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Acronyms

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

Overview of Anesthesia

- anesthesia: lack of sensation/perception

Approach to Anesthesia

Pre-operative

- pre-operative assessment
- patient optimization

Pre-operative/Peri-operative

- plan anesthetic
various types of anesthesia
pre-medication
airway management
monitors
induction
maintenance
emergence
tracheal extubation

Post-operative

- post-operative care

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, post-operative N/V
- FHx: abnormal anesthetic reactions, malignant hyperthermia, pseudocholinesterase deficiency (see *Uncommon Complications, A28*)
- medications, allergies (see *Pre-Operative Optimization: Medications, A4*)

• PMHx

- neuro: seizures, TIA/strokes, raised ICP, spinal disease, aneurysm, conditions affecting neuromuscular junction (e.g. myasthenia gravis)
- CVS: angina/CAD, MI, CHF, HTN, valvular disease, dysrhythmias, peripheral vascular disease (PVD), conditions requiring endocarditis prophylaxis, exercise tolerance, CCS/NYHA class (see **Cardiology and Cardiac Surgery, C26, C35** sidebar for New York Heart Association Classification)
- respiratory: smoking, asthma, COPD, recent upper respiratory tract infection (URTI), sleep apnea
- GI: GERD, liver disease, NPO status
- renal: acute vs. chronic renal insufficiency, dialysis, chronic kidney disease
- hematologic: anemia, coagulopathies, blood dyscrasias
- MSK: arthritides (e.g. rheumatoid arthritis, scleroderma), cervical spine pathology (e.g. cervical tumours, cervical infections/abscesses, trauma to cervical spine, previous cervical spine surgery), cervical spine instability (e.g. Trisomy 21)
- endocrine: DM, thyroid disorders, adrenal disorders
- other: morbid obesity, pregnancy, ethanol/recreational drug use

Physical Exam

- weight, height, BP, heart rate, 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 the morning air” position – **Figure 6, Panel C**)
 - Mallampati classification
 - “3-3-2 rule”
 - 3 of patient’s own fingers can be placed between the incisors (incisor distance)
 - 3 fingers along the floor of the mandible between the mentum and hyoid bone (hyoid-mental distance)
 - 2 fingers in the superior laryngeal notch (thyroid-mouth distance)
 - 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, CVP, and pulmonary artery (PA) catheters

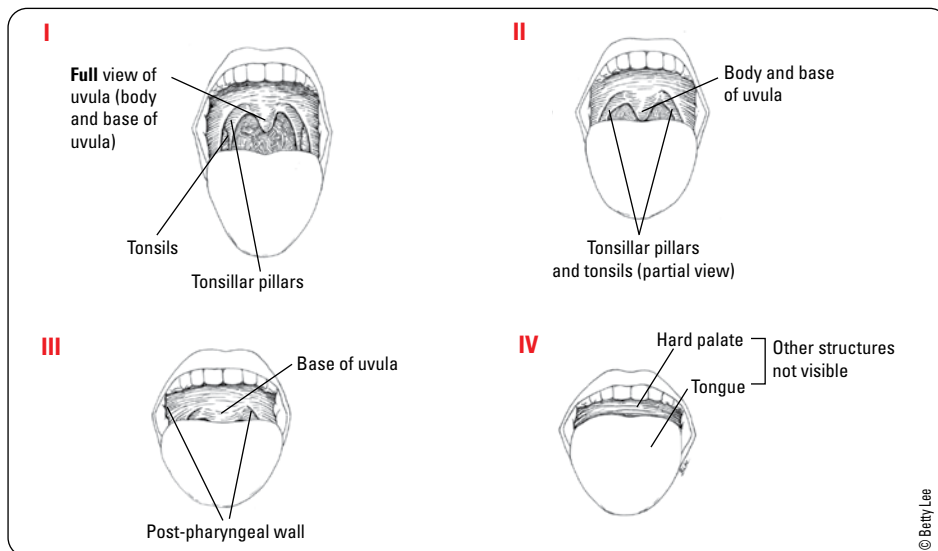


Figure 1. Mallampati classification of oral opening



Evaluation of Difficult Airway

LEMON

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



To Assess for Ventilation Difficulty

Anesthesiology 2000;92:1229-36

BONES

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

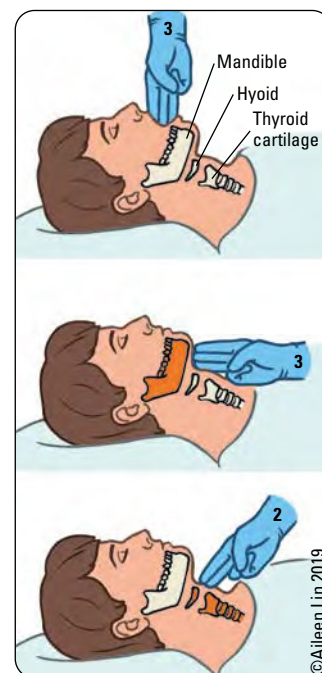


Figure 2. 3-3-2 Rule

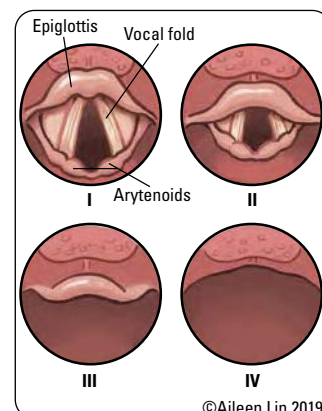


Figure 3. Laryngeal views

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	Genetically predisposed patient (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

Guidelines to the Practice of Anesthesia Revised Edition 2013. Supplement to the Canadian Journal of Anesthesia, Vol 60, Dec. 2013. Reproduced with permission © Canadian Anesthesiologists' Society

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: do not take on morning of surgery
 - ACEI and angiotensin receptor blockers: do not take on the day of surgery (controversial – they increase the risk of hypotension post-induction but have not been shown to increase mortality or adverse outcomes; therefore, some people hold and some do not)
 - warfarin (consider bridging with heparin), anti-platelet agents (e.g. clopidogrel), Xa inhibitor, direct thrombin inhibitors



Will This Patient Be Difficult to Intubate?

JAMA 2019;321:493-503

Objective: To identify risk factors and physical findings that predict difficult intubation.

Methods: Systematic review of MEDLINE and EMBASE databases.

Results: 62 studies, 33,559 patients. Physical examination findings that best predicted a difficult intubation included grade of class 3 on upper lid bite test (lower incisors cannot reach upper lip; LR 14, 95% CI 9.9-22), shorter hyomental distance (<3-5.5 cm; LR 9.4, 95% CI 4.1-10), retrognathia (mandible <9 cm from angle of jaw to tip of chin; LR 6.0, 95% CI 3.1-11), and a Mallampati score ≥3 (LR 4.1, 95% CI 3.0-5.6).



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.



Continuation versus Discontinuation of Antiplatelet Therapy for Bleeding and Ischaemic Events in Adults Undergoing Non-Cardiac Surgery

Cochrane DB Syst Rev 2018; CD012584

Objective: To compare continuation vs. discontinuation of antiplatelet therapy on the occurrence of bleeding and ischaemic events in adults undergoing non-cardiac surgery.

Methods: RCTs in Cochrane Central Register of Controlled Trials, MEDLINE, and Embase that compared adults taking single or dual antiplatelet therapy for at least two weeks, including patients with at least one cardiac risk factor. Included general, spinal, and regional anesthesia and excluded minor procedures involving only local anesthetic/sedation.

Results: 5 trials, 666 adult patients. Continuation or discontinuation had no difference on mortality at 30 d post-op (RR 1.21, 95% CI 0.34-4.27), blood loss (RR 1.37, 95% CI 0.83-2.26), or ischaemic events within 30 d of surgery (RR 0.67, 95% CI 0.25-1.77).

Conclusions: Moderate evidence supporting continuation or discontinuation of antiplatelet therapy makes no difference on bleeding requiring transfusion. Low evidence supporting no difference in mortality or ischaemic events.

