# ANESTHESIA AND RESUSCITATION

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## THE ABC's
- **AIRWAY**
  - Tracheal Intubation
  - Extubation

## BREATHING (VENTILATION)
- Manual Ventilation
- Mechanical Ventilation
- Supplemental Oxygen

## CIRCULATION
- Fluid Balance
- IV Fluid Therapy
- IV Fluid Solutions
- Blood Products
- Transfusion Reactions
- Shock

## ANESTHESIA
- Preoperative Assessment
- ASA Classification
- Postoperative Management
- Monitoring

## GENERAL ANESTHETIC AGENTS
- Definition of General Anesthesia
- IV Anesthetics (Excluding Opioids)
- Narcotics/Opioids
- Volatile Inhalational Agents
- Muscle Relaxants + Reversing Drugs

## REGIONAL ANESTHESIA
- Definition of Regional Anesthesia
- Preparation of Regional Anesthesia
- Nerve Fibres
- Epidural and Spinal Anesthesia
- IV Regional Anesthesia
- Peripheral Nerve Blocks
- Obstetrical Anesthesia

## LOCAL INFILTRATION, HEMATOMA BLOCKS

## LOCAL ANESTHETICS

## SPECIAL CONSIDERATIONS
- Atypical Plasma Cholinesterase
- Endocrine Disorders
- Malignant Hyperthermia (MH)
- Myocardial Infarction (MI)
- Respiratory Diseases

## REFERENCES
THE ABC’S - AIRWAY

- # = fracture
- most acute airway problems in an unconscious patient can be managed using simple techniques such as:
  - 100% O₂ with the patient in the lateral position (contraindicated in known suspected C-spine #)
  - head tilt via extension at the atlanto-occipital joint (contraindicated in known/suspected C-spine #)
  - jaw thrust via subluxation of temporomandibular joint (TMJ)
  - suctioning (secretions, vomitus, foreign body)
  - positioning to prevent aspiration
  - inserting oro- or naso-pharyngeal airway

- nasopharyngeal airway indicated when an oropharyngeal airway is technically difficult (e.g. trismus, mouth trauma)
  - large adult 8-9 mm, medium adult 7-8 mm, small adult 6-7 mm internal diameter

- complications of nasopharyngeal airway include
  - tube too long - enters the esophagus
  - laryngospasm
  - vomiting
  - injury to nasal mucosa causing bleeding and aspiration of clots into the trachea

- oropharyngeal airway holds tongue away from posterior wall of the pharynx
  - large adult 100 mm, medium adult 90 mm, small adult 80 mm
  - facilitates suctioning of pharynx
  - prevents patient from biting and occluding endotracheal tube (ETT)

- complications of oropharyngeal airway include
  - tube too long - may press epiglottis vs. larynx and obstruct
  - not inserted properly - can push tongue posteriorly

- more advanced techniques include
  - tracheal intubation (orally or nasally)
  - cricothyroidotomy
  - tracheostomy

TRACHEAL INTUBATION

- definition: the insertion of a tube into the trachea either orally or nasally

Indications for Intubation - the 5 P’s

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

- Protect the lungs from aspiration
  - absent protective reflexes, e.g. coma, cardiac arrest

- Positive 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

- Magill 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)
THE ABC’s - AIRWAY...CONT.

- 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, Laerdal bag, light on laryngoscope
- 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")
- "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

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)
  - gastroesophageal reflux disease (GERD)
- procedure as follows
  - patient breathes 100% O₂ for 3-5 minutes prior to induction of anesthesia (e.g. thiopental)
  - 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)
  - auscultate 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
- tumours - 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

EXTUBATION

- performed by trained, experienced personnel because reintubation may be required at any point
- laryngospasm more likely in semiconscious patient, therefore must ensure LOC is adequate
- 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, sidelying

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
THE ABC's - BREATHING (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 ET 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)
- 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

SUPPLEMENTAL OXYGEN

Low Flow Systems
- acceptable if tidal volume 300-700 mL, RR < 25, steady ventilation pattern
- nasal canula - low flow system, inspired O2 depends on flow rate and tidal volume.
  - Larger tidal volume, increased RR = lower FIO2
  - for every increase from 1 L/min O2, inspired O2 concentration increases about 4%
  - e.g. with normal tidal volume, at 1-6 L/min FIO2 = 24-44%
- facial mask - low flow system, well tolerated, will have some rebreathing at normal tidal volumes.
  - Minimize by increasing flow rate. Inspired O2 is diluted by room air
  - provides O2 concentrations of 40-60%
- facial mask with oxygen reservoir
  - provides O2 concentrations of > 60%
  - 6 L/min = 60%, each increase of 1L/min O2 increases the inspired concentration by 10%

High Flow Systems
- Venturi mask - high flow system, with mixed O2 concentrations
  - provides many O2 concentrations, e.g. 24%, 28%, 35%, and 40%
  - advantages include a consistent and predictable FIO2 and the ability to control the humidity of the gas
**THE ABC’s - CIRCULATION**

**FLUID BALANCE** (see Figure 1)
- 70 kg adult - 60% total body weight is H2O (42L) = TBW (total body water)
- for 70 kg adult
- ICF = intracellular fluid, ECF = extracellular fluid

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- ICF = intracellular fluid, ECF = extracellular fluid

- ECF volume expanded = pulmonary edema, dependent edema, S3, increased jugular venous pressure (JVP)
- ECF volume deficit = decreased JVP, hypotension, tachycardia, dry mucous membranes, decreased skin turgor, lethargy, weight loss, sunken eyes, decreased urine output, depressed fontanelle in infants
- hematocrit will decrease/increase with ECF expansion/deficit respectively
- fluid ins and outs determine total body fluid balance; altered by renal function, syndrome of inappropriate ADH secretion (SIADH), diabetes insipidus (DI), osmoles, drugs (diuretics) etc.
- adequate hydration essential prior to anesthesia

**IV FLUID THERAPY**
Total Requirement = (maintenance + deficit + ongoing losses) minus (PO intake + TPN + meds solution)

**Deficit**
- dehydration
  - mild < 5% TBW fluid loss
  - moderate 5-10% TBW fluid loss
  - severe > 10% TBW fluid loss
- total Na+ content controls ECF volume, [Na+] determines ICF volume

- hypovolemia due to volume contraction
  1. extrarenal Na+ loss
    - gastrointestinal: vomiting, nasogastric (NG) suction, drainage, fistulae, diarrhea
    - skin/resp: insensible losses (fever), sweating, burns
    - vascular: hemorrhage
  2. renal Na+ and H2O loss
    - diuretics
    - osmotic diuresis
    - hypoaldosteronism
    - salt-wasting nephropathies
  3. renal H2O loss
    - diabetes insipidus (central or nephrogenic)

- hypovolemia with normal or expanded ECF volume
  1. decreased cardiac output (CO)
  2. redistribution
    - hypoalbuminemia: cirrhosis, nephrotic syndrome
    - capillary leaking: acute pancreatitis, rhabdomyolysis, ischemic bowel

- replace water and electrolytes as determined by patients needs
- with chronic hyponatremia correction must be over > 48 hours to avoid CNS central pontine myelinolysis

<table>
<thead>
<tr>
<th>Percentage of Body Water Loss</th>
<th>Severity</th>
<th>Signs and Symptoms</th>
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<tbody>
<tr>
<td>5%</td>
<td>Mild</td>
<td>Decreased skin turgor, sunken eyes, dry mucous membranes, dry tongue, reduced sweating</td>
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<td>10%</td>
<td>Moderate</td>
<td>Oliguria, orthostatic hypotension, tachycardia, low volume pulse, cool peripheries, reduced filling of peripheral veins and central venous pressure (CVP), hemoconcentration, apathy</td>
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<td>15%</td>
<td>Severe</td>
<td>Profound oliguria and compromised CNS function with or without altered sensorium</td>
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THE ABC’s - CIRCULATION . . . CONT.

