

Anesthesiology Clerkship Rotation Handbook

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What is Anesthesia?

General anesthetics have been performed since 1846 when Morton demonstrated the first anesthetic (using ether) in Boston, USA. Local anesthetics arrived later, the first being scientifically described in 1884 when the local anesthetic effects of cocaine were described by Koller (a friend of Sigmund Freud).

General anesthesia is described as a reversible state of unconsciousness with inability to respond to a standardized surgical stimulus. In modern anesthetic practice this involves the triad of: unconsciousness, analgesia, muscle relaxation.

The exact mechanism involved is still unknown. Inhalational agents create the same endpoint as intravenous agents yet their chemical structure is different. Many theories have been postulated including:

1. Lipid solubility
2. Aqueous solubility
3. Axonal membrane action
4. Reduce synaptic transmission
5. Depression of postsynaptic response
6. Change in protein conformity
7. Alteration in sensory-motor modulation
8. Multisite expansion theory

What is known is that anesthetic agents do expand various sites with hydrophobic properties, affecting lipids, proteins, and synaptic transmission. Their action causes alteration in release of neurotransmitters especially in the thalamus.

Risks of Anesthesia

There is no such thing as a “minor anesthetic”. Death totally attributable to anesthesia is very rare ~1: 850,000, and contributory risk is 7: 10,000 (NCEPOD 1987). Factors involved with death included failure to apply knowledge, lack of care, lack of expertise, lack of knowledge, drug effect, equipment failure, failure of organization, fatigue. Six most common clinical causes of death associated with anesthesia as noted in a French survey are equipment failure, intubation difficulties, aspiration of gastric contents, post-operative respiratory depression, anaphylactoid shock, and cardiac arrest. Death rate is influenced by anesthesiologist/anesthetic factors (GA vs regional; drugs used), surgeon /surgery type (vascular, thoracic, emergency), patient factors (e.g. pediatric, obstetric, geriatric) and ASA physical status (see below).

Morbidity associated with anesthesia includes damage to teeth, nerve damage, extradural foreign bodies (e.g. catheter tips), spinal cord damage, burns, extravasations of injected drugs, awareness, aspiration.

<u>ASA*</u>	<u>% mortality**</u>	<u>ASA patient characteristics</u>
I	0.06%	Healthy patient
II	0.4%	Mild systemic disease with no limitation of function
III	4.3%	Severe systemic disease limiting functional ability
IV	23.4%	Severe systemic disease always a threat to life
V	50.7%	Moribund patient not likely to live 24 hours with or without surgery

*ASA = American Society of Anesthesiologists

** = Stats from Marx 1973

How do we know when someone is anesthetized?

Unfortunately we don't always know if they are. A variety of tests and monitors are used to try and ensure patients are asleep and unaware. These include:

1. Clinical signs: sweating, tachycardia, hypertension, reactive dilated pupils, lacrimation, frowning, limb movement. The more signs present the more likely that the patient is only lightly anesthetized.
2. Population parameters: MAC (minimum alveolar concentration) of inhalation agents tries to ensure an adequate concentration of anesthetic agent is given.
MAC: minimum concentration required to prevent 50% population from responding to a standard surgical incision over the abdomen.
MIR is the infusion rate equivalent
3. Instrumental: skin conductance (measures altered resistance with sweating), EEG analysis (e.g. BIS monitor), evoked potentials, lower esophageal sphincter contractility, isolated forearm

Awareness under anesthetic is fortunately very rare but the effects can be devastating. Usually it is not complete awareness throughout the whole operation that is reported but one brief period (e.g. during intubation). Auditory awareness is the most common form since hearing is the last sense to be anesthetized and the first to recover.

It is important to realize that vigilance is the mainstay to avoiding awareness since 70% are due to faulty anesthetic technique, 20% machine failure, 10% miscellaneous. Highest incidence occurs in obstetrics, cardiac, emergency trauma.

Preoperative Assessment

Purpose of the pre-operative visit:

1. Establish rapport with patient
2. Obtain a full history and physical examination including allergies, current medications, past anesthetic history, family anesthetic history
3. Review any investigations done; order any special investigations deemed necessary
4. Assess the risk of anesthesia and surgery and postpone or cancel if necessary
5. Prescribe any premedications
6. Formulate an anesthetic plan and discuss with patient

Pre-existing conditions of significance to anesthesia include:

Cardiovascular: Hypertension
 Ischaemic heart disease/myocardial infarction
 Valve lesions
 Pacemakers

1. Hypertension: ascertain the “normal” blood pressure range for an individual by recording serial BP readings in a non-stressful environment over time (review hospital records). Aim to have **diastolic < 110**. Gross hypertensive responses (with associated ST changes) are more common when diastolic pressure is greater than 110 and can result in an increased risk of peri-operative myocardial morbidity.

During anesthesia the aim is to maintain BP in range “normal” for that patient since end organs will be autoregulating around these pressures. Effectively treated hypertension results in less labile blood pressure i.e fewer incidences of hyper and hypo tension.

2. Ischemic heart disease: indicated by associated conditions such as diabetes, heavy smoking, hyperlipidaemia and symptoms of angina, intermittent claudication, TIA’s. Disease is usually more wide spread than symptoms may indicate. Patients’ condition should be optimized prior to surgery and may include pre-operative angioplasty, coronary artery stenting or even coronary artery bypass grafting. Investigations to consider include EKG, exercise stress testing, dobutamine stress echo, thallium perfusion scan, coronary angiography.

Previous history of myocardial infarction will significantly affect the risk of surgery depending on the time delay between infarction and surgery.

Time delay since MI	Risk of reinfarction
MI < 3 months	5.8 - 37%
MI 4-6 months	2.3 - 26%
MI > 6 months	1 - 6%

Risk of infarction extends into the postoperative period for up to 1 week with highest incidence of ischaemia occurring 3 days post op. Patients should be given supplemental oxygen for these three days post op especially if prescribed opioid analgesics.

Other scoring systems have been devised to give an assessment of cardiac risk for non-cardiac surgery. The best known is the Goldman index.

<u>Risk Factor</u>	<u>Points</u>
Third heart sound or raised JVP	11
MI in last 6 months	10
Rhythm other than sinus/PAC's	7
Abdominal/thoracic/aortic operation	3
Age > 70	5
Significant aortic stenosis	3
Emergency procedure	4
Poor condition as defined by:	3
PaO ₂ < 56mmHg (8kPa)	
PaCO ₂ > 45mmHg (6.5kPa)	
K < 3mmol/l	
HCO ₃ < 20mmol/l	
Urea > 7.5 mmol/l	
Creatinine > 270µmol/l	
SGOT abnormal	
Chronic liver disease	

5 points or less - cardiac mortality 0.2%

6-25 points – cardiac mortality 2%

>25 points – cardiac mortality 56%

3. Valve lesions: associated with a relatively fixed cardiac output.

Stenotic lesions: unable to compensate for changes in filling pressure of the right heart therefore avoid hypovolaemia, decreasing systemic vascular resistance, tachycardia.

Regurgitant lesions: more able to compensate for alterations in preload and afterload but only if there is good ventricular function. Should aim to avoid bradycardia and increases in afterload.

4. Pacemakers: ensure that they are functioning and have been checked recently. Find out what kind it is and whether on demand or not. Unipolar diathermy can be detected by the pacemaker as an arrhythmia causing it to malfunction. Try and use either bipolar diathermy or if not keep the diathermy plate away from the pacemaker and use only in short bursts. A magnet should be immediately available in the room to convert the pacemaker to fixed rate if necessary. Post operatively the pacemaker may need rechecking and reprogramming.

Respiratory: Chronic obstructive pulmonary disease
 Reactive airways disease
 Smoking
 Infection

1. COPD/Asthma: These should be optimized prior to surgery. Patients with these conditions may have problems associated with cough, bronchospasm, pneumothoraces, infection, and postoperative respiratory insufficiency. Secretions are affected by anesthesia causing them to thicken and be retained. This increases the risk of hypoxia and infection post-operatively.

Recent or ongoing respiratory infections increase airway reactivity and secretions. The risk of difficulty with intubation and ventilation, and worsening infection post operatively mean that an elective case should be cancelled until symptoms have resolved. Two weeks after resolution of symptoms is recommended.

Investigations should be considered if symptoms of cough, dyspnoea, sputum, infection, smoking are elicited or medical condition of asthma, COPD, intrathoracic tumour. Signs of wheeze, ronchi, peripheral/ central cyanosis, bronchial breathing also need further investigation. These include:

- Arterial blood gases
- Chest X-Ray
- Pulmonary function tests

Indicators of potential problems post-operatively include:

- FEV₁ < 75% predicted or < 2 litres
- FEV₁/FVC < 65% predicted
- MVV < 50% predicted
- Arterial pCO₂ >45 mmHg

Arterial pO₂ does not accurately predict outcome post operatively.

2. Smoking: common cause of cardiovascular and respiratory problems.

These include:

- CVS- increases myocardial oxygen requirements
 - coronary vasoconstriction
 - decreased oxygen carrying capabilities (~15% carboxyhemoglobin)
 - increased risk of DVT
- Resp- impaired ciliary motility (increased sputum retention)
 - reduced cellular immunity (increased infection risk)
 - increased airway reactivity (increase in risk of bronchospasm)

Encourage patients to stop smoking for 24 hours to decrease the carbon monoxide levels and reduce the effects of nicotine. 6-8 weeks are needed to return ciliary and immunological functions back to normal.

Endocrine: Diabetes mellitus
 Thyroid disease

1. Diabetes mellitus: Assessment includes usual blood sugar control, treatment regime used, presence of end-organ damage.

Aim is for blood glucose levels between 6 and 10 mmol/litre. Hypoglycaemia should be avoided as should ketoacidosis and hyperosmolar states. These should be treated prior to surgery.

Insulin dependent patients should be treated preoperatively with intermittent insulin or insulin/glucose infusion. Tablet and diet controlled diabetics should be treated by withholding oral medications alongside glucose.

All diabetics should be first on an operating list to lessen the period of preoperative starvation. A blood glucose level taken within the last hour should also be available.

Presence of complications of diabetes should be fully assessed and optimized prior to surgery.

2. Thyroid disease:

- a) Hypothyroidism – features of interest to anesthesia include:
 - i. Decreased metabolic rate with associated obesity
 - ii. Bradycardia, increased risk of myocardial ischemia
 - iii. Increased risk of atherosclerosis and pericardial effusion
 - iv. Hypothermia
 - v. Enlarged tongue making intubation more difficult
 - vi. Polyneuropathy, anemia
 - vii. Decreased drug metabolism
- b) Hyperthyroidism – features of interest to anesthesia include:
 - i. Tachycardia, increased cardiac output, arrhythmias esp. AF
 - ii. Abnormal glucose tolerance
 - iii. Thyrotoxic myopathy causing proximal muscle weakness
 - iv. Gland enlargement causing tracheal deviation, tracheal compression, tracheal collapse (with gland removal), SVC obstruction
 - v. Risk of thyrotoxic crisis with patients having partial thyroidectomy whilst still thyrotoxic. Untreated, coma and death occur.

All patients with thyroid disease should be in a euthyroid state prior to surgery. Careful assessment of the airway and trachea is required and thoracic inlet x-rays obtained. Ideally a CT scan of the trachea should be sought. Difficulty with intubation should be suspected.

Thyroid surgery can result in damage to the recurrent laryngeal nerve causing postoperative hoarseness if unilateral, and inspiratory stridor or acute respiratory obstruction if bilateral.

Thyrotoxic crisis should be treated with carbimazole, β blockade, and potassium iodide along with oxygen therapy, active cooling, sedation, and rehydration. Occasionally re-intubation and ventilation in the ICU is required.

Renal disease: patients with renal insufficiency/failure present special problems:

- a) Altered drug pharmacodynamics and pharmacokinetics due to altered protein binding (\downarrow albumin, acidosis, uraemia) and decreased or absent renal excretion.
- b) Fluid and electrolyte imbalance – Hypovolaemia (dialysis)
 - Hypervolaemia
 - Metabolic acidosis/ respiratory alkalosis
 - Hyperkalaemia
 - Hypermagnesaemia
 - Hypocalcaemia
- c) Medical conditions associated with uraemia –
 - Hypertension
 - Cardiomegaly/CHF
 - Pericardial effusion
 - Pulmonary oedema
 - Pulmonary effusion
 - ARDS
 - Depressed immunity
 - Poor wound healing
 - Coagulopathies
- d) Anaemia – normochromic, normocytic

Fluid and electrolyte abnormalities should be corrected preoperatively.

Regional anesthesia is ideal but only if there is no underlying coagulopathy.

