

Dr. Azam's....

Notes in Anesthesiology

Updated up to December 2013, 3rd Edition

Postgraduates appearing
for MD, DNB & DA Exams

Flow Charts, Diagrams & Important Tables

Edited by:
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Dedication

To **Mohammed Shafiulla**, my father, my oxygen, companion, and best friend; for being my major pillar of support and making this vision a reality. Thank you for your continual sacrifices with boundless love and limitless gratitude, for the sake of your children. I owe you a debt I can never repay.

I also would like to thank my **mom (Naaz Shafi)**, my **wife (Roohi Azam)**, my two lovely **kids (Falaq Zohaa & Mohammed Izaan)**, for their support, ideas, patience, and encouragement during the many hours of writing this book.

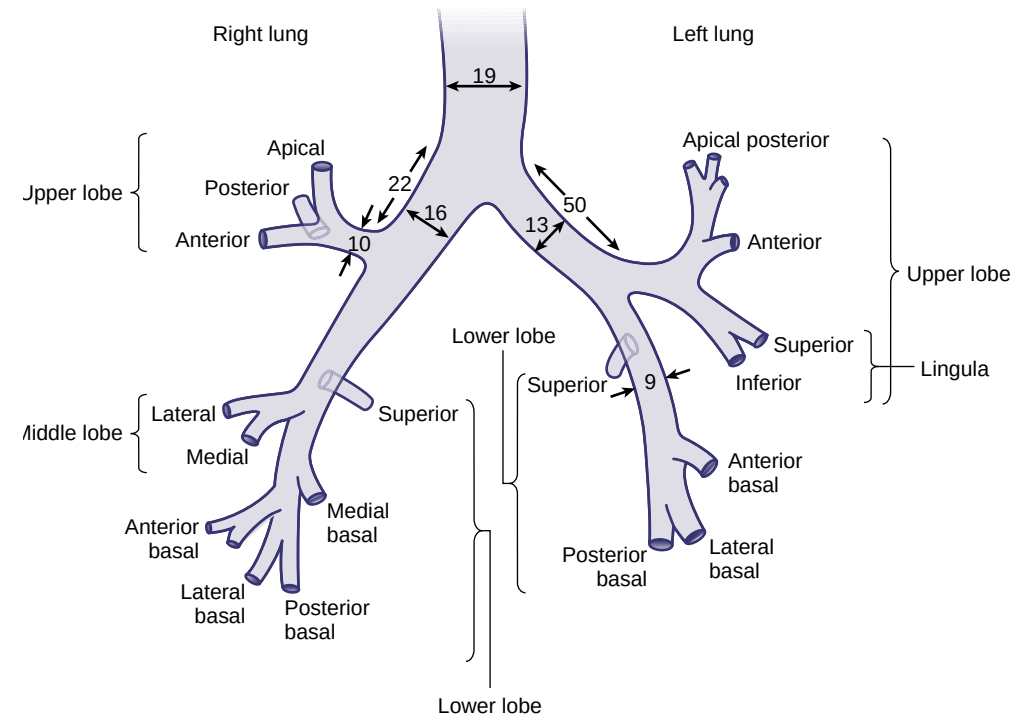
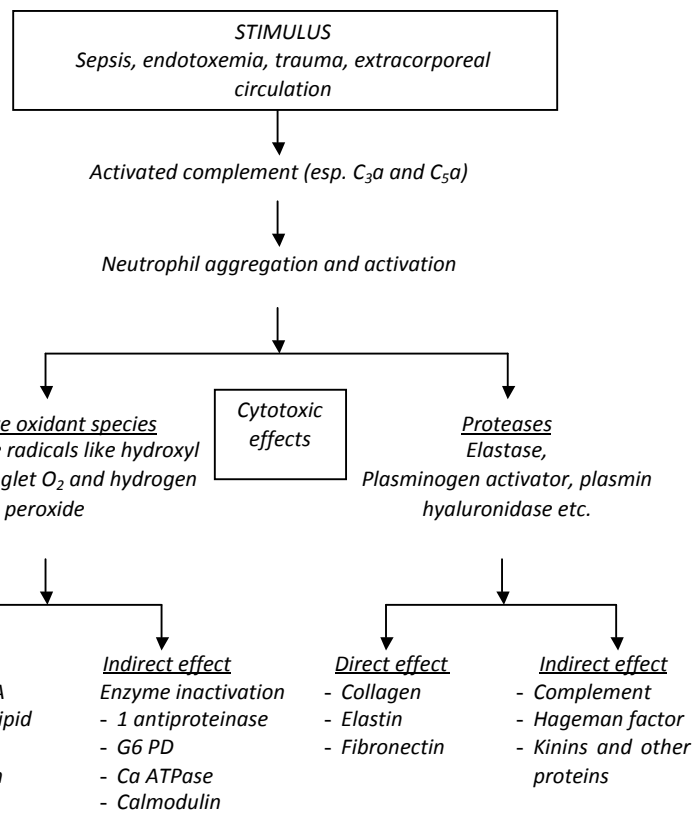
Finally, I would like to thank my teachers (**Dr. Manjunath Jajor & team**) & **Dr T. A. Patil**. The dream begins with a teacher who believes in you, who tugs and pushes and leads you to the next plateau, sometimes poking you with a sharp stick called "truth."

A NOTE TO THE READER

Anesthesiology is an ever-changing field. Standard safety precautions must be followed, but as new research and clinical experience broaden our knowledge, changes in treatment and drug therapy may become necessary or appropriate. Readers are advised to check the most current product information provided by the manufacturer of each drug to be administered to verify the recommended dose, the method and duration of administration, and contraindications.

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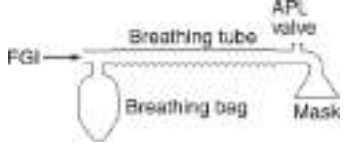
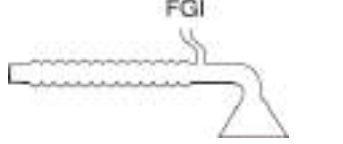
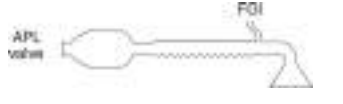
Dr. Azam



Millers: Page # 1842

Flow Chart: ARDS Pathophysiology

Classification & characteristics of various Mapleson circuit

Mapleson Class	Other Names	Configuration ¹	Spontaneous	Controlled	Comments
A	Magill attachment		Equal to minute ventilation (80 mL/kg/min)	Very high and difficult to predict	Poor choice during controlled ventilation. Enclosed Magill system is a modification that improves efficiency. Coaxial Mapleson A (Lack breathing system) provides waste-gas scavenging.
B			2 x minute ventilation	2–2½ x minute ventilation	
C	Waters' to-and-fro		2 x minute ventilation	2–2½ x minute ventilation	
D	Bain circuit		2–3 x minute ventilation	1–2 x minute ventilation	Bain coaxial modification: fresh gas tube inside breathing tube.
E	Ayre's T-piece		2–3 x minute ventilation	3 x minute ventilation (I:E =1:2)	Exhalation tubing should provide a larger volume than tidal volume to prevent rebreathing. Scavenging is difficult.
F	Jackson-Rees' modification		2–3 x minute ventilation	2 x minute ventilation	A Mapleson E with a breathing bag connected to the end of the breathing tube to allow controlled ventilation and scavenging.

4. Classify vaporizers. Discuss the effect of altered barometric pressure on the performance of vaporizers?

Vapour: A Vapour is the gaseous form of a substance which is liquid at room temperature

Vaporizer: A vaporizer is an instrument designed to change a liquid anesthetic agent in to its vapour and to add a controlled amount of this vapour to the fresh gas flow.

Classification of vaporizer:

Oldest Classification:

A. Method of regulating Output Concentration

1. Variable Bypass
2. Measured Flow

B. Method of Vaporization

1. Flow Over
2. Bubble Through
3. Flow over or Bubble Through

C. Location

1. Outside the Breathing System
2. Inside the Breathing System

D. Temperature Compensation

1. None
2. By Supplied Heat
3. By Flow Alteration

E. Specificity

1. Agent Specific
2. Multiple Agent

Older Classification:

A. Method of regulating Output Concentration

1. Variable Bypass
2. Measured Flow

B. Method of Vaporization

1. Flow Over
2. Bubble Through
3. Injection

C. Resistance

1. Plenum
2. Low resistance

D. Temperature Compensation

1. Thermocompensation
2. Supplied Heat

E. Specificity

1. Agent Specific
2. Multiple Agent

New Classification:

A. Method of regulating Output Concentration

1. Variable Bypass
2. Measured Flow

B. Method of Vaporization

1. Flow Over
2. Bubble Through
3. Injection

C. Temperature Compensation

1. Mechanical
2. Supplied Heat
3. Computerized

Preoperative Preparation:

- **Stop Smoking**
- **Dilate the airway using:**
 - Beta agonist
 - Anti-cholinergics
 - Theophylline
 - Steroids
 - Cromolyn Sodium
- **Loosen the Secretions by:**
 - Airway hydration (Humidification/Nebulization)
 - Systemic hydration orally - 3 Liters
 - Mucolytic agents & Expectorant drugs
 - Antibiotics
- **Remove secretions:**
 - Postural drainage
 - Coughing
 - Chest physiotherapy (percussion & Vibration)

Ancillary Measures:

- Treatment of co-existing disease
- Treatment of Cor-Pulmonale
- Pre-operative Digitalization

Patient Education, motivation & facilitation of post operative care: Continuation:

- Psychological preparation
- Incentive spirometry & deep breathing exercise
- Exposure to secretion removal maneuvers
- Exercise
- Weight gain/reduction
- Diet supplements.

<i>Time course</i>	<i>Beneficial effects</i>
12-24 hours	Decrease carbon monoxide and nicotine levels
48-72 hours	Normalizes carboxy Hb levels. Improves ciliary function, tissue oxygenation.
1-2 weeks	Decrease sputum production & improves smaller airway function.
4-6 weeks	Improves PFT
6-8 weeks	Normalizes immune function, drug metabolism and hepatic enzyme activity
8-12 weeks	Reduces overall post-operative pulmonary complications.

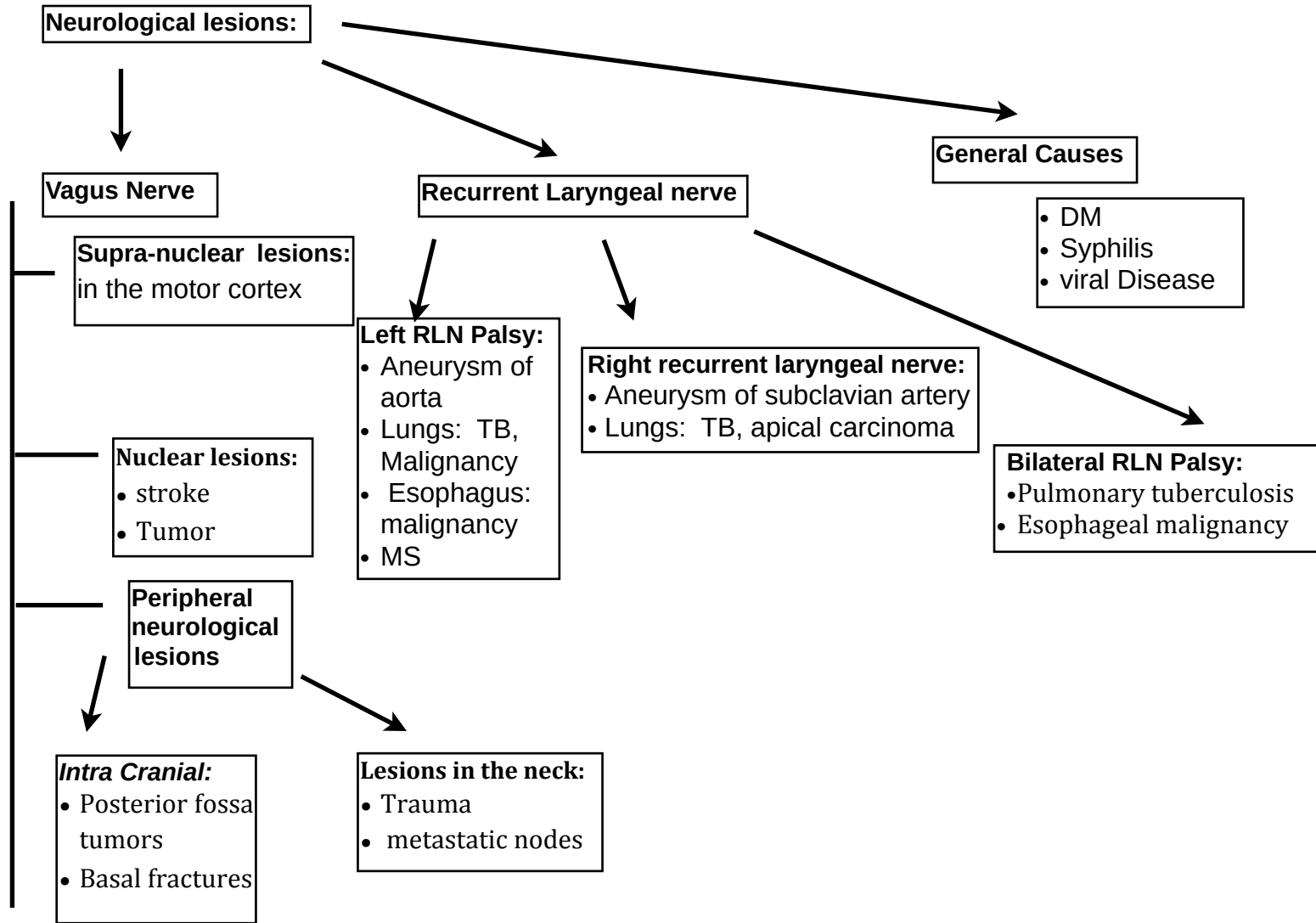
Post-operative Management:

Goals:

- To promote adequate analgesia
- To minimize the incidence of postoperative complications
- To prevent infections
- Chest physiotherapy & deep breathing exercise.
- Consider CPAP - Decreases WOB, Improves LV Compliance.
- Continuous administration of Local anesthetic with low dose opioids via epidural catheter for post operative pain relief.

9. Describe the innervations of Larynx. Briefly discuss various palsies following Nerve injury. Continuation:

Vocal Cord palsy

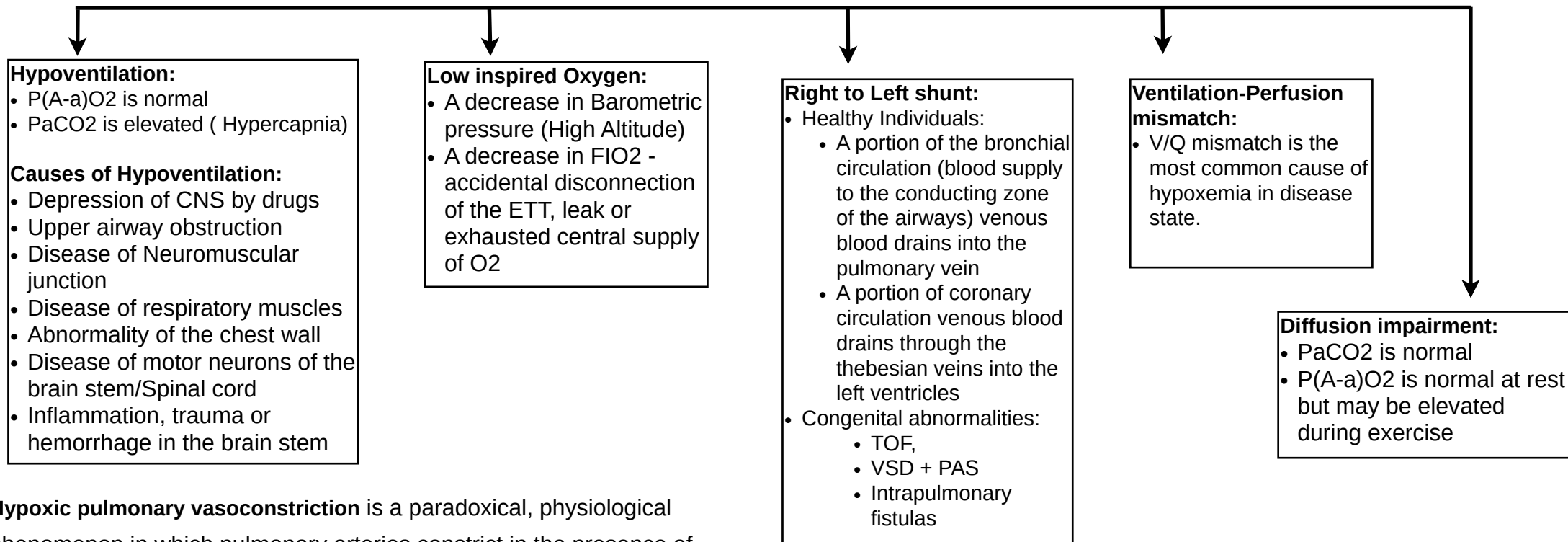


57. What are the major causes of Hypoxemia? What is Hypoxic Pulmonary Vasoconstriction? How can general anesthesia worsens V/Q mismatch?

Five Causes of Hypoxemia ($\downarrow$ PaO₂):

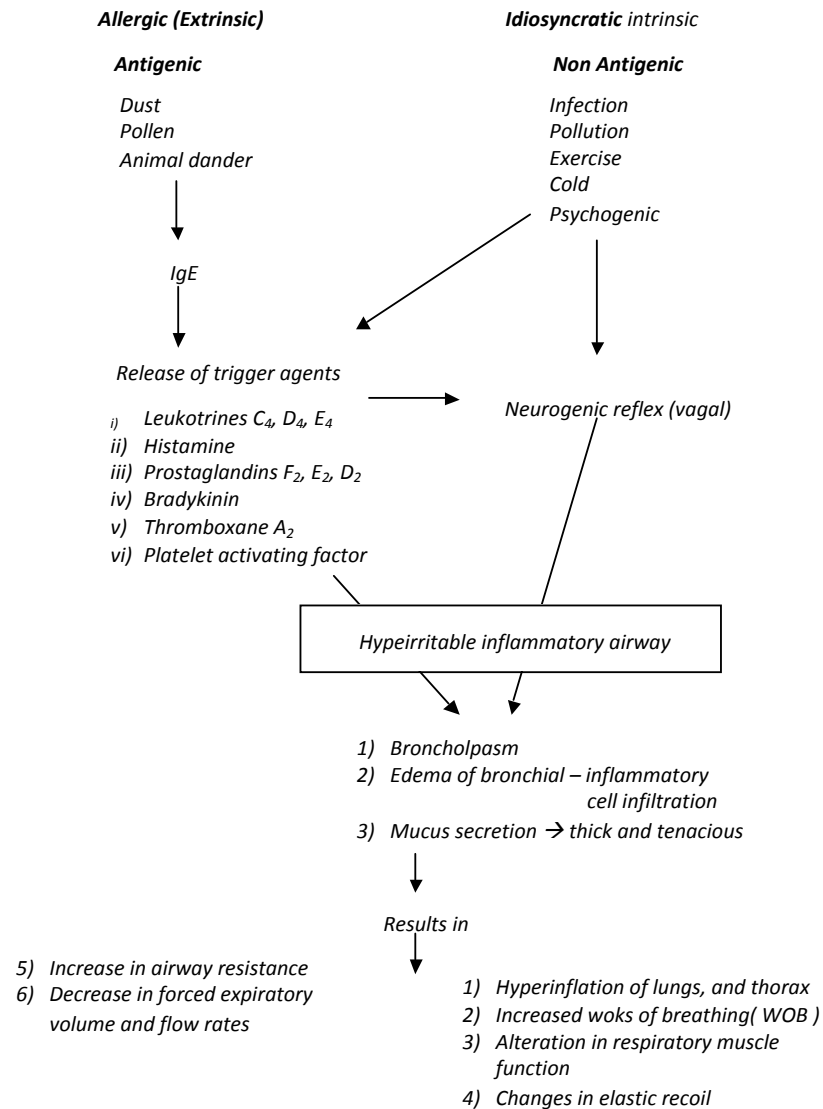
1. Hypoventilation
2. Low inspired Oxygen
3. Right to Left shunt
4. Ventilation-Perfusion mismatch
5. Diffusion impairment

Five Causes of Hypoxemia ($\downarrow$ PaO₂):



Hypoxic pulmonary vasoconstriction is a paradoxical, physiological phenomenon in which pulmonary arteries constrict in the presence of hypoxia without hypercapnia, redirecting blood flow to alveoli with a higher oxygen content.

