A Anesthesia and Perioperative Medicine

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Acronyms

2,3-BPG	2,3-Bisphosphoglycerate
ACC	American College of Cardiology
ACh	acetylcholine
AChE	acetylcholinesterase
ACV	assist-control ventilation
AHA	American Heart Association
ALS	amyotrophic lateral sclerosis
ARDS	acute respiratory distress syndrome
atm	atmosphere
CCS	Canadian Cardiovascular Society
CK	creatine kinase
CO	cardiac output
CSF	cerebrospinal fluid
CVP	central venous pressure
DIC	disseminated intravascular coagulation
$ETCO_2$	End-Tidal CO_2
ETT	endotracheal tube
FiO2	fraction of oxygen in inspired air
FFP	fresh frozen plasma
FRC	functional residual capacity
GA	general anesthetic
GERD	gastroesophageal reflux disease
Hb(i)	initial hematocrit
Hb(f)	final hematocrit
Hct	hematocrit
HES	hydroxyethyl starch
ICP	intracranial pressure
ICS	inhaled corticosteroids
IOP	intraocular pressure
LA	local anesthetic
LABA	long-acting β-agonist
LES	lower esophageal sphincter
LMA	laryngeal mask airway
LOC	level of consciousness
MAC	minimum alveolar concentration
MAP	mean arterial pressure
MH	malignant hyperthermia
MS	multiple sclerosis
NMJ	neuromuscular junction
NYHA	New York Heart Association
OCS	oral costicosteroids
OR	operating room
$PaCO_2$	arterial partial pressure of carbon dioxide
PaO_2	arterial partial pressure of oxygen
PC	patient-controlled
PCA	patient-controlled analgesia
PCV	pressure-controlled ventilation
PEEP	positive end-expiratory pressure
PNS	parasympathetic nervous system
PONV	post-operative nausea and vomiting
PPV	positive pressure ventilation
RSI	rapid sequence induction
SABA	short-acting β-agonist
SCh	succinylcholine
SIADH	syndrome of inappropriate antidiuretic hormone
SNS	sympathetic nervous system
SV	stroke volume
SVR	systemic vascular resistance
TBW	total body water
TIVA	total intravenous anesthetic
TURP	transurethral resection of prostate
V/Q	ventilation/perfusion
VT	ventricular tachycardia
VTE	venous thromboembolism

Overview of Anesthesia

- anesthesia: lack of sensation/perception
- **approach to anesthesia**

1. pre-operative assessment
2. patient optimization
3. plan anesthetic
   - various types of anesthesia
   - pre-medication
   - airway management
   - monitors
   - induction
   - maintenance
   - emergence
   - extubation
4. post-operative care

6 As of General Anesthesia
Anesthesia
Anxiolysis
Amnesia
Areflexia (muscle relaxation - not always required)
Autonomic stability
Analgesia

Types of Anesthesia

- **general**
  - general anesthesia (GA)
  - total IV anesthesia (TIVA)
- **regional**
  - spinal, epidural
  - peripheral nerve block
  - IV regional
- **local**
  - local infiltration
  - topical

Note that different types of anesthesia can be combined (general + regional)

Pre-Operative Assessment

Purpose
- identify concerns for medical and surgical management of patient
- allow for questions to help allay any fears or concerns patient and/or family may have
- arrange further investigations, consultations and treatments for patients not yet optimized
- plan and consent for anesthetic techniques

History and Physical

History
- age, gender
- indication for surgery
- surgical/anesthetic Hx: previous anesthetics, any complications, previous intubations, medications, drug allergies, post-operative N/V
- FHx: abnormal anesthetic reactions, malignant hyperthermia, pseudocholinesterase deficiency (see *Uncommon Complications*, A28)

- PMHx
  - CNS: seizures, TIA/strokes, raised ICP, spinal disease, aneurysm
  - CVS: angina/CAD, MI, CHF, HTN, valvular disease, dysrhythmias, peripheral vascular disease (PVD), conditions requiring endocarditis prophylaxis, exercise tolerance, CCS/NYHA class (Cardiology and Cardiac Surgery, C35 sidebar for *NYHA Classification*)
  - respiratory: smoking, asthma, COPD, recent upper respiratory tract infection, sleep apnea
  - GI: GERD, liver disease, NPO status
  - renal: insufficiency, dialysis, chronic kidney disease
  - hematologic: anemia, coagulopathies, blood dyscrasias
  - MSK: conditions associated with difficult intubations – arthritides (e.g. rheumatoid arthritis), cervical tumours, cervical infections/abscesses, trauma to cervical spine, previous cervical spine surgery, Trisomy 21, scleroderma, conditions affecting neuromuscular junction (e.g. myasthenia gravis)
  - endocrine: DM, thyroid disorders, adrenal disorders
  - other: morbid obesity, pregnancy, ethanol/other drug use

Physical Exam

- weight, height, BP, pulse, respiratory rate, oxygen saturation
- focused physical exam of the CNS, CVS, and respiratory systems
- general assessment of nutrition, hydration, and mental status
- airway assessment is done to determine intubation difficulty (no single test is specific or sensitive) and ventilation difficulty
  - cervical spine stability and neck movement – upper cervical spine extension, lower cervical spine flexion ("sniffing position")
  - Mallampati classification
  - "3-2-1 rule"
    - thyromental distance (distance of lower mandible in midline from the mentum to the thyroid notch); <3 finger breadths (<6 cm) is associated with difficult intubation
    - mouth opening (<2 finger breadths is associated with difficult intubation)
    - anterior jaw subluxation (<1 finger breadth is associated with difficult intubation)
  - tongue size
  - dentition, dental appliances/prosthetic caps, existing chipped/loose teeth – pose aspiration risk if dislodged and must inform patients of rare possibility of damage
  - nasal passage patency (if planning nasotracheal intubation)
  - assess potential for difficult ventilation
- examination of anatomical sites relevant to lines and blocks
  - bony landmarks and suitability of anatomy for regional anesthesia (if relevant)
  - sites for IV, central venous pressure (CVP), and pulmonary artery (PA) catheters

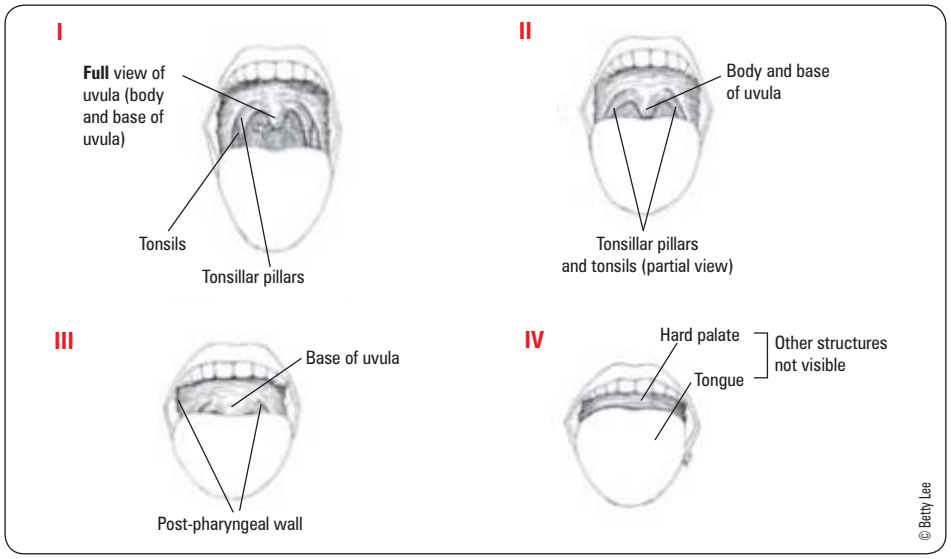

Figure 1. Mallampati classification of oral opening

Evaluation of Difficult Airway

LEMON
Look – obesity, beard, dental/facial abnormalities, neck, facial/neck trauma
Evaluate – 3-2-1 rule
Mallampati score
Obstruction – stridor, foreign bodies
Neck mobility

To Assess for Ventilation Difficulty

BONES
Beard
Obesity (BMI>26)
No teeth
Elderly (age>55)
Snoring Hx (sleep apnea)

Pre-Operative Investigations

- routine pre-operative investigations are only necessary if there are comorbidities or certain indications

Table 1. Suggested Indications for Specific Investigations in the Pre-Operative Period

Test	Indications
CBC	Major surgery requiring group and screen or cross and match; chronic cardiovascular, pulmonary, renal, or hepatic disease; malignancy; known or suspected anemia; bleeding diathesis or myelosuppression; patient >1 yr of age
Sickle Cell Screen	Patients from geographic areas with high prevalence of sickle cell disease and/or genetically predisposed patients (hemoglobin electrophoresis if screen is positive)
INR, aPTT	Anticoagulant therapy, bleeding diathesis, liver disease
Electrolytes and Creatinine	Hypertension, renal disease, DM, pituitary or adrenal disease; vascular disease, digoxin, diuretic, or other drug therapies affecting electrolytes
Fasting Glucose Level	DM (repeat on day of surgery)
Pregnancy (β-hCG)	Women of reproductive age
ECG	Heart disease, DM, other risk factors for cardiac disease; subarachnoid or intracranial hemorrhage, cerebrovascular accident, head trauma
Chest Radiograph	Patients with new or worsening respiratory symptoms/signs

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American Society of Anesthesiology Classification

- common classification of physical status at the time of surgery
- a gross predictor of overall outcome, NOT used as stratification for anesthetic risk (mortality rates)
- **ASA 1:** a healthy, fit patient
- **ASA 2:** a patient with mild systemic disease
  - e.g. controlled Type 2 DM, controlled essential HTN, obesity, smoker
- **ASA 3:** a patient with severe systemic disease that limits activity
  - e.g. stable CAD, COPD, DM, obesity
- **ASA 4:** a patient with incapacitating disease that is a constant threat to life
  - e.g. unstable CAD, renal failure, acute respiratory failure
- **ASA 5:** a moribund patient not expected to survive 24 h without surgery
  - e.g. ruptured abdominal aortic aneurysm (AAA), head trauma with increased ICP
- **ASA 6:** declared brain dead, a patient whose organs are being removed for donation purposes
- for emergency operations, add the letter E after classification (e.g. ASA 3E)

Pre-Operative Optimization

- in general, prior to elective surgery
  - any fluid and/or electrolyte imbalance should be corrected
  - extent of existing comorbidities should be understood and these conditions should be optimized prior to surgery
  - medications may need adjustment

Medications

- pay particular attention to cardiac and respiratory medications, opioids and drugs with many side effects and interactions
- **pre-operative medications to consider**
  - prophylaxis
    - risk of GE reflux: sodium citrate and/or ranitidine and/or metoclopramide 30 min-1 h prior to surgery
    - risk of infective endocarditis, GI/GU interventions: antibiotics
    - risk of adrenal suppression: steroid coverage
    - anxiety: consider benzodiazepines
    - COPD, asthma: bronchodilators
    - CAD risk factors: nitroglycerin and β-blockers
- **pre-operative medications to stop**
  - oral antihyperglycemics: stop on morning of surgery
  - ACE inhibitors and angiotension receptor blockers: may stop on morning of surgery (controversial)

Impact of Anesthesia Management Characteristics on Severe Morbidity and Mortality
Anesth 2005;102:257-268
Study: Case-control study of patients undergoing anesthesia.
Patients: 807 cases and 883 controls were analyzed among a cohort of 869,483 patients undergoing anesthesia between 1995-1997. Cases were defined as patients who either remained comatose or died within 24 h of receiving anesthesia. Controls were defined as patients who neither remained comatose nor died within 24 h of receiving anesthesia.
Intervention: General, regional, or combined anesthesia to patients undergoing a surgical procedure.
Main Outcome: Coma or death within 24 h of receiving anesthesia.
Results: The incidence of 24 h post-operative death was 8.8 per 10,000 anesthetics (95% CI 8.2-9.5) and the incidence of coma was 0.5 (95% CI 0.3-0.6). Anesthesia management risk factors that were associated with a decreased risk of morbidity and mortality were equipment check with protocol and documentation, directly available anesthesiologist with no change during anesthesia, 2 persons present at emergence of anesthesia, reversal of muscle relaxation, and post-operative pain medication.

Aspirin® in Patients undergoing Non-Cardiac Surgery
NEJM 2014;370:1494-1503
Purpose: This study evaluated the effect of low-dose ASA on the risk of death or non-fatal MI in 10,010 patients undergoing non-cardiac surgery. Patients were randomized into two groups in a double-blind process.
Methods: RCT in which ASA or placebo to be taken shortly before surgery and in the early post-operative period. The primary outcome was a composite of death or non-fatal MI.
Results: Death or non-fatal MI occurred in 7.0% in the ASA group and 7.1% in the placebo group. Major bleeding was more common in the ASA group than the placebo group.
Conclusion: Administration of ASA before surgery and throughout the early post-surgical period had no significant effect on the incidence of death or MI but increased the risk of major bleeding.

  - warfarin (consider bridging with heparin), anti-platelet agents (e.g. clopidogrel)
    - discuss perioperative use of ASA, NSAIDs with surgeon (± patient's cardiologist/internist)
    - in patients undergoing non-cardiac surgery, starting or continuing low-dose aspirin in the perioperative period does not appear to protect against post-operative MI or death, but increases the risk of major bleeding
      – note: this does not apply to patients with bare metal stents or drug-eluting coronary stents
- **pre-operative medications to adjust**
  - insulin (consider insulin/dextrose infusion or holding dose), prednisone, bronchodilators

Hypertension

- BP <180/110 is not an independent risk factor for perioperative cardiovascular complications
- target SBP <180 mmHg, DBP <110 mmHg
- assess for end-organ damage and treat accordingly

Coronary Artery Disease

- ACC/AHA Guidelines (2014) recommend that at least 60 days should elapse after a MI before a non-cardiac surgery in the absence of a coronary intervention
  - this period carries an increased risk of re-infarction/death
  - if operative procedure is essential and cannot be delayed then invasive intra- and post-operative ICU monitoring is required to reduce the above risk
- mortality with perioperative MI is 20-50%
- perioperative β-blockers
  - may decrease cardiac events and mortality (controversial, as recent data suggests stroke risk)
  - continue β-blocker if patient is routinely taking it prior to surgery
  - consider initiation of β-blocker in
    - patients with CAD or indication for β-blocker
    - intermediate or high risk surgery, especially vascular surgery

Effects of Extended-Release Metoprolol Succinate in Patients undergoing Non-Cardiac Surgery (POISE Trial): A Randomized Controlled Trial
Lancet 2008;371:1839-1847
Purpose: To investigate the role of β-blockers (metoprolol) perioperatively in patients with known vascular disease undergoing non-cardiac surgery.
Methods: Patients from 190 centres in 23 countries were eligible if they were age >45, undergoing non-cardiac surgery, and were known to have significant vascular disease. Patients were randomized to either the metoprolol group or placebo. Participants received metoprolol (or placebo) 100 mg 2-4 h prior to surgery, 6 h after surgery, and then 20 mg daily for 30 d. The primary endpoint was a composite of cardiovascular death, non-fatal myocardial infarction, and non-fatal cardiac arrest. Analysis was by intention to treat.
Results: 8,331 patients were recruited into the study, with 8,331 completing the 30 d course. Use of metoprolol was found to significantly reduce the risk of cardiovascular death, non-fatal MI, or non-fatal cardiac arrest vs. placebo (hazard ratio 0.84, $p<0.05$) but significantly increased the rate of stroke (hazard ratio 2.17, $p<0.01$) and overall risk of death (hazard ratio 1.33, $p<0.05$).
Conclusion: Use of perioperative β-blockers (metoprolol) in patients with known vascular disease provides both risks and benefits, and these must be considered for each patient individually.

