



Anaesthesia

For Undergraduate

Anesthesia Department
King Saud University

Clinical Anesthesia for undergraduate

Introduction

The scope of anaesthetic practice

Whilst the perioperative anaesthetic care of the surgical patient is the core of specialty work many anaesthetists have a much wider scope of practice which may include:

- The preoperative preparation of surgical patients
- The resuscitation and stabilization of patients in the Emergency Department
- Pain relief in labour and obstetric anaesthesia
- Intensive care medicine
- Transport of acutely ill and injured patients
- Pre-hospital emergency care
- Pain medicine including:
 - The relief of post-operative pain
 - Acute pain medicine and the management of acute teams
 - Chronic and cancer pain management
- The provision of sedation and anaesthesia for patients undergoing various procedures outside the operating theatre. Examples of this include different endoscopic procedures, interventional radiology and dental surgery [this list is not exclusive]

The Department of Anesthesia at the King Saud University is involved in providing the Undergraduate Education Program for Year 4 Anesthesia Rotation. The Anesthesia Rotation occurs as two weeks rotation.

This rotation should provide the following learning outcomes :

- a) The role of anaesthetist in preoperative assessment .
- b) The basic airway and circulatory management in patients under anaesthesia .
- c) Describe the principles of applied Anatomy, physiology and pharmacology with regards to Anesthesiology.
- d) The basic role of emergency room settings and how to manage common emergency room cases.
- e) Acquire some clinical skills in patient assessments and to demonstrate competence in the performance of basic technical procedures related to anesthesia practice.
- f) Basic management of fluids and electrolytes in patients undergoing anesthesia.
- g) Anesthesiologists are also involved in taking care of patients with acute and chronic pain syndromes in pain management clinics, ambulatory care and research process.

The principal sub-disciplines of the practice of anesthesiology include, cardiothoracic anesthesia, ambulatory anesthesia, obstetric anesthesia, neuroanesthesia, pediatric anesthesia, regional anesthesia, critical care medicine and acute and chronic pain management.

Standards and protocols

Three important and illustrative protocols are described here:

- The American Society of Anesthesiologists' standards for basic anesthetic monitoring apply to all anesthesia care, The key elements are as follows:
 - Qualified anesthesia personnel shall be present in the room throughout the course of all general anesthetics, regional anesthetics, and monitored anesthesia care.
 - Continually monitoring of the patient ,ventilation , circulation and temperature
 - Communication with surgeons.

Preoperative evaluation

The overall goals of the preoperative assessment are to reduce perioperative morbidity and mortality and to allay patient anxiety.

Preanesthesia evaluation is designed to provide patients an opportunity to discuss the anesthetic plan before surgery with their anesthesiologist. In the preoperative clinic an anesthesiologist will evaluate the medical condition of all patients, and in conjunction with the patient, will formulate a plan for the Perioperative anesthetic care. Special emphasis is given to airway evaluation, cardiopulmonary status, liver and kidney disease and any necessary labs, chest X-rays and ECG are performed in the clinic.

The patients' old charts are reviewed for any previous anesthesia related problems, and for comorbidities, including hypertension, coronary artery disease, pulmonary disease, diabetes, renal and hepatic disease, neurological disease, and among others social habits, Previously treated medical conditions are reviewed thoroughly, as is the patients' surgical history.

The Medical History:

The main objective of good medical history is to uncover and assess the severity of any pathologic condition that would influence the selection of intraoperative techniques, monitoring and anesthetics. The focus should be on the preanesthetic problem areas pertaining to history, physical examination and the surgical condition. The medical problems should be characterized by date and time of onset, severity, functional limitation due to the medical condition and response to therapy. Other important areas of emphasis are, allergies, the current medication list and the review of systems.

Cardiac History

Recent onset of chest pain, severity of chest pain, history of myocardial infarction, exercise tolerance and response to treatment. History of hypertension (controlled or uncontrolled), valvular heart disease, symptomatic dysrhythmias and patients' functional status is assessed according to the New York Heart Association classification.

New York Heart Association classification

Class I: Cardiac disease without limitation of physical activity

Class II: Slight limitation of physical activity

Ordinary physical activity results in angina or fatigue

Class III: Marked limitation of physical activity

Class IV: Angina at rest, increased with activity .

HISTORY, SYMPTOMS AND SIGNS ASSOCIATED WITH HIGH PERIOPERATIVE CARDIOVASCULAR RISK

- Myocardial infarction within the past 6 months
- Poor left ventricular function
- Poorly controlled cardiac failure
- Resting diastolic blood pressure > 110 mmHg
- Poorly controlled/untreated arrhythmia
- Age > 70
- Significant aortic stenosis

Pulmonary History

History of shortness of breath, asthma, COPD, emphysema and smoking history.

Recent upper respiratory tract infections with fever and sputum production. The patients' exercise tolerance and sleep pattern should be assessed.

Renal and liver disease

Organ dysfunction can affect the metabolism and clearance of certain intravenous as well as inhalational anesthetics. Serious bleeding problem can occur with renal (platelet dysfunction) or liver disease (deficient clotting factors).

GI reflux disease :

If present, patients are prone to aspiration of gastric contents. Hiatal hernia is believed to increase the risk of aspiration, as is diabetes, history of chronic narcotic ingestion, obesity and pregnancy.

Diabetes :

If present, close monitoring of blood glucose should be considered perioperatively. Diabetes also affect gastric emptying, having a significant impact on preoperative medication selection and management.

Rheumatoid arthritis especially if treated with steroids and ankylosing spondylosis with involvement of C-spine (difficult airway, possible atlanto-axial subluxation etc).

Alcoholism and drug abuse:

Increased tolerance to many sedative and narcotics

Family History:

Specific history of previous anesthetic problems, history of malignant hyperthermia, enzyme deficiency and other familial and inherited diseases.

Allergies:

Antibiotics, anesthetics, analgesics, sedatives/ hypnotics.

Medications:

Appropriate instructions must be given to the patient preoperatively regarding their medication management.

Bleeding:

Abnormal platelet function or hereditary deficiency of clotting factors, aspirin therapy and liver disease.

NPO status

Should be part of the checklist preoperatively so that proper anesthesia induction technique can be planned.

PHYSICAL EXAMINATION

A) AIRWAY EVALUATION

The airway evaluation is an integral part of preanesthesia evaluation. The examination of airway should always include Overall appearance

- Neck: stout or thin, long or short? Sunken cheeks and Presence of beard may make mask fit difficult.
- Mouth : Mouth opening (measured in cm or fingerbreadth)
- Anterior displacement of mandible
- Tongue size
- Visibility of uvula
- Protrusion of upper incisors
- Loose or damaged teeth; prostheses,
- Movement: Flexion/ extension of neck .

- Sniffing position .
- Palpation : Trachea in midline.
- Distance from mentum to hyoid .
- Nose : Both nares patent . Protuberant nose suggests poor mask fit and difficult mask ventilation.

*There are **three** preoperative airway examinations that attempt to predict the ease of endotracheal intubation.*

1: Size of tongue in relation to the size of oral cavity

MALAMPATI CLASSIFICATION (Modification)

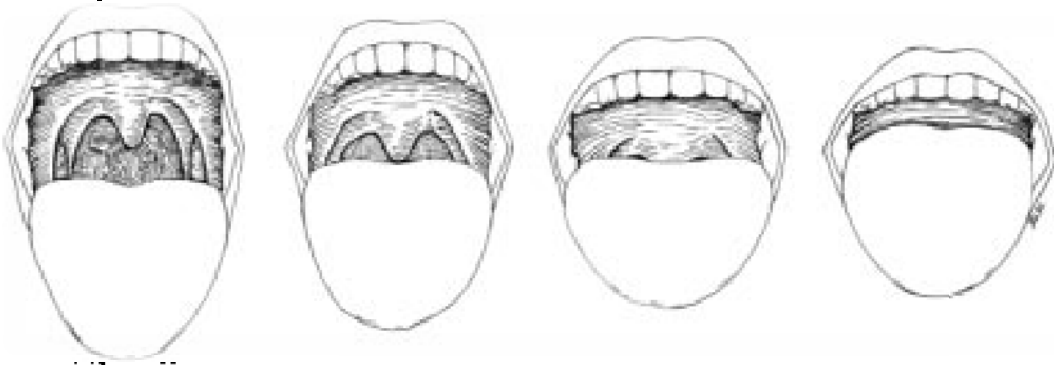
Patient is asked to open mouth widely

Class 1: Soft palate, fauces, uvula, anterior and posterior faucial pillars can be seen.

Class 2: Soft palate, fauces, uvula can be seen. The tongue masks anterior and posterior faucial pillars.

Class 3: Soft palate and the base of uvula can be seen only.

Class 4: Only hard palate is visible.



LARYNGOSCOPIC VIEW

Grade I: Visualization of entire laryngeal aperture.

Grade II: Visualization of only posterior portion of the laryngeal aperture.

Grade III: Visualization of only the epiglottis.

Grade IV: Visualization of only the soft palate.

2: Atlanto-occipital joint extension

The alignment of the oral, pharyngeal and laryngeal axes into a straight line (sniffing position). This will allow less of the tongue obscuring the laryngeal view and there will be much less need for displacing the tongue anteriorly.

3: Thyro-mental distance:

The space anterior to the larynx determines how readily the laryngeal axis will fall in line with the pharyngeal axis when the atlanto-occipital joint is extended. When there is a large mandibular space, the tongue is easily contained within this large compartment and does not have to be pulled maximally forward in order to reveal the larynx. The distance between inside the mandible to hyoid bone should be greater than 6 cm or three

fingerbreadths. Prior surgical history and intubation record should always be available (when applicable).

Other medical conditions with involvement of airway and C-spine include:

Rheumatoid Arthritis involving, cervical spine, TMJ and Cricoarytenoid joint
TMJ Dysfunction (impedes mouth opening)

Acromegaly

Cancer of head and neck, particularly, involving upper airway and trachea.

History of prior radiation treatment of neck (for cancer treatment)

Obstructive sleep apnea

Prior airway surgery

Facial trauma with mandibular fracture (CSF rhinorrhea, etc)

B) NECK: Neck examination should be performed as part of airway evaluation. Presence of carotid bruit, midline masses which can deviate or compress the trachea.

C) LUNGS: Presence of any abnormal lung sounds (wheezing, rales) merit further evaluation of the patients' pulmonary status.

D) HEART: Assessment should include heart rate, rhythm and presence or absence of murmur and distention of jugular (JVD).

E) Examination of the extremities and back is part of the preoperative evaluation.

ASA Physical Status Classification

Class 1: A normal healthy patient

Class 2: A patient with mild systemic disease that results in no functional limitation.

Class 3: A patient with severe systemic disease that results in functional limitation.

Class 4: A patient with severe systemic disease that is a constant threat to life.

Class 5: A moribund patient that is not expected to survive for 24 hours with or without the operation.

Class 6: A declared brain-dead patient whose organs are being removed for donor purposes.

The modification E is added to the ASA physical status classification to indicate that the case is done emergently.

Preoperative labs tests are indicated either to confirm the findings on abnormal physical examination or that will help the anesthesiologist to manage the patient's problems perioperatively.

F) EKG: Male or female, 50 years of age and older with coexisting cardio-pulmonary risk factors.

G) Chest X-rays: Chest x-rays are not indicated for any asymptomatic patient who is less than 75 years of age and has no cardio-pulmonary risk factors. Chest X-ray may be helpful in diagnosing the existence of tracheal deviation, mediastinal mass, lung mass, aortic aneurysm, pulmonary edema, pneumonia, atelectasis, fracture of clavicle and cardiomegaly.

Laboratory studies :

Routine laboratory screening tests are rarely useful. Tests should be selected based on the patient's medical condition and the proposed surgical procedure. A brief review of current guidelines follows:

- Hematological studies may be indicated if there are concerns about pre- or intraoperative blood loss, anemia, or coagulopathy.
 - Recent hematocrit/hemoglobin level.
 - Platelet function may be assessed by a history of easy bruising, excessive bleeding from gums and minor cuts, and family history. A positive finding in this category warrants additional laboratory evaluation and possibly a consultation with a hematologist.
 - Coagulation studies are ordered only when clinically indicated (e.g., history of a bleeding diathesis, aspirin or anticoagulant use, liver disease, or serious systemic illness) or if postoperative anticoagulation is planned.
- Serum chemistry studies are ordered only when specifically indicated by history for patients who have chronic renal, cardiovascular, hepatic, or intracranial disease as well as for those with diabetes or morbid obesity.

Informed consent :

- ✓ Involves discussing the anesthetic plan, alternatives, and potential complications in terms understandable to the layperson.
- ✓ It is preferable that this discussion be conducted in the patient's native language.
- ✓ Furthermore, written forms should also be available in the patient's native language..

References

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2. Anesthesia Patient Safety Foundation (<http://www.apsf.org>) accessed January 30, 2006.
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4. Cooper JB, Newbower RS, Kitz RJ. An analysis of major errors and equipment failures in anesthesia management: considerations for prevention and detection. *Anesthesiology* 1984;60:34-42.
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The anesthesia plan

The plan should include the following:

- A premedication.
- Need standard ASA monitors. However, if the patient may experience large hemodynamic fluctuations, invasive monitoring should be considered (e.g., central venous pressure for volume monitoring, arterial line for potential hemodynamic instability).
- A review of anesthetic options; general anesthesia, regional anesthesia, and combinations thereof should be reviewed and options appropriate for the patient listed in the final assessment.
- Plan for postoperative pain control.

Table 1.1. ASA guidelines for NPO status preoperatively

	Clear Age Liquids	Breast Milk	Nonhuman Milk/ Snack	Light	Fried Fatty Foods/Meat
Infan	2 hours	4 hours	6 hours		8 hours
Child	2 hours	4 hours	6 hours		8 hours
Adult	2 hours	N/A	6 hours		8 hours

Premedication

- Guidelines for prophylaxis for pulmonary aspiration
 - Histamine (H₂) antagonists produce a dose-related decrease in gastric acid production. Cimetidine (Tagamet), 200 to 400 mg orally, intramuscularly or intravenously, and ranitidine (Zantac), 150 to 300 mg orally or 50 to 100 mg intravenously or intramuscularly, significantly reduce the volume and the acidity of gastric secretions.
 - Metoclopramide (Reglan) enhances gastric emptying by increasing lower esophageal sphincter tone and simultaneously relaxing the pylorus. An oral dose of 10 mg is given 1 to 2 hours before anesthesia.
 - Midazolam (Versed), 1 to 3 mg intravenously or intramuscularly, is a short-acting benzodiazepine that provides excellent amnesia and sedation.
 - Lorazepam (Ativan) may also be used (1 to 2 mg orally or intravenously) but can cause more prolonged amnesia and postoperative sedation. It should not be given intramuscularly.
- Opioids are not usually given as premedication unless the patient has significant pain

THE MONITORS

I. Standard ASA_monitoring for general anesthesia , monitored anesthesia care and regional anesthesia :-

1. Oxygenation (oxygen analyzer, pulse oximetry),

2. Ventilation (capnography, minute ventilation), respiratory rate (under regional anesthesia)
3. Circulation (electrocardiogram [ECG], arterial blood pressure, perfusion assessment),
4. Temperature.

II. Cardiovascular system.

Cardiovascular system. The circulatory system is responsible for oxygen delivery to and removal of waste products from the organs, and this must be maintained during anesthesia.

- Circulation
 - Signs and symptoms of perfusion abnormalities
 - Central nervous system: mental status changes, neurologic deficits.
 - Cardiovascular system: chest pain, shortness of breath, ECG abnormalities, wall motion abnormalities on echocardiogram.
 - Renal: decreased urine output, elevated blood urea nitrogen and creatinine, decreased fractional excretion of sodium.
 - Gastrointestinal: abdominal pain, decreased bowel sounds, hematochezia.
 - Peripheral: cool limbs, poor capillary refill, diminished pulses.
- ECG. The ECG monitors the conduction of electrical impulses through the heart.
 - Rhythm detection is best seen in lead II.
 - Arterial blood pressure. Automated noninvasive blood pressure is the most common noninvasive method of measuring blood pressure in the operating room.

- Invasive blood pressure monitoring uses an indwelling arterial catheter coupled through fluid-filled tubing to a pressure transducer. The transducer converts pressure into an electrical signal to be displayed.
 - Indications
 - Need for tight blood pressure control (e.g., induced hyper- or hypotension).
 - Hemodynamically unstable patient.
 - Frequent arterial blood sampling.
 - Inability to utilize noninvasive blood pressure measurements.
- Central venous pressure (CVP) and cardiac output
 - CVP is measured by coupling the intravascular space to a pressure transducer using fluid-filled tubing. Pressure is monitored at the level of the vena cava or the right atrium.
 - Indications
 - Measurement of the right heart filling pressures to assess intravascular volume and right heart function.
 - Drug administration to the central circulation.
 - Intravenous access for patients with poor peripheral access.
 - Indicator injection for cardiac output determination (e.g., green dye cardiac output).
 - Access for insertion of pulmonary artery catheter.
 - Range. The CVP is normally 2 to 6 mm Hg.

III. Respiratory system.

Respiratory system monitoring include pulse oximetry, capnography, a fraction of inspired oxygen analyzer, and a disconnect alarm.

How does the pulse oximeter works?

Pulse oximeter combines the principles of oximetry and plethysmography to noninvasively measure oxygen saturation in arterial blood. The pulse oximeter probe contains two light emitting diodes at wavelengths of 940nm and 660 nm. Oxygenated and reduced hemoglobin differ in light absorption (940 and 660 nm respectively). Thus the change in light absorption during arterial pulsation is the basis of oximetry determination. The ratio of the absorption at the two wavelengths is analyzed by a microprocessor to record the oxygen saturation.

What is Capnometry?

Capnometry is the measurement of end-tidal carbon dioxide tension. This provides valuable information to the anesthesiologist. The presence of end tidal CO₂ aids in confirming endotracheal intubation. Alteration in the slope of the graph can give clues to the presence of airway obstruction. A rapid fall in reading may signify extubation, air embolism or low cardiac output with hypovolemia.

IV. Central nervous system (level of consciousness) monitoring

- Bispectral index (BIS) assess central nervous system depression during general anesthesia. It is based on the surface electroencephalogram (EEG), which predictably changes in amplitude and frequency as the depth of anesthesia increases.

V. Temperature monitoring

- Indications
 - Infants and small children are prone to thermal lability due to their high surface area to volume ratio.

- Adults subjected to large evaporative losses or low ambient temperatures (as occur with exposed body cavity, large volume transfusion of unwarmed fluids, or burns) are prone to hypothermia.
- Malignant hyperthermia is always a possible complication, and temperature monitoring should always be available.
- Monitoring site
 - Tympanic membrane temperature
 - Rectal temperature
 - Nasopharyngeal temperature,
 - Esophageal temperature monitoring reflects the core temperature well. The probe should be located at the lower third of the esophagus and rarely may be misplaced in the airway.
 - Blood temperature measurements may be obtained with the thermistor of a PAC.

VI. Neuromuscular blockade monitoring:

Neuromuscular blockade is monitored during surgery to guide repeated doses of muscle relaxants and to differentiate between the types of block. All techniques for assessing neuromuscular blockade use a peripheral nerve stimulator (PNS) to stimulate a motor nerve electrically.

Suggested Reading

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THE AIRWAY

The larynx lies at C4/C5 in adults and at C3/C4 in pediatric age group. In children the cricoid ring is the narrowest part as compared to glottic opening is the narrowest part in adults. The epiglottis is crescent shaped in adults and is long and omega shaped in children.

Oxygen Therapy

Aim: to prevent or at least minimize tissue hypoxia.

Indication:

1. When oxygen tension is less than 60 mmHg in a healthy patient. If patient has chronic lung disease may accept a lower oxygen tension before treatment.

2. Post operatively supplemental oxygen may be given if $SaO_2 < 92\%$ especially if anemic, hypotensive, septic
3. Delivery of medication (e.g. nebulized salbutamol)
4. Treatment of carbon monoxide poisoning

Hypoxemia: Causes of tissue hypoxia:

Low inspired oxygen tension

Hypoventilation

Poor matching of ventilated areas of lung with those areas being perfused

Impaired blood flow to tissues: Low cardiac output

Hypotension

Arterial occlusion

Impaired oxygen carrying capacity:

Low hemoglobin concentration

Abnormal hemoglobin (e.g. sickle cell)

Poisoned hemoglobin (methemoglobin, carboxyhemoglobin)

Impaired oxygen utilization by tissues: cyanide poisoning

Excess oxygen utilization: Thyrotoxicosis

Malignant hyperthermia

Delivery systems:

Nasal cannulae – inspired oxygen concentration is dependent on the oxygen flow rate, the nasopharyngeal volume and the patient's inspiratory flow rate. The nasopharynx acts as an oxygen reservoir between breaths. With each inspiratory breath oxygen is taken from this storage but also entrained directly. Increases inspired oxygen concentration by 3-4%. Oxygen flow rates greater than 3 liters are poorly tolerated by patients due to drying and crusting of the nasal mucosa. **Face masks** : Three types of facemask are available; open, Venturi, non-rebreathing.

Open facemasks :

Are the most simple of the designs available. They do not provide good control over the oxygen concentration being delivered to the patient causing variability in oxygen treatment. A 6l/min flow rate *is the minimum* necessary to prevent the possibility of rebreathing. Maximum inspired oxygen concentration ~ 50-60%.

Venturi facemasks

Are so named since they rely on entraining room air with the oxygen flow. This ensures both a high flow rate (greater than the patients inspiratory flow rate) and a guaranteed oxygen concentration. They should be used in patients with COPD/emphysema where accurate oxygen therapy is needed. Arterial blood gases can then be drawn so correlation between oxygen therapy for hypoxemia and potential risk of CO₂ retention can be made. Masks are available for delivering 24%, 28%, 35%, 40%, 50%.

Non-rebreathing facemasks have an attached reservoir bag and one-way valves on the sides of the facemask. The reservoir bag is of sufficient volume to meet the inspiratory flow rate of the patient and the one way valves prevent entrainment of room air. With flow rates of 10 liters an oxygen concentration of 95% can be achieved. These masks provide the highest inspired oxygen concentration for non-intubated patients.

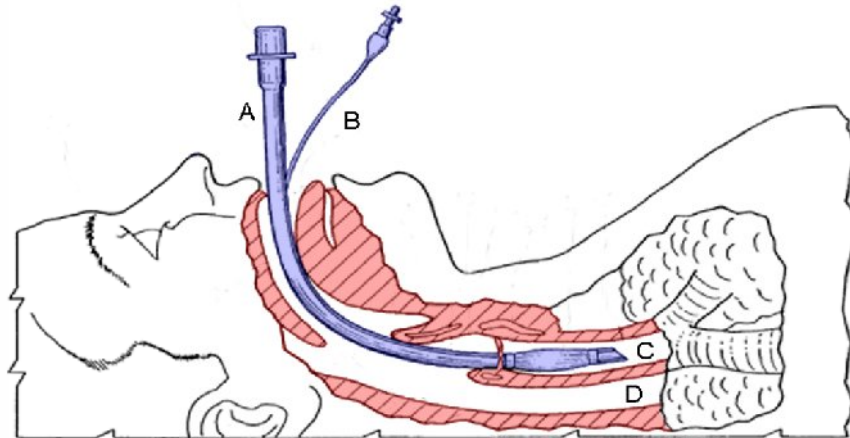
Ambu-bags -

Used in resuscitations away from the OR setting these can deliver a maximum of 50% with no reservoir bag attached but 100% if an oxygen reservoir is attached.

Hazards of oxygen therapy -

Oxygen therapy can have both respiratory and non-respiratory complications. These are usually related to prolonged treatment at high concentrations and include:

- ✚ Absorption atelectasis – Alveoli that contain 100% oxygen and have good blood flow going by can have all their oxygen taken up causing collapse of the alveolus. Just adding a small amount of nitrogen to the inspiratory mix can prevent this collapse by splinting the alveolus open since nitrogen is relatively insoluble and so very slowly absorbed across the alveolar membrane.
- ✚ Hypoventilation – Occurs in COPD patients who rely on their hypoxic drive for respiration. High inspiratory oxygen concentrations will correct this hypoxia but at the same time remove the respiratory drive. These patients begin to hypoventilate and can develop critical CO₂ retention. If giving oxygen to these patients start at a low concentration and monitor therapy with regular arterial blood gas sampling.
- ✚ Pulmonary toxicity – Prolonged high concentrations of oxygen result in the production of free radicals which are cytotoxic to cellular DNA, proteins and lipids. The resulting injury gives a clinical picture similar to ARDS (adult respiratory distress syndrome). The same toxicity results in bronchopulmonary dysplasia in newborn/premature babies.