GA should involve drugs not reliant on renal excretion and not nephrotoxic (renal insufficiency, transplant patients). Fluid management should be carefully monitored (arterial line, CVP) and **avoid succinylcholine** (hyperkalaemic response). Postoperative analgesia should ideally be with an epidural (if no contraindications) or PCA fentanyl. Morphine and demerol have active metabolites which are renally excreted and should be avoided if possible. If a-v fistulae are present these must be padded and protected – do not use this arm for non-invasive blood pressure monitoring or for siting an intravenous.

Nervous system:

Hereditary conditions
Intracranial tumours/Head injuries
Spinal injuries

- 1. Hereditary conditions:** Malignant hyperthermia
Succinylcholine apnea

Malignant hyperthermia – autosomal dominant inheritance with variable penetrance.

Incidence ~ 1: 15,000 (children) and ~1:50,000 (adults). Abnormality of calcium flux in the sarcoplasmic reticulum results in high levels of intracellular calcium.

Triggering agents are the volatile agents and succinylcholine. Can occasionally be triggered by stress outside of operating room environment.

Anesthesia of these patients involves avoidance of these agents and using an anesthetic machine that does not have traces of volatile agents within it. These patients should be first on the list and a machine flushed clear for 30 minutes and new circuit tubing attached.

If a reaction occurs it is characterized by:

Profound muscle contraction

Massive increase in metabolism with uncoupling of oxidative phosphorylation

Increased carbon dioxide production

Increased oxygen requirements

Increased body temperature by 2°C per hour

Blood gases show: hypoxia, hypercarbia, acidosis, hyperkalaemia

Myoglobinuria and renal failure

Mortality if untreated is 70% and 10% treated. Treatment is supportive with active cooling, hyperventilation and bicarbonate therapy. Definitive treatment is with dantrolene 1mg/kg intravenously up to 10mg/kg. Treatment may need to be repeated.

Succinylcholine apnea – inherited atypical pseudocholinesterases (~1:3000)

Succinylcholine is effectively two acetylcholine molecules attached together. It requires cholinesterase for metabolism. If an atypical enzyme or low concentration of enzyme is present this results in prolonged duration of action; hours or days rather than 5 minutes. If known about avoidance of succinylcholine is recommended although if rapid securement of the airway is the primary goal then it may still be used accepting the long duration of action. Prolonged action can also occur if patients have low plasma levels of cholinesterase (e.g. pregnancy, chronic liver disease).

If prolonged effect occurs then it will mean prolonged ventilation will be necessary with sedation until its effect has worn off. If prolonged ventilation is contra-indicated e.g. severe respiratory disease may consider treating with fresh frozen plasma accepting the risk of giving blood products.

2. Intracranial tumors/head injury:

Anesthesia is aimed at maintaining good cerebral perfusion in the presence of raised ICP (intracranial pressure).

Remember:

$$CPP = MAP - (ICP + CVP)$$

Raised ICP can be made better or worse by anesthesia. Aim to maintain a good mean arterial pressure and employ techniques to decrease ICP and CVP. Remember over-treatment of hypertension in the face of raised ICP will worsen cerebral perfusion and increase the risk of cerebral hypoxia.

Maneuvers that increase ICP include:

coughing, straining, obstructing venous drainage from the brain, hypercarbia, positive pressure ventilation

Anesthetic agents influence ICP:

- ↑ICP – volatile agents including nitrous oxide (cause cerebral vasodilatation)
ketamine
- ↓ICP – propofol, pentothal (barbiturate) have cerebral protection properties
(decrease brain metabolism and promote cerebral vasoconstriction)

PaCO₂ should be kept in the low normal range. Hypoventilation results in hypercarbia that causes cerebrovasodilatation. This then causes an increase in ICP. Hyperventilating patients to reduce CO₂ will give vasoconstriction but only for a limited time. The CSF will recalibrate to this new CO₂ level and any vasoconstriction present will be abolished. Diuretics (mannitol, lasix) and steroids (for tumours) are medical therapies aimed at decreasing ICP. Mechanical removal of CSF via a catheter can also be used (surgically placed).

When assessing a patient who has intracranial pathology to decide whether they require intubation the **Glasgow coma score** is used. Score ranges from 3-15 and measured on best verbal, motor and eye response to standard stimulus of voice command and knuckle pressure over sternum (pain).

Eye opening: spontaneous (4): to speech (3): to pain (2): absent (1).

Motor response: obeys commands (6): localizes to pain (5): withdraws to pain (4): abnormal flexion (3): extension (2): no response (1).

Verbal response: orientated (5): confused (4): inappropriate words (3): incomprehensible sounds (2): no verbal response (1).

Scores of 8 or less indicate need for ventilation. Trends are just as important as absolute values.

3. Spinal injuries: Unstable fractures Spinal cord damage

Unstable fractures – these can occur throughout the vertebral column. Those of most interest to anesthesiologists are the cervical spine injuries. Normally at intubation there is flexion of the lower cervical spine and extension of the upper cervical spine. This position aligns the larynx with the oropharynx so allowing easy visualization of the vocal cords. Patients with unstable c-spine fractures may need to be intubated. In these cases NO movement of the c-spine should be allowed (hard collar or manual inline stabilization), neurological assessment is carried out before intubation, and intubation is usually done awake with an intubating bronchoscope. Neurological assessment is then repeated post intubation to ensure no damage to the cord has occurred during the procedure.

Spinal cord damage – cause problems for the anesthesiologist both in the acute phase and chronically.

Acutely, spinal cord damage results in spinal shock. This causes a sudden loss of sympathetic tone with preservation of parasympathetic tone and is characterized by hypotension, bradycardia, arrhythmias. Treatment should be aggressive with vasopressors and fluids.

Chronically, hyperreflexia with uncontrolled sympathetic discharge occurs. Effect occurs in 80% patients with lesions above T6. Response causes severe hypertension and coronary vasoconstriction. It can occur during anesthesia and can be blocked with α -antagonists, ganglion blocking agents, deep anesthesia, spinal/epidural anesthesia at a level above that of the stimulus.

- Denervated muscle can develop excess numbers of acetylcholine receptors over the surface of the affected muscle as well as alterations at the neuromuscular junction. This causes an exaggerated release of potassium ions when succinylcholine is used and results in a hyperkalemic cardiac arrest. A similar effect can be seen with burns and crush injuries. Succinylcholine is contraindicated in these patients.

Musculoskeletal system: Rheumatoid arthritis
 Myopathies

1. Rheumatoid arthritis: systemic connective tissue disease affecting 1% adult males and 3% adult females. Problems of significance to anesthesia include: airway management, respiratory complications, cardiovascular complications, hematological/neurological/renal effects, drug therapies.

Airway – laxity of the atlanto-axial joint ligaments with erosion of the odontoid peg can cause subluxation during flexion with compression of the spinal cord. 25% patients have cervical spine instability BUT only 7% have symptoms. Always get extension and flexion cervical spine x-rays before surgery. A gap of more than 3mm between the odontoid peg and the posterior arch of the axis is diagnostic of subluxation. The lower cervical spine can become fused leading to fixed flexion deformity. Other airway joints involved include the cricoarytenoids (hoarseness, stridor, airway obstruction), temporomandibular joints (\downarrow mouth opening). Airway involvement may result in these patients being difficult to intubate and needing awake fiberoptic intubation.

Respiratory – diffuse infiltration with fibrosis of localized rheumatoid nodules. Pleural effusions may be present. Involvement of the costovertebral joints results in restriction to ventilation.

Cardiovascular – asymptomatic pericarditis (35%), rarely tamponade, conduction defects, valvular lesions (aortic regurgitation), generalized vasculitis.

Haematological - normocytic normochromic anemia, iron deficiency anemia, Felty's syndrome

Neurological – peripheral neuropathies, spinal cord lesions, mononeuritis multiplex, carpal tunnel syndrome

Renal – nephritis, amyloidosis

Drug therapies – NSAID'S (gastric erosion, renal impairment), steroids (suppress pituitary-adrenal response to stress), gold and penicillamine (↓ platelets, ↓ granulocytes, nephropathy, nephrotic syndrome).

2. Myopathies:

Myasthenia gravis causes fatiguable muscle weakness due to failure of neuromuscular transmission (↓ functioning post synaptic acetylcholine receptors at the neuromuscular junction). Problems concerning anesthesia include sensitivity to non-depolarising muscle relaxants (e.g. rocuronium) but usually a normal response to succinyl choline. Their muscle weakness can increase the risk of aspiration and also make it difficult to wean these patients off the ventilator post operatively (may need admission to ICU).

Myotonic syndromes (e.g. dystrophia myotonica) result in delayed muscle relaxation. Problems for anesthesia include increased sensitivity to non-depolarising muscle relaxants, myotonic response to succinyl choline (avoid), increased sensitivity to opioids, barbiturates, and volatile agents. Admission to ICU may be needed due to prolonged recovery of muscle function and prolonged sedative effect of the anesthetic agents.

Advanced age: These patients may present with problems related to both ageing body systems and an increased prevalence of specific diseases.

CVS: ↓ vessel elasticity, ↑ SVR, systemic hypertension, conduction defects (may need pacing), ↓ cardiac output by 3% per decade, ↓ ability to increase heart rate, increased incidence of IHD, CHF, valvular heart disease, PVD.

Resp: ↓ compliance, FEV₁ and FVC, vital capacity, inspiratory reserve, reduced response to hypoxia and hypercarbia and decreased protective reflexes. COPD and emphysema more common.

CNS: ↓ neuronal density (30% by 80 yrs), ↓ cerebral blood flow and brain transmitters. Increased sensitivity to anesthetic agents, sedatives, opioids, and local anesthetics. Autonomic neuropathy may be present. Increased incidence of CVA, dementia, parkinsonism, depression, deafness, poor vision.

Renal: ↓ renal blood flow, GFR, concentrating ability (1% loss in function per yr after 30 yrs). Reduced renal clearance of drugs, raised blood urea but stable blood creatinine. BPH, prostate Ca, bladder tumours more common.

Hepatic: ↓ hepatic blood flow and drug clearance (1% loss of function/yr after 30 yr).

Endocrine: ↑ adipose tissue with ↓ muscle bulk and total body water. BMR ↓ by 1%/yr after 30yr and impaired thermoregulation.

Pharmacology: Altered drug absorption, protein binding, metabolism and excretion with added problem that one third of patients over 75yr are usually on 3 or more medications chronically.

Airway Assessment

Done at the preoperative visit examination of the airway attempts to identify those patients in whom intubation +/- ventilation may be a problem. Ideally the test used should be both sensitive and specific for predicting difficulty. However, no single test used is ideal.

Incidence of difficulty is low ~1:65; failure to intubate even lower ~1:2000 patients.

Causes of difficult intubation:

Congenital: Pierre Robin, craniofacial dystocies

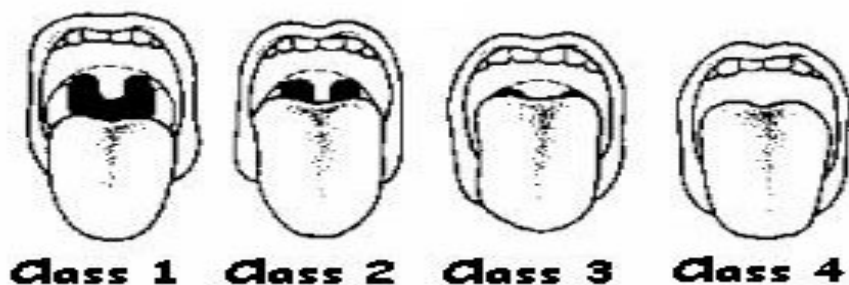
Anatomical: variants of normal e.g. prominent teeth, obesity, pregnancy

Acquired: trismus, soft tissue swelling, scarring, malignancy, infection

Predicting difficult intubation:

Mallampati classification:

- 1) soft palate, faucial pillars, uvula all visible
- 2) soft palate, faucial pillars and base of uvula visible
- 3) soft palate only visible*
- 4) hard palate only visible*



Thyromental distance: greater than 6.5cm between the thyroid cartilage and mental process of the mandible.

Ability to prognath:

- 1) subluxation of bottom incisors in front of top incisors possible
- 2) bottom incisors in line with upper incisors*
- 3) bottom incisors remain posterior to upper incisors*

Extension of the upper cervical spine:

Patients should be able to extend the upper cervical spine greater than 90°. If extension is at 90° then some difficulty may be experienced. If patient is unable to extend to 90° greatly increased difficulty with intubation will be experienced.