Maintenance
- average healthy adult requires approximately 2,500 mL water/day
  - 200 mL/day gastrointestinal (GI) losses
  - 800 mL/day insensible losses (respiration, perspiration)
- 1,500 mL/day urine (be aware of renal failure)
- increased requirements with fever, sweating, GI losses (vomiting, diarrhea, nasogastric (NG) suction), adrenal insufficiency, hyperventilation, and polyuric renal disease
- decreased requirements with anuria/oliguria, SIADH, highly humidified atmospheres, and congestive heart failure (CHF)
- 4/2/1 rule to calculate maintenance requirements
  - 4 mL/kg/hour first 10 kg
  - 2 mL/kg/hour second 10 kg
  - 1 mL/kg/hour for remaining weight > 20 kg
- maintenance lytes: Na+: 3 mEq/kg/day, K+: 1 mEq/kg/day
  - e.g. 50 kg patient maintenance requirements
    - fluid = 40 + 20 + 30 = 90 mL/hour = 2160 mL/day
    - Na+ = 150 mEq/day (therefore give 66 mEq/L)
    - K+ = 50 mEq/day (therefore give 22 mEq/L)
- above patient’s requirements roughly met via 2/3 D5W 1/3 NS (2/3 + 1/3) (with 20 mEq/KCl) @ 100 mL/hour

Ongoing Losses
1. tubes
   - Foley catheter, NG, surgical drains
2. third spacing (other than ECF, ICF)
   - pleural, GI, retroperitoneal, peritoneal
   - evaporation via exposed viscera, burns
3. blood loss
   - losses replaced approximately 1:1 on an ongoing basis

Input (PO Intake/TPN/MEDS Solutions)
- PO intake (includes metabolic water from food)
- total parenteral nutrition (TPN) contains substantial water
- antibiotic vehicle, blood products, packed red blood cells (PRBCs) with saline, etc.
- if concurrent, decrease IV to prevent fluid overload

IV FLUID SOLUTIONS
- replacement fluids include crystalloid and colloid solutions
- remember that crystalloid/colloid improves perfusion: BUT NOT O2 CARRYING CAPACITY OF BLOOD

Crystalloid Infusion
- salt containing solutions that distributes within ECF
- maintain euvoolemia in patient with blood loss - 3 mL crystalloid infusion
intusion per 1 mL of blood lost for volume replacement
- if large volumes to be given use balanced fluid such as Plasmalyte or Ringer’s lactate, as too much normal saline (NS) may lead to hyperchloremic metabolic acidosis

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<thead>
<tr>
<th>Table 2. IV Fluid Solutions</th>
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<tr>
<td>ECF</td>
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<td>meq/L</td>
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* converted from Lactate

Colloid Infusion (see Blood Products section)
- collected from donor blood (fresh frozen plasma (FFP), albumin, PRBCs) or synthetic (pentastarch products)
- distributes within intravascular volume
- 1:1 ratio (infusion:blood loss) only in terms of replacing volume
THE ABC's - CIRCULATION . . . CONT.

Initial Distribution of IV Fluids (1 Litre)
- H₂O follows ions/molecules to their respective compartments

<table>
<thead>
<tr>
<th>Table 3. Initial Distribution of IV Fluids (1 Litre)</th>
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<tr>
<td><strong>Solution</strong></td>
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<tr>
<td>NS</td>
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<td>1/2 NS</td>
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<td>1/3 NS</td>
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<tr>
<td>Ringers</td>
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<tr>
<td>D5W*</td>
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<tr>
<td>2/3 1/3</td>
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<tr>
<td>Colloid</td>
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* assuming glucose metabolized

BLOOD PRODUCTS

Red Blood Cells: RBC's (U = Unit)
- 1 U PRBCs = +/- 300 mL
- 1 U PRBCs increases hemoglobin (Hb) by approx 10 g/L in a 70 kg patient
- PRBCs may be diluted with colloid/crystalloid to decrease viscosity
- decision to transfuse based on initial blood volume, premorbid Hb level, present volume status, expected further blood loss, patient health status
- MASSIVE transfusion > 1 x blood volume/24 hours

Autologous RBCs
- replacement of blood volume with one's own RBCs
- marked decrease in complications (infectious, febrile, etc.)
- alternative to homologous transfusion in elective procedures, but only if adequate Hb, and no infection
- pre-op phlebotomy with hemodilution prior to elective surgery (up to 4 U collected > 2 days before surgery)
- intraoperative salvage and filtration (cell saver)

Non-RBC Products
- FFP (fresh frozen plasma)
  - 10-15 mL/kg
  - to prevent/treat bleeding due to coagulation factor depletion
  - for liver failure, factor deficiencies, massive transfusions
  - contains all plasma clotting factors and fibrinogen close to normal plasma levels
- factors
  - cryoprecipitate (1 U/7-10 kg) or preps (von Willebrand Factor (VWF), factor VIII, etc.)
- platelets
  - 1 concentrate/10 kg
  - thrombocytopenia, massive transfusions, impaired platelet function
- albumin
  - selective intravascular volume expander
- erythropoietin
  - can be used preoperatively to stimulate erythropoiesis
- pentaspan
  - colloid, don't give > 2 L/70 kg/24 hours

TRANSFUSION REACTIONS

Immune - Nonhemolytic
1. FEBRILE - most common mild reaction, 0.5%-4% of transfusions
   - due to alloantibodies to WBC, platelet, or other donor plasma antigens
   - fever likely caused by pyrogens liberated from lysed cells
   - more common if previous transfusion
   - mild fever < 38° with or without rigors, fever may be > 38° with restlessness and shivering
   - nausea, facial flushing, headache, myalgias, hypotension, chest and back pain (less common)
   - near completion of transfusion or within 2 hours
   - up to 40% with mild reactions will not experience another reaction with future transfusions
   - with severe/recurrent reactions, future transfusions may cause leukocyte depletion
- management - rule out fever due to hemolytic reaction or bacterial contamination
  - mild < 38° - decrease infusion rate and antipyretics
  - severe - stop transfusion, antipyretics, antihistamines, symptomatic treatment

A8 – Anesthesia
MCCQE 2002 Review Notes
2. ALLERGIC - mild allergic reaction occurs in about 3% of transfusions
   • due to IgE alloantibodies vs. substances in donor plasma
   • mast cells activated with histamine release
   • usually occurs in pre-exposed e.g. multiple transfusions, multiparous
   • often have history of similar reactions
   • abrupt onset pruritic erythema/urticaria on arms and trunk, occasionally with fever
   • less common - involvement of face, larynx, and bronchioles

