**Anesthesia Preoperative Evaluation**

1. The overall goal of the preoperative evaluation is to reduce perioperative morbidity and mortality and alleviate patient anxiety.

2. Anesthesia preoperative history and physical
   A. Note the date and time of the interview, the planned procedure, and a description of any extraordinary circumstances regarding the anesthesia.
   B. Current medications and allergies: history of steroids, chemotherapy and herb and dietary supplements.
   C. Cigarette, alcohol, and illicit drug history, including most recent use.
   D. Anesthetic history, including specific details of any problems.
   E. Prior surgical procedures and hospitalizations.
   F. Family history, especially anesthetic problems.
      Birth and development history (pediatric cases).
   G. Obstetrical history: last menstrual period (females).
   H. Medical history; evaluation, current treatment, and degree of control.
   I. Review of systems, including general, cardiac, pulmonary, neurologic, liver, renal, gastrointestinal, endocrine, hematologic, psychiatric.
   J. History of airway problems (difficult intubation or airway disease, symptoms of temporomandibular joint disease, loose teeth, etc).
   K. Last oral intake.
   L. Physical exam, including airway evaluation (see below), current vital signs, height and body weight, baseline mental status, evaluation of heart and lungs, vascular access.
   M. Overall impression of the complexity of the patient’s medical condition, with assignment of ASA Physical Status Class (see below).
   N. Anesthetic plan (general anesthesia, regional, spinal, MAC). The anesthetic plan is based on the patient's medical status, the planned operation, and the patient’s wishes.
   O. Documentation that risks and benefits were explained to the patient.

3. Preoperative laboratory evaluation
   A. Hemoglobin: menstruating females, children less than 6 months or with suspected sickle cell disease, history of anemia, blood dyscrasia or malignancy, congenital heart disease, chronic disease states, age greater than 50 years (65 years for males), patients likely to experience large blood loss.
   B. WBC count: suspected infection or immunosuppression.
   C. Platelet count: history of abnormal bleeding or bruising, liver disease, blood dyscrasias, chemotherapy, hypersplenism.
   D. Coagulation studies: history of abnormal bleeding, anticoagulant drug therapy, liver disease, malabsorption, poor nutrition, vascular procedure.
   E. Electrolytes, blood glucose, BUN/creatinine: renal disease, adrenal or thyroid disorders, diabetes mellitus, diuretic therapy, chemotherapy.
   F. Liver function tests: patients with liver disease, history of or exposure to hepatitis, history of alcohol or drug abuse, drug therapy with agents that may affect liver function.
   G. Pregnancy test: patients for whom pregnancy might complicate the surgery, patients of uncertain status by history and/or examination.
   H. Electrocardiogram: age 50 or older, hypertension, current or past significant cardiac disease or circulatory disease, diabetes mellitus in a person age 40 or older. An EKG
showing normal results that was performed within 6 months of surgery can be used if there has been no intervening clinical event.

I. Chest x-ray: asthma or chronic obstructive pulmonary disease with change of symptoms or acute episode within the past 6 months, cardiothoracic procedures.

J. Urinalysis: genito-urologic procedures; surgeon may request to rule out infection before certain surgical procedures.

K. Cervical spine flexion/extension x-rays: patients with rheumatoid arthritis or Down’s syndrome. Routine screening in asymptomatic patients is generally not required.

L. Preoperative pulmonary function tests (PFTs)

1. There is no evidence to suggest that pulmonary function tests are useful for purposes of risk assessment or modification in patients with cigarette smoking or adequately treated brochosptic disease.

2. Candidates for preoperative PFTs

A. Patients considered for pneumonectomy.
B. Patients with moderate to severe pulmonary disease scheduled for major abdominal or thoracic surgery.
C. Patients with dyspnea at rest.
D. Patients with chest wall and spinal deformities.
E. Morbidity obese patients.
F. Patients with airway obstructive lesions.

---

**Airway Evaluation**

1. Preoperative evaluation: assessed by historical interview (ie, history of difficult intubation, sleep apnea) and physical examination and occasionally with radiographs, PFTs, and direct fiber-optic examination. The physical exam is the most important method of detecting and anticipating airway difficulties.

2. Physical exam

A. Mouth

2. Dentition: ascertain the presence of loose, cracked, or missing teeth; dental prostheses; and co-existing dental abnormalities.
3. Macroglossia: will increase diffcultly of intubation.

B. Neck/Chin

1. Anterior mandibular space (thyromental distance): the distance between the hyoid bone and the inside of the mentum (mental prominence) or between the notch of the thyroid cartilage to the mentum. An inadequate mandibular space is associated with a hyomental distance of <3 cm or a thyromental distance of <6 cm.
2. Cervical spine mobility (atlanto-occipital joint extension).

3. Airway classification

A. Mallampati classification (relates tongue size vs pharyngeal size).

   Class 1: able to visualize the soft palate, fauces, uvula, anterior and posterior tonsillar pillars.
   Class 2: able to visualize the soft palate, fauces, and uvula. The anterior and posterior tonsillar pillars are hidden by the tongue.
   Class 3: only the soft palate and base of uvula are visible.
   Class 4: only the soft palate can be seen (no uvula seen).

B. Laryngoscopic view grades
Grade 1: full view of the entire glottic opening.
Grade 2: posterior portion of the glottic opening is visible.
Grade 3: only the epiglottis is visible.
Grade 4: only soft palate is visible.

4. Predictors of difficult intubation
A. Anatomic variations: micrognathia, prognathism, large tongue, arched palate, short neck, prominent upper incisors, buckteeth, decreased jaw movement, receding mandible or anterior larynx, short stout neck.
B. Medical conditions associated with difficult intubations
1. Arthritis: patients with arthritis may have a decreased range of neck mobility.
2. Tumors: may obstruct the airway
3. Infections: of any oral structure may obstruct the airway.
4. Trauma.
5. Down’s Syndrome:
6. Obesity: massive amount of soft tissue about the head and upper trunk can impair mandibular and cervical mobility, increased incidence of sleep apnea.

ASA Physical Status Classification
The ASA (American Society of Anesthesiologists) physical status classification has been shown to generally correlate with the perioperative mortality rate (mortality rates given below).
ASA 1: a normal healthy patient (0.06-0.08%).
ASA 2: a patient with a mild systemic disease (controlled hypertension [0.27-0.4%]).
ASA 3: a patient with a severe systemic disease that limits activity (angina, COPD, prior myocardial infarction [1.8-4.3%]).
ASA 4: a patient with an incapacitating disease that is a constant threat to life (CHF, renal failure [7.8-23%]).
ASA 5: a moribund patient not expected to survive 24 hours (ruptured aneurysm [9.4-51%]).
ASA 6: brain-dead patient whose organs are being harvested.
8. For emergent operations, add the letter ‘E’ after the classification.

Preoperative Fasting Guidelines
1. Recommendations (applies to all ages)

<table>
<thead>
<tr>
<th>Ingested Material</th>
<th>minimum Fasting Period (hrs)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clear liquids</td>
<td>2</td>
</tr>
<tr>
<td>Breast milk</td>
<td>4</td>
</tr>
<tr>
<td>Infant formula</td>
<td>6</td>
</tr>
<tr>
<td>Non-human milk</td>
<td>6</td>
</tr>
<tr>
<td>Light solid foods</td>
<td>6</td>
</tr>
</tbody>
</table>

Clear liquids include water, sugar-water, apple juice, non-carbonated soda, pulp-free juices, clear tea, black coffee.
Medications can be taken with up to 150 mL of water in the hour preceding induction of anesthesia.
PHARMACOLOGY

Local Anesthetics

1. Mechanism of action of local anesthetics
   A. Local anesthetics prevent increases in neural membrane permeability to sodium ions, slowing the rate of depolarization so that threshold potential is never reached and no action potential is propagated.
   B. Most local anesthetics bind to sodium channels in the inactivated state, preventing subsequent channel activation and the large transient sodium influx associated with membrane depolarization. Rapidly firing nerves are more sensitive and, therefore, are blocked first.

2. Metabolism
   A. Esters
      1. Ester local anesthetics are predominantly metabolized by pseudocholinesterase (plasma cholinesterase). Cerebrospinal fluid lacks esterase enzymes, so the termination of action of intrathecally injected ester local anesthetics depends upon their absorption into the bloodstream.
      2. P-aminobenzoic acid, a metabolite of ester local anesthetics, has been associated with allergic reactions.
   B. Amides
      1. Metabolized by microsomal enzymes in the liver; the amide linkage is cleaved through initial N-dealkylation followed by hydrolysis.
      2. Metabolites of prilocaine (o-toluidinederivatives), which accumulate after large doses (greater than 10 mg/kg), convert hemoglobin to methemoglobin. Benzocaine can also cause methemoglobinemia.

3. Physiochemical factors
   A. Lipid solubility: increased lipid solubility increases potency.
   B. Protein binding: the greater the protein binding (alpha1-acid glycoprotein), the longer the duration of action.
   C. pKa: determines the onset time. pKa is the pH at which 50% of the local anesthetic is in the charged form and 50% uncharged. Local anesthetics with a pKa closer to physiologic pH will have a higher concentration of nonionized base and a more rapid onset.
   D. Ion trapping refers to the accumulation of the ionized form of a local anesthetic in acidic environments due to a pH gradient between the ionized and non-ionized forms. This can occur between a mother and an acidotic fetus (ie, fetal distress), resulting in the accumulation of local anesthetic in fetal blood.
   E. pH of the drug solution: increasing the pH of the drug solution will increase the fraction of the non-ionized form, resulting in a faster onset. Most local anesthetic solutions are prepared commercially as a water-soluble HCL salt (pH 6-7). Agents with epinephrine added are made more acidic (pH 4-5) because epinephrine is unstable in alkaline environments.
   F. Minimum concentration of local anesthetic (Cm) is the minimum concentration of local anesthetic that will block nerve impulse conduction and is analogous to the minimum alveolar concentration (MAC).

4. Rate of systemic absorption of local anesthetics
   (from high to low): intravenous > tracheal > intercostal > caudal > paracervical > epidural
brachial plexus > sciatic/femoral > subcutaneous.

5. Spread of anesthesia and blockade
   A. Differential blockade of nerve fibers: myelinated fibers are more readily blocked then unmyelinated ones.
   B. Local anesthetics deposited around a peripheral nerve diffuse along a concentration gradient to block nerve fibers on the outer surface before more centrally located fibers.
   C. Sequence of clinical block (progresses in order): sympathetic block with peripheral vasodilation and skin temperature elevation, loss of pain and temperature sensation, loss of proprioception, loss of touch and pressure sensation, motor paralysis.

6. Preparations
   A. Hydrochloride salts: this preparation increases the solubility in water and is usually acidic to enhance the formation of the water-soluble ionized form. Plain solutions usually have an adjusted pH of 6, while solutions containing a vasoconstrictor have an adjusted pH of 4 because of the lability of catecholamine molecules at vasoconstrictor pH.
   B. Antimicrobial preservatives: usually added to multidose vials. Only preservative-free solutions should be used in spinal, epidural, or caudal anesthesia to prevent neurotoxic effects.
   C. Antioxidant: added to slow the breakdown of local anesthetics.

7. Adjuvants
   A. Epinephrine
      1. May be added to local anesthetics to produce local vasoconstriction, to limit systemic absorption, to prolong the duration of effect, tp decrease surgical bleeding, to increase the intensity of the block by direct alpha agonist effect on antinociceptive receptors in the spinal cord, and to assist in the evaluation of a test dose.
      2. The maximum dose of epinephrine should not exceed 10 mcg/kg in pediatric patients and 200-250 mcg in adults.
      3. Epinephrine should not be used in peripheral nerve blocks in areas with poor collateral blood flow or in intravenous regional techniques.
   B. Phenylephrine has been used like epinephrine, but with no advantage.
   C. Sodium bicarbonate
      1. Raises the pH and increases the concentration of nonionized free base.
      2. The addition of sodium bicarbonate (eg, 1 mL 8.4% sodium bicarbonate is added to each 10 mL of 1% lidocaine) speeds onset, improves quality of block, prolongs blockade by increasing the amount of free base available and decreases pain during subcutaneous infiltration.

8. Toxicity and effects
   A. Allergic reactions
      1. Ester-type local anesthetics: may cause allergic reactions form the metabolite para-aminobenzoic acid and persons sensitive to sulfa drugs (eg, sulfonamides or thiazide diuretics).
      2. Ester local anesthetics may produce allergic reactions in persons sensitive to sulfa drugs.
      3. Allergic reactions to amides are extremely rare and are probably related to the preservative and not the amide itself.
      4. Local hypersensitivity reactions: may produce local erythema, urticaria, edema, or dermatitis.
   B. Local toxicity
      1. Cauda equina syndrome
2. Chloroprocaine has been associated with neurotoxicity. The cause of this neural toxicity may be the low pH of chloroprocaine (pH 3.0).

C. System toxicity

1. Cardiovascular toxicity
   A. Local anesthetics depress myocardial automaticity (spontaneous phase IV depolarization) and reduce the duration of the refractory period (manifesting as prolonged PR interval and widening QRS).
   B. Myocardial contractility and conduction velocity are depressed at higher concentrations. Smooth muscle relaxation causes some degree of vasodilation (with the exception of cocaine).
   C. Cardiac dysrhythmia or circulatory collapse is often a presenting sign of local anesthetic overdose during general anesthesia.
   D. Intravascular injection of bupivacaine has produced severe cardiotoxic reactions, including hypotension, atrioventricular heart block, and dysrhythmias such as ventricular fibrillation. Pregnancy, hypoxemia, and respiratory acidosis are predisposing risk factors. Ropivacaine lacks significant cardiac toxicity because it dissociates more rapidly from sodium channels. Levobupivacaine has less cardiotoxic effects than bupivacaine.
   E. Cocaine: only local anesthetic that causes vasoconstriction at all doses.

2. Respiratory effects
   A. Lidocaine depresses the hypoxic drive (response to low PaO2).
   B. Apnea can result from phrenic and intercostal nerve paralysis or depression of the medullary respiratory center following direct exposure to local anesthetic agents (eg, postretrobulbar apnea syndrome).

3. Central nervous system toxicity
   A. Early symptoms of overdose include circumoral numbness, tongue paresthesia, and dizziness. Sensory complaints may include tinnitus and burred vision. Excitatory signs (eg, restlessness, agitation, nervousness, paranoia) often precede central nervous system depression (slurred speech, drowsiness, unconsciousness).
   B. Tonic-clonic seizures may result from selective blockade of inhibitory pathways. Respiratory arrest often follows seizure activity.
   C. CNS toxicity is exacerbated by hypercarbia, hypoxia, and acidosis.