*= increased difficulty with intubation

Wilson classification: gives an overall score which combines the above measurements

	0	1	2
Weight	<90 kg	90-110	>110
Head and neck movement	>90	~90	<90
Jaw movement (incisor gap)	>5	<5	<5
Subluxation	>0	0	<0
Receding mandible	N	Mod	Severe
Buck teeth	N	Mod	Severe

SCORE > 4 GET HELP

A summation score using a combination of any two of the above will increase the chance of identifying a difficult airway; the more tests used the greater the accuracy. It should be remembered though that a “normal” examination can still result in a difficult intubation. It is these patients which give the most trouble simply because they were unexpected.

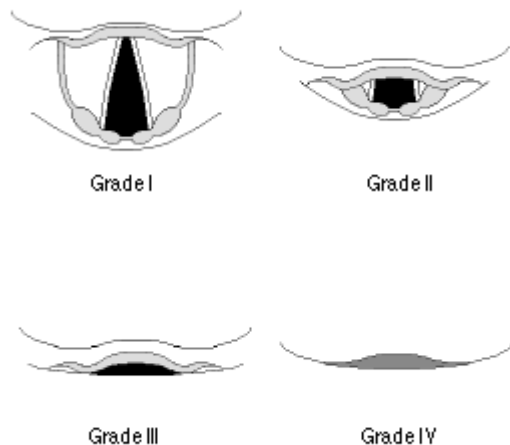
Difficult mask ventilation: Unable to maintain SaO₂ >90% despite 100%O₂, positive pressure ventilation and starting SaO₂>90%.

Characteristics associated with difficult mask ventilation include:

- Age>55yrs
- BMI>26
- Beard
- Absent teeth
- History of snoring

Difficult intubation: More than three attempts at laryngoscopy or greater than 10 minutes to properly insert an endotracheal tube using conventional laryngoscopy (assumes experienced anesthesiologist and optimum patient positioning).

Incidence = 1-18%



Cormack and Lehane classification of the larynx via direct laryngoscopy.
Grades III and IV are associated with increased difficulty with intubation.

Predicted difficult intubation:

REMEMBER: Patients do not die from failure to intubate. They die from lack of oxygen. If you feel you may be unable to ventilate **do not** paralyze.

Expert help should be sought.

Variety of techniques can be used. These include:

- 1) **awake** fiberoptic intubation
- 2) **awake** retrograde intubation
- 3) **awake** intubation through a laryngeal mask
- 4) **awake** visualization using a McCoy blade or Bullard laryngoscope
- 5) **awake** intubation with lighted stylet
- 6) **awake** tracheostomy

If the patient is unsuitable for awake intubation then attempt can be made using inhalational anesthesia and intubating with the patient deeply anesthetized **but** breathing spontaneously.

Unpredicted difficult intubation:

These are the patients that give trouble.

These patients are anesthetized and paralysed and require firstly confirmation of ability to ventilate and then attempt at intubation.

If ventilation is possible then the risk to the patient decreases since the anesthesiologist can support ventilation and keep the patient oxygenated and anesthetized until the muscle relaxant wears off and the patient breathes spontaneously.

The view at laryngoscopy is usually a Grade III i.e. epiglottis only is visible. Rarely a grade IV occurs i.e. unable to recognize anything. These are rare since we can usually predict them.

Airway adjuncts can be employed to increase the success at intubation. These include:

- Styleted tube
- Gum elastic bougie
- McCoy blade
- Light wand
- Laryngeal mask/ Intubating LMA
- Bullard laryngoscope
- Combitube
- Fibreoptic intubating bronchoscope

With each attempt at intubation the anesthesiologist should be trying to change something in order to increase the chance of success. Remember multiple attempts increase the risk of trauma to the airway. Maximum number of attempts at laryngoscopy is 3. Always ventilate between attempts to maintain oxygenation.

To act as a guide there are two airway algorithms available to aid management. These are seen overleaf. One is published by the American Society of Anesthesiologists (ASA) and the other by the Canadian Anesthesiologist Society (CAS).

Premedication

Ideal premedication should be easy to administer, reliable onset, with no side effects.

Specific aims of premedication include:

Amnesia – e.g. benzodiazepines can give both antegrade and retrograde amnesia. Useful in anxious patients but effect can be unpredictable.

Anxiolysis – the preoperative visit is more useful than sedatives but consider using benzodiazepines.

Antacid – H2 blockers decreased gastric acid production, maxeran increase gastric emptying, sodium citrate neutralizes any gastric acid present.

Antiemetic - usually given intraoperatively rather than preoperatively.

Analgesic – Tylenol and an NSAID are commonly used to give preemptive analgesia.

Antisialagogue – desirable when ketamine is used since increased salivation can be a problem, for awake fiberoptic intubations, and oral surgery procedures.

Autonomic actions – β blockade helps attenuate the hypertensive response to laryngoscopy and intubation and give myocardial protection against ischaemic events intraoperatively. Atropine or glycopyrrolate may be administered for prevention of bradycardias caused by vagal response to surgical stimulus.

Allergy prophylaxis - atopic patients should be pretreated with H1 antagonists for 24 hours preop and H2 antagonists 1-2 hours preop.

Other prophylaxis - DVT prevention, steroid cover if on systemic steroids in last 6 months. Infection risk e.g. endocarditis prevention.

Concurrent medications should also be reviewed to ensure necessary cardiac meds/ asthma therapy, diabetic management etc. is continued appropriately preoperatively.

Anesthetic equipment and monitoring

Anesthetic equipment can be categorized into:

Supply of gases

- pipeline (oxygen, nitrous oxide, air)
- cylinders (oxygen, nitrous oxide, air)

Anesthetic machine

- flowmeters (measure flow of gases from pipeline/cylinders)
- vaporizers (anesthetic agents e.g. isoflurane, sevoflurane)
- breathing circuit (transfer anesthetic gases from machine to patient)
- ventilator (acts as a “bag squeezer” for the anesthetist)
- suctioning (for removal of oral secretions/blood /vomit)

Airway equipment

- tracheal tubes (variety of sizes available)
- laryngoscopes (usually blade size 3: females, size 4: males)
- facemask (adult sizes of 3, 4, 5)
- oral airways (adult sizes 8cm, 9cm, 10cm)
- filters (bacterial/viral filter plus humidifies and warms inspiratory gases)
- airway adjuncts e.g. stilette, bougie, laryngeal mask

Monitors

- non-invasive blood pressure
- ECG
- pulse oximetry
- capnography (CO2 monitor)
- oxygen analyzer (essential)
- temperature probe
- nerve stimulator
- anesthetic agent analyzer

Specialized monitors

- arterial line (invasive blood pressure)
- central venous line (cvp monitoring)
- pulmonary artery flotation catheter (monitors function of right and left side of the heart)
- BIS monitor (depth of anesthesia)
- trans esophageal echocardiography

Accessory equipment

- blood/fluid warmers
- air warmer blanket

All of the above are available in every OR. The anesthetic machine is checked every morning to ensure that it is working properly. This check helps prevent patients from being harmed by either faulty machinery or faulty delivery of gases (especially oxygen). The oxygen analyzer is essential since this is the ONLY way of ensuring that what you believe to be coming out of the pipeline or cylinder is actually oxygen. Fatalities occurred prior to their routine use.

Other specialized equipment may also be needed which are kept in a specific area of the operating suite. These include the Difficult Airway Cart, equipment for patients with malignant hyperthermia or latex allergy, equipment needed to place double-lumen tubes (tubes which are used in thoracic surgery to allow lung separation and one lung ventilation).

Central Venous Lines

Indications and Placement:

Involves cannulation of a vein in the thorax via a peripheral vein. It is performed for:

- a) vascular access e.g. dialysis, TPN, infusion of irritant or potent drugs, rapid volume resuscitation
- b) measurement of central venous pressure
- c) cardiac catheterization, pulmonary artery catheterization, and transvenous pacing.

May be performed at various sites including:

Internal jugular vein: easy to perform and reliable.

May cause pneumothorax, damage to carotid artery, brachial plexus, phrenic nerve, thoracic duct (on left), sympathetic chain.

External jugular vein: easy to perform since it is a very superficial vein and so clearly visible.

Relatively safe in patients who have abnormal coagulation. Can be more difficult to thread the catheter through the junction with the subclavian vein. A J- shaped wire helps.

Subclavian vein: more convenient and comfortable for long-term use.

Less chance of correct placement.

Greater risk of pneumothorax or haemothorax.

May damage subclavian artery and unable to apply direct pressure to stop bleeding.

Vein held open by surrounding tissues even with severe circulatory collapse.

Arm veins: minimal risk of serious complications.

Threading of a “long-line” can be difficult especially the cephalic vein due to valves at the junction with the axillary vein. Abducting the arm helps.

50% chance of correct placement.

CVP trace and factors affecting it.

The normal venous tracing has three positive waves (a, c, v) and two negative deflections (x, y). They represent the following:

- ‘A’ wave – due to right atrial contraction and begins before the first heart sound. It is absent in atrial fibrillation and inconsistent in various heart blocks. Large ‘a’ waves associated with obstruction to atrial emptying (tricuspid stenosis, RVH, pulmonary stenosis, pulmonary hypertension).
- ‘C’ wave - due to bulging of the tricuspid valve into right atrium at onset of ventricular contraction. Occurs after first heart sound and the QRS complex.
- ‘X’ descent - due to atrial relaxation and downward displacement of the tricuspid valve during ventricular systole. It is absent in tricuspid regurgitation and replaced by a large V wave.
- ‘V’ wave - due to filling of the right atrium with a closed tricuspid valve.
- ‘Y’ descent - due to opening of the tricuspid valve and blood flowing into the right ventricle.

Central venous pressure represents the pressure within the right atrium and great veins of the thorax and is usually measured via manometry or transducer. It is usually measured with the patient lying flat and is measured in cmH₂O above a point level with the right atrium e.g. mid axillary line. Normally 0-8cmH₂O and is usually measured at end expiration. The catheter tip should lie in the superior vena cava above the pericardial reflection to decrease the risk of arrhythmias and cardiac tamponade should erosion occur.

Factors causing an increase in CVP:

- raised intrathoracic pressure e.g. IPPV, coughing, pneumothorax, hemothorax, mediasinal emphysema. CVP normally rises during expiration with spontaneous ventilation.
- Impaired cardiac function e.g. outlet obstruction, cardiac failure, cardiac tamponade. N.B. CVP is a measurement of right heart function. It may be normal in left ventricular failure and pulmonary oedema, and raised in right heart failure with normal left ventricular function.
- Circulatory overload
- Venospasm
- Superior vena caval obstruction (normal venous waveform may be lost).
- Pulmonary emboli (single, multiple, fat, air, particulate matter).
- Pulmonary artery hypertension
- COPD, Cor Pulmonale.
- Increased intraperitoneal pressures e.g. postoperative ileus.
- Artifacts, e.g. plugged catheter tip, tip in right ventricle or pulmonary artery, catheter misdirected into small branch.

Factors causing a decrease in CVP:

- reduced venous return e.g. hypovolaemia, vasodilatation.
- Reduced intrathoracic pressure, e.g. inspiration during spontaneous ventilation.

Complications

These can be summarized as:

- Arterial puncture
- Pneumothorax
- Haemothorax
- Chylothorax (thoracic duct damaged)
- Hydro/TPN thorax
- Hydro/hemomediastinum
- Hydro/hemopericardium
- Arrhythmias
- Malposition
- Air embolism
- Nerve injury
- Infection
- Upper airway obstruction due to hematoma formation

Pulmonary Artery Catheters (reference only)

Indications and Placement:

Performed using flow-directed, balloon-tipped catheters. The catheters may have some of the following features:

- 70cm long with marks every 10cm
- channels:
 - distal opening at the tip
 - proximal (30cm from tip)
 - inflation channel for balloon tip (1-1.5cc air)
 - connections to a thermistor a few cm from tip for dilutional outputs
 - fiberoptic bundles for continuous oximetry
- facilities for pacing, Doppler imaging, continuous output monitoring.

Indicated for preoperative use and within the ICU for:

- investigating cardiac shunts
- in patients where the right heart function does not correlate with left heart function, e.g. LVF or infarction, severe bundle branch block, pulmonary hypertension, cardiac tamponade, constrictive pericarditis, valvular heart disease.
- Monitoring mixed venous O₂ saturation as a continuous indicator of cardiac output and tissue perfusion

There are no outcome studies which show improvement in morbidity, mortality or length of hospital stay due to insertion of a PAFC. Hence there are no absolute indications for their insertion. However there are accepted complications associated with their use and each patient must therefore have an assessment of the risks vs. benefits of having one used.

Contraindications to use include:

- lack of operator skill
- presence of a transvenous pacemaker
- artificial tricuspid or pulmonary valve
- heart block (can use the PAFC for pacing though)
- coagulopathy is a relative contraindication

Venous access is via central venous cannulation, most typically the right internal jugular vein is used.

Pressure trace, calculations made, and factors affecting outputs.

