the patients to be placed on their bed in the **supine position** with the legs **abducted** using a pillow to prevent dislocation of the prosthesis.
- Patients are usually mobilized at **24-48 hours** and simple IM/ subcutaneous opioids with regular paracetamol or NSAIDs are usually sufficient for postoperative analgesia in a simple THR.
- If an epidural has been inserted, a postoperative infusion can be used but needs to **cease** prior to mobilization.

PCA is a suitable alternative if pain relief is needed for an extended period.

LECTURE TEN (10)

Anesthesia for obese patient

Introduction

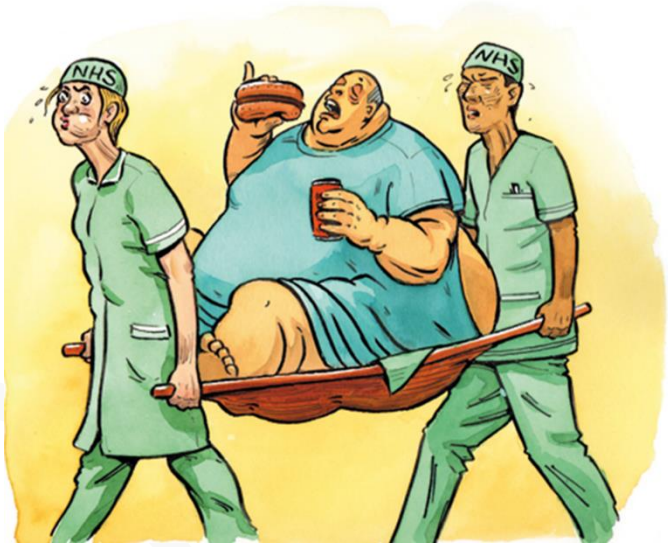
- **Obesity** (body weight of 20% or more above ideal weight) is associated with increased morbidity and mortality and a wide spectrum of medical and surgical diseases.
- Risk of premature death is doubled in the obese population and risk of death resulting from cardiovascular disease is increased fivefold in the obese compared with the nonobese.
- **Obesity** is a global health problem and the prevalence varies with socio-economic status.
- **Morbid obesity** is defined in terms of body mass index (BMI).
- BMI is calculated by establishing a ratio between the patient's **weight** and **height** as follows: **Body mass index (BMI) = weight in kg/height in m2**
- BMI values are classified as follows:

- **BMI of 18.5–24.9 = normal**
- **BMI of 25.0–29.9 = overweight**
- **BMI of 30.0–34.9 = class I obesity**
- **BMI of 35.0–39.9 = class II obesity**
- **BMI of 40.0 or greater = class III obesity (sever or morbid obesity)**

▪ Interestingly, the regional distribution of excess fat is thought to be more predictive than BMI for morbidity and mortality.

▪ Excessive abdominal fat, "**central obesity**" is particularly predictive for NIDDM, dyslipidaemia and cardiovascular disease.

- Waist circumferences need to be sex and race specific.



Pathophysiology

Obesity is a complex, multifactorial disease (mechanisms of fat storage, genetic, psychologic). Most simply, it occurs when net energy intake exceeds net energy expenditure over a prolonged period of time.

Cardiovascular Disorders

a. Systemic Hypertension. Obesity-induced hypertension is related to the effects of hyperinsulinemia on the sympathetic nervous system and extracellular fluid volume. Insulin also activates adipose tissue to release angiotensinogen. Increased circulatory cytokines cause vascular damage and fibrosis, increasing arterial stiffness. Left ventricular (LV) hypertrophy can develop. Pulmonary hypertension is common and most likely reflects the impact of chronic arterial hypoxemia or increased pulmonary blood volume (or both). Weight loss significantly improves, or even resolves, hypertension

b. Coronary Artery Disease. Obesity seems to be an independent risk factor for the development of ischemic heart disease and is more common in individuals with central /android fat distribution.

c. Congestive Heart Failure Systemic hypertension causes concentric LV hypertrophy and a progressively noncompliant left ventricle, which, when combined with hypervolemia, increases the risk of congestive heart failure. Obesity-induced cardiomyopathy is associated with hypervolemia and increased cardiac output. Insulin resistance also appears to play a role via cardiac steatosis, lipoapoptosis, and activation of cardiac genes that promote LV remodeling. Some of these structural and functional changes may reverse with significant weight loss.