- discuss perioperative use of ASA, NSAIDs with surgeon ($\pm$ patient's cardiologist/internist)
- in patients undergoing non-cardiac surgery, starting or continuing low-dose ASA 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
- herbal supplements: stop one week prior to elective surgery (ephedra, garlic, ginkgo, ginseng, kava, St. John's Wort, Valerian, Echinacea)
- **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 d 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 (but increases risk of perioperative strokes)
 - 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

Respiratory Diseases

- smoking
 - adverse effects: altered mucus secretion and clearance, decreased small airway calibre, altered oxygen carrying capacity, increased airway reactivity, 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 β_2 -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 in the short-term)
 - delay elective surgery for poorly controlled asthma (increased cough or sputum production, active wheezing)
 - ideally, 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

Aspiration

- increased risk of aspiration with:
 - decreased LOC (drugs/alcohol, head injury, CNS pathology, trauma/shock)
 - delayed gastric emptying (non-fasted within 8 h, diabetes, narcotics)
 - decreased sphincter competence (GERD, hiatus hernia, nasogastric tube, pregnancy, obesity)
 - increased abdominal pressure (pregnancy, obesity, bowel obstruction, acute abdomen)
 - unprotected airway (laryngeal mask vs. ETT)
- management
 - manage risk factors if possible
 - utilize protected airway (i.e. endotracheal tube)
 - reduce gastric volume and acidity
 - delay inhibiting airway reflexes with muscular relaxants
 - employ rapid sequence induction (*see Rapid Sequence Induction, A15*)



Perioperative β -blockers for Preventing Surgery-related Mortality and Morbidity

Cochrane DB Syst Rev 2018; CD004476

Objectives: Effects of perioperative β -blockers for prevention of surgery-related mortality and morbidity in patients undergoing any type of surgery while under general anesthesia.

Outcomes:

Cardiac surgery: Perioperative β -blockers associated with reduced supraventricular (RR 0.44, 95% CI 0.36-0.53) and ventricular (RR 0.37, 95% CI 0.24-0.58) arrhythmias in the aftermath of surgery. Effect on mortality, AMI, stroke, CHF, hypotension, and bradycardia unclear.

Non-cardiac surgery: Perioperative β -blockers associated with decreased incidence of supraventricular arrhythmias (RR 0.73, 95% CI 0.57-0.94) and AMI (RR 0.73, 95% CI 0.61-0.87). However, this is potentially offset by increased all-cause mortality (RR 1.25, CI 95% 1.00-1.57) with potential increase in stroke rate (RR 1.59, 95% CI 0.93-2.71).



β -blockers

- β_1 -receptors are located primarily in the heart and kidneys
- β_2 -receptors are located in the smooth muscle (i.e. bronchi, uterus)
- Non-selective β -blockers block β_1 and β_2 -receptors (labetalol*, carvedilol**, nadolol). Caution is required with non-selective β -blockers, particularly in patients with respiratory conditions where β_2 blockade can result in airway reactivity

*labetalol is both an α - and β -blocker

**carvedilol is also both an α and β blocker

Fasting Guidelines

Fasting Guidelines Prior to Surgery (Canadian Anesthesiologists' Society)

- before elective procedures, the minimum duration of fasting should be:
 - 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 (see Table 1)
- anemia
 - pre-operative treatments to increase hemoglobin (PO or IV iron supplementation, erythropoietin or pre-admission blood collection in certain populations)
- coagulopathies
 - discontinue or modify anticoagulation therapies (warfarin, clopidogrel, ASA, apixaban, dabigatran) 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 (DM)
 - 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 central, peripheral and autonomic nervous systems
 - preoperative guidelines for DM:
 - ♦ 1. verify target blood glucose concentration with frequent glucose monitoring: <10 mmol/L in critical patients, <7.8 mmol/L in stable patients)
 - ♦ 2. use insulin therapy to maintain glycemic goals
 - ♦ 3. hold biguanides, α -glucosidase inhibitors, thiazolidinediones, sulfonylureas and GLP-1 agonists on the morning of surgery
 - ♦ 4. consider cancelling nonemergency procedures if patient presents with metabolic abnormalities (DKA, HHS, etc.) or glucose reading above 22.2-27.7 mmol/L
 - formulate intraoperative glucose management plan based on type (1 vs. 2), glucose control, and extent of end organ damage
- hyperthyroidism and hypothyroidism
 - hyperthyroidism: can experience sudden release of thyroid hormone (thyroid storm) if not treated or well-controlled pre-operatively; 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
- severity of OSA may be determined from sleep studies and level of pressure prescribed for home CPAP device
- both obesity and OSA independently increase risk of difficult ventilation, intubation and post-operative respiratory complications

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, O₂ saturation, End Tidal CO₂, dose and route of drugs and fluids
- continuous monitoring: see *Routine Monitors for All Cases, A7*



Preoperative Anemia and Postoperative Outcomes in Non-Cardiac Surgery: a Retrospective Cohort Study

Lancet 2011; 378: 1396-1407

Objectives: Assess effect of preoperative anaemia on 30 d postoperative morbidity and mortality in patients undergoing major non-cardiac surgery.

Methods: Patients undergoing major non-cardiac surgery in 2008 from the American College of Surgeons' National Surgical Quality Improvement Program database.

Results: 227 425 adult patients. Postoperative mortality at 30 d was higher in patients with anaemia than those without (OR 1.42, 95% CI 1.31-1.54).

Outcomes: Preoperative anaemia, even to a mild degree, is independently associated with an increased risk of 30 d morbidity and mortality.



Pre-Anesthetic Checklist

MS MAIDS

Machine: connected, pressures okay, all metres functioning, vaporizers full

Suction: connected and working

Monitor: all monitors appropriate for the case

Airway: laryngoscope and blades, ETT, syringe, stylet, oral and nasal airways, tape, bag, and mask

IV: second IV set-up and ready if needed

Drugs: case-specific drugs ready and emergency medications in correct location and accessible

Special equipment: OG tube, CVP monitor, shoulder roll, etc.

Routine Monitors for All Cases

- pulse oximeter, BP monitor, electrocardiography, capnography (required for general anesthesia and deep procedural sedation, Ramsey Sedation Scale 4-6), and an 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, spirometry, manometer to measure endotracheal tube cuff pressure

Elements to Monitor

- anesthetic depth
 - end-tidal inhaled anesthetic monitoring and EEG monitoring, such as a Bispectral Index monitor, can be used as assessments of anesthetic depth
 - inadequate: blink reflex present when eyelashes lightly touched, HTN, tachycardia, tearing or sweating. However, these findings are non-specific
 - excessive: hypotension, bradycardia
- oxygenation: pulse oximetry, FiO_2
- ventilation: verify correct position of ETT, chest excursions, breath sounds, ETCO_2 analysis, end tidal inhaled anesthesia analysis
- circulation: heart rate, rhythm, BP, telemetry, oximetry, pulmonary capillary wedge pressure
- temperature
- hourly urine output

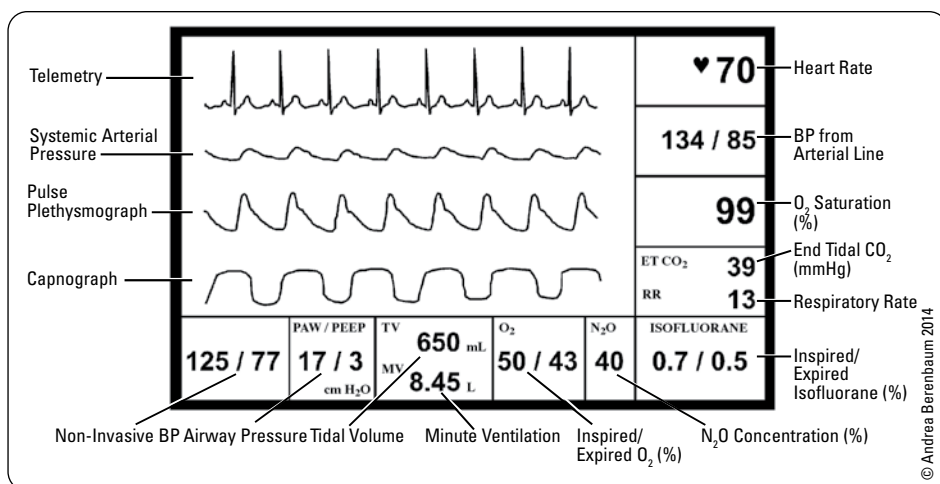


Figure 4. Typical anesthesia monitor

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)