Information obtained includes:

- mixed venous, right atrial and ventricular gas tensions and O₂ sats. Allows estimation of cardiac shunts. Continuous monitoring of venous O₂ saturation is possible.
- Measurement of right atrial and ventricular pressures, pulmonary artery pressure and pulmonary capillary wedge pressure. These are usually measured at end-systole and end-expiration.
- Measurement of right ventricular ejection fraction
- Cardiac output measurement via thermo or dye dilution or continuous methods.

Derived data includes:

- systemic vascular resistance
- pulmonary vascular resistance
- cardiac index
- stroke volume and index.

Certain criteria are needed when calculating these values to ensure their accuracy. Those needed to calculate a true wedge pressure include:

- PAWP less than or equal to the PA diastolic pressure, and less than the MPAP
- Characteristic waveform
- Fast flushing of catheter (quick elevation of waveform pressure that quickly returns to baseline; shows no blockage in catheter).
- Blood gas sample from distal tip with balloon inflated shows high O₂ sat indicating alveolar capillary gas sample.
- Transducer at level of right atrium, catheter placed in zone three where both arterial and venous pressures are greater than alveolar pressure ensuring a continuous column of blood

Values should be treated with caution in:

- LVF when LVEDP > PCWP.
- Mitral valve disease; in stenosis PCWP > LVEDP and in regurgitation large 'v' waves interfere with the waveform
- Raised intrathoracic pressure, e.g. PEEP when LVEDP > PCWP.
- Non-compliant left ventricle, LVEDP > PCWP
- Aortic regurgitation, LVEDP > PCWP
- Gradients can increase in tachycardia and increased pulmonary resistance

Calculations: (Reference only)

Cardiac output (CO) = (l/min)	heart rate x stroke volume
Cardiac index (CI) = (l/min/m ²)	$\frac{\text{cardiac output}}{\text{body surface area (BSA)}}$
SVR = (dyne.s/cm ⁵)	$\frac{\text{MAP} - \text{CVP} \times 79.9}{\text{CO}}$
SVRI = (dyne.s/cm/m ²)	$\frac{\text{SVR}}{\text{BSA}}$
PVR = (dyne.s/cm ⁵)	$\frac{\text{MPAP} - \text{PAWP} \times 79.9}{\text{CO}}$
O ₂ Delivery (DO ₂ I) = (ml/min/m ²)	CI x CaO ₂ x 10
O ₂ Consumption = (ml/min/m ²)	CI x (CaO ₂ - CvO ₂) x 10
O ₂ Extraction Ratio =	$\frac{C(a-v)O_2}{CaO_2}$
LVSWI =	$\frac{1.36 (\text{MAP} - \text{LAP})}{100} \times \text{stroke index}$

100

Complications:

These can be summarized as:

- as for central venous cannulation
- higher tendency for arrhythmias
- infection
- catheter knotting
- damage to valves, myocardium
- pulmonary artery rupture or damage
- pulmonary infarction

Arterial lines

Indications and placement:

Used for direct arterial blood pressure measurement and allows ready access for repeat gas analysis. The beat-to-beat trend allows prompt identification of changes in blood pressure and intravascular volume which would be missed using non-invasive techniques. Peripheral cannulation gives higher peak systolic pressure than more central cannulation but has fewer complication rates. Allen's test is usually performed before placement but has doubtful value. Continuous slow flushing with 1 – 3ml/hr is preferable to intermittent injection to maintain patency.

System consist of:

- a short, non-tapered, stiff cannula (reduces resonance) of appropriate size e.g. 20g for radial and brachial arteries.
- a rigid connecting tube of 1.5 – 3mm diameter of maximum length of 120cm
- one stopcock per line
- transducer with highest frequency response
- avoidance of kinks, blood clots, air bubbles in line which cause damping

Complications:

These include:

- air embolism
- thrombosis
- infection
- hemorrhage (line disconnection)
- peripheral and central embolisation of clot/debris

Waveform and information derived

The shape of the pressure waveform recorded directly in the aorta differs from the smaller arteries; the peak systolic pressure and pulse pressure increase and the aortic notch becomes more apparent the more peripherally you go. The aorta and large arteries are distended by the stroke volume during its ejection whilst during diastole the elastic recoil maintains diastolic blood flow. Smaller vessels are less compliant and so less distensible. This causes their peak pressures to be higher and they travel faster. In the elderly decrease compliance within the aorta results in similarly higher peak pressures.

Abnormal waveforms:

- *anacrotic*; aortic stenosis
- *collapsing*; hyperdynamic circulation (pregnancy, fever, anemia, hyperthyroid, AV fistula)
- *bisferiens*; aortic stenosis + aortic regurgitation
- *alternans*; LVF
- excessive damping or resonance

Information from the normal waveform include:

- arterial BP
- stroke volume and cardiac output
- myocardial contractility
- hypovolaemia
- outflow resistance

PULSE OXIMETRY

Technology:

Relies on the differences in absorption of light of oxyhemoglobin and deoxyhemoglobin.

Arterial blood oxygen saturation is calculated depending on the relative amounts of these hemoglobins present in the arterial system.

Deoxyhemoglobin absorbs more light in the 600 – 750nm range (red band)

Oxyhemoglobin absorbs more light in the 850 – 1000nm range (infrared band).

The pulse oximeter probe contains (at least) two emitting diodes that specifically emit light within the red and infrared ranges; typically 660 and 940nm.

Light emitted from the probe transmits through the patient's digit and sensors built into the same probe.

Light is emitted in a series of pulses several hundred times per second over the peak and trough of a pulse waveform.

At the trough light is absorbed by arterial, capillary, and venous blood as well as surrounding tissues.

At the peak additional light is absorbed from the increased volume of arterial blood. The probe thus calculated the relative ratios of absorption at the peak and trough to give both saturation and a pulse volume.

Accuracy:

Accurate to within 5% of in vivo sampling from 70% - 100% but calibration is done using healthy volunteers and so inaccurate at low saturation levels.

Response Time:

Within 5 – 8 secs for an initial reading and desaturation times lag by 7 – 72 seconds depending on probe location (worse on a toe).

Inaccuracies:

Low amplitude states: hypovolemia, cardiac arrest, hypotension, hypothermia, peripheral vasoconstriction, tourniquet

Arrhythmias

Anemia

Dyshemoglobins: higher than normal reading with carboxyhemoglobin, lower than normal reading with methemoglobin, ? Sickle Cell disease

Dyes and pigments: methylene blue, indigo carmine cause abnormally low readings

Ambient light

Skin pigments rarely in deeply pigmented skin since light cannot penetrate through

Nail varnish if darkly colored, multi layered; acrylic nails particular problem

Electrocautery

Motion artifact

Complications:

Skin erosions and blistering, ischemia, latex allergy trigger

Anesthetic agents: intravenous/inhalation

Intravenous agents: Pentothal
 Propofol
 Ketamine

1. Pentothal: straw coloured agent. Short acting barbiturate which has rapid onset (one arm-brain circulation). Effect lasts approximately 5-15 minutes after which patients wake up due to the agent being washed out of the brain and redistributed to muscle/fat. Patients complain of a “garlic taste” as they become anesthetized. It is very alkaline and can result in tissue necrosis if injected extravascularly. Precipitation occurs if injected into an artery causing limb ischaemia.

Induction dose : 3 - 7mg/kg intravenously

Systemic effects: CVS - ↓ blood pressure with compensatory ↑ heart rate
 CNS – powerful anticonvulsant
 Resp – respiratory depressant→ apnoea

Contraindications: allergy to barbiturates, porphyria,

2. Propofol: white coloured agent in a soya bean/egg phosphatide base prepared as a 1% solution. Phenol derivative which has rapid onset (one arm-brain circulation). Effect lasts approximately 10 minutes, quick redistribution resulting in rapid awakening. Can be painful on injection – add 10–20mg lidocaine to try and prevent this. This is the drug of choice for day case surgery since it gives very little “hangover” effect.

Induction dose: 2-2.5mg/kg intravenously (can also be used as a maintenance infusion throughout the surgery instead of inhalational agents, and as a sedative on ICU).

Systemic effects: CVS - ↓↓ blood pressure with ↓ heart rate
 CNS -? anticonvulsant, ?proconvulsant
 Resp – respiratory depression→ apnoea
 GI tract – antiemetic

Contraindications: allergy to eggs.

3. Ketamine: clear coloured agent. Produces “dissociative anesthesia”. Onset in 1min and lasts for 5-10min when given intravenously. May cause pain on injection.

Induction dose: 1-2mg/kg intravenously. Can also be given im/orally/intrathecally/extradurally. Used as analgesic in dose 0.25-0.5mg/kg iv.

Systemic effects: CNS – powerful analgesic, ↑ ICP, amnesic, visual and auditory hallucinations

CVS - ↑ blood pressure, ↑ heart rate, ↑ cardiac output, sensitizes the myocardium to catecholamines (risk of arrhythmias)

Resp – maintains upper airway tone and reflexes, ↑ salivation, bronchodilation.

GI tract - ↑ risk of nausea and vomiting

Contraindications: raised ICP, glaucoma, hypertension, angina.

Inhalational agents:

Halothane – induction and maintenance

Isoflurane - maintenance

Sevoflurane – induction and maintenance

Desflurane – maintenance

Nitrous oxide – adjunct inhalational agent

The anesthetic agents available have varying potencies i.e. varying ability to cause anesthesia. This is described as the **MAC** (or ED 50) of an agent when given with oxygen to a patient.

MAC: minimal alveolar concentration of an anesthetic needed to prevent 50% of patients from moving in response to a standard surgical incision on the abdomen.

MAC for the above agents are:

Halothane	0.75%
Isoflurane	1.15%
Sevoflurane	2.0%
Desflurane	6.0%
Nitrous oxide	105%

MAC can be altered by a variety of factors including: age, body temperature, hyper-hypometabolic states, premedications.

1. Halothane: available since the 1950’s this is the oldest agent in use in Kingston.

It is the cheapest agent available but also has the slowest onset and offset. This is because it is highly soluble and so distributes not only to the blood and brain (causing anesthesia) but to muscle and fat too. It is 20% metabolized (occurs in the liver).

Systemic effects – CVS: ↓ blood pressure, ↓ heart rate, sensitizes myocardium to catecholamines, depresses myocardial contraction

Resp: non-irritant, smells nice, bronchodilatation

GI tract: risk of hepatitis from its metabolites

2. Isoflurane: FDA approved in 1980 it is mainly used for maintenance of anesthesia but has been used occasionally in the ICU as a sedative. It is less than 1% metabolized.

Systemic effects – CVS: potent vasodilator, ↓ blood pressure due to ↓ systemic vascular resistance, ↑ heart rate

Resp: pungent, more irritant than halothane, bronchodilatation

GI tract: very small risk of hepatitis (fewer metabolites produced)

3. Sevoflurane: widely used in Japan this has only recently been approved in Canada and the US. It is 3% metabolized. It is mainly used for rapid inhalational induction of children and occasionally adults.

Systemic effects – CVS: minimal effect on blood pressure/heart rate/sensitization of myocardium to catecholamines.

Resp: smells nice, non-irritant, bronchodilatation

4. Desflurane: most recent volatile agent available. This is the least soluble volatile agent available so its time to induction and emergence from anesthesia is rapid. It is more irritating to the airway than sevoflurane so is not suitable for inhalational induction (causes coughing). It is the least metabolized of the agents ~0.02%.

Systemic effects – CVS: ↑ heart rate with sudden changes in concentration of agent, slight ↓ blood pressure

Resp: irritant, pungent smell, bronchodilatation

GI Tract: increased risk of nausea and vomiting.

5. Nitrous oxide: first discovered in 1772 it is the oldest anesthetic agent but also the weakest (MAC 105%). As such it is used as an adjunct to the other inhalational agents. Since MAC is additive when mixtures are used, the addition of nitrous oxide means that less of another agent is needed. This means that there is less risk of side effects. It is not metabolized but excreted unchanged. Also used in a 50:50 mixture for inhalational analgesia in labour.

Systemic effects – CVS: mild myocardial depressive effect, ↑ systemic vascular resistance, ↓ cardiac output

Resp: respiratory depression

CNS: analgesic

GI Tract: nausea and vomiting

Contraindications: Due to its high solubility compared with nitrogen there is a tendency for nitrous oxide to enter gas filled spaces causing expansion. As such it is contraindicated in pneumothorax, bowel obstruction, middle ear surgery. Should also be avoided in first trimester pregnancy (↑miscarriage), bone marrow suppression (affects both vit. B12 production and methionine synthetase needed in production of DNA).

Intraoperative analgesics

The mainstays of analgesics in the peri-operative period are NSAIDs, Tylenol, and opioids. These are supplemented with local anesthetics given either by the surgeon (wound infiltration, topicalization, nerve block) or the anesthesiologist (nerve block, extradurally, intrathecally).