   **management**
   - mild - slow transfusion rate, IV antihistamines
   - moderate to severe - stop transfusion, IV antihistamines, subcutaneous epinephrine, hydrocortisone, IV fluids, bronchodilators
   - prophylactic - antihistamines 15-60 minutes prior to transfusion, washed or deglycerolized frozen RBC

3. ANAPHYLACTIC - rare, potentially lethal
   • in IgA deficient patients with anti-IgA antibodies
   • immune complexes activate mast cells, basophils, eosinophils, and complement system
   = severe symptoms after transfusion of RBC, plasma, platelets, or other components with IgA
   • apprehension, urticarial eruptions, dyspnea, hypotension, laryngeal and airway edema, wheezing, chest pain, shock, sudden death

   **management**
   • circulatory support with fluids, catecholamines, bronchodilators, respiratory assistance as indicated
   • evaluate for IgA deficiency and anti-IgA antibodies
   • future transfusions must be free of IgA: washed/deglycerolized RBCs free of IgA, blood from IgA deficient donor

4. TRANSFUSION - RELATED ACUTE LUNG INJURY (TRALI)
   • form of noncardiogenic pulmonary edema
   • occurs 2-4 hours post transfusion
   • immunologic cause; not due to fluid overload or cardiac failure - is a reaction to transfusion
   • respiratory distress - mild dyspnea to severe hypoxia
   • chest x-ray - consistent with acute pulmonary edema, but pulmonary artery and wedge pressures are not elevated

   **management**
   • usually resolves within 48 hours with O2, mechanical ventilation, supportive treatment

5. IMMUNOSUPPRESSION
   • some studies show associations between perioperative transfusion and postoperative infection, earlier cancer recurrence, and poorer outcome

**Immune - Hemolytic**

- most serious and life threatening transfusion reaction
- caused by donor incompatibility with recipients’ blood

1. ACUTE - Intravascular hemolysis
   • most severe
   • often due to clerical error
   • antibody coated RBC is destroyed by activation of complement system
   • ABO incompatibility common cause, other RBC Ag-Ab systems can be involved
   • fever, chills, chest or back pain, hypotension, tachycardia, nausea, flushing, dyspnea, hemoglobinuria, diffuse bleeding due to disseminated intravascular coagulation (DIC), acute renal failure (ARF)
   • in anesthetized patients, signs include hypotension, tachycardia, wheezing, hypoxemia and hemoglobinuria

   **management**
   • stop transfusion
   • notify blood bank, confirm or rule out diagnosis - clerical check, direct Coombs', repeat grouping, Rh screen and crossmatch, serum haptoglobin
   • manage hypotension with fluids, inotropes, other blood products
   • maintain urine output with crystalloids, furosemide, dopamine, alkalinize urine
   • component treatment if DIC

2. DELAYED - Extravascular hemolysis
   • anemia, mild jaundice, fever 1-21 days post transfusion
   • incompatibility of antigen and antibody that do not bind complement
   • Ab coated RBC destroyed by macrophagic phagocytosis by in reticuloendothelial system (RES)
   • failure to recognize these antibodies at crossmatch often involved
   • low titre antibodies may be undetectable, but amnestic response in recipient = buildup of antibodies to incompatible RBC several days post transfusion

   **predisposing factors to hemolytic transfusion reactions**
   • F to M = 3:1
   • increasing age
   • blood products administered on emergent basis
THE ABC's - CIRCULATION . . . CONT.

Nonimmune
- infectious risks - HIV, hepatitis, Epstein-Barr virus (EBV), cytomegalovirus (CMV), brucellosis, malaria, salmonellosis, measles, syphilis
- hypervolemia
- electrolyte changes
  - increased K+ in stored blood
- coagulopathy
- hypothermia
- citrate toxicity
- hypocalcemia

SHOCK (see Emergency Medicine Chapter for algorithm)
- remember: hypotension is NOT synonymous with shock
- shock = inadequate organ perfusion
- general approach to treatment of shock
  - always ABCs first
  - next IDENTIFY THE CAUSE
- general management
  - O2, fluids
  - inotropes
  - monitor urine output, vitals, plus central venous pressure (CVP) +/- pulmonary capillary wedge pressure (PCWP)
- beware of complications: i.e. hypovolemic shock causing cardiac ischemia leading to cardiogenic shock, etc.

TYPES OF SHOCK
S - Septic/Spinal
H - Hemorrhage/Hypovolemia
O - Obstructive
C - Cardiogenic
K - Anaphylactic

1. SEPTIC SHOCK
   - bacterial (often Gram negative), viral, fungal
   - endotoxins/mediators cause pooling of blood in veins and capillaries
   - associated with contamination of open wounds, intestinal injury or penetrating trauma, can occur with relatively unremarkable history
   - clinical features: warm skin (fever), decreased JVP, wide pulse pressure, increased cardiac output (CO), decreased systemic vascular resistance, increased heart rate (HR)
   - initial treatment includes 1) Antibiotics, 2) Volume expansion

2. SPINAL/NEUROGENIC SHOCK
   - decreased sympathetic tone
   - hypotension without tachycardia or peripheral vasoconstriction (warm skin)

3. HYPOVOLEMIC/HEMORRHAGIC SHOCK
   - blood loss or dehydration
   - mild (< 20% blood volume)
     - decreased peripheral perfusion only of organs able to withstand prolonged ischemia (skin, fat, muscle, and bone)
     - patient feels cold, postural hypotension and tachycardia, cool, pale, moist skin, low JVP, decreased CO
     - initial treatment includes 1) Volume expansion
   - moderate (20-40%)
     - decreased perfusion of organs able to tolerate only brief periods of ischemia
     - thirst, supine hypotension and tachycardia, oliguria or anuria
   - severe (> 40%)
     - decreased perfusion of heart and brain
     - agitation, confusion, obtundation, supine hypotension and tachycardia, rapid deep respirations, anuria

4. OBSTRUCTIVE
   - cardiac compressive shock
   - increased JVP, distended neck veins, increased systemic vascular resistance (SVR)
   - insufficient cardiac output (CO)
   - occurs with tension pneumothorax, cardiac tamponade, pulmonary embolism (PE), pulmonary HTN, aortic and mitral stenosis (AS/MS)

5. CARDIOGENIC
   - myocardial dysfunction may be due to: dysrhythmias, MI, cardiomyopathy, acute valvular dysfunction
   - increased JVP, distended neck veins, increased SVR, decreased CO
6. ANAPHYLACTIC "K"
   - type I hypersensitivity
   - an acute/subacute generalized allergic reaction due to an inappropriate or excessive immune response
   - anaphylactoid reactions (similar to anaphylactic reactions) are not due to immunologic responses but
     rather due to mast cell mediator release or activation by pharmacological agents
   - treatment for moderate reaction (generalized urticaria, angioedema, wheezing, tachycardia, no
     hypotension)
     - epinephrine (1:1,000) 0.3-0.5 mg subcutaneous (SC) = 0.3-0.5 mL
     - antihistamines (Benadryl) 25 mg intramuscularly (IM)
     - ventolin 1 cc via nebulizer
   - treatment for severe reaction/evolution (severe wheezing, laryngeal/pulmonary edema, shock)
     - epinephrine IV, (via ET if no IV access)
     - TITRATE epinephrine dose to severity; begin with 1 ug /kg (e.g. 50 µ = 0.5 mL of 1:10,000
       solution), giving additional boluses q 1-2 minutes and increasing doses to achieve
       acceptable BP; may need to continue IV infusion of epinephrine for several hours
     - antihistamines over 1 minute (i.e. H1-blockers Benadryl 50 mg IV and H2-blockers famotidine)
     - steroids - initial dose 100 mg solumedrol IV, followed by the equivalent of 100 mg solucortef per hour
       (i.e. 25 mg soludromed)
ANESTHESIA . . . CONT.