4. Musculoskeletal effects: Local anesthetics are myotoxic when injected directly into skeletal muscle.

5. Other adverse effects
   A. Horner syndrome can result from blockade of B fibers in the TI-T4 nerve roots.
   B. Methemoglobinemia can be formed after large doses of prilocaine, benzocaine and EMLA cream.
   C. Decreased coagulation: Lidocaine has been demonstrated to prevent thrombosis, decrease platelet aggregation and enhance fibrinolysis of whole blood.
## Local Anesthetics: Dosages for Infiltration Anesthesia

<table>
<thead>
<tr>
<th>Drug</th>
<th>Plain Solution</th>
<th>Epinephrine Solution</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Max (mg)</td>
<td>Dose (min)</td>
</tr>
<tr>
<td>Procaine</td>
<td>400</td>
<td>30-60</td>
</tr>
<tr>
<td>Chlorprocaine</td>
<td>800</td>
<td></td>
</tr>
<tr>
<td>Lidocaine</td>
<td>300</td>
<td>30-120</td>
</tr>
<tr>
<td>Mepivacaine</td>
<td>300</td>
<td></td>
</tr>
<tr>
<td>Bupivacaine</td>
<td>175</td>
<td>120-240</td>
</tr>
<tr>
<td>Etidocaine</td>
<td>300</td>
<td></td>
</tr>
<tr>
<td>Ropivacaine</td>
<td>200</td>
<td></td>
</tr>
</tbody>
</table>

## Local Anesthetics: Dosages for Spinal Anesthesia

<table>
<thead>
<tr>
<th>Drug</th>
<th>Preparation</th>
<th>T10 Level (mg)</th>
<th>T6 Level (mg)</th>
<th>T4 Level (mg)</th>
<th>Duration Plain (min)</th>
<th>Duration w/epi (min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Procaine</td>
<td>10%</td>
<td>75</td>
<td>125</td>
<td>200</td>
<td>30-45</td>
<td>60-75</td>
</tr>
<tr>
<td>Lidocaine</td>
<td>5.0% in 7.5% glucose</td>
<td>25-50</td>
<td>50-75</td>
<td>75-100</td>
<td>45-60</td>
<td>60-90</td>
</tr>
<tr>
<td>Tetracaine*</td>
<td>1% in 10%</td>
<td>6-8</td>
<td>8-14</td>
<td>12-20</td>
<td>60-90</td>
<td>120-180</td>
</tr>
<tr>
<td>Bupivacaine</td>
<td>0.75% in 8.25%dextrose</td>
<td>6-10</td>
<td>8-14</td>
<td>12-20</td>
<td>90-120</td>
<td>120-150</td>
</tr>
<tr>
<td>Ropivacaine</td>
<td>0.2-1%</td>
<td>8-12</td>
<td>12-16</td>
<td>16-18</td>
<td>90</td>
<td>140</td>
</tr>
</tbody>
</table>

*For hypobaric spinal: tetracaine diluted with sterile water to 0.3% solution

**Preparation concentration of tetracaine is 1%; tetracaine is diluted with 5.0% glucose for hyperbaric solution and normal saline for isobaric solution

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**Neuromuscular Blocking Agents**

**Depolarizing neuromuscular blocking agents**

A. **Sucinylcholine** is the only depolarizing muscle relaxant and is made up of two joined acetylcholine molecules. Succinylcholine mimics the action of acetylcholine by depolarizing the postsynaptic membrane at the neuromuscular junction.

B. **Depolarizing blockade** is characterized by: muscle fasciculation followed by relaxation, absence of fade after tetanic or train-of-four stimulation, absence of posttetanic potentiation, potentiation of the block by anticholinesterases, and antagonism by nondepolarizing relaxants.

C. **Metabolism**

1. Succinylcholine has a rapid onset of action (30-60 seconds) with a short duration of action (5-10 minutes). Succinylcholine is rapidly metabolized by pseudocholinesterase into
succinylmonocholine (later broken down to choline and succinic acid) as it enters the circulation such that only a fraction (approximately 10%) of the injected dose ever reaches the neuromuscular junction.

2. As serum levels fall, succinylcholine molecules diffuse away from the neuromuscular junction and is broken down to choline and succinic acid in the plasma.

**D. Adverse side effects of succinylcholine**

1. **Cardiac:** Ganglionic stimulation may increase heart rate and blood pressure in adults; may produce bradycardia, junctional rhythm and sinus arrest in children after first dose and after second dose in adults (with short dose interval).
2. **Hyperkalemia**
3. **Increased intracranial pressure, increased cerebral blood flow, and increased intraocular pressure.**
4. **Increased intragastric pressure:** results from fasciculation of abdominal muscles; the increase in intragastric pressure is offset by an increase in lower esophageal sphincter tone.
5. **Myalgia and myoglobinuria**
6. **Malignant hyperthermia** may be triggered in susceptible patients.

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**Non-depolarizing neuromuscular agents**

**A. Mechanisms:** reversible competitive antagonism of Ach.

**B. Nondepolarizing blockade** is characterized by: absence of fasciculations, fade during tetanic and TOF stimulation, PTP, antagonism of block by depolarizing agents and anticholinesterases.

**C. Pharmacologic characteristics** (see table)

1. **Temperature:** Hypothermia prolongs blockade by decreasing metabolism and delaying excretion.
2. **Acid-base balance:** respiratory acidosis potentiates the blockade of most nondepolarizing relaxants and antagonizes the reversal.
3. **Electrolyte abnormalities:** Hypokalemia and hypocalcemia augment a nondepolarizing block. Hypermagnesemia potentiates blockade.
4. **Age:** Neonates have an increased sensitivity to nondepolarizing agents.
5. **Drug interactions:** Drugs that potentiate nondepolarizing relaxants include volatile agents, local anesthetics, calcium channel blockers, aminoglycosides, polymyxins, lincosamines, hexamethonium, trimethaphan, immunosuppressants, high-dose benzodiazepines, dantrolene, and magnesium.
6. **Synergistic blockade** may result when steroidal NMBD (vecuronium, rocuronium) are combined with benzylisoquinolines (atracurium).
7. **Sensitivity to neuromuscular blockade:** Muscles have different sensitivities to muscle relaxants. The most resistant to most sensitive muscles are: vocal cord, diaphragm, orbicularis oculi, abdominal rectus, adductor pollicis, masseter, pharyngeal, extraocular.
### Dosages of Muscle Relaxants

<table>
<thead>
<tr>
<th>Drug</th>
<th>Intubation</th>
<th>N2O/Opioid</th>
<th>Inhalation</th>
<th>Maintenance</th>
<th>Infusion mcg/kg/min</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vecuronium</td>
<td>0.4-0.6</td>
<td>0.3-0.4</td>
<td>0.2-0.3</td>
<td>0.1-0.15</td>
<td>4-12</td>
</tr>
<tr>
<td>Cisatracurium</td>
<td>0.15-0.2</td>
<td>0.05</td>
<td>0.03-0.04</td>
<td>0.01-0.02</td>
<td>1-2</td>
</tr>
<tr>
<td>Mivacurium</td>
<td>0.15-0.25*</td>
<td>0.1</td>
<td>0.08</td>
<td>0.05-0.1</td>
<td>3-15</td>
</tr>
<tr>
<td>Pancuronium</td>
<td>0.08-0.12</td>
<td>0.05-0.06</td>
<td>0.03</td>
<td>0.01-0.015</td>
<td></td>
</tr>
<tr>
<td>Rocuronium</td>
<td>0.6-1.2</td>
<td>0.3-0.4</td>
<td>0.2-0.3</td>
<td>0.1-0.15</td>
<td>8-12</td>
</tr>
<tr>
<td>Succinyl-choline</td>
<td>1.0-1.5</td>
<td></td>
<td></td>
<td>0.04-0.07</td>
<td>10-100</td>
</tr>
<tr>
<td>Vecuronium</td>
<td>0.1-0.2</td>
<td>0.05</td>
<td>0.03-0.04</td>
<td>0.01-0.02</td>
<td>0.8-2.0</td>
</tr>
</tbody>
</table>

### Muscle Relaxants

<table>
<thead>
<tr>
<th>Drug</th>
<th>ED95 (mg/kg)</th>
<th>Onset (min)</th>
<th>Duration (min)</th>
<th>Histamine Release</th>
<th>Elimination and Misc</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atracurium</td>
<td>0.25</td>
<td>3-5</td>
<td>20-35</td>
<td>+</td>
<td>Hofmann elimination and ester hydrolysis, laudanosine</td>
</tr>
<tr>
<td>Cisatracurium</td>
<td>0.05</td>
<td>1-2</td>
<td>60</td>
<td>None</td>
<td>Hofmann elimination</td>
</tr>
<tr>
<td>d-Tubo-curarine</td>
<td>0.51</td>
<td>3-5</td>
<td>60-90</td>
<td>+++</td>
<td>70% renal; 20% biliary; autonomic ganglia block</td>
</tr>
<tr>
<td>Doxacurium</td>
<td>0.03</td>
<td>4-6</td>
<td>60-90</td>
<td>None</td>
<td>35% renal</td>
</tr>
<tr>
<td>Gallamine</td>
<td>3</td>
<td>4-5</td>
<td>70-80</td>
<td>None</td>
<td>80-100% renal; muscarinic block</td>
</tr>
<tr>
<td>Metocurine</td>
<td>0.28</td>
<td>3-5</td>
<td>60-90</td>
<td>+</td>
<td>80-100% renal; autonomic ganglia blockade</td>
</tr>
<tr>
<td>Mivacurium</td>
<td>0.09</td>
<td>2-3</td>
<td>12-20</td>
<td>+</td>
<td>plasma cholinesterase</td>
</tr>
<tr>
<td>Pancuronium</td>
<td>0.07</td>
<td>3-5</td>
<td>60-90</td>
<td>None</td>
<td>70% renal; 15-20% liver; muscarinic block (10-15% HR increase)</td>
</tr>
<tr>
<td>Pipecuronium</td>
<td>0.06</td>
<td>3-5</td>
<td>60-90</td>
<td>None</td>
<td>90% renal; 10% liver</td>
</tr>
<tr>
<td>Rapacuronium</td>
<td>0.75-1.0</td>
<td>1-2</td>
<td>15-20</td>
<td>+</td>
<td>50% hepatic; 25% renal</td>
</tr>
<tr>
<td>Rocuronium</td>
<td>0.3</td>
<td>1-2</td>
<td>20-35</td>
<td>None</td>
<td>10-25% renal; 50-70% biliary; 10-20% hepatic</td>
</tr>
<tr>
<td>Succinyl-choline</td>
<td>0.25</td>
<td>1</td>
<td>5-10</td>
<td>Rare</td>
<td>Plasma cholinesterase muscarinic and nicotinic stim</td>
</tr>
<tr>
<td>Vecuronium</td>
<td>0.06</td>
<td>3-5</td>
<td>20-35</td>
<td>None</td>
<td>10-20% renal; 40-60% biliary; 20% hepatic</td>
</tr>
</tbody>
</table>

### Anticholinesterases

1. **Mechanism of action:** cholinesterase inhibitors inactivate acetylcholinesterase by reversibly binding to the enzyme increasing the amount of acetylcholine available to compete with the nondepolarizing agent.

2. **In excessive doses,** acetylcholinesterase inhibitors can paradoxically potentiate a nondepolarizing neuromuscular blockade and prolong the depolarization blockade of succinylcholine.
3. Anticholinesterases increases acetylcholine at both nicotinic and muscarinic receptors. Muscarinic side effects can be blocked by administration of atropine or glycopyrrolate. See table for muscarinic side effects.

4. Cholinergic receptors
   A. Nicotinic receptors (2 subtypes)
   1. NM: found at the neuromuscular junction in skeletal muscle.
   2. NN: found in autonomic ganglia (sympathetic and parasympathetic), the adrenal medulla, and the CNS.
   B. Muscarinic receptors (5 subtypes; all found within the CNS)
      1. M1: located in autonomic ganglia and various secretory glands.
      2. M2: found mainly in the heart and brainstem.
      4. M4: found in the neostriatum.
      5. M5: found in the substantia nigra.

<table>
<thead>
<tr>
<th>Anticholinesterases</th>
<th>Edrophonium</th>
<th>Neostigmine</th>
<th>Pyridostigmine</th>
<th>Physostigmine</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dose (mg/kg)</td>
<td>0.5-1.0</td>
<td>0.035-0.07 (up to 5 mg)</td>
<td>0.15-0.35</td>
<td>0.01-0.03 (per dose)</td>
</tr>
<tr>
<td>Onset (min)</td>
<td>1-3</td>
<td>7-10</td>
<td>10-13</td>
<td>5</td>
</tr>
<tr>
<td>Duration (min)</td>
<td>40-70</td>
<td>65-80</td>
<td>80-130</td>
<td>30-300</td>
</tr>
<tr>
<td>Renal Excretion (%)</td>
<td>70</td>
<td>50</td>
<td>75</td>
<td>metabolized by plasma esterases</td>
</tr>
<tr>
<td>Atropine (mcg/kg)</td>
<td>7-10</td>
<td>15-30</td>
<td>15-20</td>
<td></td>
</tr>
<tr>
<td>Glycopyrrolate</td>
<td>do not use</td>
<td>7 mcg/kg</td>
<td>7 mcg/kg</td>
<td>anticholinergic overdose treatment</td>
</tr>
<tr>
<td>Misc</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Muscarinic Side Effects of Cholinesterase Inhibitors

<table>
<thead>
<tr>
<th>Organ System</th>
<th>Muscarinic Side Effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiovascular</td>
<td>Decreased heart rate, dysrhythmias</td>
</tr>
<tr>
<td>Pulmonary</td>
<td>Bronchospasm, increased bronchial secretions</td>
</tr>
<tr>
<td>Cerebral</td>
<td>Diffuse excitation (physostigmine only)</td>
</tr>
<tr>
<td>Gastrointestinal</td>
<td>Intestinal spasm, increased salivation</td>
</tr>
<tr>
<td>Genitourinary</td>
<td>Increased bladder tone</td>
</tr>
<tr>
<td>Ophthalmologic</td>
<td>Pupillary constriction</td>
</tr>
</tbody>
</table>

**Anticholinergics**

1. **Mechanism of action**: competitively inhibits the action of acetylcholine at muscarinic receptors with little or no effect at nicotinic receptors.

2. **Central anticholinergic syndrome**
   A. **Scopolamine and atropine** can enter the central nervous system and produce symptoms of restlessness and confusion that may progress to somnolence and unconsciousness. Other systemic manifestations include dry mouth, tachycardia, atropine flush, atropine fever, and impaired vision.
**B. Phystostigmine**, a tertiary amine anticholinesterase, is lipid-soluble and reverses central anticholinergic toxicity. An initial dose of .01-0.03 mg/kg is recommended and may need to be repeat after 15-30 minutes.

**C. Glycopyrrolate** does not easily cross the blood-brain barrier, and thus does not cause a central anticholinergic syndrome.

### Pharmacological Characteristics of Anticholinergic Drugs

<table>
<thead>
<tr>
<th></th>
<th>Atropine</th>
<th>Scopolamine</th>
<th>Glycopyrrolate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tachycardia</td>
<td>+++</td>
<td>+</td>
<td>++</td>
</tr>
<tr>
<td>Bronchodilation</td>
<td>++</td>
<td>+</td>
<td>++</td>
</tr>
<tr>
<td>Sedation</td>
<td>+</td>
<td>+++</td>
<td>0</td>
</tr>
<tr>
<td>Antisialagogues</td>
<td>++</td>
<td>+++</td>
<td>+++</td>
</tr>
<tr>
<td>Amnesia</td>
<td>+</td>
<td>+++</td>
<td>0</td>
</tr>
<tr>
<td><strong>Duration of action</strong></td>
<td><strong>IV: 15-30 min</strong></td>
<td><strong>IV: 30-60 min</strong></td>
<td><strong>IV: 2-4 hr</strong></td>
</tr>
<tr>
<td></td>
<td><strong>IM: 2-4 hr</strong></td>
<td><strong>IM: 4-6 hr</strong></td>
<td><strong>IM: 6-8 hr</strong></td>
</tr>
<tr>
<td><strong>Dose-Adult</strong></td>
<td>Premed: 0.2-0.4 mg IV</td>
<td>0.3-0.6 mg</td>
<td>0.1-0.2 mg</td>
</tr>
<tr>
<td></td>
<td>Brady: 0.4-1.0 mg IV</td>
<td>IV/IM</td>
<td>IV/IM</td>
</tr>
<tr>
<td><strong>Dose-Pediatric</strong></td>
<td>Premed: 10 mcg/kg</td>
<td>6 mcg/kg</td>
<td>4-8 mcg/kg</td>
</tr>
<tr>
<td></td>
<td>Brady: 20 mcg/kg</td>
<td>IV/IM</td>
<td>IV/IM</td>
</tr>
</tbody>
</table>

O = No effect; + = Minimal effect; ++ = Moderate effect; +++ = Marked effect

---

### Benzodiazepines

#### 1. Mechanism of action

A. Benzodiazepines selectively attach to alpha subunits to enhance the chloride channel gating function of the inhibitory neurotransmitter GABA.