OROTRACHEAL INTUBATION

Indications for Intubation - the 5 P's:

- ❑ **P**atency of airway required
 - Decreased level of consciousness (LOC)
 - Facial injuries
 - Epiglottitis
 - Laryngeal edema, e.g. burns, anaphylaxis
- ❑ **P**rotect the lungs from aspiration
 - Absent protective reflexes, e.g. coma, cardiac arrest
- ❑ **P**ositive pressure ventilation
 - Hypoventilation – many etiologies
 - Apnea, e.g. during general anesthesia
 - During use of muscle relaxants

- ❑ Pulmonary Toilet (suction of tracheobronchial tree)
 - For patients unable to clear secretions
- ❑ Pharmacology also provides route of administration for some drugs

Equipment Required for Intubation

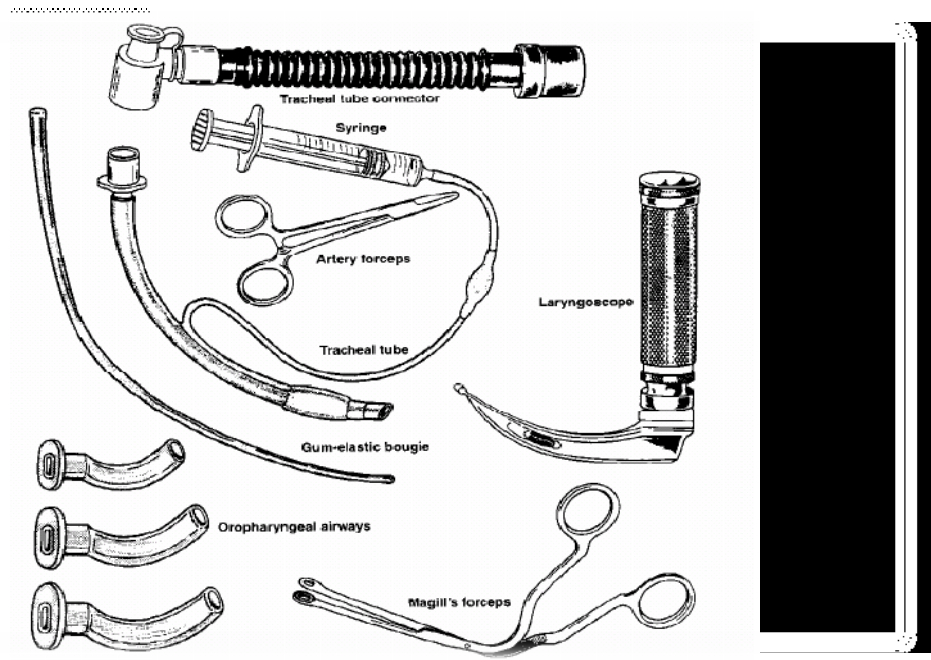
- ❑ Bag and mask apparatus (e.g. Laerdal/Ambu) to deliver O2 and to manually ventilate if necessary mask sizes/shapes appropriate for patient facial type, age
- ❑ Pharyngeal airways (nasal and oral types available) to open airway before intubation, oropharyngeal airway prevents patient biting on tube
- ❑ Laryngoscope
 - Used to visualize vocal cords
 - MacIntosh = curved blade (best for adults)
 - Magill/Miller = straight blade (best for children)
- ❑ Trachelight - an option for difficult airways
- ❑ Fiberoptic scope - for difficult, complicated intubations
- ❑ Endotracheal tube (ETT): many different types for different indications
 - Inflatable cuff at tracheal end to provide seal which permits positive pressure ventilation and

prevents aspiration

- No cuff on pediatric ETT (physiological seal at level of cricoid cartilage)
- Sizes marked according to internal diameter; proper size for adult ETT based on assessment of patient
- Adult female: 7.0 to 8.0 mm
- Adult male: 8.0 to 9.0 mm
- Child (age in years/4) + 4 or size of child's little finger = approximate ETT size
- If nasotracheal intubation, ETT 1-2 mm smaller and 5-10 cm longer
- Should always have ETT smaller than predicted size available in case estimate was inaccurate

Malleable stylet should be available; it is inserted in ETT to change angle of tip of ETT, and to facilitate the tip entering the larynx; removed after ETT passes through cords

Lubricant and local anaesthetic are optional



forceps used to manipulate ETT tip during nasotracheal intubation

- Suction, with pharyngeal rigid suction tip (Yankauer) and tracheal suction catheter
- Syringe to inflate cuff (10 ml)
- Stethoscope to verify placement of ETT
- Detector of expired CO₂ to verify placement
- Tape to secure ETT and close eyelids

Remember "SOLES"

Suction

Oxygen

Laryngoscope

ETT

Stylet, Syringe

Preparing for Intubation

- Failed attempts at intubation can make further attempts difficult due to tissue trauma
- plan and prepare (anticipate problems!) assess for potential difficulties (see Preoperative Assessment section)
- ensure equipment (as above) is available and working e.g. test ETT cuff, and means to deliver positive pressure ventilation e.g. Ventilator, Ambu bag, light on laryngoscope
- Pre-oxygenation of patient
- May need to suction mouth and pharynx first

Proper Positioning for Intubation

- FLEXION of lower C-spine and EXTENSION of upper C-spine at atlanto-occipital joint ("sniffing position")
- "sniffing position" provides a straight line of vision from the oral cavity to the glottis (axes of mouth, pharynx and larynx are aligned)

❑ Above CONTRAINDICATED in known/suspected C-spine fracture

❑ Once prepared for intubation, the normal sequence of induction can vary either:

Rapid Sequence Induction

❑ Indicated in all situations predisposing the patient to regurgitation/aspiration

- Acute abdomen
- Bowel obstruction
- Emergency operations, trauma
- Hiatus hernia with reflux
- Obesity
- Pregnancy
- Recent meal (< 6 hours)
- Gastro esophageal reflux disease (GERD)

❑ Procedure as follows

- Patient breathes 100% O₂ for 3-5 minutes prior to induction of anesthesia (e.g. thiopental and succinylcholine..I)

❑ Perform "Sellick's manoeuvre (pressure on cricoid cartilage) to compress esophagus, thereby preventing gastric reflux and aspiration

- Induction agent is quickly followed by muscle relaxant (e.g. succinylcholine), causing fasciculations then relaxation

- Intubate at time determined by clinical judgement - may use end of fasciculations if no defasciculating neuromuscular junction (NMJ) Blockers have been given
- Must use cuffed ETT to prevent gastric content aspiration
- Inflate cuff, verify correct placement of ETT, release of cricoid cartilage pressure
- Manual ventilation is not performed until the ETT is in place and cuff up (to prevent gastric distension)

Confirmation of Tracheal Placement of ETT

- ❑ Direct visualization of tube placement through cords
 - CO2 in exhaled gas as measured by capnograph
 - Visualization of ETT in trachea if bronchoscope used
- ❑ Indirect (no one indirect method is sufficient)
 - Auscultation axilla for equal breath sounds bilaterally (transmitted sounds may be heard if lung fields are auscultated) and absence of breath sounds over epigastrium
 - Chest movement and no abdominal distension
 - Feel the normal compliance of lungs when bagging patient
 - Condensation of water vapor in tube during exhalation
 - Refilling of reservoir bag during exhalation

- AP CXR: ETT tip at midpoint of thoracic inlet and carina
- ❑ Esophageal intubation is suspected when
 - Capnograph shows end tidal CO2 zero or near zero
 - Abnormal sounds during assisted ventilation
 - Impairment of chest excursion
 - Hypoxia/cyanosis
 - Presence of gastric contents in ETT
 - Distention of stomach/epigastrium with ventilation

Complications during Laryngoscopy and Intubation

❑ Mechanical

- Dental damage (i.e. chipped teeth)
- Laceration (lips, gums, tongue, pharynx, esophagus)
- Laryngeal trauma
- Esophageal or endobronchial intubation

❑ Systemic

- Activation of sympathetic nervous system (hypertension (HTN), tachycardia, dysrhythmias) since tube touching the cords is stressful
- Bronchospasm

Problems with ETT and Cuff

- ❑ Too long - endobronchial intubation
- ❑ Too short - accidental extubation

- ❑ Too large - trauma to surrounding tissues
- ❑ Too narrow - increased airway resistance
- ❑ Too soft - kinks
- ❑ Too hard - tissue damage
- ❑ Prolonged placement - vocal cord granulomas, tracheal stenosis
- ❑ Poor curvature - difficult to intubate
- ❑ Cuff insufficiently inflated - allows leaking and aspiration
- ❑ Cuff excessively inflated - pressure necrosis

Medical Conditions associated with Difficult Intubation

- ❑ Arthritis - decreased neck range of motion (ROM) (e.g. rheumatoid arthritis (RA) - risk of atlantoaxial subluxation)
- ❑ Obesity - increased risk of upper airway obstruction
- ❑ Pregnancy - increased risk of bleeding due to edematous airway, increased risk of aspiration due to decreased gastroesophageal sphincter tone
- ❑ Tumors - may obstruct airway or cause extrinsic compression or tracheal deviation
- ❑ Infections (oral)
- ❑ Trauma - increased risk of cervical spine injuries, basilar skull and facial bone fractures, and intracranial injuries
- ❑ Burns

- ❑ Down 's syndrome (DS) - may have atlantoaxial instability and macroglossia
- ❑ Scleroderma - thickened, tight skin around mouth
- ❑ Acromegaly - overgrowth and enlargement of the tongue, epiglottis, and vocal cords
- ❑ Dwarfism - associated with atlantoaxial instability
- ❑ Congenital anomalies

Predictors of Difficult Airway

Short muscular neck
Prominent upper incisors
Protruding mandible
Receding mandible
Small mouth opening
Full beard
Large tongue
Limited neck mobility
Limited mouth opening due to TMJ

EXTUBATION

❑ General guidelines:

- ✓ check that neuromuscular function and hemodynamic status is normal
- ✓ check that patient is breathing spontaneously with adequate rate and tidal volume
- ✓ allow patient to breathe 100% O₂ for 3-5 minutes
- ✓ suction secretions from pharynx
- ✓ deflate cuff, remove ETT on inspiration (vocal cords abducted)
- ✓ ensure patient breathing adequately after extubation
- ✓ ensure face mask for O₂ delivery available
- ✓ proper positioning of patient during transfer to recovery room, e.g. sniffing position, side lying.

❑ Complications Discovered at Extubation

❑ Early

- Aspiration
- Laryngospasm

❑ Late

- Transient vocal cord incompetence
- Edema (glottic, subglottic)
- Pharyngitis, tracheitis
- Damaged neuromuscular pathway (central and peripheral nervous system and respiratory muscular function), therefore no spontaneous ventilation occurs post extubation

LMAs (Laryngeal Mask Airway)

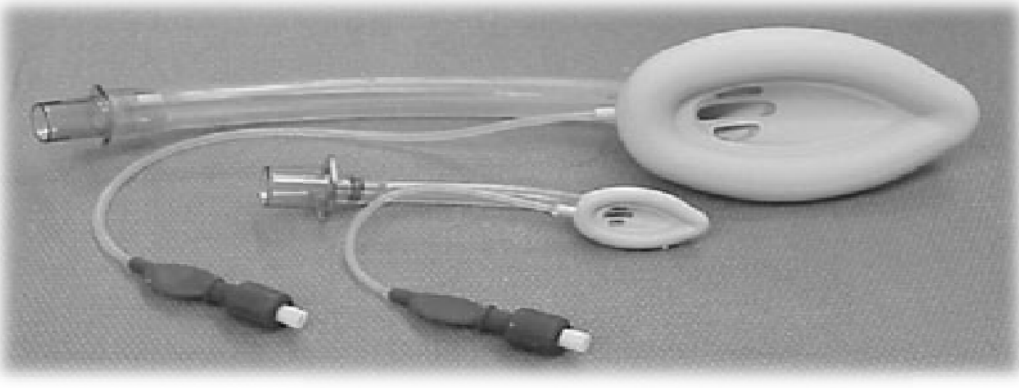
Is a reusable airway management device that can be used as an alternative to both mask ventilation and endotracheal intubation in appropriate patients. The LMA also plays an important role in management of the difficult airway. When inserted appropriately, the LMA lies with its tip resting over the upper esophageal sphincter, cuff sides lying over the pyriform fossae, and cuff upper border resting against the base of the tongue. Such positioning allows for effective ventilation with minimal inflation of the stomach.

Indications

- As an alternative to mask ventilation or endotracheal intubation for airway management. The LMA is not a replacement for endotracheal intubation when endotracheal intubation is indicated.
- In the management of a known or unexpected difficult airway.
- In airway management during the resuscitation of an unconscious patient.

Contraindications

- Patients at risk of aspiration of gastric contents (emergency use is an exception).
- Patients with decreased respiratory system compliance, because the low-pressure seal of the LMA cuff will leak at high inspiratory pressures and gastric insufflation may occur. Peak inspiratory pressures should be maintained at less than 20 cm H₂O to minimize cuff leaks and gastric insufflation.



- Patients in whom long-term mechanical ventilatory support is anticipated or required.
- Patients with intact upper airway reflexes, because insertion can precipitate laryngospasm.

Patient Age/Size	LMA SiCuff VolunETT Size (ID)
Neonates/infants to 5 kg	1 Up to 4 mL 3.5 mm
Infants, 5-10 kg	1.5 Up to 7 mL 4.0 mm
Infants/children, 10 to 20 kg	2.0 Up to 10 mL 4.5 mm
Children, 20 - 30 kg	2.5 Up to 14 mL 5.0 mm
Children, 30 kg to small adults	3.0 Up to 20 mL 6.0 cuffed
Average adults	4.0 Up to 30 mL 6.0 cuffed
Large adults	5.0 Up to 40 mL 7.0 cuffed

ETT, endotracheal tube; ID, inner diameter.

Adverse effects.

The most common adverse effect is sore throat, with an estimated incidence of 10%, and is most often related to overinflation of the LMA cuff. The primary major adverse effect is aspiration, which has been estimated to occur at a comparable incidence as with mask or endotracheal anesthesia.

VENTILATION

Manual Ventilation

- ❑ Can be done in remote areas, simple, inexpensive and can save lives
- ❑ Positive pressure supplied via self-inflating bag (e.g. Laerdal/Ambu+/O₂)
- ❑ Can ventilate via ETT or facemask - cricoid pressure reduces gastric inflation and the possibility of regurgitation and aspiration if using facemask
- ❑ Drawbacks include inability to deliver precise tidal volume, the need for trained personnel to “bag” the patient, operator fatigue, prevents operator from doing other procedures

Mechanical ventilation

❑ *Indications for mechanical (controlled) ventilation include*

- Apnea
- Hypoventilation (many causes)

- Required hyperventilation (to lower intracranial pressure (ICP))
- Intra-operative position limiting respiratory excursion, (e.g. prone, Trendelenburg)
- Use of muscle relaxants
- To deliver positive end expiratory pressure (PEEP)

❑ **Ventilator parameters include (specific to patient/procedure)**

- Tidal volume (average 10 mL/kg)
- Frequency (average 10/minute)
- PEEP
- FIO₂ (fraction of inspired oxygen) 50% - 100 %.

❑ **Types of mechanical ventilators**

1. pressure-cycled ventilators

- Delivers inspired gas to the lungs until a preset pressure level is reached
- Tidal volume varies depending on the compliance of the lungs and chest wall

2. volume-cycled ventilators

- Delivers a preset tidal volume to the patient regardless of pressure required

❑ **Complications of mechanical ventilation**

- Decreased CO₂ due to hyperventilation

- Disconnection from ventilator or failure of ventilator may result in severe hypoxia and hypercarbia
- Decreased blood pressure (BP) due to reduced venous return from increased intrathoracic pressure
- Severe alkalemia can develop if chronic hypercarbia is corrected too rapidly
- Water retention may occur as antidiuretic hormone (ADH) secretion may be elevated in patients on ventilators
- Pneumonia/bronchitis - nosocomial
- Pneumothorax
- Gastrointestinal (GI) bleeds due to stress ulcers
- Difficulty weaning

Suggested Reading

1. Brain AIJ, Verghese C, Strube PJ. The LMA Prosealâ a laryngeal mask with an oesophageal vent. *Br J Anaesthesia* 2000;84:650-654.
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3. Ferson DZ, Rosenblatt WH, Johansen MJ, et al. Use of the intubating LMA Fastrach in 254 patients with difficult-to-manage airways. *Anesthesiology* 2001;95:1175â€“1181.
4. Hurford WE. Nasotracheal intubation. *Respir Care* 1999;44:643â€“649.

5. Langeron O, Masso E, Huraux C, et al. Prediction of difficult mask ventilation. *Anesthesiology* 2000;92:1229-1236.
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Administration of General Anesthesia

Planning for Anesthesia

A good anesthetic begins with a good plan. There is no rigid format for planning anesthesia. Rather, each plan is adapted to each case. The fundamental goal of anesthetic management is to provide safety, comfort and convenience, first for the patient and second for those caring for the patient.

After a good plan, a good preparation is required for a good anesthetic.

Before every anesthetic, every anesthesiologist should go through a checklist of necessary items including, anesthesia machine, ventilator, oxygen and nitrous supply check, suction device, monitors and anesthesia cart.

Before bringing the patient to the operating room, the proper verification of patient's identity, the planned procedure and the site of the procedure should be carried out by the anesthesiologist. All the preparations should be completed before the patient enters the room including the placement of a working peripheral intravenous line.

The primary goals of general anesthesia are to maintain the health and safety of the patient while providing amnesia, hypnosis (lack of awareness), analgesia, and optimal surgical conditions (e.g., immobility).

Preoperative planning involves the integration of pre-, intra-, and postoperative care. Flexibility is an essential component in this planning; multiple approaches to induction, maintenance, and emergence should be considered.

I. Pre-op Fasting in Healthy Adults

The intake of oral fluids. Intake of water up to 2 hrs before induction of anaesthesia.

- Other clear fluids, clear tea and coffee without milk up to 2 hrs before induction of anaesthesia.
- Tea and coffee with milk are acceptable up to 6 hrs before induction of anaesthesia.
- The volume of administered fluids does not appear to have an impact on patient's residual gastric volume and gastric pH, when compared to a standard fasting regimen. Therefore, patients may have unlimited amounts of water and other clear fluid up to two hours before induction of anaesthesia.

The intake of solid foods

- A minimum pre-op fasting time of 6hrs is recommended for food (solids and milk).
- Fried or fatty meal 8hrs is recommended before induction of anaesthesia.

Chewing gum and sweets

- Chewing gum should not be permitted on the day of surgery.
- Sweets are solid food. A minimum of 6hrs pre-op fasting time is recommended.
- Preoperative evaluations (as mentioned earlier)
- Intravascular volume. The patient's volume status is evaluated either clinically or with appropriate monitors.
- Intravenous (IV) access: The size and number of IV catheters placed varies with the procedure, anticipated blood loss, and the need for continuous drug infusions. At least one 14- or 16-gauge catheter is indicated when rapid fluid or blood infusion is anticipated.
- Preoperative medications
- Monitoring. Standard monitoring (as mentioned earlier) is established before the induction of anesthesia. Invasive monitors (e.g., arterial catheter, central venous line, pulmonary artery catheter) should be placed when indicated.

II. Induction of anesthesia

- The patient's position for induction is usually supine, with extremities resting comfortably on padded surfaces in a neutral anatomic position. The head should rest comfortably on a firm support, which is raised in a sniff position.
- Routine pre-induction administration of oxygen minimizes the risk of hypoxia developing during induction of anesthesia.
 - Induction techniques.
 - IV induction
 - An induction using only inhalational anesthetics may be used to maintain spontaneous ventilation when there is a compromised airway or to defer the placement of an IV catheter (e.g., in pediatric patients).

- Airway management: The anesthetized patient's airway may be managed with a face mask, oral or nasopharyngeal airway, cuffed oro-pharyngeal airway, laryngeal mask airway (LMA), or endotracheal tube (ETT). If tracheal intubation is planned, a muscle relaxant may be given to facilitate laryngoscopy and intubation.

III. Maintenance

Maintenance begins when the patient is sufficiently anesthetized to block awareness and movements in response to surgery. Vigilance on the part of the anesthetist is required to maintain homeostasis (vital signs, acid-base balance, temperature, coagulation, and volume status) and regulate anesthetic depth.

Ventilation of the patient during general anesthesia may be spontaneous, assisted, or controlled.

- Controlled ventilation. Although LMA may be used, an ETT and mechanical ventilator are generally used if ventilation is to be controlled for a significant period of time. Initial ventilator settings in healthy patients usually consist of a tidal volume of 10 to 12 mL/kg and a respiratory rate of 8 to 10 breaths/min. Lower tidal volumes (6 to 7 mL/kg) and the addition of positive end-expiratory pressure (PEEP) reduce the likelihood of barotrauma in patients with pulmonary pathology. Peak inspiratory pressure (PIP) should be noted. High airway pressure (>25 to 30 cm H₂O in non-obese patients) or changes of PIP must be investigated immediately and may signal a breathing circuit problem, ETT obstruction or movement, altered lung compliance or resistance, change in muscle relaxation, or surgical compression.

- Assessment of ventilation. Adequate ventilation is confirmed by continual observation of the patient, auscultation of breath sounds, inspection of the anesthesia machine (e.g., reservoir breathing bag, ventilator bellows, airway pressures and gas flows), and patient monitors (e.g., capnograph, pulse oximeter). Arterial blood gas measurement and adjustments in the patient's ventilation may be required intraoperatively.

IV fluids

- Intraoperative IV fluid requirements
- IV fluids are administered to correct preoperative deficits and intraoperative losses.
 - Crystalloid solutions are used to replace maintenance fluid requirements, evaporative losses, and third space losses.
 - Colloid solutions (e.g., 5% albumin, 6% hydroxyethyl starch) may be used to replace blood loss or restore intravascular volume. To replace blood loss, colloid solutions should be administered in an approximately 1:1 ratio of volume to estimated blood loss.

IV. Recovery from general anesthesia.

During this period, the patient makes the transition from an unconscious state to an awake state with intact protective reflexes.

- Patients should be awake and responsive, with full muscle strength and adequate pain control.
- Residual muscle relaxation is reversed, and the patient may start to breathe spontaneously. Analgesic requirements should be estimated and addressed before awakening.
- Mask ventilation. A patient who has received mask ventilation should continue to breathe 100% oxygen by mask during emergence.

- Extubation. Removal of the ETT from the trachea of an intubated patient is a critical moment.
- Awake extubation. Extubation of the airway usually occurs after the patient fully regains protective reflexes. Awake extubation is indicated in patients at risk of aspiration of gastric contents, patients who have difficult airways, and patients who have just undergone tracheal or maxillofacial surgery.

V. Transport.

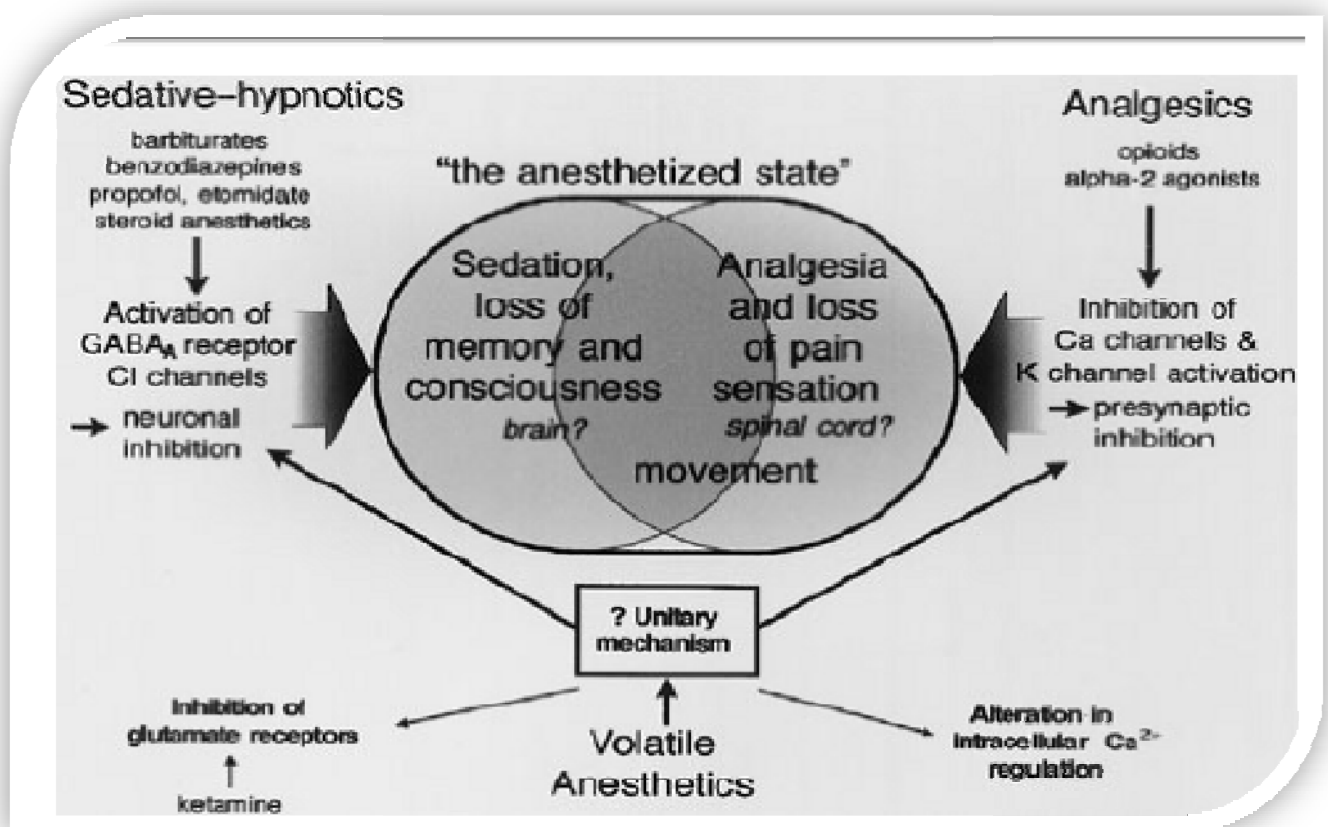
- The anesthetist should accompany the patient from the OR to the post anesthesia care unit (PACU) or ICU.
- Monitoring of blood pressure, hemoglobin saturation, and electrocardiogram is continued during transport to an ICU but generally is not needed for transport of stable patients to the PACU.
- Supplemental oxygen should be available, and the patient's airway, ventilation, and overall condition should be continually observed.
- Placing the patient in the lateral position may help to prevent aspiration and upper airway obstruction.
- Medications and airway equipment should be available during transport if the patient is unstable or if transport is over a significant distance. Upon transfer of responsibility for patient care in the PACU or ICU, the anesthetist should provide a concise but thorough summary of the patient's past medical history, intraoperative course, postoperative condition, and current therapy.