Pathophysiology of Bronchial Asthma:



Severity of expiratory airflow obstruction & Grading:

Severity	FEV (% predicted)	FEF 25-75 (% predicted)	Pao ₂ (mm Hg)	Pa Co ₂ (mm Hg)
1) Mild (asymptomatic)	65-80	60-75	> 60	< 40
2) Moderate	50-64	45-59	> 60	< 45
3) Marked	35-49	30-44	< 60	> 50
4) Sever (status asthmaticus)	< 35	< 30	< 60	> 50

Table 59-13 Characteristics of the Cohen, Arndt, and Fuji Bronchial Blockers

	Cohen Blocker	Arndt Blocker	Fuji Uniblocker
Size	9 Fr	5 Fr, 7 Fr, and 9 Fr	5 Fr, 9 Fr
Balloon shape	Spherical	Spherical or elliptical	Spherical
Guidance mechanism	Wheel device to deflect the tip	Nylon wire loop that is coupled with the fiberoptic bronchoscope	None, preshaped tip
Smallest recommended ETT for coaxial use	9 Fr (8.0 ETT)	5 Fr (4.5 ETT), 7 Fr (7.0 ETT), 9 Fr (8.0 ETT)	9 Fr (8.0 ETT)
Murphy eye	Present	Present in 9 Fr	Not present
Center channel	1.6 mm ID	1.4 mm ID	2.0 mm ID

Table 59-9 Options for Lung Isolation

Options	Advantages	Disadvantages
Double-lumen tube 1. Direct laryngoscopy 2. Via tube exchanger 3. Fiberoptically	Quickest to place successfully Repositioning rarely required Bronchoscopy to isolated lung Suction to isolated lung CPAP easily added Can alternate OLV to either lung easily Placement still possible if bronchoscopy not available	Size selection more difficult Difficult to place in patients with difficult airways or abnormal tracheas Not optimal for postoperative ventilation Potential laryngeal trauma Potential bronchial trauma
Bronchial blockers (BB) Arndt Cohen Fuji	Size selection rarely an issue Easily added to regular ETT Allows ventilation during placement Easier placement in patients with difficult airways and in children Postoperative two-lung ventilation by withdrawing blocker Selective lobar lung isolation possible CPAP to isolated lung possible	More time needed for positioning Repositioning needed more often Bronchoscope essential for positioning Nonoptimal right lung isolation due to RUL anatomy Bronchoscopy to isolated lung impossible Minimal suction to isolated lung Difficult to alternate OLV to either lung
Univent tube	Same as BBs Less repositioning compared with BBs	Same as for BBs ETT portion has higher air flow resistance than regular ETT ETT portion has larger diameter than regular ETT
Endobronchial tube	Like regular ETTs, easier placement in patients with difficult airways Longer than regular ETT Short cuff designed for lung isolation	Bronchoscopy necessary for placement Does not allow for bronchoscopy, suctioning or CPAP to isolated lung Difficult right lung OLV
Endotracheal tube (ETT) advanced into bronchus	Easier placement in patients with difficult airways	Does not allow for bronchoscopy, suctioning or CPAP to isolated lung Cuff not designed for lung isolation Extremely difficult right OLV

7. A 30 year old female ASA grade I following exploratory laparotomy is not maintaining oxygen saturation in the post operative period. Discuss the causes & Management.

Definition: Hypoxia is defined as inadequate oxygen supply to the cells and tissues of the body.

Values: Sao2 < 90%, PaO2 < 60%

Causes of Post operative Hypoxia & its treatment: (12 Causes)

Low Inspired concentration of O2:

- When FIO2 is < 0.21%. The treatment would be giving high inspired oxygen through Venturi mask.

Airway obstruction:

- Pharyngeal obstruction: Treatment would be Head tilt & chin lift.
- Laryngeal obstruction: treatment: Lift the chin while the head is on a pillow or displace jaw forward. Alternating lifting and relaxing the chin will, relax the strap muscle of the neck.
- Attempt to facilitate ventilation of gentle positive pressure via mask. When conservative measures are not effective in breaking spasm within 1min, then a muscle relaxant should be given I.V. Scoline 1.5mg/kg injected
- Bronchospasm: Bronchodilators like Beta agonist, Anticholinergics, Theophylline and Steroids

Post operative shivering:

- May also cause hypoxia. Treatment is Oxygen therapy

Sudden reduction in cardiac output:

- Can contribute to hypoxemia. Treatment: Watch out for bleeding & dehydration. Give IV fluids, blood replacement.

Diffusion Hypoxia:

- At the end of surgery when both oxygen & nitrous oxide stopped at the same time. Where N2O diffuses back into the alveoli. Treatment is to give oxygen after cutting off N2O.

Type of Surgery:

- Patients with abdomino-thoracic procedures will not respire adequately because of pain on deep inspiration. Treatment would be adequate analgesia with opioids or NSAIDS or through the epidural.

Ventilation Perfusion Mismatch:

- Usually seen under Anesthesia.

Decreased FRC:

- Seen in Pulmonary edema (Treatment will be fluid restriction, diuretics & oxygen therapy Positive pressure ventilation.), Infections(Antibiotics), Obesity (incentive spirometer & deep breathing exercises), aspiration(postural drainage, nebulization incentive spirometer & deep breathing exercises.)

Right to left intrapulmonary shunts:

- Most common cause is Atelectasis. Treatment would be Deep breathing exercise, Chest physiotherapy humidification of inspired gases, coughing & incentive spirometer.

Drug Overdosage:

- Like opioids, muscle relaxants, in adequate reversal. Treatment would be titrated dosage of opioids & to wait for adequate reversal.

Pulmonary Embolism:

- Lower limb surgeries, prolonged immobilization.treatment would be DVT stocking, early mobilization, DVT prophylaxis.

Pneumothorax:

- Result of direct injury to lungs or rib fracture.
- Treatment is 100% oxygen, insertion of chest tube & intercostal drainage & IPPV.

GRID: Question needs answered under following headings:

1. Definition
2. values
3. 12 Causes of Post operative Hypoxia & its management.

3. Pulmonary Function Test

Non-Specific - Bed Side Test

Specific Test - PFT

Test of Ventilation

Test of Distribution of Ventilation

Test of Diffusion

Test of Perfusion

Other Tests

Single Breath N₂ elimination Test
Multi-breath N₂ wash out test
Radio-active Xenon distribution

Carbon Monoxide Diffusing Capacity

radio-active Xenon injection
Radio-nucleotide
Macro-aggregated iodine

- Split Lung Function Test
 - Bronchspirometry
 - radio-active Scanning
 - Pulmonary artery occlusion test
- ABG
- A-a Gradient
- Raw
- Compliance

Non-Specific - Bed Side Function:

- Breath Holding test
- Snider's Match Blowing test
- De Bono's Whistle test
- Auscultation over the trachea
- Sphygmomanometer
- Walk test
- Stair case test.

Static Test

Dynamic Test

- Anatomical Measurements
- Do not evaluate Function

Measures rate of flow of Gas

4 Volumes
TV
IRV
ERV
RV

4 Capacities
VC
TLC
FRC
IC

MBC/MVV
FEV
PEFR
MMEFR 25 to 75 %
Respiratory Muscle Strength

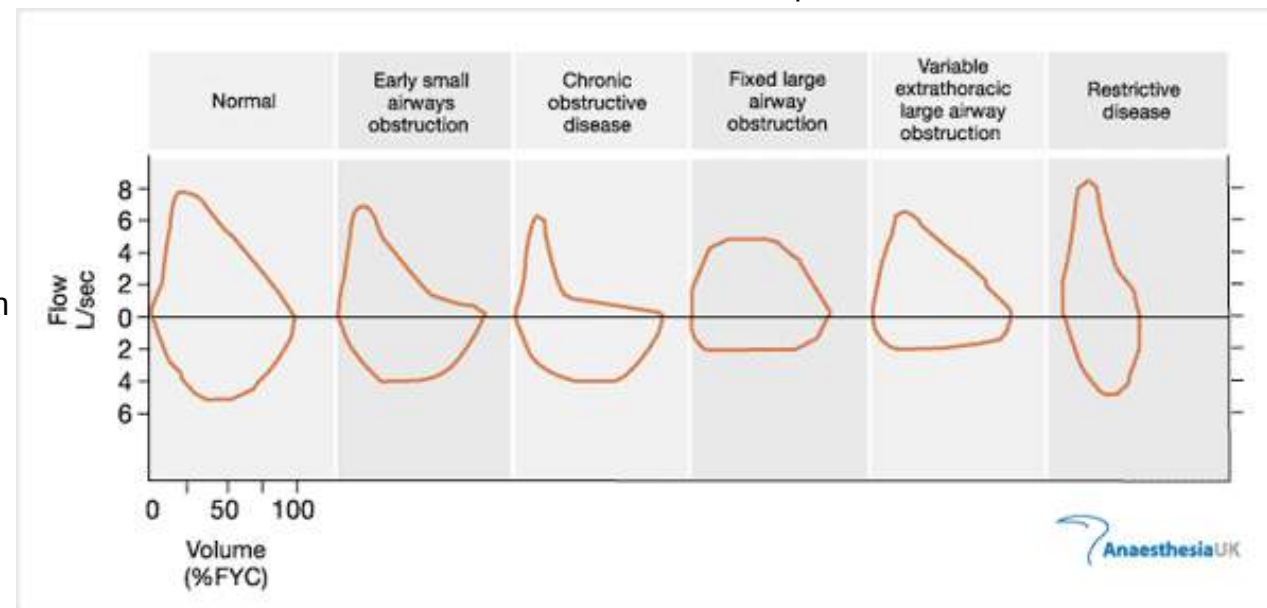
Flow Volume Loops

Fixed Obstruction

Variable Extra-Thoracic Obstruction
E-Expiration is normal

Variable Intra-Thoracic Obstruction
I-Inspiration is normal

Restrictive Lung Disease



Pulmonary Function Test

- Spirometer can measure - VT, IRV, ERV, VC, IC, FEV & MBC
- Cannot be measured by Spirometer - RV, TLC & FRC

Static Test

VT - Tidal Volume

- Total Volume of air inspired & Expired in each breath
- measured by wright's Respirometer

IRV - Inspiratory reserve volume

- Volume of air that can be inspired after normal inspiration.

ERV - Expiratory reserve volume

- Volume of air that can be expired after normal expiration
- It reflects the thoracic muscles, abdominal muscle & thoracic mobility

RV - Residual Volume

- Volume of air remaining in the lung after maximum expiration
- Measured by
 - N2 washout technique
 - Helium dilution technique

VC - Vital Capacity

- Most commonly measured
- volume of air that can be expired after maximum inspiration
- can be measured by - spirometer
- BSA: Male- 2.6l/m² Female - 2.1l/m²
- A given VC is abnormal if < 80% of predicted value
- **VC: decreased** - in pregnancy, Pulm fibrosis
 - Emphysema
 - ch. Bronchitis
 - asthma
 - Abdominal pain
 - can be < 70 to 75% in upper abdominal surgery
 - < 50 to 55% in lower abdominal surgery

- Position: Lithotomy Vc < 18%

- $VC = 3 \times VT$ for effective cough

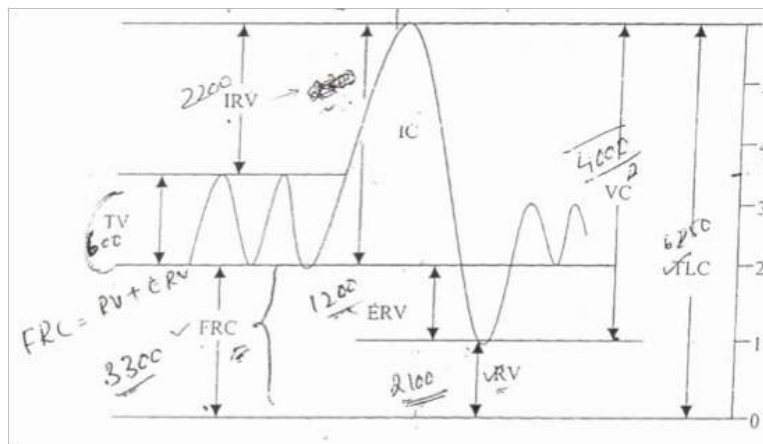
- Decrease in VC means decreased ability to cough and post operative complications

FRC

- Volume of air remaining in the lung after normal expiration
- measured by:
 - N2 washout technique
 - Helium dilution
 - Body Plethysmograph
- **FRC increased:**
 - Obstruction of airways
 - Emphysema
 - PEEP
- **FRC decreased:**
 - Under Anesthesia
 - post op old age
 - obesity
 - smoking
 - Lithotomy
 - Abdominal distention
 - Lap surgeries

TLC

- Total volume of air in lung after maximum inspiration.
- TLC decreased:
 - Pulmo fibrosis
 - obesity
 - weakness of inspiratory muscle



Dynamic Tests

MBC or MVV

- Max volume of air that can be breathed per minute by voluntary effort
- normal - 160L/min to 180 L/min

MBC is decreased in:

- old age
- emphysema
- bronchospasm
- bronchiolar obstruction

FVC & FEV

FVC

- Assess the expiratory ability
- max volume of air that can be exhaled forcefully and rapidly after maximum inspiration
- measured by Spirometer and fast moving Kymograph
- FVC done over 4 sec
- reflects the relationship between Flow & resistance

FEV1

- Max volume of gas exhaled during the 1st second of FVC maneuver
- FEV1 = 83% of VC

FVC is decreased in:

- Atelectasis
- Fibrosis
- muscle weakness
- abdominal swelling
- pain

In healthy patients
FVC=VC

PEFR:

- After max inspiration patient exhales as forcefully as he can and the max flow rate is measured and expressed in lt/min and lt/sec
- Normal:
 - 450-600 L/min
 - 300 - 500 L/min
- Measured by Wright's Respirometer
Pneumotachygraph

PEFR is decreased in:

- Obstructed Airway

Values less than 200 lt/min suggest impaired cough and increased likelihood of post op pulmonary complications

MMEFR (25% to 75%):

- Flow measured in middle half of FVC
- obtained by dividing the volume of gas between 25 - 75% by corresponding elapsed time
- Normal = 4 to 5 L/sec

Importance

- more sensitive of small airway obstruction or disease

Respiratory Muscle weakness:

- It is evaluated and measured by maximum static respiratory pressure
- PImax = -125 cm of H₂O - normal
- If PImax = -25 inability to take deep breath
- PEmax = + 200 cm of H₂O - normal
- If PEmax = + 40 cm of H₂O severely impaired coughing ability
 - seen in Emphysema
 - predictor of successful weaning
 - Evaluation of NM disorder

Condition	FEV1	FVC	FEV1 /FVC
Airway obstruction (asthma, bronchitis)	Decreased	Normal	Decreased
Stiff lungs (pneumonia, pulmonary edema)	Decreased	Decreased	Normal
Respiratory muscle weakness (myasthenia, myopathies)	Decreased	Decreased	Normal

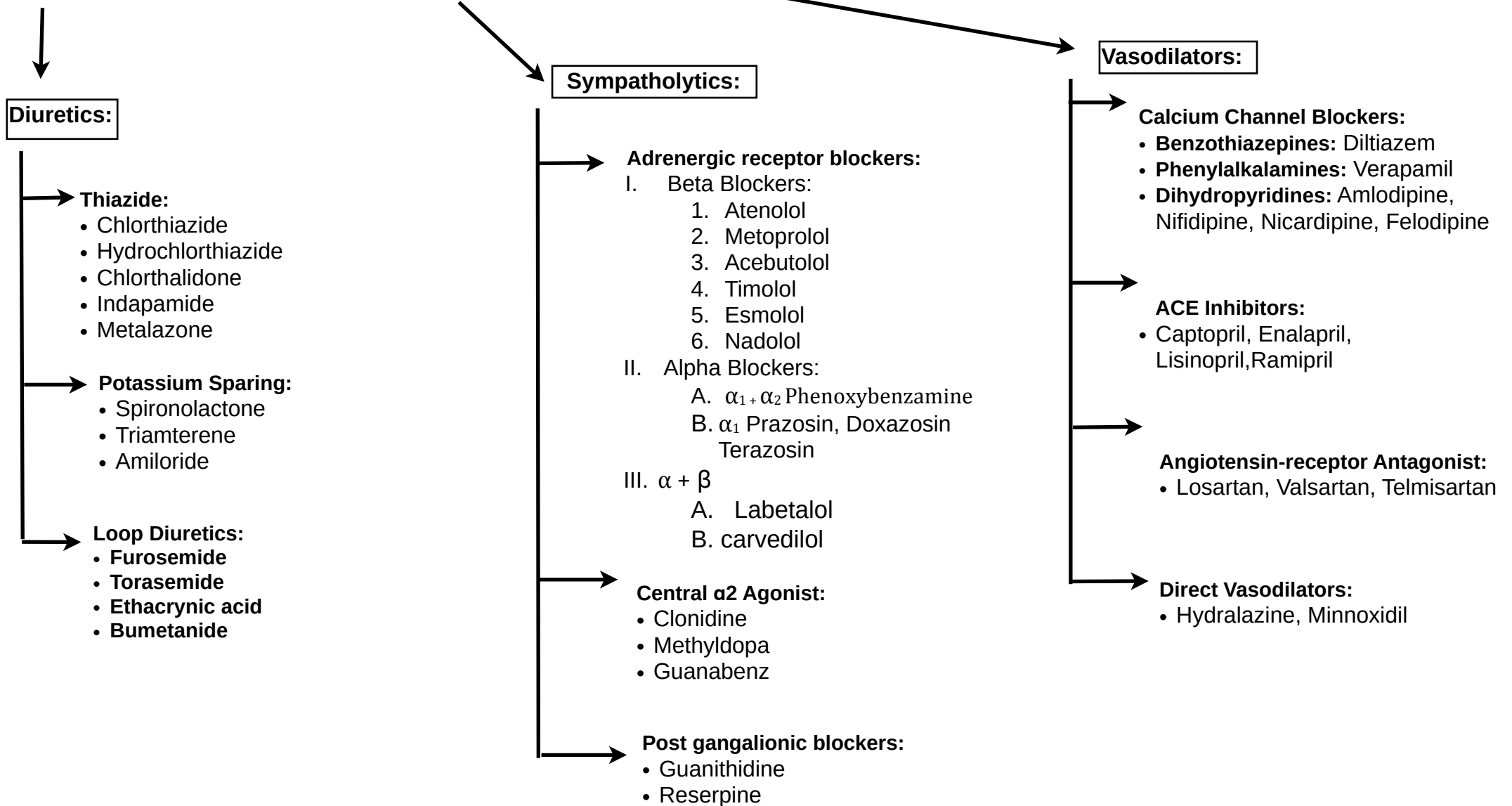
	Obstructive	Restrictive
FEV1	Decreased	Decreased
FVC	Normal	Decreased
FEV1/FVC	Decreased	Normal
FRC	Increased	Decreased
TLC	Increased	Decreased
(Raw)airway resistance	Increased	Normal
MMEFR	Decreased	Normal
MBC	Decreased	Normal
PEFR	Decreased	Decreased