Figure 2. Mallampati Classification

Drawing by Betty Lee

- bony landmarks and suitability of areas for regional anesthesia if relevant
- focus on CNS, CVS and respiratory (includes airway) systems
- general e.g. nutritional, hydration, and mental status
- pre-existing motor and sensory deficits
- sites for IV, central venous pressure (CVP) and pulmonary artery (PA) catheters, regional anesthesia

Investigations
- change in Public Hospitals Act: Hb and urinalysis no longer required as routine in all patients pre-operatively
- hospital or departmental policies and patient characteristics will dictate the necessity and/or indications for tests such as chest x-ray, Hb, etc.
- ECG often recommended for those > 40 years old
- preoperative pulmonary function tests for patients with COPD, heavy smokers with history of persistent cough, chest wall and spinal deformities, morbidly obese, elderly (> 70), and patients for thoracic surgeries
- other investigations as clinically indicated

AMERICAN SOCIETY OF ANESTHESIOLOGY (ASA) CLASSIFICATION
- common classification of physical status at time of surgery
- a gross predictor of overall outcome, NOT used as stratification for anesthetic risk (mortality rates)
  - ASA 1: a healthy, fit patient (0.06-0.08%)
  - ASA 2: a patient with mild systemic disease e.g. controlled Type 2 diabetes, controlled essential HTN, obesity (0.27-0.4%), smoker
  - ASA 3: a patient with severe systemic disease that limits activity, e.g. angina, prior MI, COPD (1.8-4.3%), DM, obesity
  - ASA 4: a patient with incapacitating disease that is a constant threat to life, e.g. CHF, renal failure, acute respiratory failure (7.8-23%)
  - ASA 5: a moribund patient not expected to survive 24 hours with/without surgery, e.g. ruptured abdominal aortic aneurysm (AAA), head trauma with increased ICP (9.4-51%)
- for emergency operations, add the letter E after classification
- from the history, physical exam, and labs/investigations the anesthetist can determine whether or not the patient is in OPTIMAL condition for the proposed surgical procedure
- goal is to optimize the non-surgical disease states prior to surgery
- in emergency cases it is not always possible to optimize coexistent or chronic disease states; goal is then to accomplish what is possible in the time available

POSTOPERATIVE MANAGEMENT
- usually begins in O.R. with discontinuation of anesthetic drugs and extubation
  (exception - if going to intensive care unit (ICU))
- patient can be transported to post-anesthesia care unit (PACU) when ABC's stable
- patient can be released from the unit when the PACU discharge criteria for ventilation, circulation, consciousness, motor function, and colour have been met
- potential complications
  - CNS: agitation, delirium, somnolence
  - Respiratory: aspiration, upper airway obstruction
  - hypoxemia, alveolar hypoventilation
  - upper airway trauma (intubation/extubation)
  - CVS: hypothermia (rewarm patient)
  - shivering (due to hypothermia or postanesthetic effect)
  - hypotension, hypertension, dysrhythmias
  - GI: nausea and vomiting
ANESTHESIA . . . CONT.

- pain control
  - goal is to provide pain relief safely with minimal disturbance of homeostasis (preoperative visit has been shown to be beneficial)
  - unrelieved pain can be the cause of many postoperative complications
  - factors influencing the degree of pain include age, personality, premedication, surgical site, and anesthetic technique
  - routes - IV, IM, oral, epidural, rectal
  - preemptive analgesia (controversial)
    - prevent/reduce noxious stimuli which potentiate peripheral and central pain mechanisms
    - in postoperative period the dose of analgesic is decreased and the side effects are less frequent
    - use - NSAID’s, opioids, local anesthetics, combined agents
  - PCA (patient controlled analgesia)
    - self-administration of small doses of opiates via pump
    - bolus dose is preset
    - lockout period is set to limit frequency of self-administration
    - requirements - oriented patient, IV, SC, or epidural access

MONITORING
- monitoring provides information that improves the safety of anesthesia and provides a means to assess physiological function
- appropriate monitors with alarms are intended to enhance but not replace the vigilance of the anesthetist
- physical examination, observation, assessment, and diagnosis remain the most important tools available to the anesthetist
- routine monitors for all cases: BP cuff, ECG, O₂ sat monitor, stethoscope, temperature probe, exposed part of patient visible, capnometer if intubated
- organ systems monitored and other devices used to monitor will vary depending on the nature, length, location, and systems involved in the surgery, and patient's pre-existing condition/diseases

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 hypotension, vasoconstriction, dyes, (e.g. nailpolish), other Hb, (e.g. CarboxyHb), compression of the limb, and movement

<table>
<thead>
<tr>
<th>PO₂ (mmHg)</th>
<th>Hb Sat (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>100</td>
<td>98</td>
</tr>
<tr>
<td>80</td>
<td>96</td>
</tr>
<tr>
<td>60</td>
<td>90*</td>
</tr>
<tr>
<td>40</td>
<td>75</td>
</tr>
<tr>
<td>27</td>
<td>50</td>
</tr>
</tbody>
</table>

* Recall Hb-O₂ saturation curve (see Respiratory Chapter)

- capnometer
  - measures exhaled CO₂, indicates adequacy of ventilation of lungs and cardiac output, confirms ETT placement
- ECG
  - changes in rate, rhythm, ST elevation/depression
- BP cuff (manual/automatic)
- stethoscope (precordial, esophageal)
- thermometer (surface or core)
- peripheral nerve stimulators (when using neuromuscular blockade or blocking drugs)
  - deliver electrical stimulus to elicit muscle responses
  - indicates degree of muscle relaxation
- machine function "monitors" - i.e. volume and pressure alarms and inspired O₂ alarms
- mass spectrometer/gas analyzer
  - identifies and measures inhaled/exhaled gases
LESS FREQUENTLY USED MONITORS
- urinary catheter and urometer
- central venous line
  - rapid fluid infusion, infusion of vasoactive drugs, measuring CVP
- arterial line
  - continuous BP monitoring, easy access allowing for frequent ABGs
- Swan-Ganz catheter- CVP, PCWP; pulmonary artery pressures, cardiac output, mixed venous blood gases, core temperature
- ICP monitoring
- EEG, brain and spinal cord evoked potentials
- transcutaneous gas measurements
- transesophageal echocardiography (TEE)