VI. Postoperative visit.

- Postoperative visit. A postoperative evaluation of the patient should be performed by the anesthetist within 24 to 48 hours of surgery and documented in the patient's medical record.
- The visit should include a review of the medical record, examination of the patient, and discussion of the patient's perioperative experience.

- Specific complications such as nausea, sore throat, dental injury, nerve injury, ocular injury, altered pulmonary function, or change in mental status should be sought.
- Questions to elicit evidence of awareness during the general anesthetic should be asked. Responses along with an evaluation and plan, if needed, should be recorded in the patient's chart.
- Complications that require further therapy or consultations should be actively managed and the patient's course should be followed until these issues are resolved.

Pharmacology of Intravenous and Inhalation Anesthetics



I. Pharmacology of intravenous (IV) anesthetics.

IV anesthetics are commonly used for induction of general anesthesia, maintenance of general anesthesia, and sedation during local or regional anesthesia.

Propofol (2,6-diisopropylphenol) is used for induction or maintenance of general anesthesia as well as for conscious sedation.

- It is prepared as a 1% isotonic oil-in-water emulsion, which contains egg lecithin, glycerol, and soybean oil.
- Mode of action: Increases activity at inhibitory gamma-aminobutyric acid (GABA) synapses. Inhibition of glutamate (N-methyl-D-aspartate [NMDA]) receptors may play a role.
 - Pharmacodynamics
 - Central nervous system (CNS)
 - Induction doses rapidly produce unconsciousness (30 to 45 seconds), followed by rapid reawakening due to redistribution. Low doses produce sedation.
 - Weak analgesic effects at hypnotic concentrations.
 - Decreases intracranial pressure (ICP) but also cerebral perfusion pressure.
 - Cardiovascular system
 - Dose-dependent decrease in preload and afterload and depression of contractility leading to decreases in arterial pressure and cardiac output.
 - Heart rate is minimally affected, and baroreceptor reflex is blunted.
 - Respiratory system
 - Produces a dose-dependent decrease in respiratory rate and tidal volume.

- Ventilatory response to hypercarbia is diminished.
- Other effect

May cause pain during IV administration in as many as 50% to 75% of patients. Pain may be reduced by administering IV in a large vein or by adding lidocaine to the solution.

Barbiturates for anesthesia include thiopental and methohexital. These medications, like propofol, rapidly produce unconsciousness (30 to 45 seconds), followed by rapid reawakening due to redistribution.

- Mode of action: Barbiturates occupy receptors adjacent to GABA receptors in the CNS and augment the inhibitory tone of GABA.
- Pharmacodynamics
 - CNS
 - Produce unconsciousness and suppress responses to pain at much higher concentrations.
 - Thiopental can cause hyperalgesia at subhypnotic concentrations (clinical relevance uncertain).
 - Produce a dose-dependent cerebral vasoconstriction and decrease in cerebral metabolism which decrease cerebral blood flow and intracranial pressure.
 - Cardiovascular system
 - Cause venodilation and depress myocardial contractility, so arterial blood pressure and cardiac output decrease in a dose-dependent manner, especially in patients who are preload dependent.
 - May increase heart rate. Very little effect on baroreceptor reflexes.
 - Respiratory system

- Produce a dose-dependent decrease in respiratory rate and tidal volume. Apnea may result for 30 to 90 seconds after an induction dose.
- Laryngeal reflexes more active than with propofol. Incidence of laryngospasm is higher.
- Adverse effects
 - Allergy. True allergies are unusual. Thiopental occasionally causes anaphylactoid reactions (hives, facial edema, hypotension).
 - Porphyria
 - Absolutely contraindicated in patients with acute intermittent porphyria, variegate porphyria, and hereditary coproporphyria.
 - Venous irritation and tissue damage
 - May cause pain at the site of administration because of venous irritation.
 - Subcutaneous infiltration or intra-arterial administration of thiopental (but not methohexital) may cause severe pain, tissue damage, arterial spasm, and necrosis. If intra-arterial administration occurs, heparin treatment, vasodilators, and/or regional sympathetic blockade may be helpful in treatment.

Benzodiazepines include midazolam, diazepam, and lorazepam.

They are often used for sedation and amnesia or as adjuncts to general anesthesia.

Mode of action: Enhance the inhibitory tone of GABA receptors.

- Pharmacodynamics

- CNS
 - Produce amnestic, anticonvulsant, anxiolytic, muscle-relaxant, and sedative-hypnotic effects in a dose-dependent manner.
 - Do not produce significant analgesia.
 - Reduce cerebral blood flow and metabolic rate.
- Cardiovascular system
 - Produce a mild systemic vasodilation and reduction in cardiac output. Heart rate is usually unchanged.
 - Hemodynamic changes may be pronounced in hypovolemic patients or in those with little cardiovascular reserve if rapidly administered in a large dose or if administered with an opioid.
- Respiratory system
 - Produce a mild dose-dependent decrease in respiratory rate and tidal volume.
 - Respiratory depression may be pronounced if administered with an opioid, in patients with pulmonary disease, or in debilitated patients.
- Dosage and administration: for midazolam.
 - Incremental IV doses of diazepam (2.5 mg) or lorazepam (0.25 mg) may be used for sedation. Appropriate oral doses are 5 to 10 mg of diazepam or 2 to 4 mg of lorazepam.
- Adverse effects
 - Drug interactions. Administration of a benzodiazepine to a patient receiving the anticonvulsant valproate may precipitate a psychotic episode.
 - Pregnancy and labor
 - May be associated with birth defects (cleft lip and palate) when administered during the first trimester.

- Cross the placenta and may lead to a depressed neonate.
- Superficial thrombophlebitis and injection pain may be produced by the vehicles in diazepam and lorazepam.
- Flumazenil is a competitive antagonist for benzodiazepine receptors in the CNS.
 - Reversal of benzodiazepine-induced sedative effects occurs within 2 min; peak effects occur at approximately 10 min. Flumazenil does not completely antagonize the respiratory depressant effects of benzodiazepines.
 - Flumazenil is shorter acting than the benzodiazepines it is used to antagonize. Repeated administration may be necessary because of its short duration of action.
 - Dose: 0.3 mg IV every 30 to 60 seconds (to a maximum dose of 5 mg).
 - Flumazenil is contraindicated in patients with tricyclic antidepressant overdose and in those receiving benzodiazepines for control of seizures or elevated intracranial pressure.

Ketamine is a congener of phencyclidine. It is a sedative-hypnotic agent with powerful analgesic properties. Usually used as an induction agent.

- Mode of action: Not well defined but includes antagonism at the NMDA receptor.
- Produces unconsciousness in 30 to 60 seconds after an IV induction dose. Effects are terminated by redistribution in 15 to 20 min. After intramuscular (IM) administration, the onset of CNS effects is delayed for approximately 5 min, with peak effect at approximately 15 min.
 - CNS

- Produces a dissociative state accompanied by amnesia and analgesia. Analgesia occurs at much lower concentrations than hypnosis, so analgesic effects persist after awakening.
- Increases cerebral blood flow (CBF), metabolic rate, and intracranial pressure. CBF response to hyperventilation is not blocked.
- Cardiovascular system
 - Increases heart rate as well as systemic and pulmonary artery blood pressures by causing centrally mediated release of endogenous catecholamines.
 - Often used to induce general anesthesia in hemodynamically compromised patients, particularly those for whom heart rate, preload and afterload, should remain high.
- Respiratory system
 - Usually depresses respiratory rate and tidal volume only mildly and has minimal effect on CO₂ response.
 - Alleviates bronchospasm by a sympathomimetic effect.
 - Laryngeal protective reflexes are relatively well-maintained, but aspiration can still occur.
- Dosage and administration:
 - Ketamine may be especially useful for IM induction in patients in whom IV access is not available (e.g., children). Ketamine is water soluble and may be administered either IV or IM.
 - A concentrated 10% solution is available for IM use only.
- Adverse effects
 - Oral secretions are markedly stimulated by ketamine.

- Emotional disturbance.
- Muscle tone is often increased.
- Increases intracranial pressure and is relatively contraindicated in patients with head trauma or intracranial hypertension.
- Ocular effects. May lead to mydriasis, nystagmus, diplopia, blepharospasm, and increased intraocular pressure; alternatives should be considered during ophthalmologic surgery.

Opioids.

Morphine, meperidine, hydromorphone, fentanyl, sufentanil, alfentanil, and remifentanil are the opioids commonly used in general anesthesia. Their primary effect is analgesia, and therefore they are used to supplement other agents during induction or maintenance of general anesthesia.

- Mode of action: Opioids bind at specific receptors in the brain, spinal cord, and on peripheral neurons. The opioids listed above are all relatively selective for μ opioid receptors.
- Pharmacodynamics
 - CNS
 - Produce sedation and analgesia in a dose-dependent manner; euphoria is common.
 - Cardiovascular system
 - Produce bradycardia in a dose-dependent manner by stimulation of the central vagal nuclei. Meperidine has a weak atropine-like effect and does not cause bradycardia.
 - The relative hemodynamic stability offered by opioids often leads to their use in sedation or

anesthesia for hemodynamically compromised or critically ill patients.

- Respiratory system
 - Produce respiratory depression in a dose-dependent manner.
 - Pupil size is decreased (miosis) by stimulation of the Edinger-Westphal nucleus of the oculomotor nerve.
 - Nausea and vomiting can occur because of direct stimulation of the chemoreceptor trigger zone. Nausea is more likely if the patient is moving.
 - Urinary retention may occur because of increased tone in the vesical sphincter and inhibition of the detrusor (voiding) reflex. May also decrease awareness of the need to urinate.
- Naloxone is a pure opioid antagonist used to reverse unanticipated or undesired opioid-induced effects such as respiratory or CNS depression.
- Peak effects are seen within 1 to 2 min; a significant decrease in its clinical effects occurs after 30 min because of redistribution.
 - Metabolized in the liver.
 - Dosage and administration: Perioperative respiratory depression in an adult can be treated with 0.04 mg IV every 2 to 3 min as needed.

II. Pharmacology of inhalation anesthetics.

Inhalation anesthetics are usually administered for maintenance of general anesthesia but also can be used for induction, especially in pediatric patients. Dosages of inhalation anesthetics are expressed as MAC, the minimum alveolar concentration at one atmosphere at which 50% of patients do not move in response to a surgical stimulus.

- Mode of action
 - Volatile anesthetics. Exact mechanisms are unknown. Various ion channels in the CNS (including GABA, glycine, and NMDA receptors) have been shown to be sensitive to inhalation anesthetics and may play a role.
 - Volatile anesthetics
 - Elimination
 - Exhalation.
 - Metabolism. Volatile anesthetics may undergo different degrees of hepatic metabolism (halothane, 15%; enflurane, 2% to 5%; sevoflurane, 1.5%; isoflurane, <0.2%; desflurane, <0.2%).
 - Volatile anesthetics effects:
 - CNS
 - Produce unconsciousness and amnesia at relatively low inspired concentrations (25% MAC).
 - Produce a dose-dependent generalized CNS depression and depression of electroencephalographic activity up to and including burst suppression..
 - Increase CBF (halothane > enflurane > isoflurane, desflurane, or sevoflurane).
 - Decrease cerebral metabolic rate (isoflurane, desflurane, or sevoflurane > enflurane > halothane)..
 - Cardiovascular system
 - Produce dose-dependent myocardial depression (halothane > enflurane > isoflurane (desflurane or sevoflurane) and systemic vasodilation (isoflurane > desflurane or sevoflurane > enflurane > halothane).
 - Heart rate tends to be unchanged.
 - Respiratory system

- Produce dose-dependent respiratory depression with a decrease in tidal volume, an increase in respiratory rate, and an increase in arterial CO₂ pressure.
- Produce airway irritation (desflurane > isoflurane > enflurane > halothane > sevoflurane) and, during light levels of anesthesia, may precipitate coughing, laryngospasm, or bronchospasm, particularly in patients who smoke or have asthma. The lower pungency of sevoflurane and halothane may make them more suitable as inhalation induction agents.
- Liver. May cause a decrease in hepatic perfusion (halothane > enflurane > isoflurane, desflurane, or sevoflurane). Rarely, a patient may develop hepatitis secondary to exposure to a volatile agent, most notably halothane
- Renal system. Decrease renal blood flow through either a decrease in mean arterial blood pressure or an increase in renal vascular resistance.

Pharmacology of Neuromuscular Blockade

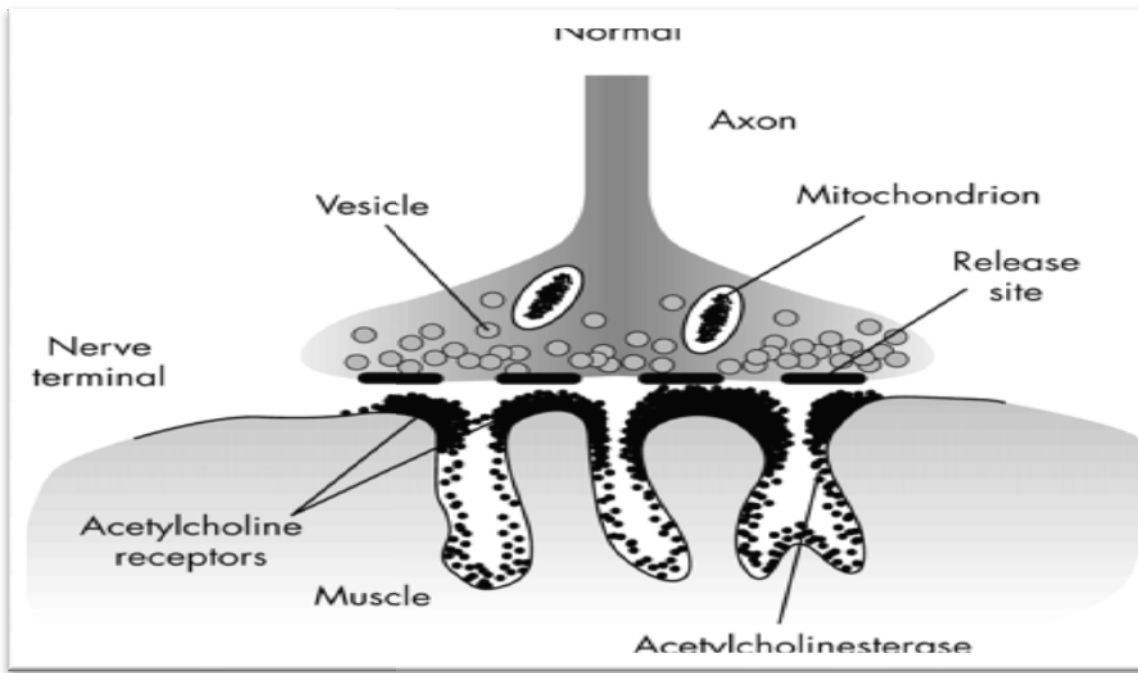
The principal pharmacologic effect of neuromuscular blocking drugs (NMBDs) is to interrupt transmission of synaptic signaling at the neuromuscular junction (NMJ) by antagonism of the nicotinic acetylcholine receptor (AChR).

I. Anatomy and physiology of the NMJ

- The NMJ comprises portions of three cell types: motor neuron, muscle fiber, and Schwann cell. It is a chemical synapse located in the

peripheral nervous system that is composed of the neuronal presynaptic terminal, where acetylcholine (ACh) is stored and released, and the postsynaptic muscle cell (motor endplate), where high densities of the AChR reside. In the nerve terminal, ACh is stored for eventual release in specialized organelles known as synaptic vesicles.

- After triggering depolarization, the ACh diffuses into the synaptic cleft where it is broken down by acetylcholinesterase (AChE) into choline and acetyl CoA. These molecules are then recycled to synthesize new ACh for use in synaptic vesicles and synaptic transmission.



II. General pharmacology of the NMJ

- All NMBDs are antagonists of the AChR. Each is designated depolarizing or nondepolarizing based on whether it induces a depolarization of the muscle membrane after binding to the receptor. The agents differ substantially in their onset, duration of blockade, metabolism, side effects, and interactions with other drugs.
- Succinylcholine (SCh) is currently the only available depolarizing NMBD.
- Nondepolarizing NMBDs are often divided by chemical class: aminosteroid derivatives (e.g., pancuronium, vecuronium, and rocuronium) and benzylisoquinolines (e.g., d-tubocurarine, cisatracurium, and mivacurium). The NMBDs also are commonly classified by duration of effect: ultrashort (SCh), short (mivacurium), intermediate (vecuronium, rocuronium, cisatracurium), and long (pancuronium, d-tubocurarine).

III. Neuromuscular blockade

Depolarizing blockade occurs when a drug mimics the action of the neurotransmitter ACh. SCh, like ACh, binds and activates the AChR, which leads to depolarization of the endplate and adjacent muscle membrane.

Depolarizing blockade from SCh ends when the molecule diffuses away from the receptor and is broken down to choline and succinic acid in the plasma. SCh is hydrolyzed by plasma cholinesterase (also called butyrylcholinesterase or pseudocholinesterase) to choline and succinic acid. This enzyme is not the same as AChE and is not found in the synaptic cleft. Inhibitors of AChE tend to affect both enzymes, however.

- Side effects of SCh are related to its transient agonist effects at both the nicotinic and muscarinic AChRs:
 - Myalgias may occur secondary to muscle fasciculations.
 - Arrhythmias, bradycardia, junctional rhythm, and sinus arrest in children after the first dose and in adults receiving a second dose within a short dose interval (i.e., 5 minutes). Pretreatment with atropine (0.4 mg IV) immediately before SCh blocks this bradycardia.
 - SCh normally causes serum K^+ to increase 0.5 to 1.0 mEq/L but dangerous hyperkalemia and cardiovascular collapse have occurred in patients with burns, upper and lower motor neuron disease, and injuries. It is advisable to avoid SCh in burned patients after the first 24 hours and for 2 years from the injury. Patients with renal failure may safely receive SCh if they are not currently hyperkalemic or acidemic.
 - A transient increase in intraocular pressure due to fascicular contractions of the extraocular muscles.
 - Increased intragastric pressure results from fasciculation of abdominal muscles.
 - SCh produces a mild brief increase in cerebral blood flow and intracranial pressure .
 - A history of malignant hyperthermia is an absolute contraindication to the use of SCh.
 - Prolonged blockade may be caused by low levels of plasma cholinesterase,

Nondepolarizing blockade is most commonly due to reversible competitive antagonism of ACh at the alpha subunits of the AChR.

- The clinical pharmacology of the commonly used nondepolarizing NMBDs:

- Cisatracurium is 1 of 10 stereoisomers that constitute atracurium. It is two to three times as potent as atracurium. Unlike atracurium, it does not produce histamine release or hemodynamic effects after rapid injection of doses as high as eight times its 95% effective dose (ED₉₅).
- Rocuronium, at a dose of 0.6 mg/kg, good to excellent intubating conditions occur by 60 seconds. It is often chosen when rapid sequence induction is necessary and SCh is contraindicated.
- Clinical choice of NMBD: Many factors must be considered simultaneously when selecting a NMBD:
 - The urgency for tracheal intubation,
 - The duration of the procedure,
 - Coexisting medical conditions that may affect the NMJ,
 - Side effects and metabolism of the drug.

IV. Monitoring neuromuscular function

Monitoring neuromuscular function

- There are several reasons to monitor neuromuscular function under anesthesia:
 - To facilitate timing of intubation.
 - To provide an objective measurement of relaxation during surgery and degree of recovery before extubation.
 - To titrate dosage according to patient response.
 - To monitor for the development of phase II block.
 - To permit early recognition of patients with abnormal plasma cholinesterase activity.
- Peripheral nerve stimulators use various patterns of stimulation: single-twitch, tetanus, TOF (Train of four).

V. Reversal of neuromuscular blockade

- Recovery from SCh-induced depolarizing blockade usually occurs in 10 to 15 min. Patients with atypical or inhibited plasma cholinesterase will have a greatly prolonged duration of blockade.
- Nondepolarizing block spontaneously recovers when the drugs diffuse from their sites of action. Reversal can be accelerated by administering agents that inhibit AChE (anticholinesterases), thereby increasing the ACh available to compete for binding sites.
- AChEs: The three principal drugs are edrophonium, neostigmine, and pyridostigmine
- Simultaneous administration of atropine or glycopyrrolate is necessary to decrease cholinergic side effects by causing muscarinic receptor blockade.

Pharmacology of Local Anesthetics (e.g. lidocaine, bupivacaine etc...)