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)

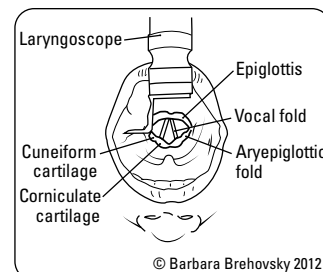


Figure 5. Landmarks for intubation

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) Patent airway Protects against aspiration Positive pressure ventilation Pulmonary toilet (suction) Pharmacologic administration during 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 ₂ O 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 <i>Complications During Laryngoscopy and Intubation, A9</i>
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% O₂ 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
 - poor/no view of glottic opening can be remediated by anterior laryngeal pressure
- laryngoscope tip placed in the epiglottic vallecula in order to visualize cord

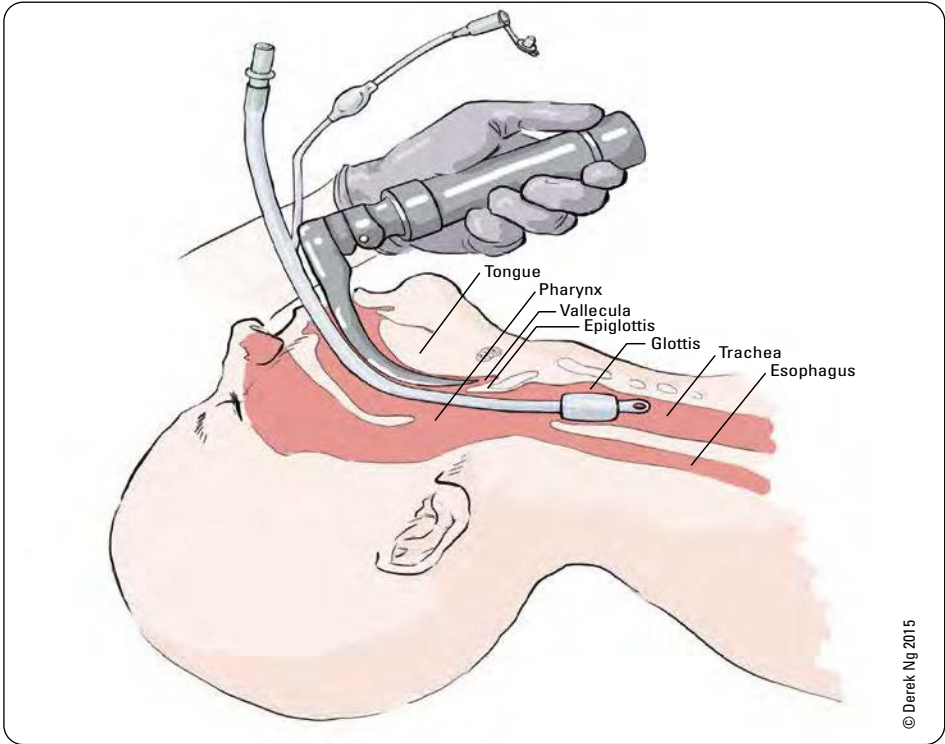


Figure 7. Sagittal view of airway with laryngoscope in vallecula



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, Syringe for tube cuff inflation



Medications that can be given through the ETT

NAVEL

- Naloxone
- Atropine
- Ventolin
- Epinephrine
- Lidocaine

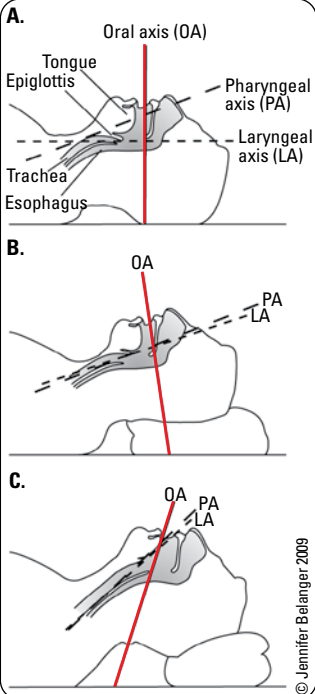


Figure 6. Anatomic considerations in laryngoscopy

- 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 Endotracheal Tube

- direct
 - visualization of ETT passing through cords
 - bronchoscopic visualization of ETT in trachea
- indirect
 - ET CO_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 whether the ETT is in the trachea
- esophageal intubation suspected when:
 - ET CO_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

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), *see Appendices, 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)



Differential Diagnosis of Poor Bilateral Breath Sounds after Intubation

DOPE

Displaced ETT
Obstruction
Pneumothorax
Esophageal intubation



Cormack-Lehane Classification of Laryngeal View (*Figure 3*)

- Grade 1: all laryngeal structures revealed
- Grade 2: posterior laryngeal 2A (posterior vocal folds) 2B (arytenoids)
- Grade 3: Larynx concealed, only epiglottis
- Grade 4: Neither glottis nor epiglottis



If you encounter difficulty with tracheal intubation, oxygenation is more important than intubation

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 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 $\text{SaO}_2 < 85\%$, frank cyanosis at $\text{SaO}_2 = 67\%$

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 $\times$ RR) results in a decrease in FiO_2
- e.g. nasal cannula (prongs)
 - well tolerated if flow rates $< 5\text{-}6$ 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

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 entrainment
 - 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, atelectrauma), 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

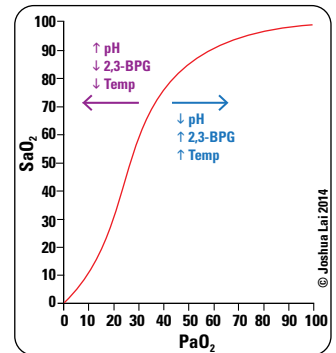


Figure 8. HbO_2 saturation curve



Composition of Air

78.1% nitrogen
20.9% oxygen
0.9% argon
0.04% carbon dioxide



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 CO₂ 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
 - in traditional PCV, tidal volume is not guaranteed thus changes in compliance and resistance affect tidal volume
- 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 CO₂ 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	Technical issues
Incorrect placement of sampling catheter	Water in capnography device
Inadequate sampling volume	Anesthetic breathing circuit error
	Inadequate fresh gas flow
	Rebreathing
	Exhausted soda lime
	Faulty circuit absorber valves
V/Q mismatch	Low bicarbonate
Pulmonary thromboembolism	
Incipient pulmonary edema	
Air embolism	



Monitoring Ventilatory Therapy

- Pulse oximetry, end-tidal CO₂ 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

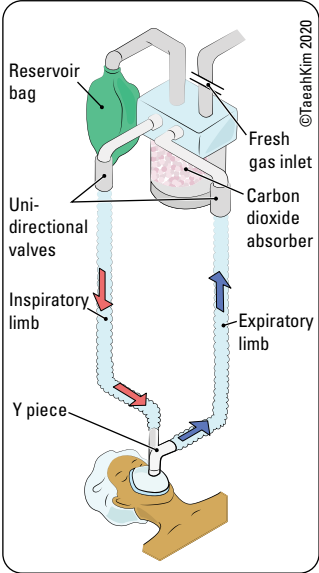


Figure 9. The anesthesia circuit

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: hypothermia, hypovolemia, hypoxia, hydrogen ions (acidosis), hypo/hyperkalemia
 - 5 Ts: tamponade (cardiac), thrombosis (pulmonary), thrombosis (coronary), tension pneumothorax, toxins (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 Appendices, A32*

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 Appendices, A33*

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. suprachoroidal hemorrhage, opioids, edrophonium, neostigmine, halothane, digoxin, β -blockers)
 - high spinal/epidural anesthesia
- for management of bradycardia, *see Appendices, A33*

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, ID19](#)
- e) spinal/neurogenic shock
 - decreased sympathetic tone
 - hypotension without tachycardia or peripheral vasoconstriction (warm skin)
- f) anaphylactic shock
 - see [Emergency Medicine, ER3](#)
- g) drugs
 - vasodilators, high spinal anesthetic interfering with sympathetic outflow
- h) other
 - transfusion reaction, Addisonian crisis, thyrotoxicosis, hypothyroid, aortocaval syndrome
 - see [Hematology](#) and [Endocrinology](#)