B. Benzodiazepine receptors mostly occur on postsynaptic nerve endings in the central nervous system.

C. Benzodiazepines undergo hepatic metabolism via oxidation and glucuronide conjugation.

#### 2. Systemic effects

A. **Central nervous system effects**
   1. Amnestic, anticonvulsant, hypnotic, muscle-relaxant, and sedative effects in a dose-dependent manner.
   2. Reduced cerebral oxygen consumption, cerebral blood flow and ICP.

B. **Cardiovascular effects**
   1. Mild systemic vasodilation and reduction in cardiac output;
   2. Pronounced effect in hypovolemic patients, those with poor cardiac reserve, or if administered with an opioids.
   3. Midazolam reduces blood pressure and SVR more than diazepam.

C. **Respiratory effects**
   1. Mild dose-dependent decrease in respiratory rate and tidal volume.
   2. Increased respiratory depression with opioids and pulmonary disease.

D. **Miscellaneous effects**
   1. Reduces MAC by up to 30%.
2. Pain during IV/IM injection and thrombophlebitis occurs with diazepam (secondary to its organic solvent propylene glycol).
3. Crosses the placenta and may lead to neonatal depression.
4. Erythromycin inhibits midazolam metabolism; cimetidine reduces metabolism of diazepam.
5. Heparin displaces diazepam from protein- binding sites and increases the free drug concentration.
6. Benzodiazepine administered to patient receiving valproate may precipitate a psychotic episode.

3. Reversal
A. Flumazenil (Mazicon, Romazicon), an imidazobenzodiazepine, is a competitive antagonist of benzodiazepines.

B. Dosage
1. Reversal of conscious sedation: 0.2 mg IV over 15 seconds; give additional 0.1 mg IV bolus every 60 seconds to achieve desired effect, to a total of 1 mg.
2. Reversal of overdose: 0.2 mg IV over 30 seconds; if necessary, give 0.3 mg IV 60 seconds later, if no effect, give 0.5 mg boluses every 60 seconds to a total of 3 mg.
3. Reversal of re sedation: 0.2 mg IV (to 1 mg/hr), or infusion 0.5 mg/hr.
4. Diagnosis in coma: 0.5 mg IV repeated up to 1.0 mg IV.
C. Duration of antagonism is brief (45-90 minutes) and may require repeated doses. Peak effect occurs in approximately 10 minutes.
D. Flumazenil may induce seizures, acute withdrawal, nausea, dizziness, agitation, or arrhythmias.
E. Flumazenil is contraindicated in patients with tricyclic antidepressant overdose and patients receiving benzodiazepines for control of seizures or elevated intracranial pressure. Use caution in patients who have received long-term treatment with benzodiazepines.

<table>
<thead>
<tr>
<th>Benzodiazepines</th>
<th>Midazolam (Versed)</th>
<th>Diazepam (Valium)</th>
<th>Lorazepam (Ativan)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Relative Potency</td>
<td>3</td>
<td>1</td>
<td>5</td>
</tr>
<tr>
<td>Induction</td>
<td>0.2-0.6 mg/kg</td>
<td>0.3-0.6 mg/kg</td>
<td></td>
</tr>
<tr>
<td>Maintenance</td>
<td>0.05 mg/kg prn or</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sedation (mg/kg)</td>
<td>0.25-1.5mcg/kg/min</td>
<td></td>
<td></td>
</tr>
<tr>
<td>IM: 0.07-0.2</td>
<td></td>
<td>IV: 0.05-0.2</td>
<td>IV/IM: 0.02-0.08</td>
</tr>
<tr>
<td>IN: 0.2-0.5</td>
<td></td>
<td>PO: 0.2-0.5</td>
<td>PO: 0.05</td>
</tr>
<tr>
<td>IV: 0.025-0.1</td>
<td></td>
<td>PR: 0.2-0.5</td>
<td></td>
</tr>
<tr>
<td>PO: 0.5-0.75</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PR: 0.3-0.5</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Duration</td>
<td>IM: 2 hrs</td>
<td>IV: 20-30 min</td>
<td>IM/IV/PO: 6-8 hrs</td>
</tr>
<tr>
<td>Elimination</td>
<td>1-7.5 hrs</td>
<td>22-50 hrs</td>
<td>10-20 hr</td>
</tr>
<tr>
<td>Half-Time</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
1. Classification of opioids receptors
   A. Mu receptor: morphine is the prototype exogenous ligand.
      1. Mu-1: the main action at this receptor is analgesia, but also responsible for miosis, nausea/vomiting, urinary retention, and pruritus. The endogenous ligands are enkephalins.
      2. Mu-2: respiratory depression, euphoria, sedation, bradycardia, ileus and physical dependence are elicited by binding at this receptor.
   B. Delta: modulation of mu receptor, physical dependence. High selective for the endogenous enkephalins, but opioids drugs still bind (leu- enkephalin and beta-endorphin).
   C. Kappa: ketocyclazocine and dynorphin are the prototype exogenous and endogenous ligands, respectively. Analgesia, sedation, dysphoria, and psychomimetic effects are produced by this receptor. Binding to the kappa receptor inhibits release of vasopressin and thus promotes diuresis. Pure kappa agonists do not produce respiratory depression.
   D. Sigma: N-allylnormetazocine is the prototype exogenous ligand. While this receptor binds many types of compounds, only levorotatory opioids isomers have opioids activity. The sigma receptor binds primarily dextrorotatory compounds. Dysphoria, hypertonia, tachycardia, tachypnea, and mydriasis are the principal effects of this receptor.

2. Systemic effects
   A. Central nervous system effects
      1. Sedation and analgesia dose-dependent; euphoria.
      2. Amnesia with large doses (not reliable).
      3. Reduces MAC.
      4. Decreases cerebral blood flow and metabolic rate.
      5. Toxicity
         A. Dysphoria and agitation may occur (higher with meperidine).
         B. Seizures may be produced by meperidine (normeperidine, major metabolite, is potent convulsant).

---

### Oral Benzodiazepines

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alprazolam (Xanax)</td>
<td>0.25-0.5 mg TID/QID (up to 4 mg/day)</td>
</tr>
<tr>
<td>Chlordiazepoxide (Librium)</td>
<td>5-25 mg TID/QID</td>
</tr>
<tr>
<td>Clonazepam (Clonopin)</td>
<td>Initial: 0.5 mg TID Maintenance: 0.05-0.2 mg/kg</td>
</tr>
<tr>
<td>Clorazepate (Tranxene)</td>
<td>7.5-15 mg BID-QID or 11.25-22.5 qhs</td>
</tr>
<tr>
<td>Diazepam (Valium)</td>
<td>2-10 mg BID-QID</td>
</tr>
<tr>
<td>Estazolam (Prosom)</td>
<td>0.5-2 mg qhs</td>
</tr>
<tr>
<td>Flurazepam (Dalmane)</td>
<td>15-30 mg qhs</td>
</tr>
<tr>
<td>Lorazepam (Ativan)</td>
<td>1-10 mg BID/TID</td>
</tr>
<tr>
<td>Oxazepam (Serax)</td>
<td>10-30 mg TID/QID</td>
</tr>
<tr>
<td>Quazepam (Doral)</td>
<td>7.5-15 mg qhs</td>
</tr>
<tr>
<td>Temazepam (Restoril)</td>
<td>15-30 mg qhs</td>
</tr>
<tr>
<td>Triazolam (Halcion)</td>
<td>0.125-0.25 mg qhs</td>
</tr>
</tbody>
</table>
C. ICP may increase if ventilation and PaCO₂ are not controlled.

B. Cardiovascular effects
   1. Minimal contractility effects, except meperidine (direct myocardial depressant); may enhance depressant effects of other agents.
   2. Bradycardia, dose-dependent, by stimulating the central nuclei on the vagus nerves increasing vagal tone; meperidine, may increase heart rate because of its atropine-like structure.

C. Respiratory effects
   1. Respiratory depression: dose-related depression on the ventilatory response to CO₂ by direct effect on respiratory centers resulting in increased arterial carbon dioxide tension, decreased breathing rate, increased tidal volume, decreased minute ventilation and decreased ventilatory response to carbon dioxide
   2. Cough suppression: dose-dependent decrease in cough reflex.

D. Pupillary constriction: opioids stimulate the Edinger-Westphal nucleus of the oculomotor nerve to produce miosis.

E. Muscle rigidity
   1. Large IV doses may produce generalized hypertonus of skeletal muscle, which, in its most severe form, can prevent ventilation.
   2. Benzodiazepine pretreatment may help in preventing rigidity.

F. Gastrointestinal effects
   1. Nausea/vomiting: direct stimulation of the chemoreceptor trigger zone.
   2. Decrease gastric motility; increase tone and secretions of GI tract.

G. Urinary retention: increases tone of ureter and vesicle sphincter, making voiding difficult (can be reversed with atropine).

H. Endocrine: may block stress response to surgery at high doses.

I. Placenta: can cross the placenta causing neonatal depression.

J. Histamine release
   1. May produce local itching, redness or hives near the site of injection and may cause a decrease in SVR, hypotension, and tachycardia.
   2. Morphine and meperidine release histamine, but fentanyl, sufentanil, alfentanil, and remifentanil do not.

K. Tolerance: both acute and chronic tolerance may occur.

L. Drug interactions: administration of meperidine in a patient taking a monoamine oxidase inhibitor may result in delirium or hyperthermia.

<table>
<thead>
<tr>
<th>Pharmacokinetics of Intravenous Opioids</th>
</tr>
</thead>
<tbody>
<tr>
<td>Comparative Potency</td>
</tr>
<tr>
<td>Comparative Potency</td>
</tr>
<tr>
<td>Peak Effect (min)</td>
</tr>
<tr>
<td>Duration(hr)</td>
</tr>
<tr>
<td>Half-Life(hr)</td>
</tr>
<tr>
<td>Clearance(mL/min/ kg)</td>
</tr>
</tbody>
</table>
### Meperidine Dosing

<table>
<thead>
<tr>
<th>Indication</th>
<th>Adult</th>
<th>Pediatric</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sedation</td>
<td>50-150 mg IV, IM</td>
<td>1-2 mg/kg IV, IM</td>
</tr>
<tr>
<td>Incremental Dosing</td>
<td>50-150 mg IV, IM q3-4 hr</td>
<td>0.5-2 mg/kg IV, IM, SC q3-4 hrs</td>
</tr>
<tr>
<td>Epidural</td>
<td>Bolus: 20-50 mg Cont Infusion: 10-50 mg/hr</td>
<td></td>
</tr>
<tr>
<td>Postop Shivering</td>
<td>12.5 mg IV</td>
<td></td>
</tr>
<tr>
<td>Continuous Infusion</td>
<td></td>
<td>0.3-1.5 mg/kg/hr</td>
</tr>
</tbody>
</table>

### Morphine Dosing

<table>
<thead>
<tr>
<th>Indication</th>
<th>Adult</th>
<th>Pediatric</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sedation</td>
<td>2-10 mg IV</td>
<td>0.05-0.1 mg/kg IV</td>
</tr>
<tr>
<td>Incremental Dosing</td>
<td>2-20 mg q2-4 hr IV, IM, SC</td>
<td>0.05-0.2 mg/kg q2-4 hr IV, IM, SC</td>
</tr>
<tr>
<td>PCA</td>
<td>Bolus: 1-3 mg       Lockout: 6-20 min Basal rate: 0-1 mg/hr</td>
<td>Bolus: 0.01-0.03 mg/kg Lockout: 6-20 min Basal rate: 0-0.03 mg/kg/hr</td>
</tr>
<tr>
<td>Epidural</td>
<td>Bolus: 2-6 mg q8-24 hr Cont Inf: 0.2-1 mg/hr</td>
<td>0.03-0.05 mg/kg (max: 0.1 mg/kg or 5 mg/24 hr)</td>
</tr>
<tr>
<td>Intrathecal</td>
<td>0.1-0.5 mg</td>
<td>0.01 mg/kg</td>
</tr>
<tr>
<td>Continuous Infusion</td>
<td>0.8-10 mg/hr IV (up to 80 mg/hr)</td>
<td>Sickle Cell/Cancer Pain: 0.025-2 mg/kg/hr Postop Pain: 0.01-0.04 mg/kg/hr</td>
</tr>
</tbody>
</table>

### Fentanyl Dosing

<table>
<thead>
<tr>
<th>Indication</th>
<th>Initial Dose</th>
<th>Supplemental Dose</th>
<th>Continuous Infusion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Premedication</td>
<td>25-100 mcg</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sedation (minor procedure)</td>
<td>0.5-2 mcg/kg</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Adjunct to GA</td>
<td>2-50 mcg/kg</td>
<td>25-50</td>
<td></td>
</tr>
<tr>
<td>General Anesthesia</td>
<td>50-150 mcg/kg</td>
<td>25-100</td>
<td>0.5-5 mcg/kg/hr</td>
</tr>
<tr>
<td>Postoperative Analgesia</td>
<td>0.5-1.5 mcg/kg</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

### Common Parenteral Opioids

<table>
<thead>
<tr>
<th>Drug</th>
<th>Equianalgesic Dose (mg)</th>
<th>Adult Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Meperidine</td>
<td>75</td>
<td>50-150 mg q2-4 hr</td>
</tr>
<tr>
<td>Morphine</td>
<td>10</td>
<td>2-20 mg q2-6 hr</td>
</tr>
<tr>
<td>Hydromorphone</td>
<td>1.5</td>
<td>1-4 mg/dose q4-6 hr</td>
</tr>
<tr>
<td>Fentanyl</td>
<td>0.1</td>
<td>1-2 mcg/kg q30-60 min</td>
</tr>
<tr>
<td>Drug</td>
<td>Adult Dosing</td>
<td>Pediatric Dosing</td>
</tr>
<tr>
<td>-------------------------</td>
<td>---------------</td>
<td>------------------</td>
</tr>
<tr>
<td>Buprenorphine (Buprenex)</td>
<td>0.4 mg IV q 4-6 hr</td>
<td>0.004 mg/kg IV q 6-8 hr</td>
</tr>
<tr>
<td>Butorphanol (Stadol)</td>
<td>0.5-2 mg IV q 3-4 hr</td>
<td>Not recommended</td>
</tr>
<tr>
<td>Dezocine (Dalgan)</td>
<td>2.5-10 mg IV q 2-4 hr</td>
<td>Not recommended</td>
</tr>
<tr>
<td>Nalbuphine (Nubain)</td>
<td>10 mg IV q 3-4 hr</td>
<td>0.1 mg/kg IV q 3-4 hr</td>
</tr>
<tr>
<td>Pentazocine (Talwin)</td>
<td>50 mg PO q 4-6 hr</td>
<td>Not recommended</td>
</tr>
</tbody>
</table>

### Opioid Antagonist

1. **Naloxone (Narcan)**
   - **Pure opioids antagonists**: administration results in displacement of opioids agonists from opioids receptors.
   - **Peak effects**: seen in 1-2 minutes; duration approximately 30 minutes.