Definition and Mode of Action

- ❑ LA are drugs that block the generation and propagation of impulses in excitable tissues: nerves, skeletal muscle, cardiac muscle, brain
- ❑ LA substances bind to a Na⁺ channel receptor on the cytosolic side of the Na⁺ channel (i.e. must be lipid soluble), inhibiting Na⁺ flux and thus blocking impulse conduction
- ❑ LA must convert to an ionized form to properly bind to receptor
- ❑ Different types of nerve fibres undergo blockade at different rates (see Regional Anesthesia section)

Absorption, Distribution, Metabolism

- ❑ LA readily crosses the blood-brain barrier (BBB) once absorbed into the blood stream
- ❑ Ester-type LA (procaine, tetracaine) broken down by plasma and hepatic esterases; metabolites excreted via kidneys
- ❑ Amide-type LA (lidocaine, bupivacaine) broken down by hepatic mixed function oxidases (P450 system); metabolites excreted via kidney

Selection of LA

- ❑ Delivery modalities include epidural, spinal, peripheral nerve blockades, local injections, topical
- ❑ Choice of LA depends on:
 - Onset of action –influenced by pKa (lower the pKa, the higher the concentration of the base form of the LA and the faster the onset of action)
 - Duration of desired effects – influenced by protein binding (long duration of action when the protein binding of LA is strong)
 - Potency – influenced by lipid solubility (agents with high lipid solubility will penetrate the nerve membrane more easily)
 - Unique needs (e.g. sensory blockade with relative preservation of motor function, for pain management)
 - Potential for toxicity

Maximum Doses for LA

- ❑ Always be aware of the maximum dose for the particular LA used
- ❑ Maximum dose usually expressed as (mg of LA) per (kg of lean body weight) and as a total maximal dose (adjusted for young/elderly/ill)

- lidocaine maximum dose: 5 mg/kg (with epinephrine: 7mg/kg)
- chlorprocaine maximum dose: 11 mg/kg (with epinephrine: 14 mg/kg)
- Bupivacaine maximum dose: 2.5 mg/kg (with epinephrine: 3 mg/kg)

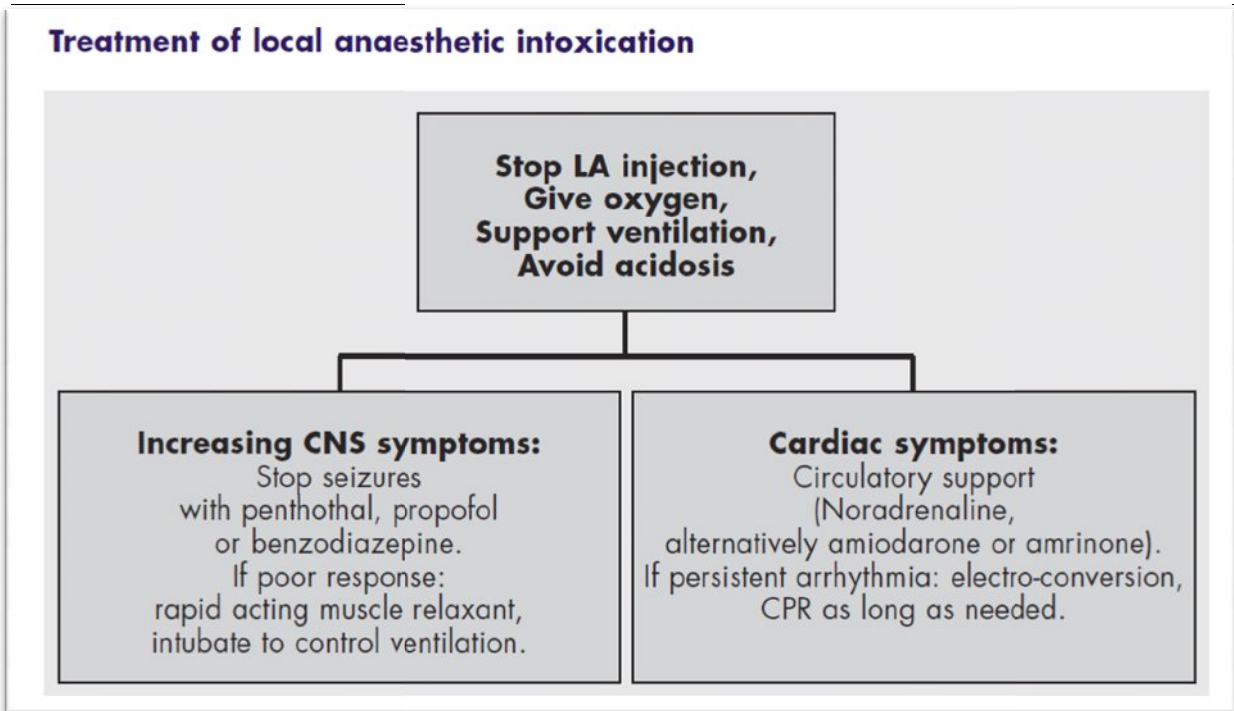
Systemic Toxicity

- Occurs by accidental intravascular injection, LA overdose, or unexpectedly rapid absorption
- Systemic toxicity manifests itself mainly at CNS and CVS
- CNS effects first appear to be excitatory due to initial block of inhibitory fibres; subsequently, block of excitatory fibres
- CNS effects (in approximate order of appearance)
 - Numbness of tongue, perioral tingling, disorientation, drowsiness
 - Tinnitus
 - Visual disturbances
 - Muscle twitching, tremors
 - Convulsions, seizures
 - Generalized CNS depression, coma, respiratory arrest
- CVS effects
 - Vasodilatation, hypotension
 - Decreased myocardial contractility
 - Dose-dependent delay in cardiac impulse transmission
 - Prolonged PR, QRS intervals
 - Sinus bradycardia
 - CVS collapse

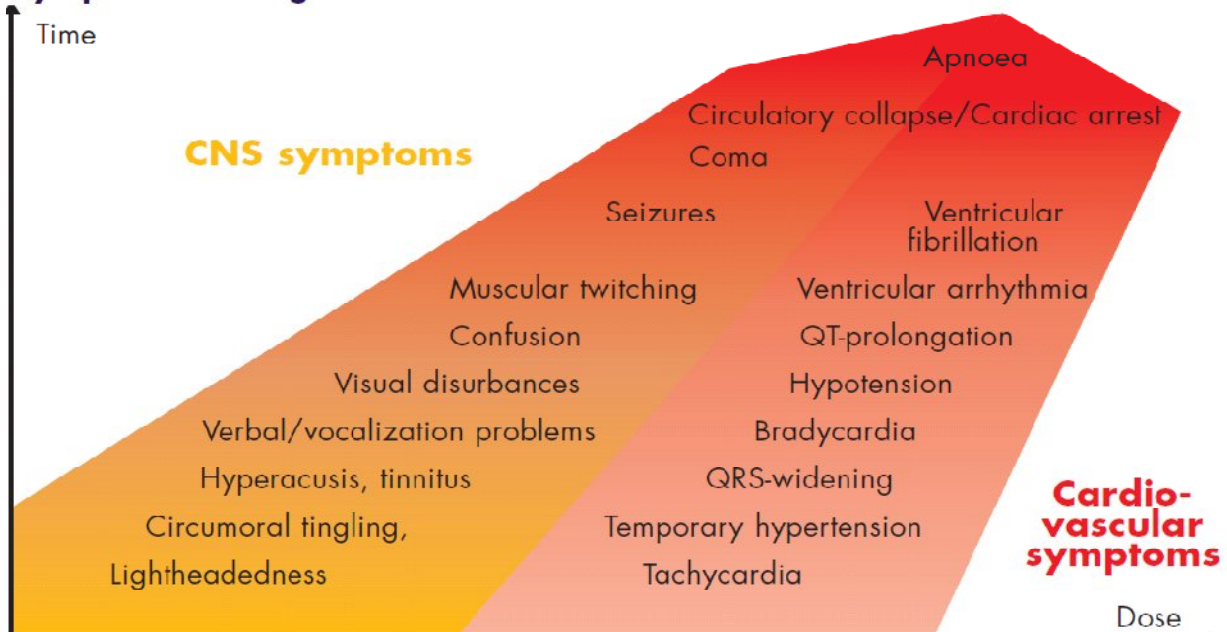
□ Treatment of systemic toxicity

- Early recognition of signs
- 100% O₂, manage ABCs
- Diazepam may be used to increase seizure threshold

- If the seizures are not controlled by diazepam, consider using:
- Thiopental, Possible ETT.



Symptoms and signs of local anaesthetic intoxication



A relative small dose of local anaesthetic, if accidentally injected intravascularly, may lead directly to seizures with both respiratory and cardiovascular problems, depending on drug and patient conditions.

Suggested Reading

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3. Kennedy, SK. Pharmacology of intravenous anesthetic agents. In: Longnecker DE,
4. Clinical Anesthesia Procedures of the Massachusetts General Hospital, 7th Edition
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REGIONAL ANESTHESIA

DEFINITION OF REGIONAL ANESTHESIA

- Local anesthetic applied around a peripheral nerve at any point along the length of the nerve (from spinal cord up to, but not including, the nerve endings) for the purposes of reducing or preventing impulse transmission
- No CNS depression (unless overdose (OD) of local anesthetic); patient conscious
- Regional anesthetic techniques categorized as follows
 - Epidural and spinal anesthesia
 - Peripheral nerve blockades
 - IV regional anesthesia

PREPARATION FOR REGIONAL ANESTHESIA

Patient Preparation

- Thorough pre-op evaluation and assessment of patient
- Technique explained to patient

- IV sedation may be indicated before block
- Monitoring should be as extensive as for general anesthesia

Nerve Localization

- Anatomical landmarks, local anatomy, e.g. line joining iliac crests cross L3-L4 interspace; axillary artery as guide to brachial plexus
- Paresthesias and peripheral nerve stimulation used as a guide to proper needle placement

Relative Indications for Regional Anesthesia

- Avoidance of some of the dangers of general anesthesia (e.g. known difficult intubation, severe respiratory failure, etc.)
- Patient specifically requests regional anesthesia
- For high quality post-op pain relief
- General anesthesia not available

Contraindications to Regional Anesthesia

- Allergy to local anesthetic
- Patient refusal, lack of cooperation
- Lack of resuscitation equipment
- Lack of IV access
- Coagulopathy
- Certain types of preexisting neurological dysfunction
- Local infection at block site

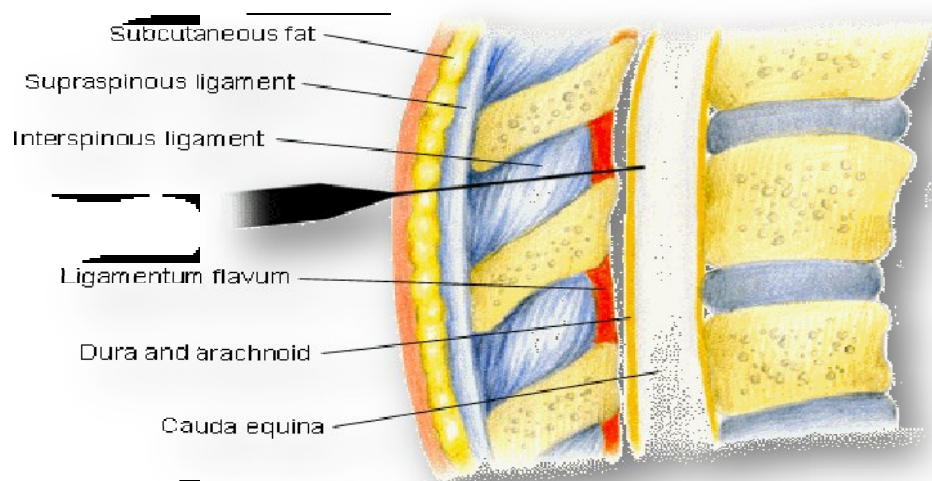
Complications of Regional Anesthesia

- Failure of technique
- Systemic drug toxicity due to overdose or intravascular injection
- Peripheral neuropathy due to intraneural injection
- Pain or hematoma at injection site

EPIDURAL AND SPINAL ANESTHESIA

Anatomy of Spinal/Epidural Area

- Spinal cord extends to L2, dural sac to S2
- Nerve roots (cauda equina) from L2 to S2
- Needle inserted below L2 should not encounter cord, thus L3-L4, L4-L5 interspace commonly used
- Structures penetrated
 - Skin, subcutaneous fat
 - Supraspinous ligament
 - Interspinous ligament • ligamentum flavum (last layer before epidural space) , dura + arachnoid for spinal anesthesia



Spinal Anesthesia

- Relatively small LA dose injected into subarachnoid space in the dural sac surrounding the spinal cord + nerve roots
- LA solution may be made hyperbaric (of greater specific gravity (SG) than the cerebrospinal fluid (CSF) by mixing with 10% dextrose, thus increasing spread of LA to the dependent (low) areas of the subarachnoid space

Epidural Anesthesia

- LA deposited in epidural space (potential space between ligamentum flavum and dura)
- Solutions injected here spread in all directions of the potential space; SG of solution does not affect spread
- Initial blockade is at the spinal roots followed by some degree of spinal cord anesthesia as LA diffuses into the subarachnoid space through the dura
- Larger dose of LA used

Spinal vs. Epidural Anesthesia

□ spinal

- Easier to perform
- Smaller dose of LA required (usually < toxic IV dose)
- Rapid blockade (onset in 2-5 minutes)
- Very effective blockade
- Hyperbaric LA solution - position of patient important

□ epidural

- Technically more difficult; greater failure rate
- Larger volume/doses of LA (usually > toxic IV dose)
- Significant blockade requires 10-15 minutes
- Effectiveness of blockade can be variable
- Use of catheter allows for continuous infusion or repeat injections
- Slower onset of side effects
- Position of patient not as important
- SG of LA solution not as important

Complications of Spinal/Epidural Anesthesia

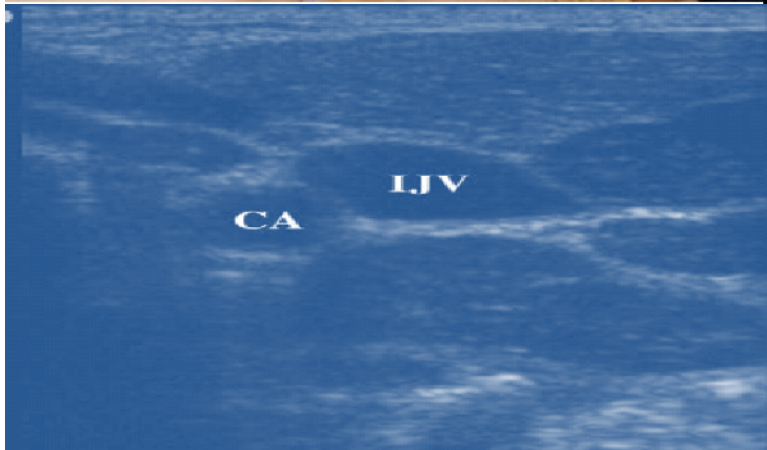
❑ Spinal anesthesia	❑ Epidural anesthesia
<ul style="list-style-type: none">➤ Failure of technique➤ Hypotension, bradycardia if block reaches T2-4 (sympathetic nervous system (SNS) block)➤ Post-spinal headache➤ Extensive spread of anesthetic ("high spinal")➤ Persistent paresthesias (usually transient)➤ Epidural or subarachnoid hematoma➤ Spinal cord trauma, infection	<ul style="list-style-type: none">➤ Failure of technique➤ Hypotension - common➤ Bradycardia if cardiac sympathetics blocked (only if ~T4 block)➤ Systemic toxicity of LA (accidental intravenous)➤ Accidental subarachnoid injection can lead to total spinal anesthesia➤ Catheter complications (shearing, kinking, vascular or subarachnoid placement)➤ Epidural or subarachnoid hematoma

Contraindications to Spinal/Epidural Anesthesia

- ❑ **Absolute contraindications** include lack of proper equipment or properly trained personnel, patient refusal, lack of IV access, allergy to LA, infection at puncture site or underlying tissues, uncorrected hypovolemia, coagulation abnormalities, raised ICP
- ❑ **Relative contraindications** include bacteremia, preexisting neurological disease, aortic/mitral valve stenosis, previous spinal surgery, other back problems, severe/unstable psychiatric disease or emotional instability

Ultrasound for regional anesthesia

The use of ultrasound for regional anesthesia is relatively new, was first described as early as 1978, but it was not until the advent of advanced ultrasound technology in the 1990's that interest in this field grew. Published reports of ultrasound guided regional anesthesia have largely focused on brachial plexus blockade in the interscalene, supraclavicular, infraclavicular and axillary regions. Recent studies examining the efficacy of ultrasound guidance for femoral, sciatic, psoas compartment, celiac plexus and stellate ganglion blocks are promising, while ultrasound visualization of the epidural space can facilitate neuraxial blockade in children, adults and parturients.



Cross-sectional image of the right IJV AND carotid artery

Evidence regarding the use of ultrasound in regional anaesthesia indicates that the use of US may:-

- Decrease the time taken to perform a block
- Decrease the time for a block to onset
- Improve the duration of the block
- Improve patient satisfaction in the block process
- Allow a successful block without the use of a nerve stimulator
- Lower the dose of local anaesthetic required for a block
- Allow regional anaesthesia in otherwise difficult circumstances
- Allow the detection of an intraneural injection
- Allow confirmation of local anaesthetic spread
- Allow confirmation of catheter placement.

Suggested Reading

1. Aida S, et al. Headache after attempted epidural block: the role of intrathecal air *Anesthesiology* 1998;88:76-81.
2. Moraca RJ, et al. The role of epidural anesthesia and analgesia in surgical practice. *Ann Surg* 2003;238:663 to 673.
3. The Second Consensus Conference on Neuraxial Anesthesia and Anticoagulation. Regional anesthesia in the anticoagulated patient: defining the risks. *Regional Anesth Pain Med* 2003;28:172 to 197.
4. Turnbull DK, et al. Post-dural puncture headache: pathogenesis, prevention and treatment. *Br J Anaesth* 2003;91(5):718-729.
5. Vibeke M, et al. Severe neurological complications after central neuraxial blockades in Sweden 1990-1999. *Anesthesiology* 2004;101:950-959.
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8. Copyright 2007© Lippincott Williams & Wilkins

Fluid Management

Physiologic changes during surgery and anesthesia lead to shifts in fluid balance. Epidural, spinal, and caudal anesthesia all may cause variable amounts of sympathetic blockade. Significant third space loss may occur, which essentially involves fluid that is still in the body, but not contributing to intravascular volume, oxygen delivery, or waste removal; this is often difficult to measure. Patients undergoing major surgical procedures require fluid replacement beyond simple blood loss, and the anesthesiologist plays a vital role in assessing and ultimately administering appropriate fluid therapy in intraoperative and postoperative clinical settings.

CRYSTALLOIDS AND COLLOIDS

Intravascular Volume Assessment and Fluid Replacement

Total body water is approximately 60 % of body weight in males and 55 % of body weight in females. (40% is intracellular and 20 % is extracellular). Intravascular volume or plasma volume is 1/4th of extracellular fluid volume. Estimated total blood volume in a 70-kg patient is approximately 4900 cc (70 Kgx70cc/Kg).

I. Goals of Fluid Resuscitation

The primary objective of perioperative fluid management is maintenance of adequate tissue perfusion. In a simple term, $DO_2 = CaO_2 \times CO \times 10$

Where, DO₂ is (delivery of oxygen), CaO₂ (total oxygen content in blood) and CO (cardiac output)

Normally, DO₂ is regulated by local and systemic factors with changes in vascular tone in response to changes in regional and systemic oxygen consumption (VO₂). In normal situations, VO₂ becomes DO₂-dependent when DO₂ is reduced to critical levels, <330 ml O₂/min/m². This is when oxygen extraction reaches a maximum. Further reductions in DO₂ result in a reduction in VO₂.

In certain conditions such as ARDS, sepsis, and severe burns, VO₂ will be dependent on DO₂ at levels that are higher than the normal DO₂. In these high-risk patients, survival has been shown to be associated with supernormal DO₂, >600 ml O₂/min/m². Studies suggest that treatment of the components of DO₂ may improve survival.

$$\text{CaO}_2 = (\text{Hb} \times \text{SaO}_2 \times 1.34) + (0.003 \times \text{PaO}_2)$$

$$\text{DO}_2 = \text{Cardiac Index} \times \text{CaO}_2 \times 10$$

Adequacy of Perfusion can be assessed by clinically and laboratory investigations:

Mental status changes
Poor Capillary refill
Skin color
Skin Temperature
Fast pulse rate
Low Urine Output

Low urinary sodium level

Acid-base status (Metabolic acidosis or alkalosis)

High Serum Lactate Levels

High Oxygen consumption

Low Mixed Venous Oxygen Saturation

In approaching the surgical patient who exhibits signs of low perfusion, such as oliguria or hypotension and tachycardia, the most common etiology is insufficient intravascular volume.

II. Assessment of crystalloid requirement

A. Determining Preoperative Fluid Deficits

Intra operative maintenance fluid requirement can be assessed by maintenance fluid requirements per hour in ml and is always replaced by isotonic saline solution

From : 1-10 Kg 4 ml/Kg /hr

From : 11-20 Kg 2 ml/Kg/hr

More than 20 add 1 ml/Kg/hr

B. Determination of intra op losses

Usual Intra-operative Fluid requirement in addition to maintenance fluids for minor to major procedures

Minimal Trauma 4 ml/kg/hr

Moderate Trauma 6 ml/kg/hr

Severe Trauma 8 ml/kg/hr

Example: A 70-kg patient, NPO for 6 hours, undergoing one-hour hernia repair.

Deficit = 40 cc/hr for 1st 10 kg BW + 20 cc/hr for 2nd 10 kg BW and 50 cc/hr for rest of 50 kg = 110 cc/hr

6 hours of NPO X 110 cc/hr = 660 pre-op deficit

Intra-op losses = 4 cc/kg/hr X 70 kg X 1 hr = 280 cc plus blood loss

C. To understand the distribution of crystalloid infused intravenously it is important to have good knowledge of distribution of water in normal individuals. Normal serum sodium and albumin concentration plays an important role in the distribution of water between intracellular and extracellular space.

III. CHOICE OF FLUID

A. Isotonic Crystalloid

Normal Saline (0.9%) (Na 154 meq/L, Cl 154 meq/L, osmolality 308 mosms/L)

Lactate Ringer's (Hartman's solution) (Na 130 meq/L, Cl 109 meq/L, K 4 meq/L, Lactate 28 meq/L, Ca 3 meq/L, osmolality 273 mosms/L)

B. Isotonic Colloid

5% Albumin (Na 145 meq/L and Cl 145 meq/L)

25% Albumin (Na 145 meq/L and Cl 145 meq/L)

6% Hetastarch (Na 154 meq/L and Cl 154 meq/L) (hydroxyethyl starch, Hespan)

C. Hypertonic Saline and hypertonic solutions with colloids

Hypertonic Solutions(3% and 7.5% NaCl) has been extensively investigated in humans. These solutions are inexpensive, are known to promote urine flow, improve microvascular blood flow, improve cardiac output and Blood pressure in hemorrhage patients following trauma but they rapidly decline after discontinuation of therapy. Hypertonic solutions restore regional cerebral blood flow and reduce brain water contents as compared to isotonic and hypotonic solutions.

FAQ

Describe the dynamics of fluid distribution between the intravascular and interstitial compartments?

The intravascular and interstitial fluid spaces compose the extracellular fluid and are in dynamic equilibrium, governed by hydrostatic and oncotic forces. Under normal circumstances, the capillary hydrostatic pressure produces an outward movement of fluid while the capillary oncotic pressure results in reabsorption. The sum of the forces leads to an egress of fluids from arterioles; about 90% of the fluid returns into the venules. The remainder of the fluid is subsequently returned to the circulation via the lymphatic system

How are body water and tonicity regulated?

Antidiuretic hormone (ADH) is a primary mechanism; it circulates unbound in plasma, has a half-life of roughly 20 minutes, and increases production of cyclic adenosine monophosphate in the distal collecting tubules of the kidney. Tubular permeability to water increases, resulting in conservation of water and sodium and production of concentrated urine. Stimuli for the release of ADH include the following:

Hypothalamic osmoreceptors have an osmotic threshold of about 289 mOsm/kg. Above this level, ADH release is stimulated.

Hypothalamic thirst center neurons regulate conscious desire for water, and are activated by an increase in plasma sodium of 2 mEq/L, an increase in plasma osmolality of 4 mOsm/L, and loss of potassium from thirst center neurons and angiotensin II.

Aortic baroreceptors and left atrial stretch receptors respond to volume depletion and stimulate hypothalamic neurons.

Discuss the synthesis of ADH?

ADH, or vasopressin, is synthesized in the supraoptic and paraventricular nuclei of the hypothalamus. It is transported attached to carrier proteins down the pituitary stalk in secretory granules into the posterior pituitary gland (neurohypophysis). There it is stored and released into the capillaries of the neurohypophysis in response to stimuli from the hypothalamus. ADH-producing neurons receive efferent innervation from osmoreceptors and baroreceptors

List conditions that stimulate and inhibit release of ADH

conditions that stimulate and inhibit release of ADH

	Stimulates ADH Release	Inhibits ADH Release
Normal physiologic states	Hyperosmolality	Hypo-osmolality
	Hypovolemia	Hypervolemia
	Upright position	Supine position
	β -Adrenergic stimulation	α -Adrenergic stimulation
	Pain and emotional stress	
	Cholinergic stimulation	
Abnormal physiologic states	Hemorrhagic shock	Excess water intake
	Hyperthermia	Hypothermia
	Increased intracranial pressure	
	Positive airway pressure	
Medications	Morphine	Ethanol
	Nicotine	Atropine
	Barbiturates	Phenytoin
	Tricyclic antidepressants	Glucocorticoids
	Chlorpropamide	Chlorpromazine
Results	Oliguria, concentrated urine	Polyuria, dilute urine

Discuss issues associated with estimating volume status in outpatients?

For most outpatients, the period of fasting would provide a rough estimate of volume deficit. A patient's hourly metabolic requirement is roughly 4 mL/kg for the first 10 kg, 2 mL/kg for the second 10 kg, and 1 mL/kg for the remainder of their weight. The actual deficit may be less than calculated due to renal conservation of fluids. Factors that may result in volume depletion include chronic hypertension, diuretic use, diabetes mellitus, alcohol ingestion, bowel preps (2-4 L may be lost), etc.

Discuss estimating volume status in acutely ill patient

Acutely ill patients are often hypovolemic due to bleeding, peritonitis, pancreatitis, sepsis, gastrointestinal losses, traumatic injury, inadequate fluid replacement, etc.

Physical findings suggesting inadequate intravascular fluid include dry mucous membranes, loss of skin turgor, capillary refill greater than 2 seconds, postural hypotension, tachycardia, and oliguria (less than 0.5 mL/h in adults, less than 1 mL/h in children). If present, central venous or (rarely) pulmonary artery catheters may assist in the diagnosis of hypovolemia.

Useful laboratory values in the assessment of volume status include hemoglobin, hematocrit, electrolytes, blood urea nitrogen (BUN), creatinine, proteins, as well as urine osmolality, specific gravity, and sodium concentration.

Are there distinct advantages to using colloids to resuscitate a patient?

Colloid advocates claim that because these solutions have an intravascular space half-life of 3-6 hours (much greater than crystalloids), they are superior resuscitation fluids. There may be some benefit to using them in profoundly hypovolemic patients, but often these patients require blood transfusions. Colloids are more expensive

than crystalloids and when compared to crystalloids in a controlled fashion, have not been shown to improve outcomes. Though only a third of a liter of crystalloid remains in the intravascular space, if given in sufficient quantities (replacing losses 3 to 1), crystalloids are excellent resuscitation fluids. It should be noted that dehydrated patients suffer from fluid losses in both intracellular and extracellular compartments and crystalloids will replete both compartments.

What colloidal solutions are available?