Respiratory disorders

Since **obesity** is a **multisystem disease** affecting all organs, there are a number of implications relevant to the conduct of anaesthesia:

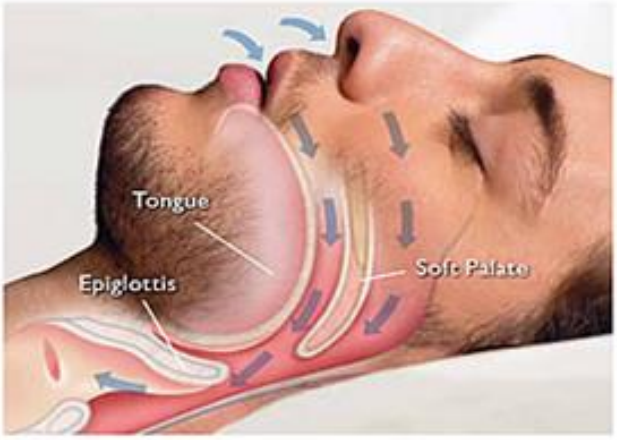
a. **Lung Volumes**. Obesity imposes a restrictive ventilatory defect because the weight of the thoracic cage and abdomen impedes the motion of the diaphragm and decreases functional residual capacity (FRC), especially in the supine position. These changes are accentuated by general anesthesia and impair the ability of obese patients to tolerate apnea (i.e., during direct laryngoscopy), resulting in rapid arterial oxygen desaturation after induction of anesthesia, often despite preoxygenation.

b. Gas Exchange and Work of Breathing. Paco2 and ventilatory response to carbon dioxide remain within a normal range in obese patients. Normocapnia is maintained by increased minute ventilation, resulting in increased work of breathing.

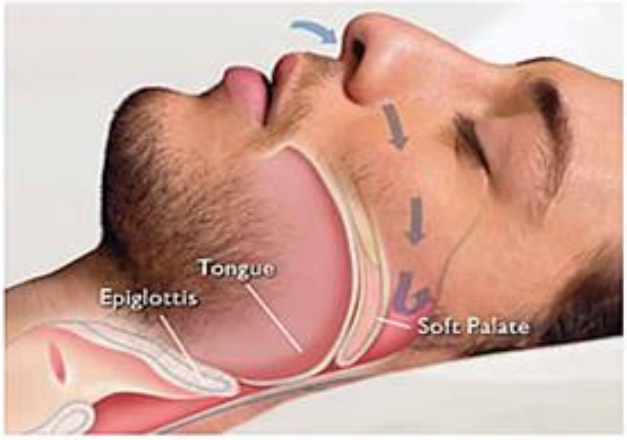
c. Lung Compliance and Resistance. Obesity is associated with a decrease in lung compliance and an increase in airway resistance that result in rapid, shallow breathing patterns and increased work of breathing that is most marked in the supine position.

d. Obstructive Sleep Apnea (OSA). OSA is cessation of breathing for more than 10 seconds during sleep, and hypopnea is a reduction in the size and number of breaths compared with normal breathing.

- ✓ At least 5% of morbidly obese patients will have OSA
- ✓ is caused by passive collapse of the pharyngeal airway during deeper planes of sleep, resulting in snoring and intermittent airway obstruction.
- ✓ hypoxemia and hypercapnia results in arousal and disruption of quality sleep thus causing the characteristic daytime somnolence.
- ✓ Pulmonary and systemic vasoconstriction, polycythemia, and right ventricular failure all occur and can cause type II respiratory failure
- ✓ treatment includes removal of precipitants, weight loss and nocturnal CPAP.



Normal breathing
During sleep, air can travel freely to and from your lungs through your airways.



Obstructive Sleep Apnoea
Your airway collapses, stopping air from traveling freely to and from your lungs and disturbing your sleep.