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, thyroid storm, pheochromocytoma (see [Endocrinology, E37](#))

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, fluids given with blood products and medications
- increasingly, Enhanced Recovery After Surgery protocols recommend consumption of clear fluids up to two hours prior to surgery
- both inadequate fluid resuscitation AND excessive fluid administration increases morbidity post-operatively

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)
- replacement of 'third space' losses is not warranted
- maintenance should not exceed 3mL/kg/hr
- 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
- 4-2-1 rule: 4 mL/h first 10 kg, 2 mL/h next 10 kg, 1 mL/h for every kg after to calculate maintenance fluid requirement
- may also add 40 kg to weight for adults to calculate maintenance fluid requirement in mL/h



Intraoperative Shock

SHOCK TED

Sepsis or Spinal shock
Hypovolemic/Hemorrhagic
Obstructive
Cardiogenic
anaphylactic
Transfusions
Endocrine (Addison's disease, hyperthyroidism)
Drugs



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



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 = $70 \times 0.6 = 42$ L)
- total Na^+ content determines ECF volume; $[\text{Na}^+]$ 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
 - hypoadosteronism
 - 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

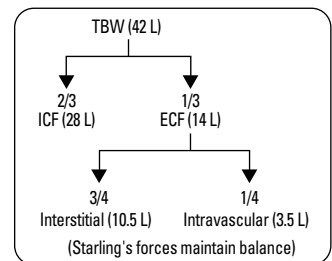


Figure 10. Total body water division in a 70 kg adult

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?

- traditionally thought that fluid loss during surgery resulted from blood loss, losses from Foley catheter, NG, surgical drains, and third spacing (sequestration of fluid into other body compartments such as GI, lung, evaporation)
- fluid therapy accounting for these losses often resulted in excess crystalloid administration
- goal-directed fluid regimens associated with lower rate of post-op complications compared to pre-determined calculations

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
- consensus guidelines recommend use of balanced crystalloid (i.e. Ringer's lactate) over normal saline for routine replacement and resuscitation
- maintain euolemia 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

- includes protein colloids (albumin and gelatin solutions) and non-protein colloids (dextrans and starches e.g. hydroxyethyl starch [HES])
- distributes within intravascular volume
- 1:1 ratio (infusion:blood loss) only in terms of replacing intravascular volume
- 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 People

Cochrane DB Syst Rev 2018;CD000567

Objectives: To assess effect of colloids vs. crystalloids in critically ill patients on mortality, need for transfusions or renal replacement therapy, and adverse events.

Methods: Systematic review of RCTs and quasi-RCTs involving trauma, burns, or medical conditions (i.e. sepsis). Searched CENTRAL, MEDLINE, and Embase.

Outcomes: 69 studies, 30 020 participants. Starches, dextrans, albumin or FFP (moderate-certainty evidence), or gelatins (low-certainty evidence) vs. crystalloids has no difference on mortality. Starches slightly increase the need for blood transfusion (moderate-certainty evidence), and albumin or FFP may make little or no difference to the need for renal replacement therapy (low-certainty evidence). Evidence for blood transfusions for dextrans, and albumin or FFP, is uncertain.

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
Pentaspán®	10%	1:1.2-1.5	18-24	28

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 (e.g. from the time when they are unconscious 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, suction on	
2. Pre-Oxygenation/Denitrogenation	100% O ₂ for 3 min or 4-8 vital capacity breaths; reduce risk of hypoxemia during apneic period following induction	
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 (most commonly Sch, occasionally high dose rocuronium) given IMMEDIATELY after induction agent
6. Ventilation	Bag-mask ventilation	DO NOT bag ventilate – can increase risk of aspiration
7. Cricoid Pressure	Posterior pressure on thyroid cartilage to improve view of vocal cords as indicated	Traditionally 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) but increasingly omitted
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	



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

Edition ed. Toronto: Sunnybrook and Women's College Health Science Centre; 2016



Rocuronium versus Succinylcholine for Rapid Sequence Induction Intubation

Cochrane DB Syst Rev 2015;CD002788.

Objective: Whether rocuronium creates intubating conditions comparable to those of succinylcholine during RSI intubations.

Methods: Systematic review of RCTs or CCTs with a dose of at least 0.6 mg/kg for rocuronium and 1 mg/kg for succinylcholine.

Results: 50 trials, 4151 participants.

Succinylcholine was superior to rocuronium for achieving excellent intubating conditions (RR 0.86, 95% CI 0.81-0.92) and clinically acceptable intubation conditions (RR 0.97, 95% CI 0.95-0.99).

Conclusion: Succinylcholine created superior intubation conditions to rocuronium in achieving excellent and clinically acceptable intubating conditions.

Induction Agents

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

Intravenous Agents

- 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
 - most commonly used is propofol or ketamine
 - a continuous propofol infusion may also be used for the maintenance phase of GA



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

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	Imidazole derivative - hypnotic
Action	Inhibitory at GABA synapse Decreased cerebral metabolic rate and blood flow, decreased ICP, decreased SVR, decreased BP, and decreased SV	Inhibitory at GABA synapse Decreased cerebral metabolism and blood flow, decreased CPP, decreased CO, decreased BP, decreased reflex tachycardia, decreased respiration	May act on NMDA (antagonistically), 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	Inhibitory at GABA synapse 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 s Lasts 4-8 min
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

*As of 2011, Thiopental has been discontinued from production for North America

Volatile Inhalational Agents

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

Table 9. Volatile Inhalational Agents

	Sevoflurane	Desflurane	Isoflurane	Enflurane	Halothane	Nitrous oxide (N ₂ O)*
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 N₂O
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 N₂O is administered
Diffusion hypoxia: during anesthesia, the washout of N₂O from body stores into alveoli can dilute the alveolar O₂, creating a hypoxic mixture if the original O₂ 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
- MAC of halogenated volatile anesthetics decrease by approximately 6% per additional decade of age in adults
- 1.2-1.3 times MAC will often ablate response to stimuli in the general population
- MAC values are roughly additive when mixing N₂O 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 N₂O = 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



Factors increasing MAC: chronic alcohol use, hyperthyroidism, hyperthermia, stimulant (amphetamines), young age
Factors decreasing MAC: acute alcohol intoxication, hypothermia, sedating drugs, advanced age, drugs (opioids, benzodiazepines)

Muscle Relaxants and Reversing Agents

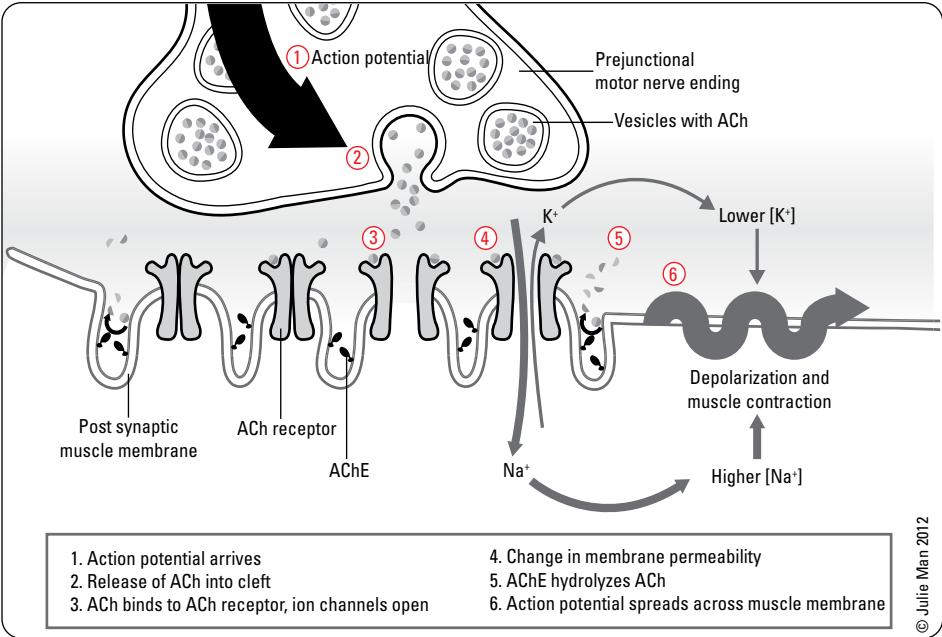


Figure 11. Review of anatomy and physiology of the neuromuscular junction (NMJ)