Flow-Volume Loops

- Provide a graphic analysis of flow at various lung volumes
- Used to differentiate between patients with upper airway and lower airway obstruction
- Flow is charted on Y-axis
- Volume is charted on X axis
- Patient is asked to inhale fully to TLC and then perform a FVC maneuver. This is followed immediately by a maximum inspiration back to TLC
- The ratio of expiratory flow to inspiratory flow at 50% of VC is normally 1 (mid VC ratio)

20. Classify Anti-Hypertensive drugs. Describe anesthetic management of hypertensive episode during anesthesia

Oral Anti-Hypertensive Drugs - Classification



Indications	Drugs	Dose	Onset	Duration
For patient with good LV function ↑ HR CI in bronchospastic disease	<i>Esmolol IV</i>	<i>0.5mg/kg over /min 50-300µg/kg/min</i>	<i>1min</i>	<i>12-20min</i>
	<i>Labetalol</i>	<i>5-20mg</i>	<i>1-2min</i>	<i>4-8hr</i>
	<i>Propanol</i>	<i>1-3mg</i>	<i>1-2min</i>	<i>4-6hr</i>
Ca+2 channel blockers Myocardial ischemia ← reflex tachycardia	<i>Nicardipine</i>	<i>0.25-0.5mg</i>	<i>1-5min</i>	<i>3-4 hr</i>
	<i>Nifedipine (S/L)</i>	<i>10mg</i>	<i>5-10min</i>	<i>4h</i>
Mod to severe HTN – most rapid	<i>Nitroprusside</i>	<i>0.5-10µg/kg/min</i>	<i>30-60 sec</i>	<i>1-5min</i>
	<i>Nitroglycerine</i>	<i>0.5-10µg/kg/min</i>	<i>1min</i>	<i>3-5min</i>
Sustained control of BP ← delayed onset reflex tachycardia ACE Inhibitors	<i>Hydralazine</i>	<i>5-20mg</i>	<i>5-20min</i>	<i>4-8hr</i>
	<i>Phentolamine</i>	<i>1-mg</i>	<i>1-10min</i>	<i>20-40mm</i>
	<i>Enalaprilat</i>	<i>0.625-1mg</i>	<i>6-14min</i>	<i>4-6h</i>
	<i>Fenoldopam</i>	<i>0.1-0.6 µg/kg/min</i>	<i>5 min</i>	<i>5 min</i>

Goldman's Cardiac Risk Index - 1977.

i.	Age > years	5
ii.	MI in previous 6 months	10
iii.	S3 gallop or ↑ JVP (0-3 cm)	11
iv.	Important aortic stenosis	3
v.	Rhythm other than sinus, Pac on last preoperative	7
vi.	>5 PVC/min at any time before	7
vii.	PaO ₂ , < 60 or PaCO ₂ > 50mm Hg, K<3.00 mEq/L or HCO ₃ < 20 mEq/l BUN > 50 or Cr> 3 mg/dl. Abnormal SGOT, signs of chronic liver disease, bed ridden from non-cardiac cause	3
viii.	Intraperitoneal, Intrathoracic, aortic surgery	3
ix.	Emergency operation	4
	Total	53

Class I	0-5 points (low risk)
Class II	6-12 points (intermediate risk)
Class III	13-25 points (high risk)
Class IV	≥ 26 points (very high)

Detsky's Modified Cardiac Risk Index- 1986

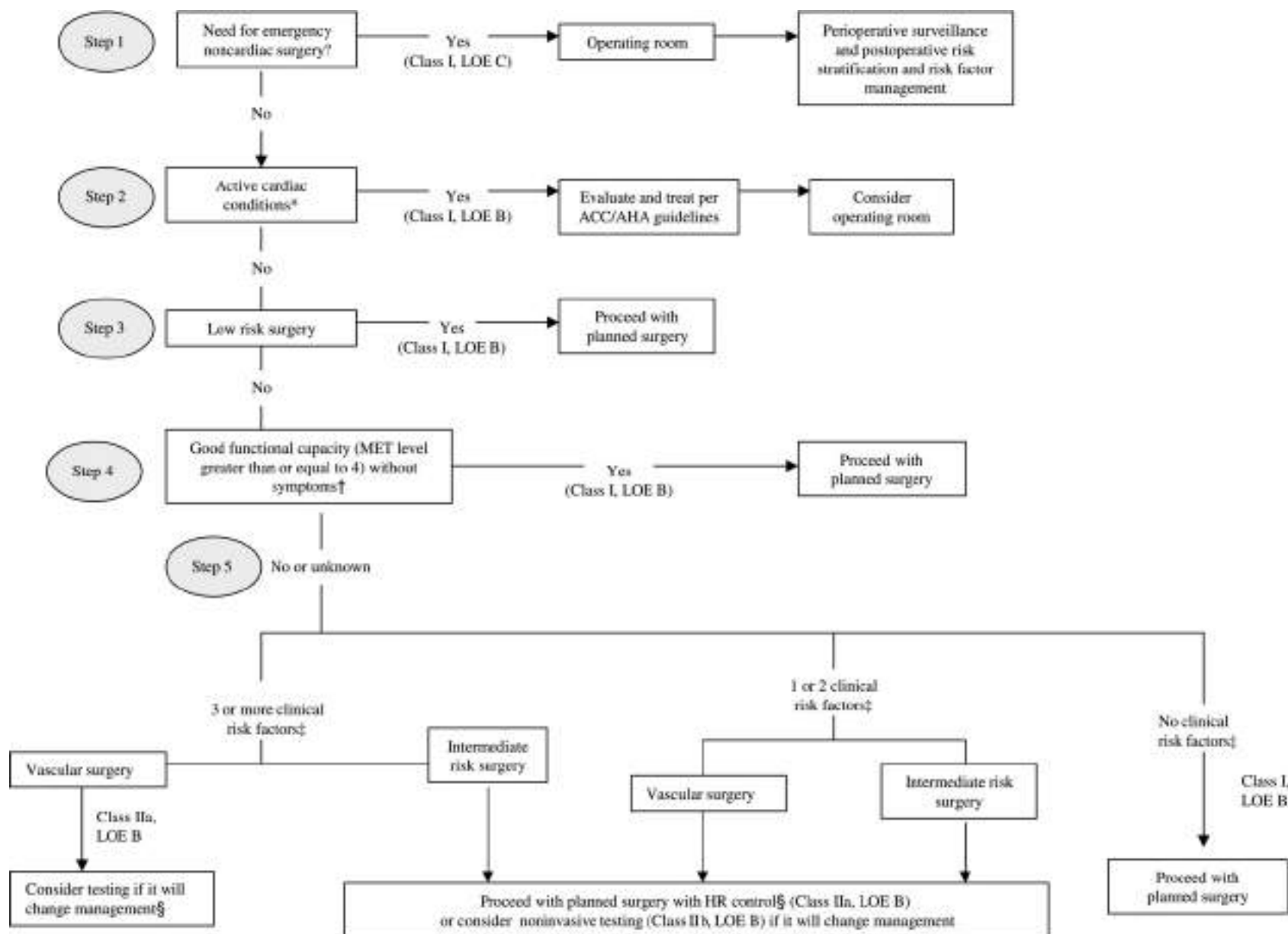
i.	Age > 70 years	5
ii.	MI < 6 months	10
iii.	MI > 6 months	5
iv.	Unstable angina	10
v.	Pulmonary edema < 1 week	10
vi.	Pulmonary edema in the past	5
vii.	Non sinus rhythm PAC	5
viii.	CCVSA class III	10
ix.	CCVSA class IV	20
x.	Critical As	20
xi.	Emergency operation	5
xii.	Poor general status	5
	Total	120

Risk of perioperative reinfarction in patients with recent MI:

Duration since last MI	Re-infarction rate
with in 3 months	30%
3 - 6 months	15%
> 6 months	6%

Lee's revised Cardiac Risk Index-- 1999.

	(Assign one point to each of the following variables)
i.	High risk type of surgery (intra-peritoneal, intra-thoracic or suprainguinal vascular procedures.
ii.	History of IHD (history of MI, positive exercise test, current complaint of ischemic chest pain or use of nitrate therapy of ECG with Q waves)
iii.	History of CHF
iv.	History of cerebro-vascular disease
v.	Insulin therapy for diabetes
vi.	Preoperative serum creatinine > 2.0 mg/gl Class I = 0 factors, Class II = 1 factor Class III = 2 factors Class IV = >2 factors



Cardiac Risk* Stratification for Noncardiac Surgical Procedures
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Risk Stratification	Procedure Examples
Vascular (reported cardiac risk often more than 5%)	Aortic and other major vascular surgery Peripheral vascular surgery
Intermediate (reported cardiac risk generally 1% to 5%)	Intraperitoneal and intrathoracic surgery Carotid endarterectomy Head and neck surgery Orthopedic surgery Prostate surgery
Low† (reported cardiac risk generally less than 1%)	Endoscopic procedures Superficial procedure Cataract surgery Breast surgery Ambulatory surgery

* Combined incidence of cardiac death and nonfatal myocardial infarction.

† These procedures do not generally require further preoperative cardiac testing.

6. Supraglottic devices

Supraglottic airway devices are devices that ventilate patient by delivering/anesthetic gases/O₂ above the level of the vocal cords and are designed to overcome the disadvantages of Et - Intubation.

Advantages:

- Avoidance of laryngoscopy
- Less invasive for respiratory tract
- Better tolerated by patients
- Placement is easier
- Improved hemodynamic stability in emergence
- Less coughing
- Less sore throat
- Less incidence of bronchospasm
- Hands free airway

Classification of SGA(Supraglottic Airway):

Based on:

- Presence or absence of cuff
- Oral/nasal route of insertion
- Anatomic location of the distal portion (sealing mechanism)
- **Cuffed perilaryngeal sealers:** E.g Ambu laryngeal mask, SSLM(softseal laryngeal mask), ILA(intubating laryngeal airway), LMA, ILMA(intubating laryngeal mask airway).
- **Cuffed pharyngeal sealers:** E.g CobraPLA (Cobra perilaryngeal airway)
- **Cuff less pre-shaped sealer:** E.g SLIPA

Supraglottic devices.continuation:

Cuffed perilaryngeal sealers			Cuffed pharyngeal sealers		Cuff less pre-shaped sealers
	Without directional sealing	With directional sealing	Without esophageal sealing cuff	With esophageal sealing cuff	
Devices	LMA, ILMA, SSLM, AMBU LM, ILA	PLMA Proseal LMA	COPA, COBRA PLA,	Laryngeal tube, combitube, easy tube	SLIPA, I-GEL
Sealing mechanism	relies on simple opposition of the cuff that surrounds the larynx	sealing site is at or around the entrance of the larynx	Pharyngeal cuff seals at the base of the tongue		Anatomically preshaped hollow airway which seals the outlet from the pharynx at the base of the tongue to the esophagus, as a result of the resilience of the walls of the shaped airway
Advantages	- Minimal risk of precipitating laryngospasm, - Well tolerated at lower anesthetic levels	- Higher seal pressure - Decreased risk of regurgitation	- Better sealing pressures - Pressure exerted perpendicular to airway channel	- Minimizes aspiration risk - Provides access to esophagus	- Easier to use, - Able to insert through limited mouth opening - Hollow structure provides protection for aspiration - Pre-shaped provides specific positioning and a stable airway
Disadvantages	- Low seal pressure - Little storage space for regurgitated liquid - Increased risk of GE insufflation.	- Harder to insert - More easily rotated out of position - Folding of tip can occur upon insertion, occluding the drainage tube.	- No sealing of the downward outlet increasing the risk of GE aspiration. - No protection from aspiration - Increased need for repositioning	- Increased mucosal trauma due to stiffer tube - Congestion of tongue with excessive increase in cuff pressure and potential lingual nerve damage - Increased need for repositioning	- Difficult to select proper size - Less flexibility in positioning - Not for use in abnormal airway anatomy.

Table 2: Classification of opioids

Traditional	Origin	Function
Strong	Naturally occurring	Pure agonists
Morphine	Morphine	Morphine
Pethidine	Codeine	Fentanyl
Fentanyl	Papavarine	Alfentanil
Alfentanil	Thebaine	Remifentanil
Remifentanil	Semisynthetic	Sufentanil
Sufentanil	Diamorphine	Partial agonist
Intermediate	Dihydrocodeine	Buprenorphine
Buprenorphine	Buprenorphine	Agonists-antagonists
Pentazocine	Synthetic	Pentazocine
Butorphanol	Phenylpiperidines:	Nalbuphine
Nalbuphine	pethidine, fentanyl, alfentanil, sufentanil	Nalorphine
Weak	Diphenylpropylamines:	Pure Antagonists
Codeine	methadone, dextropropoxyphene	Naloxone
	Morphinans:	Naltrexone
	butorphanol, levorphanol	
	Benzomorphans:	
	pentazocine	

Venturi Mask: Flow rates & percentages:

OXYGEN CONCENTRATION(%)	OXYGEN FLOW (L/min)	AIR ENTRAINMENT (L/min)
24 Blue	2-4	50-100
28 White	4-6	40-60
32 Orange	6-8	56
36 yellow	8-10	40
40 Red	8-12	24-36
60 Green	12	33

Table 23.3 Comparative elimination half-life of drugs.

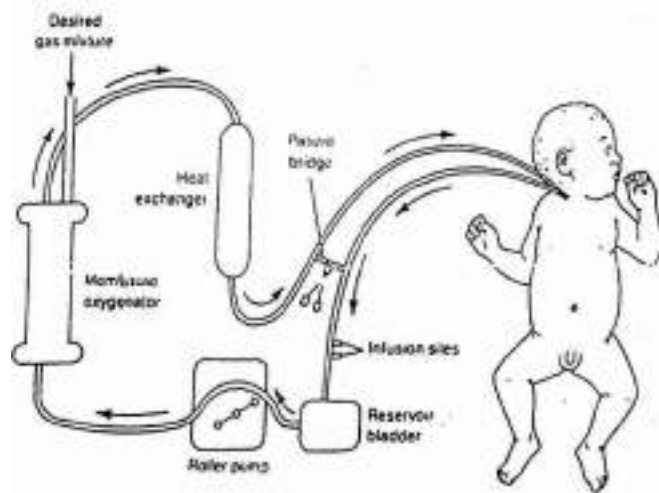
	Young	Old
Fentanyl	250 min	925 min
Alfentanil	90 min	130 min
Diazepam	24 h	72 h
Midazolam	2.8 h	4.3 h
Vecuronium	16 min	45 min

Table 27.3 Suggested medical therapies for hypertension.

Mild to moderate hypertension	• Beta blockers (labetalol, esmolol, metoprolol) • Calcium channel blockers (nicardipine) • Nitro paste • Hydralazine
Severe or refractory hypertension (consider intra-arterial BP monitoring)	• IV antihypertensive infusions – Nicardipine – Nitroglycerine – Nitroprusside

Table 27.4 Causes of postoperative tachycardia.

Noxious stimuli	• Pain, anxiety • Endotracheal tube • Distended bladder
Physiologic derangements	• Acidosis • Hypoxemia • Hypotension and hypovolemia • Hypoglycemia • Increased intracranial pressure • Myocardial ischemia
Medications	• Beta adrenergic vasopressors • Dopamine • Dobutamine • Bronchodilators
Anesthetics	• Ketamine • Isoflurane

**ECMO circuit:**

It utilizes a modified heart lung bypass machine, consisting of a venous blood drainage reservoir, a blood pump, the membrane oxygenator where the exchange of O_2 and CO_2 takes place, and a heat exchanger to maintain temperature.

The pre-operative respiratory care:

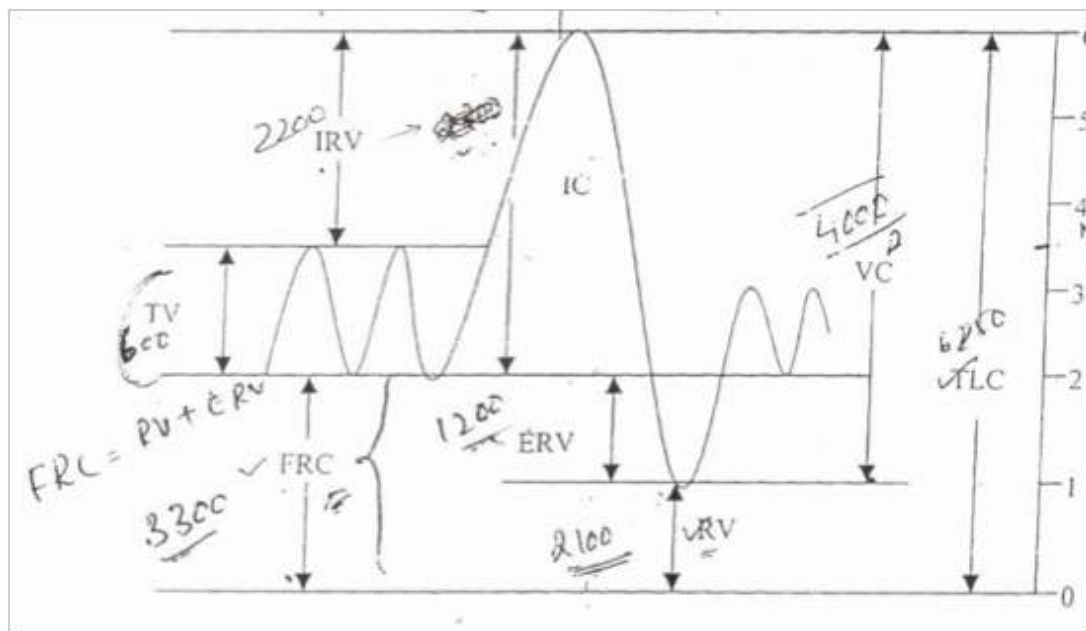
- **Stop Smoking**
- **Dilate the airway using:**
 - Beta agonist
 - Anti-cholinergics
 - Theophylline
 - Steroids
 - Cromolyn Sodium
- **Loosen the Secretions by:**
 - Airway hydration (Humidification/Nebulization)
 - Systemic hydration orally - 3 Liters
 - Mucolytic agents & Expectorant drugs
 - Antibiotics
- **Remove secretions:**
 - Postural drainage
 - Coughing
 - Chest physiotherapy(percussion & Vibration)

Ancillary Measures:

- Treatment of co-existing disease
- Treatment of Cor-Pulmonale
- Pre-operative Digitalization

Patient Education, motivation & facilitation of post operative care:

- Physiological preparation
- Incentive spirometry & deep breathing exercise
- Exposure to secretion removal maneuvers
- Exercise
- Weight gain/reduction
- Diet supplements.



	Obstructive	Restrictive
FEV1	Decreased	Decreased
FVC	Normal	Decreased
FEV1/FVC	Decreased	Normal
FRC	Increased	Decreased
TLC	Increased	Decreased
(Raw)airway resistance	Increased	Normal
MMEFR	Decreased	Normal
MBC	Decreased	Normal
PEFR	Decreased	Decreased

Condition	FEV1	FVC	FEV1 /FVC
Airway obstruction (asthma, bronchitis)	Decreased	Normal	Decreased
Stiff lungs (pneumonia, pulmonary edema)	Decreased	Decreased	Normal
Respiratory muscle weakness (myasthenia, myopathies)	Decreased	Decreased	Normal

Relationship of FRC and CC:

- FRC = CC Neonate and infants
- FRC = CC in supine position at 45 years (40-45)
- FRC = CC in upright position at 65 yrs.