Respiratory Diseases

- smoking
  - adverse effects: altered mucus secretion and clearance, decreased small airway calibre, and altered immune response
  - abstain at least 8 wk pre-operatively if possible
  - if unable, abstaining even 24 h pre-operatively has been shown to increase oxygen availability to tissues
- asthma
  - pre-operative management depends on degree of baseline asthma control
  - increased risk of bronchospasm from intubation
    - administration of short course (up to 1 wk) pre-operative corticosteroids and inhaled β2-agonists decreases the risk of bronchospasm and does not increase the risk of infection or delay wound healing
  - avoid non-selective β-blockers due to risk of bronchospasm
  - cardioselective β-blockers (metoprolol, atenolol) do not increase risk of bronchospasm in the short-term
  - delay elective surgery for poorly controlled asthma (increased cough or sputum production, active wheezing)
  - delay elective surgery by a minimum of 6 wk if patient develops URTI
- COPD
  - anesthesia, surgery (especially abdominal surgery, in particular upper abdominal surgery) and pain predispose the patient to atelectasis, bronchospasm, pneumonia, prolonged need for mechanical ventilation, and respiratory failure
  - pre-operative ABG is needed for all COPD stage II and III patients to assess baseline respiratory acidosis and plan post-operative management of hypercapnea
  - cancel/delay elective surgery for acute exacerbation

β-blockers
- β1-receptors are located primarily in the heart and kidneys
- β2-receptors are located in the lungs
- Non-selective β-blockers block β1 and β2-receptors (labetalol, carvedilol, nadolol). Caution is required with non-selective β-blockers, particularly in patients with respiratory conditions where β2 blockade can result in airway reactivity

Aspiration

- increased risk of aspiration with
  - decreased LOC
  - trauma
  - meals within 8 h
  - suspected sphincter incompetence (GERD, hiatus hernia, nasogastric tube)
  - increased abdominal pressure (pregnancy, obesity, bowel obstruction, acute abdomen)
  - laryngeal mask vs. endotracheal tube (ETT)

- management
  - reduce gastric volume and acidity
  - delay inhibiting airway reflexes with muscular relaxants
  - employ rapid sequence induction (see *Rapid Sequence Induction*, A15)

Fasting Guidelines

Fasting Guidelines Prior to Surgery (Canadian Anesthesiologists' Society)

- **8 h** after a meal that includes meat, fried or fatty foods
- **6 h** after a light meal (such as toast or crackers) or after ingestion of infant formula or non-human milk
- **4 h** after ingestion of breast milk
- **2 h** after clear fluids (water, black coffee, tea, carbonated beverages, juice without pulp)

Hematological Disorders

- history of congenital or acquired conditions (sickle cell anemia, factor VIII deficiency, ITP, liver disease)
- evaluate hemoglobin, hematocrit and coagulation profiles when indicated (Table 1)
- anemia
  - pre-operative treatments to increase hemoglobin (erythropoietin or pre-admission blood collection in certain populations)
- coagulopathies
  - discontinue or modify anticoagulation therapies (warfarin, clopidogrel, ASA) in advance of elective surgeries
  - administration of reversal agents if necessary: vitamin K, FFP, prothrombin complex concentrate, recombinant activated factor VII

Endocrine Disorders

- diabetes mellitus
  - clarify type 1 vs. type 2
  - clarify treatment – oral anti-hyperglycemics and/or insulin
  - assess glucose control with history and HbA1c; well controlled diabetics have more stable glucose levels intraoperatively
  - end organ damage: be aware of damage to cardiovascular, renal, and nervous systems, including autonomic neuropathy
  - formulate intraoperative glucose management plan based on type (1 vs. 2), glucose control, and extent of end organ damage
- hyperthyroidism
  - can experience sudden release of thyroid hormone (thyroid storm) if not treated or well-controlled preoperatively
  - treatment: β-blockers and pre-operative prophylaxis
- adrenocortical insufficiency (Addison's, exogenous steroid use)
  - consider intraoperative steroid supplementation

Obesity and Obstructive Sleep Apnea

- assess for co-morbid conditions in obese patient (independent risk factor for CVD, DM, OSA, cholelithiasis, HTN)
- previously undiagnosed conditions may require additional testing to characterize severity
- both obesity and OSA increase risk of difficult ventilation, intubation and post-operative respiratory complications
  - risk may be magnified with both diseases present

Monitoring

Canadian Guidelines to the Practice of Anesthesia and Patient Monitoring

- an anesthetist present: "the only indispensable monitor"
- a completed pre-anesthetic checklist: including ASA class, NPO policy, Hx and investigations
- a perioperative anesthetic record: HR and BP every 5 min, O_2 saturation, End Tidal CO_2, dose and route of drugs and fluids
- continuous monitoring: see *Routine Monitors for All Cases*

Routine Monitors for All Cases

- pulse oximeter, BP monitor, electrocardiography and capnography are required for general anesthesia and sedation (Ramsey Sedation Scale 4-6), agent-specific anesthetic gas monitor when inhalational anesthetic agents are used
- the following must also be available: temperature probe, peripheral nerve stimulator, stethoscope, appropriate lighting, spirometer

Pre-Anesthetic Checklist

SAMMM
Suction: connected and working
Airways: laryngoscope and blades, ETT, syringe, stylet, oral and nasal airways, tape, bag, and mask
Machine: connected, pressures okay, all meters functioning, vaporizers full
Monitors: available, connected, and working
Medications: IV fluids and kit ready, emergency medicines in correct location and accessible

Elements to Monitor

- anesthetic depth
  - inadequate: blink reflex present when eyelashes lightly touched, HTN, tachycardia, tearing or sweating
  - excessive: hypotension, bradycardia
- oxygenation: pulse oximetry, fraction of inspired O_2 (FiO_2)
- ventilation: verify correct position of ETT, chest excursions, breath sounds, $ETCO_2$ analysis, end tidal inhaled anesthesia analysis
- circulation: pulse, rhythm, BP, telemetry, oximetry, CVP, pulmonary capillary wedge pressure
- temperature
- hourly urine output

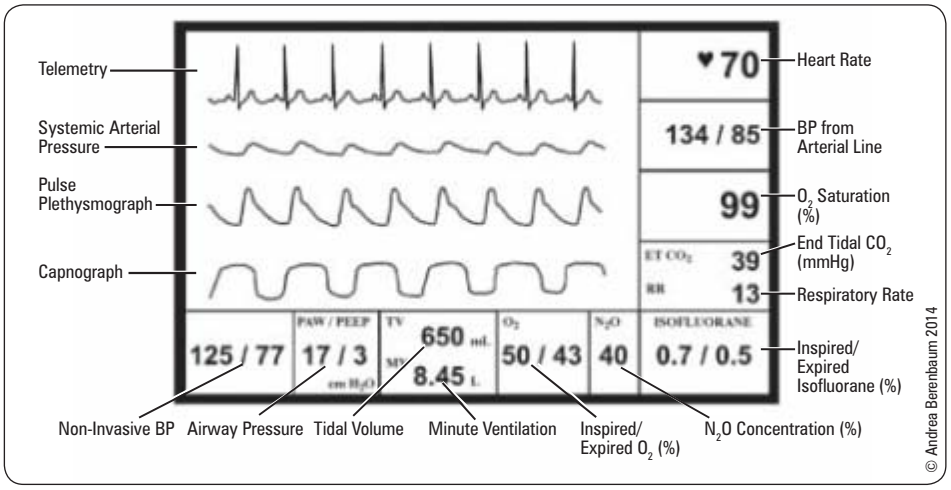

Figure 2. Typical anesthesia monitor

Airway Management

Airway Anatomy

- resistance to airflow through nasal passages accounts for approximately 2/3 of total airway resistance
- pharyngeal airway extends from posterior aspect of the nose to cricoid cartilage
- glottic opening (triangular space formed between the true vocal cords) is the narrowest segment of the laryngeal opening in adults
- the glottic opening is the space through which one visualizes proper placement of the ETT
- the trachea begins at the level of the thyroid cartilage, C6, and bifurcates into the right and left main bronchi at T4-T5 (approximately the sternal angle)

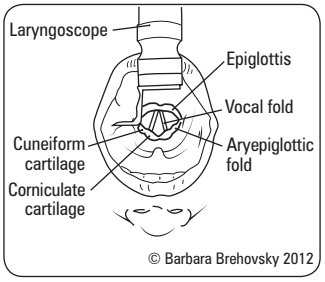

Figure 3. Landmarks for intubation

Methods of Supporting Airways

1. non-definitive airway (patent airway)
   - jaw thrust/chin lift
   - oropharyngeal and nasopharyngeal airway
   - bag mask ventilation
   - LMA
2. definitive airway (patent and protected airway)
   - ETT
   - surgical airway (cricothyrotomy or tracheostomy)

Table 2. Methods of Supporting the Airway

	Bag and Mask	Laryngeal Mask Airway (LMA)	Endotracheal Tube (ETT)
Advantages/ Indications	Basic Non-invasive Readily available	Easy to insert Less airway trauma/irritation than ETT Frees up hands (vs. face mask) Primarily used in spontaneously ventilating patient	Indications for intubation **(5 Ps)** **P**atent airway **P**rotects against aspiration **P**ositive pressure ventilation **P**ulmonary toilet (suction) **P**harmacologic administration also hemodynamic instability
Disadvantages/ Contraindications	Risk of aspiration if decreased LOC Cannot ensure airway patency Inability to deliver precise tidal volume Operator fatigue	Risk of gastric aspiration PPV <20 cm H_2O needed Oropharyngeal/retropharyngeal pathology or foreign body Does not protect against laryngospasm or gastric aspiration	Insertion can be difficult Muscle relaxant usually needed Most invasive – see *Complications During Laryngoscopy and Intubation*, A9
Other	Facilitate airway patency with jaw thrust and chin lift Can use oropharyngeal/ nasopharyngeal airway	Sizing by body weight (approx) 40-50 kg: 3 50-70 kg: 4 70-100 kg: 5	Auscultate to avoid endobronchial intubation Sizing (approx): Male: 8.0-9.0 mm Female: 7.0-8.0 mm Pediatric Uncuffed (>age 2): (age/4) + 4 mm

Tracheal Intubation

Preparing for Intubation

- failed attempts at intubation can make further attempts more difficult due to tissue trauma
- plan, prepare, and assess for potential difficulties (see *Pre-Operative Assessment*, A2)
- ensure equipment is available and working (test ETT cuff, check laryngoscope light and suction, machine check)
- pre-oxygenate/denitrogenate: patient breathes 100% O_2 for 3-5 min or for 4-8 vital capacity breaths
- may need to suction mouth and pharynx first

Proper Positioning for Intubation

- align the three axes (mouth, pharynx, and larynx) to allow visualization from oral cavity to glottis
  - "sniffing position": flexion of lower C-spine (C5-C6), bow head forward, and extension of upper C-spine at atlanto-occipital joint (C1), nose in the air
  - contraindicated in known/suspected C-spine fracture/instability
- laryngoscope tip placed in the epiglottic vallecula in order to visualize cord

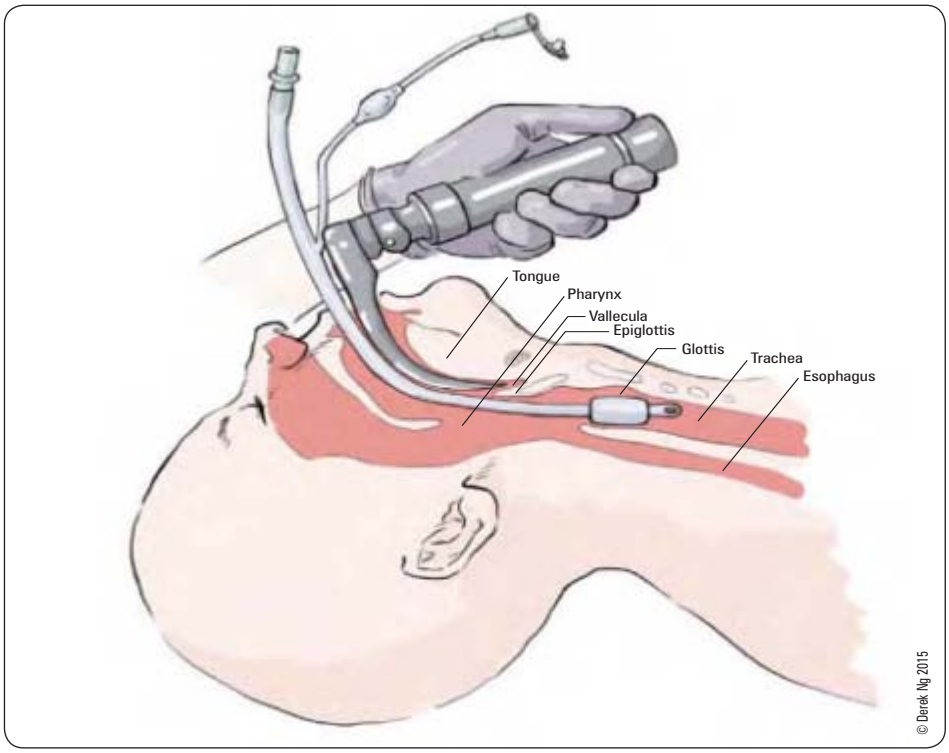

Figure 4. Saggital view of airway with laryngoscope in vallecula

Equipment for Intubation

MDSOLES
Monitors
Drugs
Suction
Oxygen source and self-inflating bag with oropharyngeal and nasopharyngeal airways
Laryngoscope
Endotracheal tubes (appropriate size and one size smaller)
Stylet, **S**yringe for tube cuff inflation

Medications that can be Given Through the ETT

NAVEL
Naloxone
Atropine
Ventolin
Epinephrine
Lidocaine

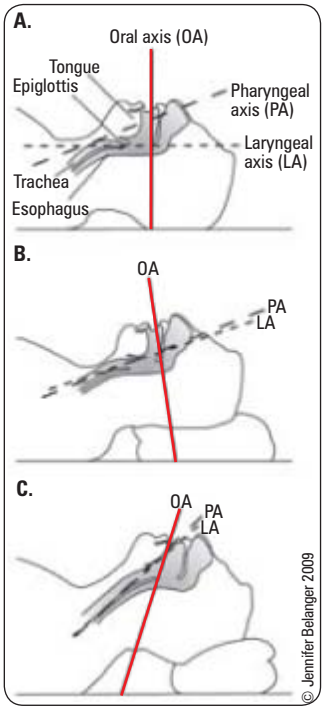

Figure 5. Anatomic considerations in laryngoscopy
A. Neutral position
B. C-spine flexion
C. C-spine flexion with atlanto-occipital extension

Tube Insertion

- laryngoscopy and ETT insertion can incite a significant sympathetic response via stimulation of cranial nerves 9 and 10 due to a "foreign body reflex" in the trachea, including tachycardia, dysrhythmias, myocardial ischemia, increased BP, and coughing
- a malpositioned ETT is a potential hazard for the intubated patient
  - if too deep, may result in right endobronchial intubation, which is associated with left-sided atelectasis and right-sided tension pneumothorax
  - if too shallow, may lead to accidental extubation, vocal cord trauma, or laryngeal paralysis as a result of pressure injury by the ETT cuff
- the tip of ETT should be located at the midpoint of the trachea at least 2 cm above the carina and the proximal end of the cuff should be placed at least 2 cm below the vocal cords
  - approximately 20-23 cm mark at the right corner of the mouth for men and 19-21 cm for women

Confirmation of Tracheal Placement of ETT

- direct
  - visualization of ETT passing through cords
  - bronchoscopic visualization of ETT in trachea
- indirect
  - $ETCO_2$ in exhaled gas measured by capnography – a mandatory method for confirming the ETT is in the airway
  - auscultate for equal breath sounds bilaterally and absent breath sounds over epigastrium
  - bilateral chest movement, condensation of water vapour in ETT visible during exhalation and no abdominal distention
  - refilling of reservoir bag during exhalation
  - CXR (rarely done): only confirms position of the tip of ETT and not that ETT is in the trachea
- esophageal intubation suspected when
  - $ETCO_2$ zero or near zero on capnograph
  - abnormal sounds during assisted ventilation
  - impairment of chest excursion
  - hypoxia/cyanosis
  - presence of gastric contents in ETT
  - breath sounds heard when auscultating over epigastrium/LUQ
  - distention of stomach/epigastrium with ventilation

Differential Diagnosis of Poor Bilateral Breath Sounds after Intubation

DOPE
Displaced ETT
Obstruction
Pneumothorax
Esophageal intubation

Complications During Laryngoscopy and Intubation

- dental damage
- laceration (lips, gums, tongue, pharynx, vallecula, esophagus)
- laryngeal trauma
- esophageal or endobronchial intubation
- accidental extubation
- insufficient cuff inflation or cuff laceration: results in leaking and aspiration
- laryngospasm (see *Extubation*, A20 for definition)
- bronchospasm

Difficult Airway

- difficulties with bag-mask ventilation, supraglottic airway, laryngoscopy, passage of ETT through the cords, infraglottic airway or surgical airway
- algorithms exist for difficult airways (*Can J Anesth* 2013;60:1119-1138; *Anesthesiology* 2003;98:3273; *Anesthesiology* 2013;118:251-270), see *ACLS Guidelines* (Figures 14, A29 and 15, A30)
- pre-operative assessment (history of previous difficult airway, airway examination) and pre-oxygenation are important preventative measures
- if difficult airway expected, consider
  - awake intubation
  - intubating with bronchoscope, trachlight (lighted stylet), fibre optic laryngoscope, glidescope, etc.
- if intubation unsuccessful after induction
  1. CALL FOR HELP
  2. ventilate with 100% O_2 via bag and mask
  3. consider returning to spontaneous ventilation and/or waking patient
- if bag and mask ventilation inadequate
  1. CALL FOR HELP
  2. attempt ventilation with oral airway
  3. consider/attempt LMA
  4. emergency invasive airway access (e.g. rigid bronchoscope, cricothyrotomy, or tracheostomy)