▪**Specific Anaesthetic considerations:**

✓ Avoid sedative premedication (difficult to maintain airway)

✓ Airway obstruction is very likely to occur in the postoperative period (give oxygen and apply CPAP if required)

✓ Regional techniques and short acting anaesthetic agents are ideal to reduce postoperative drowsiness.

✓ Consider nocturnal oxygen for up to 5 days following major surgery if available.

✓ Regional anesthesia plus noninvasive mechanical ventilation represented the preferred techniques for obese patient with respiratory problems.

Airway

▪Obese patients tend to have short, fat necks making both mask ventilation and direct laryngoscopy technically more challenging.

▪A high BMI is associated with increased risk of difficult intubation.

▪**Specific Anaesthetic considerations:**

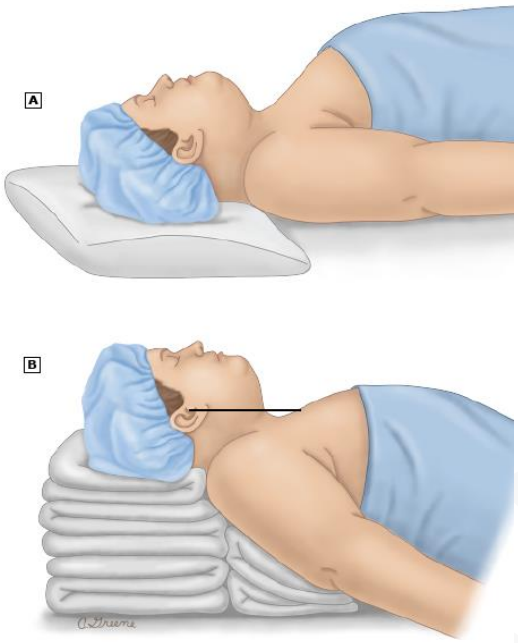
✓ Always assess the airway for prediction of difficult intubation.

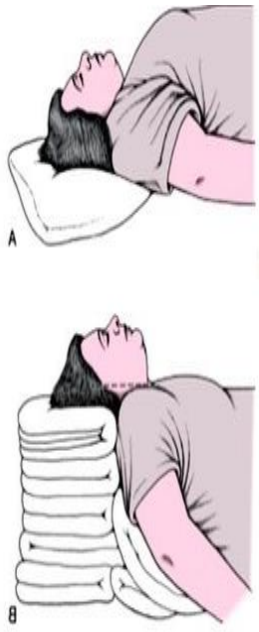
✓ Difficult mask ventilation can sometimes be transformed by placement of an oral airway; typically, laryngeal mask airways (LMAs) are used for this purpose.

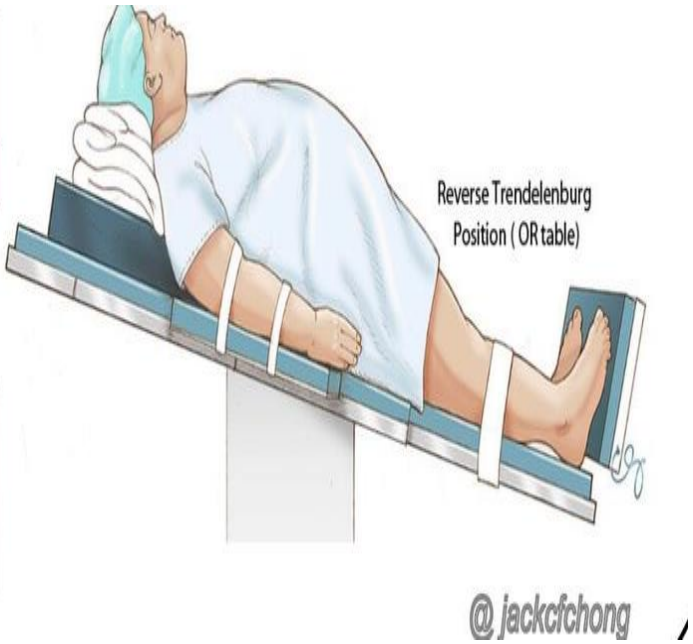
✓ Obese women are more likely to have large breasts, which can interfere with easy placement of the laryngoscope, therefore aim for a degree of head-up tilt, and if necessary, apply traction on the breasts to allow placement of the laryngoscope.