Continuation:

Diagnostic criteria:

Gurd and Wilson criteria:

Major features– petechial rash, respiratory insufficiency, cerebral signs.

Minor features– Pyrexia, tachycardia, retinal changes, jaundice, renal changes.

- Positive diagnosis requires at least one major and four minor clinical features.

Lindeque Criteria:

1. $\text{PaO}_2 < 60$ mmHg on room air.
2. $\text{PaCO}_2 > 55$ mmHg or $\text{pH} < 7.3$
3. Spontaneous respiratory rate > 35 breaths/minute.
4. Clinical signs of increase work of breathing (dyspnea and accessory muscle action).

TREATMENT:

1. **Fluid resuscitation:** The severity of FES is directly related to degree and duration of associated shock. Shock by producing pulmonary damage of its own can intensify the lung injury caused by FES. Aggressive fluid resuscitation must be initiated early to restore intravascular volume, improve preload establish optimal cardiac output. Appropriate monitoring should be used to avoid fluid overload.
2. **Albumin** has been recommended for volume resuscitation along with balanced electrolyte solution, as it not only restore blood volume but also binds fatty acids, reducing their potential for lung damage.
3. **Ventilatory support:** Lung is the primary target organ in FES necessitating the use of aggressive ventilatory therapy. Mechanical ventilation preferably with PEEP must be instituted whenever simple oxygen administration with or without CPAP is insufficient to maintain adequate oxygen saturation.
4. **Drugs:** Steroids-Limits free fatty acid induced inflammatory reaction in lungs.
5. **Aprotinin** – A protease inhibitor decreases aggregation of platelet and release of serotonin and thus limits lung injury.

GRID: Question needs answered under following headings:

1. Definition
2. Etiology
3. Pathophysiology - Theories
4. Clinical features
5. Diagnosis
6. Management.

Time course	Beneficial effects
12-24 hours	Decrease carbon monoxide and nicotine levels
48-72 hours	Normalizes carboxy Hb levels. Improves ciliary function, tissue oxygenation.
1-2 weeks	Decrease sputum production & improves smaller airway function.
4-6 weeks	Improves PFT
6-8 weeks	Normalizes immune function, drug metabolism and hepatic enzyme activity
8-12 weeks	Reduces overall post-operative pulmonary complications.

Table 27.6 Causes of postoperative mental status changes.

Hypoxemia
 Metabolic derangements
 Cerebral hypoperfusion
 Extremes of age
 Emotionally significant operations
 Presence of intraoperative recall
 Scopolamine or atropine
 Substance abuse
 Pain, nausea, pruritus

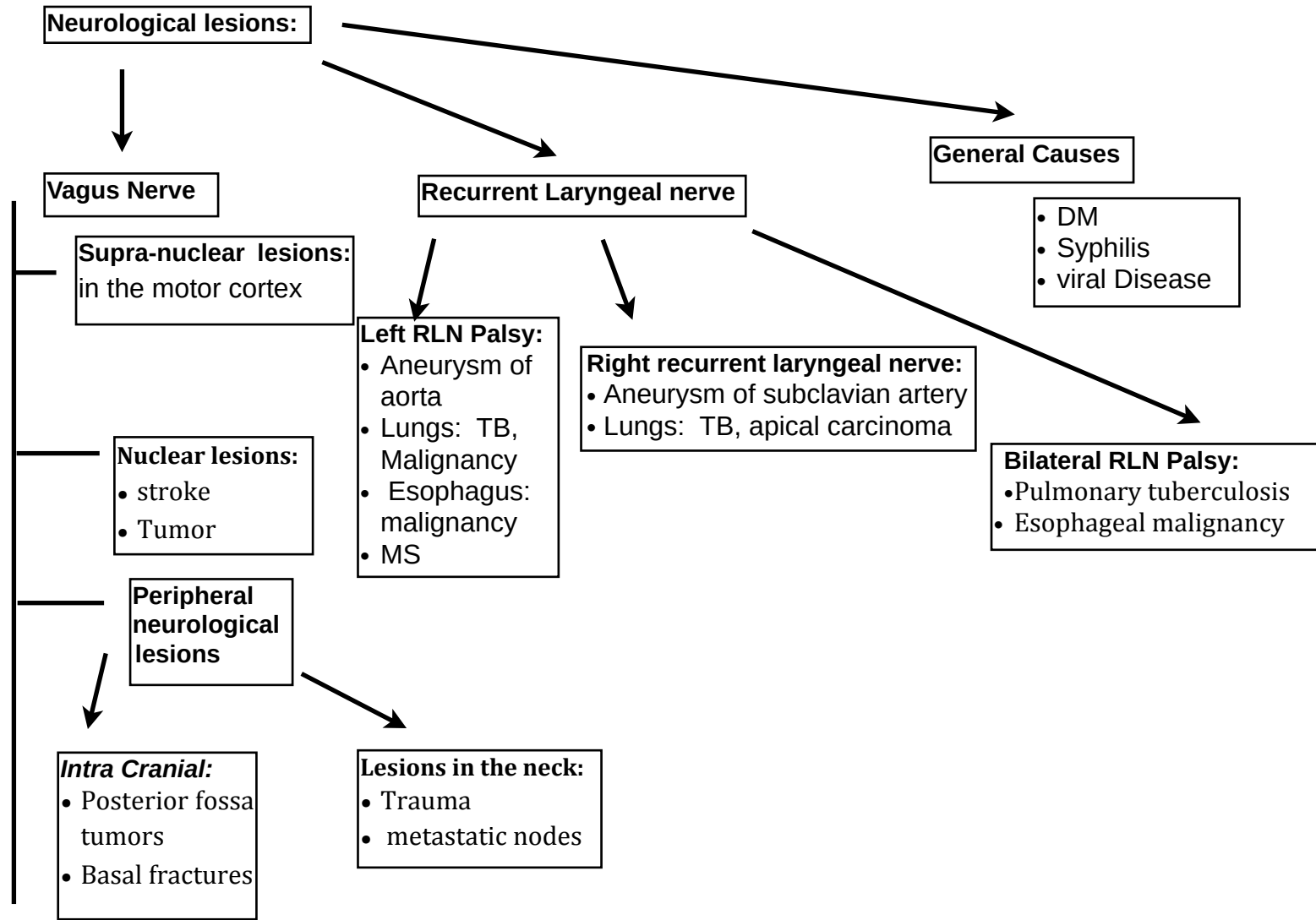
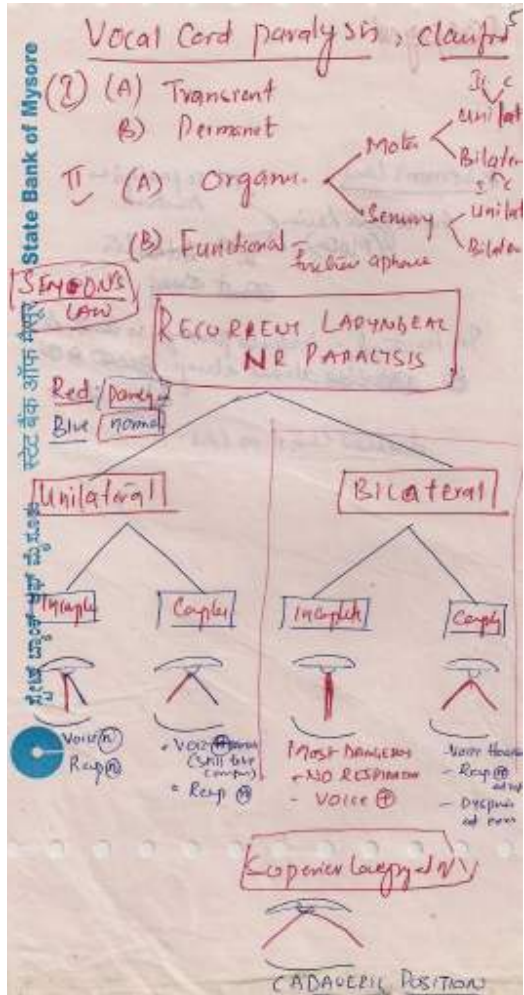
Table 27.5 Common causes of postoperative bradycardia.

Medications	• Neostigmine, edrophonium • Phenylephrine, norepinephrine • Opioids • Succinylcholine • Beta blockers • Local anesthetics • Ganglionic blockers • High spinal/epidural anesthesia
Physical causes	• Carotid sinus massage • Valsalva maneuver • Gagging • Rectal exam • Increased ocular pressure • Distended bladder • Stimulation of pharynx
Metabolic derangements	• Severe acidemia • Hypoxemia

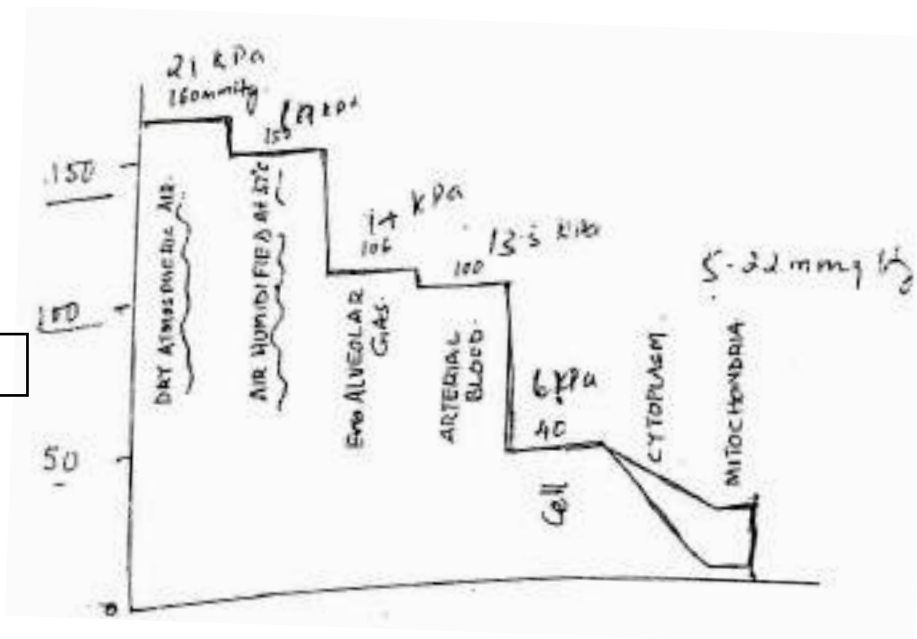
Table 27.9 Effects of hypothermia and shivering.

Hypothermia
• Increased oxygen consumption, carbon dioxide production • Elevates peripheral vascular resistance • Impairs platelet function, decreased clotting factors • Increased infection rates • May lead to cardiac dysrhythmias
Shivering
• Increased oxygen consumption (up to 200%) • Increased carbon dioxide production (up to 200%) • Impairs monitoring devices • May lead to myocardial ischemia • May precipitate ventilatory compromise

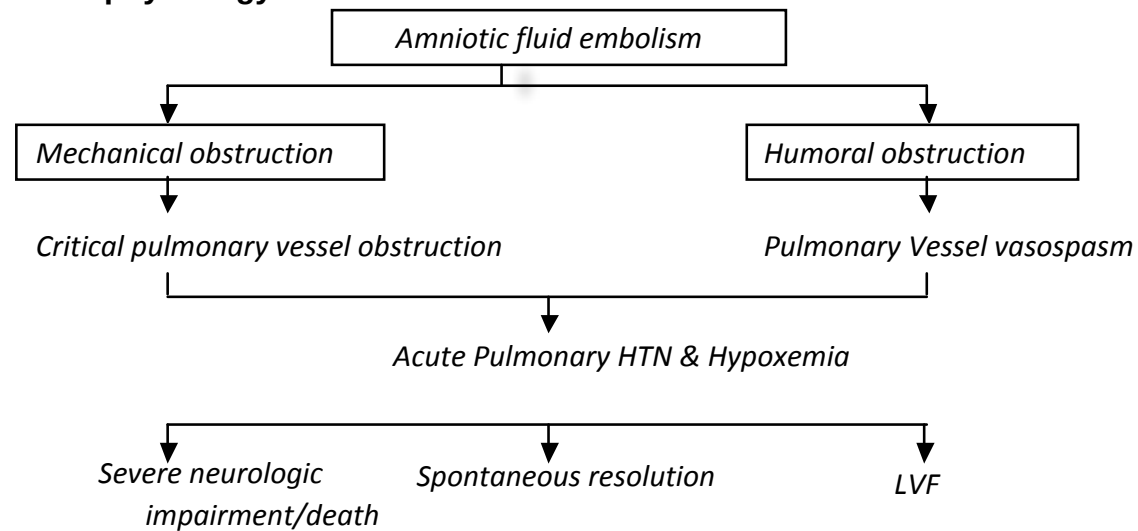
Vocal Cord palsy



1 kPa = 7.6 mmHg



Pathophysiology: Amniotic fluid embolism:

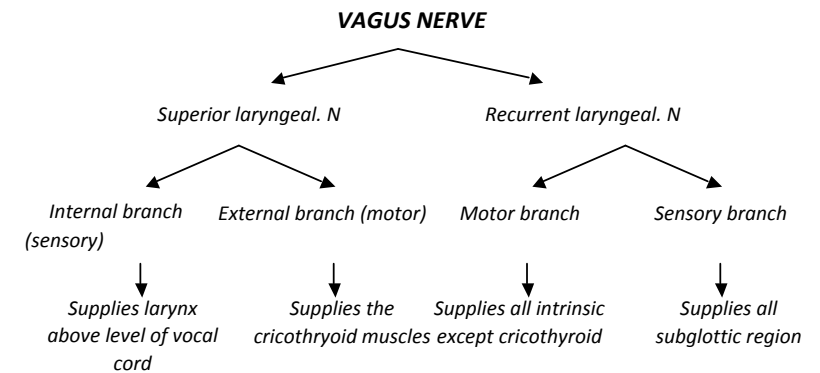


B) Biochemical and other investigation abnormalities:

A four point scoring system for lung injury has been formulated and is as follows:

1. Chest radiograph score	Score
No alveolar consolidation	0
Alveolar consolidation in one quadrant	1
Alveolar consolidation in two quadrants	2
Alveolar consolidation in three quadrants	3
Alveolar consolidation in all four quadrants	4

2. Hypoxemia score	Value	Score
PaO ₂ / FiO ₂	> 300	0
PaO ₂ / FiO ₂	225-299	1
PaO ₂ / FiO ₂	175-224	2
PaO ₂ / FiO ₂	100-174	3
PaO ₂ / FiO ₂	< 100	4
3. Respiratory system compliance score (when ventilated)	Value	Score
Compliance	> 80ml/cm. H ₂ O	0
Compliance	60-79 ml/cm. H ₂ O	1
Compliance	40-59ml/cm. H ₂ O	2
Compliance	20-39/cm. H ₂ O	3
Compliance	< 19 ml/cm. H ₂ O	4
4. Peep score (when ventilated)	Value	Score
PEEP	< 5cm. H ₂ O	0
PEEP	6-8 cm. H ₂ O	1
PEEP	9-11 cm. H ₂ O	2
PEEP	12-14 cm. H ₂ O	3
PEEP	> 15 cm. H ₂ O	4

**Nerve Supply to Larynx:**

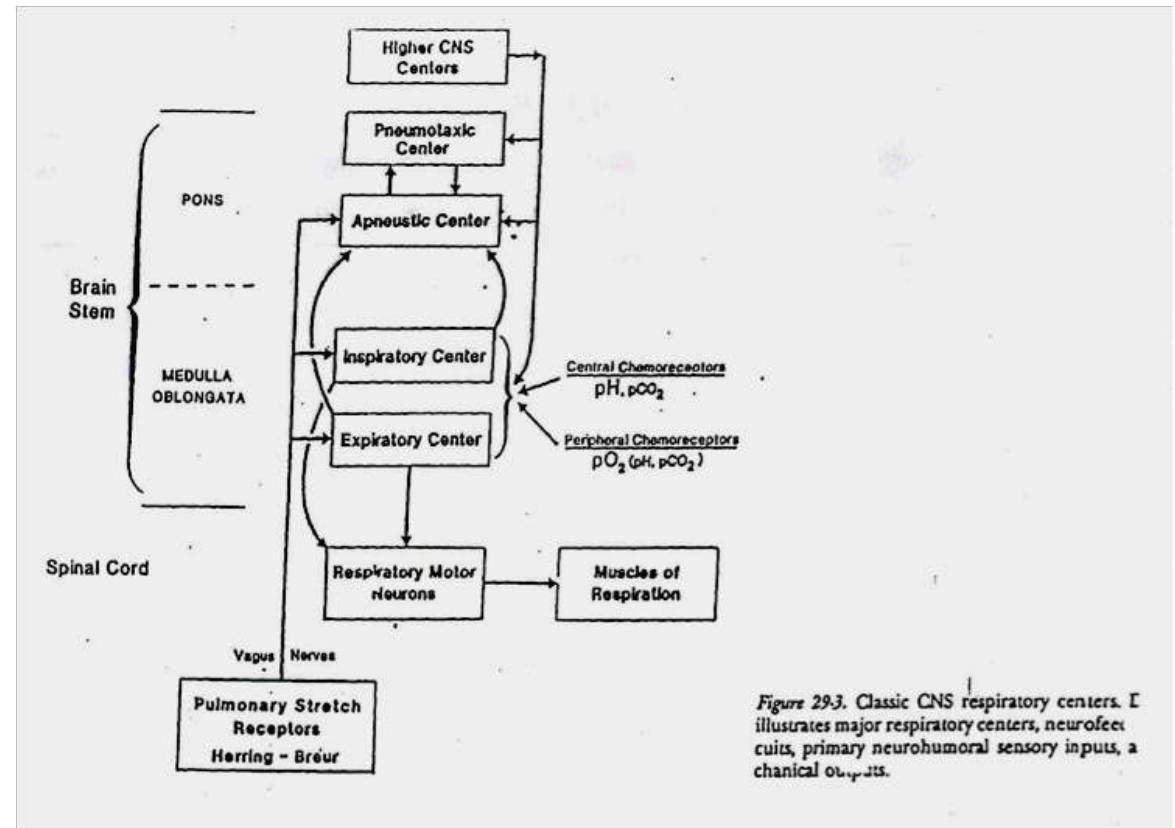
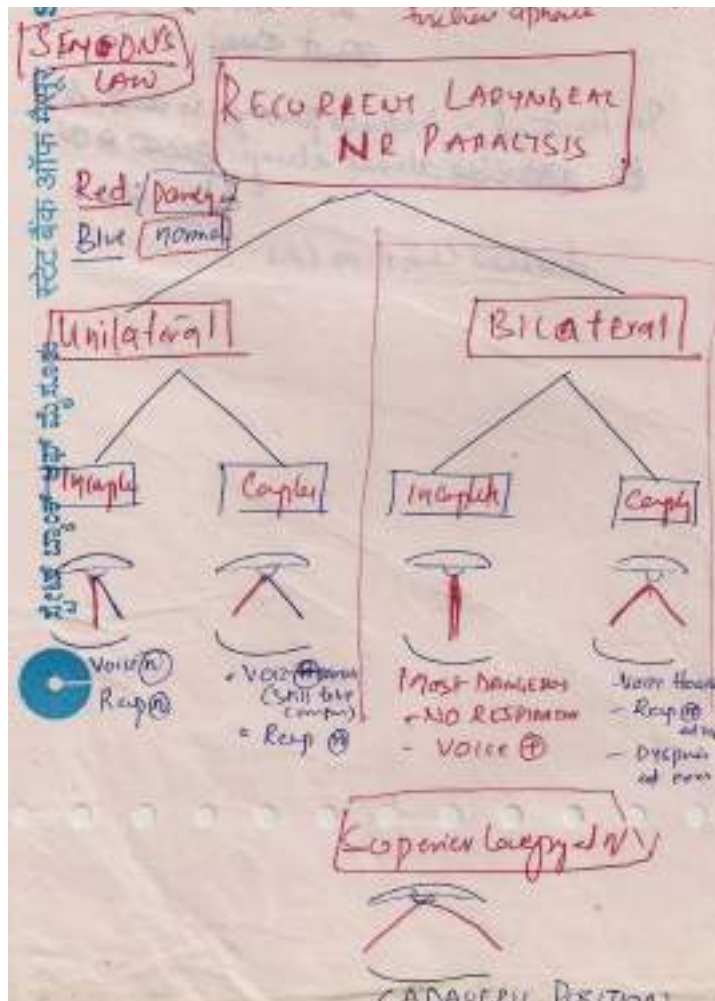


Figure 29-3. Classic CNS respiratory centers. It illustrates major respiratory centers, neurofeedbacks, primary neurohumoral sensory inputs, a chemical outputs.