GENERAL ANESTHETIC AGENTS

DEFINITION OF GENERAL ANESTHESIA (GA)
- delivery of anesthetic drugs (inhaled or infused) to produce a level of CNS depression with the following goals (the 6 A's of Anesthesia)
  1. **ANESTHESIA** - hypnosis, loss of consciousness
  2. **ANALGESIA** - pain control
  3. **AMNESIA** - loss of recall
  4. **AREFLEXIA** - muscle relaxation (this is not always required)
  5. **AUTONOMIC AREFLEXIA** - decrease sympathetic nervous system (SNS) function
  6. **ANXIOLYSIS** - pre-op and intra-op
- methods of GA are variable and complex

BALANCED ANESTHESIA
- a dynamic process incorporating a multiplicity of agents as no single anesthetic agent has been developed in which all these properties (the 6 A's) are combined in optimal proportions

PREMEDICATION
- medication may be given prior to anesthesia, i.e. benzodiazepines, opioids, glycopyrrolate
- goals
  1. provide sedation, amnesia and relief from anxiety and pain
  2. to prevent parasympathomimetic effects of the anesthetics, i.e. to prevent salivation, bronchial secretions and dysrhythmias caused by anesthetic agents and airway instrumentation

IV ANESTHETICS (EXCLUDING OPIOIDS)
- IV administration provides rapid distribution and onset of effects
- given as a bolus or as a continuous infusion, titrate to effect
- common agents used for induction are described below

**Thiopental (Sodium Thiopental, Sodium Thiopentone, STP)**
- ultrashort acting thiobarbiturate
- most commonly used as an induction agent
- prepared as a pale yellow 2.5% solution with pH 10.5 (alkaline)
- after IV bolus, rapidly distributes to vessel rich organs (brain, liver, heart, kidney), thus achieves unconsciousness in brain circulation time (approximately 30 seconds)
- rapid redistribution from vessel rich tissues to muscle and fat causes short lived effect (approximately 5 minutes)
- metabolism and elimination occur at a slower rate (T1/2 =5-12 hrs), resulting in residual effects (usually sedation) during post-anesthesia recovery which may last hours
- effects of thiopental include
  - unconsciousness
  - decreased cerebral metabolism and O2 requirements
  - reduction of cerebral blood flow
  - decrease in CO, BP, reflex tachycardia
  - respiratory depression (apnea often occurs with bolus dose)
- thiopental has no analgesic properties and at low doses actually increases the subjective feeling of pain (anti-analgesia)
- no muscle relaxant properties
- some contraindications
  - lack of equipment for intubation and resuscitation
  - potential difficult intubation
  - hypersensitivity
  - untreated hypovolemia, hypotension, shock-like states
  - cardiac failure
  - porphyria
**GENERAL ANESTHETIC AGENTS ... CONT.**

**Propofol (Diprivan)**
- unique agent in its own class (an alkyl phenol)
- used for induction and/or maintenance of anesthesia
- thick white soybean-based solution
- pharmacological effects similar to that of thiopental; thus similar contraindications but is safe for porphyria patients
- metabolism and elimination much more rapid due to increased rate of liver metabolism compared to thiopental
- less residual sedative effect, patient recovers sooner ($T_{1/2} = 0.9$ hr), thus popular for outpatient surgery since reduces post-anesthesia recovery time; decreased incidence of nausea and vomiting
- more suited for continuous infusion than STP due to rapid elimination
- more expensive

**Benzodiazepines (e.g. diazepam, midazolam, lorazepam)**
- also known as the minor tranquilizers
- used as a premedication prior to induction or as an induction agent in combination with other drugs
- oral and injectable formulations available
- act on specific brain (GABA) receptors to produce selective anti-anxiety and sedative effects; in correct doses, causes only slight depression of CVS and respiratory systems
- onset less than 5 minutes if given IV
- duration of action long but variable/somewhat unpredictable
- benzodiazepine antagonist flumazenil (Anexate)
  - competitive inhibition
  - does not affect benzodiazepine metabolism, therefore once effects of reversal wear off, sedation may return

**Neuroleptics**
- also known as the major tranquilizers, rarely used in anesthesia
- blockade of dopamine receptors at various locations in CNS
- droperidol used in low dose as antiemetic

**NARCOTICS/OPIOIDS**
- opium: natural product derived from poppy plant extract
- opiates: derived from opium (e.g. morphine, codeine)
- opioids: any drug that binds to morphine receptors (also known as opioid receptors); includes natural products, semisynthetic products, synthetic drugs, endogenous substances

**Opioid Receptors**
- found in many locations in the body, particularly in the brain, brainstem, and spinal cord
- several classes of receptors, each responsible for different effects
  - mu receptors: analgesia, respiratory depression, dependence
  - kappa receptors: spinal analgesia, sedation
  - sigma receptors: hallucinations, dysphoria
  - delta receptors: mood changes

**Indications**
- opioids used for pre-, intra-, postoperative analgesia
- also used as an induction agent, alone or as adjuvant
- reduces minimum alveolar concentration (MAC) required for volatile anesthetics
- can be administered IV, IM, PO

**General Effects of Morphine (Prototype Opioid)**
- CNS (depression) - analgesia, mood changes, sedation, respiratory depression, decreased cough reflex
- CNS (excitation) - miosis, nausea and vomiting, hyperreflexia
- CVS - vasodilatation, orthostatic hypotension
- Respiratory - central depression, bronchial constriction
- GI - constipation, biliary colic
- GU - urinary retention
- Other - histamine release, smooth muscle contraction (e.g. biliary and bladder sphincters)
### Table 4. Other Opioids Used in Anesthesia

<table>
<thead>
<tr>
<th>Agent</th>
<th>Potency*</th>
<th>Onset</th>
<th>Duration</th>
<th>Special Considerations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Morphine</td>
<td>1</td>
<td>Moderate</td>
<td>Moderate</td>
<td>Histamine release</td>
</tr>
<tr>
<td>Codeine</td>
<td>1/6-1/10</td>
<td>Moderate</td>
<td>Moderate</td>
<td>Primarily postoperative use, not for IV use</td>
</tr>
<tr>
<td>Meperidine</td>
<td>1/10</td>
<td>Moderate</td>
<td>Moderate</td>
<td>Anticholinergic, hallucination, less pupillary constriction than morphine</td>
</tr>
<tr>
<td>Fentanyl</td>
<td>100</td>
<td>Rapid</td>
<td>Short</td>
<td>Transient muscle rigidity in very high doses, good CVS stability</td>
</tr>
<tr>
<td>Sufentanyl</td>
<td>1000</td>
<td>Rapid</td>
<td>Short</td>
<td></td>
</tr>
<tr>
<td>Alfentanly</td>
<td>20</td>
<td>Rapid</td>
<td>Very short</td>
<td></td>
</tr>
</tbody>
</table>

*potency compared to morphine

### Opioid Antagonists (e.g. naloxone, naltrexone)
- opioid toxicity manifests primarily at CNS - manage ABC's
- opioid antagonists competitively inhibit opioid receptors, predominantly mu receptors
- must observe patient after administration
  - naloxone relatively short acting (T1/2 = 1 hour); effects of narcotic may return when naloxone wears off
  - naltrexone (T1/2 = 10 hours) - less likely to see return of narcotic effects unless narcotic levels very high
- relative overdose of naloxone may cause agitation, sweating, tachycardia, hypertension, re-emergence of pain, pulmonary edema, seizures