✓ LMAs and other supraglottic airway devices remain relatively contraindicated for elective use in MO patients, but are acceptable choices for emergency use.

✓ For intubation, **ramps** are recommended to achieve optimal sniffing position. These ramps are created by placing folded blankets under the patient's shoulders, neck, and occiput. The idea is to bring the patient's **chin** to a **higher** point than the **chest**.







Inflammatory Syndrome of Obesity

Obese patients have a higher rate of perioperative infections, which may be caused by impaired neutrophil function secondary to secretion of proinflammatory cytokines by adipocytes**.**

Thromboembolic Disorders

The risk of deep vein thrombosis in obese patients undergoing surgery is approximately double that of nonobese individuals due to polycythemia, increased intraabdominal pressure, and immobilization. Risk of stroke is also increased. For

every 1 unit increase in BMI above normal, there is a 4% increase in risk of ischemic stroke and 6% increase in risk of hemorrhagic stroke.

Anesthesia management

1. Preoperative Evaluation. The focus is on cardiovascular and respiratory systems and airway evaluation.

a. History. The history should focus on symptoms such as chest pain, syncope, exertional dyspnea, and symptoms suggestive of OSA. Other concerns include the presence of GERD, control of systemic hypertension, and the presence of diabetes (diagnosed or previously undiagnosed).

b. Physical Examination. Any signs of respiratory or cardiac compromise should be identified, and a detailed assessment of the airway performed. Intravenous access should be assessed.

c. Preoperative Diagnostic Testing. Tests may include electrocardiography (looking for signs of right heart strain or right or left ventricular hypertrophy), chest x-ray examination if congestive heart failure is suspected, transthoracic echocardiography to evaluate left and right ventricular function and pulmonary hypertension, and arterial blood gases if severe OSA is suspected.

d. Home Medications. Most medications should be continued perioperatively, with the exception of oral hypoglycemics, angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, anticoagulants, and nonsteroidal anti-inflammatory drugs. Proton pump inhibitors should be taken the morning of surgery, and deep venous thromboembolism prophylaxis should be considered (low-molecular-weight heparin or unfractionated heparin).

e. Continuous Positive Airway Pressure (CPAP) or Bi-Level Positive Airway Pressure (BiPAP). If such treatment is used at home, the patient should bring the mask so that this therapy can be continued in the perioperative period.

2- Intraoperative Management

a. Positioning. Specially designed operating room tables may be needed, and special transfer devices (such as air transfer mattresses) can minimize the risk of injury to patient or staff. “Ramping” the patient may allow better ventilator mechanics. Pressure points require special attention. Neutral arm position is preferred when possible.
b. Laparoscopic Surgery. Pneumoperitoneum during laparoscopy causes physiologic changes that may be accentuated in obesity (increased venous return if abdominal pressure is 10 mm Hg or less, and decreased venous return with abdominal pressure 20 mm Hg or more with decreased cardiac output; hypercarbia, acidosis, and increased pulmonary vascular resistance; higher myocardial oxygen demand). Trendelenburg positioning may further compromise ventilation.

c. Choice of Anesthesia. **Local or regional anesthesia is preferable to general anesthesia if feasible.**

i. Regional Anesthesia. In obese patients' regional anesthesia may be technically difficult, as bony landmarks are obscured. Local anesthetic requirements for spinal and epidural anesthesia in obese patients may be as much as 20% lower than in nonobese patients.

ii. Regional Anesthesia

A-premedication

Use of benzodiazepines is controversial owing to risk of upper airway obstruction.

(b) Airway Management.