• Post operative respiratory Complications are:

1. Airway obstruction:

- a. Laryngospasm, bronchospasm - Treatment of cause, Oxygen supplementation, bronchodilators.

2. Hypoxemia: SaO₂ < 90%

• Immediate (1 to 2 Hrs):

- Opioids,
- Loss of pulmonary vasoconstriction,
- shivering
- V/Q mismatch,
- Diffusion Hypoxia

• Late (> 2 Hrs):

- Atelectasis,
- Pulmonary Embolism,
- Pneumothorax,
- Pulmonary Edema

3. Cor-Pulmonale: Digitalization & Diuretics

4. Pulmonary infection:

- Pneumonia

5. Hypoventilation.

- Hypercarbia: Alveolar hypoventilation secondary to pain, inadequate analgesia, inadequate reversal of NMB. - Oxygen supplementation.

6. Aspiration:

7. Atelectasis:

- Decreased FRC,
- Pain,
- splinting of diaphragm,
- decrease sputum clearance.

8. Delayed ambulation

9. DVT & PE:

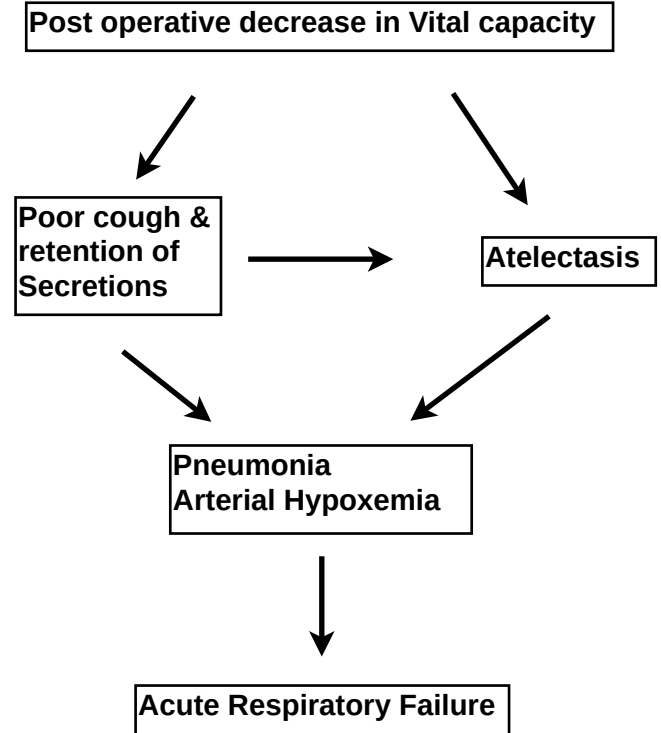
- Polycythemia
- intraoperative hypothermia & Dehydration:
 - LMWH, TED Stockings, early mobilization and Hydration

10. Need for Mechanical ventilation, prolonged ICU stay/ hospital stay

Mechanism for Post operative pulmonary complications:

- VC is ↓ by 25% (<15ml/kg - vital capacity): Decrease ability to cough.
- Residual Volume ↑ by 13% following GA
- ERV ↓ by 25% after lower abdominal surgery & 60% after upper abdominal and thoracic surgery.
- Risk increases as duration of surgery exceeds 3 hours to 4hrs.

Pathogenesis:



Treatment of PPC:

- | | |
|---|--|
| <ul style="list-style-type: none"> • Oxygen supplementation • Incentive Spirometry & Deep breathing exercises • Chest Physiotherapy - Percussion & vibration. • Bronchodilators | <ul style="list-style-type: none"> • Non - Invasive Ventilation - CPAP. • Early Mobilization • Early Nutrition • Cor-pulmonaleDiuretics, Digoxin • Avoiding Respiratory depressants. • Antibiotics & Analgesics • Invasive ventilation - Lastly |
|---|--|

Problems During OLV	Etiology
Hypoxemia	Intrapulmonary shunt during one-lung ventilation
Sudden severe hypotension	Surgical compression of the heart or great vessels
Sudden changes in ventilating pressure or volume	Movement of endobronchial tube/blocker, air leak
Arrhythmias	Direct mechanical irritation of the heart
Bronchospasm	Direct airway stimulation, increased frequency of reactive airways disease
Massive hemorrhage	Surgical blood loss from great vessels or inflamed pleura
Hypothermia	Heat loss from the open hemithorax

Table 59-18 Suggested Ventilation Parameters for One-Lung Ventilation

Parameter	Suggested	Guidelines/ Exceptions
Tidal volume	5-6 mL/kg	Maintain: Peak airway pressure < 35 cm H ₂ O Plateau airway pressure < 25 cm H ₂ O
Positive end-expiratory pressure	5 cm H ₂ O	Patients with COPD: no added PEEP
Respiratory rate	12 breaths/min	Maintain normal PaCO ₂ ; Pa-ETCO ₂ will usually increase 1-3 mm Hg during OLV
Mode	Volume or pressure controlled	Pressure control for patients at risk of lung injury (e.g., bullae, pneumonectomy, post lung transplantation)

Severity of expiratory airflow obstruction & Grading:

Severity	FEV (% predicted)	FEF 25-75 (% predicted)	Pao ₂ (mm Hg)	Pa Co ₂ (mm Hg)
1) <i>Mild (asymptomatic)</i>	65-80	60-75	> 60	< 40
2) <i>Moderate</i>	50-64	45-59	> 60	< 45
3) <i>Marked</i>	35-49	30-44	< 60	> 50
4) <i>Sever (status asthmaticus)</i>	< 35	< 30	< 60	> 50

Table 59-9 Options for Lung Isolation

Options	Advantages	Disadvantages
Double-lumen tube 1. Direct laryngoscopy 2. Via tube exchanger 3. Fiberoptically	Quickest to place successfully Repositioning rarely required Bronchoscopy to isolated lung Suction to isolated lung CPAP easily added Can alternate OLV to either lung easily Placement still possible if bronchoscopy not available	Size selection more difficult Difficult to place in patients with difficult airways or abnormal tracheas Not optimal for postoperative ventilation Potential laryngeal trauma Potential bronchial trauma
Bronchial blockers (BB) Arndt Cohen Fuji	Size selection rarely an issue Easily added to regular ETT Allows ventilation during placement Easier placement in patients with difficult airways and in children Postoperative two-lung ventilation by withdrawing blocker Selective lobar lung isolation possible CPAP to isolated lung possible	More time needed for positioning Repositioning needed more often Bronchoscope essential for positioning Nonoptimal right lung isolation due to RUL anatomy Bronchoscopy to isolated lung impossible Minimal suction to isolated lung Difficult to alternate OLV to either lung
Univent tube	Same as BBs Less repositioning compared with BBs	Same as for BBs ETT portion has higher air flow resistance than regular ETT ETT portion has larger diameter than regular ETT
Endobronchial tube	Like regular ETTs, easier placement in patients with difficult airways Longer than regular ETT Short cuff designed for lung isolation	Bronchoscopy necessary for placement Does not allow for bronchoscopy, suctioning or CPAP to isolated lung Difficult right lung OLV
Endotracheal tube (ETT) advanced into bronchus	Easier placement in patients with difficult airways	Does not allow for bronchoscopy, suctioning or CPAP to isolated lung Cuff not designed for lung isolation Extremely difficult right OLV

• Complications related to single lung ventilation:

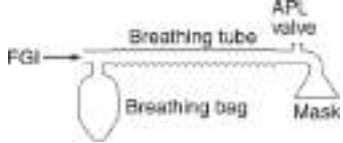
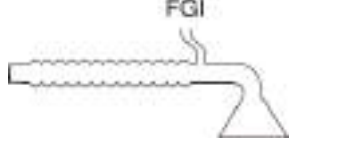
Pulmonary Effects	Cardiovascular Effects	CO2 Insufflation Effects
Hypoxaemia	Hypotension	Hypotension
Hypercapnia	Hypertension	Bradycardia
Impaired HPV	Arrhythmias	Co2 air embolism
Pulmonary Edema	Mediastinal Shift	Subcutaneous Emphysema
Atelectasis		
Pneumonia		

Complications more with Rt Lung as Vascular supply is 10% more compared to the left side.

Table 59-13 Characteristics of the Cohen, Arndt, and Fuji Bronchial Blockers

	Cohen Blocker	Arndt Blocker	Fuji Uniblocker
Size	9 Fr	5 Fr, 7 Fr, and 9 Fr	5 Fr, 9 Fr
Balloon shape	Spherical	Spherical or elliptical	Spherical
Guidance mechanism	Wheel device to deflect the tip	Nylon wire loop that is coupled with the fiberoptic bronchoscope	None, preshaped tip
Smallest recommended ETT for coaxial use	9 Fr (8.0 ETT)	5 Fr (4.5 ETT), 7 Fr (7.0 ETT), 9 Fr (8.0 ETT)	9 Fr (8.0 ETT)
Murphy eye	Present	Present in 9 Fr	Not present
Center channel	1.6 mm ID	1.4 mm ID	2.0 mm ID

Classification & characteristics of various Mapleson circuit

Mapleson Class	Other Names	Configuration ¹	Spontaneous	Controlled	Comments
A	Magill attachment		Equal to minute ventilation (80 mL/kg/min)	Very high and difficult to predict	Poor choice during controlled ventilation. Enclosed Magill system is a modification that improves efficiency. Coaxial Mapleson A (Lack breathing system) provides waste-gas scavenging.
B			2 x minute ventilation	2–2½ x minute ventilation	
C	Waters' to-and-fro		2 x minute ventilation	2–2½ x minute ventilation	
D	Bain circuit		2–3 x minute ventilation	1–2 x minute ventilation	Bain coaxial modification: fresh gas tube inside breathing tube.
E	Ayre's T-piece		2–3 x minute ventilation	3 x minute ventilation (I:E =1:2)	Exhalation tubing should provide a larger volume than tidal volume to prevent rebreathing. Scavenging is difficult.
F	Jackson-Rees' modification		2–3 x minute ventilation	2 x minute ventilation	A Mapleson E with a breathing bag connected to the end of the breathing tube to allow controlled ventilation and scavenging.

4. Classify vaporizers. Discuss the effect of altered barometric pressure on the performance of vaporizers?

Vapour: A Vapour is the gaseous form of a substance which is liquid at room temperature

Vaporizer: A vaporizer is an instrument designed to change a liquid anesthetic agent in to its vapour and to add a controlled amount of this vapour to the fresh gas flow.

Classification of vaporizer:

Oldest Classification:

A. Method of regulating Output Concentration

1. Variable Bypass
2. Measured Flow

B. Method of Vaporization

1. Flow Over
2. Bubble Through
3. Flow over or Bubble Through

C. Location

1. Outside the Breathing System
2. Inside the Breathing System

D. Temperature Compensation

1. None
2. By Supplied Heat
3. By Flow Alteration

E. Specificity

1. Agent Specific
2. Multiple Agent

Older Classification:

A. Method of regulating Output Concentration

1. Variable Bypass
2. Measured Flow

B. Method of Vaporization

1. Flow Over
2. Bubble Through
3. Injection

C. Resistance

1. Plenum
2. Low resistance

D. Temperature Compensation

1. Thermocompensation
2. Supplied Heat

E. Specificity

1. Agent Specific
2. Multiple Agent

New Classification:

A. Method of regulating Output Concentration

1. Variable Bypass
2. Measured Flow

B. Method of Vaporization

1. Flow Over
2. Bubble Through
3. Injection

C. Temperature Compensation

1. Mechanical
2. Supplied Heat
3. Computerized

Preoperative Preparation:

- **Stop Smoking**
- **Dilate the airway using:**
 - Beta agonist
 - Anti-cholinergics
 - Theophylline
 - Steroids
 - Cromolyn Sodium
- **Loosen the Secretions by:**
 - Airway hydration (Humidification/Nebulization)
 - Systemic hydration orally - 3 Liters
 - Mucolytic agents & Expectorant drugs
 - Antibiotics
- **Remove secretions:**
 - Postural drainage
 - Coughing
 - Chest physiotherapy (percussion & Vibration)

Ancillary Measures:

- Treatment of co-existing disease
- Treatment of Cor-Pulmonale
- Pre-operative Digitalization

Patient Education, motivation & facilitation of post operative care: Continuation:

- Psychological preparation
- Incentive spirometry & deep breathing exercise
- Exposure to secretion removal maneuvers
- Exercise
- Weight gain/reduction
- Diet supplements.

<i>Time course</i>	<i>Beneficial effects</i>
12-24 hours	Decrease carbon monoxide and nicotine levels
48-72 hours	Normalizes carboxy Hb levels. Improves ciliary function, tissue oxygenation.
1-2 weeks	Decrease sputum production & improves smaller airway function.
4-6 weeks	Improves PFT
6-8 weeks	Normalizes immune function, drug metabolism and hepatic enzyme activity
8-12 weeks	Reduces overall post-operative pulmonary complications.

Post-operative Management:

Goals:

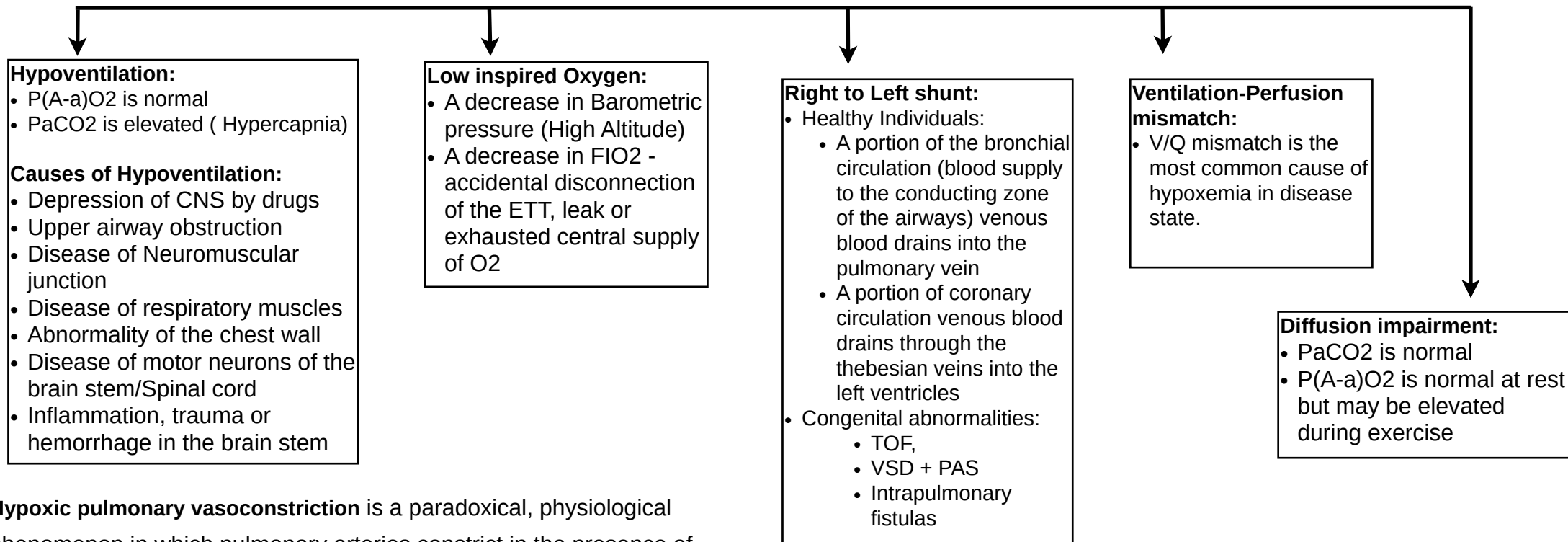
- To promote adequate analgesia
- To minimize the incidence of postoperative complications
- To prevent infections
- Chest physiotherapy & deep breathing exercise.
- Consider CPAP - Decreases WOB, Improves LV Compliance.
- Continuous administration of Local anesthetic with low dose opioids via epidural catheter for post operative pain relief.

57. What are the major causes of Hypoxemia? What is Hypoxic Pulmonary Vasoconstriction? How can general anesthesia worsens V/Q mismatch?

Five Causes of Hypoxemia ($\downarrow$ PaO₂):

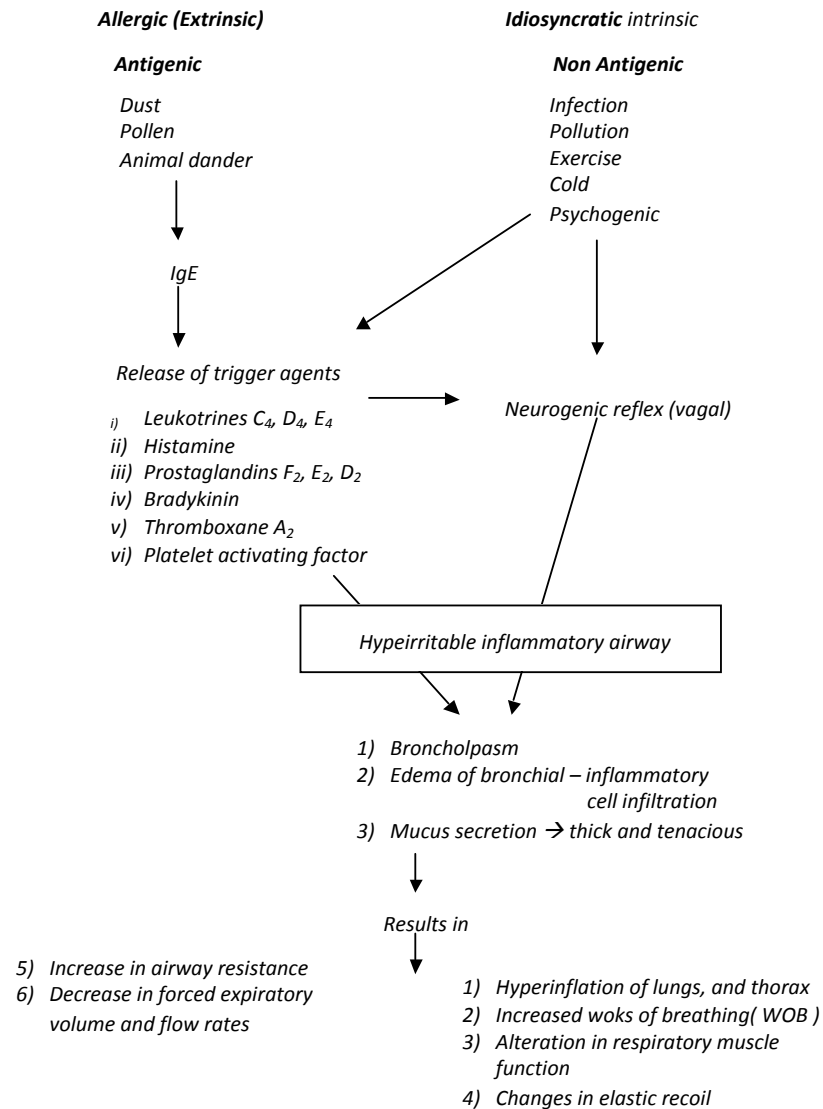
1. Hypoventilation
2. Low inspired Oxygen
3. Right to Left shunt
4. Ventilation-Perfusion mismatch
5. Diffusion impairment

Five Causes of Hypoxemia ($\downarrow$ PaO₂):



Hypoxic pulmonary vasoconstriction is a paradoxical, physiological phenomenon in which pulmonary arteries constrict in the presence of hypoxia without hypercapnia, redirecting blood flow to alveoli with a higher oxygen content.