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



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 <i>Malignant Hyperthermia, A28</i>) 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

- sugammadex is a selective relaxant binding agent
- neostigmine, pyridostigmine, edrophonium are acetylcholinesterase inhibitors
- 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, increased peristalsis, and bronchoconstriction)

Table 12. Reversal Agents for Non-Depolarizing Relaxants

Agent	Pyridostigmine	Neostigmine	Edrophonium	Sugammadex
Onset	Slow	Intermediate	Intermediate	Fast
Mechanism of Action	(acetylcholinesterase inhibitors) 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*			Encapsulates and inactivates rocuronium and vecuronium → ↓ amount of agent available to bind to receptors in NMJ
Dose	0.1-0.4 mg/kg	0.04-0.08 mg/kg	0.5-1 mg/kg	2-16 mg/kg
Recommended Anticholinergic	Glycopyrrolate	Glycopyrrolate	Atropine	NA
Dose of Anticholinergic (per mg)	0.05 mg	0.2 mg	0.014 mg	NA

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

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
 - 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% O₂ 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 O₂ 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% O₂, low-dose propofol (0.5-1.0 mg/kg) optional, low-dose succinylcholine (approximately 0.25 mg/kg) and reintubate 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 and/or anxiolysis may be indicated before block
- monitoring should be as extensive as for general anesthesia

Epidural and Spinal Anesthesia

- most common for surgeries performed below level of umbilicus but can be extended to any level (useful in thoracic, abdominal and lower extremity surgeries). Typically placed in thoracic or lumbar spine

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



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



Landmarking Epidural/Spinal Anesthesia

- Spinous processes should be maximally flexed
- L4 spinous processes found between iliac crests
- T7 landmark at the tip of the scapula

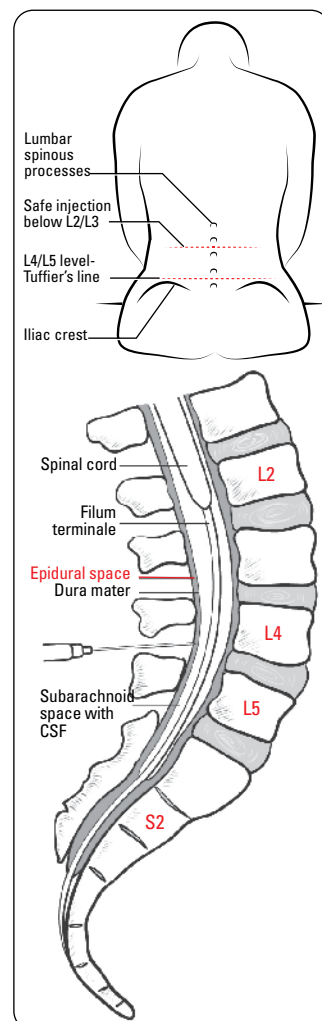
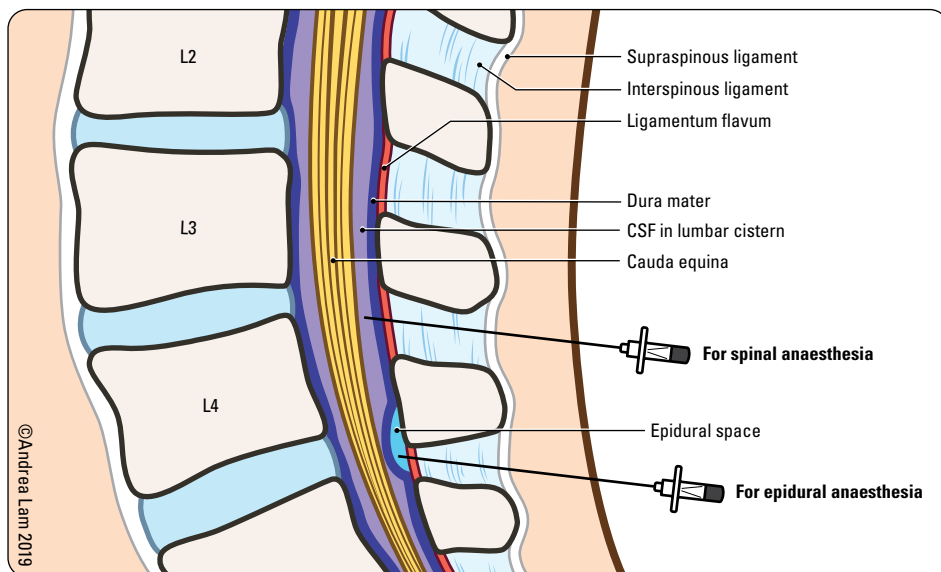


Classic Presentation of Dural Puncture Headache

- 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 CSF 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 12. Landmarks for placement of epidural/spinal anesthesia****Figure 13. Sagittal cross-section of the anatomy of neuraxial anesthesia****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

Local Anesthesia

Local Anesthetic Agents

- see Table 14, A22, 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 (e.g. procaine, tetracaine) are broken down by plasma and hepatic esterases; metabolites excreted via kidneys
- amide-type LA (lidocaine, bupivacaine) 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 bupivacaine at low doses)
 - potential for toxicity

Table 14. Local Anesthetic Agents

	Maximum Dose	Maximum Dose with Epinephrine	Potency	Duration	Onset
chloroprocaine	11 mg/kg	14 mg/kg	Low	15-30 min	Fast
lidocaine	5 mg/kg	7 mg/kg	Medium	1-2 h	Fast
bupivacaine	2.5 mg/kg	3 mg/kg	High	3-8 h	Slow
ropivacaine	2.5 mg/kg	3 mg/kg	High	2-8 h	Medium
mepivacaine	5 mg/kg	7 mg/kg	Medium	3-6 h	Fast

Systemic Toxicity

- see Table 14, A22 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% O₂, manage ABCs
- diazepam or other anticonvulsant to prevent potential onset of seizures
- manage arrhythmias
- Intralipid® 20% to bind local anesthetic in circulation

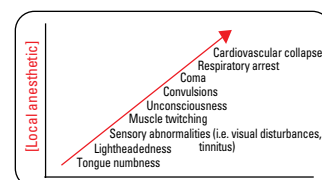


Figure 14. Local anesthetic systemic toxicity

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

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



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

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

Uncontrolled/Poorly Controlled Pain

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 ICP)
- 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 $\pm$ inotropic agents

Hypertension

- pain, hypercapnia, hypoxemia, increased intravascular fluid volume, and sympathomimetic drugs can cause hypertension
- IV nitroglycerin, hydralazine, calcium channel blockers 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

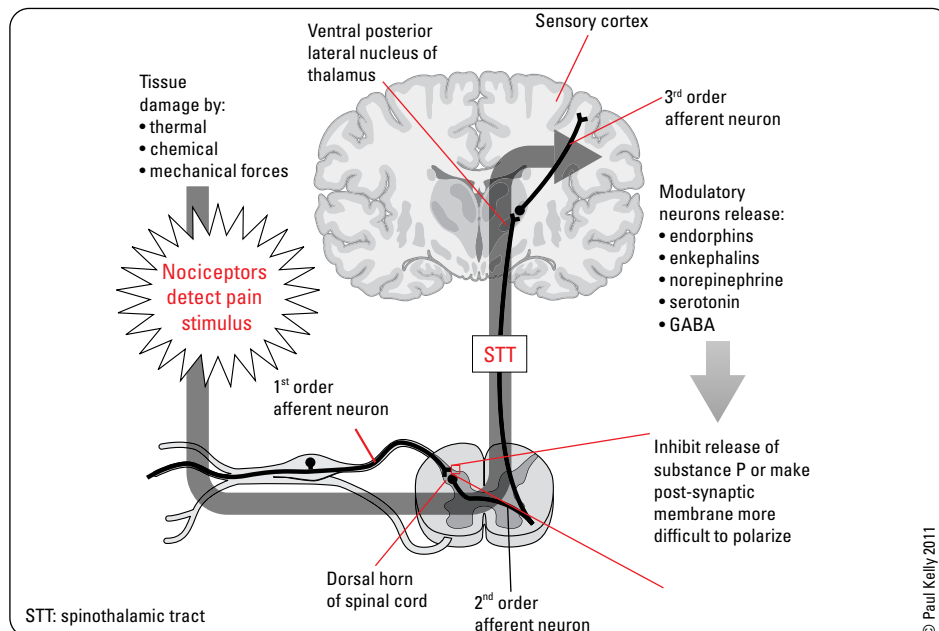


Figure 15. Acute pain mechanism



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: N₂O, opioids, volatile agents



Dexamethasone versus Standard Treatment for Postoperative Nausea and Vomiting in Gastrointestinal Surgery: Randomised Controlled Trial (DREAMS Trial)

BMJ 2017;357:j1455

Objectives: Whether preoperative dexamethasone reduces postoperative vomiting in patients undergoing elective bowel surgery and whether it is associated with other measurable benefits during recovery from surgery.