Predicting Difficult Intubation in Apparently Normal Patients
Anesth 2005;103:429-437
Purpose: To assess widely available bedside tests and widely used laryngoscopic techniques in the prediction of difficult intubations.
Study: Meta-analysis.
Patients: 35 studies encompassing 50,760 patients.
Definitions: Difficult intubation was defined usually as Cormack–Lehane grade of 3 or greater, but some authors reported the requirement of a special technique, multiple unsuccessful attempts, or a combination of these as the accepted standard for difficult intubation.
Results: The overall incidence of difficult intubation was 5.8% (95% CI 4.5-7.5%) for the overall patient population, 6.2% (95% CI 4.6-8.3%) for normal patients excluding obstetric and obese patients, 3.1% (95% CI 1.7-5.5%) for obstetric patients, and 15.8% (95% CI 14.3-17.5%) for obese patients Mallampati score: SN:49% SP:86% PLR:3.7 NLR:0.5; thyromental distance: SN:20% SP:94% PLR:3.4 NLR:0.8; sternomental distance: SN:62% SP:82% PLR:5.7 NLR:0.5; mouth opening: SN:22% SP:97% PLR: 4.0 NLR:0.8; Wilson risk-sum: SN:46% SP:89% PLR:5.8 NLR:0.6; combination Mallampati and thyromental distance: SN:36% SP:87% PLR:9.9 NLR:0.6.
Conclusions: A combination of the Mallampati score and thyromental distance is the most accurate at predicting difficult intubation. The PLR (9.9) is supportive of the test as a good predictor of difficult intubation.
PLR: positive likelihood ratio; NLR: negative likelihood ratio; SN: sensitivity; SP: specificity

Oxygen Therapy

- in general, the goal of oxygen therapy is to maintain arterial oxygen saturation (SaO_2) > at a minimum, 90%
- small decrease in saturation below SaO_2 of 90% corresponds to a large drop in arterial partial pressure of oxygen (PaO_2)
- in intubated patients, oxygen is delivered via the ETT
- in patients not intubated, there are many oxygen delivery systems available; the choice depends on oxygen requirements (FiO_2) and the degree to which precise control of delivery is needed
- cyanosis can be detected at SaO_2 <85%, frank cyanosis at SaO_2 = 67%

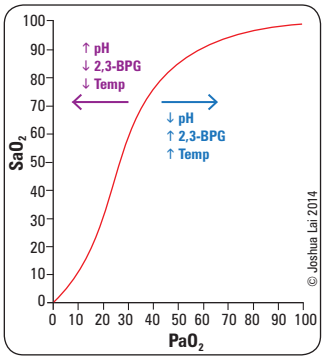

Figure 6. HbO_2 saturation curve

Low Flow Systems

- provide O_2 at flows between 0-10 L/min
- acceptable if tidal volume 300-700 mL, respiratory rate (RR) <25, consistent ventilation pattern
- dilution of oxygen with room air results in a decrease in FiO_2
- an increase in minute ventilation (tidal volume x RR) results in a decrease in FiO_2
- e.g. nasal cannula (prongs)
  - well tolerated if flow rates <5-6 L/min; drying of nasal mucosa at higher flows
  - nasopharynx acts as an anatomic reservoir that collects O_2
  - delivered oxygen concentration (FiO_2) can be estimated by adding 4% for every additional litre of O_2 delivered
  - provides FiO_2 of 24-44% at O_2 flow rates of 1-6 L/min

Reservoir Systems

- use a volume reservoir to accumulate oxygen during exhalation thus increasing the amount of oxygen available for the next breath
- simple face mask
  - covers patient's nose and mouth and provides an additional reservoir beyond nasopharynx
  - fed by small bore O_2 tubing at a rate of at least 6 L/min to ensure that exhaled CO_2 is flushed through the exhalation ports and not rebreathed
  - provides FiO_2 of 55% at O_2 flow rates of 10 L/min
- non-rebreather mask
  - a reservoir bag and a series of one-way valves prevent expired gases from re-entering the bag
  - during the exhalation phase, the bag accumulates with oxygen
  - provides FiO_2 of 80% at O_2 flow rates of 10-15 L/min

Composition of Air
78.1% nitrogen
20.9% oxygen
0.9% argon
0.04% carbon dioxide

High Flow Systems

- generate flows of up to 50-60 L/min
- meet/exceed patient's inspiratory flow requirement
- deliver consistent and predictable concentration of O_2
- Venturi mask
  - delivers specific FiO_2 by varying the size of air entrapment
  - oxygen concentration determined by mask's port and NOT the wall flow rate
  - enables control of gas humidity
  - FiO_2 ranges from 24-50%

Ventilation

- ventilation is maintained with PPV in patients given muscle relaxants
- assisted or controlled ventilation can also be used to assist spontaneous respirations in patients not given muscle relaxants as an artificial means of supporting ventilation and oxygenation

Mechanical Ventilation

- indications for mechanical ventilation
  - apnea
  - hypoventilation/acute respiratory acidosis
  - intraoperative positioning limiting respiratory excursion (e.g. prone, Trendelenburg)
  - required hyperventilation (to lower ICP)
  - deliver positive end expiratory pressure (PEEP)
  - increased intrathoracic pressure (e.g. laparoscopic procedure)
- complications of mechanical ventilation
  - airway complications
    - tracheal stenosis, laryngeal edema
    - alveolar complications
    - ventilator-induced lung injury (barotrauma, volutrauma, atelectatrauma), ventilator-associated pneumonia (nosocomial pneumonia), inflammation, auto-PEEP, patient-ventilator asynchrony
    - cardiovascular complications
    - reduced venous return (secondary to increased intrathoracic pressure), reduced cardiac output, hypotension

Changes in peak pressures in ACV and tidal volumes in PCV may reflect changes in lung compliance and/or airway resistance – patient may be getting better or worse

Positive End Expiratory Pressure (PEEP)
- Positive pressure applied at the end of ventilation that helps to keep alveoli open, decreasing V/Q mismatch
- Used with all invasive modes of ventilation

Tracheostomy
- Tracheostomy should be considered in patients who require ventilator support for extended periods of time
- Shown to improve patient comfort and give patients a better ability to participate in rehabilitation activities

- neuromuscular complications
  - muscle atrophy
  - increased intracranial pressure
- metabolic
  - decreased CO_2 due to hyperventilation
  - alkalemia with over correction of chronic hypercarbia

Ventilator Strategies

- mode and settings are determined based on patient factors (e.g. ideal body weight, compliance, resistance) and underlying reason for mechanical ventilation
- hypoxemic respiratory failure: ventilator provides supplemental oxygen, recruits atelectatic lung segments, helps improve V/Q mismatch, and decreases intrapulmonary shunt
- hypercapnic respiratory failure: ventilator augments alveolar ventilation; may decrease the work of breathing, allowing respiratory muscles to rest

Modes of Ventilation

- assist-control ventilation (ACV) or volume control (VC)
  - every breath is delivered with a pre-set tidal volume and rate or minute ventilation
  - extra controlled breaths may be triggered by patient effort; if no effort is detected within a specified amount of time the ventilator will initiate the breath
- pressure control ventilation (PCV)
  - a minimum frequency is set and patient may trigger additional breaths above the ventilator
  - all breaths delivered at a preset constant inspiratory pressure
- synchronous intermittent mandatory ventilation (SIMV)
  - ventilator provides controlled breaths (either at a set volume or pressure depending on whether in VC or PCV, respectively)
  - patient can breathe spontaneously (these breaths may be pressure supported) between controlled breaths
- pressure support ventilation (PSV)
  - patient initiates all breaths and the ventilator supports each breath with a pre-set inspiratory pressure
  - useful for weaning off ventilator
- high-frequency oscillatory ventilation (HFOV)
  - high breathing rate (up to 900 breaths/min in an adult), very low tidal volumes
  - used commonly in neonatal and pediatric respiratory failure
  - occasionally used in adults when conventional mechanical ventilation is failing
- non-invasive positive pressure ventilation (NPPV)
  - achieved without intubation by using a nasal or face mask
  - BiPAP: increased pressure (like PSV) on inspiration and lower constant pressure on expiration (i.e. PEEP)
  - CPAP: delivers constant pressure on both inspiration and expiration

Table 3. Causes of Abnormal End Tidal CO_2 Levels

Hypocapnea (Decreased CO_2)	Hypercapnea (Increased CO_2)
Hyperventilation	Hypoventilation
Hypothermia (decreased metabolic rate)	Hyperthermia and other hypermetabolic states
Decreased pulmonary blood flow (decreased cardiac output)	Improved pulmonary blood flow after resuscitation or hypotension
Technical issues Incorrect placement of sampling catheter Inadequate sampling volume	Technical issues Water in capnography device Anesthetic breathing circuit error Inadequate fresh gas flow Rebreathing Exhausted soda lime Faulty circuit absorber valves
V/Q mismatch Pulmonary thromboembolism Incipient pulmonary edema Air embolism	Low bicarbonate

Monitoring Ventilatory Therapy
- Pulse oximetry, end-tidal CO_2 concentration
- Regular arterial blood gases
- Assess tolerance regularly

Patients who develop a pneumothorax while on mechanical ventilation require a chest tube

Causes of Intraoperative Hypoxemia
Inadequate oxygen supply
e.g. breathing system disconnection, obstructed or malpositioned ETT, leaks in the anesthetic machine, loss of oxygen supply
Hypoventilation
Ventilation-perfusion inequalities e.g. atelectasis, pneumonia, pulmonary edema, pneumothorax
Reduction in oxygen carrying capacity
e.g. anemia, carbon monoxide poisoning, methemoglobinemia, hemoglobinopathy
Leftward shift of the hemoglobin-oxygen saturation curve
e.g. hypothermia, decreased 2,3-BPG, alkalosis, hypocarbia, carbon monoxide poisoning
Right-to-left cardiac shunt

A Comparison of Four Methods of Weaning Patients from Mechanical Ventilation
NEJM 1995;332:345-350
Study: Prospective, randomized, multicentre trial.
Participants: 130 of 546 patients who received mechanical ventilation and were considered ready for weaning but had respiratory distress during a 2 h trial of spontaneous breathing.
Intervention: One of four weaning techniques following standardized protocol.
Outcome: Median duration of weaning.
Results: The median duration of weaning for intermittent mandatory ventilation, pressure-support ventilation, intermittent (multiple) trials of spontaneous breathing, and once-daily trial of spontaneous breathing was 5 d, 4 d, and 3 d, respectively. The rate of successful weaning was higher with once-daily trial of spontaneous breathing than with intermittent mandatory ventilation (rate ratio 2.83; 95% CI 1.36-5.89; $p<0.006$) or pressure-support ventilation (ratio 2.05; 95% CI 1.04-4.04; $p<0.04$). There was no significant difference in the rate of success between once-daily trials and multiple trials of spontaneous breathing.
Conclusions: Once-daily or multiple trials of spontaneous breathing led to extubation more quickly than intermittent mandatory or pressure-support ventilation.

Intraoperative Management

Temperature

Causes of Hypothermia (<36.0°C)
- intraoperative temperature losses are common (e.g. 90% of intraoperative heat loss is transcutaneous), due to
  - OR environment (cold room, IV fluids, instruments)
  - open wound
- prevent with forced air warming blanket and warmed IV fluids

Causes of Hyperthermia (>37.5-38.3°C)
- drugs (e.g. atropine)
- blood transfusion reaction
- infection/sepsis
- medical disorder (e.g. thyrotoxicosis)
- malignant hyperthermia (see *Uncommon Complications*, A28)
- over-zealous warming efforts

Impact of Hypothermia (<36°C)
- Increased risk of wound infections due to impaired immune function
- Increases the period of hospitalization by delaying healing
- Reduces platelet function and impairs activation of coagulation cascade increasing blood loss and transfusion requirements
- Triples the incidence of VT and morbid cardiac events
- Decreases the metabolism of anesthetic agents prolonging post-operative recovery

Heart Rate

Cardiac Arrest
- pulseless arrest occurs due to 4 cardiac rhythms divided into shockable and non-shockable rhythms
  - shockable: ventricular fibrillation (VF) and ventricular tachycardia (VT)
  - non-shockable: asystole and pulseless electrical activity (PEA)
- for VF/VT, key to survival is good early CPR and defibrillation
- for asystole/PEA, key to survival is good early CPR and exclusion of all reversible causes
- reversible causes of PEA arrest (5 Hs and 5 Ts)
  - 5 Hs: **h**ypothermia, **h**ypovolemia, **h**ypoxia, **h**ydrogen ions (acidosis), **h**ypo/**h**yperkalemia
  - 5 Ts: **t**amponade (cardiac), **t**hrombosis (pulmonary), **t**hrombosis (coronary), **t**ension pneumo**t**horax, **t**oxins (overdose/poisoning)
  - when a patient sustains a cardiac arrest during anesthesia, it is important to remember that there are other causes on top the Hs and Ts to consider (i.e. local anesthetic systemic toxicity (LAST), excessive anesthetic dosing and others)
- for management of cardiac arrest, see *ACLS Guidelines* (Figure 16), A31

Intraoperative Tachycardia
- tachycardia = HR >150 bpm; divided into narrow complex supraventricular tachycardias (SVT) or wide complex tachycardias
- SVT: sinus tachycardia, atrial fibrillation/flutter, accessory pathway mediated tachycardia, paroxysmal atrial tachycardia
- wide complex tachycardia: VT, SVT with aberrant conduction
- causes of sinus tachycardia
  - shock/hypovolemia/blood loss
  - anxiety/pain/light anesthesia
  - full bladder
  - anemia
  - febrile illness/sepsis
  - drugs (e.g. atropine, cocaine, dopamine, epinephrine, ephedrine, isoflurane, isoproterenol, pancuronium) and withdrawal
  - Addisonian crisis, hypoglycemia, transfusion reaction, malignant hyperthermia
- for management of tachycardia, see *ACLS Guidelines* (Figure 17), A32

Intraoperative Bradycardia
- bradycardia = HR <50 bpm; most concerning are 2nd degree (Mobitz type II) and 3rd degree heart block, which can both degenerate into asystole
- causes of sinus bradycardia
  - increased parasympathetic tone vs. decreased sympathetic tone
  - must rule out hypoxemia
  - arrhythmias (see Cardiology and Cardiac Surgery, C16)
  - baroreceptor reflex due to increased ICP or increased BP
  - vagal reflex (oculocardiac reflex, carotid sinus reflex, airway manipulation)
  - drugs (e.g. SCh, opioids, edrophonium, neostigmine, halothane, digoxin, β-blockers)
  - high spinal/epidural anesthesia
- for management of bradycardia, see *ACLS Guidelines* (Figure 18), A32

Blood Pressure

Causes of Intraoperative Hypotension/Shock

- shock: condition characterized by inability of cardiovascular system to maintain adequate end-organ perfusion and delivery of oxygen to tissues

a) hypovolemic/hemorrhagic shock
  - most common form of shock, due to decrease in intravascular volume

b) obstructive shock
  - obstruction of blood into or out of the heart
  - increased JVP, distended neck veins, increased systemic vascular resistance, insufficient cardiac output (CO)
  - e.g. tension pneumothorax, cardiac tamponade, pulmonary embolism (and other emboli – i.e. fat, air)

c) cardiogenic shock
  - increased JVP, distended neck veins, increased systemic vascular resistance, decreased CO
  - e.g. myocardial dysfunction, dysrhythmias, ischemia/infarct, cardiomyopathy, acute valvular dysfunction

d) septic shock
  - see Infectious Diseases, ID21

e) spinal/neurogenic shock
  - decreased sympathetic tone
  - hypotension without tachycardia or peripheral vasoconstriction (warm skin)

f) anaphylactic shock
  - see Emergency Medicine, ER38

g) drugs
  - vasodilators, high spinal anesthetic interfering with sympathetic outflow

h) other
  - transfusion reaction, Addisonian crisis, thyrotoxicosis, hypothyroid, aortocaval syndrome
  - see Hematology, H52 and Endocrinology, E33

Intraoperative Shock

SHOCKED
Sepsis or **S**pinal shock
Hypovolemic/**H**emorrhagic
Obstructive
Cardiogenic
anaphylacti**K**
Extra/other
Drugs

BP = CO x SVR, where CO = SV x HR
SV is a function of preload, afterload, and contractility

Causes of Intraoperative Hypertension

- inadequate anesthesia causing pain and anxiety
- pre-existing HTN, coarctation, or preeclampsia
- hypoxemia/hypercarbia
- hypervolemia
- increased intracranial pressure
- full bladder
- drugs (e.g. ephedrine, epinephrine, cocaine, phenylephrine, ketamine) and withdrawal
- allergic/anaphylactic reaction
- hypermetabolic states: malignant hyperthermia, neuroleptic malignant syndrome, serotonin syndrome (see Psychiatry, PS44), thyroid storm, pheochromocytoma (see Endocrinology, E25)

Fluid Balance and Resuscitation

- total requirement = maintenance + deficit + ongoing loss
- in surgical settings this formula must take into account multiple factors including pre-operative fasting/decreased fluid intake, increased losses during or before surgery, fluid shifting during surgery, fluids given with blood products and medications

What is the Maintenance?