Pathophysiology of Bronchial Asthma:



Severity of expiratory airflow obstruction & Grading:

Severity	FEV (% predicted)	FEF 25-75 (% predicted)	Pao ₂ (mm Hg)	Pa Co ₂ (mm Hg)
1) Mild (asymptomatic)	65-80	60-75	> 60	< 40
2) Moderate	50-64	45-59	> 60	< 45
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7. A 30 year old female ASA grade I following exploratory laparotomy is not maintaining oxygen saturation in the post operative period. Discuss the causes & Management.

Definition: Hypoxia is defined as inadequate oxygen supply to the cells and tissues of the body.

Values: Sao2 < 90%, PaO2 < 60%

Causes of Post operative Hypoxia & its treatment: (12 Causes)

Low Inspired concentration of O2:

- When FIO2 is < 0.21%. The treatment would be giving high inspired oxygen through Venturi mask.

Airway obstruction:

- Pharyngeal obstruction: Treatment would be Head tilt & chin lift.
- Laryngeal obstruction: treatment: Lift the chin while the head is on a pillow or displace jaw forward. Alternating lifting and relaxing the chin will, relax the strap muscle of the neck.
- Attempt to facilitate ventilation of gentle positive pressure via mask. When conservative measures are not effective in breaking spasm within 1min, then a muscle relaxant should be given I.V. Scoline 1.5mg/kg injected
- Bronchospasm: Bronchodilators like Beta agonist, Anticholinergics, Theophylline and Steroids

Post operative shivering:

- May also cause hypoxia. Treatment is Oxygen therapy

Sudden reduction in cardiac output:

- Can contribute to hypoxemia. Treatment: Watch out for bleeding & dehydration. Give IV fluids, blood replacement.

Diffusion Hypoxia:

- At the end of surgery when both oxygen & nitrous oxide stopped at the same time. Where N2O diffuses back into the alveoli. Treatment is to give oxygen after cutting off N2O.

Type of Surgery:

- Patients with abdomino-thoracic procedures will not respire adequately because of pain on deep inspiration. Treatment would be adequate analgesia with opioids or NSAIDS or through the epidural.

Ventilation Perfusion Mismatch:

- Usually seen under Anesthesia.

Decreased FRC:

- Seen in Pulmonary edema (Treatment will be fluid restriction, diuretics & oxygen therapy Positive pressure ventilation.), Infections(Antibiotics), Obesity (incentive spirometer & deep breathing exercises), aspiration(postural drainage, nebulization incentive spirometer & deep breathing exercises.)

Right to left intrapulmonary shunts:

- Most common cause is Atelectasis. Treatment would be Deep breathing exercise, Chest physiotherapy humidification of inspired gases, coughing & incentive spirometer.

Drug Overdosage:

- Like opioids, muscle relaxants, in adequate reversal. Treatment would be titrated dosage of opioids & to wait for adequate reversal.

Pulmonary Embolism:

- Lower limb surgeries, prolonged immobilization.treatment would be DVT stocking, early mobilization, DVT prophylaxis.

Pneumothorax:

- Result of direct injury to lungs or rib fracture.
- Treatment is 100% oxygen, insertion of chest tube & intercostal drainage & IPPV.

GRID: Question needs answered under following headings:

1. Definition
2. values
3. 12 Causes of Post operative Hypoxia & its management.

3. Pulmonary Function Test

Non-Specific - Bed Side Test

Specific Test - PFT

Test of Ventilation

Test of Distribution of Ventilation

Test of Diffusion

Test of Perfusion

Other Tests

Single Breath N₂ elimination Test
Multi-breath N₂ wash out test
Radio-active Xenon distribution

Carbon Monoxide Diffusing Capacity

radio-active Xenon injection
Radio-nucleotide
Macro-aggregated iodine

- Split Lung Function Test
 - Bronchspirometry
 - radio-active Scanning
 - Pulmonary artery occlusion test
- ABG
- A-a Gradient
- Raw
- Compliance

Non-Specific - Bed Side Function:

- Breath Holding test
- Snider's Match Blowing test
- De Bono's Whistle test
- Auscultation over the trachea
- Sphygmomanometer
- Walk test
- Stair case test.

Static Test

Dynamic Test

- Anatomical Measurements
- Do not evaluate Function

Measures rate of flow of Gas

4 Volumes
TV
IRV
ERV
RV

4 Capacities
VC
TLC
FRC
IC

MBC/MVV
FEV
PEFR
MMEFR 25 to 75 %
Respiratory Muscle Strength

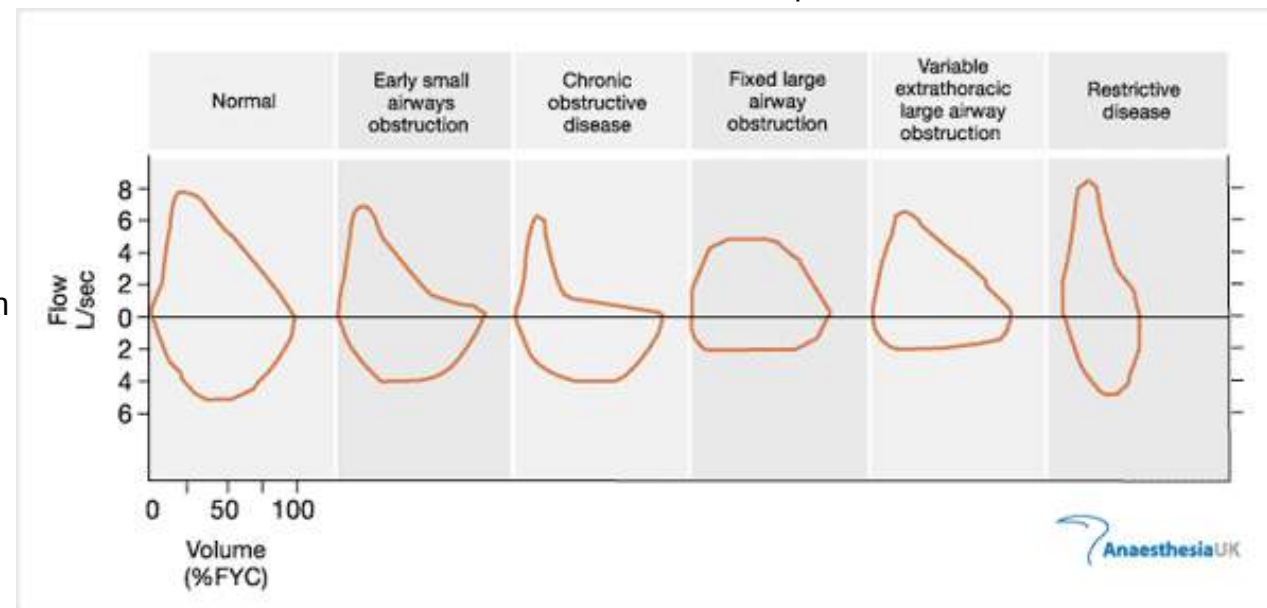
Flow Volume Loops

Fixed Obstruction

Variable Extra-Thoracic Obstruction
E-Expiration is normal

Variable Intra-Thoracic Obstruction
I-Inspiration is normal

Restrictive Lung Disease



Pulmonary Function Test

- Spirometer can measure - VT, IRV, ERV, VC, IC, FEV & MBC
- Cannot be measured by Spirometer - RV, TLC & FRC

Static Test

VT - Tidal Volume

- Total Volume of air inspired & Expired in each breath
- measured by wright's Respirometer

IRV - Inspiratory reserve volume

- Volume of air that can be inspired after normal inspiration.

ERV - Expiratory reserve volume

- Volume of air that can be expired after normal expiration
- It reflects the thoracic muscles, abdominal muscle & thoracic mobility

RV - Residual Volume

- Volume of air remaining in the lung after maximum expiration
- Measured by
 - N2 washout technique
 - Helium dilution technique

VC - Vital Capacity

- Most commonly measured
- volume of air that can be expired after maximum inspiration
- can be measured by - spirometer
- BSA: Male- 2.6l/m² Female - 2.1l/m²
- A given VC is abnormal if < 80% of predicted value
- **VC: decreased** - in pregnancy, Pulm fibrosis
 - Emphysema
 - ch. Bronchitis
 - asthma
 - Abdominal pain
 - can be < 70 to 75% in upper abdominal surgery
 - < 50 to 55% in lower abdominal surgery

- Position: Lithotomy Vc < 18%

- $VC = 3 \times VT$ for effective cough

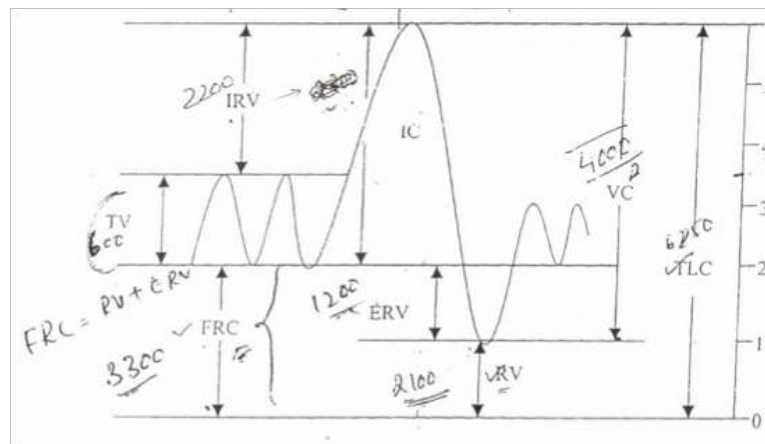
- Decrease in VC means decreased ability to cough and post operative complications

FRC

- Volume of air remaining in the lung after normal expiration
- measured by:
 - N2 washout technique
 - Helium dilution
 - Body Plethysmograph
- **FRC increased:**
 - Obstruction of airways
 - Emphysema
 - PEEP
- **FRC decreased:**
 - Under Anesthesia
 - post op old age
 - obesity
 - smoking
 - Lithotomy
 - Abdominal distention
 - Lap surgeries

TLC

- Total volume of air in lung after maximum inspiration.
- TLC decreased:
 - Pulmo fibrosis
 - obesity
 - weakness of inspiratory muscle



Dynamic Tests

MBC or MVV

- Max volume of air that can be breathed per minute by voluntary effort
- normal - 160L/min to 180 L/min

MBC is decreased in:

- old age
- emphysema
- bronchospasm
- bronchiolar obstruction

FVC & FEV

FVC

- Assess the expiratory ability
- max volume of air that can be exhaled forcefully and rapidly after maximum inspiration
- measured by Spirometer and fast moving Kymograph
- FVC done over 4 sec
- reflects the relationship between Flow & resistance

FEV1

- Max volume of gas exhaled during the 1st second of FVC maneuver
- FEV1 = 83% of VC

FVC is decreased in:

- Atelectasis
- Fibrosis
- muscle weakness
- abdominal swelling
- pain

In healthy patients
FVC=VC

PEFR:

- After max inspiration patient exhales as forcefully as he can and the max flow rate is measured and expressed in lt/min and lt/sec
- Normal:
 - 450-600 L/min
 - 300 - 500 L/min
- Measured by Wright's Respirometer
Pneumotachygraph

PEFR is decreased in:

- Obstructed Airway

Values less than 200 lt/min suggest impaired cough and increased likelihood of post op pulmonary complications

MMEFR (25% to 75%):

- Flow measured in middle half of FVC
- obtained by dividing the volume of gas between 25 - 75% by corresponding elapsed time
- Normal = 4 to 5 L/sec

Importance

- more sensitive of small airway obstruction or disease

Respiratory Muscle weakness:

- It is evaluated and measured by maximum static respiratory pressure
- PImax = -125 cm of H₂O - normal
- If PImax = -25 inability to take deep breath
- PEmax = + 200 cm of H₂O - normal
- If PEmax = + 40 cm of H₂O severely impaired coughing ability
 - seen in Emphysema
 - predictor of successful weaning
 - Evaluation of NM disorder

Condition	FEV1	FVC	FEV1 /FVC
Airway obstruction (asthma, bronchitis)	Decreased	Normal	Decreased
Stiff lungs (pneumonia, pulmonary edema)	Decreased	Decreased	Normal
Respiratory muscle weakness (myasthenia, myopathies)	Decreased	Decreased	Normal

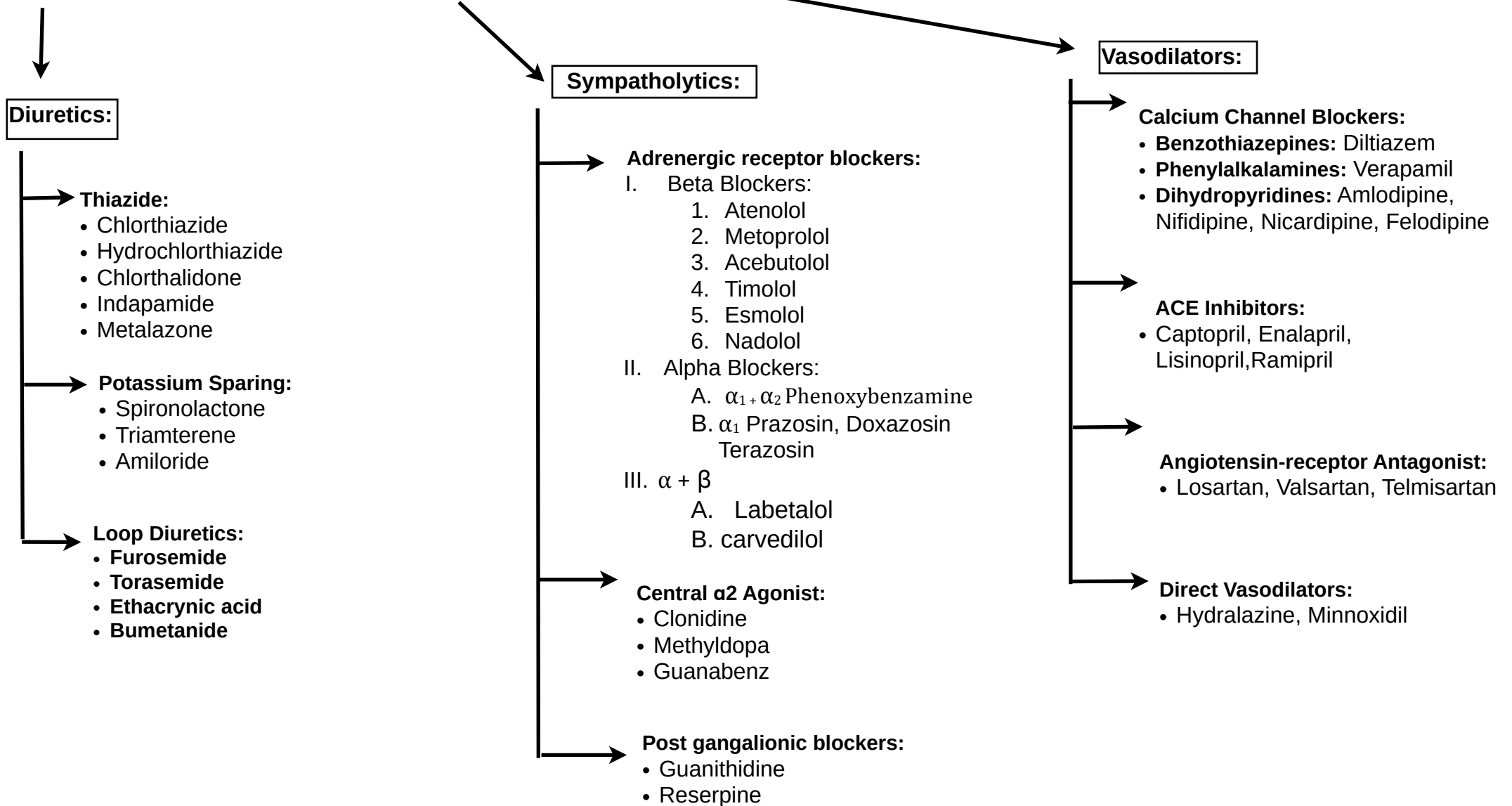
	Obstructive	Restrictive
FEV1	Decreased	Decreased
FVC	Normal	Decreased
FEV1/FVC	Decreased	Normal
FRC	Increased	Decreased
TLC	Increased	Decreased
(Raw)airway resistance	Increased	Normal
MMEFR	Decreased	Normal
MBC	Decreased	Normal
PEFR	Decreased	Decreased

Flow-Volume Loops

- Provide a graphic analysis of flow at various lung volumes
- Used to differentiate between patients with upper airway and lower airway obstruction
- Flow is charted on Y-axis
- Volume is charted on X axis
- Patient is asked to inhale fully to TLC and then perform a FVC maneuver. This is followed immediately by a maximum inspiration back to TLC
- The ratio of expiratory flow to inspiratory flow at 50% of VC is normally 1 (mid VC ratio)

20. Classify Anti-Hypertensive drugs. Describe anesthetic management of hypertensive episode during anesthesia

Oral Anti-Hypertensive Drugs - Classification



Indications	Drugs	Dose	Onset	Duration
For patient with good LV function ↑ HR CI in bronchospastic disease	Esmolol IV	0.5mg/kg over /min 50-300µg/kg/min	1min	12-20min
	Labetalol	5-20mg	1-2min	4-8hr
	Propanol	1-3mg	1-2min	4-6hr
Ca+2 channel blockers Myocardial ischemia ← reflex tachycardia	Nicardipine	0.25-0.5mg	1-5min	3-4 hr
	Nifedipine (S/L)	10mg	5-10min	4h
Mod to severe HTN – most rapid	Nitroprusside	0.5-10µg/kg/min	30-60 sec	1-5min
	Nitroglycerine	0.5-10µg/kg/min	1min	3-5min
Sustained control of BP ← delayed onset reflex tachycardia	Hydralazine	5-20mg	5-20min	4-8hr
	Phentolamine	1-mg	1-10min	20-40mm
	Enalaprilat	0.625-1mg	6-14min	4-6h
ACE Inhibitors	Fenoldopam	0.1-0.6 µg/kg/min	5 min	5 min

Goldman's Cardiac Risk Index - 1977.

i.	Age > years	5
ii.	MI in previous 6 months	10
iii.	S3 gallop or ↑ JVP (0-3 cm)	11
iv.	Important aortic stenosis	3
v.	Rhythm other than sinus, Pac on last preoperative	7
vi.	>5 PVC/min at any time before	7
vii.	PaO ₂ , < 60 or PaCO ₂ > 50mm Hg, K<3.00 mEq/L or HCO ₃ < 20 mEq/l BUN > 50 or Cr> 3 mg/dl. Abnormal SGOT, signs of chronic liver disease, bed ridden from non-cardiac cause	3
viii.	Intraperitoneal, Intrathoracic, aortic surgery	3
ix.	Emergency operation	4
	Total	53

Class I	0-5 points (low risk)
Class II	6-12 points (intermediate risk)
Class III	13-25 points (high risk)
Class IV	≥ 26 points (very high)