Method: Pragmatic two arm parallel group randomised trial with blinded postoperative care and outcome assessment.

Results: Patients receiving additional 8 mg of IV dexamethasone at induction is associated with lower rates of vomiting within 24 hours of surgery (NNT=13) and reduced need for antiemetics up to 72h (NNT=8) vs. standard of care.

Conclusions: A single dose of dexamethasone at induction of anesthesia significantly reduces incidence of PONV and need for rescue antiemetics post-operatively with no increase in adverse events.

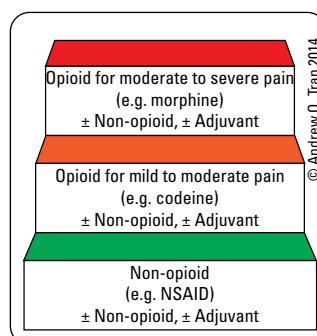


Figure 16. WHO analgesia ladder

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. Not ideal as analgesic effect depends on highly variable CYP2D6 metabolism
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 Decreased use for pain management due to potential toxicity compared to other opioids. Typically reserved to treat post-operative shivering. Absolute contraindication in patients taking MAO-inhibitors
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 (Oxyne®)	15 mg PO	10-20 mg PO (no IV)	Late (30-45 min)	Long (8-12 h)	Do not split, crush, or chew tablet (but can be difficult to swallow)
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)	Less pruritus, N&V, and sedation compared to morphine
Fentanyl	100 µg IV	2-3 µg/kg IV	Rapid (<5 min)	Short (0.5-1 h)	Transient muscle rigidity in very high doses



Cautionary Use of NSAIDs in Patients with

- Asthma
- Coagulopathy
- GI ulcers
- Renal insufficiency
- Pregnancy, 3rd trimester



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		



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 versus Non-Patient Controlled Opioid Analgesia for Postoperative Pain

Cochrane DB Syst Rev 2015;CD003348

Purpose: To evaluate the efficacy of patient controlled analgesia (PCA) versus non-patient controlled opioid analgesia for as-needed opioid analgesia for postoperative pain relief.

Methods: Meta-analyses of RCTs 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: 49 studies with a total of 1725 patients receiving PCA and 1687 patients assigned to a control group. PCA had a lower VAS pain intensity score vs. non-patient controlled analgesia over most time intervals in the first 48h. PCA was associated with higher patient satisfaction and consumed higher amounts of opioids than controls. PCA was also associated with higher incidence of pruritus but not other adverse events.

Conclusions: Moderate to low quality evidence that PCA is an efficacious alternative to non-patient controlled systemic analgesia for postoperative pain control.

Table 16. Opioids (continued)

Agent	Relative Dose to 10 mg Morphine IV	Moderate Dose	Onset	Duration	Special Considerations
Remifentanyl	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
Methadone (opioid agonist)	Morphine to methadone conversion is variable based on patient's morphine dose. Ranges from 1/4 to 1/20	15-40 mg/d in divided doses	Rapid (8 min)	15-90 (24h average)	Can only be prescribed by federally/provincially licensed physicians Acts through both NMDA and mu receptors Challenging due to variable equianalgesic dose and half-life After titration, accumulates in tissue for once/twice daily dosing Metabolized by CYP3A4 Caution with high doses, may cause QT prolongation, baseline ECG required
Buprenorphine (opioid agonist antagonist)	Varies depending on route of administration (pill/film, transdermal)	Film: 2 mg up to max of 24 mg	Moderate (30 min)	6-8 h	For moderate to severe chronic pain and opioid addiction Ceiling effect for respiratory depression but not analgesia High affinity to mu-opioid receptors, very resistant to reversal with opioid antagonists

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

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 3 mo period
- pain of duration or intensity that persists beyond normal tissue healing and adversely affects functioning
- in the perioperative period, consider continuing regular long-acting analgesics and augmenting with regional techniques, adjuvants, additional opioid analgesia and non-pharmacological techniques

Central Sensitization

- central sensitization: hyperalgesia (increased sensitivity to pain) as a result of central nervous system mechanisms
- may have nociceptive and neuropathic components; dysregulation of analgesic pathways implicated
- plays a role in fibromyalgia

Chronic Post-Surgical Pain

- chronic post-surgical pain (CPSP): pain that develops after surgery and persists for at least 2 mo
- primary predictor of CPSP is history of chronic pain; other risk factors include female gender, surgical procedure/approach, poor social supports, catastrophizing behaviour



Epidural versus Non-Epidural or No Analgesia for Pain Management in Labour

Cochrane DB Syst Rev 2018;CD000331

Objectives: To assess effectiveness and safety of all types of epidural analgesia when compared with non-epidural or no pain relief during labour.

Methods: Systematic review of RCTs comparing epidural with any form of pain relief not involving regional blockade, or no pain relief in labour.

Results: 52 studies involving over 11000 women were included; 34 studies compared epidural analgesia with opioids. Epidural analgesia was associated with lower pain intensity, higher satisfaction, and decreased need for additional pain relief vs. opioids. While it was also associated with increased risk of assisted vaginal birth (RR 1.44, 95% CI 1.29-1.60), post-hoc analysis of studies conducted after 2005 eliminates this risk (RR 1.19, 95% CI 0.97-1.46). Women with epidural experienced more hypotension, motor blockage, fever, and urinary retention with less risk of respiratory depression and nausea/vomiting. There was no difference in neonatal outcomes, admission to NICU, caesarean section rates, or maternal long-term backache.

Conclusions: Epidural analgesia may be more effective in reducing pain during labour and increasing maternal satisfaction than non-epidural methods and, when considering modern approaches, is not associated with increased instrumentation. Epidural analgesia had no impact on the risk of caesarean section or long-term backache, and did not appear to have an immediate effect on neonatal status as determined by Apgar scores or in admissions to NICU.



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

Obstetrical Anesthesia

Anesthesia Considerations in Pregnancy

- airway
 - possible difficult airway as tissues becomes edematous and friable especially in labour
- respiratory
 - decreased FRC and increased O₂ 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 (supine hypotensive syndrome) – therefore for surgery, a pregnant patient is positioned in left uterine displacement (approximately 15°C) 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; fentanyl can be considered
- inhalational analgesia
 - easy to administer, makes uterine contractions more tolerable, but does not relieve pain completely
 - 50% nitrous oxide is insufficient alone but good safety profile for mother + child
- neuraxial anesthesia
 - provides excellent analgesia with minimal depressant effects
 - hypotension is the most common complication
 - maternal BP monitored q2-5min for 15-20 min after initiation and regularly thereafter
 - epidural usually given as it preferentially blocks sensation, leaving motor function intact

Options for Caesarean Section

- neuraxial: spinal or epidural
- general: used if contraindications or time precludes regional blockade (*see Regional Anesthesia, Epidural and Spinal Anesthesia, A20*)

Pediatric Anesthesia

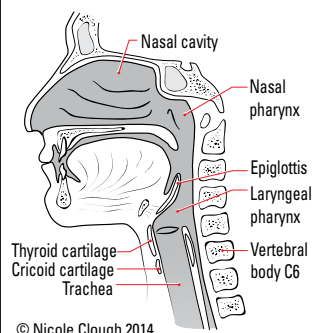
Respiratory System

- in comparison to adults, anatomical differences in infants include:
 - large head, short trachea/neck, large tongue, larynx positioned more superior and anterior, 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