Difficulties with mask ventilation and tracheal intubation may occur (because of fat face and cheeks, short neck, large tongue, excessive palatal and pharyngeal soft tissue, restricted mouth opening, limited mandibular mobility, or large breasts). Emergency airway equipment should be readily available. Rapid decreases in arterial oxygenation

may be seen with direct laryngoscopy and tracheal intubation, and adequate preoxygenation is critical. Awake intubation may be a consideration in some patients. Obese patients are traditionally presumed to be at increased risk of pulmonary aspiration during the induction of anesthesia, but the risk of pulmonary aspiration is related to difficult tracheal intubation rather than to BMI. Use of a laryngeal mask airway (LMA) is successful in aiding intubation in 96% of obese patients and is successful in establishing ventilation in less than 1 minute in almost 100% of obese patients.

(c) Management of Ventilation.

Controlled ventilation using large tidal volumes is often applied in an attempt to offset the decreased FRC. Positive end-expiratory pressure may improve ventilation/perfusion matching and arterial oxygenation in obese patients, but adverse effects on cardiac output and oxygen delivery may offset these benefits. Patients should be maintained in a semi-upright position during spontaneous ventilation during emergence.

Pharmacokinetics.

The dosage of injectable drugs is hard to predict. Total blood volume may be increased, but fat has relatively low blood flow, and doses based on total weight may be relatively excessive. In general, loading doses should be based on lean body weight. Lean body weight is total body weight minus fat weight. In clinically severe obesity, lean body weight accounts for 20% to 40% of excess body weight. Cardiac output is increased, which can affect drug distribution after intravenous injection. Repeat injections should be based on pharmacologic response but can result in cumulative drug effects owing to storage of drug in fat. Liver dysfunction may decrease drug clearance from the circulation. Awakening of obese patients is more prompt after exposure to desflurane or sevoflurane than after administration of either

isoflurane or propofol. Ketamine and dexmedetomidine may be useful anesthetic adjuncts for patients who are susceptible to narcotic-induced respiratory depression.

Monitoring.

The technical difficulty of placing intravenous catheters and invasive monitors may be increased by the presence of obesity. Invasive arterial monitoring should be used for the morbidly obese with severe cardiopulmonary disease and for those patients in whom a poor fit of the noninvasive blood pressure cuff is likely because of the severe conical shape of the upper arms or unavailability of appropriately sized cuffs. Transesophageal echocardiography and/or pulmonary artery catheterization may be needed in patients with CHF or pulmonary hypertension. For surgeries performed with the patient under local or regional anesthesia, capnography is recommended to decrease the risk of undetected airway obstruction.

Fluid Management.

Fluid management should be based on lean body weight. Urinary output during laparoscopic surgery does not necessarily reflect volume status.

3. Postoperative Management

a. **Extubation**. When obese patients are fully recovered from the depressant effects of anesthetics, extubation is considered. Ideally, obese patients should recover in a head-up to sitting position. A history of OSA or obesity hypoventilation syndrome mandates intense postoperative monitoring to ensure maintenance of a patent upper airway and acceptable oxygenation and ventilation.

b. **Transport**. Transport should occur with the patient awake, in a semi-upright position, and receiving supplemental oxygen.

c. **Postoperative Analgesia**. Opioid depression of ventilation in obese patients is a concern, and the intramuscular route of administration may be unreliable owing to the unpredictable absorption of drugs. Patient-controlled analgesia or neuraxial

opioids are commonly used.

Nonsteroidal anti-inflammatory agents may reduce narcotic requirements.

Intravenous acetaminophen has been recently approved by the FDA. Ketamine and dexmedetomidine may be useful. Patients with OSA are at risk for development of postoperative hypoxemia. Adequacy of ventilation should be assessed for 24 to 48 hours postoperatively.

d. Respiratory Monitoring and Management.

Adequacy of ventilation should be assessed for 24 to 48 hours postoperatively. CPAP or BIPAP, if used at home, should be resumed.

e. Discharge to an Unmonitored Setting.

When pain is controlled and patient is no longer at significant risk of postoperative respiratory depression, he or she may be discharged to an unmonitored setting.

f. Postoperative Complications.

Wound infection is twice as common in obese as in nonobese patients. Mechanical ventilation may be required in patients who have a history of CO2 retention. Risks associated with OSA may extend for several days into the postoperative period. Likelihood of deep vein thrombosis and pulmonary embolism is increased.