### Volatile Inhalational Agents
- exact mechanism of action unknown: currently thought to be due to anesthetic molecules embedding into plasma membranes of cells, causing disruption of ion channels
- agents are delivered via respiratory system; partial pressure gradients cause diffusion of inhaled agents from alveoli to blood to brain (target organ)
- for a given anesthetic gas at steady state
  - alveolar partial pressure = arterial partial pressure = brain partial pressure
- monitoring the end-tidal alveolar concentration of inhaled anesthetic agent provides a good estimate of brain anesthetic tension and anesthetic depth
- Minimum Alveolar Concentration (MAC)
  - \( = \% \) concentration of anesthetic agent in alveolar gas at steady state that will prevent movement in 50% of subjects in response to a standard surgical stimulus eg. skin incision
  - gas concentrations often expressed as multiples of MAC,
    - e.g. if an agent has a MAC of 1.5% then 0.5 MAC = 0.75% and 2 MAC = 3.0%
  - MACs are additive,
    - e.g. 0.5 MAC of agent A plus 0.5 MAC of agent B will provide a gas mixture with a MAC of 1.0

### Table 5. Volatile Inhalational Agents

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Halothane, Enflurane, Isoflurane, Sevoflurane</th>
<th>Nitrous Oxide (N₂O)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Characteristics</td>
<td>• Liquid, colorless, non-flammable, non-explosive</td>
<td>• Gas, colorless, mild sweet odor at room temperature (stored as liquid under pressure)</td>
</tr>
<tr>
<td></td>
<td>• Vaporizer delivers controlled concentration of anesthetic agents to respiratory system of patient via anesthetic machine</td>
<td>• 104%(^1) (weak anesthetic)</td>
</tr>
<tr>
<td>MAC</td>
<td>• 0.75% 1.68% 1.15%</td>
<td>• 0%</td>
</tr>
<tr>
<td>Metabolism(^2)</td>
<td>• 20% 2% 0.2%</td>
<td>• Second gas effect(^3)</td>
</tr>
<tr>
<td>Effects</td>
<td>• CNS: increase cerebral blood flow, decrease cerebral O₂ consumption</td>
<td>• Analgesia, allows for use of lower dose of more potent anesthetic</td>
</tr>
<tr>
<td></td>
<td>• Resp: respiratory depression (decreased tidal volume (TV), increased rate), decreased response to respiratory CO₂ reflexes, bronchodilation</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• CVS: myocardial depression, vasodilatation</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• MSK: muscle relaxation, potentiation of other muscle relaxants, uterine relaxation</td>
<td></td>
</tr>
<tr>
<td>Uses</td>
<td>• Maintenance of anesthetic state</td>
<td></td>
</tr>
</tbody>
</table>

\(^*\) potency compared to morphine

\(^1\) MAC\(^1\)

\(^2\) MAC\(^2\)

\(^3\) MAC\(^3\)
GENERAL ANESTHETIC AGENTS . . . CONT.

Table 5. Volatile Inhalational Agents (continued)

<table>
<thead>
<tr>
<th>Adverse Effects</th>
<th>Halothane, Enflurane, Isoflurane, Sevoflurane</th>
<th>Nitrous Oxide (N₂O)</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Halothane rarely implicated in postoperative hepatitis</td>
<td></td>
<td>• During emergence, N₂O can diffuse rapidly from the blood to the alveoli, resulting in a dilution of O₂ in the alveoli (&quot;diffusion hypoxia&quot;) it is therefore necessary to provide 100% O₂ for several minutes until N₂O is eliminated</td>
</tr>
<tr>
<td>• Toxicity mostly at CNS (decreased autonomic functions, hypotension, respiratory arrest)</td>
<td></td>
<td>• Bone marrow depression</td>
</tr>
<tr>
<td>• Hypersensitivity, malignant hyperthermia, (see above)</td>
<td></td>
<td>• Chronic neuropathy</td>
</tr>
<tr>
<td>• Airway obstruction, cardiac failure, severe CVS disease, raised ICP</td>
<td></td>
<td>• Tends to diffuse into closed air spaces causing increased pressure and volume (important if there is trapped air e.g. air embolus, pneumothorax, blocked nasal sinuses, etc.)</td>
</tr>
<tr>
<td>Contraindications</td>
<td></td>
<td>• Bowel obstruction</td>
</tr>
<tr>
<td>• Lack of equipment/skill to intubate/resuscitate, illness requiring high inspired O₂</td>
<td></td>
<td>• Any abdominal surgery where an increased volume of bowel gas would interfere</td>
</tr>
<tr>
<td>• Hypersensitivity, malignant hyperthermia, (see above)</td>
<td></td>
<td>• Pneumothorax</td>
</tr>
<tr>
<td>• Airway obstruction, cardiac failure, severe CVS disease, raised ICP</td>
<td></td>
<td>• Pneumocephalus</td>
</tr>
<tr>
<td>• Hypersensitivity, malignant hyperthermia, (see above)</td>
<td></td>
<td>• Large alveolar bullae</td>
</tr>
</tbody>
</table>

1. A MAC of 104% is possible in a pressurized chamber only
2. Oxidative metabolism in liver, remainder is eliminated via the respiratory system
3. SECOND GAS EFFECT: Even though N₂O is poorly soluble in blood, large amounts are taken up from the alveoli during induction because it is administered in such large quantities (2-6 L/minute). As a result, the remaining gases (e.g. isoflurane, enflurane) become more concentrated in the alveoli and therefore their uptake is enhanced

MUSCLE RELAXANTS + REVERSING DRUGS

- mild muscle relaxation can be attained by increasing the depth of general anesthesia with potent inhalational agents but the amount required for useful muscle relaxation is too high to be practical, thus specific muscle relaxant drugs preferable
- muscle relaxants cause variable degrees of neuromuscular blockade (paralysis), depending on dose
- muscle relaxation often desired during surgical procedures for various reasons
  - prevent muscle stretch reflex and suppresses muscle resting tone
  - facilitate intubation
  - facilitate controlled ventilation
  - allow access to the surgical field (intraoperative surgery)
- muscle relaxants classified on the basis of the type of neuromuscular blockade they provide
  - Depolarizing Neuromuscular Relaxants
  - Non-depolarizing Neuromuscular Relaxants
- and according to their duration of action
  - short
  - intermediate
  - long
- both act at post-synaptic nicotinic acetylcholine (ACh) receptor at the neuromuscular junction (NMJ)
- actions potentiated by all potent inhalational agents
- nerve stimulator used intraoperatively to assess block level
**Table 6. Muscle Relaxants**