- average healthy adult requires approximately 2500 mL water/d
  - 200 mL/d GI losses
  - 800 mL/d insensible losses (respiration, perspiration)
  - 1500 mL/d urine (beware of renal failure)
- 4:2:1 rule to calculate maintenance requirements (applies to crystalloids only)
  - 4 mL/kg/h first 10 kg
  - 2 mL/kg/h second 10 kg
  - 1 mL/kg/h for remaining weight >20 kg
- increased requirements with fever, sweating, GI losses (vomiting, diarrhea, NG suction), adrenal insufficiency, hyperventilation, and polyuric renal disease
- decreased requirements with anuria/oliguria, SIADH, highly humidified atmospheres, and CHF
- maintenance electrolytes
  - Na^+: 3 mEq/kg/d
  - K^+: 1 mEq/kg/d
- 50 kg patient maintenance requirements
  - fluid = 40 + 20 + 30 = 90 mL/h = 2160 mL/d = 2.16 L/d
  - Na^+ = 150 mEq/d (therefore 150 mEq / 2.16 L/d ≈ 69 mEq/L)
  - K^+ = 50 mEq/d (therefore 50 mEq / 2.16 L/d ≈ 23 mEq/L)
- above patient's requirements roughly met with 2/3 D5W, 1/3 NS
  - 2/3 + 1/3 at 100 mL/h with 20 mEq KCl per litre

What is the Deficit?

- patients should be adequately hydrated prior to anesthesia
- total body water (TBW) = 60% or 50% of total body weight for an adult male or female, respectively (e.g. for a 70 kg adult male TBW = 70 x 0.6 = 42 L)
- total Na^+ content determines ECF volume; [Na^+] determines ICF volume
- hypovolemia due to volume contraction
  - extra-renal Na^+ loss
    - GI: vomiting, NG suction, drainage, fistulae, diarrhea
    - skin/resp: insensible losses (fever), sweating, burns
    - vascular: hemorrhage
  - renal Na^+ and H_2O loss
    - diuretics
    - osmotic diuresis
    - hypoaldosteronism
    - salt-wasting nephropathies
  - renal H_2O loss
    - diabetes insipidus (central or nephrogenic)
  - hypovolemia with normal or expanded ECF volume
    - decreased CO
    - redistribution
      - hypoalbuminemia: cirrhosis, nephrotic syndrome
      - capillary leakage: acute pancreatitis, rhabdomyolysis, ischemic bowel, sepsis, anaphylaxis
- replace water and electrolytes as determined by patient's needs
- with chronic hyponatremia, correction must be done gradually over >48 h to avoid central pontine myelinolysis

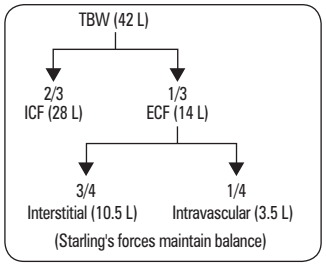

Figure 7. Total body water division in a 70 kg adult

Table 4. Signs and Symptoms of Dehydration

Percentage of Body Water Loss	Severity	Signs and Symptoms
3%	Mild	Decreased skin turgor, sunken eyes, dry mucous membranes, dry tongue, reduced sweating
6%	Moderate	Oliguria, orthostatic hypotension, tachycardia, low volume pulse, cool extremities, reduced filling of peripheral veins and CVP, hemoconcentration, apathy
9%	Severe	Profound oliguria or anuria and compromised CNS function with or without altered sensorium

What are the Ongoing Losses?

- losses from Foley catheter, NG, surgical drains
- third-spacing (other than ECF, ICF)
  - pleura, GI, retroperitoneal, peritoneal
  - evaporation via exposed viscera, burns
- blood loss
- ongoing loss due to surgical exposure and evaporative losses

IV Fluids

- replacement fluids include crystalloid and colloid solutions
- IV fluids improve perfusion but NOT O_2 carrying capacity of blood

Initial Distribution of IV Fluids

- H_2O follows ions/molecules to their respective compartments

Crystalloid Infusion

- salt-containing solutions that distribute only within ECF
- maintain euvolemia in patient with blood loss: 3 mL crystalloid infusion per 1 mL of blood loss for volume replacement (i.e. 3:1 replacement)
- if large volumes are to be given, use balanced fluids such as Ringer's lactate or Plasmalyte®, as too much normal saline (NS) may lead to hyperchloremic metabolic acidosis

Colloid Infusion (see *Blood Products*, A15)

- includes protein colloids (albumin and gelatin solutions) and non-protein colloids (dextrans and starches e.g. hydroxyethol starch [HES])
- distributes within intravascular volume
- 1:1 ratio (infusion:blood loss) only in terms of replacing intravascular volume
- HES colloids remain in intravascular space (metabolized by plasma serum amylase and renally excreted); two available in Canada: Voluven® and Pentaspan®
- the use of HES solutions is controversial because of recent RCTs and meta-analyses highlighting their renal (especially in septic patients) and coagulopathic side effects, as well as a lack of specific indications for their use
  - colloids are being used based on mechanistic and experimental evidence but there is a paucity of definitive studies investigating their safety and efficacy; routine use of colloids should be avoided

Colloids vs. Crystalloids for Fluid Resuscitation in Critically Ill Patients
Cochrane DB Syst Rev 2012;6:CD000567
Purpose: To evaluate the effects of colloids compared to crystalloids for fluid resuscitation, specifically when used in critically ill patients.
Methods: A meta-analysis was performed looking at randomized controlled trials comparing colloid vs. crystalloid use in patients requiring volume replacement. Pregnant women and neonates were excluded. Primary outcome was overall mortality.
Results: Results were broken down based on specific colloid. For albumin (or plasma protein fraction) the relative risk (RR) was 1.01 (95% CI 0.93-1.10) as compared to crystalloid. For hydroxyethyl starch the RR was 1.10 (95% CI 0.91-1.32). Modified gelatin had a RR of 0.91 (95% CI 0.49-1.72) and Dextran had a RR of 1.24 (95% CI 0.94-1.65). For colloids mixed in a hypertonic crystalloid as compared to isotonic crystalloid the RR was 0.88 (91% CI 0.71-1.06).
Conclusions: There is no evidence that use of colloids improves survival in trauma patients, burn patients, or post-operative patients when compared to crystalloid solutions. Given the increased cost of colloids as compared to crystalloids, it is recommended that crystalloids be the fluid of choice in these patients.

Table 5. IV Fluid Solutions

		ECF	Ringer's Lactate	0.9% NS	0.45% NS in D5W	D5W	2/3 D5W + 1/3 NS	Plasmalyte
mEq/L	Na^+	142	130	154	77	-	51	140
	K^+	4	4	-	-	-	-	5
	Ca^{2+}	4	3	-	-	-	-	-
	Mg^{2+}	3	-	-	-	-	-	3
	Cl^-	103	109	154	77	-	51	98
	HCO_3^-	27	28*	-	-	-	-	27
mOsm/L		280-310	273	308	154	252	269	294
pH		7.4	6.5	5.0	4.5	4.0	4.3	7.4

*Converted from lactate

Table 6. Colloid HES Solutions

	Concentration	Plasma Volume Expansion	Duration (h)	Maximum Daily Dose (mL/kg)
Voluven®	6%	1:1	4-6	33-50
Pentaspan®	10%	1:1.2-1.5	18-24	28

Calculating Acceptable Blood Losses (ABL)

- Blood volume
  term infant 80 mL/kg
  adult male 70 mL/kg
  adult female 60 mL/kg
- Calculate estimated blood volume (EBV) (e.g. in a 70 kg male, approx. 70 mL/kg)
  EBV = 70 kg x 70 mL/kg = 4900 mL
- Decide on a transfusion trigger, i.e. the Hb level at which you would begin transfusion, (e.g. 70 g/L for a person with Hb(i) = 150 g/L)

Hb(f) = 70 g/L

- Calculate

$$\text{ABL} = \frac{\text{Hb(Hi)} - \text{Hb(Hf)}}{\text{Hb(Hi)}} \times \text{EBV}$$
$$= \frac{150 - 70}{150} \times 4900$$
$$= 2613 \text{ mL}$$

- Therefore in order to keep the Hb level above 70 g/L, RBCs would have to be given after approximately 2.6 L of blood has been lost

Transfusion Infection Risks

Virus	Risk per 1 unit pRBCs
HIV	1 in 21 million
Hepatitis C virus	1 in 13 million
Hepatitis B virus	1 in 7.5 million
HTLV	1 in 1-1.3 million
Symptomatic Bacterial Sepsis	1 in 40,000 from platelets and 1 in 250,000 from RBC
West Nile virus	No cases since 2003

Source: Callum JL, Pinkerton PH. Bloody Easy. Fourth Edition ed. Toronto: Sunnybrook and Women's College Health Science Centre; 2016

Blood Products

- see Hematology, H52

Induction

Routine Induction vs. Rapid Sequence Induction

- routine induction is the standard in general anesthesia, however a RSI is indicated in patients at risk of regurgitation/aspiration (see *Aspiration*, A5)
- RSI uses pre-determined doses of induction drugs given in rapid succession to minimize the time patient is at risk for aspiration (i.e. from the time when they are asleep without an ETT until the time when the ETT is in and the cuff inflated)

Table 7. Comparison of Routine Induction vs. RSI

Steps	Routine Induction	RSI
1. Equipment Preparation	Check equipment, drugs, suction, and monitors; prepare an alternative laryngoscope blade and a second ETT tube one size smaller	Check equipment, drugs, suction, and monitors; prepare an alternative laryngoscope blade and a second ETT tube one size smaller; suction on
2. Pre-Oxygenation/ Denitrogenation	100% O_2 for 3 min or 4-8 vital capacity breaths	100% O_2 for 3 min or 4-8 vital capacity breaths
3. Pre-Treatment Agents	Use agent of choice to blunt physiologic responses to airway manipulation 3 min prior to laryngoscopy	Use agent of choice to blunt physiologic responses to airway manipulation; if possible, give 3 min prior to laryngoscopy, but can skip this step in an emergent situation
4. Induction Agents	Use IV or inhalation induction agent of choice	Use pre-determined dose of fast acting induction agent of choice
5. Muscle Relaxants	Muscle relaxant of choice given after the onset of the induction agent	Pre-determined dose of fast acting muscle relaxant (e.g. SCh) given IMMEDIATELY after induction agent
6. Ventilation	Bag-mask ventilation	DO NOT bag ventilate – can increase risk of aspiration
7. Cricoid Pressure	Backwards upwards rightwards pressure (BURP) on thyroid cartilage to assist visualization if indicated	Sellick maneuver, also known as cricoid pressure, to prevent regurgitation and assist in visualization (2 kg pressure with drowsiness, 3 kg with loss of consciousness)
8. Intubation	Intubate, inflate cuff, confirm ETT position	Intubate once paralyzed (~45 s after SCh given), inflate cuff, confirm ETT position; cricoid pressure maintained until ETT cuff inflated and placement confirmed
9. Secure Machines	Secure ETT, and begin manual/machine ventilation	Secure ETT, and begin manual/machine ventilation

Induction Agents

- induction in general anesthesia may be achieved with intravenous agents, volatile inhalation agents, or both

Solubility of Volatile Anesthetics in Blood
Least Soluble to Most Soluble
Nitrous oxide < desflurane < sevoflurane < isoflurane < halothane

Intravenous Agents

- see Table 8
- IV induction agents are non-opioid drugs used to provide hypnosis, amnesia and blunt reflexes
- these are initially used to draw the patient into the maintenance phase of general anesthesia rapidly, smoothly and with minimal adverse effects
  - examples include propofol, sodium thiopental (not available in North America), or ketamine
  - a continuous propofol infusion may also be used for the maintenance phase of GA

Table 8. Intravenous Induction Agents

	Propofol (Diprivan®)	Thiopental (sodium thiopental, sodium thiopentone)	Ketamine (Ketalar®, Ketaject®)	Benzodiazepines (midazolam [Versed®], diazepam [Valium®], lorazepam [Ativan®])	Etomidate
Class	Alkylphenol – hypnotic	Ultra-short acting thiobarbiturate – hypnotic	Phencyclidine (PCP) derivative – dissociative	Benzodiazepines – anxiolytic	Imadazole derivative - hypnotic
Action	Inhibitory at GABA synapse Decreased cerebral metabolic rate and blood flow, decreased ICP, decreased SVR, decreased BP, and decreased SV	Decreased time Cl– channels open, facilitating GABA and suppressing glutamic acid Decreased cerebral metabolism and blood flow, decreased CPP, decreased CO, decreased BP, decreased reflex tachycardia, decreased respiration	May act on NMDA, opiate, and other receptors Increased HR, increased BP, increased SVR, increased coronary flow, increased myocardial O_2 uptake CNS and respiratory depression, bronchial smooth muscle relaxation	Causes increased glycine inhibitory neurotransmitter, facilitates GABA Produces antianxiety and skeletal muscle relaxant effects Minimal cardiac depression	Decreases concentration of GABA required to activate receptor CNS depression Minimal cardiac or respiratory depression
Indications	Induction Maintenance Total intravenous anesthesia (TIVA)	Induction Control of convulsive states, obstetric patients	Major trauma, hypovolemia, obstetric bleeding, severe asthma because sympathomimetic	Used for sedation, amnesia, and anxiolysis	Induction Poor cardiac function, severe valve lesions, uncontrolled hypertension
Caution	Patients who cannot tolerate sudden decreased BP (e.g fixed cardiac output or shock)	Allergy to barbiturates Uncontrolled hypotension, shock, cardiac failure Porphyria, liver disease, status asthmaticus, myxedema	Ketamine allergy TCA medication (interaction causes HTN and dysrhythmias) History of psychosis Patients who cannot tolerate HTN (e.g. CHF, increased ICP, aneurysm)	Marked respiratory depression	Post-operative nausea and vomiting Venous irritation
Dosing	IV induction: 2.5-3.0 mg/kg (less with opioids) Unconscious <1 min Lasts 4-6 min $t_{1/2}$ = 55 min Decreased post-operative sedation, recovery time, N/V	IV induction: 3-5 mg/kg Unconscious about 30 s Lasts 5 min Accumulation with repeat dosing – not for maintenance $t_{1/2}$ = 5-10 h Post-operative sedation lasts hours	IV induction 1-2 mg/kg Dissociation in 15 s, analgesia, amnesia, and unconsciousness in 45-60 s Unconscious for 10-15 min, analgesia for 40 min, amnesia for 1-2 h $t_{1/2}$ = ~3 h	Onset less than 5 min if given IV Duration of action long but variable/somewhat unpredictable	IV induction 0.3 mg/kg Onset 30-60 seconds Lasts 4-8 minutes
Special Considerations	0-30% decreased BP due to vasodilation Reduce burning at IV site by mixing with lidocaine	Combining with rocuronium causes precipitates to form	High incidence of emergence reactions (vivid dreaming, out-of-body sensation, illusions) Pretreat with glycopyrrolate to decrease salivation	Antagonist: flumazenil (Anexate®) competitive inhibitor, 0.2 mg IV over 15 s, repeat with 0.1 mg/min (max of 2 mg), $t_{1/2}$ of 60 min Midazolam also has amnestic (antegrade) effect and decreased risk of thrombophlebitis	Adrenal suppression after first dose, cannot repeat dose or use as infusion Myoclonic movements during induction

Volatile Inhalational Agents

- examples include sevoflurane, desflurane, isoflurane, enflurane, halothane, and nitrous oxide
- see Table 9