COMMONLY ADMINISTERED COLLOID

Colloid	Benefits and Risks
Albumin (5% 25%)	Expensive; allergic reactions; question its use where there is a loss of alveolar integrity
Hetastarch	Currently constituted in either NS or RL; administer less than 20 mL/kg to avoid antiplatelet effects; renally excreted; increases serum amylase
Dextran (40 or 70)	Anaphylactic reactions; interferes with platelet function and crossmatching; increases hepatic transaminases

What is the normal range for serum osmolality?

Different sources quote different ranges, but, in general, normal serum osmolality ranges between 285 and 305 mOsm/L. A quick rough estimate is to double the sodium concentration. A more accurate estimate of osmolality can be obtained using the following equation: $2 \times [\text{Na}^+] + [\text{Glucose}]/18 + \text{BUN}/2.8$, where the values in the brackets are the concentrations of the substances (sodium in mEq/L, glucose and BUN in

g/L).

How do you estimate fluid loss during a surgical procedure?

This is an inexact process. Gather as much information as you can. Estimating the volume found in suction canisters and subtracting whatever volume has been used for irrigation can assess blood loss. Surgical sponges can be weighed; a large laparotomy sponge can hold more than 100 mL of blood. Blood loss can also be occult, soaking into surgical drapes or running down on to the floor. Measure gastric aspirate. Significant peritoneal fluid collections may require replacement. Especially in intra-abdominal procedures, insensible losses should be taken into consideration. For moderate surgical trauma (e.g., open cholecystectomy), estimate about 4 mL/kg/h; for more extensive procedures, such as enteral resections, estimate 6-8 ml/kg/h; and for major vascular resections (e.g., abdominal aortic aneurysms), estimate 10-20 ml/kg/h.

What is meant by third-space losses? What are the effects of such losses?

In certain clinical conditions, such as major intra-abdominal operations, hemorrhagic shock, burns, and sepsis, patients develop fluid requirements that are not explained by externally measurable losses. Losses are internal, a temporary sequestration of intravascular fluid into a functionless "third space," which may not readily participate in the dynamic fluid exchanges at the microcirculatory level. The volume of this internal loss is proportional to the degree of injury, and its composition is similar to plasma or interstitial fluid. The creation of the third space necessitates further fluid infusions to maintain intravascular volume, adequate cardiac output, and perfusion, and third space fluids will persist until the patient's primary problem has resolved.

Is blood pressure a good sign of hypovolemia?

Blood pressure is not significantly affected until approximately 30% of blood volume is lost. Early compensatory mechanisms, including peripheral vasoconstriction and tachycardia, may mask significant volume loss.

What clinical findings support a diagnosis of hypervolemia?

The patient may have rales upon lung auscultation, frothy secretions in the endotracheal tube, edematous mucous membranes and conjunctiva (though edematous conjunctiva by itself is not enough to make the diagnosis, especially when the patient has been prone), polyuria, and peripheral edema. Like hypovolemia, hypervolemia is best diagnosed when a constellation of findings, and not just a single finding, is present.

You have concerns that an edematous patient may develop an obstructed airway from edema after extubation. Can any test assist your decision making?

If the patient otherwise meets extubation criteria and is spontaneously breathing, disconnect the endotracheal tube (ET) from the ventilator circuit, occlude the tube lumen, deflate the ET cuff, and assess whether air movement around the tube is adequate. Failure to breathe around the deflated tube suggests intubation may not be wise. Even if air flows around the deflated cuff, if the patient was a difficult intubation, it may be best to leave the patient intubated, elevate the patient's head, and reassess at a later time.

References

1. What is the average human blood volume?

The estimated total blood volume (EBV) of an average adult male is about 75 mL/kg and that of the average female about 65 mL/kg. In a 70-kg man, of the approximate 5-L blood volume, 3 L is plasma and 2 L is red cells.

2. What are the physiologic adaptations to acute normovolemic anemia?

During surgery, acute blood loss is usually replaced with crystalloid solutions, resulting in acute normovolemic hemodilution. Compensatory changes include tachycardia, increased cardiac output, redistribution of blood to the tissues that are oxygen supply dependent (e.g., heart and brain), and increased oxygen extraction.

3. What is an acceptable minimum preoperative hematocrit?

There are numerous reports of severe anemia being well tolerated in surgical patients. Elective surgery has been successfully performed with preoperative hemoglobin as low as 6 gm/dL. Patients with chronic renal failure have had successful outcomes despite significant levels of anemia. Patients refusing blood products for religious reasons have tolerated profound decreases in hemoglobin concentration.

Emphasis should be placed on considering whether patients have sufficient oxygen-carrying capacity to meet their metabolic demands. Factors that are important in this consideration include the patient's age, general health, coexisting (particularly cardiopulmonary) disease, the setting (e.g., is the patient septic, having acute organ dysfunction, or acutely hemorrhaging), and predictions of expected or ongoing blood losses.

Although the following recommendations are insufficiently supported by strong clinical evidence, they provide rough guidelines. In the absence of cardiovascular instability and without substantial ongoing bloodloss, perioperative transfusion should be considered at these hematocrit

values:

- **Healthy patients:** 18-21%
- **Patients with well-compensated systemic disease:** 24%
- **Patients with symptomatic cardiac or cerebrovascular disease:** 30%

4. How is blood loss estimated?

Estimating blood loss is challenging. Measure blood suctioned into canisters and estimate loss into surgical sponges and drapes. Blood loss may be occult; blood may be hidden in surgical drapes or in body cavities behind surgical packing. Large hematomas and femur fractures, for example, may have large inapparent blood losses. When blood loss is vigorous and ongoing, serial hematocrits are necessary.

6. For what infectious agents is donor blood screened?

Blood is tested for the following infectious disease markers:

- **Syphilis**
- **Viral hepatitis:** Antibody to hepatitis B core antigen; hepatitis C antibody
- **Human immunodeficiency virus (HIV) retrovirus:** Antibodies to HIV: anti-HIV-1, anti-HIV-2, and HIV antigen p24
- **Human T cell lymphotropic virus type I/II:** Antibody to HTLV I and II

7. What types of red cell products are available?

- **Red blood cells:** Indicated for increasing oxygen-carrying capacity and volume expansion during significant hemorrhage. A unit with citrate, phosphate, dextrose-adenine (CPDA-1) anticoagulant has a volume of 300-350 mL with a hematocrit of about 70%. Additives may reduce the hematocrit to 60%.
- **Red blood cells deglycerolized:** Rare blood types may be frozen for prolonged storage.

- **Leukocyte-reduced red blood cells:** For patients with previous febrile transfusion reactions.
- **Washed red blood cells:** Useful in patients who have had severe transfusion reactions.
- **Whole blood:** Rarely used because component therapy is the best practice and conserves a precious resource.

8. What is the usual survival time for stored red blood cells? What is a storage lesion?

Whole blood or red cells may be stored for 35 days when preserved with CPDA-1 and for 42 days when AS-1 (Adsol) or AS-3 (Nutrice) is added.

Changes in stored blood that reduce post-transfusion viability include reduction in red cell deformability, altered red cell adhesiveness, and reduction in 2,3-diphosphoglycerate and adenosine triphosphate (ATP). Proinflammatory cytokines also accumulate. These "storage lesions" may alter immune function, render the patient prone to infection, and influence mortality and morbidity. In fact, there are concerns that administering blood may not increase tissue oxygen availability! This is particularly true of blood units nearing their expiration dates. At the current level of knowledge, it may be premature to declare banked blood under current storage recommendations unsafe, but it reinforces the concept that the risks and benefits of every transfused unit should be considered before transfusion is initiated.

9. How much will a unit of red blood cells increase hematocrit? What intravenous solutions are acceptable and unacceptable during transfusion?

One unit of blood will increase the hematocrit by 3%. To dilute blood, the recommended practice is to use normal saline. Saline dilution will facilitate infusion and minimize hemolysis. Hypotonic solutions and calcium-containing solutions should be avoided to prevent hemolysis and clot formation, respectively.

A standard blood administration set with a pore size of 170 μm is recommended. Because surgical patients are prone to hypothermia, the best practice is to warm all blood units administered intraoperatively. Warming is absolutely essential when patients have cold agglutinins.

10. What are some of the complications of massive blood transfusion?

Massive transfusion is defined as the administration of more than one blood volume within several hours. Complications include:

- Coagulopathy secondary to dilutional thrombocytopenia, lack of labile coagulation factors V and VIII, and disseminated intravascular coagulation
- Metabolic disturbances associated with banked blood, including hyperkalemia, hypocalcemia (citrate toxicity), acidosis, and impaired oxygen delivery due reduced 2,3-diphosphoglycerate
- Hypothermia

11. Review the types of transfusion reactions and their causes?

- Hemolytic transfusion reactions (ABO incompatibility, most commonly caused by clerical errors and transfusion of the wrong unit).
- Anaphylactic reactions are due to binding of IgE and present with bronchospasm, edema, redness, and hypotension and require urgent treatment with epinephrine, fluid infusions, corticosteroids and antihistamines, and other therapies as indicated by severity and progression of symptoms.
- Febrile reactions may be an early sign of hemolytic transfusion reaction (but other symptoms should be present) or bacterial contamination of the blood product. Febrile nonhemolytic

transfusion reactions usually occur in patients who have had prior transfusions; headache, nausea, and malaise are associated symptoms. The reaction is due to leukocyte antibodies and leukocyte-depleted red blood cells may be indicated for these patients. Antipyretics may decrease the symptoms if given prior to the transfusion; meperidine may decrease the severity of chills.

- Acute lung injury is indicative of acute respiratory distress; it is noted within hours after transfusion and is secondary to a transfusion-related inflammatory reaction and altered permeability within the pulmonary circulation.
- Urticarial reactions secondary to mast cell degranulation do not require the transfusion be stopped; antihistamines may be given.

12. Review the clinical manifestations and management of a hemolytic transfusion reaction?

Most reactions occur during or shortly after a transfusion. Clinical manifestations include fever; chills; chest, flank, and back pain; hypotension; nausea; flushing; diffuse bleeding; oliguria or anuria; and hemoglobinuria. General anesthesia may mask some of the clinical manifestations, and hypotension, hemoglobinuria, and diffuse bleeding may be the only signs. When a reaction occurs:

- Stop the transfusion immediately and remove the blood tubing.
- Alert the blood bank and send a recipient and donor blood specimen for compatibility testing.
- Treat hypotension aggressively with intravenous fluids and pressor agents.
- Maintain urine output with intravenous hydration. Mannitol and loop diuretics are used on occasion.
- Massive hemolysis can result in hyperkalemia. Follow serum potassium levels and observe the ECG for changes of hyperkalemia.
- Disseminated intravascular coagulation may occur. The best

treatment is identifying and treating the underlying cause. Follow prothrombin, partial thromboplastin, fibrinogen, and D-dimer levels.

- Check urine and plasma hemoglobin levels and verify hemolysis with direct antiglobulin (Coombs') test, bilirubin, and plasma haptoglobin levels.

KEY POINTS: TRANSFUSION THERAPY

1. There is no set hemoglobin/hematocrit level at which transfusion is required. The decision should be individualized to the clinical situation, taking into consideration the patient's health status.
2. In the operating room, monitor ongoing blood loss closely so as not to "fall behind."
3. If blood is needed in an emergency, type O-packed cells and/or type-specific blood may be used.
4. Hemolytic transfusion reactions can be catastrophic and are most often due to clerical or management system errors. Thoroughly check all blood units before administration.
5. Although rare, transmission of blood-borne diseases can occur with blood transfusion. Transfuse no more blood than absolutely necessary.

13. What alternatives are there to transfusion of donor blood?

- Autologous transfusion (the collection and reinfusion of the patient's own blood)
- Intraoperative collection and reinfusion of blood lost during surgery
- Intraoperative isovolemic hemodilution (the reduction of hematocrit or hemoglobin by withdrawal of blood and simultaneous intravascular replacement with crystalloid)

- Use of hemoglobin solutions

14. What are the indications for platelet therapy?

Platelets may be administered when there is either a quantitative or qualitative deficiency in platelets. Usually platelets will be administered when the platelet count decreases to $50 \times 10^9/L$ but under certain circumstances a higher platelet count is desirable. Examples include craniotomy, when the patient is on medications with antiplatelet effects, uremia, a history of bleeding abnormalities from other causes, large surgical blood losses, etc. A unit of platelets will increase the platelet count by $5-10 \times 10^9/L$.

15. What are the indications for administering fresh frozen plasma (FFP)?

When microvascular bleeding is noted and prothrombin time or partial thromboplastin time exceeds 1.5 the control value, FFP should be considered. The usual dose is 10-15 mL/kg. FFP will also reverse the anticoagulant effects of warfarin (5-8 mL/kg). (Administration of vitamin K will have the same result but will take 6-12 hours to become effective.) Volume expansion is not an indication for FFP.

16. What is cryoprecipitate? When should it be administered?

Cryoprecipitate is the cold-insoluble white precipitate formed when FFP is thawed at $1-6^{\circ}C$. It is removed by centrifugation, refrozen, and thawed immediately prior to use. Cryoprecipitate contains factor VIII, von Willebrand's factor, fibrinogen, and factor XIII. It is used to replace fibrinogen, factor VIII deficiencies, and factor XIII deficiencies. It has been used to treat von Willebrand's disease (unresponsive to desmopressin) and hemophilia. There is now a purified factor VIII concentrate more appropriate for use in these selected problems. One unit of cryoprecipitate per 10 kg body weight will increase fibrinogen levels by 50 mg/dL. Since cryoprecipitate lacks factor V, for the treatment of disseminated intravascular coagulation, FFP is also necessary.

Suggested Reading

American Society of Anesthesiologists Task Force on Perioperative Blood Transfusion and Adjuvant Therapies. Practice guidelines for perioperative blood transfusion and adjuvant therapies. Approved October 22, 1995, last amended October 25, 2005. Available at <http://www.asahq.org/publicationsAndServices/practiceparam.htm#blood>. Accessed January 22, 2006.

Circulatory Shock

Description

Inadequate tissue perfusion resulting in organ dysfunction. Classified as:

- Hypovolemic shock:
 - Cardiac output is severely reduced due to loss of intravascular volume.
 - Blood loss is most common cause.
- Cardiogenic shock:
 - Cardiac output is severely compromised by loss of myocardial muscle function, valvular dysfunction, or arrhythmia.
 - Myocardial infarction is most common cause.
- Obstructive shock:
 - Cardiac output reduced due to obstruction of venous return to the heart (vena cava syndrome), compression of the heart, (pericardial tamponade, tension pneumothorax), or disrupted cardiac outflow (aortic dissection, pulmonary embolism).
- Distributive shock:
 - Reduced systemic vascular resistance and effective circulating volume. Dilated peripheral vessels shunt blood flow away from perfusion of critical organs.
 - Most commonly due to sepsis and inflammation, which cause vasodilation and increased capillary permeability.
- Neurogenic:
 - Venous pooling in periphery (most often owing to spinal shock or drug overdose); behaves much like hypovolemic shock.

Etiology

- Hypovolemic shock:
 - Hemorrhagic: Blood loss owing to trauma or gastrointestinal bleeding
 - Nonhemorrhagic: 3rd-space loss of plasma volume (pancreatitis, bowel obstruction, infarction, anaphylaxis, diarrhea, burns)
- Cardiogenic shock:
 - Acute myocardial infarction (>40% of left ventricular myocardium)
 - Arrhythmia (heart block, ventricular tachycardia, atrial fibrillation with rapid ventricular response, etc.)
 - Acute valvular dysfunction: Mitral valve due to papillary muscle rupture following inferior myocardial infarctions or after rupture of chordae; aortic or mitral valve dysfunction, for example in bacterial endocarditis
 - Ventricular septal rupture following anterior/septal myocardial infarction
- Obstructive shock:
 - Pericardial tamponade
 - Inferior/Superior vena caval obstruction usually owing to neoplasms
 - Aortic dissection
 - Massive pulmonary embolism
- Distributive shock: High-output shock is a result of sepsis, toxic shock, burns, or anaphylaxis. Severe noninfectious systemic inflammatory response can be due to sepsis, but also can independently cause distributive shock.
- Neurogenic: Venous pooling when loss of sympathetic innervation causes loss of venous tone (e.g., acute spinal injury, general or spinal **anesthesia**, or overdose of sedative drugs)

Diagnosis

Signs and Symptoms

- Signs/symptoms of underlying disease
 - Upper gastrointestinal (UGI) tract bleeding (hematemesis, melena)
 - Sepsis (fever, chills, symptoms of underlying infection such as dysuria, costovertebral angle tenderness in urosepsis)
 - Myocardial infarction (chest pain, diaphoresis, nausea, vomiting, S₄ or S₃ gallop, new heart murmur, rales)
 - Jugular-venous distention (JVD), pulsus paradoxus in pericardial tamponade
- Signs/Symptoms of underperfusion of organ systems:
 - Brain: Confusion, anxiety, agitation, coma
 - Kidney: Oliguria
 - Skin: Peripheral cyanosis, sluggish capillary refill, mottling, and/or coolness; may be overly perfused (flushed and warm) in high-output (septic) shock
 - Gastrointestinal tract: Absence of bowel sounds
 - Circulation: Thready pulses, tachycardia, hypotension, secondary cardiac ischemia (ST depression), or heart failure.

Tests

- Pulmonary artery (Swan-Ganz) catheterization
 - Serial measurement of cardiac output; central venous, pulmonary arterial, and pulmonary arterial occlusion pressures (left atrial pressure); and vascular resistance
 - Mixed venous blood gases can be drawn from the catheter.

- Was standard of care, but recent studies suggest increased mortality with use, thus this practice is currently controversial (1)[B].
- ECG:
 - Valvular failure
 - Pericardial effusions
 - Echo-guided pericardiocentesis
- Endoscopy if suspect GI bleed

Lab

- Specific to shock:
 - Elevated lactate (>2 mmol/L) suggests anaerobic metabolism from underperfusion.
 - Reduced mixed venous P_{O_2} obtained from the pulmonary artery indicates vigorous extraction of oxygen from tissues owing to underperfusion.
- For underlying diseases responsible for shock:
 - ECG, creatine kinase levels (serial), troponin levels (serial)
 - Arterial blood gases
 - Gram stain and culture of infected sites
 - Blood cultures
 - CBC (serial determination of hemoglobin and hematocrit in bleeding patients)
 - Type and cross bleeding patients

Imaging

Depends on suspected underlying etiology

Treatment

Initial Stabilization

- Support airway, breathing, circulation

- Continuous ECG monitoring with frequent assessment of BP, respiratory status, and urine output

General Measures

- Therapy involves aggressive volume resuscitation with control of the underlying etiology and monitoring of end-organ perfusion.
- Maintain $\text{Sao}_2 >92\%$ with supplemental oxygen. Intubate and mechanically ventilate if patient cannot oxygenate adequately or has markedly increased breathing effort.
- Correct plasma volume deficits rapidly with crystalloids and/or packed RBCs.
- Correction of coagulation derangement by administration of coagulation factors (fresh frozen plasma, cryoprecipitate) and platelets if coagulopathy (prolonged prothrombin time, partial thromboplastin time, or platelet count $<50 \times 10^9/\text{L}$) is present in a patient who is bleeding.
- Tachyarrhythmias (other than sinus tachycardia) should be promptly corrected by electrocardioversion. Insert transvenous pacemaker to correct bradyrhythmias.
- Vasopressors to correct hypotension or low cardiac output owing to myocardial failure or hypotension caused by low vascular resistance
- Treat underlying conditions, for example:
 - Hemorrhagic:
 - Trauma
 - GI blood loss may require endoscopic or surgical intervention
 - Sepsis:
 - Empiric antibiotic therapy
 - Hydrocortisone and vasopressin improve BP. Consider when patients do not improve in response to fluids, antibiotics, and low doses of vasopressors.
 - Cardiogenic shock:

- Therapy should help reduce cardiac ischemia (oxygen, nitrates) and accomplish rapid reperfusion of injured, but potentially viable, myocardium. A balloon pump may temporize by providing improved coronary blood flow during and after diagnostic testing and revascularization therapy.
- If shock is a result of acute failure of the mitral or aortic valve, surgical valve replacement may be lifesaving.

Special Therapy

IV-Fluids : Infusion of crystalloids and colloids to support BP

Medication (Drugs)

- In addition to specific treatment of underlying condition, use medications to support perfusion .
- Dopamine IV effects depend on dosing:
 - Augments contractility, cardiac output and heart rate at moderate doses via β -1 effects. Increases BP by a combination of increased cardiac output and, at higher doses, vasoconstriction (via α -effects).
- Norepinephrine IV: Augments BP by increasing vascular resistance (α). Cardiac output in septic shock maintained/increased (β -1).
- Phenylephrine IV: Pure α -agonist that increases BP via vasoconstriction
- Dobutamine IV: Inotropic agent with β -1 effects:
 - Augments contractility and cardiac output (β -1); does not raise BP because of vasodilation (β -2).
- Vasopressin: Used in patients refractory to inotropic agents. Increases vascular smooth muscle tone, may increase mean arterial pressure (3)[B]
- Activated protein C: In the PROWESS study, APC was shown to reduce mortality from severe sepsis at 28 days from 31–25% (4)[B].

Treatment of DIC is critical to prevent sepsis-related organ failure. Activated protein C may help by effects on microcirculation ([5,6](#))[B].

- Corticosteroids: Low doses of hydrocortisone and fludrocortisone may reduce mortality in shock from sepsis ([7,8](#))[B].
- Precautions
 - Myocardial oxygen consumption is increased by increased heart rate, afterload, and contractility.
 - Pressors can increase myocardial ischemia if present and may precipitate or worsen tachyarrhythmias. Use in lowest possible dose for as limited period as possible.

Surgery

In hemorrhagic and septic shock, surgery may be indicated to correct the underlying pathology.

Disposition

ICU care

Prognosis

Mortality is determined by a complex interaction of primary disease causing shock, age, coexisting chronic disease, and shock severity as marked by the number of acute organ system failures that follow shock.

Complications

- Multiple organs may be damaged by underperfusion during shock.
- Acute tubular necrosis
- Ischemic hepatitis
- Ischemic bowel
- Abdominal compartment syndrome
- Adult respiratory distress syndrome
- Encephalopathy and/or cerebrovascular accident

Patient Monitoring

Careful monitoring in intensive care

FAQ

Q: What is the most frequent form of shock?

A: Hypovolemic

Q: What is vasopressin's mechanism of action?

A: Acts at V¹ receptors, producing vasoconstriction and increased reactivity of vascular smooth muscle to catecholamines.

Q: What are the risks associated with the use of PA catheters?

Pneumothorax, ventricular arrhythmias, line infection, venous thrombosis, and pulmonary artery rupture.

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Acute Pain Management

I. Definitions

- Pain (per the International Association for the Study of Pain): An unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage. The pain system provides information on noxious stimuli that allows the body to respond to the injury (immediate, withdrawal from a noxious source; long-term, protection of the injured part by guarding). Pain may be somatic, visceral, neuropathic, or sympathetically maintained.

II. Assessment of pain in adults

In the assessment of pain intensity, rating scale techniques are often used. The most commonly used forms are:

• The **Category Rating Scales** (e.g. none, mild, moderate, severe, unbearable or 1-5)

• The **Visual Analogue Scales (VAS)** (e.g. 10 cm line with anchor points at each end). The VAS has been shown to be more sensitive to change and is therefore more widely used. These scales may also be incorporated into pain *diaries*.

III. Types of Pain

- Somatic pain is described as aching, gnawing, and/or sharp in quality. It is generally well localized and initiated by nociceptor activation in cutaneous and deep tissues. Examples of somatic pain include acute postoperative pain and bone fractures.
- Visceral pain is also associated with tissue injury, specifically infiltration, compression, and distention of viscera. It is usually described as dull and aching in quality and poorly localized, and it

may be referred to other sites. Examples include abdominal pain due to constipation.

- Neuropathic pain results from injury to the peripheral or central nervous system (CNS). Shooting, electrical, or burning pain often is superimposed on a chronic background of burning and aching sensations. Examples include postherpetic neuralgia (PHN) and diabetic neuropathy

IV. Acute pain

Acute pain

- Basics
 - Acute pain follows injury to the body and generally disappears with wound healing. It often is associated with physical signs of autonomic hyperactivity. Unfortunately, the most common reason for unrelieved pain is the failure of medical staff to routinely and systematically evaluate the patient's pain and provide adequate pain relief.
 - Postoperative pain management. Patients expected to have significant postoperative pain should be given adequate treatment before emergence from general anesthesia. The sudden onset of severe pain during recovery can lead to emergence delirium, undesirable cardiovascular effects, and impaired ventilation. Reestablishing analgesia can be more difficult in this circumstance.
 - Postoperative analgesia can be provided with intravenous (IV) boluses or infusions of opioids (nurse or patient-controlled), oral and parenteral NSAIDs, or neuraxial infusion of opioids and/or local anesthetics. Initial analgesia is most often established by titration with incremental IV or epidural bolus doses. Some of the pharmacologic agents often used to treat acute pain are as follows:
 - Nonopioid analgesics

- Aspirin, acetaminophen, and NSAIDs are all useful in the management of acute and chronic pain. These agents differ significantly from opioid analgesics because the intensity of the analgesic effect is more limited; they do not produce tolerance or physical dependence, and they are antipyretic. Both ASA and the NSAIDs work by inhibiting the cyclooxygenase pathway, which in turn stops the production of various prostaglandins that can sensitize free nerve endings to painful stimuli.
- Opioids : Systemic opioids have long been the treatment of choice for acute postoperative pain and for severe chronic pain in combination with adjuvant medications.