<table>
<thead>
<tr>
<th></th>
<th>Non-depolarizing (Competitive)</th>
<th>Depolarizing (Non-competitive)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Agents</strong></td>
<td>d-Tubocurarine, pancuronium, doxacurium, atracurium, vecuronium, mivacurium, rocuronium</td>
<td>Succinylcholine</td>
</tr>
<tr>
<td><strong>Action at ACh Receptor</strong></td>
<td>Competitively bind at NMJ without causing depolarization</td>
<td>Binds receptor with depolarization causing fasciculations, sustained receptor availability to ACh depolarization prevents action potential from propagating at junction causing temporary paralysis</td>
</tr>
<tr>
<td><strong>Onset</strong></td>
<td>Slower (2-4 minutes)</td>
<td>Rapid (30-60 seconds)</td>
</tr>
<tr>
<td><strong>Duration</strong></td>
<td>Intermediate to long (20-60 minutes)</td>
<td>Short (5 minutes)</td>
</tr>
<tr>
<td><strong>Use</strong></td>
<td>Muscle relaxation for intubation or intraoperatively, facilitation of mechanical ventilation in some ICU patients, reduction of fasciculations and post-op myalgias secondary to SCh</td>
<td>Muscle relaxation for intubation short procedures, ECT to eliminate muscular component of convulsions</td>
</tr>
<tr>
<td><strong>Reversibility</strong></td>
<td>Yes, with anticholinesterase agents such as Neostigmine</td>
<td>No pharmacological reversal available</td>
</tr>
<tr>
<td><strong>Response to Peripheral Nerve Stimulation with Partial Block</strong></td>
<td>Lower than normal twitch height</td>
<td>Lower than normal twitch height</td>
</tr>
<tr>
<td></td>
<td>Gradual fade of twitch height with single twitch stimulus applied as a train of four (TOF) and with tetanus</td>
<td>NO fade of twitch height with single twitch stimulus applied as a train of four (TOF) or with tetanus</td>
</tr>
</tbody>
</table>

### Succinylcholine (SCh)

- SCh = physically resembles two ACh molecules joined end to end
- metabolism of SCh by plasma cholinesterase, 1/3,000 have atypical plasma cholinesterase (pseudocholinesterase) resulting in abnormally long duration of paralysis
- side effects of SCh
  1. SCh also binds to autonomic cholinergic receptors
     - muscarinic receptors in heart can cause sinus bradycardia
       (especially in children or with repeat bolus in less than 10 minutes)
     - muscarinic receptors in salivary glands resulting in increased secretions, especially in children
  2. hyperkalemia
     - potassium release due to persistent depolarization
     - increase of 0.5 mEq/L with standard bolus
     - increase of 4.0 to 8.0 mEq/L in severe burns, denervated muscles (plegias), major trauma, tetanus; but use of SCh is generally safe in the first 24 hours
  3. other side effects
     - increased ICP/intraocular pressure (IOP)/intragastric pressure
     - triggers malignant hyperthermia
     - sustained contraction in myotonia
     - fasciculations
- defasciculation: a small dose of non-depolarizing agent given before SCh may reduce some side effects (fasciculations, increased ICP, IOP, myalgia); however, SCh efficacy is decreased, thus SCh has to be given in a 30-50% higher dose
GENERAL ANESTHETIC AGENTS . . . CONT.

- contraindications to SCh use
  - upper and lower motor neuron lesions (UMN/LMN), burns, etc.
  - allergy, hypersensitivity
  - malignant hyperthermia
  - lack of necessary skill or equipment to intubate
  - suspected difficult intubation (e.g. facial/neck trauma, unstable cervical spine, etc.)
  - hyperkalemia
  - myotonia congenita, muscular dystrophy
  - decreased levels/atypical plasma cholinesterase (pseudocholinesterase)
  - open eye injury

Reversing Agents for Non-depolarizing Blockade (e.g. Neostigmine, Pyridostigmine)
- reversible anticholinesterases
- inhibit enzymatic degradation of ACh; increases ACh at nicotinic receptors, displacing the non-depolarizing muscle relaxant
- if non-depolarizing blockade is COMPLETE, increasing amount of ACh has little effect; therefore anticholinesterase has little effect and should not be administered until the block is PARTIAL
- blockade assessed with nerve stimulator before administration of reversal
  (no twitch response = 100% blockade)
- with reversal, ACh concentration will increase at muscarinic (before nicotinic) sites causing bradycardia, salivation etc.
- therefore simultaneous administration of atropine or glycopyrrolate is necessary to decrease cholinergic side effects by causing muscarinic receptor blockade

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 crosses 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
- infection
NERVE FIBRES

Different types categorized as follows:

1. MYELINATED A FIBERS (largest to smallest)
   - alpha: motor function, proprioception
   - beta: pressure and touch, some motor function
   - gamma: muscle spindle tone
   - delta: pain and temperature

2. THIN MYELINATED B FIBERS
   - preganglionic axons

3. UNMYELINATED C FIBERS
   - pain and temperature

Order of blockade with local anesthetic (LA):

<table>
<thead>
<tr>
<th>Fibres</th>
<th>Function</th>
</tr>
</thead>
<tbody>
<tr>
<td>B</td>
<td>sympathetic blockade</td>
</tr>
<tr>
<td>A-delta and C</td>
<td>pain</td>
</tr>
<tr>
<td>A-beta and A-gamma</td>
<td>touch</td>
</tr>
<tr>
<td>A-alpha</td>
<td>motor, proprioception and vibration</td>
</tr>
</tbody>
</table>

Since sympathetic blockade (with hypotension, bradycardia) occurs early, it is a potentially dangerous side effect of spinal/epidural anesthesia.

Titration of LA dosage for differential blockade, e.g. can block pain but preserve motor function.

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 hematomas
  - Spinal cord trauma, infection
REGIONAL ANESTHESIA...CONT.

- 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

IV REGIONAL ANESTHESIA

- provides very good anesthesia and muscle relaxation for operations up to 1.5 hours on the upper/lower extremity (UE/LE)
- more commonly used for upper extremity
- primary blockade at nerve trunks
- significant secondary blockade at sensory nerve endings and NMJ
- risk of systemic LA toxicity (i.e. tourniquet failure)

Advantages

- reliable
- relatively simple technique
- very few absolute contraindications

Contraindications

- patient refusal
- allergy or hypersensitivity to LA
- thrombophlebitis
- conditions where a tourniquet cannot be used (e.g. sickle cell disease)

Technique involves

1. cannulation of peripheral vein
2. exsanguination of limb by elevation and bandage application
3. arterial tourniquet inflated to a pressure of 100 mm Hg above patient's systolic pressure
4. inject low concentration lidocaine (e.g. 0.5%) without epinephrine via cannula

Note: pain at site of tourniquet can be avoided by using a double tourniquet - anesthesia is induced with proximal tourniquet inflated, then distal cuff inflated and proximal cuff deflated

PERIPHERAL NERVE BLOCKS

- e.g. brachial plexus block, ankle block, digital ring block
- relatively safe – avoid intraneural injection and neurotoxic agents
- provides good operating conditions

OBSTETRICAL ANESTHESIA (see Obstetrics Chapter)