Table 9. Volatile Inhalational Agents

	Sevoflurane	Desflurane	Isoflurane	Enflurane	Halothane	Nitrous oxide (N_2O)*
MAC (% gas in O_2)	2.0	6.0	1.2	1.7	0.8	104
CNS	Increased ICP	Increased ICP	Decreased cerebral metabolic rate Increased ICP	ECG seizure-like activity Increased ICP	Increased ICP and cerebral blood flow	—
Resp	Respiratory depression (severely decreased TV, increased RR), decreased response to respiratory CO_2 reflexes, bronchodilation					—
CVS	Less decrease of contractility, stable HR	Tachycardia with rapid increase in concentration	Decreased BP and CO, increased HR, theoretical chance of coronary steal**	Stable HR, decreased contractility	Decreased BP, CO, HR, and conduction Sensitizes myocardium to epinephrine-induced arrhythmias	Can cause decreased HR in pediatric patients with existing heart disease
MSK	Muscle relaxation, potentiation of other muscle relaxants, uterine relaxation					

*Properties and Adverse Effects of N_2O
Due to its high MAC, nitrous oxide is combined with other anesthetic gases to attain surgical anesthesia. A MAC of 104% is possible in a pressurized chamber only
Second Gas Effect
Expansion of closed spaces: closed spaces such as a pneumothorax, the middle ear, bowel lumen and ETT cuff will markedly enlarge if N_2O is administered
Diffusion hypoxia: during anesthesia, the washout of N_2O from body stores into alveoli can dilute the alveolar [O_2], creating a hypoxic mixture if the original [O_2] is low
**Coronary steal: isoflurane causes small vessel dilation which may compromise blood flow to areas of the heart with fixed perfusion (e.g. stents, atherosclerosis)

MAC (Minimum Alveolar Concentration)

- the alveolar concentration of a volatile anesthetic at one atmosphere (atm) of pressure that will prevent movement in 50% of patients in response to a surgical stimulus (e.g. abdominal incision)
- potency of inhalational agents is compared using MAC
- 1.2-1.3 times MAC will often ablate response to stimuli in the general population
- MAC values are roughly additive when mixing N_2O with another volatile agent; however, this only applies to movement, not other effects such as BP changes (e.g. 0.5 MAC of a potent agent + 0.5 MAC of N_2O = 1 MAC of potent agent)
- MAC-intubation: the MAC of anesthetic that will inhibit movement and coughing during endotracheal intubation, generally 1.3 MAC
- MAC-block adrenergic response (MAC-BAR): the MAC necessary to blunt the sympathetic response to noxious stimuli, generally 1.5 MAC
- MAC-awake: the MAC of a given volatile anesthetic at which a patient will open their eyes to command, generally 0.3-0.4 of the usual MAC

Muscle Relaxants and Reversing Agents

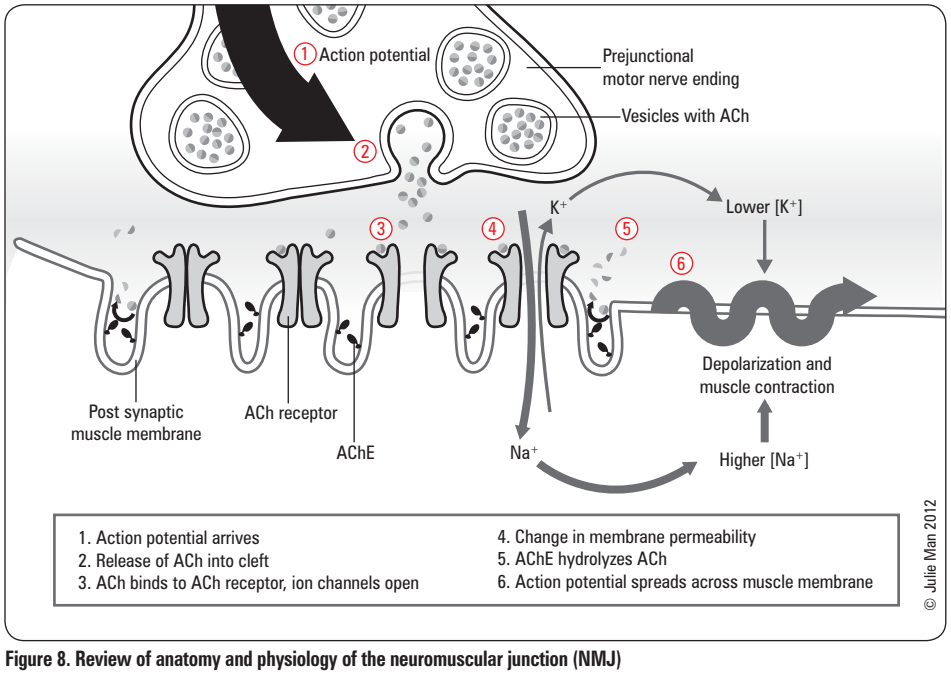

Figure 8. Review of anatomy and physiology of the neuromuscular junction (NMJ)

Muscle Relaxants

- two types of muscle relaxants
  1. depolarizing muscle relaxants: succinylcholine (SCh)
  2. non-depolarizing muscle relaxants: rocuronium, mivacurium, vecuronium, cistracurium, pancuronium
- block nicotinic cholinergic receptors in NMJ
- provides skeletal muscle paralysis, including the diaphragm, but spares involuntary muscles such as the heart and smooth muscle
- never use muscle relaxants without adequate preparation and equipment to maintain airway and ventilation
- muscle relaxation produces the following desired effects
  1. facilitates intubation
  2. assists with mechanical ventilation
  3. prevents muscle stretch reflex and decreases muscle tone
  4. allows access to the surgical field (intracavitary surgery)
- nerve stimulator (i.e. train of four) is used intraoperatively to assess the degree of nerve block; no twitch response seen with complete neuromuscular blockade
- see Tables 10 and 11, for more details including mechanism of action

Plasma Cholinesterase
Plasma cholinesterase is produced by the liver and metabolizes SCh, ester local anesthetics, and mivacurium. A prolonged duration of blockade by SCh occurs with:
(a) decreased quantity of plasma cholinesterase, e.g. liver disease, pregnancy, malignancy, malnutrition, collagen vascular disease, hypothyroidism
(b) abnormal quality of plasma cholinesterase, e.g. normal levels but impaired activity of enzymes, genetically inherited

Table 10. Depolarizing Muscle Relaxants (Non-Competitive): Succinylcholine (SCh)

Mechanism of Action	Mimics ACh and binds to ACh receptors causing prolonged depolarization; initial fasciculation may be seen, followed by temporary paralysis secondary to blocked ACh receptors by SCh
Intubating Dose (mg/kg)	1-1.5
Onset	30-60 s – rapid (fastest of all muscle relaxants)
Duration	3-5 min – short (no reversing agent for SCh)
Metabolism	SCh is hydrolyzed by plasma cholinesterase (pseudocholinesterase), found only in plasma and not at the NMJ
Indications	Assist intubation Increased risk of aspiration (need rapid paralysis and airway control (e.g. full stomach), hiatus hernia, obesity, pregnancy, trauma) Short procedures Electroconvulsive therapy (ECT) Laryngospasm
Side Effects	1. SCh also stimulates muscarinic cholinergic autonomic receptors (in addition to nicotinic receptors; may cause bradycardia, dysrhythmias, sinus arrest, increased secretions of salivary glands (especially in children) 2. Hyperkalemia Disruption of motor nerve activity causes proliferation of extrajunctional (outside NMJ) cholinergic receptors Depolarization of an increased number of receptors by SCh may lead to massive release of potassium out of muscle cells Patients at risk 3rd degree burns 24 h-6 mo after injury Traumatic paralysis or neuromuscular diseases (e.g. muscular dystrophy) Severe intra-abdominal infections Severe closed head injury Upper motor neuron lesions 3. Can trigger MH (see Malignant Hyperthermia, A28) 4. Increased ICP/intraocular pressure/intragastric pressure (no increased risk of aspiration if competent lower esophageal sphincter) 5. Fasciculations, post-operative myalgia – may be minimized if small dose of non-depolarizing agent given before SCh administration
Contraindications	
Absolute	Known hypersensitivity or allergy, positive history of malignant hyperthermia, myotonia (m. congenita, m. dystrophica, paramyotonia congenital), high risk for hyperkalemic response
Relative	Known history of plasma cholinesterase deficiency, myasthenia gravis, myasthenic syndrome, familial periodic paralysis, open eye injury

Table 11. Non-Depolarizing Muscle Relaxants (Competitive)

Mechanism of Action	Competitive blockade of postsynaptic ACh receptors preventing depolarization					
Classification	**Short**		**Intermediate**		**Long**	
	Mivacuronium	Rocuronium	Vecuronium	Cisatracurium	Pancuronium	
Intubating Dose (mg/kg)	0.2	0.6-1.0	0.1	0.2	0.1	
Onset (min)	2-3	1.5	2-3	3	3-5	
Duration (min)	15-25	30-45	45-60	40-60	90-120	
Metabolism	Plasma cholinesterase	Liver (major) Renal (minor)	Liver	Hofmann Eliminations	Renal (major)	Liver (minor)
Indications	Assist intubation, assist mechanical ventilation in some ICU patients, reduce fasciculations and post-operative myalgias secondary to SCh					
Side Effects						
Histamine Release	Yes	No	No	No	No	
Other	—	—	—	—	Tachycardia	
Considerations	Increased duration of action in renal or liver failure	Quick onset of rocuronium allows its use in rapid sequence induction Cisatracurium is good for patients with renal or hepatic insufficiency			Pancuronium if increased HR and BP desired	

Reversing Agents

- neostigmine, pyridostigmine, edrophonium
- reversal agents are acetylcholinesterase inhibitors
  - inhibits enzymatic degradation of ACh; increases amount of ACh at nicotinic and muscarinic receptors, displacing non-depolarizing muscle relaxant
  - administer reversal agents when there has been some recovery of blockade (i.e. muscle twitch)
  - can only reverse the effect of non-depolarizing muscle relaxants
- anticholinergic agents (e.g. atropine, glycopyrrolate) are simultaneously administered to minimize muscarinic effect of reversal agents (i.e. bradycardia, salivation and increased bowel peristalsis)

Table 12. Reversal Agents for Non-Depolarizing Relaxants

Cholinesterase Inhibitor	Neostigmine	Pyridostigmine	Edrophonium
Onset and Duration	Intermediate	Longest	Shortest
Mechanism of Action	Inhibits enzymatic degradation of ACh, increases ACh at nicotinic and muscarinic receptors, displaces non-depolarizing muscle relaxants Muscarinic effects of reversing agents include unwanted bradycardia, salivation, and increased bowel peristalsis*		
Dose	0.04-0.08 mg/kg	0.1-0.4 mg/kg	0.5-1 mg/kg
Recommended Anticholinergic	Glycopyrrolate	Glycopyrrolate	Atropine
Dose of Anticholinergic (per mg)	0.2 mg	0.05 mg	0.014 mg

*Atropine and glycopyrrolate are anticholinergic agents administered during the administration of reversal agents to minimize muscarinic effects

Analgesia

- options include opioids (e.g. morphine, fentanyl, hydromorphone), NSAIDS, acetaminophen, ketamine, gabapentin, local, and regional anesthetic (see Table 15, A25)

Maintenance

- general anesthesia is maintained using volatile inhalation agents and/or IV agents (i.e. propofol infusion)
- analgesia (usually IV opioids) and muscle relaxants are also given as needed

Extubation

- criteria: patient must no longer have intubation requirements (see Table 2, A8)
  - patency: airway must be patent
  - protection: airway reflexes intact
  - patient must be oxygenating and ventilating spontaneously
- general guidelines
  - ensure patient has normal neuromuscular function (peripheral nerve stimulator monitoring) and hemodynamic status
  - ensure patient is breathing spontaneously with adequate rate and tidal volume
  - allow ventilation (spontaneous or controlled) with 100% O_2 for 3-5 min
  - suction secretions from pharynx, deflate cuff, remove ETT on inspiration (vocal cords abducted)
  - ensure patient is breathing adequately after extubation
  - ensure face mask for O_2 delivery available
  - proper positioning of patient during transfer to recovery room (supine, head elevated)

Complications of Extubation

- early extubation: aspiration, laryngospasm
- late extubation: transient vocal cord incompetence, edema (glottic, subglottic), pharyngitis, tracheitis

Laryngospasm

- defined as forceful involuntary spasm of laryngeal muscles caused by stimulation of superior laryngeal nerve (by oropharyngeal secretions, blood, extubation)
- causes partial or total airway obstruction
- more likely to occur in semi-conscious patients
- prevention: extubate while patient is still deeply under anesthesia or fully awake
- treatment: apply sustained positive pressure with bag-mask ventilation with 100% oxygen, low-dose propofol (0.5-1.0 mg/kg) optional, low-dose succinylcholine (approximately 0.25 mg/kg) and reintubation if hypoxia develops

Regional Anesthesia

- local anesthetic agent (LA) 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 purpose of reducing or preventing impulse transmission
- no CNS depression (unless overdose of local anesthetic); patient remains conscious
- regional anesthetic techniques categorized as follows:
  - epidural and spinal anesthesia (neuraxial anesthesia)
  - peripheral nerve blocks
  - IV regional anesthesia (e.g. Bier block)

Patient Preparation

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

Benefits of Regional Anesthesia
- Reduced perioperative pulmonary complications
- Reduced perioperative analgesia requirements
- Decreased PONV
- Reduced perioperative blood loss
- Ability to monitor CNS status during procedure
- Improved perfusion
- Lower incidence of VTE
- Shorter recovery and improved rehabilitation
- Pain blockade with preserved motor function

Epidural and Spinal Anesthesia

- most useful for surgeries performed below level of umbilicus

Anatomy of Spinal/Epidural Area

- spinal cord extends to L2, dural sac to S2 in adults
- 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 (outside to inside)
  - skin
  - subcutaneous fat
  - supraspinous ligament
  - interspinous ligament
  - ligamentum flavum (last layer before epidural space)
  - dura + arachnoid for spinal anesthesia

Landmarking Epidural/Spinal Anesthesia
Spinous processes should be maximally flexed
L4 spinous processes found between iliac crests
Common sites of insertion are L3-L4 and L4-L5

Classic Presentation of Dural Puncture Headache
- Onset 6 h-3 d after dural puncture
- Postural component (worse when sitting)
- Occipital or frontal localization
- ± tinnitus, diplopia

Table 13. Epidural vs. Spinal Anesthesia

	Epidural	Spinal
Deposition Site	LA injected in epidural space (space between ligamentum flavum and dura) 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	LA injected into subarachnoid space in the dural sac surrounding the spinal cord and nerve roots
Onset	Significant blockade requires 10-15 min Slower onset of side effects	Rapid blockade (onset in 2-5 min)
Effectiveness	Effectiveness of blockade can be variable	Very effective blockade
Difficulty	Technically more difficult; greater failure rate	Easier to perform due to visual confirmation of CSF flow
Patient Positioning	Position of patient not as important; specific gravity not an issue	Hyperbaric LA solution – position of patient important
Specific Gravity/Spread	Epidural injections spread throughout the potential space; specific gravity of solution does not affect spread	LA solution may be made hyperbaric (of greater specific gravity than the cerebrospinal fluid by mixing with 10% dextrose, thus increasing spread of LA to the dependent (low) areas of the subarachnoid space)
Dosage	Larger volume/dose of LA (usually > toxic IV dose)	Smaller dose of LA required (usually < toxic IV dose)
Continuous Infusion	Use of catheter allows for continuous infusion or repeat injections	None
Complications	Failure of technique Hypotension Bradycardia if cardiac sympathetics blocked (only if ~T2-4 block), e.g. "high spinal" Epidural or subarachnoid hematoma Accidental subarachnoid injection can produce spinal anesthesia (and any of the above complications) Systemic toxicity of LA (accidental intravenous) Catheter complications (shearing, kinking, vascular or subarachnoid placement) Infection Dural puncture	Failure of technique Hypotension Bradycardia if cardiac sympathetics blocked (only if ~T2-4 block), e.g. "high spinal" Epidural or subarachnoid hematoma Post-spinal headache (CSF leak) Transient paresthesias Spinal cord trauma, infection
Combined Spinal-Epidural	Combines the benefits of rapid, reliable, intense blockade of spinal anesthesia together with the flexibility of an epidural catheter	