2. **Side effects**
   - **Pain**: may lead to abrupt onset of pain.
   - **Sudden antagonism**: can activate the sympathetic nervous system, resulting in cardiovascular stimulation.

3. **Dosage**
   - **Bolus**: adult: 0.04 mg IV in titrated bolus every 2-3 minutes until the desired effect; ped: 1-4 mcg/kg titrated.
   - **Continuous infusion**: 5 mcg/kg/hr IV, will prevent respiratory depression without altering the analgesia produced by neuraxial opioids.

### Nonsteroidal Anti-Inflammatory Drugs (NSAIDs)

1. **Mechanism of action**: anti-inflammatory effects are due to the inhibition of cyclooxygenase, which prevents the formation of inflammatory mediators such as prostaglandins and thromboxanes.

2. **Adverse effects**
   - **Common**: GI effects (gastritis, peptic ulcer, GI bleeding, abdominal pain, nausea, vomiting, diarrhea), decreased hemostasis (platelet dysfunction), surgical bleeding, renal dysfunction and failure, drug interactions.
   - **Other effects**: hepatic necrosis, asthma, vasomotor rhinitis, antineurotic edema, urticaria, laryngeal edema, hypotension, impede cartilage repair.

3. **Contraindications to NSAID use**: history of peptic ulcer disease or intolerance to NSAIDs; bleeding, bleeding diatheses, or anticoagulant therapy; renal failure, renal dysfunction, or risk factors for renal dysfunction; old age (particularly with risk factors); prophylactic use in major surgery.
### Nonsteroidal Anti-Inflammatory Drugs (NSAIDs)

<table>
<thead>
<tr>
<th>Generic Name</th>
<th>Dosage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acetaminophen (Tylenol)</td>
<td>Adult: 500-1000 mg PO q4 h</td>
</tr>
<tr>
<td></td>
<td>Ped: 10-15 mg/kg PO q4 h; 15-20 mg/kg PR q4 h</td>
</tr>
<tr>
<td>Acetylsalicylic Acid</td>
<td>Adult: 325-650 mg PO q4 h</td>
</tr>
<tr>
<td></td>
<td>Ped: 10-15 mg/kg PO q4 h; 15-20 mg/kg PR q4</td>
</tr>
<tr>
<td>Celecoxib (Celebrex)</td>
<td>Adult: 100 mg BID or 200 mg qd; 200 mg BID</td>
</tr>
<tr>
<td>Choline Mg Trisalicylate (Trilisate)</td>
<td>Adult: 500-1000 mg PO q8-12 hr</td>
</tr>
<tr>
<td></td>
<td>Ped: 7.5-15 mg/kg PO q6-8 h</td>
</tr>
<tr>
<td>Choline Salicylate (Arthropan)</td>
<td>Adult: 870 mg q3-4 h</td>
</tr>
<tr>
<td>Diclofenal Sodium (Voltaren)</td>
<td>Adult: 25-75 mg q8-12 hr</td>
</tr>
<tr>
<td>Diflunisal (Dolobid)</td>
<td>Adult: 250-500 mg q8-12 hr</td>
</tr>
<tr>
<td>Etodolic Acid (Lodine)</td>
<td>Adult: 200-400 mg q6-8 hr</td>
</tr>
<tr>
<td>Fenoprofen Calcium (Nalfon)</td>
<td>Adult: 300-600 mg PO q6 hr</td>
</tr>
<tr>
<td>Flurbiprofen (Anasid)</td>
<td>Adult: 50-100 mg BID/TID</td>
</tr>
<tr>
<td>Ibuprofen (Motrin)</td>
<td>Adult: 200-800 mg PO q6-8 hr</td>
</tr>
<tr>
<td></td>
<td>Ped: 5-10 mg/kg PO q6-8 hr</td>
</tr>
<tr>
<td>Indomethacin (Indocin)</td>
<td>Adult: 25-50 mg q8-12 hr</td>
</tr>
<tr>
<td>Ketoprofen (Orudis)</td>
<td>Adult: 25-75 mg PO q6-8 hr</td>
</tr>
<tr>
<td>Ketorolac (Toradol)</td>
<td>Adult: 10 mg PO q6-8 hr; 30-60 mg IV q6 h</td>
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<td></td>
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<td>MeclofenamateSodium (Meclomen)</td>
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<td>Meloxicam (Mobic)</td>
<td>Adult: 7.5-15 mg qd</td>
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<td></td>
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<td>Naproxen Sodium (Anaprox)</td>
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<tr>
<td>Piroxicam (Feldene)</td>
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<td>Ped: 0.2-0.3 mg/kg/day qd</td>
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<td>Salsalate (Disalcid)</td>
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<tr>
<td>Sulindac (Clinoril)</td>
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<tr>
<td>Tolmetin (Tolectin)</td>
<td>Adult: 200-600 mg q8 hr</td>
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<tr>
<td></td>
<td>Ped: 15-30 mg q6-8 hr</td>
</tr>
</tbody>
</table>

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**Intravenous Induction Agents**

**Sodium thiopental (Pentothal)** and other barbiturates.

**A. Preparation:** thiopental is prepared as a 2.5% solution, water-soluble, pH of 10.5, and stable for up to 1-2 weeks if refrigerated.

**B. Mechanism of action:** depresses the reticular activating system, reflecting the ability of barbiturates to decrease the rate of dissociation of the inhibitory neurotransmitter GABA.
C. Pharmacokinetics

1. Short duration of action (5-10 minutes) following IV bolus reflects high lipid solubility and redistribution from the brain to inactive tissues.
2. Protein binding parallels lipid solubility, decreased protein binding increases drug sensitivity.
3. Protein binding of thiopental in neonates is about half that in adults, suggesting a possible increased sensitivity to this drug in neonates.
4. Fat is the only compartment in which thiopental continues to accumulate 30 minutes after injection.

D. Effects on organ systems

1. Cardiovascular: Induction doses cause a decrease in blood pressure (peripheral vasodilation) and tachycardia (a central vagolytic effect).
2. Respiratory: barbiturate depression on; the medullary ventilatory center decreases the ventilatory response to hypercapnia and hypoxia. Laryngospasm and hiccuping are more common after methohexital than after thiopental.
3. Cerebral: Barbiturates constrict cerebral vasculature, decreasing cerebral blood flow and intracranial pressure. Barbiturates cause a decline in cerebral oxygen consumption (up to 50% of normal) and slowing of the EEG (an exception is methohexital which activates epileptic foci). This effect may provide some brain protection from transient episodes of focal ischemia (eg, cerebral embolism) but probably not from global ischemia (eg, cardiac arrest).
4. Renal: Barbiturates decrease renal blood flow and glomerular filtration rate in proportion to the fall in blood pressure.
5. Hepatic: Hepatic blood flow is decreased.

E. Adverse effects

1. Barbiturates are contraindicated in patients with acute intermittent porphryia, variegate porphyria, and hereditary coprophryia.
2. Venous irritation and tissue damage (reflects possible barbiturate crystal formation); intra-arterial injection results in severe pain and possible gangrene.
3. Myoclonus and hiccuping.

---

Propofol

A. Mechanisms of action: propofol increases the inhibitory neurotransmission mediated by gamma-aminobutyric acid.

B. Pharmacokinetics: highly lipid solubility. Short duration of action results from a very short initial distribution half-life (2-8 minutes). Elimination occurs primarily through hepatic metabolism to inactive metabolites. Recovery from propofol is more rapid and accompanied by less hangover than other induction agents.

C. Effects on organ systems

1. Cardiovascular: decrease in arterial blood pressure secondary to a drop in systemic vascular resistance, contractility, and preload. Hypotension is more pronounced than with thiopental. Propofol markedly impairs the normal arterial baroreflex response to hypotension.
2. Respiratory: Propofol causes profound respiratory depression. Propofol induced depression of upper airway reflexes exceeds that of thiopental.
3. Cerebral: decreases cerebral blood flow and intracranial pressure. Propofol has
antiemetic, antipruritic, and anticonvulsant properties.

**D. Other effects**

1. **Venous irritation:** Pain may be reduced by prior administration of opioids or lidocaine.
2. Propofol is an emulsion and should be used with caution if lipid disorder present. Propofol is preservative free.
4. Allergic reactions may reflect patient sensitivity to the solvent, isopropylphenol structure of propofol, or sulfite preservative.
5. Occasional myoclonic movement.
6. Subhypnotic doses (10-15 mg) can help treat nausea/vomiting.

---

**Ketamine**

**A. Mechanism of action:** Ketamine blocks polysynaptic reflexes in the spinal cord, inhibiting excitatory neurotransmitter effects. Ketamine functionally dissociates the thalamus from the limbic cortex, producing a state of dissociative anesthesia.

**B. Structure:** Ketamine is a structural analogue of phencyclidine (PCP).

**C. Pharmacokinetics:** metabolized in the liver to multiple metabolites.

**D. Effects on organ systems**

1. **Cardiovascular:** Ketamine increases arterial blood pressure, heart rate, and cardiac output. The direct myocardial depressant effects of ketamine (large doses) are unmasked by sympathetic blockade or patients who are catecholamine depleted.
2. **Respiratory:** Ventilation is minimally affected with normal doses of ketamine. Ketamine is a potent bronchodilator.
3. **Cerebral:** Ketamine increases cerebral oxygen consumption, cerebral blood flow, and intracranial pressure.

**E. Drug interactions:** Nondepolarizing muscle relaxants are potentiated by ketamine. The combination of ketamine and theophylline may predispose patients to seizures.

**F. Adverse effects**

1. **Increased salivation** (can be attenuated by pretreatment with an anticholinergic).
2. **Emergence delirium:** characterized by visual, auditory, proprioceptive and confusional illusions; reduced by benzodiazipine (midazolam) premedication.
3. **Myoclonic movements.**
4. **Increased ICP.**
5. **Eyes:** nystagmus, diplopia, blepharospasm, and increased intraocular pressure.

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<thead>
<tr>
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<th>Propofol</th>
<th>Thiopental</th>
<th>Ketamine</th>
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<tr>
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<td>3-5</td>
<td>1-2 (4-8 mgIM)</td>
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</tr>
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<tr>
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</table>

### Inhaled Anesthetics

**Sevoflurane**

**A. Advantages**
1. Well tolerated (non-irritant, sweet odor), even at high concentrations, making this the agent of choice for inhalational induction.
2. Rapid induction and recovery (low blood:gas coefficient)
3. Does not sensitize the myocardium to catecholamines as much as halothane.
4. Does not result in carbon monoxide production with dry soda lime.

**B. Disadvantages**
1. Less potent than similar halogenated agents.
2. Interacts with CO₂ absorbers. In the presence of soda lime (and more with barium lime) compound A (a vinyl ether) is produced which is toxic to the brain, liver, and kidneys. Thus it is recommended that, in the presence of soda lime, fresh gas flow rates should not be less than 2 L/min, and use of barium lime is contraindicated.
3. About 5% is metabolized and elevation of serum fluoride levels has led to concerns about the risk of renal toxicity. In theory, sevoflurane should be avoided in the presence of renal failure.
4. Postoperative agitation may be more common in children then seen with halothane.

**Isoflurane**

**A. Advantages**
1. Suitable for virtually all types of surgery.

**B. Disadvantages**
1. May have coronary steal effect.
2. Pungent odor makes unsuitable for inhalational induction.

**Halothane**

**A. Advantages**
1. Potent inhalational agent.
2. Sweet, nonirritating odor suitable for inhalational induction.

**B. Disadvantages**
1. Requires preservative.
2. Risk of halothane hepatitis (dysfunction).
3. Sensitizes myocardium to catecholamines more than other agents.
4. Causes vagal stimulation, which can result in marked bradycardia.
5. Potent trigger for malignant hyperthermia.
6. Relaxes uterine muscle.

C. Recommendations
1. Avoid repeat exposure within 6 months.
2. History of unexplained jaundice or pyrexia after a previous halothane anesthetic is a contraindication to repeat exposure.
3. Use caution with epinephrine. Avoid concentrations >1:100,000.

Nitrous oxide

A. Advantages
1. Powerful analgesic properties.
2. Decreases the MAC and accelerates the uptake of these agents.
3. Appears to be safe in patients with MH susceptibility.
5. No effect on smooth muscle.

B. Disadvantages
1. Decreases myocardial contractility (offset by stimulating effect on the SNS, increasing SVR). Also increases PVR in patients with preexisting pulmonary hypertension.
2. 35 times more soluble than nitrogen in blood, thus causing a rapid increase in the size of air-filled spaces. Also leads to diffusion hypoxia when N2O is stopped.
3. Supports combustion and can contribute to fires.
4. Increases risk of postoperative nausea and vomiting.
5. May increase intracranial pressure by increasing cerebral blood flow.
6. Inhibits methionine synthetase (prolonged exposure may lead to megaloblastic bone marrow changes).
7. Long-term use can lead to peripheral neuropathy.
8. Possible teratogenic effect
Common indications for endotracheal intubation: provide patent airway, protection from aspiration, facilitate positive-pressure ventilation, operative position other than supine, operative site near or involving the upper airway, airway maintenance by mask is difficult, disease involving the upper airway, one-lung ventilation, altered level of consciousness, tracheobronchial toilet, severe pulmonary or multisystem injury.

Confirmation of endotracheal Intubation
A. Direct visualization of the ET tube passing though the vocal cords.
B. Carbon dioxide in exhaled gases
C. Bilateral breath sounds.
D. Absence of air movement during epigastric auscultation.
E. Condensation (fogging) of water vapor in the tube during exhalation.
F. Refilling of reservoir bag during exhalation.
G. Maintenance of arterial oxygenation.
H. Chest x-ray: the tip of ET tube should be between the carina and thoracic inlet or approximately at the level of the aortic notch or at the level of T5.

Complications of endotracheal intubation
A. Complications occurring during intubation: aspiration, dental damage (chip tooth), laceration of the lips or gums, laryngeal injury, esophageal intubation, endobronchial intubation, activation of the sympathetic nervous system (high BP and HR), bronchospasm.
B. Complications occurring after extubation: aspiration, laryngospasm, transient vocal cord incompetence, glottic or subglottic edema, pharyngitis or tracheitis.

Endotracheal tube recommendations
A. Endotracheal tube size (mm): for children older then 2 years ETT can be estimated by:
   \[
   \text{Age/4 + 4.}
   \]
B. Length of Insertion (cm) of ETT
   1. Under 1 year: 6 + Wt(kg).
   2. Over 2 years: 12 + Age/2.
   3. Multiply internal diameter (mm) of ETT by 3 to give insertion (cm).
   4. Add 2-3 cm for nasal tube.
C. Pediatrics: generally use uncuffed tubes in patients under 10 years. When a cuff tube is used maintain endotracheal leak at 15-20 cm H2O2

Endotracheal Intubation under Anesthesia

Preparation for intubation
A. Preoperative evaluation of the airway will help determine the route (oral or nasal) and method (awake or anesthetized) for tracheal intubation.
B. Equipment: laryngoscope with working light, endotracheal tubes of appropriate sizes, malleable stylet, oxygen supply, functioning suction catheter, functioning IV, and appropriate anesthetic drugs.
C. Cricoid pressure (Sellick’s maneuver): used to minimize the spillage of gastric contents
into the pharynx during the period of time from induction of anesthesia (unconsciousness) to successful placement of a cuffed tracheal tube. An assistant’s thumb and index finger exert downward pressure on the cricoid cartilage (approximately 5 kg pressure) so as to displace the cartilaginous cricothyroid ring posteriorly and thus compress the esophagus against the underlying cervical vertebrae.