** If the rectal route is to be used when a patient is anesthetized the patient must give consent beforehand otherwise it constitutes assault**

NSAID's: Usually given preoperatively via the oral or rectal route as part of a premedication or rectally in the OR. Care should be taken when administering to patients with renal insufficiency, gastric ulcers, concurrent anticoagulant therapy, and reactive airways disease. They are a co analgesic with action at the peripheral portion of the pain pathway. They act by reducing the activity of cyclooxygenase so decreasing the production of prostaglandins, prostacyclins, and thromboxane which sensitize pain receptors.

Tylenol (acetaminophen): An analgesic and antipyretic that does not possess anti-inflammatory properties. It inhibits central prostaglandin synthesis and does not have an action on platelet function or cause gastric irritation. It is usually given orally or rectally as part of a premedication. Alternatively it is given rectally in the OR.

NB: Care should be taken in patients with impaired liver function since it is metabolized in the liver via glutathione and sulphate pathways. If these pathways become saturated metabolism results in a toxin to hepatocytes that causes centrilobular necrosis. If severe liver transplant will be necessary or death will occur.

Opioids: Also known as narcotic analgesics. They act not only on pain transmission but also on the emotional component of pain perception. This action occurs at a variety of receptor sites [μ (mu), κ (kappa), δ (delta) ϵ (epsilon)]. The various opioids act on these receptors to a greater or lesser extent. This results in variation both in action and side effect profile for each opioid.

Opioid effects include: analgesia, respiratory depression, euphoria, miosis, bradycardia, dependence, nausea, pruritis, hypothermia, hallucinations, antitussive, histamine release (bronchospasm, hypotension), urinary retention (intrathecal/extradural route).

Opioids are usually given via the intravenous, intrathecal or extradural route by the anesthesiologist. The most common ones used in the OR are morphine and fentanyl.

1. Morphine: Excellent analgesic. The 'gold standard' by which all other opioids are compared. Derived from poppy seeds it was first used in the 4th century BC but only isolated in 1806. Used intraoperatively to provide more prolonged analgesia that will be effective both intraoperatively and in the post-operative period. Most common analgesic ordered for post operative pain control both by the acute pain service (via PCA route) and surgical team. Care needed in patients with renal insufficiency since morphine is broken down to an active (analgesic) metabolite that is renally excreted. Patients with renal insufficiency tend to require less drug dosage and less frequently. This includes the elderly.

Dose/ route of administration: 0.1-0.15mg/kg iv/im/sc
 ~100µg intrathecal
 2.5-5mg extradural

Onset : 3-5min iv
 60-90 min im/sc

2. Fentanyl: Shorter acting synthetic opioid that has 100 times more analgesic potency than morphine. It has less effect on blood pressure and no histamine release (*cf* morphine).

Dose/route of administration: 1-50mcg/kg iv intraoperatively (higher dose in cardiac anesthesia)
 0.25-1 mcg.kg iv post operatively
 10-15mcg intrathecally
 1mcg/kg extradurally

Onset of action: 1-2 min
 Duration of action: 20-30 min

Muscle relaxants

Muscle relaxants were introduced into anesthetic practice with the advent of thoracic surgery since it wasn't possible for a patient to breath spontaneously with an open chest wound. The first drug used for this purpose was curare in 1912. Other drugs have since been developed that have a better side effect profile, quicker onset and offset, and less reliance on organ function for excretion.

Muscle relaxants are used in anesthesia to allow easier surgical access and to aid ventilation both in the OR and occasionally in ICU.

There are two types of muscle relaxants used: Depolarizing agents
 Non-depolarizing agents

The terms refer to the drug's action at the postsynaptic acetylcholine receptor.

Depolarizing agents: bind to the acetylcholine receptor, act like acetylcholine causing depolarization at the neuromuscular junction. However it stays bound to the receptor for a longer period so effectively deactivating the receptor until the drug is broken down. The drug is metabolized by plasma cholinesterase enzyme: a process that takes between 3-5 min usually).

1.Succinylcholine (structure: two acetylcholine molecules joined together).

The most commonly used drug of this type.

Dose: 1-2mg/kg iv
 Onset of action: 60 seconds
 Duration of action: 5 minutes

Side effects: myalgia pains, histamine release, transiently raised intraocular pressure, transiently raised ICP, increase in potassium levels (can be fatal in renal failure/burns/crush injuries), anaphylaxis, malignant hyperthermia, prolonged action if have abnormal pseudocholinesterase enzyme, bradycardia.

There is no reversal agent available to counteract the effects of succinylcholine. Effect wears off when the drug has been metabolized.

Despite all of these side effects there is no other muscle relaxant available yet which provides such rapid onset and offset. Until one is discovered succinylcholine will continue to be used.

Non Depolarizing agents: bind to the acetylcholine receptor in a competitive manner with acetylcholine. They do not cause activation of the postsynaptic receptors (do not see muscle twitching). Effect of the drug wears off as it is metabolized and leaves the receptor surface. It is possible to reverse the effects of these drugs by giving an anticholinesterase (e.g. edrophonium, neostigmine). These drugs increase the concentration of acetylcholine at the neuromuscular junction enabling it to *competitively* remove the muscle relaxant from the receptor surface. The most common non-depolarizing drug of this type used in our institution is rocuronium. However you may also see the drugs vecuronium, cis-atracurium, and pancuronium used.

1. Rocuronium

Dose: 0.6mg/kg

Onset of action: 1 minute

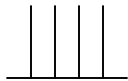
Duration of action: 20 min

Side effects: transient tachycardia with injection

When muscle relaxants are used the anesthesiologist may use a nerve stimulator to help in assessing how paralyzed a patient is during surgery and especially at the end of surgery. This allows us to ensure optimal conditions for the surgeon and helps in deciding when it is safe to reverse the effect of a muscle relaxant.

The nerve stimulator is attached to a peripheral nerve (e.g. wrist, facial) and a series of four twitches are applied over 2 seconds (train of four). Depending on whether a depolarizing relaxant or non-depolarizing relaxant has been used there will be a different pattern of twitches elicited.

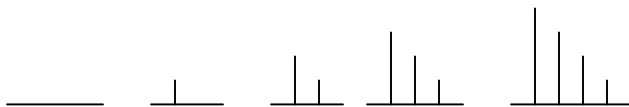
Normally: all four twitches are of equal height and strength



Depolarizing: all four twitches initially disappear (fully paralyzed) and then gradually return in strength but all the twitches are of equal strength at any test period.



Nondepolarizing: all four twitches disappear (fully paralyzed) and then gradually see twitches return but initially may only have one twitch. With time the number of twitches increases, as does the strength of contraction. You see a *fade* in strength between the first twitch and subsequent twitches. Four twitches are needed for safe reversal of muscle relaxation but the strength can be less with each twitch.

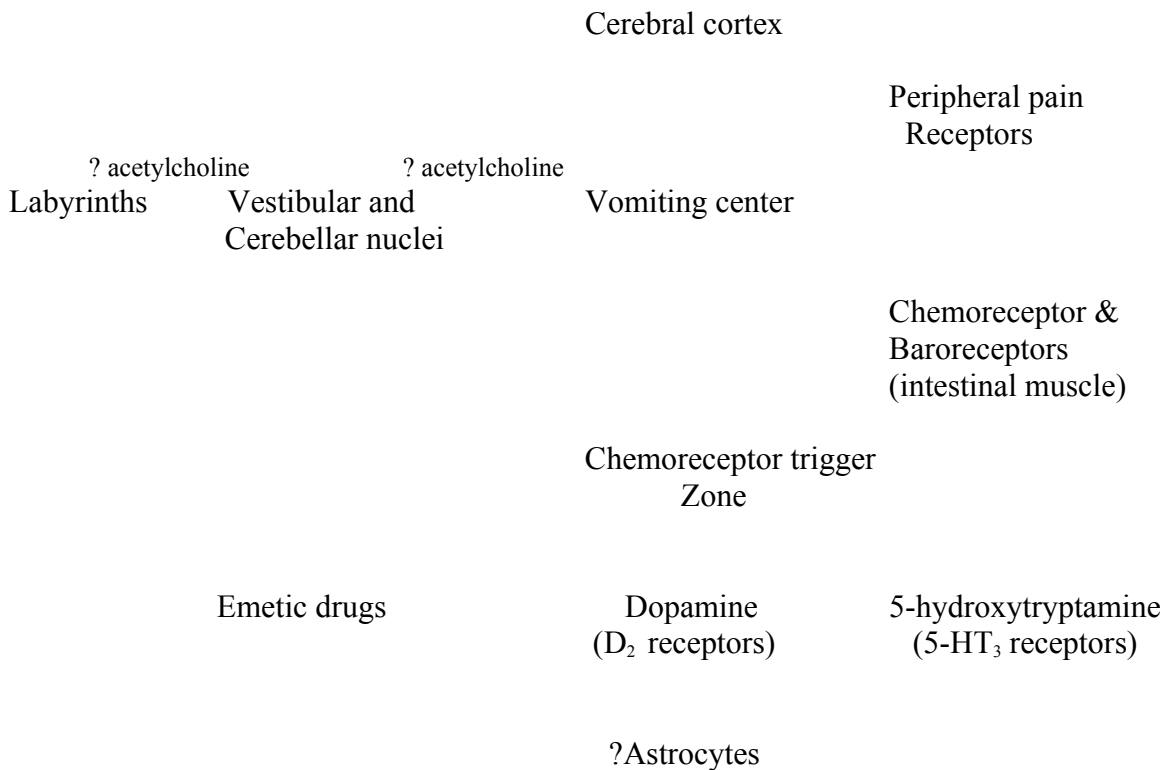


When anticholinergics are given one of their side effects is bradycardia. To prevent this atropine or glycopyrrolate is given at the same time as neostigmine (or edrophonium). Glycopyrrolate causes less tachycardia and no central confusion unlike atropine. However atropine possesses some antiemetic properties and has less antisialagogue effect.

Antiemetics

Vomiting is a reflex action involving retrograde passage of gastric contents through the mouth. It is centralized in the vomiting center located in the reticular formation of the medulla and receives afferent impulses from:

- GI tract, abdominal organs and peritoneum via the vagus nerve and sympathetic fibres
- Heart via vagus nerve
- Vestibular apparatus
- Chemoreceptor trigger zone
- Higher centers.



Causes of nausea and vomiting related to anesthesia include:

- gastric distension from facemask ventilation
- use of nitrous oxide/ neostigmine
- opioid analgesics
- gynecological/abdominal procedures
- middle ear surgery/ squint surgery
- young women esp. if menstruating at time of surgery

- anxiety
- raised ICP, hypoxaemia, hypotension
- movement of patient whilst recovering from anesthetic (go slowly around the corners with patients on stretchers!!)

Antiemetic drugs tend to be receptor specific. The main emetic in the perioperative period is opioid use and it is this, which the anesthesiologist is treating. Opioids cause nausea via dopamine receptors located in the chemoreceptor trigger zone (Area Postrema in the floor of the fourth ventricle). Effective antiemetics include: butyrophenones (droperidol, haloperidol) and phenothiazines [prochlorperazine (Stemetil)].

Other antiemetics include:

- metoclopramide (dopamine, 5HT₃ receptors plus increases gastric emptying)
- ondansetron (5-HT₃ receptors)
- Gravol (anticholinergic)

Local Anesthetic Agents

Cocaine was the first agent discovered to have local anesthetic properties (1884) and was mainly used for topical anesthesia of the cornea and nasal mucosa. It is still used today for anesthesia and vasoconstriction of the nasal mucosa for ENT procedures. Since that time other, safer agents have been discovered. Those commonly seen now include lidocaine (1947), chlorprocaine (1952), bupivacaine (1963), ropivacaine (1991).

Local anesthetics tend to be poorly soluble weak acids and comprise both hydrophilic and hydrophobic portions. Their speed of onset and duration of action is partly related to their pKa. The pKa is the pH at which a solution is half in its un-ionized, lipid soluble form and half in its ionized, water-soluble form. The pKa of the most commonly used local anesthetics are: lidocaine 7.9, bupivacaine 8.1. Hence lidocaine has a quicker onset of action than bupivacaine since its pKa is closer to tissue pH. When local anesthetic drug is injected it diffuses across the axonal membrane in its un-ionized, lipid soluble form. Its ability to do this is therefore affected by the pH of the surrounding tissues. If the tissues are acidic (e.g. infection) the local anesthetic will stay in its water-soluble form and will not be able to diffuse across the axonal membrane into the nerve. Once within the axon the local anesthetic converts to its ionized form and attaches itself to the sodium channels. Again its ability to do this is dependent on the intracellular pH. If intracellular pH is relatively alkali the local anesthetic will be ineffective, as it will remain in its unionized, lipid soluble form. Binding reversibly to these sodium channels the nerve becomes unable to depolarize and thus propagate an action potential.