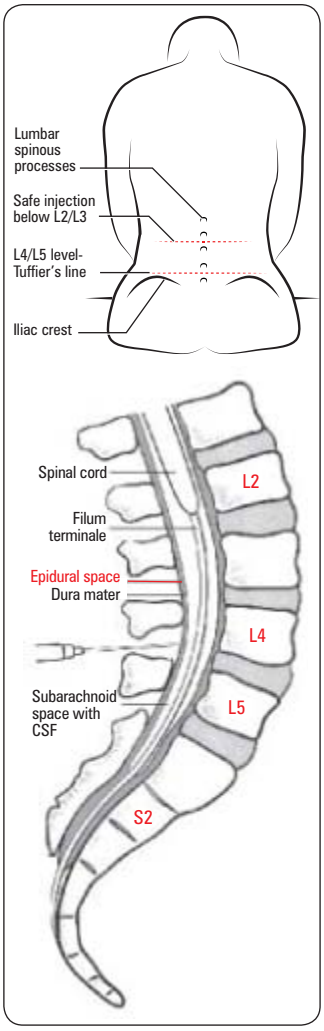

Figure 9. Landmarks for placement of epidural/spinal

Contraindications to Spinal/Epidural Anesthesia

- absolute contraindications
  - lack of resuscitative drugs/equipment
  - patient refusal
  - allergy to local anesthetic
  - infection at puncture site or underlying tissues
  - coagulopathies/bleeding diathesis
  - raised ICP
  - sepsis/bacteremia
  - severe hypovolemia
  - cardiac lesion with fixed output states (severe mitral/aortic stenosis)
  - lack of IV access
- relative contraindications
  - pre-existing neurological disease (demyelinating lesions)
  - previous spinal surgery, severe spinal deformity
  - prolonged surgery
  - major blood loss or maneuvers that can compromise reaction

Peripheral Nerve Blocks

- deposition of LA around the target nerve or plexus
- ultrasound guidance and peripheral nerve stimulation (needle will stimulate target nerve/plexus) may be used to guide needle to target nerve while avoiding neural trauma or intraneural injection
- most major nerves or nerve plexi can be targeted (brachial plexus block, femoral nerve block, sciatic nerve block, etc.)
- performed with standard monitors
- approximately 2-4 per 10,000 risk of late neurologic injury
- resuscitation equipment must be available

Contraindications to Peripheral Nerve Blockade

- absolute contraindications
  - allergy to LA
  - patient refusal
- relative contraindications
  - certain types of pre-existing neurological dysfunction (e.g. ALS, MS, diabetic neuropathy)
  - local infection at block site
  - bleeding disorder

Reduction of Post-Operative Mortality and Morbidity with Epidural or Spinal Anaesthesia: Results from Overview of Randomized Trials
BMJ 2000;321:1-12
Purpose: To obtain reliable estimates of the effects of neuraxial blockade with epidural or spinal anesthesia on post-operative morbidity and mortality after various surgeries with or without general anesthesia.
Study: Systematic review of all trials with randomization to intraoperative neuraxial blockade vs. control group.
Patients: 141 trials including 9,559 patients.
Main Outcomes: All cause mortality, MI, PE, DVT, transfusion requirements, pneumonia, other infections, respiratory depression, and renal failure
Results: With neuraxial blockade, overall mortality was reduced by about one third. Neuraxial blockade reduced the risk of PE by 55%, DVT by 44%, transfusion requirements by 50%, pneumonia by 39%, and respiratory depression by 59%. There were also reductions in MI and renal failure.These mortality reductions are irrespective of surgical group, type of blockade (epidural or spinal), or whether neuraxial blocker was combined with general anesthetic.
Conclusions: Neuraxial blockade reduces post-operative mortality and other serious complications.

Local Anesthesia

Local Anesthetic Agents

- see Table 14, for list of LA agents

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 bind to receptors on the cytosolic side of the Na^+ channel, inhibiting Na^+ flux and thus blocking impulse conduction
- different types of nerve fibres undergo blockade at different rates

Absorption, Distribution, Metabolism

- LA readily crosses the blood-brain barrier (BBB) once absorbed into the bloodstream
- ester-type LA (procaine, tetracaine) are broken down by plasma and hepatic esterases; metabolites excreted via kidneys
- amide-type LA (lidocaine, bupivicaine) are broken down by hepatic mixed-function oxidases (P450 system); metabolites excreted via kidneys

Selection of LA

- choice of LA depends on
  - onset of action: influenced by pKa (the 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 (longer duration of action when protein binding of LA is strong)
  - potency: influenced by lipid solubility (agents with high lipid solubility penetrate the nerve membrane more easily)
  - unique needs (e.g. sensory blockade with relative preservation of motor function by bupivicaine at low doses)
  - potential for toxicity

Table 14. Local Anesthetic Agents

	Maximum Dose	Maximum Dose with Epinephrine	Potency	Duration
chloroprocaine	11 mg/kg	14 mg/kg	Low	15-30 min
lidocaine	5 mg/kg	7 mg/kg	Medium	1-2 h
bupivacaine	2.5 mg/kg	3 mg/kg	High	3-8 h
ropivacaine	2.5 mg/kg	3 mg/kg	High	2-8 h

Systemic Toxicity

- see Table 16, A25 for maximum doses, potency, and duration of action for common LA agents
- occurs by accidental intravascular injection, LA overdose, or unexpectedly rapid absorption

CNS Effects
- CNS effects first appear to be excitatory due to initial block of inhibitory fibres, then subsequent block of excitatory fibres
- effects in order of appearance
  - numbness of tongue, perioral tingling, metallic taste
  - disorientation, drowsiness
  - tinnitus
  - visual disturbances
  - muscle twitching, tremors
  - unconsciousness
  - convulsions, seizures
  - generalized CNS depression, coma, respiratory arrest

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, get help
- 100% O_2, manage ABCs
- diazepam or sodium thiopental may be used to increase seizure threshold
- manage arrhythmias (see *ACLS Guidelines*, A31-32)
- Intralipid® 20% to bind local anesthetic in circulation

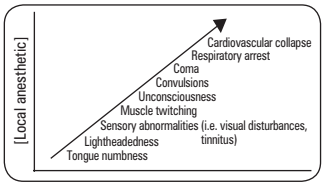

Figure 10. Local anesthetic systemic toxicity

Local Infiltration and Hematoma Blocks

Local Infiltration
- injection of tissue with LA, producing a lack of sensation in the infiltrated area due to LA acting on nerves
- suitable for small incisions, suturing, excising small lesions
- can use fairly large volumes of dilute LA to infiltrate a large area
- 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

Where Not to Use LA with Epinephrine
"Ears, Fingers, Toes, Penis, Nose"

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

Topical Anesthetics

- various preparations of local anesthetics available for topical use, may be a mixture of agents (EMLA cream is a combination of 2.5% lidocaine and prilocaine)
- must be able to penetrate the skin or mucous membrane

Post-Operative Care

- pain management should be continuous from OR to post-anesthetic care unit (PACU) to hospital ward and home

Common Post-Operative Anesthetic Complications

Nausea and Vomiting
- hypotension and bradycardia must be ruled out
- pain and surgical manipulation also cause nausea
- often treated with dimenhydrinate (Gravol®), metoclopramide (Maxeran®; not with bowel obstruction), prochlorperazine (Stemetil®), ondansetron (Zofran®), granisetron (Kytril®)

Confusion and Agitation
- ABCs first – confusion or agitation can be caused by airway obstruction, hypercapnea, hypoxemia
- neurologic status (Glasgow Coma Scale, pupils), residual paralysis from anesthetic
- pain, distended bowel/bladder
- fear/anxiety/separation from caregivers, language barriers
- metabolic disturbance (hypoglycemia, hypercalcemia, hyponatremia – especially post-TURP)
- intracranial cause (stroke, raised intracranial pressure)
- drug effect (ketamine, anticholinergics, serotonin)
- elderly patients are more susceptible to post-operative delirium

Respiratory Complications
- susceptible to aspiration of gastric contents due to PONV and unreliable airway reflexes
- airway obstruction (secondary to reduced muscle tone from residual anesthetic, soft tissue trauma and edema, or pooled secretions) may lead to inadequate ventilation, hypoxemia, and hypercapnia
- airway obstruction can often be relieved with head tilt, jaw elevation, and anterior displacement of the mandible. If the obstruction is not reversible, a nasal or oral airway may be used

Hypotension
- must be identified and treated quickly to prevent inadequate perfusion and ischemic damage
- reduced cardiac output (hypovolemia, most common cause) and/or peripheral vasodilation (residual anesthetic agent)
- first step in treatment is usually the administration of fluids ± inotropic agents

Hypertension
- pain, hypercapnia, hypoxemia, increased intravascular fluid volume, and sympathomimetic drugs can cause hypertension
- sodium nitroprusside or β-blocking drugs (e.g. esmolol and metoprolol) can be used to treat hypertension

Pain Management

Definitions
- pain: perception of nociception, which occurs in the brain
- nociception: detection, transduction, and transmission of noxious stimuli

Pain Classifications
- temporal: acute vs. chronic
- mechanism: nociceptive vs. neuropathic

Acute Pain
- pain of short duration (<6 wk) usually associated with surgery, trauma, or acute illness; often associated with inflammation
- usually limited to the area of damage/trauma and resolves with healing

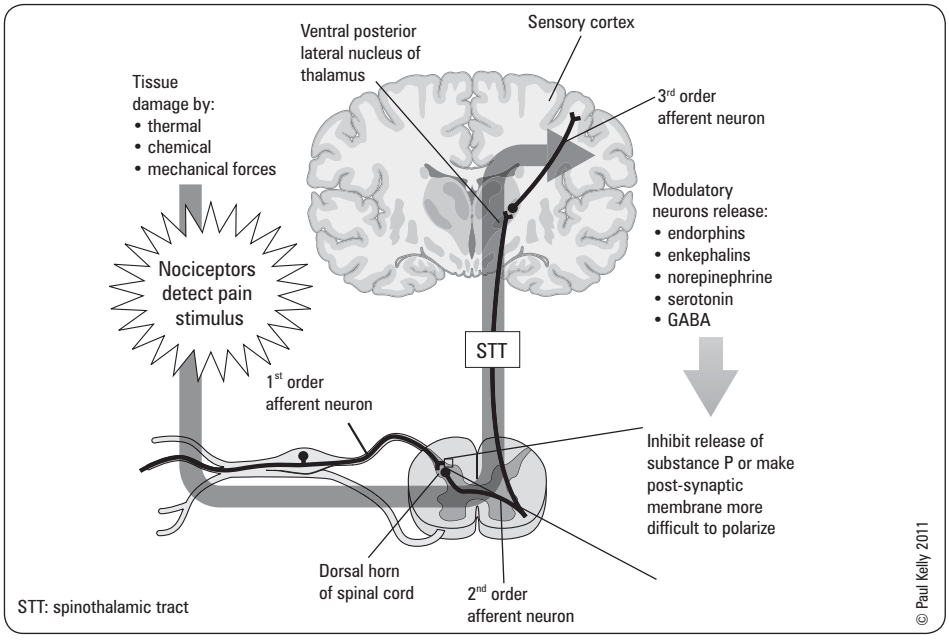

Figure 11. Acute pain mechanism

Risk Factors for Post-Operative Nausea and Vomiting (PONV)
- Young age
- Female
- History of PONV
- Non-smoker
- Type of surgery: ophtho, ENT, abdo/pelvic, plastics
- Type of anesthetic: N_2O, opioids, volatile agents

Drugs for Preventing Post-Operative Nausea and Vomiting

Cochrane DB Syst Rev 2006;3:CD004125

Purpose: To evaluate the efficacy of antiemetics in preventing PONV.

Methods: A meta-analysis was performed looking at randomized controlled trials comparing an antiemetic to either a second antiemetic or placebo. Trials looking at dosing and/or timing of medication administration were also included. PONV was used as the primary outcome.

Results: 737 studies involving 103,237 patients. Eight drugs significantly reduced the occurence of PONV, namely: droperidol, metoclopramide, ondansetron, tropisetron, dolasetron, dexamethasone, cyclizine, and granisetron. Relative risk (RR) versus placebo varied between 0.60, and 0.80. Side effects included a significant increase in drowsiness for droperidol (RR 1.32) and headache for ondansetron (RR 1.16). The cumulative number needed to treat was 3.57.

Conclusion: Antiemetic medication is effective for reducing the occurrence of PONV. However, further investigation needs to be done to determine whether antiemetics can cause more severe (and likely rare) side effects, which could alter how liberally they are used.

Opioid Conversion

	Parenteral (IV)	Equivalent Oral Dose
Morphine	10 mg	30 mg
Hydromorphone	2 mg	4 mg
Codeine	120 mg	200 mg
Oxycodone	N/A	20 mg
Fentanyl IV	100 μg	N/A

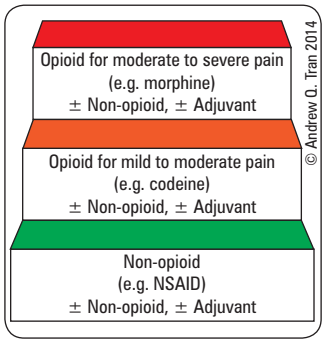

Figure 12. WHO analgesia ladder

Pharmacological Management of Acute Pain

- ask the patient to rate the pain out of 10, or use visual analog scale, to determine severity
- pharmacological treatment guided by WHO analgesia ladder
- patient controlled analgesia (PCA)
  - involves the use of computerized pumps that can deliver a constant infusion and bolus breakthrough doses of parenterally-administered opioid analgesics
  - limited by lockout intervals
  - most commonly used agents: morphine and hydromorphone
  - see Table 17, A26 for suggested infusion rate, PCA dose and lockout intervals

Table 15. Commonly Used Analgesics

	Acetaminophen	NSAIDs	Opioids
Examples	Tylenol®	Aspirin®, ibuprofen, naproxen ketorolac (IV)	Oral: codeine, oxycodone, morphine, hydromorphone Parenteral: morphine, hydromorphone, fentanyl
Indications	First-line for mild acute pain	Mild-moderate pain	Oral: moderate acute pain Parenteral: moderate-severe acute pain
Mechanism of Action	Unclear, hypothesized cyclooxygenase-2 (COX-2) inhibition Unclear, hypothesized modulation of endogenous cannabinoid system	Non-selective COX-1 and 2 inhibition reducing proinflammatory prostaglandin synthesis	Dampens nociceptive transmission between 1st and 2nd order neurons in the dorsal horn Activates ascending modulatory pathways resulting in release of inhibitory neurotransmitters Inhibits peripheral inflammatory response and hyperalgesia Affects mood and anxiety – alleviates the affective component of perceived pain
Dosing/ Administration	Limited by analgesic ceiling beyond which there is no additional analgesia Opioid-sparing Max dose of 4 g/24 h	Limited by analgesic ceiling beyond which there is no additional analgesia Opioid-sparing Significant inter-individual variation in efficacy	No analgesic ceiling (except for codeine) Can be administered intrathecally (spinal block) or by continuous infusion
Side Effects/ Toxicity	Considered relatively safe Liver toxicity in elevated doses	Gastric ulceration/bleeding Decreased renal perfusion Photosensitivity Premature closure of the ductus arteriosus in pregnancy	Respiratory depression Constipation and abdominal pain Sedation N/V Pruritus Confusion (particularly in the elderly) Dependence

Table 16. Opioids

Agent	Relative Dose to 10 mg Morphine IV	Moderate Dose	Onset	Duration	Special Considerations
Codeine	200 mg PO	15-30 mg PO	Late (30-60 min)	Moderate (4-6 h)	Primarily post-operative use, not for IV use
Meperidine (Demerol®)	75 mg IV	2-3 mg/kg IV	Moderate (10 min)	Moderate (2-4 h)	Anticholinergic, hallucinations, less pupillary constriction than morphine, metabolite build up may cause seizures
Morphine	10 mg IV 20 mg PO	0.2-0.3 mg/kg IV 0.4-0.6 mg/kg PO	Moderate (5-10 min)	Moderate (4-5 h)	Histamine release leading to decrease in BP
Oxycodone Controlled Release (Oxyneo®)	15 mg PO	10-20 mg PO (no IV)	Late (30-45 min)	Long (8-12 h)	Do not split, crush, or chew tablet
Oxycodone Regular Tablet (Oxy IR®)	15 mg PO (no IV)	5-15 mg PO	Moderate (15 min)	Moderate (3-6 h)	Percocet® = oxycodone 5 mg + acetaminophen 325 mg
Hydromorphone (Dilaudid®)	2 mg IV 10 mg PO	40-60 μg/kg IV 2-4 mg PO	Moderate (15 min)	Moderate (4-5 h)	
Fentanyl	100 μg IV	2-3 μg/kg IV	Rapid (<5 min)	Short (0.5-1 h)	Transient muscle rigidity in very high doses
Remifentanil	100 μg IV	0.5-1.5 μg/kg IV	Rapid (1-3 min)	Ultra short (<10 min)	Only use during induction and maintenance of anesthesia

In general, parenteral route is 2-3x more potent than oral

Cautionary Use of NSAIDs in Patients with
- Asthma
- Coagulopathy
- GI ulcers
- Renal insufficiency
- Pregnancy, 3rd trimester

Common Side Effects of Opioids
- N/V
- Constipation
- Sedation
- Pruritus
- Abdominal pain
- Urinary retention
- Respiratory depression

When prescribing opioids, consider:
- Breakthrough dose
- Anti-emetics
- Laxative

PCA Parameters
- Loading dose
- Bolus dose
- Lockout interval
- Continuous infusion (optional)
- Maximum 4 h dose (limit)

Advantages of PCA
- Improved patient satisfaction
- Fewer side effects
- Accommodates patient variability
- Accommodates changes in opioid requirements

Patient Controlled Opioid Analgesia vs. Conventional Opioid Analgesia for Post-Operative Pain

Cochrane DB Syst Rev 2006;4:CD003348

Purpose: To evaluate the efficacy of patient controlled analgesia (PCA) as compared to conventional 'as-needed' analgesia administration providing pain relief in post-operative patients.