D. Induction of anesthesia prior to tracheal intubation may include injected and/or inhaled anesthetic drugs.

**Orotracheal intubation**

A. **Head position**: place the head in the “sniffing” position if there is no cervical spine injury. The sniffing position is characterized by flexion of the cervical spine and extension of the head at the atlanto-occipital joint (achieved by placing pads under the occiput to raise the head 8-10 cm). This position serves to align the oral, pharyngeal, and laryngeal axes such that the passage from the lips to the glottic opening is most nearly a straight line. The height of the OR table should be adjusted to bring the patient’s head to the level of the anesthesiologist’s xiphoid cartilage.

B. Hold the laryngoscope in the palm of the left hand and introduce the blade into the right side of the patient’s mouth. Advance the blade posteriorly and toward the midline, sweeping the tongue to the left. Check that the lower lip is not caught between the lower incisors and the laryngoscope blade. The placement of the blade is dependent on the blade used.

1. **Macintosh (curve) blade**: The tip of the curved blade is advanced into the valleculum (the space between the base of the tongue and the pharyngeal surface of the epiglottis).

2. **Miller (straight) blade**: The tip of the straight blade is passed beneath the laryngeal surface of the epiglottis, epiglottis is then lifted to expose the vocal cords.

C. Regardless of the blade used, lift the laryngoscope upward and forward, in the direction of the long axis of the handle, to bring the larynx into view. Do not use the upper incisors as a fulcrum for leverage because this action may damage the upper incisors and may push the larynx out of sight.

D. The vocal cords should be visualized prior to endotracheal placement. The glottic opening is recognized by its triangular shape and pale white vocal cords. Posteriorly, the vocal cords terminate in the arytenoid cartilages. The tube should be seen to pass between the cords, anterior to the arytenoids. Insert the tube into the pharynx with the right hand from the right side of the mouth; it should pass without resistance through the vocal cords (about 1-2 cm). The endotracheal tube cuff should lie in the upper trachea but beyond the larynx.

E. Once the endotracheal tube is in place, inflate the cuff, confirm endotracheal intubation and secure the endotracheal tube. In order to minimize the pressure transmitted to the tracheal mucosa, the cuff should be inflated with the least amount of air necessary to create a seal during positive pressure ventilation. For patients intubated outside the operating room, obtain a portable chest x-ray following intubation to confirm tube placement and bilateral lung expansion.

**Nasotracheal intubation**

A. A vasoconstrictor should be applied before nasal instrumentation. After anesthesia is induced the mask ventilation is established, the endotracheal tube can be placed.

B. Generously lubricate the nare and endotracheal tube. Soften the endotracheal tube tip by immersing it in hot water. The endotracheal tube should be advanced through the nose directly backward toward the nasopharynx with the Murphy eye orientated anteriorly facing the epiglottis. A loss of resistance marks the entry into the oropharynx.

C. The laryngoscope and Magill forceps can be used to guide the endotracheal tube into the
trachea under direct vision (if needed). A fiberoptic bronchoscope can be utilized to direct the tube into the trachea.

**Rapid sequence induction/intubation**

**Indications:** patients who are at risk for aspiration (eg, history of recent meal, gastroesophageal reflux, pregnancy, trauma) and there is reasonable certainty that intubation should not be difficult.

---

### Fiberoptic-Assisted Tracheal Intubation

**Indications:** upper airway obstruction, mediastinal mass, subglottic edema, congenital upper airway abnormalities, immobile cervical vertebrae, verify position of a double-lumen endobronchial tube.

**Nasal technique:** after the patient’s nares and nasopharynx are anesthetized and vasoconstricted, the tracheal tube is passed through the naris into the posterior nasopharynx. The lubricated bronchoscope is then passed through the tracheal tube until the epiglottis and glottic opening are visualized continuing until the carina is identified. Pass the tube over the scope while the view of the carina is maintained.

**Oral technique:** an intubating oral airway (or bite block) is inserted after topicalization of the posterior tongue, soft palate, and lateral oropharyngeal areas. The tracheal tube is inserted about 8-10 cm into the airway and the bronchoscope passed through the tube. The posterior tongue, epiglottis, glottis and carina should be visualized in order. The tube is then passed over the scope while keeping the carina in view.

---

### Transtracheal Ventilation (Cricothyrotomy)

**Indications:** can be used as a temporizing measure if mask ventilation and oxygenation become inadequate or is not possible.

**Technique:** a catheter (12- or 14-gauge) is connected to a jet-type ventilator, which in turn is connected to an oxygen source capable of delivering gas at a pressure around 50 psi, and inserted into the trachea through the cricothyroid membrane. The gas is delivered intermittently by a hand-held actuator. The duration of ventilation is best assessed by watching the rise and fall of the chest: an I:E ratio of 1:4 seconds is recommended. Oxygenation usually improves rapidly, however, retention of carbon dioxide may limit the duration of the usefulness of the technique.

**Complications:** catheter displacement (caused by high pressures created by jet ventilation), pneumomediastinum.

---

### Laryngeal Mask Airway

**Indications for LMA**

A. In place of a face mask or endotracheal tube.

B. In place of an endotracheal tube, when breathing is being controlled, as long as the inflation pressure is not more than 30 cm H2O.

C. To aid in the management of the difficult airway (ie, the LMA can be used as a guide for fiberoptic intubation).
Contraindications for LMA
A. The LMA does not provide an airtight seal of the airway and, thus, does not protect against gastric regurgitation and pulmonary aspiration.
B. When controlled ventilation is likely to require a high-inflation pressure of more than 30 cm H2O.

Insertion of the LMA-Classic
A. Propofol (2.5-3.0 mg/kg) is the agent of choice for LMA insertion. Propofol relaxes the jaw and pharyngeal muscles better than thiopental.
B. The leading edge of the deflated cuff should be wrinkle-free and facing away from the aperture. Lubricate only the back side of the cuff with a water soluble lubricant.
C. The LMA is held like a pencil and is inserted blindly in the midline with concavity forward while pressing on the anterior shaft with the tip of the index finger toward the hard palate and guiding it toward the pharynx.
D. When the upper esophageal sphincter is reached, a characteristic resistance is felt. The cuff is then inflated with air (the cuff should be inflated without holding the tube to enable the expanding cuff to find its correct position in the pharynx).
E. When correctly placed, the black vertical line on the posterior aspect of the tube should always face directly backward, toward the head of the patient.
F. The LMA should be left in place until the patient can open his mouth on command. During emergence, the patient should not be stimulated (ie, suctioned), and the cuff should not be deflated until the patient can open his mouth on command.
G. A bite block (or folded gauze) is inserted in the mouth to protect the LMA.

Complications of the LMA
A. Possibility of regurgitation and pulmonary aspiration.
B. Oral and pharyngeal mucosa injury during insertion of the LMA.
C. Laryngospasm and coughing (may occur in lightly anesthetized patient).
D. Negative pressure pulmonary edema after improper placement in spontaneously breathing patient.
E. The failure to function properly in the presence of local pharyngeal or laryngeal disease.
F. The need for neck extension in the patient with cervical spine disorder.

<table>
<thead>
<tr>
<th>Laryngeal Mask Airway (LMA) Sizes</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Size</strong></td>
</tr>
<tr>
<td>--------</td>
</tr>
<tr>
<td>1</td>
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<tr>
<td></td>
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<tr>
<td>2</td>
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<tr>
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<tr>
<td>4</td>
</tr>
<tr>
<td>5</td>
</tr>
<tr>
<td>6</td>
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</tbody>
</table>

ETT= endotracheal tube; * = cuffed tube; ID = inner diameter
**Mechanical Ventilation**

### Types of mechanical ventilators

A. **Time cycled**: the tidal volume is delivered and inspiration ends after a preset time interval.

B. **Volume cycled**: the tidal volume is delivered and inspiration ends after a preset time interval.

C. **Pressure cycled**: the tidal volume is delivered and inspiration ends when a preset volume is delivered.

### Modes of mechanical ventilation

A. **Intermittent positive-pressure ventilatory modes (IPPV)**
   1. **Controlled mechanical ventilation (CMV)**: mechanical breaths are delivered at a preset rate and tidal volume regardless of the pt effort.
   2. **Assist-control ventilation (AC)**: A preset minute ventilation is delivered regardless of the patient’s effort. Ventilator senses patient- initiated spontaneous breath and delivers a preset tidal volume as well.
   3. **Intermittent mandatory ventilation (IMV)**: the ventilator provides tidal volume breaths at a preset fixed rate. In between ventilator-delivered breaths, the patient is able to breathe spontaneously at any rate, tidal volume, or pattern.
   4. **Synchronized intermittent mandatory ventilation (SIMV)**: similar to IMV, ventilatory breaths timed to coincide with spontaneous effort.
   5. **Continuous positive airway pressure (CPAP)**: a preset level of positive airway pressure is maintained throughout the respiratory cycle. The patient must be spontaneously breathing.
   6. **Inspiratory pressure support ventilation (IPS)**: a preset pressure is obtained when the patient initiates an inspiratory effort.

B. **Pressure-controlled ventilation**
   1. Maximum airway pressure is set on the ventilator, and tidal volume becomes the dependent variable.
   2. The duration of inspiration is determined by setting either the inspiratory time or the I:E ratio. Tidal volume is the product of inspiratory flow and inspiratory time.
   3. The primary advantage of pressure-controlled ventilation is reduction in peak airway pressure and potential improvement of gas exchange.

C. **High-frequency ventilation**
   1. **High-frequency positive pressure ventilation (HFPPV)**: similar to conventional ventilation, however, tidal volumes are very small, and cycling frequencies are very fast (60-300).
   2. **High-frequency jet ventilation (HFJV)**: a small diameter injecting catheter positioned in the central airway pulses gas along the luminal axis under high pressure at a rapid cycling rate.

D. **Pressure-controlled inverse ratio ventilation (PC-IRV)**: set by choosing a prolonged inspiratory time such that the time spend during inspiration exceeds expiratory time.

### Positive end-expiratory pressure (PEEP)

**Function of PEEP**: increases oxygenation by maximizing the ventilation-perfusion relationship in the lung. PEEP does this by maximizing the FRC (functional residual capacity), keeping lung volumes greater than closing capacity, therefore maintaining airways
open and functional.

**Adverse effects of PEEP:** decreased cardiac output, hypotension, worsening hypoxia, barotrauma, increased ICP, decreased urine output.

### Ventilator Settings

**FIO2:** normally start with 40% otherwise use 90-100% until first ABG available (1% decrease in FIO2 = decrease PaO2 by 7).

**PEEP:** initially none; start with 5 cm H2O and increase in 3-5 cm H2O increments if PaO2 less than 60 mm Hg with FIO2 > 50%; over 10 cm H2O normally requires pulmonary artery catheter.

**Rate:** start at 10-14 (for infants start at 25-30).

**Tidal volume:** 10-15 mL/kg (infants 8-12 mL/kg).

**Mode:** IMV, SIMV, CPAP, A/C, PSV.

### Oxygen Therapy

**Nasal cannulas:** FIO2 increases by 3-4%/liter of O2 given (up to 40%); high flow rates may cause dry mucous membranes, gastric distension, headaches.

**Face masks**

A. **Simple mask:** insufficient flow rates may cause CO2 retention.

B. **Venturi mask (air entrainment mask):** useful in COPD; if back pressure develops on jet, less room air enters and FIO2 can elevate unpredictably.

C. **Partial rebreathing mask:** simple mask with a valveless reservoir bag and exhalation ports; collapsed reservoir bag indicates air leak or inadequate flow of oxygen.

D. **Nonrebreathing mask:** simple mask with reservoir bag and unidirectional valve; requires tight seal and high flow rate to deliver maximum FIO2.

E. **Tracheostomy mask:** provides humidity and controlled oxygen; FIO2 should be analyzed for each individual patient.

F. **Aerosol face tent:** delivers oxygen form variable oxygen nebulizer over mouth and nose.

### Oxygen tents

A. Insufficient flow rates may cause CO2 retention; can be used to provide humidified air.

B. **Disadvantages:** development of oxygen gradient; sparks in or near tent may be hazardous; claustrophobia in older children; requires close monitoring of patient and apparatus.

### Oxygen Delivery Systems

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<th>FIO2 Delivered (%)</th>
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<td>Nasal cannula</td>
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<td>Venturi mask</td>
<td>24-50 (mask specific)</td>
<td>Variable</td>
</tr>
</tbody>
</table>
**Functional fluid compartments**

A. Total body water (TBW): 60% (adult males) and 50% (adult females) of ideal body weight (IBW).

B. Intracellular fluid (ICF): comprises approximately 35% of IBW or 60% of TBW. Principal potassium containing space.

C. Extracellular fluid (ECF): accounts for 25% of IBW or 40% of TBW and is subdivided into interstitial fluid (ISF) and blood volume (BV; about 8% of TBW). Principal sodium containing space.

**Daily electrolyte requirements**

A. Na: 2-3 mEq/kg/24 hours  B. K: 1-2 mEq/kg/24 hours  C. Cl: 2-3 mEq/kg/24 hours

**Perioperative fluid replacement**

A. Normal maintenance requirements (hourly rate based on weight)
   1. First 10 kg: 4 mL/kg/hr or 100 mL/kg/day
   2. Second 10 kg: add 2 mL/kg/hr or 50 mL/kg/day
   3. >20 kg: add 1 mL/kg/hr or 20 mL/kg/day

B. Fluid deficit: primarily NPO deficit caused by patient fasting.
   1. NPO deficit = hourly maintenance rate x number of hours NPO
   2. Replace the first half pre-op and the remaining over the next 2-3 hours.

C. Intraoperative fluid loss: primarily third-space (redistribution) and evaporative losses; amount based on degree of tissue trauma.
   1. Minimal (eg, herniorrhaphy): 0-2 mL/kg/hr
   2. Moderate (eg, cholecystectomy): 2-4 mL/kg/hr
   3. Severe (eg, bowel resection): 4-8 mL/kg/hr

D. Blood loss (see blood therapy section)
   1. Replace each mL of blood loss with 3 mL of crystalloid, 1 mL colloid solution or 1 mL PRBC.
   2. Transfusion of red blood cells as necessary to maintain hematocrit.

**Calculated osmolality** = 2 Na + glucose/18 +BUN/2.8 + ethanol/4.6 + isopropanol/6 + methanol/3.2 + ethylene glycol/6.2 (norm 280-295).

<table>
<thead>
<tr>
<th>Fluid</th>
<th>Glu</th>
<th>Na</th>
<th>Cl</th>
<th>K</th>
<th>Ca</th>
<th>HCO₃</th>
<th>Kcal/ L</th>
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<tbody>
<tr>
<td>D5W</td>
<td>50</td>
<td></td>
<td></td>
<td></td>
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<td>NS</td>
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<td>154</td>
<td>154</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>D51/4NS</td>
<td>50</td>
<td>38.5</td>
<td>38.5</td>
<td>4</td>
<td>3</td>
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<td>LR</td>
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<td>130</td>
<td>110</td>
<td>4</td>
<td>3</td>
<td>27</td>
<td>&lt;10</td>
</tr>
</tbody>
</table>
Transfusions

1. Blood loss management
   A. Estimated blood volume (EBV).
      1. 95-100 mL/kg for premature infant.
      2. 85-90 mL/kg for full-term infant.
      3. 80 mL/kg for infants up to 12 months.
      4. 70-75 mL/kg for adult men.
      5. 65-70 mL/kg for adult women.
   B. Max allowable blood loss = [EBV x (Hct – target Hct)]/ Hct.
   C. Replace every 1 mL blood loss with 3 mL crystalloid or 1 cc PRBC.