They are active both peripherally and centrally on autonomic, sensory and motor fibers. The degree of blockade is dependent on the dose used; the higher the dose the more nerve fibers are affected.

1. Lidocaine:

Uses: local anesthesia, class Ib antiarrhythmic agent

Doses: Infiltration/nerve block 5mg/kg (no epinephrine), 7mg/kg (with epinephrine)

Intravenous block (Bier's block using a tourniquet) 3mg/kg (no epinephrine)

Intravenous (tachyarrhythmias) 1mg/kg over 2 min then infusion

Side effects: mainly related to plasma concentration. Has narrow therapeutic range so care in patients with cardiac/hepatic failure.

Toxicity: initially lightheadedness/tingling around mouth

- Visual/auditory disturbances
- Muscle twitching
- Cerebral excitation/convulsions
- Coma
- Respiratory arrest
- Cardiovascular collapse

Treatment includes: STOP giving any more lidocaine!

100% oxygen via facemask if conscious and breathing spontaneously

Tracheal tube placement and ventilation if unconscious

Hyperventilate to give alkalosis so decreasing CNS toxicity by keeping the drug non-ionized and unable to attach to the sodium channel

↓BP give fluid bolus +/- ephedrine 5mg iv bolus

CNS excitation give diazepam 5-10mg iv or pentothal 50mg iv

2. Bupivacaine:

Uses: local anesthetic (slower onset but more prolonged action compared to lidocaine)

Dose: 2mg/kg (no effect on dose by using epinephrine)

Side effects: same as lidocaine but has more toxic effect on the myocardium causing severe myocardial depression and cardiovascular collapse at an earlier stage than lidocaine.

This can be very difficult to treat. *Bupivacaine is contraindicated for intravenous nerve block techniques.*

Regional Anesthesia/Analgesia

Regional anesthetic techniques include epidural block and spinal (subarachnoid) block. Both can be used for intraoperative anesthesia but epidurals can also be used for intra-operative analgesia (combined with a GA) and postoperative analgesia.

1.Epidural Analgesia/Anesthesia

Involves placement of local anesthetic into the epidural (extradural) space via a catheter as either bolus dosing or for continuous infusion.

Anatomically divided into cervical, thoracic, lumbar and caudal epidurals.

Indications are:

1. Sole anesthetic technique for surgery +/- sedation
2. Intra-operative analgesia in conjunction with a general anesthetic
3. Analgesia for labor and delivery
4. Post-operative analgesia

Effects: Similar to a spinal anesthetic but comes on more gradually. The level of the block can be controlled more easily than a spinal and there is a more gradual onset of hypotension due to sympathetic blockade. The density of block and degree of muscle relaxation varies on the dose of local anesthetic given (more drug causes more muscle relaxation). There is an increased incidence of inadequate (“patchy”) block compared with spinal anesthesia.

Solutions used: Lidocaine 1-2% +/- CO₂
Bupivacaine 0.1% - 0.5% solution

The local anesthetics cause muscle relaxation, sympathetic block (causing hypotension)

Additives used: Fentanyl 25mcg/ml (intra-operative/postoperative analgesia)
Hydromorphone 10-20mcg (post operative analgesia)
Preservative free morphine 1-5mg (post operative analgesia)

Opioid side effects include nausea, vomiting, itching, urine retention, sedation, respiratory depression.

Other additives that can be used include sodium bicarbonate, carbon dioxide, and epinephrine. These speed up the onset of the block and are usually used for emergency LSCS under epidural anesthesia.

The local anesthetics and opioids can be used either singly or in combination. Combination decreases the total dose of each and helps decrease side effects. Solutions can be given either as bolus dosing or via an infusion pump. The latter is more common for post operative/labor analgesia as it gives continuous analgesia with less risk of breakthrough pain.

Contraindications include:

1. Patient refusal
2. Infection either generalized or local
3. Hypovolaemia/shock
4. Raised ICP
5. Abnormal coagulation (platelets $<100 \times 10^9$ INR >1.2)
6. Allergy to local anesthetics

Complications include:

1. Hypotension
2. Post dural puncture headache (if needle accidentally pierces the dura)
3. Transient backache for 24 hours
4. Neurological injury e.g. cord ischaemia, direct trauma to the cord, nerve root paraesthesia, arachnoiditis, meningitis, epidural abscess/hematoma
5. Infection
6. Bloody tap (needle or catheter into a blood vessel)
7. High spinal
8. Shivering
9. Anaphylaxis to local anesthetic drug (very rare)

Epidurals may be more difficult to site if patients have had previous back surgery/scoliosis but it is not a contraindication to trying. The block may not be predictable in these patients due to altered spread of the local anesthetic.

2. Spinal anesthesia

Consists of a single dose of local anesthetic injected into the subarachnoid space. Site of injection is more limited than that of epidurals and is confined to interspaces L3-4, L4-5, L5-S1. At these levels there is minimal chance of damaging the spinal cord which usually ends at L1-2 in adults.

Indications:

1. Sole anesthetic for surgery +/- sedation
2. Short acting analgesia for labor if require analgesia quickly due to rapid progress of labor (i.e. epidural onset too slow)

Effect: rapid onset (3-5 min) of profound muscle relaxation and sensory block with sympathetic nerve block also. The smaller sympathetic nerve fibres are first affected followed by sensory and then motor fibers. Usually the sensory block is two segments higher than the motor block. Sudden hypotension can occur with patients complaining of nausea.

Solutions used: Bupivacaine 0.5%

Heavy Bupivacaine 0.75% (dextrose added to make the solution heavier than csf).

Additives: fentanyl 10-20 mcg (intra-operative analgesia)
 Preservative free morphine 100mcg (postoperative analgesia)

Contraindications:

These are the same as for epidural anesthesia with the addition of patients with fixed cardiac output states (e.g. aortic stenosis). These patients are unable to compensate for any drop in blood pressure and would be at high risk of a cardiac arrest if given a spinal. Patients with active neurological disease e.g. spinal stenosis, spina bifida are also unsuitable for spinal anaesthesia.

Complications:

1. Hypotension
2. Post dural puncture headache
3. Neurological damage: direct trauma, spinal hematoma, cord ischemia secondary to severe hypotension, arachnoiditis, aseptic meningitis, infection
4. Backache
5. Anaphylaxis to local anesthetics (very rare)

Obstetrical Anesthesia

Obstetrical anesthesia is unique in that you are caring for two patients; the mother and fetus. Both patients have an impact on the conduct of anesthesia and any management plan must bear this in mind.

Physiological changes of pregnancy:

- CVS: ↑ intravascular volume
 ↑ heart rate ~15 beats/min
 ↑ cardiac output by 40%
 ↑ stroke volume ~30%
 ↓ systemic vascular resistance
 aortocaval compression (left lateral tilt needed if supine to avoid this)
- RESP: ↑ minute ventilation ~50% due to increase tidal volume
 ↓ arterial pCO₂
 ↓ FRC due to upward displacement of diaphragm
 ↑ oxygen consumption ~20%
- Airway: Venous engorgement of airway mucosa
 Edema of airway mucosa
 Worsening Mallampati score when in labor especially with ++ iv fluids

** Every pregnant patient has a difficult airway until proven otherwise. It is only easy once the tube is securely placed in the trachea. Oxygen consumption and decreased FRC make the risk of hypoxia high whilst altered anatomy can make instrumentation and visualization more challenging**

GIT: delayed gastric emptying
 ↑ gastroesophageal reflux (aspiration risk at intubation)

Coagulation: ↑ fibrinogen and factors VII, IX, X, XII predispose to thromboembolism
 Dilutional anemia

Renal: ↑ renal blood flow
 ↑ GFR ~ 40%

Pregnant patients requiring elective surgery during their pregnancy should have this delayed until at least the second trimester to avoid the theoretical teratogenic effect of anesthetic agents on the fetus. If abdominal surgery is needed there is an increased risk of spontaneous abortion.

Obstetrical analgesia was originally opposed on moral and religious grounds. However that changed following Queen Victoria's use of chloroform for analgesia in 1853. The option of regional techniques was introduced in the early 1900's but became especially popular after the 1960's.

The pain of labor varies in site and intensity depending on the stage. During the early stages pain is concentrated at the T11 and T12 dermatomes. As labor progresses pain is referred to T10 and L1 dermatomes. With distension of the pelvic floor, vagina and perineum pain is also felt through the sacral routes S2, S3, S4.

Analgesia options for labor and delivery include:

Natural (no medications) childbirth: breathing exercises
 Autohypnosis
 Acupuncture
 White noise
 TENS (see "acute pain management")

Advantages: generally safe for mother and fetus

Disadvantages: variable efficacy

Inhalational agents: Nitrous oxide: oxygen mixture
 Enflurane/Isoflurane (in low concentration with oxygen)

Advantages: on demand delivery so only receive analgesia when need it
relatively safe for mother and fetus

Disadvantages: variable efficacy (50:50 chance get some relief)
nausea and drowsiness possible in mother
neonatal depression

Parenteral agents: Narcotics (meperidine, fentanyl)
Sedatives (rarely used due to depressant effect on fetus)

Advantages: relatively good analgesics

Disadvantages: neonatal depression (max. risk 2 hours after im meperidine)
sedation, nausea, vomiting

These agents can be given either im by the nursing staff or via the iv route using a PCA pump. The latter offers more immediate analgesia and only when needed as opposed to having a slow release of narcotic giving analgesia both during and in-between contractions.

Regional analgesia: Epidural
Spinal
Combined spinal/epidural

* refer to regional section for more details of these techniques*

Epidural analgesia can be given as either a continuous infusion or as an intermittent bolus technique. It can be used for the duration of labor and if a LSCS is required the epidural can be reinforced using a higher concentration of local anesthetic. Solutions used can be either local anesthetic alone (bupivacaine 0.25 –0.5%) or a mixture of local anesthetic (0.1% bupivacaine) and opioid (fentanyl 2mcg/ml). It is the most effective analgesia technique available and can be used for the whole duration of labor and delivery.

Disadvantages include: it is an invasive technique
Side effects include hypotension, headache, itching, backache for 24 hrs post delivery, nausea, prolonged second stage of labor (arguable), risk of instrumental delivery, shivering, urinary retention.

Spinal analgesia can be used in patients who have rapidly progressing labor and require good analgesia. Its onset time is more rapid than an epidural (2 min *cf* 10 – 20 min) but it is a single shot technique only. It also recedes more quickly than an epidural (~ 1.5 hr). Local anesthetic +/- opioid are again used but a much smaller dose of each is needed.

Disadvantages include: same as for epidural but more risk of a spinal headache and hypotension, less risk of backache.

Combined spinal /epidural (CSE) has the advantages of both techniques i.e. rapid onset of analgesia and capability of continuing analgesia if labor lasts longer than the effect of

the spinal. It is technically more difficult to site and also the most invasive since two needles are used and dural puncture is necessary. It forms the basis of the 'walking epidural' promoted by some centers. Ironically most women in labor who request this form of analgesia are so exhausted by the time they receive analgesia that the last thing they want to do is walk around!!

Caesarean section

This can be performed under a regional technique or under general anesthesia. Due to the anatomical and physiological changes of pregnancy general anesthesia is considered to have more risk of morbidity and mortality to the extent that regional anaesthesia is the norm. For elective LSCS a spinal anesthetic is usually performed since it comes on more rapidly and gives a more guaranteed block *cf* epidurals. Epidurals are confined to those patients in whom a more controlled and gradual block is advantageous and where surgery may take longer to complete e.g. morbidly obese, pre-eclamptic patients. General anesthesia is seen in cases where regional anaesthesia is contraindicated or when it is necessary to deliver the baby ASAP e.g. abruption, cord prolapse, footling/frank breech, severe fetal bradycardia.

Associated problems with general anesthesia include:

1. Difficult/failed intubation
2. Gastric aspiration
3. Depression of the fetus
4. Awareness – high risk in obstetric patients since avoid opioids and use low anesthetic concentrations until baby delivered; use low anesthetic concentrations after delivery to avoid uterine relaxation and postpartum hemorrhage.

Remember: obstetric patients can have concurrent diseases as well as being pregnant; these may impact both on the mother and the fetus and potentially alter your management.

Medical conditions seen particularly in obstetrics include: pregnancy induced hypertension, preeclampsia, eclampsia, DIC, antepartum hemorrhage, placenta previa, HELLP syndrome, amniotic fluid embolism, pulmonary embolism.