Routes of administration include the following:

- Oral: In patients who can take oral medications immediately after surgery, rapid-acting opioids (oxycodone/acetaminophen, hydrocodone, codeine) may be sufficient and provide a good transition to analgesic therapy at home. The oral route is optimal for patients with chronic pain because of its convenience and flexibility.
- Intramuscular: Although commonly used, painful intramuscular (IM) injections are rarely necessary
- Rectal: Although not commonly used, rectal administration is a good alternative that permits rapid absorption and avoids hepatic first-pass metabolism.
- IV: This is the most commonly used route for immediate postoperative pain control. Parenteral opioids are also used when oral medications fail , a patient-controlled analgesia (PCA) system can be used.

- PCA: This device will deliver a preset dose of opioid on demand as an IV bolus. The prescription must specify an incremental dose, a lock-out interval, and a maximum 1-hour total dose. This is a safe tool for postoperative pain management, and it also provides patients with a sense of control.
- Transdermal: Fentanyl is lipid soluble and is readily absorbed through skin. A fentanyl patch is extremely convenient because steady blood levels are attained with a system that is changed only every 3 days. A 25- μ g/hour patch is equivalent to 10 mg of IV morphine administered every 8 hours.
- Epidural: Narcotics can be deposited in the epidural space. This is most safely done in the perioperative setting if the patient is monitored with oximetry. Most commonly, patients are given local anesthetic-opioid infusions, but a bolus dose of preservative-free morphine can be used (2 to 5 mg) and provides analgesia up to 18 to 22 hours. If a patient receives epidural opioids, great care must be taken
- Local anesthetics. Epidural catheters: Epidural analgesia can provide excellent intraoperative as well as postoperative analgesia. They are commonly placed for the following surgeries: thoracic or abdominal surgery, especially in patients with significant underlying pulmonary disease; lower limb surgery where early progression to ambulation is important; lower extremity vascular surgery where a sympathectomy would be advantageous.

Suggested Reading

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The Post anesthesia Care Unit

I. General considerations.

General considerations. For most patients, recovery from anesthesia is uneventful. Nevertheless, when postoperative complications occur, they may be sudden and life-threatening. The postanesthesia care unit (PACU) is designed to provide close monitoring and care to patients recovering from anesthesia and sedation, during the transition to a fully awake state, and before transfer to general hospital wards.

The PACU is staffed by a dedicated team, which consists of anesthesiologists, nurses, and aides. It is located in immediate proximity to the operating room (OR), with access to radiology and the laboratory. Drugs and equipment for routine care (O₂, suction, monitors) and advanced support (mechanical ventilators, pressure transducers, infusion pumps, code cart) must be readily available.

II. Admission to the PACU

- Transport from the OR is carried out under direct supervision of the anesthetist, preferably with the head of the bed elevated or with the patient in the lateral decubitus position to maximize airway patency. Oxygen delivered via facemask is indicated in most patients to prevent hypoxemia due to hypoventilation or diffusion hypoxia
- Report. Upon arrival, vital signs are recorded and the anesthetist provides a complete report to the PACU team. The anesthetist remains in charge of the care of the patient until the PACU team is ready to take over.
- The report includes the following:

- Patient identification, age, surgical procedure, diagnosis, a summary of prior medical history, medications, allergies, and preoperative vital signs.
- Location and size of intravascular catheters.
- Premedication, antibiotics, anesthetic drugs for induction and maintenance, opioids, muscle relaxants, and reversal agents. Vasoactive drugs, bronchodilators, and other relevant drugs administered should be listed.

III. Monitoring.

Monitoring. Close observation of the patient's level of consciousness, breathing pattern, and peripheral perfusion is of utmost importance .

Vital signs are monitored and charted at regular intervals according to the patient's need. Standard monitoring includes respiratory rate, continuous electrocardiogram, manual or automated oscillometric blood pressure, and pulse oximetry. Temperature should also be monitored and recorded.

IV. General complications.

Respiratory and hemodynamic issues are the most frequent complications in the PACU.

Hemodynamic complications

- Hypotension. The differential diagnosis is aided by a review of the patient's history and the intraoperative management. The anesthetist who performed the case can be contacted to help interpret the current events. In most cases, the following differential diagnosis algorithm is helpful.

- Hypovolemia is the most common cause of hypotension in the PACU and administration of a fluid bolus during the initial assessment is generally a safe maneuver. Ongoing hemorrhage, inadequate fluid replacement, osmotic polyuria, and fluid sequestration (intestinal obstruction, ascites) are among the causes of hypovolemia in the PACU. Nonspecific signs include hypotension, tachycardia, tachypnea.
- Impaired venous return occurs when mechanical forces decrease the venous return
- Hypertension is most commonly observed in patients with preexisting hypertensive disease, particularly if antihypertensive medications were held preoperatively.

The management of hypertension can be supplemented or substituted with a fast-onset, short-acting IV medication.

- Alpha and Beta Adrenergic blockers. Labetalol, 5 to 20 mg IV bolus or up to 2 mg/min as IV infusion, esmolol, 10 to 100 mg IV or as an infusion at 25 to 300 µg/kg/minute, and propranolol, 0.5- to 1.0-mg IV increments, may be used.
- Calcium-channel blockers. Verapamil, 2.5- to 5-mg increments IV, or nicardipine infusion initiated at 5 to 15 mg/hour, followed by 0.5 to 2.2 mg/hour.
- Hydralazine, 5 to 20 mg IV, is a pure vasodilator that may induce reflex tachycardia.
- Nitrates. Nitroglycerin, starting at 25 µg/minute IV, is preferentially a venodilator, useful for coexisting myocardial ischemia.
- Dysrhythmias. Increased sympathetic outflow, hypoxemia, hypercarbia, electrolyte and acid-base imbalance, myocardial

ischemia, increased ICP, drug toxicity, thyrotoxicosis, and malignant hyperthermia are possible etiologies of perioperative dysrhythmias.

- Common supraventricular dysrhythmias
 - Sinus tachycardia may be secondary to pain, agitation, hypovolemia, fever, hyperthermia, hypoxemia, hypercarbia, congestive heart failure, and pulmonary embolism. The symptomatic treatment with B-blockers should be instituted only after its etiology is addressed, unless it constitutes a risk for myocardial ischemia.
 - Sinus bradycardia may result from a high neuroaxial anesthetic block, opioid administration (with the exception of meperidine), vagal stimulation, α_2 -adrenergic blockade, and increased ICP. Symptomatic treatment with anticholinergic muscarinic agents, atropine, 0.2 to 0.4 mg IV, or glycopyrrolate, 0.2 mg IV, is indicated when hypotension is present or for severe bradycardia.
 - Paroxysmal supraventricular tachydysrhythmias occur with a higher incidence in patients over 70 years of age; after abdominal, thoracic, or major vascular procedures; and in patients with preoperative premature atrial contractions. They include paroxysmal atrial tachycardia, multifocal atrial tachycardia, junctional tachycardia, atrial fibrillation, and flutter. These rhythms may cause significant hypotension.
 - Synchronized cardioversion should be used if the patient is hemodynamically unstable, as per the ACLS protocol

- Adenosine, 6 mg followed by 12 mg IV, administered rapidly has a high success rate in converting paroxysmal atrial tachycardia to sinus rhythm.
- Verapamil, 2.5- to 5-mg increments IV, or diltiazem 5- to 20-mg IV push or as an infusion (bolus: 0.25 to 0.35 mg/kg IV followed by infusion rate of 5 to 15 mg/hour IV), will slow the ventricular response.
- Amiodarone is the antidysrhythmic of choice to ensure rate control for atrial dysrhythmias in the setting of a decreased myocardial function (congestive heart failure, ejection fraction <40%).
- Stable ventricular dysrhythmias. PVCs and stable nonsustained ventricular tachycardia generally do not require treatment. However, a search for reversible causes (hypoxemia, myocardial ischemia, acidosis, hypokalemia, hypomagnesemia, irritation due to a central venous catheter) should be conducted.

VI. Respiratory and airway complications

The main events were inadequate oxygenation and/or ventilation, upper airway obstruction, laryngospasm, and aspiration.

- Hypoxemia. Causes of hypoxemia include the following:
 - Atelectasis, with subsequent increased intrapulmonary shunting, is a predictable effect of a decreased FRC caused by general anesthesia. Deep breathing (with or without holding) and incentive spirometry are equally effective in rapidly reexpanding small areas of alveolar collapse.

Noninvasive ventilation (NIV) has been shown to further improve atelectasis and oxygenation in postoperative patients.

- Hypoventilation causes hypoxemia by promoting alveolar collapse and increasing the CO_2 partial pressure in the alveolar air.
- Upper airway obstruction is most often caused by inadequate recovery of the airway reflexes and tone.
- Bronchospasm may cause hypoventilation, CO_2 retention, and hypoxemia
- Aspiration of gastric contents
- Pulmonary edem. Cardiogenic edema occurs mostly in individuals with preexisting cardiac disease . It may be precipitated by fluid overload, dysrhythmias, and myocardial ischemia. Evaluation by a cardiologist may be indicated, particularly when aggressive management of conditions such as unstable angina or acute valvular disease is being considered. Inotropic agents, diuretics, and vasodilators are the mainstay of treatment. The use of NIV can obviate the need for intubation in patients with severe hypoxia pending the response to the medical treatment.
- Pulmonary embolism seldom occurs immediately postoperatively.
- Upper airway obstruction may occur during recovery from anesthesia.

Principal signs are the lack of adequate air movement, intercostal and suprasternal retractions, and discoordinate abdominal and chest wall motion during inspiration. Complete upper airway obstruction is silent, whereas partial

obstruction is accompanied by snoring .While 100% O₂ is given by mask, swift airway management is required from the PACU anesthesiologist. Oftentimes, a chin lift, with or without jaw thrust, may be all it takes to relieve the obstruction

- Incomplete recovery from general anesthesia and/or neuromuscular blockade Decreased strength and coordination of the intrinsic and extrinsic airway musculature causes the tongue to fall backward and occlude the airway. Patency is reestablished by inserting a nasal or oral airway, by manually assisting ventilation, or by intubating the trachea.
- Laryngospasm may be precipitated by light anesthesia and irritation of the glottis by secretions, blood, or a foreign body .
- Airway edema may occur during bronchoscopy, esophagoscopy, and surgery of the head and neck.

. Treatment of upper airway edema includes the following:

- Administration of warmed, humidified 100% O₂ by face mask.
- Head elevation and fluid restriction.
- Nebulization of racemic epinephrine 2.25% solution, 0.5 to 1.0 mL in normal saline, or L-epinephrine, 2 mL of a 1:1,000 solution, which may be repeated in 20 minutes if needed.
- Dexamethasone, 4 to 8 mg IV every 6 hours for 24 hours.

- Administration of Heliox (helium:oxygen, 80:20) can dramatically improve gas exchange and the work of breathing while awaiting the response to other medical treatments.
 - Reintubation of the trachea must be considered early because distortion of airway anatomy may occur rapidly, especially with allergic reactions.
- Wound hematoma. Bleeding at the surgical site may complicate thyroid, parathyroid surgery, neck dissections, and carotid endoarterectomy.
- Vocal cord (VC) paralysis may occur after thyroid, parathyroid, thoracic, and tracheal surgery or a traumatic endotracheal intubation.
- Delayed awakening
 - The most frequent cause of delayed awakening is persistent effects of anesthetics
 - Metabolic causes of delayed awakening include hypothermia, sepsis, preexisting encephalopathies, hypoglycemia, and electrolyte or acid-base derangements. Cerebral edema from the inadvertent infusion of hypotonic crystalloid solutions has been reported.
- Neurologic damage may be the result of a stroke or may be due to peripheral nerve injury. Strokes in the perioperative period have an incidence of 0.08% to 2.9% and may be ischemic or hemorrhagic. Early diagnosis of a stroke may be difficult because symptoms such as slurred speech, visual changes, dizziness, agitation, confusion, psychosis, numbness, muscular weakness, and paralysis may overlap with the manifestations of residual anesthetics.

Equipments and Practice in Anesthesia

For Medical Students

Part 1

Airway Management

Devices to Supplementary

Oxygen supply

Refers to an oxygen



Provide Oxygen

cylinder or wall unit that connects to an administration device to deliver oxygen to the patient.

When the patient is receiving oxygen from one of these systems, be sure to check the following equipment:

- Valve handles to open the cylinder
- pressure gauge
- flow meter
- Tubing connecting the oxygen supply to the patient's oxygen administration device

Oxygen therapy is required for

Respiratory failure in many conditions like severe asthma, chronic bronchitis, pneumonia, and myocardial infarction, etc.

4 devices to provide supplementary oxygen:

1. Oxygen supply (cylinder or wall unit)
2. Nasal cannula
3. Face mask
4. Venturi mask

Nasal Cannula

The nasal cannula is a low-flow oxygen administration system designed to add oxygen to room air when the patient inspires.

A nasal cannula provides up to 44% oxygen.

In this low-flow system, inspired air mixes with room air.

The ultimate inspired oxygen concentration is determined by the oxygen flow rate through the cannula and how deeply the patient breathes (tidal volume).

Increasing the oxygen flow by 1 L/min (starting with 1 L/min) will increase the inspired oxygen concentration by approximately 4%:



1 L/min:	21%to24%
2 L/min:	25% to 28%
3L/min:	29%to32%
4L/min:	33%to36%
5 L/min:	37% to 40%
6L/min:	41%to44%

Face Mask:

A simple face mask delivers low oxygen flow to the patient's nose and mouth.

A partial rebreathing mask consists of a face mask with an attached reservoir bag

A face mask can supply up to 60% oxygen with flow rates of 6 to 10 L/min

A face mask with oxygen reservoir (nonrebreathing mask) provides up to 90% to 100% oxygen with flow rates of 9 to 15 L/min. In this system a constant flow of oxygen enters an attached reservoir.

Use a face mask with a reservoir for patients who:

- Are seriously ill, responsive, and have adequate ventilation but require high oxygen concentrations
- May avoid endotracheal intubation if acute interventions produce a rapid clinical effect (eg, patients with acute pulmonary edema, chronic obstructive pulmonary disease [COPD], or severe asthma)
- Have relative indications for endotracheal intubation but maintain an intact gag reflex

- d. Have relative indications for intubation but have clenched teeth or other physical barriers to immediate intubation
- e. The above patients may have a diminished level of consciousness and may be at risk for nausea and vomiting.

Face Mask

Venturi Mask :

enables a more reliable and controlled delivery of oxygen



concentrations from 24% to 50%.

Use the Venturi mask for

- patients who retain carbon dioxide (CC2).
- Patients who have chronic high levels of CO₂ in their blood and moderate-to-severe hypoxemia may develop respiratory depression if the drive stimulating them to breathe (oxygen) is reduced.
- A Venturi mask can accurately control the inspired oxygen concentration.
- Use this mask in patients with COPD, who usually have chronic hypercarbia (high O₂) and mild to moderate hypoxemia.

Delivered oxygen concentrations can be adjusted to 24%, 28%, 35%, and 40% using a flow rate of 4-8 L/min and 40% to 50% using a flow rate of 10-12 L/min.

Observe the patient closely for respiratory depression. Use a pulseoximeter to quickly titrate to the preferred level of oxygen administration.

Venturi Mask



Administration of high oxygen concentrations to patients with end-stage COPD may produce respiratory depression because the increase in P_{aO_2} eliminates the stimulant effect of hypoxemia on the respiratory centers.



Never withhold oxygen from patients who have respiratory distress and severe hypoxemia simply because you suspect a hypoxic ventilatory drive. If oxygen administration depresses ventilation, support ventilation.

Opening the Airway



The tongue may obstruct the airway in unresponsive patients.

Head-tilt, Chin-lift Maneuver: Done in the absence of spinal injury.

Jaw Thrust: Done when spinal injury is suspected.

Airway Adjuncts

Oropharyngeal airway



Insert oropharyngeal airway - Direct method

1. Select correct size of oropharyngeal airway by measuring from the angle of the mouth to the angle of the mandible.
2. Positioning at the head of the patient, open the mouth using tongue-jaw-lift or cross fingers technique
3. Insert a tongue depressor into the mouth and hold down the tongue
4. Holding the oropharyngeal airway with the convex curvature towards the operator, insert the airway directly into the mouth, following the mid-line, until the flange reaches the lips
5. Remove the tongue depressor and inspect the airway position to make sure that the lips are not trapped between the teeth and the airway
6. Using a bag-mask device, provide 2 or 3 ventilations observing for adequate chest rise



The (OPA) displaces the tongue. Oropharyngeal airways keep the airway open during bag-mask ventilation. These devices help suction the mouth and throat and prevent the patient from biting and occluding a tracheal tube.



Gag reflex:

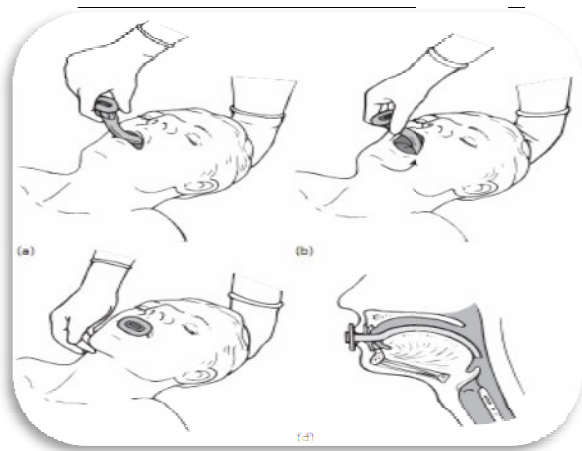
A reflex that causes the patient to retch when the back of the throat is stimulated.

The airway should be used only in the unconscious patient.

Hazards

A long oropharyngeal airway may press the epiglottis against the entrance of the larynx, producing complete airway obstruction.

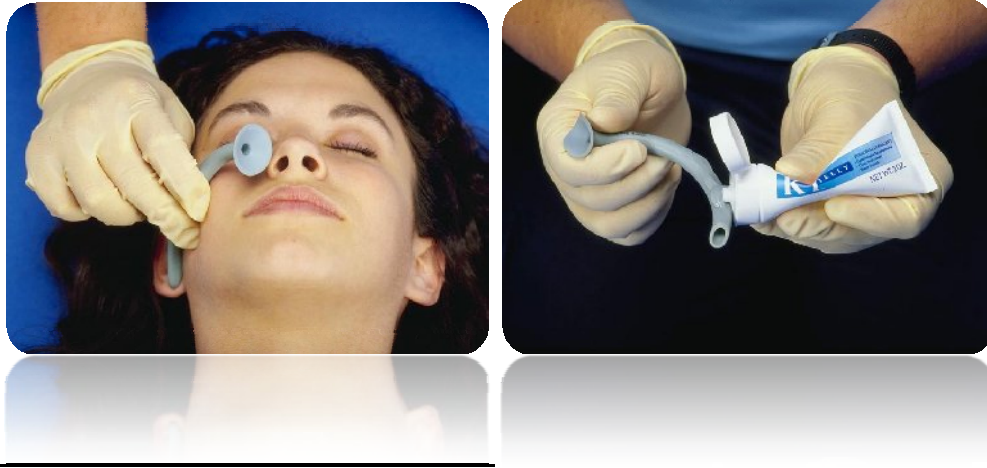
If the airway is not inserted properly, it may push the tongue posteriorly, aggravating upper airway obstruction.



Insert oropharyngeal airway – Indirect method

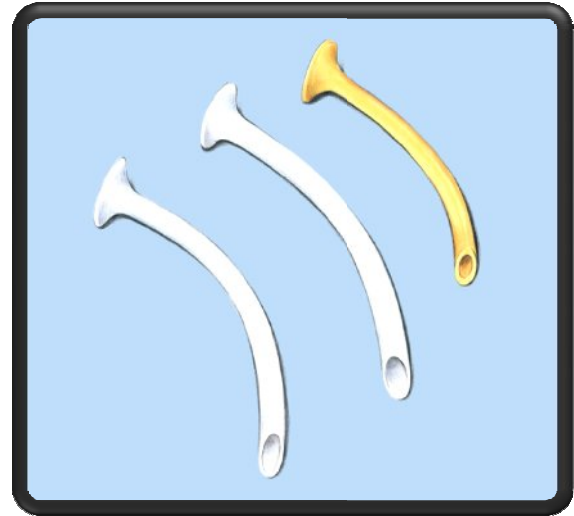
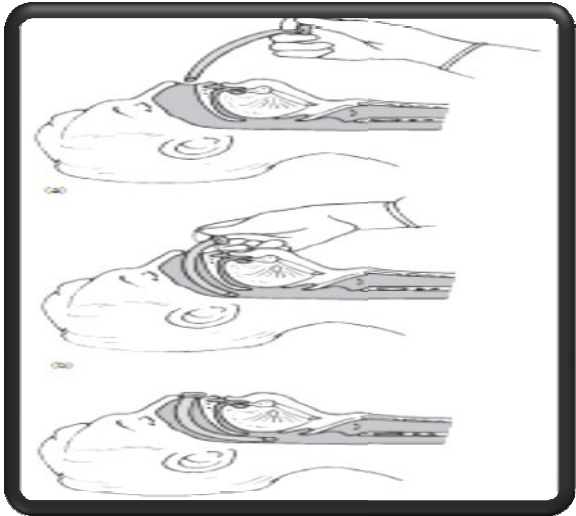
1. Steps 1 & 2, as above
3. Holding the oropharyngeal airway with the concave curvature towards the head of the manikin, insert it into the mouth of the patient
4. As the tip of the airway reaches the posterior pharynx, rotate the airway through 180°, continue to advance the airway until the flange

Nasopharyngeal airway



Insert nasopharyngeal airway

1. Select correct size of nasopharyngeal airway by measuring from the tip of the nose to the tragus of the ear.
2. Inspect the nostrils of the manikin, looking for any obvious obstructions or dried secretions
3. Lubricate the airway with a water-soluble lubricant
4. Hold the airway with the convex curvature towards the operator and the bevel of the airway tip towards the nasal septum
5. Gently insert the airway following the floor of the selected nostril. Use a slight twisting motion if a small amount of resistance is felt. Do not force it.
6. Continue to insert the airway until the flange reaches the nostrils
7. Using a bag-mask device, provide 2 or 3 ventilations observing for adequate chest rise .



Hazards

A long nasopharyngeal airway may enter the esophagus.

Although a nasopharyngeal airway is better tolerated by semiconscious patients, its use may also precipitate laryngospasm and vomiting.

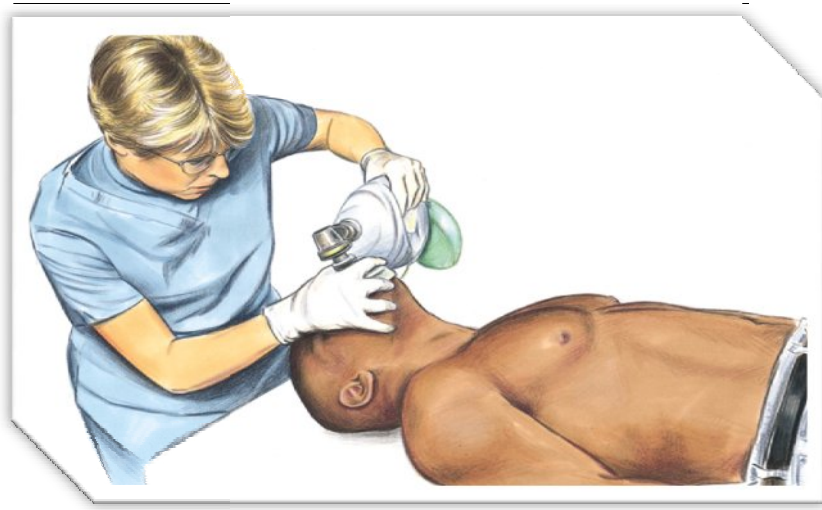
Techniques for Oxygenation and Ventilation

Bag-Mask Ventilation

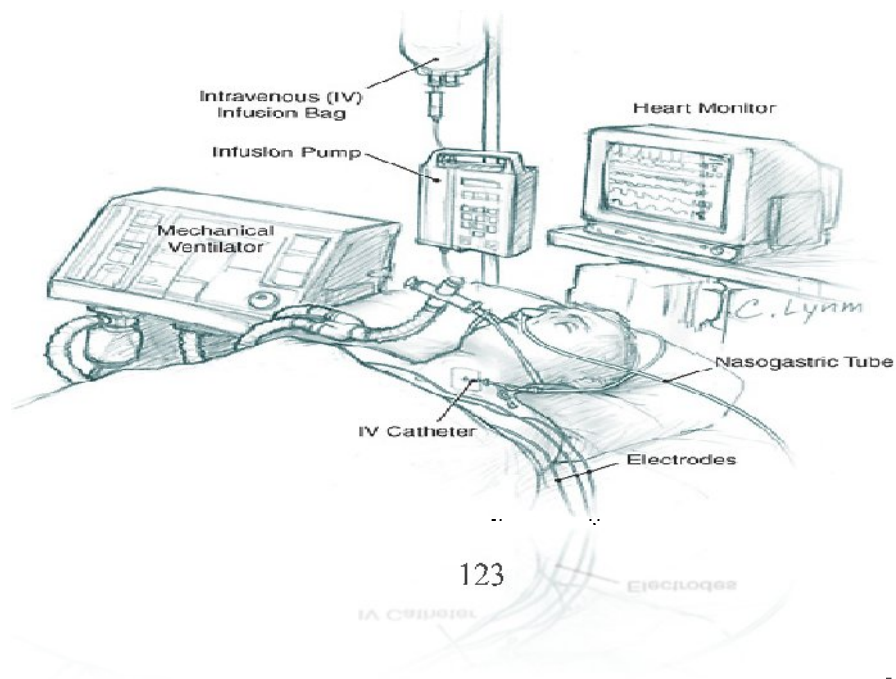
- Consists of a self-inflating bag and a nonrebreathing valve, may be used with a face mask or an advanced airway .
- Can provide bag-mask ventilation with room air or oxygen if they use a self-inflating bag.
- This device provides positive-pressure ventilation when used without an advanced airway and therefore may produce gastric inflation and its complications.