D. PRBC transfusion guidelines
   1. one unit PRC increases Hct about 3% and Hb about 1 g/dL in adults.
   2. 3 mL/kg PRC increases Hb about 1 g/dL.
   3. 10 mL/kg PRBC increases Hct about 10%.

E. Fluid replacement equivalents
   1. Crystalloid: 3 cc/1 cc estimated blood loss (EBL).
   2. Colloid: 1 cc/cc EBL.
   3. Whole blood: 1 cc/cc EBL.
   4. Packed red blood cells (pRBC): 1/2 cc/cc EBL.

Compatibility testing

A. Type specific: ABO-Rh typing only; 99.80% compatible.
B. Type and screen: ABO-Rh and screen; 99.94% compatible.
C. Type and crossmatch: ABO-Rh, screen, and crossmatch; 99.95% compatible. Crossmatching confirms ABO-Rh typing, detects antibodies to the other blood group systems, and detects antibodies in low titers.
D. Screening donor blood: hematocrit is determined, if normal, the blood is typed, screened for antibodies, and tested for hepatitis B, hepatitis C, syphilis, HIV-1, HIV-2, and human T-cell lymphotropic viurses I and II. ALT is also measured as a surrogate marker of nonspecific liver infection.

Blood component therapy

A. Whole blood: 40% hematocrit; used primarily in hemorrhagic shock.
B. Packed red blood cells (PRBC): volume 250-300 mL with a hematocrit of 70-80%; increases adult hemoglobin approximately 1 g/dL.
C. Platelets
   1. One unit of platelets will increases platelet count 5000-10,000/mm³; usual dose is 1 unit of platelets per 10 kg body weight; single-donor platelets obtained by apheresis are equivalent to 6 platelet concentrate; platelets are stored at room temp; ABO compatibility is not mandatory.
   2. A normal platelet count is 150,00-440,000/mm³. Thrombocytopenia is defined as <150,000/mm³. Intraoperative bleeding increases with counts of 40,000-70,000/mm³, and spontaneous bleeding can occur at counts <20,000/mm³. During surgery platelet transfusions are probably not required unless count is less then 50,000/mm³.
D. Fresh frozen plasma (FFP): 250 cc/bag; contains all coag factors except platelets; 10-15 mL/kg will increase plasma coag factors to 30% of normal; fibrinogen levels increase by 1 mg per mL of plasma transfused; reversal of warfarin requires 5-8 mL/kg of FFP. ABO compatibility is required.

E. Cryoprecipitate: 10-20 mL/bag; contains 100 units factor VIII-C, 100 units factor vWF, 60 units factor XIII, and 250 mg fibrinogen; indications include hypofibrinogenemia, von Willebrand disease, hemophilia A and preparation of fibrin glue; ABO compatibility not mandatory.

F. Albumisol: 5% and 25% (heat treated at 60 degrees C for 10 hrs).

Complications of transfusions

A. Immune complications

1. Hemolytic reactions
   A. Acute hemolytic reactions
      1. Occurs when ABO-incompatible blood is transfused resulting in acute intravascular hemolysis; severity of a reaction often depends on how much incompatible blood has been given.
      2. Symptoms include fever, chills, chest pain, anxiety, back pain, dyspnea; in anesthetized patients, the reaction is manifested by rise in temperature, unexplained tachycardia, hypotension, hemoglobinuria, and diffuse oozing from surgical site. Free hemoglobin in the plasma or urine is presumptive evidence of a hemolytic reaction.
      3. Risk of fatal hemolytic transfusion reaction: 1:600,000 units.

   B. Delayed hemolytic reactions
      1. Occurs because of incompatibility of minor antigens (eg, Kidd, Kelly, Duffy, etc) are characterized by extravascular hemolysis.
      2. The hemolytic reaction is typically delayed 2-21 days after transfusion, and symptoms are generally mild, consisting of malaise, jaundice, and fever. Treatment is supportive.

2. Nonhemolytic reactions
   A. Febrile reactions
      Most common nonhemolytic reaction (0.5-1.0% of RBC transfusions and up to 30% of platelet transfusions); due to recipient antibodies against donor antigens present on leukocytes and platelets; treatment includes stopping or slowing infusion and antipyretics.

   B. Urticarial reactions
      1. Characterized by erythema, hives, and itching without fever.
      2. Occur in 1% of transfusions and are thought to be due to sensitization of the patient to transfused plasma proteins.
      3. Treated with antihistaminic drugs.

   C. Anaphylactic reactions
      1. Anaphylactic reactions are rare; about 1:500,000.
      2. Patients with IgA deficiency may be at increased risk of the presence of anti-IgA antibodies that react with transfused IgA.

   D. Transfusion related acute lung injury (TRALI)
      1. Due to transfusion of antileukocytic or anti-HLA antibodies that interact with and cause the patient’s white cells to aggregate in the pulmonary circulation.
      2. Risk is 1:6000.
      3. Treatment is supportive, mimicking the treatment of ARDS.

   E. Graft-vs-host disease
      1. Most commonly seen in immune-compromised patients.
2. Cellular blood products contain lymphocytes capable of mounting an immune response against the compromised host.

F. Posttransfusion purpura
1. Due to the development of platelet alloantibodies; the platelet count typically drops precipitously 1 week after transfusion.

G. Immune suppression
1. Transfusion of leukocyte-containing blood products appears to be immunosuppressive (can improve allograft survival following renal transplants).
2. Blood transfusions may increase the incidence of serious infections following surgery or trauma.
3. Blood transfusions may worsen tumor recurrence and mortality rate following resections of many cancer.

B. Infectious complications
1. Viral infections
   A. Hepatitis: risk of HBV: 1:137,000; HCV: Hepatitis C: 1:1,000,000.
   B. HIV/AIDS: risk of HIV: 1:1,900,000
   C. Other viral infections
2. Parasitic infections: very rare; include malaria, toxoplasmosis, and Chagas’ disease.
3. Bacterial infections

C. Massive transfusions: is defined as the replacement of a patient's total blood volume in less than 24 hours, or as the acute administration of more than half the patient's estimated blood volume per hour.

D. Other complications
1. Metabolic abnormalities
   A. Decreased pH secondary to increased hydrogen ion production.
   B. Increase potassium: due to cell lysis; increases with length of storage.
   C. Decrease in 2,3 DPG: consumed by RBCs; P50 decreases to 18 mm Hg after 1 week and 15 mm Hg after 3 weeks.
   D. Citrate toxicity:
2. Microaggregates consisting of platelets and leukocytes form during storage of whole blood. Micropore filters may decrease help remove these particles.
3. Hypothermia: the use of blood warmers (except for platelets) greatly decreases the likelihood of transfusion-related hypothermia.
4. Coagulopathy disorders
   A. Usually occurs only after massive transfusion(greater than 10 units).
   B. Dilutional thrombocytopenia: common cause of abnormal bleeding in massive transfusion, responds quickly to platelet transfusions.
   C. Low Factors V and VIII: factors V and VII are very labile in stored blood and may decrease to levels as low as 15-20% normal, however, this is usually enough for hemostasis.
   D. Disseminated Intravascular Coagulation: a hypercoagulable state caused by activation of the clotting system leading to deposition of fibrin in microvasculature which causes a secondary activation of fibrinolysis. resulting in consumption of factors and platelets
### Contraindications to peridural anesthesia

**A. Absolute contraindications:** lack of patient consent, localized infection at injection site, allergy to local anesthetics, increased intracranial pressure, coagulopathy or other bleeding diathesis, severe hypovolemia, severe aortic or mitral stenosis.

**B. Relative contraindications:** localized infection peripheral to regional site, demyelinating CNS disease, patients taking platelet inhibiting drugs.

**C. Controversial:** prior back surgery at site of injection, inability to communicate with patient, complicated surgery.

### Anatomy

**A. Spinal canal:** extends from the foramen magnum to the sacral hiatus.

**B. Spinal cord:** spinal cord extends the length of the vertebral canal during fetal life, ends at L3 at birth, and moves progressively cephalad to reach the adult position of L1-L2 by 2 years of age.

**C. Subarachnoid space:** subarachnoid space lies between the pia mater and the arachnoid and extends from S2 to the cerebral ventricles.

**D. Epidural space** contains nerve roots, fat, lymphatic and blood vessels, and areolar tissue.

**E. Course of anatomy:** skin, subcutaneous tissue, supraspinous ligament, interspinous ligament, ligamentum flavum, epidural space, and dura.

### Physiological changes with spinal and epidural anesthesia

**A. Neural blockade**

1. **Sequence of neural blockade**
   - A. Sympathetic block with peripheral vasodilation and skin temperature elevation.
   - B. Loss of pain and temperature sensation.
   - C. Loss of proprioception.
   - D. Loss of touch and pressure sensation.
   - E. Motor paralysis.

2. The above sequence of neural blockade occurs because smaller C fibers are blocked more easily than the larger sensory fiber, which in turn are blocked more easily than motor fibers. As a result, the level of autonomic blockade for a spinal anesthetic extends above the level of the sensory blockade by 2-3 segments, while the motor blockade is 2-3 segments below the sensory blockade. During epidural anesthesia there is not a zone of differential nervous system blockade, and the zone of differential motor blockade averages 4 segments below the sensory level.

3. With epidural anesthesia, the local anesthetics act directly on the spinal nerve roots located in the lateral part of the space. To a lesser extent, diffusion of local anesthetic solutions from the epidural space into the subarachnoid space produces spinal cord effects. As a result, the onset of the block is slower than with spinal anesthesia, and the intensity of the sensory and motor block is less.

**B. Cardiovascular**

1. **Hypotension:** The degree of hypotension is directly proportional to the degree of sympathetic blockade.

2. **Blockade above T4** interrupts cardiac sympathetic fibers, leading to bradycardia,
Preparation

**Ropi**

**Lidocaine**

Drug

Local

Complications of epidural anesthesia

Factors influencing epidural anesthesia

1. **Respiratory:** With ascending height of the block into the thoracic area, there is a progressive, ascending intercostal muscle paralysis. The dia-phragmatic ventilation is mediated by the phrenic nerve, and typically will remain unaffected even during high cervical blockade.

2. **Visceral effects**
   1. **Bladder:** Sacral blockade results in an atonic bladder.
   2. **Intestine:** With sympathectomy, vagal tone dominates and results in a small, contracted gut with active peristalsis.

Spinal Anesthesia

Factors influencing spinal anesthetic

A. Primary factors: baricity of anesthetic solution, position of the patient, drug dosage, site of injection.

B. Other factors: age, CSF volume, curvature of spine, drug volume, intra-abdominal pressure, needle direction, patient height, pregnancy.

Vasoconstrictors

A. Enhances the quality and duration of spinal anesthesia by decreasing the uptake and clearance of local anesthetic from CSF.

B. Agents: epinephrine (0.1-0.2 mg) and phenylephrine (1-2 mg)

<table>
<thead>
<tr>
<th>Local Anesthetics: Dosages for Spinal Anesthesia</th>
</tr>
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<tbody>
<tr>
<td>Drug</td>
</tr>
<tr>
<td>------</td>
</tr>
<tr>
<td>Lidocaine</td>
</tr>
<tr>
<td>Bupivacaine</td>
</tr>
<tr>
<td>Ropivacaine</td>
</tr>
</tbody>
</table>

*For hypobaric spinal: tetracaine diluted with sterile water to 0.3% solution

**Preparation concentration of tetracaine is 1%; tetracaine is diluted with 5.0% glucose for hyperbaric solution and normal saline for isobaric solution

Epidural Anesthesia

Factors influencing epidural anesthesia

A. Local anesthetic selected.

B. Mass of drug injected (dose, volume, and concentration).

C. Addition of vasoconstrictors to reduce systemic absorption.

D. Site injection.

E. Patients over 40 years of age.

F. Pregnancy (hormonal and/or mechanical factors).

Epidural insertion sites: cervical interspaces through T4 are best accessed by a median approach, while a paramedian approach for T4-T9 and a median approach for T9-L5.

Complications of epidural anesthesia (in addition to those listed above)

A. Dural puncture: unintentional dural puncture occurs in 1% of epidural injections performed.
B. Catheter complications
1. Inability to insert the catheter.
2. Catheter can be inserted into an epidural vein.
3. Catheters can break off or become knotted within the epidural space.
5. **Intravascular injection:** may result in local anesthetic overdose where large amounts of local anesthetic are used.
6. Direct spinal cord injury.
7. **Bloody tap:** may result from perforation of an epidural vein.

**Clinical pharmacology of epidural agents**

A. Dosing
1. **Adults:** 1-2 mL of local anesthetic per segment to be blocked.
2. **Time to two-segment regression**
   A. The time it takes for a sensory level to decrease by two dermatone levels.
   B. When a two-segment regression has occurred, one can reinject one-third to half the initial activation dose.

B. **Hydrophilic opioids** (morphine, hydromorphone)
1. **Properties:** slow onset, long duration, high CSF solubility, extensive CSF spread.
2. **Advantages:** prolonged single-dose analgesia, thoracic analgesia with lumbar administration, minimal dose compared to IV administration.
3. **Disadvantages:** delayed onset of analgesia, unpredictable duration, higher incidence of side effects, delayed respiratory depression.

C. **Lipophilic opioids** (fentanyl, sufentanil)
1. **Properties:** rapid onset, short duration, low CSF solubility, minimal CSF spread.
2. **Advantages:** rapid analgesia, decreased side effects, ideal for continuous infusion or PCEA.
3. **Disadvantages:** systemic absorption, brief single-dose analgesia, limited thoracic analgesia with lumbar administration.

D. **Additives**
1. **Epinephrine** may be added to a maximum concentration of 1:200,000.
2. **Bicarbonate,** 1 cc for each 10 mL of local anesthetic, can be added to speed up onset.

E. **Side effects of neuraxial opioids (see table).**

<table>
<thead>
<tr>
<th>Local Anesthetics: Dosages for Epidural Anesthesia</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Drug</strong></td>
</tr>
<tr>
<td>----------</td>
</tr>
<tr>
<td>Lidocaine</td>
</tr>
<tr>
<td>Mepivacaine</td>
</tr>
<tr>
<td>Bupivacaine</td>
</tr>
<tr>
<td>Ropivacaine</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Neuraxial Opioids: Side Effects and Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Problem</strong></td>
</tr>
<tr>
<td>Pruritus</td>
</tr>
</tbody>
</table>
| Change | Diphenhydramine 25-50 mg IV  
Naloxone 40-80 mcg IV | intrathecal morphine |
|---|---|---|
| Nausea/ Vomiting | Metoclopramide 5-10 mg IV  
Nalbuphine 5-10 mg IV/IM  
Naloxone 40-80 mcg IV | |
| Respiratory Depression | Naloxone 0.1 mg IV prn | Watch for synergism with other sedatives |
| Urinary Retention | Urinary Catheter | |
| Blood Pressure Changes | Fluid hydration  
Ephedrine  
Phenylephrine | Most likely after meperidine (local anesthetic effects) |

### Complications of Neuraxial Blocks

1. **Backache**: usually benign, mild and self-limited; can treat with tylenol, ASA, NSAIDs, warm or cold packs; may be a clinical sign of a more serious complications, such as epidural hematoma or abscess.