Patients having multiple pregnancies (twins, triplets etc) are at increased risk of developing the above complications as well as premature delivery.

Pediatric Anesthesia: the basics

The pediatric population varies from the adult population in a variety of ways. Broken down into systems they include:

Respiratory:

Airway

Relatively large head, short neck, large tongue.

Narrow nasal passages but are obligate nose breathers.

Larynx is higher, more anterior, and narrowest at the level of the cricoid (C3/4).

Epiglottis is U-shaped and angled at 45°.

Carina is wider, and higher up at level of T2 (T4 adults).

Pulmonary

Chest wall is compliant and FRC low.

Increased V/Q mismatch.

Airway closure occurs at end expiration.

Tidal volume is relatively fixed (horizontal ribs, weak intercostals, large abdomen).

Oxygen consumption is high. Neonates 6ml/kg/min *cf* 3ml/kg/min adult.

Cardiovascular:

Blood volume at birth ~85ml/kg. Transfusion should be started if >10% of blood volume lost.

Hemoglobin at birth ~180, falling to 100 at 3 months.

Pulse rate is high and BP low (110 beats/min and 95/55 at 2 years).

Sinus arrhythmia is common.

Large percentage of heart muscle is non-contractile (60% *cf* 30% adults).

Fixed stroke volume therefore increase cardiac output by increasing heart rate

Nervous system:

Myelination incomplete during first year of life.

Increased sensitivity to non-depolarising muscle relaxants in first month.

The MAC of anesthetic agents is increased in both infants and neonates.

Narcotics readily depress the ventilatory response to CO₂.

Control of breathing is altered; increased incidence of periodic breathing and sleep apnoea.

Sympathetic response to bleeding is reduced.

Renal:

Extracellular fluid increased at birth (40% vs 30%) so volume of distribution of water-soluble drugs is increased e.g. suxamethonium.

Renal function immature reaching 80% maturity at 1 month. Neonates are unable to handle large fluid or salt loads.

Electrolyte requirements: sodium 2-3 mmol/kg/day, potassium 1-2 mmol/kg/day.

Fluid requirements calculated by:

Weight	Rate
Up to 10kg	4ml/kg/hr or 100ml/kg/day
10 – 20kg	2ml/kg/hr or 1000ml +50[wt(kg) -10]ml/kg/day
20 – 30kg	1ml/kg/hr or 1500ml +20[wt(kg) - 20]ml/kg/day

Temperature:

High surface area:body weight ratio.

Head is a large proportion of the body surface.

Large heat losses if head exposed during surgery.

Immature response to hypothermia (unable to shiver and poor vasoconstriction).

Heat production via brown fat that increases oxygen requirements.

Term infants cope with small changes in temperature but ideally all children should be in a thermoneutral environment.

The critical temperature is that below which the naked subject is unable to maintain body temperature.

The critical temperature for an adult is 6°C, whilst for a term infant it is 23°C.

Glucose homeostasis:

Children less than one year of age are at high risk of hypoglycaemia during preoperative fasting and postoperatively.

Healthy children aged 1-5 years can maintain normal glucose homeostasis after 8 hours fasting.

Respiratory Therapy

1. Oxygen Therapy: Aim: to prevent or at least minimize tissue hypoxia.

Indication:

1. When oxygen tension is less than 60 mmHg in a healthy patient. If patient has chronic lung disease may accept a lower oxygen tension before treatment.
2. Post operatively supplemental oxygen may be given if $SaO_2 < 92\%$ especially if anemic, hypotensive, septic
3. Delivery of medication (e.g. nebulized salbutamol)
4. Treatment of carbon monoxide poisoning

Causes of tissue hypoxia:

Hypoxemia: Low inspired oxygen tension
 Hypoventilation
 Poor matching of ventilated areas of lung with those areas being perfused

Impaired blood flow to tissues: Low cardiac output
 Hypotension
 Arterial occlusion

Impaired oxygen carrying capacity: Low hemoglobin concentration
 Abnormal hemoglobin (e.g. sickle cell)
 Poisoned hemoglobin (methemoglobin, carboxyhemoglobin)

Impaired oxygen utilization by tissues: cyanide poisoning

Excess oxygen utilization: Thyrotoxicosis
 Malignant hyperthermia

Delivery systems:

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

Face masks – Three types of facemask are available; open, Venturi, non-rebreathing.

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

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

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

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

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

Absorption atelectasis – Alveoli that contain 100% oxygen and have good blood flow going by can have all their oxygen taken up causing collapse of the alveolus. Just adding a small amount of nitrogen to the inspiratory mix can prevent this collapse by splinting the alveolus open since nitrogen is relatively insoluble and so very slowly absorbed across the alveolar membrane.

Hypoventilation – Occurs in COPD patients who rely on their hypoxic drive for respiration. High inspiratory oxygen concentrations will correct this hypoxia but at the same time remove the respiratory drive. These patients begin to hypoventilate and can develop critical CO₂ retention. If giving oxygen to these patients start at a low concentration and monitor therapy with regular arterial blood gas sampling.

Pulmonary toxicity – Prolonged high concentrations of oxygen result in the production of free radicals which are cytotoxic to cellular DNA, proteins and lipids. The resulting injury gives a clinical picture similar to ARDS (adult respiratory distress syndrome). The same toxicity results in bronchopulmonary dysplasia in newborn/premature babies.

Retinopathy of prematurity – Babies develop abnormal disorganized, blood vessel formation in the retina with resulting fibrosis, retinal detachment and blindness. Maintain arterial oxygen tension below 140 mmHg to try and prevent this occurring.

Other symptoms – retrosternal chest pain, coughing, severe dyspnoea, nausea, vertigo, muscle twitching, convulsions.

2. Intubation:

Usually done with patients asleep but tube may be placed with the patient awake and the airway topicalized first using local anesthetic agent. Intubation can be performed through the nose or mouth and occasionally via a tracheostomy site.

- Indications:
1. Maintain a clear airway
 2. Protect against aspiration of gastric contents
 3. Prevent/relieve upper airway obstruction (N.B. intubation will not relieve the airway obstruction associated with asthma since the obstruction is at the level of the small airways not the trachea/upper airway. Intubation is performed in these patients when respiratory failure/fatigue is present).
 4. To facilitate positive pressure ventilation
 5. To allow bronchopulmonary toilet (suctioning)

Complications of Intubation:

During Intubation:

Trauma – leaning on the eyes, corneal abrasions, injury to neck or jaw, teeth, lips, mucosa, tongue, pharynx, larynx (including vocal cord haematoma, subluxation of the arytenoids) laryngeal nerves, trachea. May cause infection, bleeding, surgical emphysema.

Hypertensive response – associated with increased sympathetic activity and tachycardia. Causes increase in ICP. Due to laryngoscopy, coughing, straining. Can be reduced by giving anti-hypertensives e.g. β blockers, hydralazine, nitroglycerine, SNP; benzodiazapines, iv lidocaine 1-2mg/kg, fentanyl 6-8 μ g/kg, alfentanil 30-50 μ g/kg or sufentanil 0.5-1 μ g/kg. All should be given 1-2 min prior to intubation.

Arrhythmias – especially if hypoxaemia or hypercarbia are present.

Laryngospasm, bronchospasm if attempted too early.

Aspiration of gastric contents.

Misplaced tube or difficult/failed intubation.

Once intubated:

Tube displacement - extubation, endobronchial intubation.

Disconnection from fresh gas flows.

Airway obstruction – sputum, blood, foreign body, compression by mouth gags, cuff herniation, patient biting down.

Tube ignition in laser surgery.

Late complications:

Nasal, oral and cord ulceration.

Nerve damage due to stretching or compression .Slow recovery.

Tracheal stenosis – due to prolonged intubation.

Sinusitis – prolonged nasal intubation.

Problems at Extubation

Supraglottic, glottic and subglottic oedema - most significant complication at extubation associated with morbidity and mortality. Mainly seen in paediatric patients but also in adults usually as an allergic response to the tube or lubricant. Use of iv steroids or racemic epinephrine nebulisers may help in these cases.

Laryngeal dysfunction – protective reflexes can be subnormal for quite a period of time post extubation and suspicion of aspiration must be considered. The longer the period of intubation the longer it takes to have the return of normal airway reflexes.

Sore throat – up to 90% incidence especially if a large tube was used, lubricant was present and the patient was female.

Vocal cord paralysis – secondary to surgical trauma of the vagus or recurrent laryngeal nerve, but also from cuff compression of the recurrent laryngeal nerve at the thyroid lamina.

Miscellaneous - laryngospasm, bronchospasm, sore jaw, dysphonia, tracheal collapse.

3. Positive pressure ventilation:

Indication

1. Inability to maintain adequate oxygenation despite maximum non-invasive therapy (O₂, physio, humidification, bronchodilators, steroids etc)
3. Fatigue secondary to increased work of breathing e.g. asthmatics
4. Therapeutic e.g. neuromuscular disorders, management of flail chest, head injury management
5. When require neuromuscular blockade
6. Thoracic surgery

Problems with positive pressure ventilation:

- CVS: decreased venous return, decreased cardiac output, increase CVP measurement
- Resp: decrease in lung compliance, alveolar airway closure, barotrauma, pneumothorax
- Renal: decreased renal perfusion, decreased GFR, decreased urine output ~40%
- GIT: ileus
- CNS: raised ICP

Ventilator settings commonly see:

- Tidal volume ~ 8-10ml/kg
- Respiratory rate ~ 8 – 14/min for adults
- Inspiratory oxygen concentration ~ 24% – 100%

Resuscitation Drugs : the basics

- Epinephrine: α and β agonist used in anaphylaxis, asystole, low cardiac output states
 Dose: 1mg iv bolus = 1ml of 1:1000 (asystolic arrest)
 0.1 mg iv bolus = 1 ml of 1:10,000 solution (anaphylaxis)
 0.01- 0.1 mcg/kg/min iv infusion
 Can be given via endotracheal tube (twice iv dose)
- Atropine: anticholinergic agent used for bradycardias, asystolic arrest
 Dose: 10 –20 mcg/kg iv/im
 Can be given via the endotracheal tube
- Ephedrine: acts indirectly causing release of norepinephrine and directly via stimulation of α and β receptors
 Dose: 5 mg iv boluses repeated as necessary (or 0.07mg/kg iv bolus)
 N.B. Tachyphylaxis occurs with large doses i.e. starts to become ineffective with repeated doses.
- Dopamine: acts on dopaminergic, β and α receptors depending on dose use
 Dose: 1 – 5mcg/kg/min iv dopaminergic on renal receptors
 5 – 10mcg/kg/min iv beta receptor effects dominate
 >15mcg/kg/min iv alpha receptor effects dominate
- NTG: Dilatation of arteries and veins (venous dilatation at low concentrations, both at higher concentrations). Used to improve coronary perfusion to subendocardium and reduce preload
 Dose: 1 – 10mcg/kg/min iv

Lidocaine: Ventricular dysrhythmias
 Dose: 1mg/kg iv bolus over 2 minutes (may be repeated x1) then 15-50mcg/kg/min iv infusion

Fluid Management

When assessing the fluid requirements of a patient intraoperatively you need to take into account their normal maintenance needs, their losses due to preoperative fasting (+/- bowel prep), and intraoperative losses (frank blood loss, evaporative losses, urine output, ng tube losses).

Maintenance fluids: On the wards usually given as 3.3% dextrose/0.3% saline +/- potassium. This solution is rarely given in the OR. Hourly need calculated on the 4:2:1 rule and deficit calculated from time of last known fluid intake. Therefore intraoperatively need to replace this AND what there requirements are for during the surgical procedure.

Weight	ml/kg/hr	~70 kg	~13kg
0 – 10 kg	4	40ml	40ml
11 – 20 kg	2	20ml	06ml
21 + kg	1	50ml	-
		Total: <u>110ml/hr</u>	<u>46ml/hr</u>

Intraoperative losses: Usually given as either normal saline or lactated Ringer's solution. Given for normal maintenance needs, evaporative loss and for blood loss initially. Crystalloid needs to be given at the rate of 3ml for every 1ml blood loss. Aim to keep urine output at least 0.5ml/kg/hr accepting that urine output can decrease due to positive pressure ventilation. Evaporative losses can be difficult to estimate and depends on the size of the surgical field, the size of the patient and ambient room temperature. Classify surgery into minor/moderate/major.

Minor (superficial e.g. appendectomy) ~ 5ml/kg/hr

Moderate (e.g. gastrectomy) ~ 10ml/kg/hr

Major (e.g. aortic aneurysm repair) ~ 15ml/kg/hr

Blood replacement: Classically aim is to maintain Hb ~100 since this is thought to have good oxygen carrying capacity and be of a viscosity to allow optimum blood flow. However opinion has changed, as the risks of blood transfusion are significant. Now a risk/benefit analysis is used to weigh up the risk of anemia vs the risk of giving blood for an individual patient. Some of the risks of giving blood have been addressed through autologous blood donation preoperatively. This system is not foolproof though and does have some infection/transfusion risks attached to it.