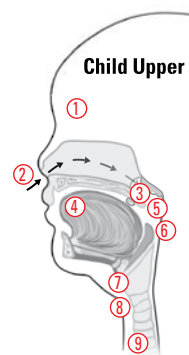
- blood volume at birth is approximately 80 mL/kg; transfusion should be started if >10% of blood volume is lost
- children have a high HR and low BP
- CO is dependent on HR, not stroke volume because of low heart wall compliance; therefore, bradycardia severe compromise in CO

Adult Upper Airway



© Nicole Clough 2014

Child Upper Airway



1. Large head
2. Newborns are obligate nasal breathers
3. Adenoid and tonsils
4. Larger tongue in proportion to mouth
5. Smaller pharynx
6. Larger and more flaccid epiglottis
7. Larynx is more superior and anterior
8. Narrowest point at cricoid cartilage
9. Trachea is more narrow and less rigid

© Andrew Q. Tran 2014 (after Leanne Chan 2011)

Figure 17. 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

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, there is an autonomic imbalance
- infant brain is 12% of body weight and receives 34% of CO (adult: 2% body weight and 14% CO)

Glucose Maintenance

- infants <1 yr 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

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 ETCO_2 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

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



Signs of Malignant Hyperthermia

- Unexplained rise in ETCO_2
- Increase in minute ventilation
- Tachycardia
- Rigidity
- Hyperthermia (late sign)

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 ETCO₂ 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 Ca²⁺ 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 ETCO₂, 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% O₂
 Hyperventilate
 Dantrolene 2.5 mg/kg every 5 min
 Bicarbonate
 Glucose and insulin
 IV fluids; cool patient to 38°C
 Fluid output; consider furosemide
 Fast Heart [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 as it will cause rebound neuromuscular blockade once cholinesterase inhibitor effect is terminated)

Appendices

Difficult Tracheal Intubation in Unconscious Patient

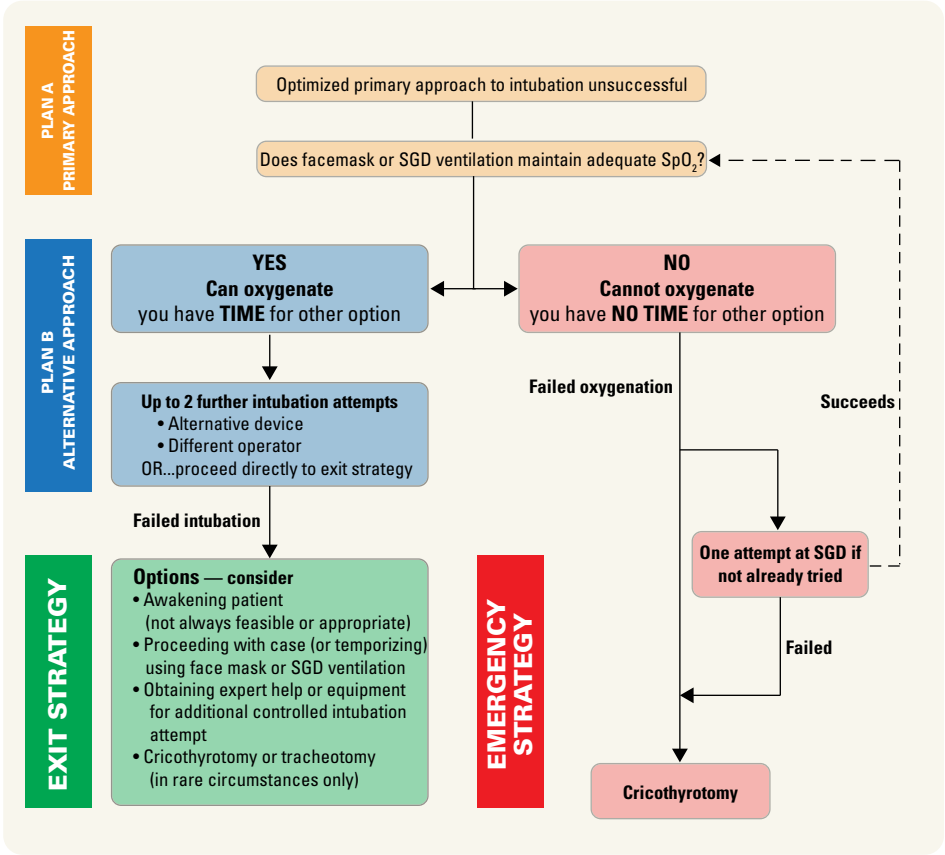


Figure 18. Difficult tracheal intubation encountered in the unconscious patient
SGD = supraglottic device