- all patients entering the delivery room potentially require anesthesia, whether planned or as an emergency
- adequate anesthesia of obstetric patients requires a clear understanding of maternal and fetal physiology
- options for pain relief during parturition (labour) are
  1) psychoprophylaxis – Lamaze method
     - patterns of breathing and focused attention of fixed object
  2) systemic medication
     - easy to administer but risk of maternal or neonatal depression
     - common drugs: opioids, tranquilizers, ketamine
  3) regional anesthesia
     - provides excellent analgesia with minimal depressant effects in mother and fetus
     - hypotension as a consequence of sympathectomy is the most common complication
     - maternal BP monitored q2-5 min for 15-20 min after initiation and regularly thereafter
     - techniques used: epidural, combined spinal epidural, pudendal blocks, spinal, paracervical, lumbar sympathetic blocks
  4) inhalational analgesia
     - easy to administer, makes uterine contractions more tolerable, but does not relieve pain completely
     - 50% nitrous oxide

Anesthesia for cesarean section

1. Regional - spinal or epidural
2. General - used when contraindications to regional or time precludes regional blockade

Potential complications

- pulmonary aspiration – due to increased gastroesophageal reflux
- hypotension and/or fetal distress - caused by occlusion of the inferior vena cava (IVC)/aorta by the gravid uterus (aortocaval compression) therefore corrected by turning patient in the left lateral decubitus (LLD) position
- unintentional total spinal anesthesia
- LA induced seizures – as a result of intravascularization of LA
- postdural puncture headache
- nerve injury - rare
LOCAL INFILTRATION, HEMATOMA BLOCKS

Local Infiltration
- injection of tissue with LA, producing a lack of sensation in the infiltrated area due to LA acting on nerve endings
- one of the simplest and safest techniques of providing anesthesia
- suitable for small incisions, suturing, excising small lesions
- can use fairly large volumes of dilute LA to infiltrate a large area (see maximum dose below)
- low concentrations of epinephrine (1:100,000-1:200,000) cause vasoconstriction thus reducing bleeding and prolonging the effects of LA by reducing systemic absorption (contraindicated in fingers, nose, penis, toes and ears)

Fracture Hematoma Block
- special type of local infiltration for pain control in the manipulation of certain fractures
- hematoma created by fracture is infiltrated with LA to anesthetize surrounding tissues
- sensory blockade may be only partial
- no muscle relaxation

LOCAL ANESTHETICS (LA)

- Local Anesthetics (e.g. lidocaine, bupivicaine, mepivacaine, chlorprocaine and "TAC" - mixture of tetracaine, adrenaline and cocaine)

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, bupivicaine) 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)
- bupivicaine 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
LOCAL ANESTHETICS (LA) . . . CONT.

- CVS effects
  - vasodilation, 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 (increases seizure threshold)
    - SCh (stops muscular manifestations of seizures, facilitates intubation)

SPECIAL CONSIDERATIONS

ATYPICAL PLASMA CHOLINESTERASE
- plasma cholinesterase also known as Pseudocholinesterase
- plasma cholinesterase variants decrease SCh hydrolysis (metabolism) and thus prolong muscle paralysis
- suspect if patient has personal or family history of anesthetic related complications
- treatment
  - ABCs
  - ventilate till normal muscle strength returns as no SCh direct antagonist exists

ENDOCRINE DISORDERS
- adrenocortical insufficiency
  - Addison’s, exogenous steroids
  - treatment with steroids pre-, peri-, post-op
- diabetes mellitus (DM)
  - hypo/hyperglycemia due to drugs + stress
  - hypoglycemia masked by anesthesia
  - treatment: dextrose/insulin, monitor blood glucose
- pheochromocytoma
  - adrenergic crisis with surgical manipulation
  - prevention with alpha + beta adrenergic blockade pre-op
- “thyroid storm” (rare)
  - especially labour/delivery, anesthesia
  - treatment: beta blockers + pre-op prophylaxis

MALIGNANT HYPERTHERMIA (MH)
- hypermetabolic disorder of skeletal muscle
- autosomal dominant (AD) pattern of genetic inheritance (possibly)
- incidence of 1-5:100,000, may be associated with skeletal muscle abnormalities such as ptosis, hernia, scoliosis
- intracellular hyperCa²⁺ (due to altered Ca²⁺ sequestration) with resultant hypercatabolism and decreased ATP
- anesthetic drugs triggering MH crises include
  - volatile anesthetics: enflurane, halothane, isoflurane and sevoflurane (any drug ending in “ane”)
  - depolarizing relaxants SCh, decamethonium

Signs and Symptoms
- immediate or hours after contact with trigger agent
- increased end-tidal endotracheal CO₂ monitoring on capnograph
- tachycardia/dysrhythmia
- tachypnea/cyanosis
- increased temperature - may be delayed
- hypertension
diaphoresis
- trismus (masseter spasm) common but not specific for MH
  - (occurs in 1% of children given SCh with halothane anesthesia)

Lab
- hyper CO₂, hypoxia (early)
- metabolic acidosis
- respiratory acidosis
- hyperkalemia
- myoglobinemia/myoglobinuria
- increased creatine kinase (CK)
SPECIAL CONSIDERATIONS . . . CONT.

Complications
- death/coma
- disseminated intravascular coagulation (DIC)
- muscle necrosis/weakness
- myoglobinuric renal failure
- electrolyte abnormalities (i.e., iatrogenic hypokalemia)

Prevention
- suspect possible MH in patients presenting with a family history of problems/death with anesthetic
- dantrolene prophylaxis no longer routine
- avoid all triggers
- central body temp and ET CO₂ monitoring
- use regional anesthesia if possible
- use equipment “clean” of trigger agents

Management
- discontinue inhaled anesthetic and Sch, terminate procedure
- hyperventilate with 100% O₂
- Dantrolene 1 mg/kg, repeating until stable or 10 mg/kg maximum reached
  (Dantrolene interferes with calcium release into myoplasm from sarcoplasmic reticulum)
- treat metabolic/physiologic derangements accordingly
- control body temperature
- diligent monitoring (especially CVS, lytes, ABGs, urine output)

MYOCARDIAL INFARCTION (MI)
- ELECTIVE surgery should not be carried out within 6 months of an MI:
  this period carries increased risk of reinfarction/death
- classic reinfarction risk is quoted as:
  - <3 months after MI - 37% patients may reinfarct
  - 3-6 months after MI - 15%
  - >6 months after MI - risk remains constant at 5%
  - reinfarction carries a 50% mortality rate
- if operative procedure is essential and cannot be delayed,
  the risk may be lessened by invasive monitoring + post-op ICU
  monitoring to 6%, 3% and 1%, respectively for the above time periods
- mortality with perioperative MI is 20-50%
- infarct rate in absence of prior MI is 0.13%

RESPIRATORY DISEASES
- ventilation and delivery of volatile anesthetics can be complicated by pulmonary disease states
  (e.g., volume changes, decreased diffusion, hyperactive airways, laryngeal spasm, obesity,
  altered compliance, secretions, etc.)
- anticipate + optimize pre-operatively to prevent intra-op and post-op problems

ASPIRATION SYNDROME
- severity of gastric aspiration related to volume of the aspirate, quality of aspirate
  (i.e., acidity and presence of contaminated particles) and the health status of the patient
- avoid inhibiting airway reflexes, reduce gastric volume and acidity, employ rapid sequence induction

REFERENCES