2. **Postdural puncture headache (PDPH)**
   A. Characteristics of a postdural puncture headache: postural component (made worse by upright position), frontal or occipital location, tinnitus, diplopia, young females, use of a large-gauge needle.
   B. **Mechanism**: usually due to a continued leak of CSF through the hole in the dura mater, resulting in low CSF pressure, which causes traction on meningeal vessels and nerves.
   C. **Incidence**: the overall incidence is approximately 5-10%.

3. **Treatment of a postdural puncture headache**
   1. Oral Analgesics.
   2. Bed rest.
   3. Hydration (IV fluids, PO fluids, caffeine containing beverages).
   4. Caffeine infusion (500 mg caffeine and sodium benzoate in 1 liter of isotonic crystalloid given over 1-2 hours).
   5. Epidural blood patch (placement of 10-20 cc of autologous blood in the epidural space). The success rate is approximately 95% (90% respond after initial patch; 90% of initial non-responders after second patch).

3. **Urinary retention**
   A. Local anesthetic block of S2-S4 root fibers decreases urinary bladder tone and inhibits the voiding reflex.
   B. Neuraxial opioids can interfere with normal voiding.

4. **Maternal fever**: commonly seen in epidural analgesia for labor resulting from epidural induced shivering or inhibition of sweating and hyperventilation.

5. **Transient neurologic symptoms (TNS)**
   A. Characterized by back pain radiating to the legs without sensory or motor deficits,
occurring after the resolution of the block; usually resolves spontaneously within several days; pathogenesis unclear.

6. High or total spinal anesthesia
   A. Can result in hypotension, bradycardia, and respiratory insufficiency.
   B. Treatment consists of supporting airway, ventilation, and circulation.

7. Subdural injection (during attempted epidural anesthesia)
   A. Similar presentation as high spinal but with slower onset.
   B. Treatment is supportive.

8. Systemic toxicity
   A. Extreme high levels of local anesthetics affects the CNS (seizure and unconsciousness) and the cardiovascular system (hypotension, arrhythmias, cardiovascular collapse).

9. Cauda equina syndrome
   A. Characterized by bowel and bladder dysfunction together with evidence of multiple nerve root injury.

10. Meningitis and arachnoiditis

11. Epidural abscess
   A. Reported incidence: 1:6500 to 1:500,000 epidurals.
   B. Four classic clinical stages
      1. Back or vertebral pain that is intensified by percussion over spine.
      2. Nerve root or radicular pain develops.
      3. Motor and/or sensory deficits or sphincter dysfunction.
      4. Paraplegia or paralysis.
   C. If suspected: remove catheter (if placed), blood cultures, MRI or CT scan.

12. Spinal or epidural hematoma
   A. Incidence of clinically significant hematoma:
      1:150,000 for epidurals and 1:220,000 for spinals.
   B. Majority occur in patients with abnormal coagulation.

Postanesthesia Care Unit

Postoperative Hemodynamic Complications

1. Hypotension
   A. Causes: arterial hypoxemia, hypovolemia (most common), decreased myocardial contractility (myocardial ischemia, pulmonary edema), decreased systemic vascular resistance (neuroaxial anesthesia, sepsis), cardiac dysrhythmias, pulmonary embolus, pneumothorax, cardiac tamponade, spurious (large cuff).
   B. Treatment: fluid challenge; pharmacologic treatment includes inotropic agents (dopamine, dobutamine, epinephrine) and alpha receptor agonists (phenylephrine, epinephrine). CVP and PA catheter monitoring may be needed to guide therapy.

2. Hypertension
   A. Causes: enhanced SNS activity (pain, gastric distension, bladder distension), preoperative hypertension, hypervolemia, hypoxemia, spurious (small cuff), increased intracranial pressure, and vasopressors.
   B. Treatment: management begins with identification and correction of the initiating cause; various medications can be used to treat hypertension including beta-blockers
3. Cardiac dysrhythmias
A. Causes: arterial hypoxemia, hypercarbia, hypovolemia, pain, electrolyte and acid-base imbalances, myocardial ischemia, increased ICP, drug toxicity (digitalis), hypothermia, anticholinesterases and malignant hyperthermia.
B. Treatment: supplemental oxygen should be given while the etiology is being investigated; most dysrhythmias do not require treatment.

Postoperative Respiratory and Airway Complications
1. Respiratory problems are the most frequently encountered complications in the PACU, with the majority related to airway obstruction, hypoventilation, or hypoxemia.
2. Hypoxemia
A. Causes: right-to-left intrapulmonary shunt (atelectasis), mismatching of ventilation-to-perfusion (decreased functional residual capacity), decreased cardiac output, alveolar hypoventilation, diffusion hypoxia, upper airway obstruction, bronchospasm, aspiration of gastric contents, pulmonary edema, pneumothorax and pulmonary embolism, obesity, advanced age, and posthyperventilation hypoxia.
B. Clinical signs of hypoxia (restlessness, tachycardia, cardiac irritability hypertension, hypotension) are nonspecific; obtunded, bradycardia, hypotension, and cardiac arrest are late signs.
C. Increased intrapulmonary shunting relative to closing capacity is the most common cause of hypoxemia following general anesthesia.
D. Treatment: oxygen therapy with or without positive airway pressure. Additional treatment should be directed at the underlying cause.
3. Hypoventilation
A. Causes: drug-induced central nervous system depression (residual anesthesia), suboptimal ventilatory muscle mechanics, increased production of carbon dioxide, decreased ventilatory drive, pulmonary, and respiratory muscle insufficiency (pre-existent respiratory disease, inadequate reversal of neuromuscular blockade, inadequate analgesia, and bronchospasm).
B. Hypoventilation in the PACU is most commonly caused by residual depressant effects of anesthetic agents on respiratory drive or persistent neuromuscular blockade.
C. Treatment: should be directed at the underlying cause. Marked hypoventilation may require controlled ventilation until contributory factors are identified and corrected.
4. Upper airway obstruction (stridor)
A. Causes: include incomplete anesthetic recovery, laryngospasm, airway edema, wound hematoma, and vocal cord paralysis. Airway obstruction in unconscious patients is most commonly due to the tongue falling back against the posterior pharynx.
B. Treatment: supplemental oxygen while corrective measures are undertaken. Jaw thrust, head-tilt, oral or nasal airways often alleviate the problem.
5. Laryngospasm and laryngeal edema
A. Laryngospasm is a forceful involuntary spasm of the laryngeal musculature caused by sensory stimulation of the superior laryngeal nerve. Triggering stimuli include pharyngeal secretions or extubating in stage 2. The large
negative intrathoracic pressures generated by the struggling patient in laryngospasm can cause pulmonary edema.

B. Treatment of laryngospasm: initial treatment includes 100% oxygen, anterior mandibular displacement, and gentle CPAP (may be applied by face mask). If laryngospasm persists and hypoxia develops, succinylcholine (0.25-1.0 mg/kg; 10-20 mg) should be given in order to paralyze the laryngeal muscles and allow controlled ventilation.

C. Treatment of glottic edema and subglottic edema: administer warm, humidified oxygen by mask, inhalation of racemic epinephrine 2.25% (0.5-1 mL in 2 mL NS), repeated every 20 minutes, dexamethasone 0.1-0.5 mg/kg IV may be considered. Reintubation with a smaller tube may be helpful.

Postoperative Neurologic Complications

1. Delayed awakening: the most frequent cause of a delayed awakening is the persistent effect of anesthesia or sedation. Other causes include recurarization, severe hypothermia, hypoglycemia, and neurologic disorders.

2. Emergence delirium (agitation): is characterized by excitement, alternating with lethargy, disorientation, and inappropriate behavior. Potential causes include arterial hypoxemia, hypercapnia, pain, unrecognized gastric dilation, urinary retention, and previous administration of atropine. Treatment includes haloperidol, titrated in 1-2 mg IV increments. Benzodiazepines may be added if agitation is severe. Phystostigmine (0.5-2.0 mg IV) may reverse anti-cholinergic delirium.

Postoperative Nausea and Vomiting

1. Risk factors
   A. Patient risk factors: short fasting status, anxiety, younger age, female, obesity, gastroparesis, pain, history of postoperative nausea/vomiting or motion sickness.
   B. Surgery-related factors: gynecological, abdominal, ENT, ophthalmic, and plastic surgery; endocrine effects of surgery; duration of surgery.
   C. Anesthesia-related factors: premedicants (morphine and other opioids), anesthetics agents (nitrous oxide, inhalational agents, etomidate, methohexital, ketamine), anticholinesterase reversal agents, gastric distention, longer duration of anesthesia, mask ventilation, intraoperative pain medications, regional anesthesia(lower risk).
   D. Postoperative factors: pain, dizziness, movement after surgery, premature oral intake, opioid administration.

Postanesthesia Care Unit Pain Control

1. Moderate-to-severe postoperative pain in the PACU
   A. Meperidine 25-150 mg (0.25-0.5 mg/kg in children).
   B. Morphine 2-4 mg (0.025-0.05 mg/kg in children).
   C. Fentanyl 12.5-50 mcg IV.

2. Nonsteroidal anti-inflammatory drugs are an effective complement to opioids. Ketorolac 30 mg IV followed by 15 mg q6-8h.

3. Patient-controlled and continuous epidural analgesia should be started in the PACU.
Treatmen t of Postoperative Nausea and Vomiting (PONV)

<table>
<thead>
<tr>
<th>Drug</th>
<th>Adult Dose</th>
<th>Peds Dose</th>
<th>Duration</th>
<th>Caution Use In</th>
</tr>
</thead>
<tbody>
<tr>
<td>Droperidol</td>
<td>0.625-1.25mg IV/IM</td>
<td>50-75mg/kg IV/IM</td>
<td>3-4 hr</td>
<td>Parkinson, hypovolemia</td>
</tr>
<tr>
<td>Metoclopramide</td>
<td>10 mg IV/IM (max 20 mg)</td>
<td>0.1 mg/kg (max 5 mg)</td>
<td>1-2 hr</td>
<td>GI obstruction, seizures, Parkinson</td>
</tr>
<tr>
<td>Trimethobenzamide</td>
<td>200 mg IM/PR</td>
<td>&lt;14 kg: 100 mg &gt;14 kg: 100-200 mg</td>
<td>6-8 hr</td>
<td>Benzocaine allergy, Reye’s syndrome</td>
</tr>
<tr>
<td>Ondansetron</td>
<td>4-8 mg IV</td>
<td>0.1 mg/kg IV</td>
<td>4-6 hr</td>
<td>Prolong cardiac conduction</td>
</tr>
<tr>
<td>Granisetron</td>
<td>1-3 mg IV</td>
<td>10 mcg/kg IV</td>
<td>24 hr</td>
<td>Liver disease</td>
</tr>
<tr>
<td>Dexamethasone</td>
<td>8-10 mg IV</td>
<td>0.15-1 mg/kg</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Betamethasone</td>
<td>12 mg IV</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Miscellaneous Postanesthesia Complications**

1. **Renal dysfunction:** oliguria (urine output less then 0.5 mL/kg/hour) most likely reflects decreased renal blood flow due to hypovolemia or decreased cardiac output.
2. **Bleeding abnormalities:** causes include inadequate surgical hemostasis or coagulopathies.
3. **Shivering** (hypothermia)
   A. Shivering can occur secondary to hypothermia or the effects of anesthetic agents (most often volatile anesthetics).
   B. Shivering should be treated with warming measures (Bair Huggar system). Small doses of meperidine (12.5-25 mg) IV.

**Postanesthesia Care Unit Discharge Criteria**

1. All patients should be evaluated by an anesthesiologist prior to discharge; patients should have been observed for respiratory depression for at least 30 minutes after the last dose of parenteral narcotic.
   A. Patients receiving regional anesthesia should show signs of resolution of both sensory and motor blockade prior to discharge.
   B. Other minimum discharge criteria include stable vital signs, alert and oriented (or to baseline), able to maintain adequate oxygen saturation, free of nausea/vomiting, absence of bleeding, adequate urine output, adequate pain control, stabilization or resolution of any problems, and movement of extremity following regional anesthesia.
### Drug Overdoses

#### Overdoses: general approach

- Overdoses account for 15% of acute medical emergencies.
- 65% of drugs involved belong to the patient, a relative, or friend.
- 30% of self-poisonings involve multiple drugs.
- 50% of patients will have taken alcohol as well.
- The history may be unreliable. Question any witnesses or family about where a patient was found and any possible access to drugs. Examination may reveal clues as to the likely poison (e.g. pinpoint pupils with opiates) and signs of solvent or ethanol abuse and iv drug use should be noted.

#### Management

- **Priorities are**
  - Resuscitate the patient
  - Reduce absorption of the drug if possible
  - Give specific antidote if available.

- **Monitor their airway (place in the recovery position) and breathing, BP, temperature, acid-base and electrolytes, and treat seizures or dysrhythmias. Intubate if GCS \( \leq 8 \), and not reversible with naloxone or flumazenil.**

- **Take account of any active medical problems that the patient may have, e.g. iv drug users may have concurrent septicaemia, hepatitis, SBE, pulmonary hypertension, or HIV-related disease.**

- **Measures to reduce gut absorption include**
  - Gastric lavage is only effective if used up to 1 hour post OD. It is contraindicated if corrosive substances or hydrocarbons have been ingested. Protect the airway with endotracheal intubation if conscious level is impaired.
  - Activated charcoal (50 g as a single dose) will absorb many drugs if given within 1 hour of ingestion although its effectiveness falls off rapidly thereafter. Drugs not absorbed by charcoal include iron, lithium, salts, alkalis, acids, ethanol, methanol, ethylene glycol, and organic solvents.
  - Repeated administration of activated charcoal (50g every 4 hours) may also accelerate whole body clearance of some drugs by interrupting enterohepatic cycling, e.g. phenobarbitone, phenytoin, carbamazepine, digoxin, paraquat, dapsone, quinine, and slow-release preparations such as theophylline. Charcoal is rather unpleasant to drink repeatedly and will be more reliably taken if given down a nasogastric tube.
  - PEG bowel lavage: in whole bowel irrigation, a solution of polyethylene glycol (not to be confused with ethylene glycol!!) is given orally or by NG tube at 2L/h in adults. It is continued until the rectal effluent becomes clear.

**Indications:** ingestion of serious substances such as sustained-release or enteric-coated preparations. May be used for lithium, iron, arsenic, lead oxide, or zinc sulphate.
Contraindications: bowel obstruction, perforation, ileus, or in patients seriously ill e.g. haemodynamic instability.

- Ipecac-induced emesis is no longer used.

Always seek advice from the local poisons unit

**Assessment of poisoning in the unconscious patient**

<table>
<thead>
<tr>
<th>Sign</th>
<th>Consider</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypoventilation</td>
<td>Opiates, ethanol, benzodiazepines</td>
</tr>
<tr>
<td>Hyperventilation</td>
<td>Metabolic acidosis (aspirin, paracetamol), gastric aspiration, carbon monoxide</td>
</tr>
<tr>
<td>Pinpoint pupils</td>
<td>Opiates, organophosphates</td>
</tr>
<tr>
<td>Dilated pupils</td>
<td>Methanol, anticholinergics, tricyclics, LSD</td>
</tr>
<tr>
<td>Bradycardia</td>
<td>β-blockers, digoxin, opiates</td>
</tr>
<tr>
<td>Tachyarrhythmias</td>
<td>Tricyclics, anti-cholinergics, caffeine, theophylline, lithium, digoxin</td>
</tr>
<tr>
<td>Hyperthermia</td>
<td>Ecstasy, amphetamines, anti-cholinergics</td>
</tr>
<tr>
<td>Pyramidal signs, ataxia, hypotonia, hyper-reflexia and extensor plantars</td>
<td>Tricyclics or anti-cholinergic agents</td>
</tr>
<tr>
<td>Hypertension</td>
<td>Cocaine, amphetamines, ecstasy</td>
</tr>
</tbody>
</table>

NB: Occasionally patients present where poisoning is suspected but not known. Even where the history suggests self-poisoning be aware that serious underlying disease may be present.