Detsky's Modified Cardiac Risk Index- 1986

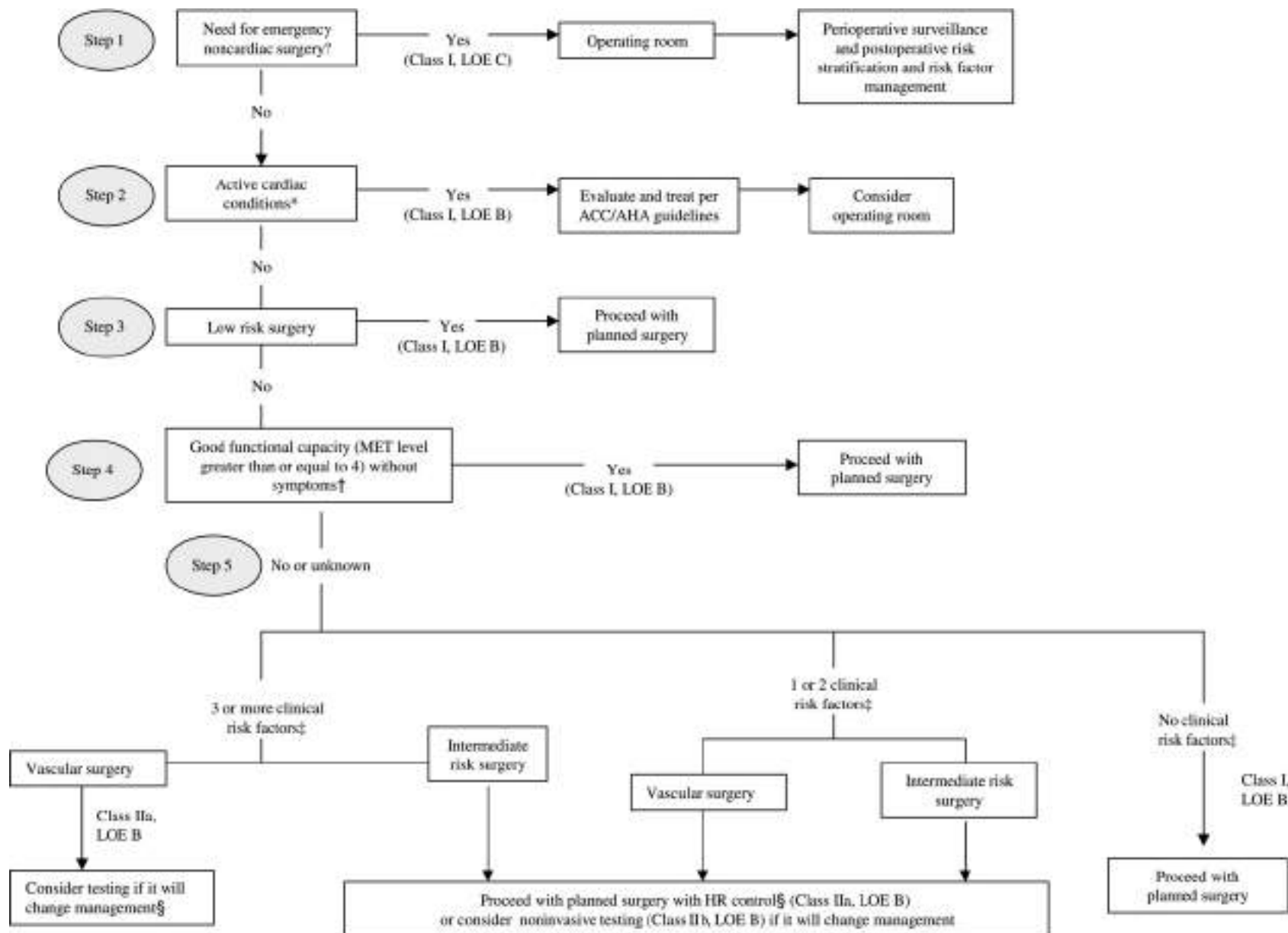
i.	Age > 70 years	5
ii.	MI < 6 months	10
iii.	MI > 6 months	5
iv.	Unstable angina	10
v.	Pulmonary edema < 1 week	10
vi.	Pulmonary edema in the past	5
vii.	Non sinus rhythm PAC	5
viii.	CCVSA class III	10
ix.	CCVSA class IV	20
x.	Critical As	20
xi.	Emergency operation	5
xii.	Poor general status	5
	Total	120

Risk of perioperative reinfarction in patients with recent MI:

Duration since last MI	Re-infarction rate
with in 3 months	30%
3 - 6 months	15%
> 6 months	6%

Lee's revised Cardiac Risk Index-- 1999.

	(Assign one point to each of the following variables)
i.	High risk type of surgery (intra-peritoneal, intra-thoracic or suprainguinal vascular procedures.
ii.	History of IHD (history of MI, positive exercise test, current complaint of ischemic chest pain or use of nitrate therapy of ECG with Q waves)
iii.	History of CHF
iv.	History of cerebro-vascular disease
v.	Insulin therapy for diabetes
vi.	Preoperative serum creatinine > 2.0 mg/gl Class I = 0 factors, Class II = 1 factor Class III = 2 factors Class IV = >2 factors



Cardiac Risk* Stratification for Noncardiac Surgical Procedures
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Risk Stratification	Procedure Examples
Vascular (reported cardiac risk often more than 5%)	Aortic and other major vascular surgery Peripheral vascular surgery
Intermediate (reported cardiac risk generally 1% to 5%)	Intraperitoneal and intrathoracic surgery Carotid endarterectomy Head and neck surgery Orthopedic surgery Prostate surgery
Low† (reported cardiac risk generally less than 1%)	Endoscopic procedures Superficial procedure Cataract surgery Breast surgery Ambulatory surgery

* Combined incidence of cardiac death and nonfatal myocardial infarction.

† These procedures do not generally require further preoperative cardiac testing.

6. Supraglottic devices

Supraglottic airway devices are devices that ventilate patient by delivering/anesthetic gases/O₂ above the level of the vocal cords and are designed to overcome the disadvantages of Et - Intubation.

Advantages:

- Avoidance of laryngoscopy
- Less invasive for respiratory tract
- Better tolerated by patients
- Placement is easier
- Improved hemodynamic stability in emergence
- Less coughing
- Less sore throat
- Less incidence of bronchospasm
- Hands free airway

Classification of SGA(Supraglottic Airway):

Based on:

- Presence or absence of cuff
- Oral/nasal route of insertion
- Anatomic location of the distal portion (sealing mechanism)
- **Cuffed perilaryngeal sealers:** E.g Ambu laryngeal mask, SSLM(softseal laryngeal mask), ILA(intubating laryngeal airway), LMA, ILMA(intubating laryngeal mask airway).
- **Cuffed pharyngeal sealers:** E.g CobraPLA (Cobra perilaryngeal airway)
- **Cuff less pre-shaped sealer:** E.g SLIPA

Supraglottic devices.continuation:

Cuffed perilaryngeal sealers			Cuffed pharyngeal sealers		Cuff less pre-shaped sealers
	Without directional sealing	With directional sealing	Without esophageal sealing cuff	With esophageal sealing cuff	
Devices	LMA, ILMA, SSLM, AMBU LM, ILA	PLMA Proseal LMA	COPA, COBRA PLA,	Laryngeal tube, combitube, easy tube	SLIPA, I-GEL
Sealing mechanism	relies on simple opposition of the cuff that surrounds the larynx	sealing site is at or around the entrance of the larynx	Pharyngeal cuff seals at the base of the tongue		Anatomically preshaped hollow airway which seals the outlet from the pharynx at the base of the tongue to the esophagus, as a result of the resilience of the walls of the shaped airway
Advantages	- Minimal risk of precipitating laryngospasm, - Well tolerated at lower anesthetic levels	- Higher seal pressure - Decreased risk of regurgitation	- Better sealing pressures - Pressure exerted perpendicular to airway channel	- Minimizes aspiration risk - Provides access to esophagus	- Easier to use, - Able to insert through limited mouth opening - Hollow structure provides protection for aspiration - Pre-shaped provides specific positioning and a stable airway
Disadvantages	- Low seal pressure - Little storage space for regurgitated liquid - Increased risk of GE insufflation.	- Harder to insert - More easily rotated out of position - Folding of tip can occur upon insertion, occluding the drainage tube.	- No sealing of the downward outlet increasing the risk of GE aspiration. - No protection from aspiration - Increased need for repositioning	- Increased mucosal trauma due to stiffer tube - Congestion of tongue with excessive increase in cuff pressure and potential lingual nerve damage - Increased need for repositioning	- Difficult to select proper size - Less flexibility in positioning - Not for use in abnormal airway anatomy.

Classification and Management of Blood Pressure for Adults Aged 18 years or Older
JNC 7 Report

BP classification	Systolic BP, mmHg	Diastolic BP, mmHg	Management	
			Initial Drug Therapy	
			Without compelling indications	With compelling indications
Normal	< 120	< 80		
Prehypertension	120 – 139	80 – 89	No antihypertensive drug indicated	Drug(s) for the compelling indications
Stage 1 hypertension	140 – 159	90 – 99	Thiazide-type diuretics for most; may consider ACE inhibitor, ARB, β -blocker, CCB, or combination	Drug(s) for the compelling indications Other antihypertensive drugs (diuretics, ACE inhibitor, ARB, β -blocker, CCB) as needed
Stage 2 hypertension	≥ 160	≥ 100	2-Drug combination for most (usually thiazide-type diuretic and ACE inhibitor or ARB or β -blocker or CCB)	Drug (s) for the compelling indications Other antihypertensive drugs (diuretics, ACE inhibitor, ARB, β -blocker, CCB) as need

Symptoms and Signs of OP Poisoning

MILD	MODERATE	SEVERE
Increased salivation	Excessive salivation	Symptoms for mild and moderate poisoning increased, plus:
Contraction of pupil of eye (miosis)	Small pupil with visual disturbance	Depressed consciousness or coma
Lacrimation (tears in eye)	Lacrimation	Reduced respiration
Nausea	Sweating	Cyanosis
Headache	Vomiting, diarrhea	Convulsions
Weakness (of muscles) or tremor	Headache, drowsiness	Cardiac failure
Dizziness	Weakness increased, muscle tremor, increased muscle tension	
	Disturbed gait (ataxia)	
	Bronchospasm and bronchial hypersecretion	
	Breathlessness increased	

PARAMETERS	HALOTHANE	ENFLURANE	ISOFLURANE	SEVOFLURANE	DESFLURANE	Nitrous Oxide N2O
HISTORY	SUCKLING - 1951	E = Epilepsy	S = Steal, L = Laryngospasm	- S = not to be used with Soda Lime - sevoflurane appeared in the literature in 1971 - Introduced into clinical practice initially in Japan in 1990		- Priestly - 1772 - Fresh Blue cylinder (750 - Psi) - Pin Index = 3,5
BASIC PHARMACOLOGY	2-CHLORO,2-BROMO,111-TRIFLUOROETHANE	Clear colourless liquid with a sweet smell (should be protected from light).		1,1,1,3,3,3-hexafluoro-2-(fluoromethoxy)propane		- Laughing gas - Colourless - Odorless - Obtained by heating Ammonium nitrate at 240 degree centigrade
PHARMACOKINETICS	MAC = 0.75%, Boiling point = 50.2,Blood-gas coefficient = 2.54 Clear colourless liquid, available in Amber bottle. PRESERVATIVE: 0.01% Thymol used to prevent decomposition to light. organic odour	MAC = 1.63%, Boiling point = 56.5,Blood-gas coefficient = 1.90	MAC = 1.17%, Boiling point = 48.5,Blood-gas coefficient = 1.40	MAC = 2%(1.8%), Boiling point = 58.5,Blood-gas coefficient = 0.69 (Hence speeds Induction & recovery)	MAC = 6.6%, Boiling point = 22.8,Blood-gas coefficient =0.42 (Blood -gas coefficient close to N2O) Special vaporizer TEC - 6	MAC = 104%, Boiling point = - 88,Blood-gas coefficient = 0.46
PHARMACODYNAMICS						
CNS	↑ ICP, ↓ CMRO2 25%	Convulsions, ↑ CBF, ↓ CMRO2	depress CNS, Not epileptic & not an analgesic	- Sevoflurane of 0.5 to 1.0 MAC – maintains auto regulation ↑ CBF, ↑ ICP, ↓ CMRO2	↑ CBF, ↑ ICP, ↑ CMRO2	

PARAMETERS	HALOTHANE	ENFLURANE	ISOFLURANE	SEVOFLURANE	DESFLURANE	Nitrous Oxide N2O
RS	• ↑ PaCO2, • Bronchodilator, • ↓ TV, • ↑ RR, • ↑ alveolar ventilation	• Inhibit HPV, • ↓ MV, • Respiratory depressant	• irritating to airways - leading to coughing & laryngospasm • ↓ TV, • ↑ RR, • ↓ FRC • Hypoxemia	• Pleasant to inhale, • ↓ MV, • ↑ RR. • Abolishes HPV Extremely irritating to airways, • ↑ Secretion (bronchial & Salivary) • Potent respiratory depressant • ↓ TV, • ↑ RR, • ↓ MV	• ↓ TV, • Inhibits Carotid body hypoxic drive • Pulmonary vasoconstriction - ↑ PVR	
CVS	• Sensitizes heart to catecholamines • ↓ Myocardial oxygen consumption & depressant • ↓ coronary blood flow, • ↓ Heart rate	• ↑ heart rate • ↓ BP • ↓ Cardiac output • ↓ LV after load	• ↑ Heart rate • ↓ Myocardial O2 consumption • Hypotension • Potent coronary vasodilator & Coronary Steal Phenomenon • DOES NOT SENSITIZES TO EXOGENOUS MYOCARDIUM	• ↓ SVR • Dose dependent decrease in CO ↓ Myocardial contractility • ↓ SVR, • ↓ cardiac work • Causes coronary vasodilatation • DOES NOT SENSITIZES TO EXOGENOUS MYOCARDIUM	• Direct Myocardial depressant	
NEUROMUSCULAR	• Muscle relaxant • Relaxes uterine muscles • Causes halothane shakes	• Relaxation of all abdominal muscles • Relaxes uterine muscles • Augments muscle blocking agents				Analgesic
KIDNEY	• ↓ GFR • ↓ RBF					
LIVER	• ↓ HBF, • ↓ Clearance	Minimal or No change in portal vein flow Compensated by An Increase in hepatic artery blood flow				

PARAMETERS	HALOTHANE	ENFLURANE	ISOFLURANE	SEVOFLURANE	DESFLURANE	Nitrous Oxide N2O
METABOLISM	3 weeks to clear - Oxidative & - reductive (<1%) Enzymes Responsible: - P-450 & 2EI (Oxidative) - CYP 2A6 & CYP 3A4 (Reductive) - Metabolites are TFA-trifluoroacetic acid & Inorganic Cl, FI, Br	Only OXIDATIVE P-450 & 2EI (Hepatic)				
		TFA-trifluoroacetic acid & Inorganic FI	TFA-trifluoroacetic acid	- Hexafluoro isopropanol, Inorganic FI .	TFA-trifluoroacetic acid	
ELIMINATION	60 to 80 % via Lung 20% metabolized in liver	Mostly Lung	Mostly Lung		Mostly Lung	By Lung
OTHERS POINTS	Hepatotoxicity - results in Hepatic necrosis	production of carbon monoxide - due to Soda & Baralyme leading to ↑ Temperature	Rapid awaking	compound A [fluoromethyl-2,2-difluoro-1-(trifluoromethyl)vinyl ether] also called PIFE (pentafluoroisopropenyl fluoromethyl ether) and Compound B [1,1,1,3,3-pentafluoro-2-(fluoromethoxy)-3-methoxypropane] also called PMFE (pentafluoromethoxy isopropyl fluoromethyl ether) on contact with the <u>soda lime</u>		- N2O Toxicity, - N2O impurities - irreversibly oxidizes cobalt atom of Vit B12 inhibiting B12-dependent Enzyme - Bone marrow depression

PARAMETERS	HALOTHANE	ENFLURANE	ISOFLURANE	SEVOFLURANE	DESFLURANE	Nitrous Oxide N2O
CONTRAINDICATIONS	Malignant Hyperthermia (MH)	Epilepsy, MH, & Renal Failure	MH, Coronary Steal	MH, Its toxic hence avoided in Renal, hepatic, & brain tumors	MH, Causes Laryngospasm - hence not used in children under 2 years of age	- It is 35 times more soluble than N₂. - So fills & expand any air-containing cavities: Air Embolism, Pneumothorax, Intracranial air, Lung Cyst, Intraocular air bubbles, Tympanoplasty, Endotracheal tube cuff

Venturi Mask: Flow rates & percentages:

OXYGEN CONCENTRATION(%)	OXYGEN FLOW (L/min)	AIR ENTRAINMENT (L/min)
24 Blue	2-4	50-100
28 White	4-6	40-60
32 Orange	6-8	56
36 yellow	8-10	40
40 Red	8-12	24-36
60 Green	12	33

There is increased sequelae of hypertension, such as heart failure, ischaemic heart disease, renal failure which increases the perioperative complications.