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

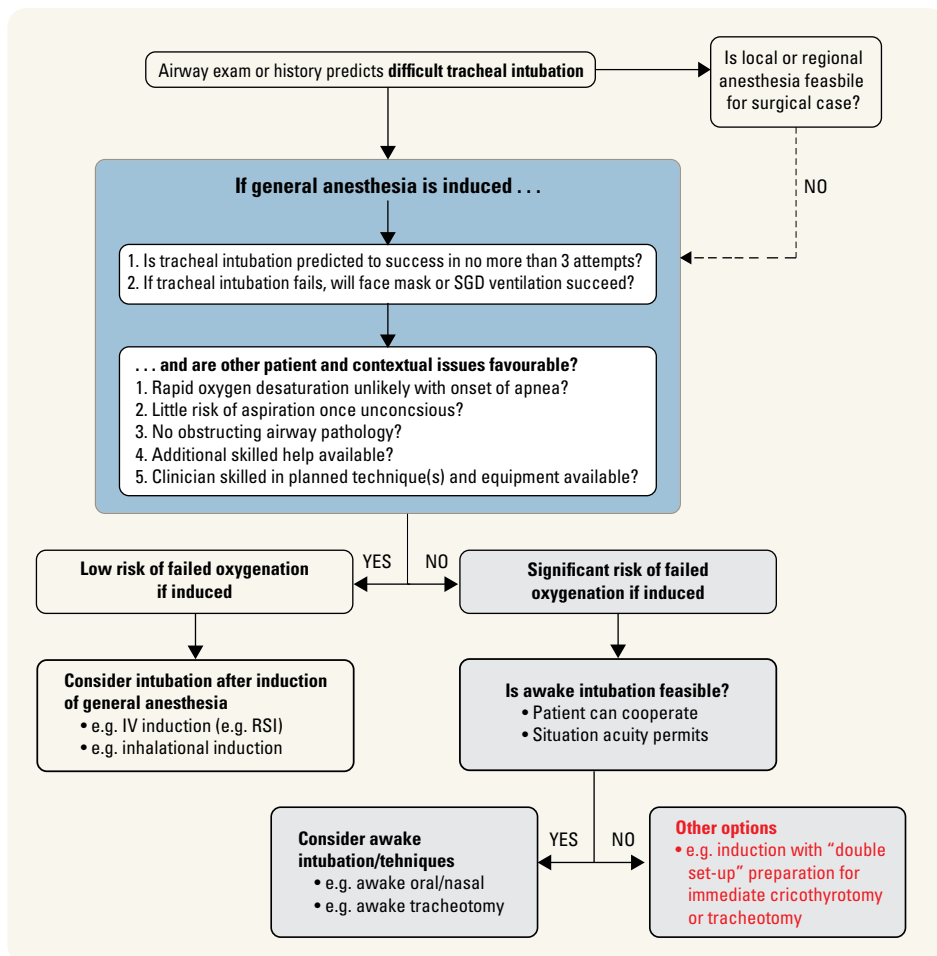


Figure 19. Anticipated difficult tracheal intubation

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

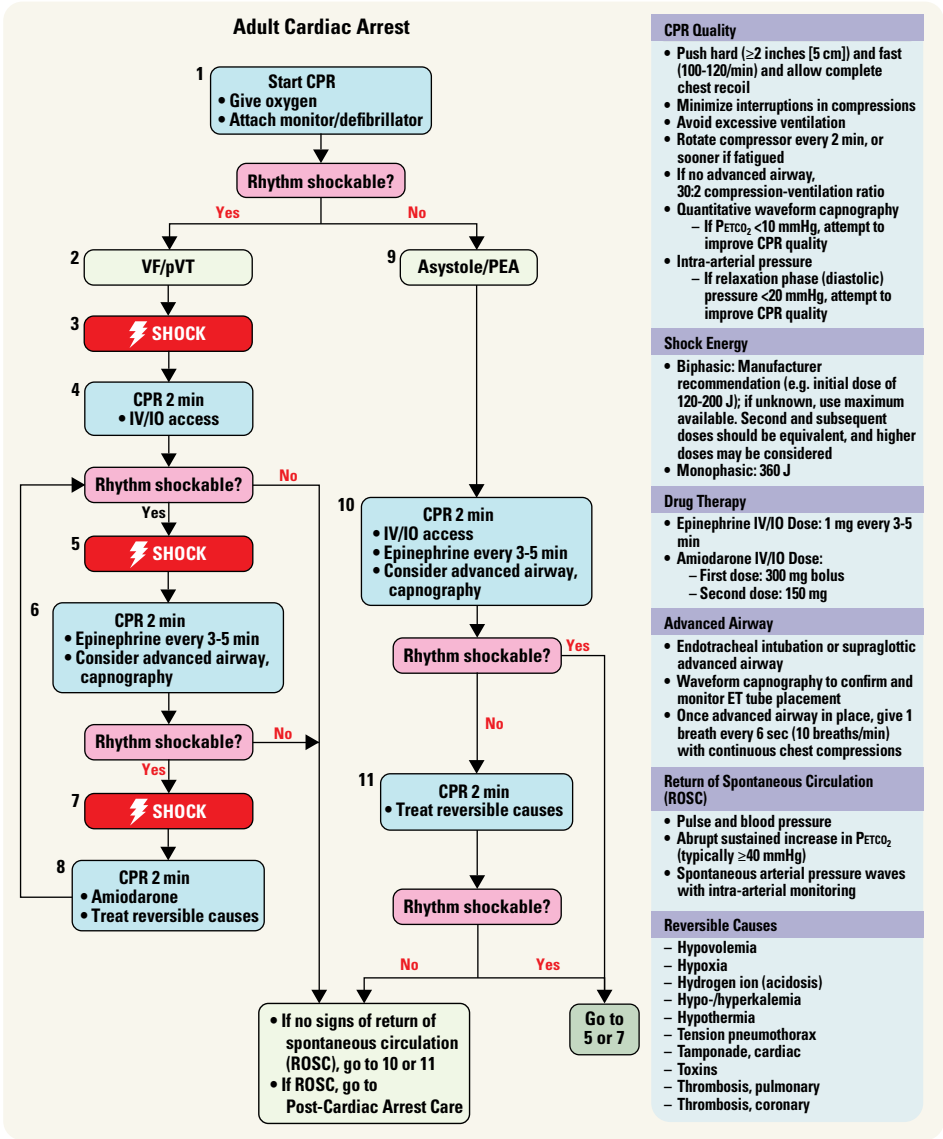


Figure 20. Adult cardiac arrest algorithm

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.

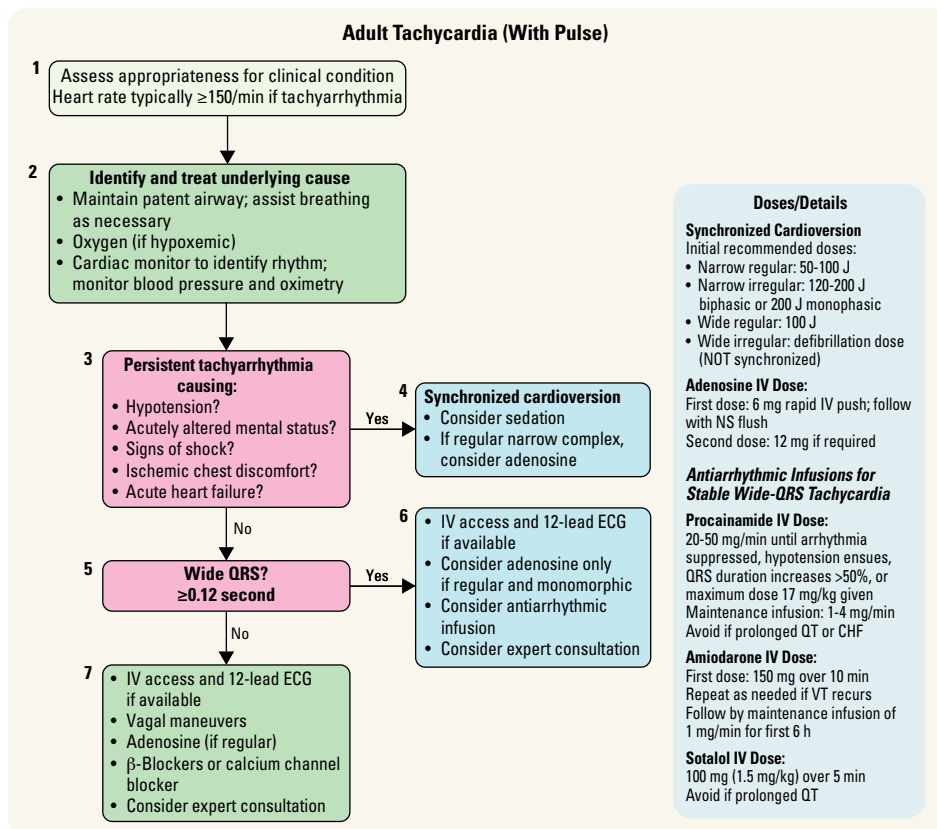


Figure 21. Adult tachycardia algorithm

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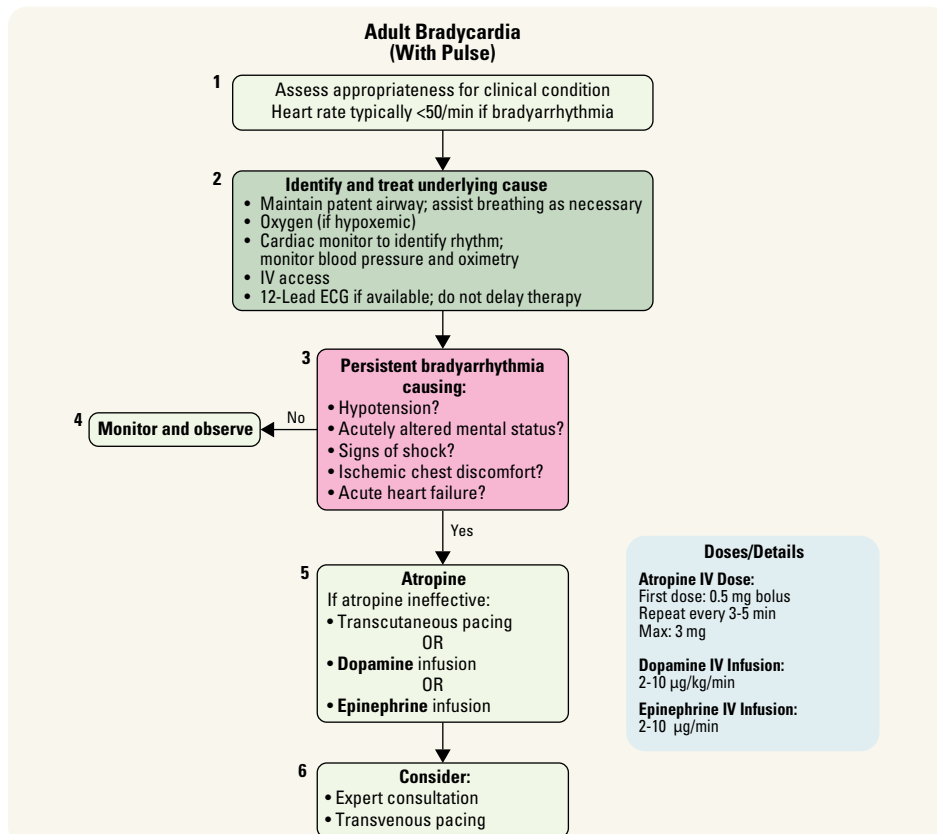


Figure 22. Adult bradycardia algorithm

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