Methods: Meta-analyses of randomized controlled trials comparing PCA vs. conventional administration of opioid analgesia. Assessment employed a visual analog scale (VAS) for pain intensity along with overall analgesic consumption, patient satisfaction, length of stay, and adverse side effects.

Results: 55 studies with a total of 2,023 patients receiving PCA and 1,838 patients with standard as-needed opioid administration. PCA provided significantly better pain control through 72 h post-operatively, but patients consumed significantly more opioids (>7 mg morphine/24 h, $p<0.05$) Significantly more patients reported pruritus in the PCA group compared to control with a number needed to harm of 13. No significant difference in overall length of stay in hospital, sedation level, N/V, or urinary retention.

Conclusions: PCA is more effective than standard as-needed administration for reducing post-operative pain. However, patients using PCA consume more opioids overall and have more pruritus.

Table 17. Opioid PCA Doses

Agent	PCA Dose	PCA Lockout Interval	PCA 4 h Maximum
Morphine	1 mg	5 min	30 mg
Hydromorphone	0.2 mg	5 min	6 mg
Fentanyl	25-50 μg	5 min	400 μg

Opioid Antagonists (naloxone, naltrexone)

- indication: opioid overdose (manifests primarily at CNS, e.g. respiratory depression)
- mechanism of action: competitively inhibit opioid receptors, predominantly μ receptors
  - naloxone is short-acting ($t_{1/2}$ = 1 h); effects of narcotic may return when naloxone wears off; therefore, the patient must be observed closely following its administration
  - naltrexone is longer-acting ($t_{1/2}$ = 10 h); less likely to see return of opioid effects
- side effects: relative overdose of naloxone may cause nausea, agitation, sweating, tachycardia, hypertension, re-emergence of pain, pulmonary edema, seizures (essentially opioid withdrawal)

Neuropathic Pain

- see Neurology, N41

Chronic Pain

- chronic pain: greater than 3 mo, or recurrent pain that occurs at least 3 times throughout three month period
- pain of duration or intensity that persists beyond normal tissue healing and adversely affects functioning
- may have nociceptive and neuropathic components; dysregulation of analgesic pathways implicated
- in the perioperative period, consider continuing regular long-acting analgesics and augmenting with regional techniques, adjuvants, additional opioid analgesia and non-pharmacological techniques

Obstetrical Anesthesia

Anesthesia Considerations in Pregnancy

- airway
  - possible difficult airway as tissues becomes edematous and friable especially in labour
- respiratory
  - decreased FRC and increased O_2 consumption cause more rapid desaturation during apnea
- cardiovascular system
  - increased blood volume > increased RBC mass results in mild anemia
  - decreased SVR proportionately greater than increased CO results in decreased BP
  - prone to decreased BP due to aortocaval compression – therefore for surgery, a pregnant patient is positioned in left uterine displacement using a wedge under her right flank
- central nervous system
  - decreased MAC due to hormonal effects
  - increased block height due to engorged epidural veins
- gastrointestinal system
  - delayed gastric emptying
  - increased volume and acidity of gastric fluid
  - decreased LES tone
  - increased abdominal pressure
  - combined, these lead to an increased risk of aspiration – therefore for surgery, a pregnant patient is given sodium citrate 30 cc PO immediately before surgery to neutralize gastric acidity

Options for Analgesia during Labour

- psychoprophylaxis – Lamaze method
  - patterns of breathing and focused attention on fixed object
- systemic medication
  - easy to administer, but risk of maternal or neonatal respiratory depression
  - opioids most commonly used if delivery is not expected within 4 h
- inhalational analgesia
  - easy to administer, makes uterine contractions more tolerable, but does not relieve pain completely
  - 50% nitrous oxide
- neuraxial anesthesia
  - provides excellent analgesia with minimal depressant effects
  - hypotension is the most common complication
  - maternal BP monitored q2-5 min for 15-20 min after initiation and regularly thereafter
  - epidural usually given as it preferentially blocks sensation, leaving motor function intact

The Effect of Epidural Analgesia on Labour, Maternal, and Neonatal Outcomes: A Systematic Review
Am J Obstet Gynecol 2002;186:S69-77
Study: Meta-analysis of 14 studies with 4,324 women.
Selection Criteria: RCTs and prospective cohort studies between 1980-2001 comparing epidural analgesia to parenteral opioid administration during labour.
Types of Participants: Healthy women with uneventful pregnancies.
Intervention: Participants were randomized to either epidural analgesia or parenteral opioid administration during labour.
Outcomes and Results: Maternal – there were no differences between the 2 groups in first-stage labour length, incidence of Cesarean delivery, incidence of instrumented vaginal delivery for dystocia, nausea, or mid-to-low back pain post-partum. However, second-stage labour length was longer (mean=15 min) and there were greater reports of fever and hypotension in the epidural group. Also, lower pain scores and greater satisfaction with analgesia were reported among the epidural group. There was no difference in lactation success at 6 wk and urinary incontinence was more frequent in the epidural group immediately post-partum, but not at 3 mo or 1 yr (evidence from PC studies only) Neonatal – there were no differences between the 2 groups for incidence of fetal heart rate abnormalities, intrapartum meconium, poor 5-min Apgar score, or low umbilical artery pH. However, the incidence of poor 1-min Apgar scores and need for neonatal naloxone were higher in the parenteral opioid group.
Conclusions: Epidural analgesia is a safe intrapartum method for labour pain relief and women should not avoid epidural analgesia for fear of neonatal harm, Cesarean delivery, breastfeeding difficulties, long-term back pain, or long-term urinary incontinence.

Nociceptive Pathways in Labour and Delivery
Labour
- Cervical dilation and effacement stimulates visceral nerve fibres entering the spinal cord at T10-L1

Delivery
- Distention of lower vagina and perineum causes somatic nociceptive impulses via the pudendal nerve entering the spinal cord at S2-S4

Options for Caesarean Section
- neuraxial: spinal or epidural
- general: used if contraindications or time precludes regional blockade

Pediatric Anesthesia

Respiratory System
- in comparison to adults, anatomical differences in infants include:
  - large head, short trachea/neck, large tongue, adenoids, and tonsils
  - narrow nasal passages (obligate nasal breathers until 5 mo)
  - narrowest part of airway at the level of the cricoid vs. glottis in adults
  - epiglottis is longer, U shaped and angled at 45°; carina is wider and is at the level of T2 (T4 in adults)
- physiologic differences include
  - faster RR, immature respiratory centres which are depressed by hypoxia/hypercapnea (airway closure occurs in the neonate at the end of expiration)
  - less oxygen reserve during apnea – decreased total lung volume, vital and functional reserve capacity together with higher metabolic needs
  - greater V/Q mismatch – lower lung compliance due to immature alveoli (mature at 8 yr)
  - greater work of breathing – greater chest wall compliance, weaker intercostals/diaphragm, and higher resistance to airflow

Cardiovascular System
- blood volume at birth is approximately 80 mL/kg; transfusion should be started if >10% of blood volume lost
- children have a high HR and low BP
- CO is dependent on HR, not stroke volume because of low heart wall compliance; therefore, bradycardia severe compromise in CO

Temperature Regulation
- vulnerable to hypothermia
- minimize heat loss by use of warming blankets, covering the infant's head, humidification of inspired gases, and warming of infused solutions

Central Nervous System
- MAC of halothane is increased compared to the adult (0.75% adult, 1.2% infant, 0.87% neonate)
- NMJ is immature for the first 4 wk of life and thus there is an increased sensitivity to non-depolarizing relaxants
- parasympathetics mature at birth, sympathetics mature at 4-6 mo thus autonomic imbalance
- infant brain is 12% of body weight and receives 34% of CO (adult: 2% body weight and 14% CO)

Glucose Maintenance
- infants less than 1 yr old can become seriously hypoglycemic during pre-operative fasting and post-operatively if feeding is not recommenced as soon as possible
- after 1 yr, children are able to maintain normal glucose homeostasis in excess of 8 h

Pharmacology
- higher dose requirements because of higher TBW (75% vs. 60% in adults) and greater volume of distribution
- barbiturates/opioids more potent due to greater permeability of BBB
- muscle relaxants
  - non-depolarizing
    - immature NMJ, variable response
  - depolarizing
    - must pre-treat with atropine or may experience profound bradycardia and/or sinus node arrest due to PNS > SNS (also dries oral secretions)
    - more susceptible to arrhythmias, hyperkalemia, rhabdomyolysis, myoglobinemia, masseter spasm and malignant hyperthermia

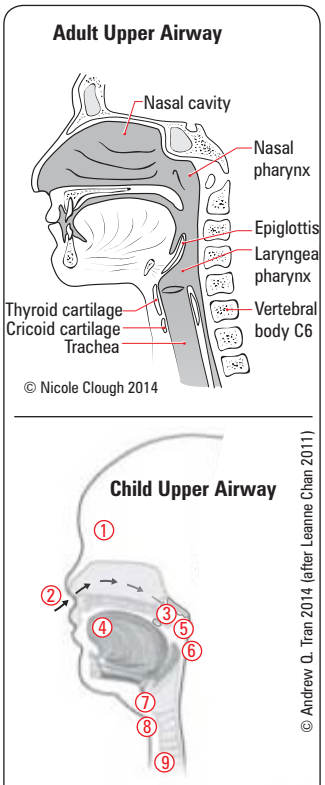

Figure 13. Comparison of pediatric vs. adult airway

To increase alveolar minute ventilation in neonates, increase respiratory rate, not tidal volume.
Neonate: 30-40 breaths/min
Age 1-13: (24 – [age/2]) breaths/min

ETT Sizing in Pediatrics
Diameter (mm) of tracheal tube in children after 1 year = (age/4) + 4
Length (cm) of tracheal tube = (age/2) + 12

Uncommon Complications

Malignant Hyperthermia

- hypermetabolic disorder of skeletal muscle
- due to an uncontrolled increase in intracellular Ca^{2+} (because of an anomaly of the ryanodine receptor which regulates Ca^{2+} channel in the sarcoplasmic reticulum of skeletal muscle)
- autosomal dominant inheritance
- incidence of 1-5 in 100,000, may be associated with skeletal muscle abnormalities such as dystrophy or myopathy
- anesthetic drugs triggering MH include
  - all inhalational agents except nitrous oxide
  - depolarizing muscle relaxants: SCh

Clinical Picture

- onset: immediate or hours after contact with trigger agent
  - increased oxygen consumption
  - increased ET_{CO2} on capnograph
  - tachycardia/dysrhythmia
  - tachypnea/cyanosis
  - diaphoresis
  - hypertension
  - increased temperature (late sign)
- muscular symptoms
  - trismus (masseter spasm) common but not specific for MH (occurs in 1% of children given SCh with halothane anesthesia)
  - tender, swollen muscles due to rhabdomyolysis
  - trunk or total body rigidity

Signs of Malignant Hyperthermia
- Unexplained rise in ET_{CO2}
- Increase in minute ventilation
- Tachycardia
- Rigidity
- Hyperthermia (late sign)

Complications

- coma
- DIC
- rhabdomyolysis
- myoglobinuric renal failure/hepatic dysfunction
- electrolyte abnormalities (e.g. hyperkalemia) and secondary arrhythmias
- ARDS
- pulmonary edema
- can be fatal if untreated

Prevention

- suspect MH in patients with a family history of problems/death with anesthetic
- avoid all trigger medications, use vapour free equipment, use regional anesthesia if possible
- central body temp and ET_{CO2} monitoring

Malignant Hyperthermia Management (Based on Malignant Hyperthermia Association of the U.S. [MHAUS] Guidelines, 2008)

1. notify surgeon, discontinue volatile agents and succinylcholine, hyperventilate with 100% oxygen at flows of 10 L/min or more, halt the procedure as soon as possible
2. dantrolene 2.5 mg/kg IV, through large-bore IV if possible
   - repeat until there is control of signs of MH; up to 30 mg/kg as necessary
3. bicarbonate 1-2 mEq/kg if blood gas values are not available for metabolic acidosis
4. cool patients with core temperature >39°C
   - lavage open body cavities, stomach, bladder, rectum; apply ice to surface; infuse cold saline IV
   - stop cooling if temperature is <38°C to prevent drift to <36°C
5. dysrhythmias usually respond to treatment of acidosis and hyperkalemia
   - use standard drug therapy except Ca^{2+} channel blockers as they may cause hyperkalemia and cardiac arrest in presence of dantrolene
6. hyperkalemia
   - treat with hyperventilation, bicarbonate, glucose/insulin, calcium
   - bicarbonate 1-2 mEq/kg IV, calcium chloride 10 mg/kg or calcium gluconate 10-50 mg/kg for life-threatening hyperkalemia and check glucose levels hourly
7. follow ET_{CO2}, electrolytes, blood gases, creatine kinase (CK), core temperature, urine output/colour with Foley catheter, coagulation studies
   - if CK and/or potassium rises persistently or urine output falls to <0.5 mL/kg/h, induce diuresis to >1 mL/kg/h urine to avoid myoglobinuric renal failure
8. maintain anesthesia with benzodiazepines, opioids, and propofol
9. transfer to ICU bed

Basic Principles of MH Management

"Some Hot Dude Better Get Iced Fluids Fast"
Stop all triggering agents, give 100% O_2
Hyperventilate
Dantrolene 2.5 mg/kg every 5 min
Bicarbonate
Glucose and insulin
IV fluids; cool patient to 38°C
Fluid output; consider furosemide
Tachycardia: be prepared to treat VT

Abnormal Pseudocholinesterase

- pseudocholinesterase hydrolyzes SCh and mivacurium
- individuals with abnormal pseudocholinesterase will have prolonged muscular blockade
- SCh and mivacurium are contraindicated in those with abnormal pseudocholinesterase
- if SCh or mivacurium are given accidentally, treat with mechanical ventilation until function returns to normal (do not use cholinesterase inhibitors rebound neuromuscular blockade once drug effect is terminated)

Appendices

Difficult Tracheal Intubation in Unconscious Patient

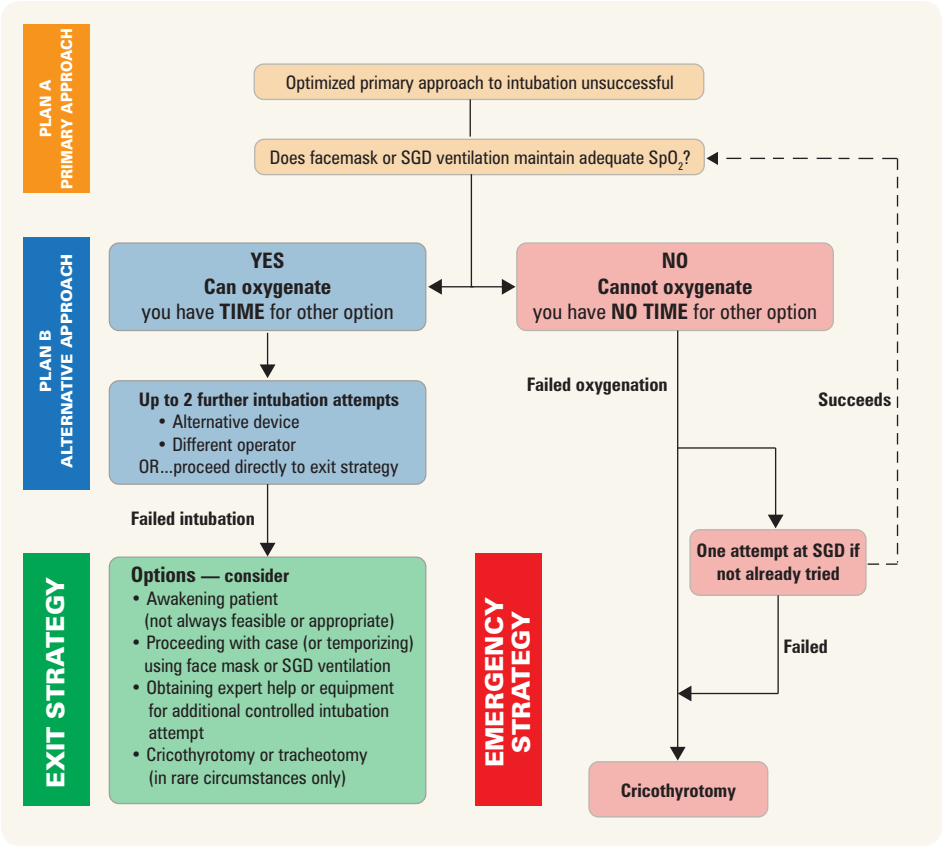

Figure 14. Difficult tracheal intubation encountered in the unconscious patient
SGD = supraglottic device

Reprinted with permission. Law JA, et al. The difficult airway with recommendations for management – Part 1 – Difficult tracheal intubation encountered in an unconscious/induced patient. *Can J Anesth* 2013;60:1089–1118.