Bag-Mask Ventilation 1 person

1. Attach the correct size of mask to the bag-mask device
2. Attach oxygen tubing and reservoir
3. Set oxygen flow rate to 10 – 15 L/min to provide 100% oxygen
4. Using the CE technique, apply the mask to the face and provide an effective, airtight seal
5. Provide 2 or 3 effective ventilations observing adequate chest rise



MECHANICAL VENTILATION



Indications for mechanical (controlled) ventilation include

- apnea
- hypoventilation (many causes)
- required hyperventilation (to lower intracranial pressure (ICP))
- intra-operative position limiting respiratory excursion, (e.g. prone, Trendelenburg)
- Use of muscle relaxants
- To deliver positive end expiratory pressure (PEEP)

Ventilator parameters include (specific to patient/procedure)

- tidal volume (average 10 mL/kg)
- frequency (average 10/minute)
- PEEP
- FIO₂ (fraction of inspired oxygen)

Types of mechanical ventilators

1.pressure-cycled ventilators

- delivers inspired gas to the lungs until a preset pressure level is reached
- tidal volume varies depending on the compliance of the lungs and chest wall

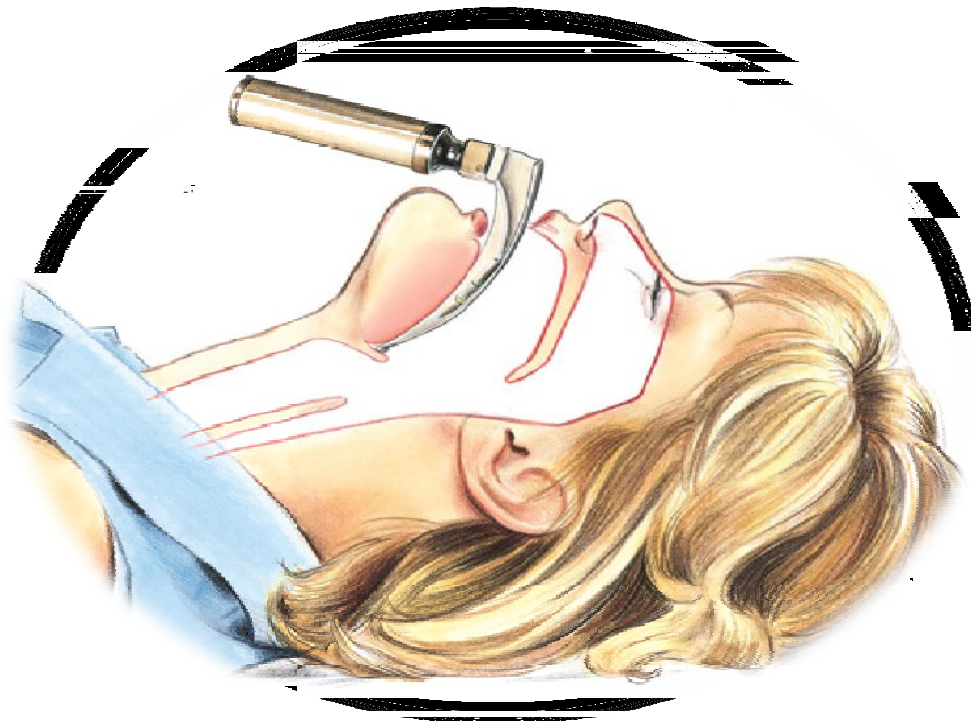
2.volume-cycled ventilators

- delivers a preset tidal volume to the patient regardless of pressure required

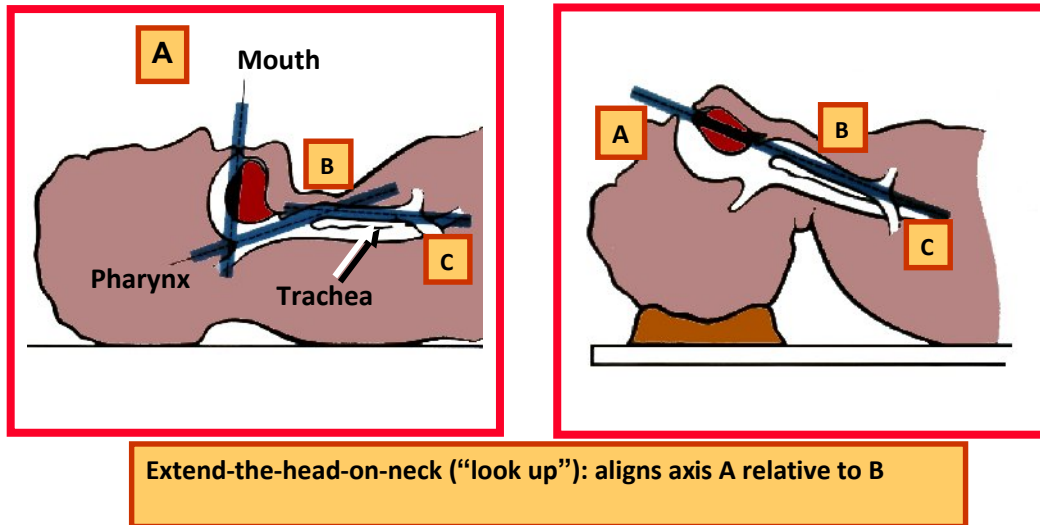
complications of mechanical ventilation

- decreased CO₂ due to hyperventilation
- Disconnection from ventilator or failure of ventilator may result in severe hypoxia and hypercarbia
- decreased blood pressure (BP) due to reduced venous return from increased intrathoracic pressure
- Severe alkalemia can develop if chronic hypercarbia is corrected too rapidly
- Water retention may occur as antidiuretic hormone (ADH) secretion may be elevated in patients on ventilators
- Pneumonia/bronchitis - nosocomial
- pneumothorax
- Gastrointestinal (GI) bleeds due to stress ulcers
- Difficulty weaning

Endotracheal intubation



Aligning Axes of Upper Airway



Equipment Required for Intubation

- 1) bag and mask apparatus (e.g. Laerdal/Ambu)
- 2) mask sizes/shapes appropriate for patient facial type, age
- 3) pharyngeal airways (nasal and oral types available)
- 4) laryngoscope MacIntosh = curved blade (best for adults)
- 5) Magill/Miller = straight blade (best for children)
- 6) Trachelight - an option for difficult airways
- 7) Fiberoptic scope - for difficult, complicated intubations
- 8) Endotracheal tube (ETT): many different types for different indications - inflatable cuff at tracheal end to provide seal which permits positive pressure ventilation - adult female: 7.0 to 8.0 mm - adult male: 8.0 to 9.0 mm - child (age in years/4) + 4 or size of child's little finger = approximate ETT size if nasotracheal intubation,

ETT 1-2 mm smaller and 5-10 cm longer , should always have ETT smaller than predicted size available in case estimate was inaccurate

- 9) malleable stylet should be available
- 10) lubricant and local anaesthetic are optional
- 11) Magill forceps used to manipulate ETT tip during nasotracheal intubation
- 12) suction, with pharyngeal rigid suction tip (Yankauer) and tracheal suction catheter
- 13) syringe to inflate cuff (10 ml)
- 14) stethoscope to verify placement of ETT
- 15) detector of expired CO₂ to verify placement
- 16) tape to secure ETT and close eyelids

Technique of Adult Intubation

1. Assume ventilation is in progress.
2. Connect laryngoscope blade and handle; check light: check cuff on endotracheal tube.
3. Hold laryngoscope in left hand.
4. Insert laryngoscope in right side of mouth, moving tongue to the left.
5. Visualize epiglottis, then vocal cords.
6. Insert ETT (endotracheal tube).
7. Inflate cuff with 4-6 ml of air.

8. Confirm correct placement of ETT clinically by ventilating, visualizing bilateral lung inflations, and auscultation of both sides of the chest and over the epigastrium with a stethoscope.
9. Confirm correct placement of ETT by using an appropriate device such as an ETCO₂ detector or esophageal detector device
10. Fix the tube in place .

Attempts of intubation 30sec.

failed attempts at intubation can make further attempts difficult due to tissue trauma

Plan and prepare (anticipate problems!)

Assess for potential difficulties (see Preoperative Assessment section)

Ensure equipment

Preoxygenation of patient

May need to suction mouth and pharynx first

Proper Positioning for Intubation

FLEXION of lower C-spine and EXTENSION of upper C-spine at atlanto-occipital joint (“sniffing position”) above

CONTRAINDICATED in known/suspected C-spine fracture

Cricoid Pressure Maneuver

During endotracheal intubation applying cricoid pressure by another person. may protect against regurgitation of gastric contents and helps ensure tube placement in the tracheal orifice

Indications for Intubation - the 5 P's

1. Patency of airway required

- decreased level of consciousness (LOC)
- facial injuries
- epiglottitis
- laryngeal edema, e.g. burns, anaphylaxis

2. Protect the lungs from aspiration

- absent protective reflexes, e.g. coma, cardiac arrest

3. Positive pressure ventilation

- hypoventilation – many etiologies
- apnea, e.g. during general anesthesia
- during use of muscle relaxants

4. Pulmonary Toilet (suction of tracheobronchial tree)

- for patients unable to clear secretions

5. Pharmacology also provides route of administration for some drugs

Complications

During Laryngoscopy and Intubation

Mechanical

Dental damage (i.e. chipped teeth)

Laceration (lips, gums, tongue, pharynx, esophagus)

Laryngeal trauma

Esophageal or endobronchial intubation

Systemic

activation of sympathetic nervous system (hypertension (HTN), tachycardia, dysrhythmias) since tube touching the cords is stressful
bronchospasm

Problems with ETT and Cuff

Too long - endobronchial intubation

Too short - accidental extubation

Too large - trauma to surrounding tissues

Too narrow - increased airway resistance

Too soft - kinks

Too hard - tissue damage

Prolonged placement - vocal cord granulomas, tracheal stenosis

Poor curvature - difficult to intubate

Cuff insufficiently inflated - allows leaking and aspiration

Cuff excessively inflated - pressure necrosis

Confirm correct placement of ETT by using an appropriate device such as an ETCO₂ detector or esophageal detector device

Difficult Airway Equipment List

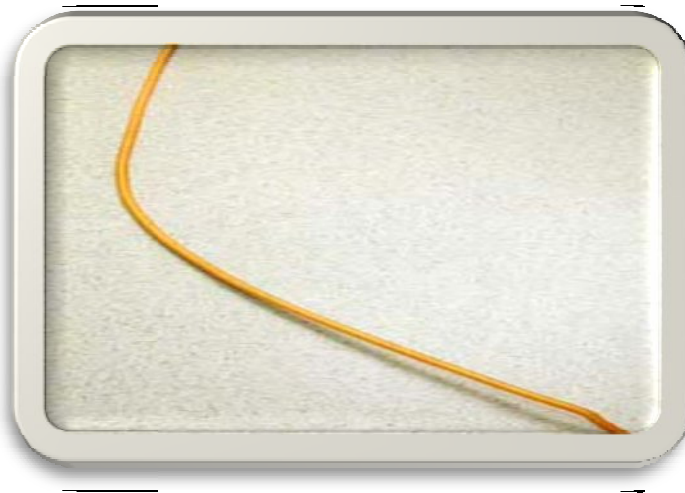
Recommended equipment for routine airway management

- Facemasks
- Oropharyngeal airways: three sizes
- Nasopharyngeal airways: three sizes
- Laryngeal Mask Airways
- Tracheal tubes in a range of sizes
- Two working laryngoscope handles
- Macintosh blades: sizes 3 & 4
- Tracheal tube introducer ("gum-elastic" bougie)
- Malleable stylet
- Magill forceps

Recommended equipment for management of unanticipated difficult intubation

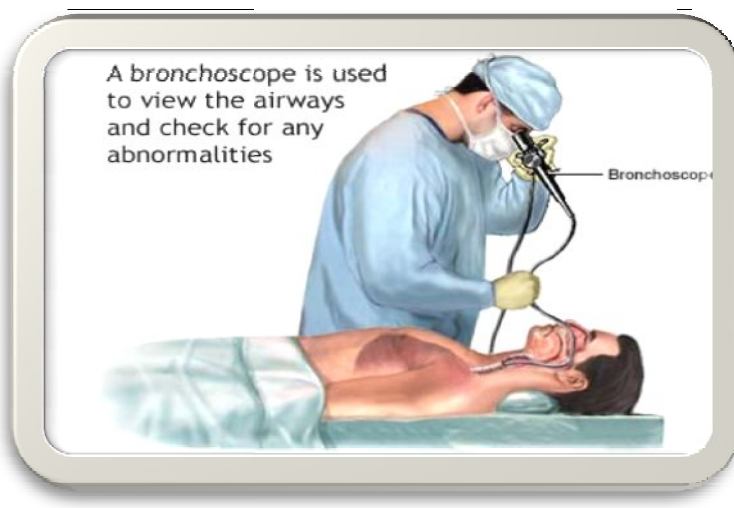
- Bullard type laryngoscope
- Trachlight
- Combitu

- At least one alternative blade (e.g. straight, McCoy)
-



- Intubating Laryngeal Mask Airway (ILMATM) set (size 3, 4, 5 with dedicated tubes and pusher)
- Tracheal tubes – reinforced and microlaryngeal size 5 & 6mm
- Flexible fiberoptic laryngoscope (with portable/battery light source)
- Proseal laryngeal Mask Airway (ProSeal LMATM)
- Cricothyroid cannula (e.g. Ravussin) with High pressure jet ventilation system (e.g. Manujet) OR
- Large bore cricothyroid cannula (e.g. Quicktrach)
- Surgical cricothyroidotomy kit

Tracheal tube introducer ("gum-elastic" bougie)



Flexible fiberoptic laryngoscope

Laryngeal Mask Airway (LMA)



1. Select the appropriate size of LMA for the intubation manikin.
2. Test the LMA cuff, partially deflate it and lubricate the back of the cuff with a water-soluble lubricant.
3. Open the mouth using the “crossed fingers” technique or by performing a tongue-jaw lift; do not hyperextend neck.
4. Insert tube into mouth and place it so that the curvature is the same as that of the pharynx.
5. Insert the tube into the mouth, directing it posteriorly until resistance is felt.
6. Inflate the cuff with the appropriate amount of air corresponding to the size of the tube.
7. Confirm correct placement of the LMA clinically by ventilating, visualizing bilateral lung inflations, and auscultation of both sides of the chest and over the epigastrium with a stethoscope.
8. Confirm correct placement of LMA by using an appropriate device such as an ETCO₂ detector or oesophageal detector device

ENDOTRACHEAL TUBE SUCTIONING

Purpose: To facilitate removal of secretions and maintain patency of the ET tube.

Equipment:

1. Sterile suction catheter of appropriate size (not more than 2/3 the size of the ET tube)
2. Sterile disposable gloves
3. Supplemental oxygen source and oxygen tubing
4. Resuscitation bag
5. Vacuum source and connecting tubing
6. Sterile H₂O
7. Syringe with sterile normal saline
8. Personal Protective equipment

Contraindications/Hazards/Complications:

1. Hypoxia
2. Vagal stimulation: Cardiac arrhythmia
3. Tracheitis.
4. Damage to mucosal membranes.
5. Airway occlusions.
6. Sudden death.
7. Bleeding disorders.

Procedure:

1. Evaluate patient to determine if suction is necessary (i.e. breath sounds, tactile fremitus, etc.).
2. Assemble equipment

3. Prepare resuscitation bag (check that oxygen is on) and verify that suction is set at 60-80 mmHg.
5. Carefully open catheter kit.
6. Pre-oxygenate infant for a brief period (one or two minutes with the FiO₂ increased by 10-15% above the delivered FiO₂).
8. Pick up vacuum control end of catheter with gloved hand then attach to vacuum connecting tubing.
9. Disconnect patient from ventilator and instill 1/2cc of sterile normal saline to irrigate, if necessary.
10. Bag patient with increased FiO₂ (5-10% above set FiO₂); observe color and oxygenation trends during the procedure.
11. Introduce catheter into ETT and watch cm markings on catheter. Match cm markings from ETT and catheter and pass 1 cm. Apply suction for no longer than 15 seconds.
12. Again bag patient with increased FiO₂ (10 - 15% above set FiO₂.
Observe patient's heart rate, color, breath rate, etc.
13. After completion of endotracheal suction, nares and oropharynx should be suctioned gently prior to disposal of the catheter.
14. Once the catheter and glove have been disposed of, the vacuum connecting tubing should be rinsed with sterile H₂O. The oxygen concentration must be returned to pre-suctioning FiO₂, if indicated.
15. Auscultation will determine whether a second suctioning episode is indicated.
16. The color, consistency and amount of secretions aspirated should be documented.

Part 2

Intravenous Access



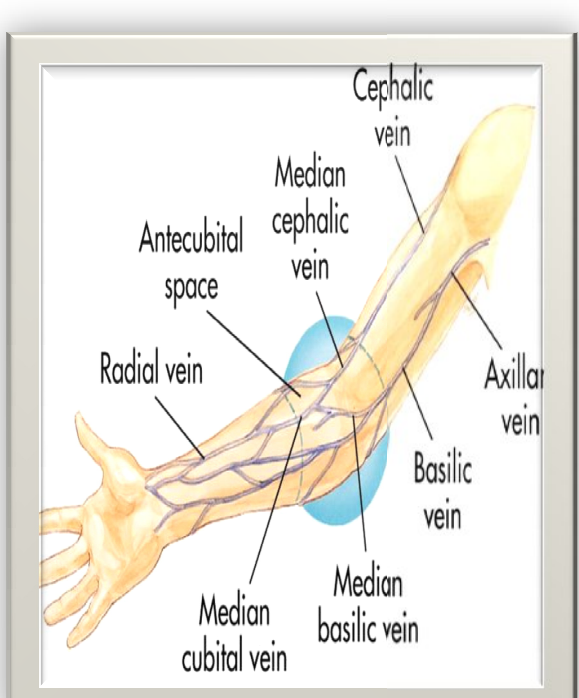
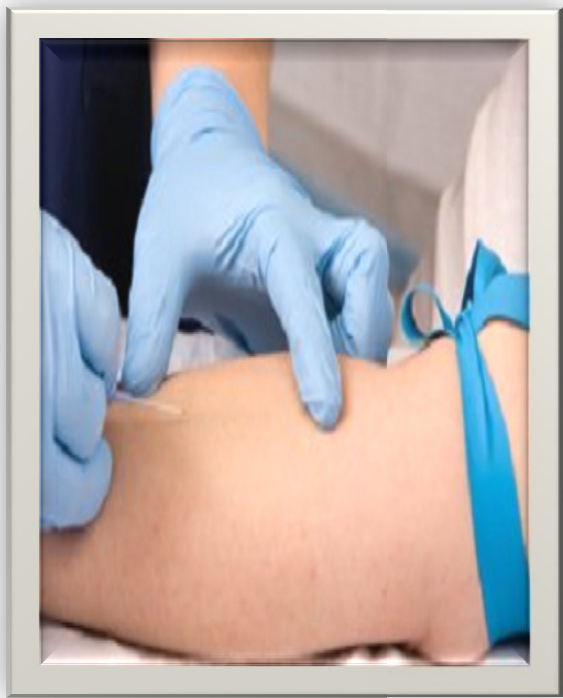
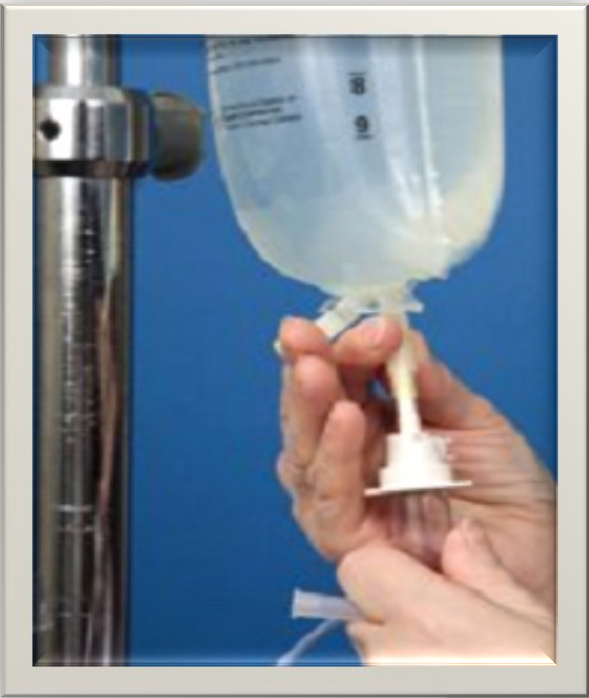
PERIPHERAL VEINS

A. Forearm Vein

1. Apply tourniquet proximally.
2. Locate vein and cleanse the overlying skin with alcohol or povidone-iodine.
3. Anesthetize the skin if a large bore cannula is to be inserted in an awake patient.
4. Hold vein in place by applying pressure on vein distal to the point of entry.
5. Puncture the skin with bevel of needle upward about $\frac{1}{2}$ to 1 centimetre from the vein and enter the vein either from the side or from above.
6. Note blood return and advance the catheter either over or through the needle, depending on which type of catheter-needle device is employed. Remove the tourniquet.
7. Withdraw and remove the needle and attach the intravenous tubing.
8. Cover the puncture site with povidone-iodine ointment and a sterile dressing and tape in place, excluding the point of connection of the intravenous tubing.

Large-bore catheter used for fluid replacement(14 to 16 gauge)

Smaller bore catheter used for “keep open” lines(18 to 20 gauge)



B. External Jugular Vein

1. **Patient in supine, at least 15° head down position, head turned away toward the opposite side.**

2. Cleanse skin, use lidocaine if patient is awake and large bore needle is used.

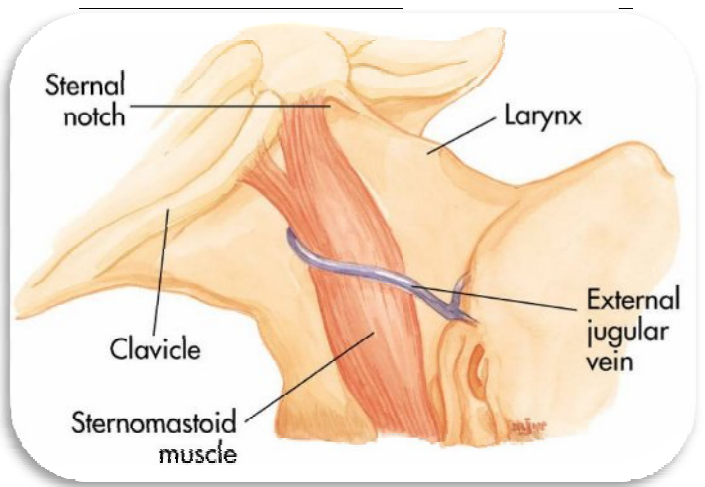
3. Align needle in the direction of the vein with the point aimed toward the ipsilateral shoulder.

4. Make venipuncture midway between angle of jaw and midclavicular line; "tourniquet" the vein lightly with one finger above the clavicle.