**Drug overdoses and antidotes**

<table>
<thead>
<tr>
<th>Drug</th>
<th>Action</th>
<th>Antidote/Therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anti-depressants</td>
<td>Activated charcoal</td>
<td>Diazepam for convulsions, cardiac monitoring</td>
</tr>
<tr>
<td>Aspirin</td>
<td>Protect airway</td>
<td>Alkaline diuresis, haemodialysis</td>
</tr>
<tr>
<td>Benzodiazepines</td>
<td>Check BP, HR, and breathing</td>
<td>Flumazenil if severe</td>
</tr>
<tr>
<td>β-blockers</td>
<td>Calcium gluconate</td>
<td>Anti-cholinergics</td>
</tr>
<tr>
<td>Calcium antagonists</td>
<td>Give 100% oxygen</td>
<td>Treat cerebral oedema with mannitol, consider hyperbaric oxygen</td>
</tr>
<tr>
<td>Carbon monoxide</td>
<td>Give 100% oxygen</td>
<td>Sodium thiosulphate, dicobalt edetate</td>
</tr>
<tr>
<td>Cyanide</td>
<td>Check K⁺ and ECG</td>
<td>Digibind® (digoxin-binding antibody)</td>
</tr>
<tr>
<td>Digoxin</td>
<td>Gastric emptying</td>
<td>Infuse ethanol, 4-methyl pyrazole</td>
</tr>
<tr>
<td>Ethylene glycol</td>
<td>Gastric emptying</td>
<td>Dimercaprol, penicillamine, sodium calcium edetate</td>
</tr>
<tr>
<td>Heavy metals</td>
<td>Gastric emptying</td>
<td>Desferrioxamine</td>
</tr>
<tr>
<td>Iron tablets</td>
<td>Gastric emptying</td>
<td>Diuresis/dialysis</td>
</tr>
<tr>
<td>Lithium</td>
<td>Convulsions may occur. Treat with diazepam</td>
<td>None</td>
</tr>
<tr>
<td>Mefenamic acid</td>
<td>Monitor U&amp;Es, glucose</td>
<td>Infuse ethanol, phenytoin for seizures, dialysis if severe</td>
</tr>
<tr>
<td>Methanol</td>
<td>Gastric emptying, remove clothes, and decontaminate</td>
<td>Atropine, pralidoxime</td>
</tr>
<tr>
<td>Organophosphorus insectides</td>
<td>Ensure breathing is adequate</td>
<td>Naloxone</td>
</tr>
<tr>
<td>Opiates</td>
<td>Gastric emptying if within 4 hours</td>
<td>Fuller's Earth (or bentonite or activated charcoal), intravenous vitamin E may be of benefit</td>
</tr>
<tr>
<td>Paracetamol</td>
<td>Check plasma potassium urgently</td>
<td>Repeat dose of activated charcoal</td>
</tr>
</tbody>
</table>

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Cardiopulmonary Resuscitation (CPR)

- Cardiopulmonary resuscitation (CPR) is systematic therapy that is aimed at sustaining vital organ function until natural cardiac function can be restored.
- The major components of resuscitation from cardiac arrest are airway, breathing, circulation, drugs, and electrical therapy (ABCDE). Traditionally, these have been divided into basic life support (BLS) and advanced cardiac life support (ACLS)

**BASIC LIFE SUPPORT**

**Goal of BLS:**
Increasing the survival rates of cardiac and respiratory arrest through the training of laypersons to:
- Recognize the symptoms of inadequate circulation or respiration.
- Immediately activate the Emergency Medical Services (EMS).
- Support the circulation and respiration via cardiopulmonary resuscitation (CPR) and rescue breathing.

**BLS Protocol (3As)**
- Assessment: Determine unresponsiveness of the patient.
- Activate the EMS system immediately by calling 9-1-1.
- ABCs of CPR (airway, breathing, and circulation).

**Airway with C-Spine Control**
- Position the patient supine on a flat surface using “logroll” technique.
- Open the airway using head tilt–chin lift maneuver or the jaw thrust maneuver.

**Breathing**
- Look, listen, and feel for adequate breaths (approximately 3 to 5 seconds).
- Perform rescue breathing (mouth-to-mouth, etc.):
  - Give two initial breaths over 1 second.
  - Deliver 10 to 12 breaths per minute; 8 to 10 if advanced airway in place.

**Circulation**
- Determine pulselessness by checking carotid artery pulse. If there is no pulse, begin chest compressions:
  - Proper hand position is on the lower half of the sternum.
  - Push hard, push fast. Allow the chest to recoil after each compression.
- Rate of chest compressions should be 80 to 100 per minute.
- For one-rescuer CPR, ratio is 20 compressions to 2 breaths.
- For two-rescuer CPR, ratio is 15 compressions to 2 breaths.

**Risk Factors**
- Large, poorly chewed pieces of food.
- Excessive alcohol intake.
- Dentures.
- Children swallowing small objects (toys, beads, marbles, thumbtacks).
- Children eating foods that require adequate chewing (hot dogs, peanuts, popcorn, candy).
- Children running/playing while eating.
Management of Partial Airway Obstruction
Do not interfere with any choking victim who is able to cough or speak. Coughing is the most effective way to clear a foreign body from the airway, and the ability to speak indicates that adequate ventilation is still occurring.

Signs of Complete Airway Obstruction
- High-pitched, stridorous sounds during inhalation
- Weak and ineffective coughing
- Respiratory distress
- Inability to speak
- Cyanosis

Heimlich Maneuver
- In a Standing (Conscious) Victim
  Stand behind victim and wrap arms around waist.
  Make fist and place thumb of fist slightly above the navel of the victim’s abdomen.
  Grasp fist with the other hand and quickly thrust inward and upward into victim’s abdomen.
  Repeat until object is dislodged or patient becomes unconscious.
- In an Unconscious Victim
  Lay victim supine.
  Straddle victim, place heel of palm just above navel (well below the xiphoid), and deliver quick inward and upward abdominal thrusts (up to five).
  Open the mouth of the unconscious victim and perform a finger sweep using a hooking motion of the index finger along the base of the tongue to dislodge the foreign body.
  Reposition the head and attempt rescue breathing.
  Repeat the sequence of the Heimlich maneuver, finger sweep, and rescue breathing attempts until victim resumes breathing or definitive help arrives.

ADVANCED CARDIAC LIFE SUPPORT (ACLS)

Goals
To provide rapid assessment and definitive management of the cardiac arrest situation using cardiac monitoring equipment, advanced airway management, as well as electrical and pharmacologic therapy.

Primary Survey
Focus on the ABCs of CPR and keep in mind defibrillation. First “A-B-C-D”
- Airway—open the airway (maintaining C-spine control).
- Breathing—assess breathlessness and provide rescue breathing.
- Circulation—give chest compressions (CPR).
- Defibrillation—shock ventricular fibrillation and pulseless ventricular tachycardia.

Secondary Survey
Secondary survey of ACLS focuses on the same ABCs in more detail: Establishing a definitive airway, establishing access to the circulation, assessing cardiac rhythms, pharmacologic interventions, etc.

Second “A-B-C-D”
- Airway—laryngeal mask airway (LMA) or endotracheal intubation.
- Breathing—assess bilateral chest rise and bilateral breath sounds.
- Circulation—establish intravenous (IV) access, determine the cardiac rhythm, and give the appropriate medication for that rhythm.
- Differential diagnosis—why did the arrest occur? Are there any causes that are reversible and have a specific therapy?
Airway

Nasal airway—rubber nasal trumpet inserted into the nostril and passed into the posterior pharynx keeps the tongue from falling back and obstructing the airway.

Oral airway—curved rigid airway, inserted using a tongue blade so that the distal edge prevents the tongue from falling backward. Often incorrectly used as a “bite block.” Should be used only in unconscious patients with absent gag reflexes (i.e., it will cause gagging if any gag reflex remains).

Laryngeal mask airway—a supraglottic airway management device. Distal tip of LMA cuff presses against upper esophageal sphincter, upper border rests against tongue (see Figure 2-1). Cuff is then inflated, forming a seal over the larynx and permitting positive pressure ventilation.

Endotracheal intubation—establishes a definitive airway that also protects against aspiration of blood, vomit, and pharyngeal secretions. Several cardiac medications can be given directly through the endotracheal tube (ETT). Usual ETT dose is 2 to 2.5 times the IV dose followed by 10 mL of normal saline flush and several ventilations by bag-valve ventilation. Intravenous and intraosseous routes are preferred for administration of resuscitation medications.

Breathing

Assess the status of ventilations after intubation (listen for equal breath sounds over both lung fields and make sure there are no sounds of gastric insufflation) and adjust the tube as necessary.

Assess the movement of the chest wall with ventilations.

If in a hospital setting, obtain a stat portable chest x-ray (CXR).

Confirm ETT placement with an end-tidal CO₂ monitor.

If there is any doubt of placement, consider extubation and reintubation under direct visualization with a laryngoscope.

Circulation

Establish IV access (easiest access is usually the antecubital vein).

Normal saline is the fluid of choice in the resuscitation setting.

Determine cardiac rhythm.

Differential Diagnosis

Continually ask yourself, “What caused this arrest?”

Examine the rhythm and consider all the possible causes.

Treat each of those possible causes that are reversible and/or have a specific therapy.
“Golden Hour” of Trauma
Period immediately following trauma in which rapid assessment, diagnosis assessment, diagnosis, and stabilization must occur.

Prehospital Phase
Control of airway and external hemorrhage, immobilization, and rapid transport of patient to nearest appropriate facility.

Preparation
Gown up, glove up, face shields on!
Standard precautions!
Set up: Airway equipment, monitor, O2, urinary catheter (Foley), IV and blood tubes (complete blood count, chemistry, prothrombin time/partial thromboplastin time, type and cross, human chorionic gonadotropin, toxicologies), chest tube tray, etc.

Trauma History
Whenever possible, take an AMPLE history:
✓ Allergies
✓ Medications/Mechanism of injury
✓ Past medical history/Pregnant?
✓ Last meal
✓ Events surrounding the mechanism of injury

Primary Survey
Initial assessment and resuscitation of vital functions.
Prioritization based on ABCs of trauma care.

ABCs
✓ Airway (with cervical spine precautions)
✓ Breathing and ventilation
✓ Circulation (and Control of hemorrhage)
✓ Disability (neurologic status)
✓ Exposure/Environment control
✓ Foley

Airway and C-Spine
Assess patency of airway.
Use jaw thrust or chin lift initially to open airway.
Clear foreign bodies.
Insert oral or nasal airway when necessary. Obtunded/unconscious patients should be intubated. Surgical airway—cricothyroidotomy is used when unable to intubate airway.

Breathing and Ventilation
Inspect, auscultate, and palpate the chest.
Ensure adequate ventilation and identify and treat injuries that may immediately impair ventilation:
Tension pneumothorax
Flail chest and pulmonary contusion
Massive hemothorax
Open pneumothorax

Control of Hemorrhage
Place two large-bore (14 or 16G) IVs.
Assess circulatory status (capillary refill, pulse, skin color) (see Shock section below).
Control of life-threatening hemorrhage using **direct pressure**; do not “clamp” bleeding vessels with hemostats.

**Disability**
- Rapid neurologic exam.
- Establish pupillary size and reactivity, and level of consciousness using the AVPU or Glasgow Coma Scale.

**Exposure/Environment/Extras**
- Completely undress the patient, most often with the help of your trauma shears.
- Hook up monitors (cardiac, pulse oximetry, blood pressure, etc.).

**Foley Catheter**
- Placement of a urinary catheter is considered part of the resuscitative phase, which takes place during the primary survey.
- Important for monitoring urinary output, which is a reflection of renal perfusion and volume status.
- Adequate urinary output:
  - Adult: 0.5 cc/kg/hr
  - Child (> 1 year of age): 1.0 cc/kg/hr
  - Child (< 1 year of age): 2.0 cc/kg/hr
- Foley is contraindicated when urethral transection is suspected, such as in the case of a pelvic fracture. If transection is suspected, perform retrograde urethrogram before Foley.

**Signs of Urethral Transection**
- Blood at the meatus
- A “high-riding” prostate
- Perineal or scrotal hematoma

**Gastric Intubation**
- Placement of nasogastric (NG) or orogastric (OG) tube may reduce risk of aspiration by decompressing stomach, but does not assure full prevention.

Begins during the primary survey.
- Life-threatening injuries are tended to as they are identified.

**Intravenous Catheters**
- The rate of maximal fluid administration is directly related to the internal diameter of the IV catheter (to the fourth power of the radius according to Poiseuille’s law) and inversely related to the length of the tubing.

**Intravenous Fluid**
- Fluid therapy should be initiated with 1 to 2 L of an isotonic (either lactated Ringer’s or normal saline) crystalloid solution (see below).
- Pediatric patients should receive an IV bolus of 20 cc/kg.

**Crystalloid versus Colloid**
- Crystalloids are sodium-based solutions that provide a transient increase in intravascular volume.
  - Approximately one third of an isotonic solution will remain in the intravascular space. The remainder almost immediately distributes to the extravascular and interstitial spaces. This occurs because crystalloid solutions easily diffuse across membranes.
  - Colloids have a harder time diffusing across membranes, thus remaining in the intravascular space for longer periods of time thereby requiring smaller volumes for resuscitation. However, it is costly and carries the risks of transfusion reactions and viral transmission.
Neither crystalloids nor colloids have been shown to be superior for volume resuscitation. Therefore, volume resuscitation begins with crystalloids.

“3 to 1 Rule”
Used as a rough estimate for the total amount of crystalloid volume needed acutely to replace blood loss.

Shock
Inadequate delivery of oxygen on the cellular level secondary to tissue hypoperfusion. In traumatic situations, shock is the result of hypovolemia until proven otherwise.

Hypovolemic Shock
Caused by the acute loss of blood in most cases. Blood volume estimate based on body weight in kilograms:
   - Adults: 7% of weight
   - Peds: 8 to 9% of weight For example, 70-kg adult \((70 \cdot 7\% = 4.9 \text{ L of blood})\).

Radiologic and Diagnostic Studies
X-rays of the chest, pelvis, and lateral cervical spine usually occur concurrently with early resuscitative efforts; however, their procedure should never interrupt the resuscitative process. Diagnostic peritoneal lavage (DPL) and Focused Abdominal Sonogram for Trauma (FAST) are also tools used for the rapid detection of intraabdominal bleeding that often occurs early in the resuscitative process

Secondary Survey
Begins once the primary survey is complete and resuscitative efforts are well under way. Includes a head-to-toe evaluation of the trauma patient and frequent reassessment of status.

Neurologic examination, procedures, radiologic examination, and laboratory testing take place at this time if not already accomplished.

Tetanus Prophylaxis
Immunize as needed.
CLINICAL ANESTHESIA
AND EMERGENCY

For

Undergraduate Medical Student

By

Staff Members
Anesthesia and Intensive Care Department
Damietta Faculty of Medicine
Al-Azhar University
2011
With my best wishes
Tarik Sarhan