Difficult Tracheal Intubation

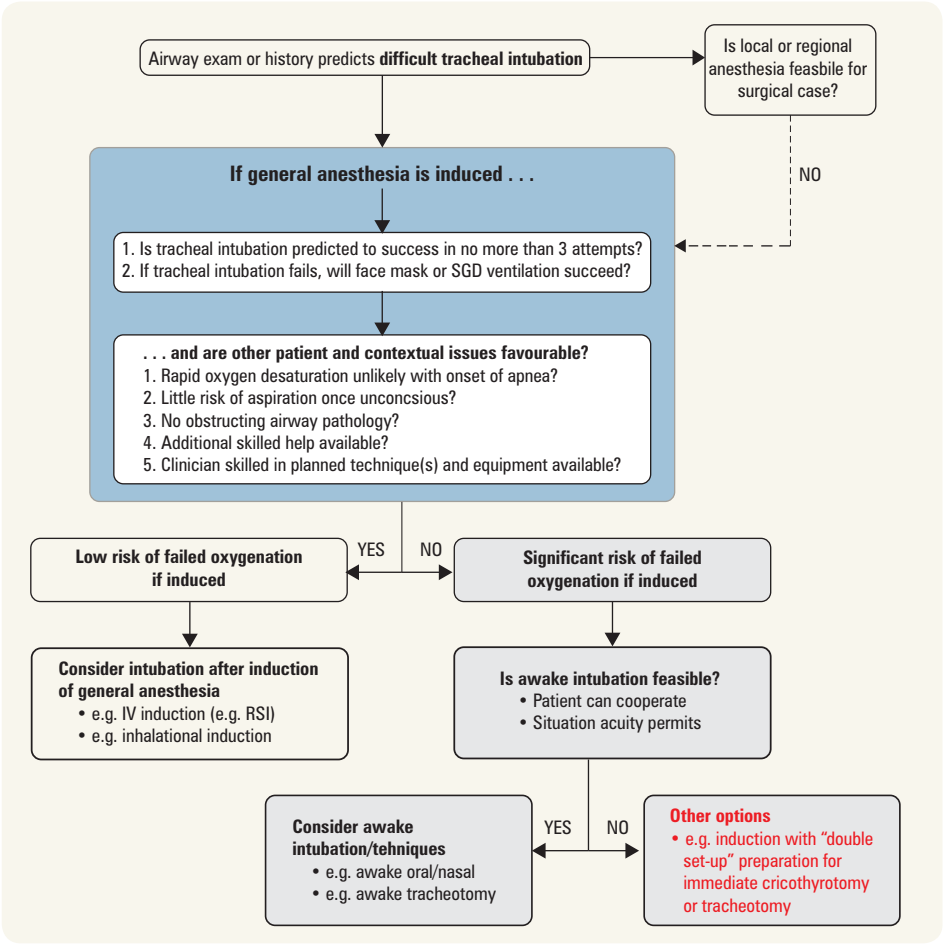

Figure 15. Anticipated difficult tracheal intubation
IV = intravenous; RSI = rapid sequence induction/intubation; SGD = supraglottic device

Reprinted with permission: Law JA, et al. The difficult airway with recommendations for management – Part 2 – The anticipated difficult airway. *Can J Anesth* 2013;60:1119-1138.

Advanced Cardiac Life Support Guidelines

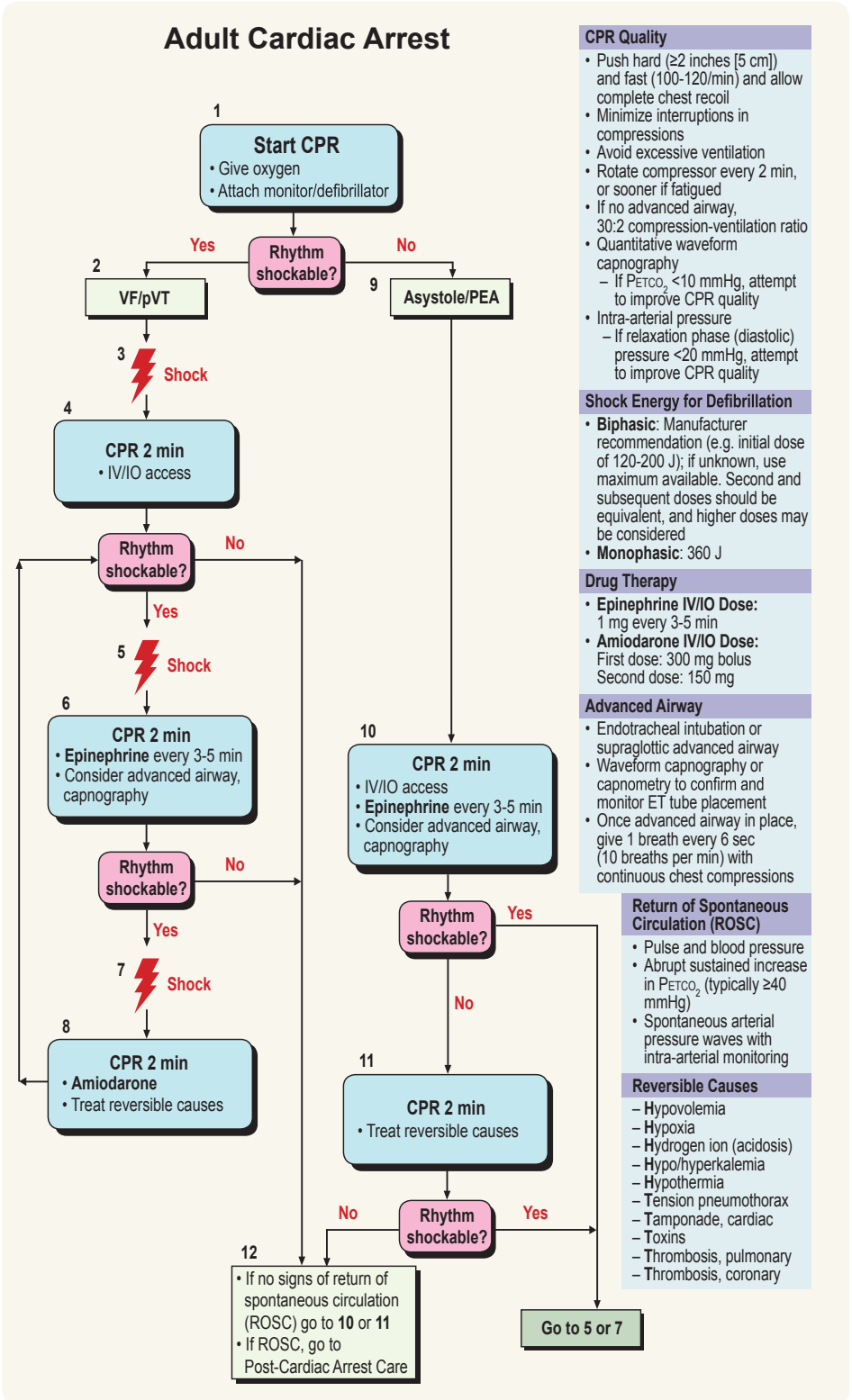

Figure 16. Adult cardiac arrest algorithm

Reprinted with permission: Link, M. S., Berkow, L. C., Kudenchuk, P. J., Halperin, H. R., Hess, E. P., Moitra, V. K., ... & White, R. D. (2015). Part 7: Adult Advanced Cardiovascular Life Support 2015 American Heart Association Guidelines Update for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. *Circulation 2015,* 132(18 suppl 2), S444-S464. ©2015 American Heart Association, Inc.

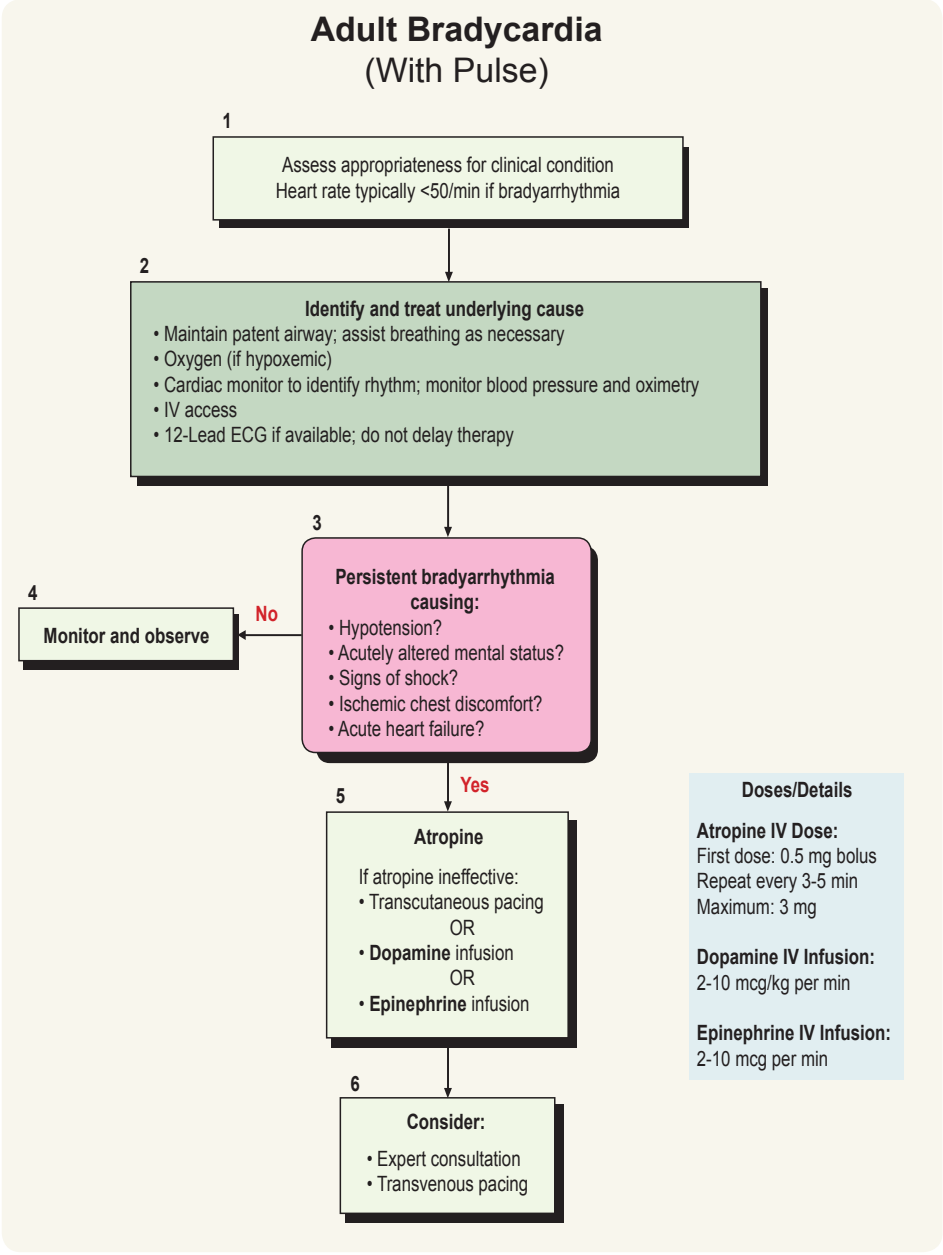

Figure 17. Adult tachycardia algorithm

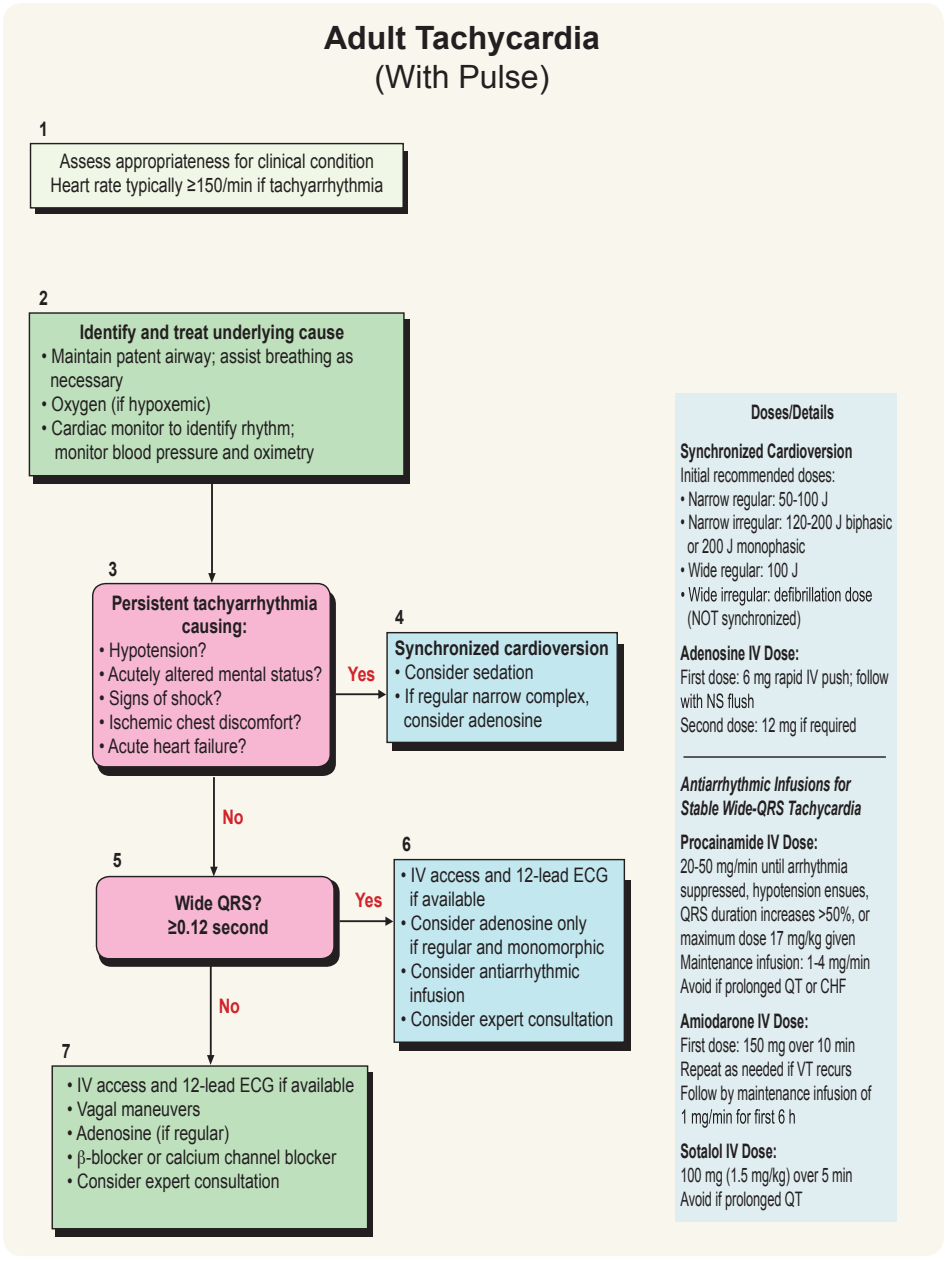

Figure 18. Adult bradycardia algorithm