5. Note blood return.

6. Advance catheter and remove needle; attach to IV tubing

7. Cover puncture site and affix catheter in place.



Central Line Placement

Indications:

- ❗ Lack of peripheral veins
- ❗ Inability to cannulate peripheral veins
- ❗ Infusion of irritant substances
- ❗ Delivery of incompatible medications in a multi-lumen Catheter
- ❗ Delivery of parenteral nutrition

- 💡 Central venous access for monitoring or temporary transvenous cardiac pacing

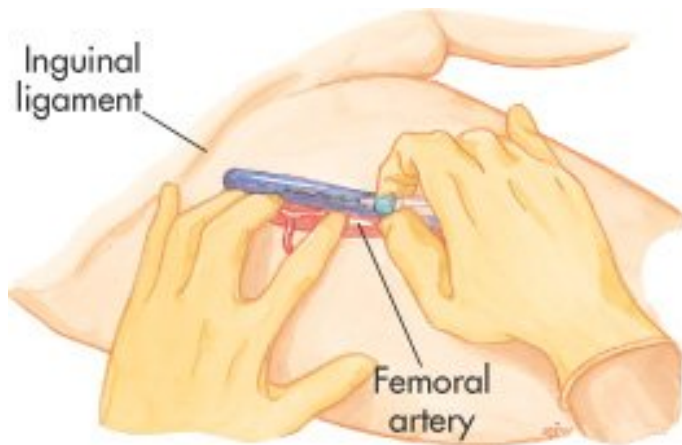
Contraindications:

- 💡 No absolute contraindications
- 💡 Avoid sites of sepsis
- 💡 Apical emphysema or bullae contraindicate infraclavicular or supraclavicular approaches to the subclavian vein
- 💡 Carotid artery aneurysm precludes using internal jugular vein on same side
- 💡 Reconsider central venous cannulation in hypocoagulation and hypercoagulation states or if septicemia is present

Complications:

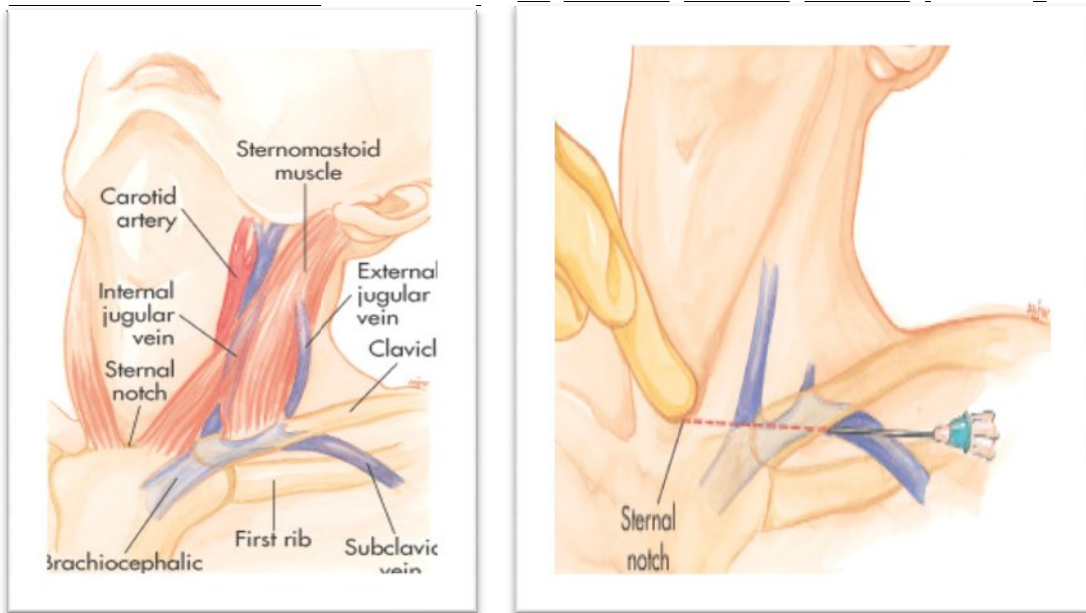
- 💡 Pneumothorax
- 💡 Bleeding (hemothorax)
- 💡 Infection
- 💡 Arterial puncture
- 💡 Hematoma
- 💡 Venous thrombosis
- 💡 Air embolism (reduced with head down position)
- 💡 Catheter embolus
- 💡 Thoracic duct obstruction or injury (reduced with right side insertion)
- 💡 Catheter malposition
- 💡 Pleural effusion
- 💡 Nerve injury
- 💡 Pericardial effusion
- 💡 Myocardial perforation and tamponade
- 💡 Cardiac arrhythmias

A. femoral Vein



1. Cleanse the overlying skin with povidone-iodine;
2. Locate the femoral artery either by its pulsation or by finding the midpoint of a line drawn between the anterior superior iliac spine and the symphysis pubis.
3. Infiltrate the skin with lidocaine if the patient is awake.
4. Make the puncture with the needle attached to a 5 or 10 ml syringe two fingerbreadths below the inguinal ligament, medial to the artery, directing the needle cephalad at a 60° angle with the skin or frontal plane
5. Maintain suction on the syringe and insert the needle slowly until blood appears in the syringe, indicating that the lumen of the vein has been entered.
6. Remove the syringe and insert catheter with the needle more parallel to the frontal plane.
7. Withdraw the needle, leaving the catheter in place.
8. Connect to intravenous tubing.
9. Cover the puncture site with povidone-iodine ointment and a sterile dressing and secure the catheter and tubing in place.
9. Remove needle and connect catheter to IV

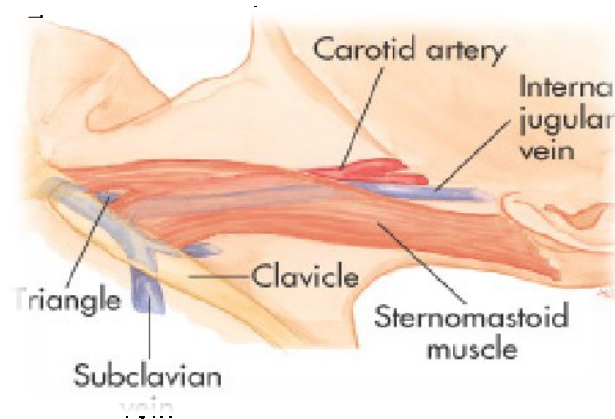
B. Subclavian Vein



1. Patient in supine, at least 15° head down position, head turned away.
2. Cleanse skin, use lidocaine if patient awake.
3. Introduce needle attached to a syringe 1 centimetre below the junction of the middle and medial thirds of the clavicle.
4. Hold the syringe and needle parallel to the frontal plane (the plane of the back of the patient).
5. Direct the needle medially, slightly cephalad, and posteriorly behind the clavicle toward the posterior superior angle of the sternal end of the clavicle.
6. Establish a good point of reference by firmly pressing the fingertip into the suprasternal notch to locate the deep side of the superior angle of the clavicle and directing the course of the needle slightly behind the fingertip.
7. Advance needle while withdrawing plunger of syringe.

8. When blood appears and vein entered, rotate bevel of needle caudally; remove syringe and insert catheter to predetermined depth.
9. Remove needle and connect catheter to IV tubing.
10. Cover puncture site, and affix catheter in place.

C. Internal Jugular Vein, Middle Or Central Route



1. Patient in supine, at least 15° head down position, head turned away.
2. Cleanse skin, use lidocaine if patient awake.
3. Introduce needle attached to syringe in the center of triangle formed by two lower heads of sternomastoid muscle and clavicle.
4. Direct needle caudally, parallel to sagittal plane, at 30° posterior angle with frontal plane.
5. If vein not entered, withdraw needle and redirect it 5 to 10 degrees laterally.
6. Advance needle while withdrawing plunger of syringe.
7. When blood appears and vein entered, remove syringe and insert catheter to predetermined depth.
8. Remove needle and connect catheter to IV tubing.
9. Cover puncture site, and affix catheter in place.

Arterial lines

Indications

- Multiple arterial blood samples
- **Arterial blood gases**
- Continuous blood pressure

Radial Artery • Preferred site: Accessible, compressible, easy to clean and maintain

- Collateral circulation
- Small size limits success

Allen's Test

- Normal response <7 seconds
- Inadequate collateral flow .14 seconds

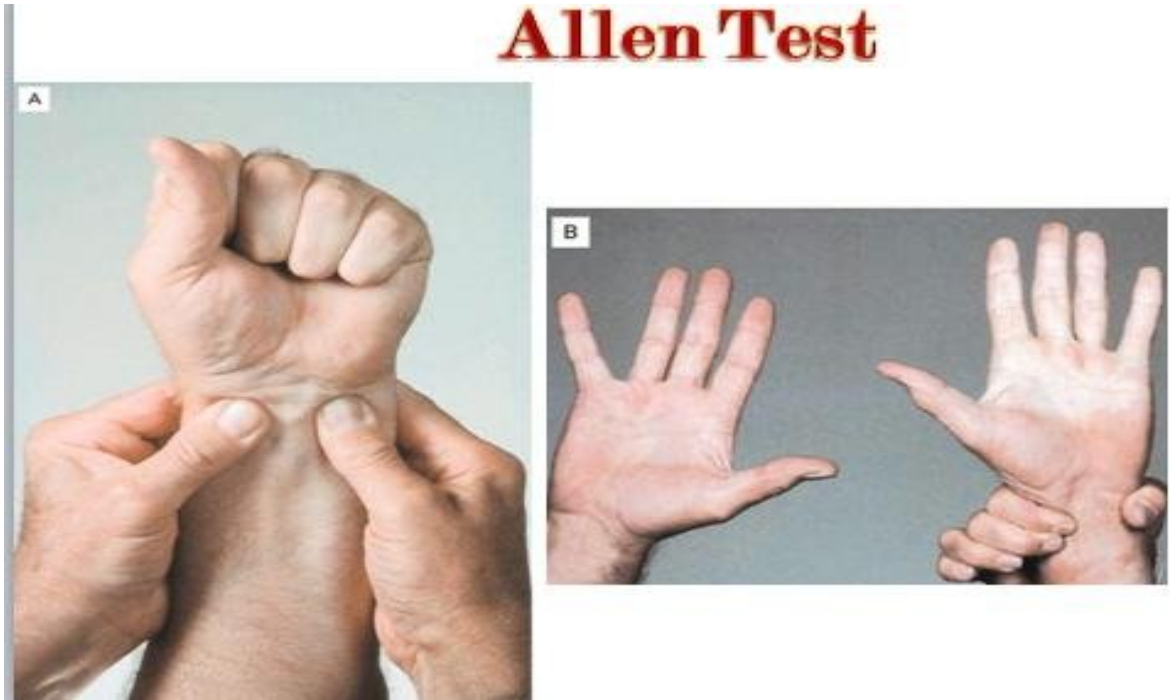
Technique

- Position arm
- Wear gloves
- Prep skin
- Anesthesia
- Inset catheter-needle device at 30 angle to skin surface
- Through and through technique, advance catheter over needle
- Check waveform and blood return
- Suture in place

Complications

- Arterial Occlusions-Arterial injury
- Thrombosis/distal ischemia Ischemic necrosis of digits-rare
- Catheter related septicemia Infection

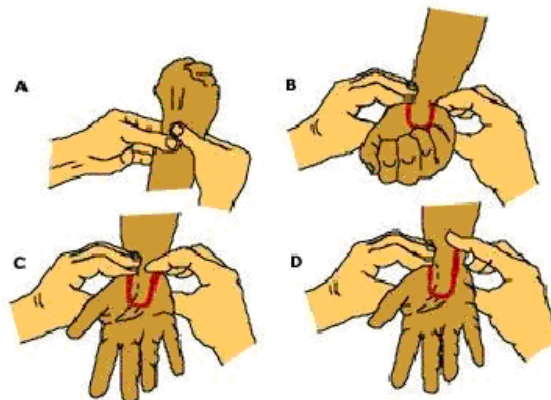
Allen Test



In the Allen test, the patient is instructed to make a fist, which will empty the blood from the hand and fingers

(A). The examiner's thumbs are then pressed down across the thenar and hypothenar eminences to the wrist to occlude the radial and ulnar arteries. The patient then opens the hand, making sure not to overextend the fingers, since this can cause a false positive result. The pressure on the ulnar artery is then released while the radial artery is still compressed

(B). The hand does not fill with blood. Note the paleness of the hand on the right compared with the hand on the left, indicating occlusion of the ulnar artery distal to the wrist (positive test result). If there is prompt return of color to the hand (indicating a negative test result), the pressure on the radial artery is released while the ulnar artery remains compressed.



Part 3

Regional anaesthesia

Regional anesthetic techniques categorized as follows

- Epidural and spinal anesthesia
- Peripheral nerve blockades
- IV regional anesthesia

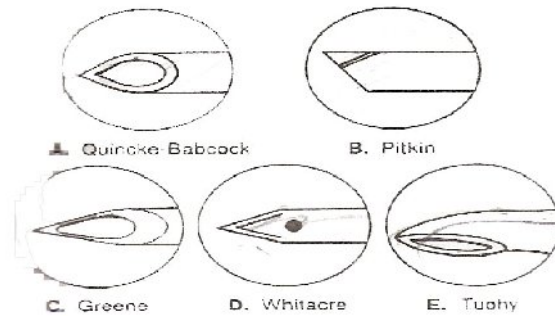
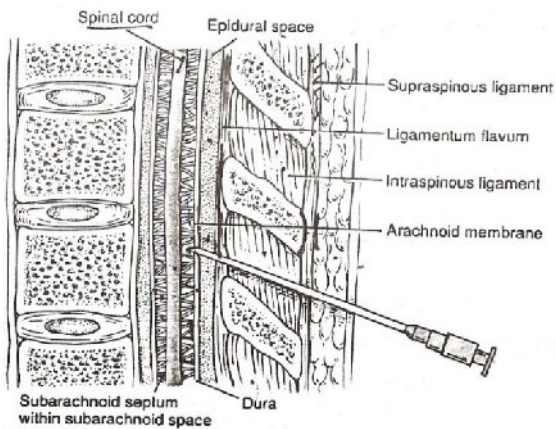
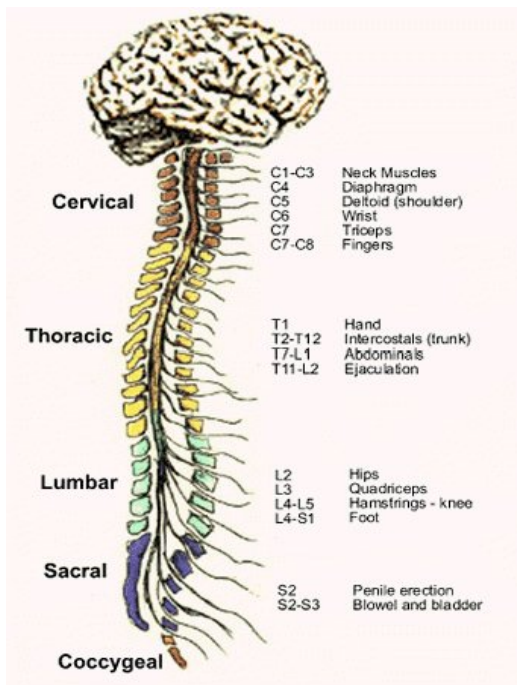
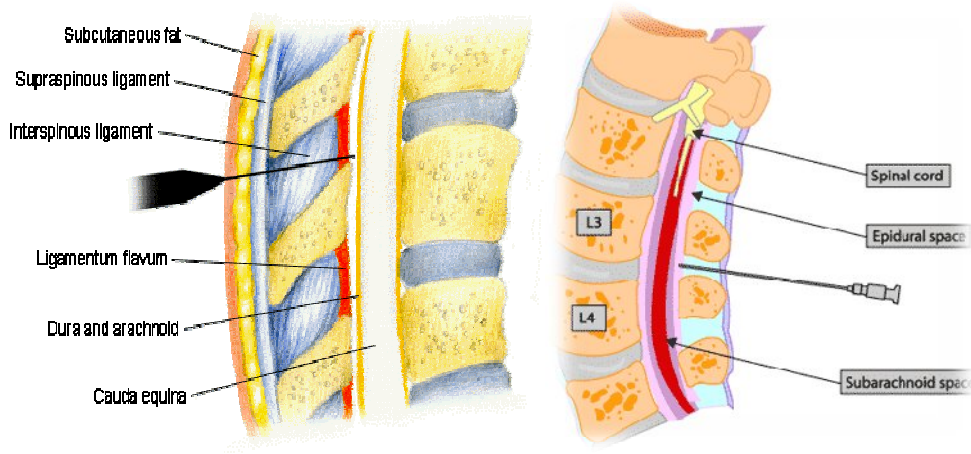


Figure 16-10. Spinal needles.



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EPIDURAL AND SPINAL ANESTHESIA

Anatomy of Spinal/Epidural Area

- spinal cord extends to L2, dural sac to S2
- nerve roots (cauda equina) from L2 to S2
- needle inserted below L2 should not encounter cord, thus L3-L4, L4-L5 interspace commonly used

Structures penetrated

- skin, subcutaneous fat
- supraspinous ligament
- interspinous ligament
- ligamentum flavum (last layer before epidural space)
- dura + arachnoid for spinal anesthesia

Spinal Anesthesia

- relatively small LA dose injected into subarachnoid space in the dural sac surrounding the spinal cord + nerve roots

- LA solution may be made hyperbaric (of greater specific gravity (SG) than the cerebrospinal fluid (CSF) by mixing with 10% dextrose, thus increasing spread of LA to the dependent (low) areas of the subarachnoid space

Epidural Anesthesia

- LA deposited in epidural space (potential space between ligamentum flavum and dura)
- solutions injected here spread in all directions of the potential space; SG of solution does not affect spread
- initial blockade is at the spinal roots followed by some degree of spinal cord anesthesia as LA diffuses into the subarachnoid space through the dura
- larger dose of LA used

Spinal vs. Epidural Anesthesia

spinal

- easier to perform
- smaller dose of LA required (usually < toxic IV dose)
- rapid blockade (onset in 2-5 minutes)
- very effective blockade
- hyperbaric LA solution - position of patient important

epidural

- technically more difficult; greater failure rate
- larger volume/doses of LA (usually > toxic IV dose)
- significant blockade requires 10-15 minutes
- effectiveness of blockade can be variable
- use of catheter allows for continuous infusion or repeat injections
- slower onset of side effects

- position of patient not as important
- SG of LA solution not as important

Complications of Spinal/Epidural Anesthesia

spinal anesthesia

- failure of technique
- hypotension, bradycardia if block reaches T2-4 (sympathetic nervous system (SNS) block)
- post-spinal headache
- extensive spread of anesthetic ("high spinal")
- persistent paresthesias (usually transient)
- epidural or subarachnoid hematoma
- spinal cord trauma, infection

epidural anesthesia

- failure of technique
- hypotension - common
- bradycardia if cardiac sympathetics blocked (only if T2-4 block)
- systemic toxicity of LA (accidental intravenous)
- accidental subarachnoid injection can lead to total spinal anesthesia
- catheter complications (shearing, kinking, vascular or subarachnoid placement)
- epidural or subarachnoid hematoma

Contraindications to Spinal/Epidural Anesthesia

Absolute contraindications include

- lack of proper equipment or properly trained personnel, patient refusal, lack of IV access, allergy to LA, infection at puncture site or

underlying tissues, uncorrected hypovolemia, coagulation abnormalities, raised ICP

Relative contraindications include

- bacteremia, preexisting neurological disease, aortic/mitral valve stenosis, previous spinal surgery, other back problems, severe/unstable psychiatric disease or emotional instability

PERIPHERAL NERVE BLOCKS

Brachial plexus block, ankle block, digital ring block
relatively safe – avoid intraneural injection and neurotoxic agents
provides good operating conditions



Part 4

MONITORING

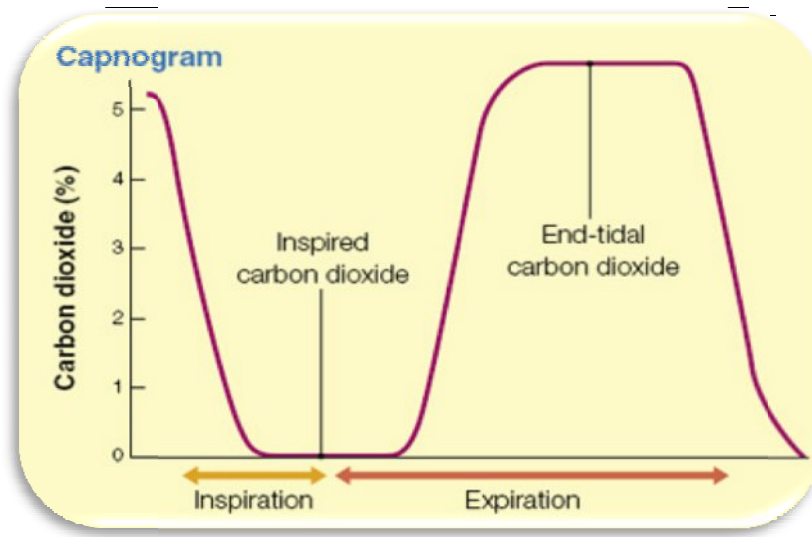
COMMONLY USED MONITORING DEVICES

pulse oximeter



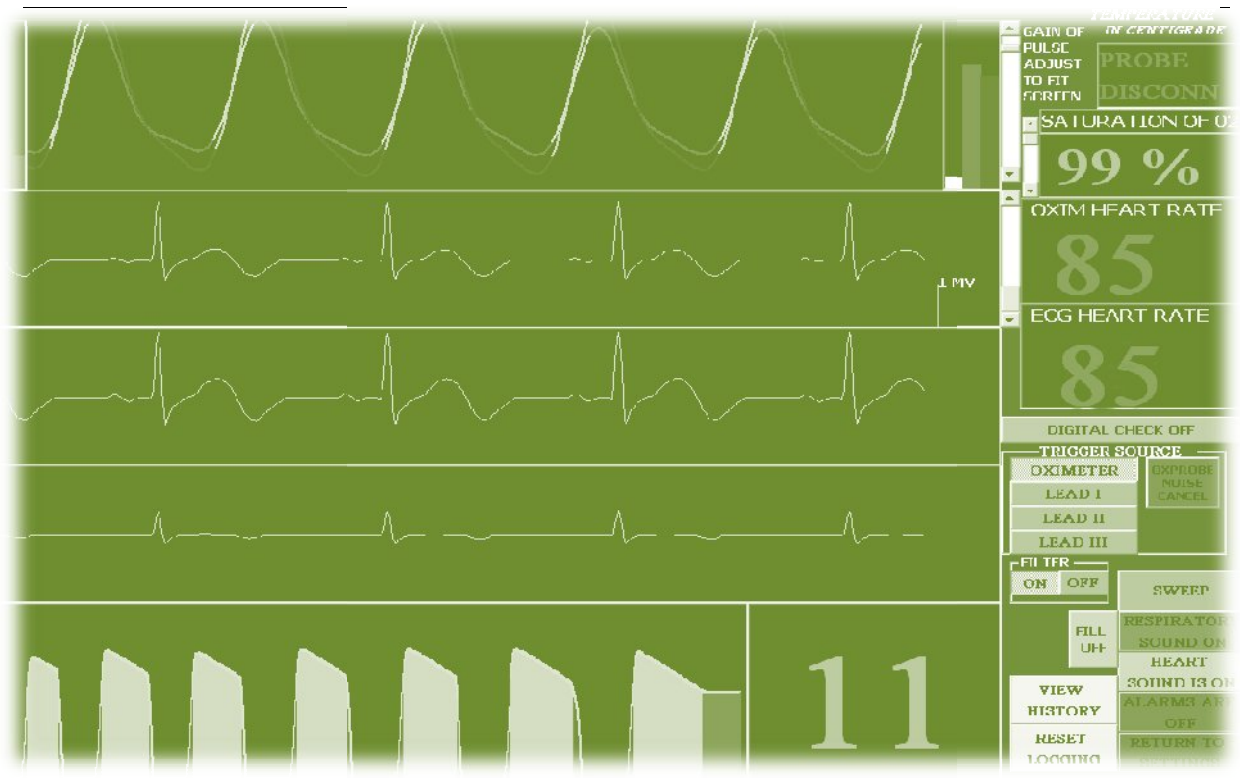
- measures SaO₂ by red and infrared light absorption by Hb; oxygenated and deoxygenated Hb have different absorption characteristics
- non-invasive
- can show pulse waveforms on suitably equipped monitors
- if ventilation is accidentally terminated, the SaO₂ may remain normal for several minutes in a well oxygenated patient due to the high partial pressure of O₂ remaining in the lungs
- inaccurate with
 - i. hypotension,
 - ii. vasoconstriction
 - iii. dyes, (e.g. nailpolish),
 - iv. other Hb (e.g. CarboxyHb)
 - v. compression of the limb
 - vi. movement

Capnometer



- Measures exhaled CO₂ indicates adequacy of ventilation of lungs and cardiac output, confirms ETT placement
2. ECG • changes in rate, rhythm, ST elevation/depression
 3. BP cuff (manual/automatic)
 4. stethoscope (precordial, esophageal)
 5. thermometer (surface or core)
 6. peripheral nerve stimulators (when using neuromuscular blockade or blocking drugs)
deliver electrical stimulus to elicit muscle responses indicates degree of muscle relaxation

7. machine function "monitors" - i.e. volume and pressure alarms and inspired O₂ alarms
8. mass spectrometer/gas analyzer • identifies and measures inhaled/exhaled gases



Patient Monitoring

Examples of Multiparameter Patient Monitors