1. What are the infection risks to a patient if blood is given?
2. What is blood tested for?

Apart from infection other risks include:

1. Immunological reactions – Immediate hemolysis (ABO incompatibility)
 Delayed hemolysis (7 – 10 days later will manifest as renal failure)
 Reaction to platelets/WBC (fever, dyspnoea)
 Febrile reaction of unknown etiology
2. Metabolic reactions - Hyperkalemia
 Citrate toxicity
 Hypocalcemia
 Alkalosis/acidosis
3. Fluid overload

Intraoperatively keep a close eye on blood losses both in swabs and suction canisters. If considering blood replacement take into account preoperative Hb level, patients blood volume, losses up to now, replacement volume given, and blood losses expected in the near future. Transfusion is usually considered when patients have lost ~ 20% estimated blood volume. A “hemocue” level can be easily performed to confirm the degree of anemia. If significant for that individual (e.g. <100 if have significant CVS disease, <70 if healthy adult and further blood loss expected) then blood is given.

Adequacy of fluid replacement is monitored via arterial blood pressure, pulse rate and urine output. If need more accurate assessment can use invasive blood pressure monitoring via an arterial line (see fluctuation in BP with each ventilator breath if under filled), central venous pressure line (right side of the heart pressure) or a pulmonary artery flotation catheter (if have cardiac pathology where left ventricular pressures need to be calculated).

Postoperative Recovery Period

This covers the period from the end of surgery until the patient is alert and physiologically stable. During this time patients are in a designated recovery area and measurements of heart rate, blood pressure and oxygen saturation are recorded every 15 minutes. Additional intravenous analgesics/antiemetics may be required to achieve good symptom control prior to transfer to the ward.

Problems during the recovery period include:

- Resp: hypoventilation, hypercapnia, hypoxia, airway obstruction, bronchospasm, aspiration of gastric contents
 CVS: hypotension, hypertension, ischemia, arrhythmias, infarction, ongoing blood loss
 CNS: confusion, agitation, seizures, CVA
 GIT: nausea, vomiting

Metabolic: shivering, hypothermia (intra and post op), hypoglycemia, hyperglycemia, TURP syndrome, hypothyroidism, hypocalcemia, thyrotoxic crisis

Reactions secondary to anesthesia: inadequate reversal of muscle relaxation, malignant hyperthermia, dystonic reactions, emergence phenomenon, central anticholinergic syndrome, headache, sore throat

HYPOTHERMIA

Routes of heat loss: Percentages will change depending on the environment but are approximately:

Radiation ~ 40%
 Convection ~ 30%
 Evaporation ~ 20%
 Respiration ~10% (8% humidification of inspired air, 2% heating inspired air)

Causes in OR:

Patient related:

Neonates: a larger surface area: body weight ratio esp. if head exposed
 decreased ability to shiver
 poor ability to vasoconstrict
 temperature generation derived from brown fat
 risk of heat loss 2-2.5x that of adults
 critical temperature for term infant 23°C *cf* adult 6°C

Elderly: impaired thermoregulation

Anesthesia related:

Anesthetic agents impair hypothalamic thermoregulation and cause vasodilatation
 Regional anesthesia increases vasodilatation
 Decreased metabolic rate
 Muscle relaxants prevent shivering
 Loss of behavioral techniques to generate heat
 Inability of vasoconstriction or piloerection
 Infusion of cold intravenous fluids
 Inhalation of cold, dry anesthetic gases

i.e. Redistribution of blood flow from the core to the periphery, increased tendency to radiant, convection and respiratory heat loss

Surgery related:

Exposure of large areas of skin, abdominal and thoracic viscera,
 Blood loss
 Irrigation with fluids cooler than body temp

Prolonged surgery
 Cold operating room environment
 Turnover of OR atmosphere via ventilation system causing rapid air movement

i.e. Increased evaporative, convection and radiant heat loss

Effects:

Cardiovascular: Anesthetized patient: ↓HR, ↓CO, ↓BP, failure of pulse oximetry monitoring due to ↓ peripheral perfusion

Unanesthetized patient: ↑HR, ↑BP, ↑CO, ↑oxygen consumption
 ↑cutaneous vasoconstriction, failure of pulse oximetry monitoring due to ↓peripheral perfusion

ECG changes associated include: sinus bradycardia, prolonged PR, widening QRS, prolonged QT

Arrhythmias common below 28°C: nodal rhythms, PVC's, AV block, VF

Arrhythmias common below 20°C: VF, asystole

Respiratory: PaCO₂ and PaO₂ decrease 4.5% per degree decrease in temp
 Hypoxic ventilatory drive depressed
 Respiratory rate driven by hypercarbia
 ↓RR, left shift of the oxygen dissociation curve
 If not anesthetized increase in O₂ requirements 5x due to shivering
 Hypoxia possible in PACU with shivering

CNS: Sedation at 33°C, increasing impairment of awareness at 31 °C,
 LOC below 30°C
 ↓ cerebral metabolic rate 7 – 10% per degree decrease
 Flat EEG below 20°C

Renal: ↓GFR and ↓RBF by 60% at 25°C but inhibited tubular resorption and ↓ADH secretion so maintain urine output until <20°C

Hepatic: ↓ hepatic blood flow ∞ ↓CO
 ↓metabolic/excretory function

Blood: ↑viscosity 2-3% per degree decrease temp
 ↓ plasma volume (causes the ↑ hematocrit)
 Platelet aggregation, rouleau formation → resistance to blood flow
 Impaired coagulation Thrombocytopenia
 Impaired function of WBC with ↑ infection risk
 ↑ use of blood products needed perioperatively

Metabolic: ↓BMR → ↓CO₂ production and O₂ consumption
 ↓ drug metabolism
 hyperglycemia common
 pancreatitis with prolonged severe hypothermia

Prevention:

Prevention of hypothermia on transport to OR
 OR environment kept as warm as comfortable for OR team to work in
 Anesthesia gases should be warmed and humidified
 Intravenous and irrigation fluids should be warmed
 Use of warming blankets
 Minimize area of exposure of patient

Acute Pain Management

Inadequate pain relief remains one of the most serious deficiencies in postoperative patient care.

- Issues:**
1. Wide interpersonal variability in analgesia requirements.
 2. Regular scheduled doses offer benefit over “as required” doses.
 3. Rescue analgesia is essential.
 4. A multimodal approach to pain relief offers better analgesia.
 5. Pain must be assessed regularly and objectively.
 6. Prescriptions for *prn* drugs encourages cycles of recurring pain.

Routes of Administration:

Oral Perioperative gastric emptying and gut function is unreliable.
 Oral route only suitable in immediate post operative period for very minor surgery.
 Simple analgesics include antiprostinoids *e.g.* aspirin, diclofenac.

Stronger analgesics *e.g.* morphine, diladid, codeine cause delay in gastric emptying and small bowel function so absorption can be unreliable. A large ileal or first pass metabolism occurs causing decreased bioavailability. Hence the equipotent dose of oral morphine is approximately 150-200% of the parenteral dose. *Note conversion of parenteral morphine to oral morphine for dosing is an increase of 2-3 times.* These drugs are especially useful in the later postoperative period.

- Sublingual** Buprenorphine, a partial agonist of the μ opioid receptor, is given by this route. It avoids the risk of delayed gastric emptying and first pass metabolism. It is inherently safe since accidental swallowing creates reduced bioavailability. It does cause a great degree of nausea and vomiting in the first 24 hours of use along with dizziness.
- Transcutaneous** Fentanyl can be given by this route. It is mainly seen in palliative care where the slow release nature allows long term control. It is unsuitable for postoperative acute pain management where analgesia needs change daily and the depot effect of the fentanyl means sudden alterations in treatment cannot be achieved. The depot effect also means that if respiratory depression occurs simply removing the patch will not reverse the respiratory depression.
- Intramuscular** Opioid analgesics are frequently given by this route. Peak onset of action is achieved slowly so analgesia is not immediate. Peak blood levels are also likely to be less a similar dose given intravenously. Hence the gradual onset of symptoms of overdose makes this potentially safer. Disadvantages are that injections are painful, doses are fixed and doses may be too small (inadequate analgesia) or too large (side-effects). The fixed time interval allows breakthrough pain to develop and increased suffering for the patient.
- Intravenous** Intermittent doses of opioid analgesics are given perioperatively. There is rapid onset of action but these systemic fluctuations result in increased risk of side effects, especially respiratory depression. Systemic side effects are less likely if the drugs are titrated to analgesic effect. A variation on this route where the patient is in control is “patient controlled analgesia (PCA)”.
- Subcutaneous** This is an alternative route to intramuscular injection. It is especially useful in very thin, malnourished patients with little muscle bulk. Absorption is very similar to im injections.
- Rectal** Drugs given this way do not suffer from first pass metabolism and are unaffected by gastric emptying. The rectal mucosa is richly supplied with blood vessels and so predictable absorption occurs. Cultural attitudes limit its use.

- Inhalational** This route is seen mainly in emergency departments and on labour/delivery suites. Nitrous oxide/oxygen mixture (50-50) is inhaled by the patient just prior to the peak onset of the pain. Nitrous oxide is relatively insoluble in blood so limiting its absorption. This also means that it is rapidly excreted so giving short lived analgesia. Side effects include nausea, vomiting, and drowsiness.
- Local anesthetic Techniques** Use of bupivacaine for nerve blocks provides several hours of analgesia post operatively. It results in good analgesia and avoids the side effects of opioids. They may be repeated postoperatively and a catheter may be inserted to allow continuous infusion or bolus “top-ups”.
- Epidural/Spinal** Local anesthetics may be instilled into the epidural or subarachnoid space. Catheter techniques allow continuous infusion of local anesthetic/opioid solutions for up to three or four days postoperatively. Their use intraoperatively results in constricted bowel and so may improve surgical access. They provide muscle relaxation, abolish the stress response to surgery and decrease the risk of blood loss and DVT formation.
Refer to the regional section of this handout for more details.
- Cryoanalgesia** Provides long lasting nerve blocks. Usually done on intercostals nerves exposed during thoracic surgery and can provide analgesia for up to several months. The disadvantages are that the posterior ramus of the intercostals nerve is not blocked and there may be an increased risk of chronic pain at the surgical site in the long term.
- TENS** Electrodes are placed around an incision or over a dermatome where pain is perceived. Low frequency pulses of electricity stimulate the large nerve fibres which transmit to the dorsal horn cells. Acts via the gate theory of pain perception. Does not usually provide adequate analgesia post operatively but may offer use for laboring parturient or patients with chronic pain syndromes.

Patient controlled analgesia

Within any individual there is an underlying endogenous opioid level. Patients with high endogenous levels require less analgesia requirements than someone with low endogenous levels. Unfortunately predicting which patient is which isn't possible. Hence the PCA pump was developed which allows the patients to control their own analgesia within set parameters set by the attending anesthesiologist.

- Pump controls***
1. Drug choice varies between institutions with morphine being the most popular. Demerol and fentanyl can also be used.
 2. *Dose varies depending on the drug used, the patient's status, surgery performed. The correct bolus dose gives adequate analgesia with minimal side effects.*

3. *Loading dose enables good control of pain before allowing the patient to take over control. It is very important to ensure good analgesia before this handover occurs.*
4. *Lockout interval is a safety feature preventing too frequent dosages. It denies analgesia for a set period of time after a dose has been given. It should be greater than the length of time needed for maximal effect.*
5. Maximum dosage sets the maximum dose allowed in any hour.
6. Background infusion gives a constant infusion of analgesia independent of the patient dosing. It helps prevent “breakthrough” pain when sleep prevents the patient from pushing the button. It increases the risk of respiratory depression. This mode is mainly used in patients who are not opioid naïve and take significant opioid analgesics on a regular basis at home.

Use of PCA Patients need to be assessed regularly for the following:

1. pain score at rest and with movement
2. sedation
3. respiratory rate
4. pulse, blood pressure
5. nausea, vomiting, pruritis

These should be done hourly for the first 4 hours then two hourly until the PCA is discontinued.

Antiemetics and co-analgesics should be ordered to work alongside the PCA. Sedatives and additional analgesics should not be prescribed whilst a patient is on a PCA without first clearing it with the acute pain service team. The patient is the only person who should press the button.

NB: The PCA requires there to be a degree of background pain in order to work. The patient must be sufficiently competent to understand how it works. The controls are such that it may not be physically possible for the patient to press the button. PCA has been used successfully in pediatric patients, sometimes with the nurse pressing the button.