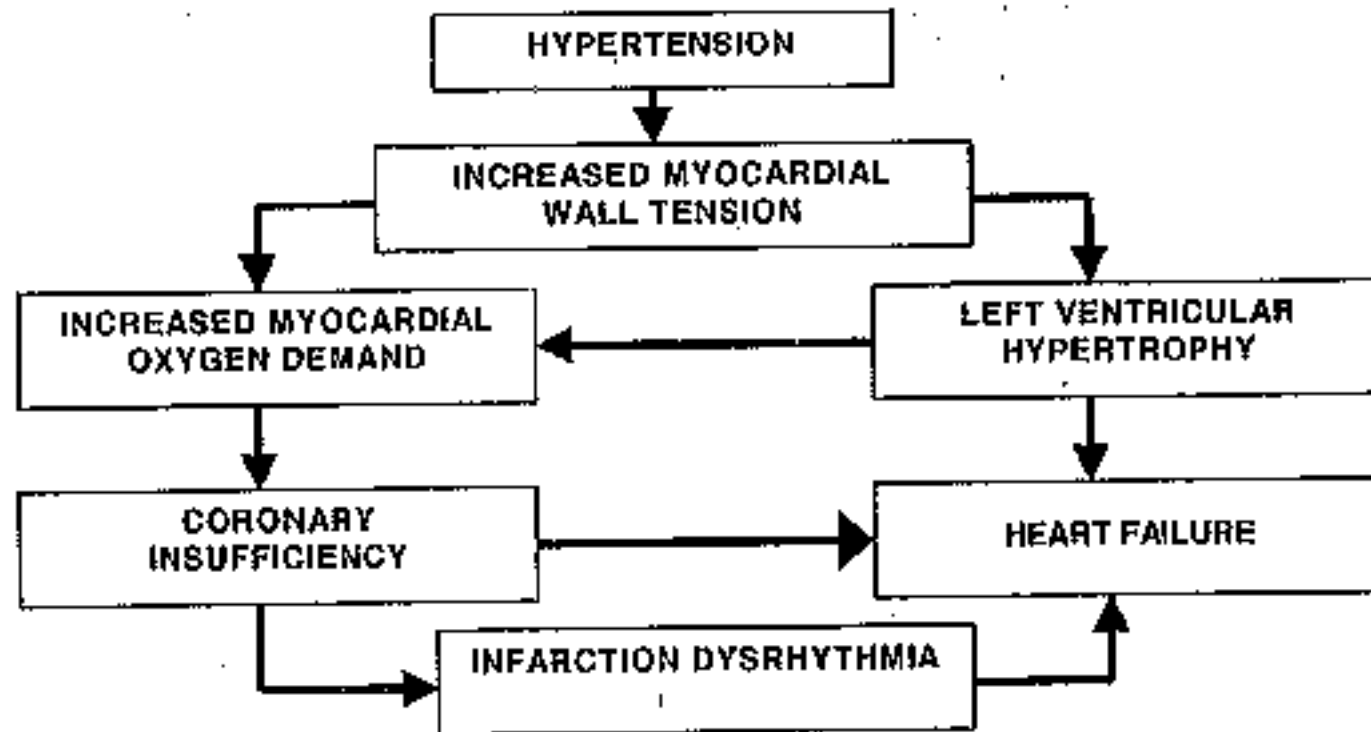


Table summarizes simple acid base disorders and expected compensation:

Primary Acid-Base Disorder	Primary Defect	Effect on pH	Compensatory Response	Expected Range of Compensation	Limits of Compensation
Respiratory Acidosis	Hypoventilation ($\uparrow \text{PCO}_2$)	$\downarrow$	HCO_3^- Generation	$\uparrow [\text{HCO}_3^-] = 1-4 \text{ mEq/L for each } 10 \text{ mm Hg } \uparrow \text{PCO}_2$	$[\text{HCO}_3^-] = 45 \text{ mEq/L}$
Respiratory Alkalosis	Hyperventilation ($\downarrow \text{PCO}_2$)	$\uparrow$	HCO_3^- Consumption	$\downarrow [\text{HCO}_3^-] = 2-5 \text{ mEq/L for each } 10 \text{ mm Hg } \downarrow \text{PCO}_2$	$[\text{HCO}_3^-] = 12-15 \text{ mEq/L}$
Metabolic Acidosis	Loss of HCO_3^- or gain of H^+ ($\uparrow \text{HCO}_3^-$)	$\downarrow$	Increase in Ventilation ($\downarrow \text{PCO}_2$)	$\uparrow \text{PCO}_2 = 1.5([\text{HCO}_3^-]) + 8$	$\text{PCO}_2 = 12-14 \text{ mm Hg}$
Metabolic Alkalosis	Gain of HCO_3^- or loss of H^+ ($\downarrow \text{HCO}_3^-$)	$\uparrow$	Decrease in Ventilation ($\uparrow \text{PCO}_2$)	$\uparrow \text{PCO}_2 = 0.6 \text{ mm Hg for each } 1 \text{ mEq/L } ([\text{HCO}_3^-])$	$\text{PCO}_2 = 55 \text{ mm Hg}$

Table 3: Oral Antihypertensive Drugs

Drug	Usual dose range, total mg/d (Frequency per day)	Selected side effects and comments
Diuretics		
Chlorthalidone	12.5-50 (1)	Small, little hypokalemia, cholesterol and glucose levels; mild renal abnormalities; decreased potassium, sodium and magnesium levels; increased uric acid; calcium levels; and blood dyscrasias; photosensitivity; paroxysmal hypertension
Hydrochlorothiazide	12.5-50 (1) or 25	
Loop diuretics		
Furosemide only	20-40 (2-3)	(Only non-sulfonamide diuretic, electrolyte) (Start careful potassium, no hyperkalemia)
Furosemide	40-80 (2-3) b.i.d.	
Potassium-sparing agents		
Amiloride/hydrochlorothiazide	5-10 (1)	Hyperkalemia Gynecomastia
Spartanone	25-100 (1) or 50	
Tamoxifen	20-100 (1)	
Adenosine inhibitors		
Peripheral agents		
✓ Sildenafil/monosulfate	10-100 (1)	Postural hypotension, dizziness
✓ Reserpine	0.25-0.75 (1)	Nasal congestion, angina, depression, retention of papilloedema Sedation, dry mouth, bradycardia, orthostatic hypotension (More withdrawn) Hepatic and autoimmune disorders
Central agents		
✓ Clonidine hydrochloride	0.2-1.2 (2-3) t.i.d.	Rebound hypertension, orthostatic hypotension, dry mouth, sedation
✓ Guanabenz acetate	0.37-1 (1)	
✓ Mefenorex	200-300 (2) B.i.d.	
β-blockers		
Obetaxol mesylate	1-15 (1)	Rebound hypertension, orthostatic hypotension, dry mouth, sedation
Propranolol hydrochloride	2-30 (2-3)	
Terbutaline hydrochloride	1-20 (1)	
α-blockers		
Alone		
Alone	25-100 (1-2) B.i.d.	Postural hypotension, bradycardia
Alone	50-300 (2) B.i.d.	
Alone	50-300 (2)	
Alone	40-120 (2) B.i.d.	
Combination α and β blockers		
Carvedilol	12.5-50 (1)	Headaches, intolerance, tachycardia (Less common)
Labetalol hydrochloride	200-1200 (2)	
Direct vasodilators		
Hydralazine hydrochloride	50-300 (2)	Conduction defects, worsening of systolic dysfunction, gingival hyperplasia (Nausea, headache) (Constipation) Edema of lower limbs, fatigue, headache, gingival hypertrophy
Calcium antagonists		
Long-acting		
Diltiazem hydrochloride	120-360 (2)	Common cough; low angiotensin, hyperkalemia, tachycardia, autonomic
Verapamil hydrochloride	100-480 (2)	
Short-acting		
Amlodipine mesylate	2.5-10 (1)	Common cough; low angiotensin, hyperkalemia, tachycardia, autonomic
Nifedipine	2.5-20 (1)	
Nitroglycerin hydrochloride	60-90 (2)	
Nitroglycerin	30-120 (1)	
Angiotensin-converting enzyme inhibitors		
Enalapril maleate	25-150 (2-3)	Common cough; low angiotensin, hyperkalemia, tachycardia, autonomic
Lisinopril	5-40 (1-2)	
Perindopril maleate	5-10 (1-2)	
Quinapril	10-40 (1-2)	
Trandolapril	1-2.5 (1-2)	
Angiotensin II receptor antagonists		
Losartan potassium	25-100 (1-2)	Angiotensin (very rare), hyperkalemia
Valsartan	80-320 (1)	
Telmisartan	20-50 (1)	

Table 4: Parenteral Drugs for Treatment of Hypertensive Emergencies

Drug	Dose	Onset of action	Duration of action	Adverse effects	Special indications
Vasodilators					
✓ Sulfamethoxazole	0.25-10 mg/kg per minute as IV infusion (maximal dose for 10 min only)	Immediate	1-2 min	Nausea, vomiting, muscle twitching, sweating, hypotension and cyanide intoxication	Most hypertensive emergencies; caution with high intracranial pressure or aortic aneurysm (continuous infusion)
✓ Nitroglycerin	5-100 µg/min as IV infusion	2-5 min	3-5 min	Headache, vomiting, methemoglobinemia, tolerance with prolonged use	Coronary ischemia (MI, angina, etc.)
✓ Hydralazine hydrochloride	10-20 mg IV 10-50 mg IM	10-20 min 20-30 min	3-6 h	Tachycardia, flushing, headache, vomiting, aggravation of angina	Echthyma
✓ Labetalol hydrochloride	20-80 mg IV bolus every 10 min 0.5-2 mg/min IV infusion	5-10 min	3-8 h	Bradycardia or AV block, vomiting, scalp tingling, burning in 2nd, 3rd, 4th, 5th, 6th, 7th, 8th, 9th, 10th, 11th, 12th, 13th, 14th, 15th, 16th, 17th, 18th, 19th, 20th, 21st, 22nd, 23rd, 24th, 25th, 26th, 27th, 28th, 29th, 30th, 31st, 32nd, 33rd, 34th, 35th, 36th, 37th, 38th, 39th, 40th, 41st, 42nd, 43rd, 44th, 45th, 46th, 47th, 48th, 49th, 50th, 51st, 52nd, 53rd, 54th, 55th, 56th, 57th, 58th, 59th, 60th, 61st, 62nd, 63rd, 64th, 65th, 66th, 67th, 68th, 69th, 70th, 71st, 72nd, 73rd, 74th, 75th, 76th, 77th, 78th, 79th, 80th, 81st, 82nd, 83rd, 84th, 85th, 86th, 87th, 88th, 89th, 90th, 91st, 92nd, 93rd, 94th, 95th, 96th, 97th, 98th, 99th, 100th, 101st, 102nd, 103rd, 104th, 105th, 106th, 107th, 108th, 109th, 110th, 111th, 112th, 113th, 114th, 115th, 116th, 117th, 118th, 119th, 120th, 121st, 122nd, 123rd, 124th, 125th, 126th, 127th, 128th, 129th, 130th, 131st, 132nd, 133rd, 134th, 135th, 136th, 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Table 5: Antihypertensive Drugs Used in Pregnancy

Suggested drug	Comments
Central α -agonists	Methyldopa is the <u>drug of choice</u> recommended by the NHBPEP Working Group
β -blockers	Atenolol and metoprolol appear to be safe and <u>effective in late pregnancy</u> . Labetalol hydrochloride also appears to be effective (α - and β -blockers)
Calcium antagonists	Potential synergism with magnesium sulfate may lead to precipitous hypotension
ACE inhibitors, angiotensin II receptors blockers	Fetal abnormalities, including death, can be caused, and these drugs should not be used in pregnancy
Diuretics	Diuretics are recommended for chronic hypertension if proscribed before gestation or if patients appear to be salt-sensitive. They are <u>not recommended</u> in preeclampsia
Direct vasodilators	Hydralazine hydrochloride is the <u>parenteral drug of choice</u> based on its long history of <u>safety and efficacy</u> .

Table 1 : Neuro-ophthalmic Reflexes*

Reflex	Pathway		Precipitating Factors	Symptoms and Signs	Prophylaxis and treatment
	Afferent	Efferent			
Oculo Cardiac	Long and short ciliary N to ciliary ganglion	Motor nucleus of vagus	Pressure, torsion, pulling on extraocular muscles	Slow: Bradycardia Ectopic beats Sinus arrest	IM / IV Atropine Or Glycopyrrolate
Oculo Respiratory	Same as oculocardiac	Via connection between trigeminal sensory nucleus and pneumatic center and medullar resp. centre		Shallow breathing bradypnoea or respiratory arrest	Controlled ventilation on children undergoing squint surgery
Oculo Ematic		Reflex action	Traction on extraocular muscles	Vomiting	

*Source : Shashi Kiran et al (1999) :Anaesthesia for ophthalmic surgery :Hospital ; IV, 4,

Table 56-1 Anticoagulant Agents

Drug	Site of Action	Route	Plasma Half-life	Excretion	Antidote	Stop before Procedure	Prolongation of PT/aPTT
Unfractionated heparin	Ila/Xa	IV/SC	1.5 hr	Hepatic	Protamine	6 hr	No/Yes
LMWH	Xa	SC	4.5 hr	Renal	Protamine (partial reversal)	12-24 hr	No/No
Streptokinase	Plg	IV	23 min	Hepatic	Antifibrinolytics	3 hr	Yes/Yes
t-PA	Plg	IV	<5 min	Hepatic	Antifibrinolytics	1 hr	Yes/Yes
Warfarin	Vitamin K–dependent factors	Oral	2-4 days	Hepatic	Vitamin K rfVIIa PCCs Plasma	2-4 days	Yes/No
Pentasaccharide	Xa	SC	14-17 hr	Renal	rfVIIa	4 days	No/No
Bivalirudin	Ila	IV	25 min	Hepatic	None	2-3 hr	Yes/Yes
Argatroban	Ila	IV	45 min	Hepatic	None	4-6 hr	*Yes/Yes
Hirudin	Ila	IV	1.5 hr	Renal	PMMA, dialysis	8 hr	*Yes/Yes
APC	Va/VIIIa	IV	2 hr	Hepatic	None	12 hr	No/Yes
Ximelagatran	Ila	Oral	3 hr	Renal	None	24 hr	Yes/Yes

*Argatroban and lepirudin may increase the normal PT 4 to 5 seconds.

APC, activated protein C; HIT, heparin-induced thrombocytopenia; IV, intravenous; LMWH, low molecular weight heparin; PCCs, prothrombin complex concentrates; Plg, plasminogen; PMMA, polymethyl methacrylate; rFVIIa, recombinant factor VIIa; SC, subcutaneous; Ila, thrombin; t-PA, tissue plasminogen activator.

From Roberts HR, Monroe DM, Escobar MA: Current concepts of hemostasis: Implications for therapy. Anesthesiology 100:722-730, 2004, with permission.

Table 56-2 Antiplatelet Agents

Drug	Site of Action	Route	Plasma Half-life	Metabolism	Antidote	Stop before Procedure	Prolongation of PT/aPTT
Aspirin	COX 1-2	Oral	20 min	Hepatic	None	7 days	No/No
Dipyridamole	Adenosine	Oral	40 min	Hepatic	None	24 hr	No/No
Clopidogrel	ADP	Oral	7 hr	Hepatic	None	5 days	No/No
Ticlopidine	ADP	Oral	4 days	Hepatic	None	10 days	No/No
Abciximab	GPIIb-IIIa	IV	30 min	Renal	None	72 hr	No/No
Eptifibatide	GPIIb-IIIa	IV	2.5 hr	Renal	None	24 hr	No/No
Tirofiban	GPIIb-IIIa	IV	2 hr	Renal	Hemodialysis	24 hr	No/No

ADP, adenosine diphosphate; COX, cyclooxygenase diphosphate; IV, intravenous; GP, glycoprotein.

From Roberts HR, Monroe DM, Escobar MA: Current concepts of hemostasis: Implications for therapy. *Anesthesiology* 100:722-730, 2004, with permission.

Table 59-19 Therapies for Desaturation during One-Lung Ventilation

Severe or precipitous desaturation: Resume two-lung ventilation (if possible).

Gradual desaturation:

1. Ensure that delivered FiO_2 is 1.0.
2. Check position of double-lumen tube or blocker with fiberoptic bronchoscopy.
3. Ensure that cardiac output is optimal; decrease volatile anesthetics to < 1 MAC.
4. Apply a recruitment maneuver to the ventilated lung (this will transiently make the hypoxemia worse).
5. Apply PEEP 5 cm H_2O to the ventilated lung (except in patients with emphysema).
6. Apply CPAP 1-2 cm H_2O to the nonventilated lung (apply a recruitment maneuver to this lung immediately before CPAP).
7. Intermittent reinflation of the nonventilated lung
8. Partial ventilation techniques of the nonventilated lung:
 - a. Oxygen insufflation
 - b. High-frequency ventilation
 - c. Lobar collapse (using a bronchial blocker)
9. Mechanical restriction of the blood flow to the nonventilated lung

Table 60-13 Goals for Anesthetic Care of Patients with Aortic Stenosis and Aortic Regurgitation

	Left Ventricular Preload	Heart Rate	Contractile State	Systemic Vascular Resistance	Pulmonary Vascular Regurgitation
Aortic stenosis	↑	↓ (sinus)	Maintain	↑	Maintain
Aortic regurgitation	↑	↑	Maintain	↓	Maintain

From Sukernik MR, Martin DE: Anesthetic management for the surgical treatment of valvular heart diseases. In Hensley FA, Martin DE, Gravlee GP (eds): A Practical Approach to Cardiac Anesthesia, 4th ed. Philadelphia, Lippincott Williams & Wilkins, 2008, pp 316-347.

Table 60-14 ACC/AHA Classification of Chronic Heart Failure

Stage	Description
A—High risk for heart failure	Hypertension, diabetes mellitus, CAD, family history of cardiomyopathy
B—Asymptomatic heart failure	Previous MI, LV dysfunction, valvular heart disease
C—Symptomatic heart failure	Structural heart disease, dyspnea and fatigue, impaired exercise tolerance
D—Refractory end-stage heart failure	Marked symptoms at rest despite maximal medical therapy

ACC/AHA, American College of Cardiology/American Heart Association; CAD, coronary artery disease; LV, left ventricular; MI, myocardial infarction.

From Hunt SA, Baker DW, Chin MH, et al: ACC/AHA guidelines for the evaluation and management of chronic heart failure in the adult: Executive summary. A Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee to Revise the 1995 Guidelines for the Evaluation and Management of Heart Failure): Developed in Collaboration with the International Society for Heart and Lung Transplantation; Endorsed by the Heart Failure Society of America. Circulation 104:2996-3007, 2001.

Problems During OLV	Etiology
Hypoxemia	Intrapulmonary shunt during one-lung ventilation
Sudden severe hypotension	Surgical compression of the heart or great vessels
Sudden changes in ventilating pressure or volume	Movement of endobronchial tube/blocker, air leak
Arrhythmias	Direct mechanical irritation of the heart
Bronchospasm	Direct airway stimulation, increased frequency of reactive airways disease
Massive hemorrhage	Surgical blood loss from great vessels or inflamed pleura
Hypothermia	Heat loss from the open hemithorax

Box 60-6 Pulmonary Vasodilator Agents**1. Phosphodiesterase III Inhibitors**

- Inhibition of myocardial type III phosphodiesterase leads to an increase in myocardial cAMP, which increases the influx of intracellular Ca^{2+} and has a positive inotropic effect
- Unique mechanism of inotropic effects independent of β -receptor stimulation
- Bypass β -adrenergic receptors in patients with preexisting heart failure
- Additive effect with catecholamines
- Pulmonary vasodilation
- Coronary vasodilation

2. B-Type Natriuretic Peptide

- Recombinant human B-type natriuretic peptide
- Site of action: guanylate cyclase receptor of vascular smooth muscle and endothelial cells
- Effect: increased intracellular cGMP and smooth muscle cell relaxation, dose-dependent reduction in PCWP and systemic arterial pressure in patients with heart failure, increased permeability of vascular endothelium, inhibition of the renin-angiotensin-aldosterone axis
- Dose: 2- $\mu\text{g/kg}$ bolus followed by a 0.01- to 0.03- $\mu\text{g/kg/min}$ infusion
- Plasma half-life: 18 minutes

3. Prostaglandin I_2 (PGI_2)

- Endogenous prostaglandin
- Synthesized by the cyclooxygenase arm of the arachidonic acid metabolic pathway
- Potent vasodilator
- Inhibits neutrophil activation
- Stabilizes cell membranes
- Enhances myocardial inotropy by activating adenylate cyclase and increasing intracellular cAMP

4. Nitric Oxide

- Selective pulmonary dilator; less potent than inhaled prostacyclin
- Activates guanylate cyclase
- Improves ventilation-perfusion distribution
- No effect on the cardiac or stroke index
- Complex and expensive technology necessary for safe and effective use
- Prolonged use can cause rebound pulmonary vasoconstriction
- Toxic metabolites: nitrogen dioxide and methemoglobin

5. Prostaglandin E_1 (PGE_1)

- Endogenous prostaglandin
- Synthesized by the cyclooxygenase arm of the arachidonic acid metabolic pathway
- Potent pulmonary and systemic vasodilator
- Cleared from the circulation during its first pass through the lung

cAMP, cyclic adenosine monophosphate; cGMP, cyclic guanosine monophosphate; PCWP, pulmonary capillary wedge pressure.

Table 60-16 Pulsatile Ventricular Assist Devices

Device	Length of Support	Position	Ventricular Support	Drive Mechanism
Abiomed BVS 5000	Short term	Extracorporeal	LV, RV, BV	Pneumatically driven atrial and ventricular chambers
Thoratec VAD	Short to medium term	Extracorporeal	LV, RV, BV	Pneumatically driven sac
HeartMate IP and VE	Long term as a bridge to transplantation or recovery or as destination therapy (HeartMate VE)	Intracorporeal, abdominal (preperitoneal or intraperitoneal)	LV	Pneumatically or electrically driven, flexible, textured polyurethane diaphragm
Novacor	Long term as a bridge to transplantation or recovery	Intracorporeal, abdominal (preperitoneal or intraperitoneal)	LV	Polyurethane pump sac compressed by electrically driven pusher plates

BV, biventricular; BVS, biventricular support; IP, implantable pneumatic; LV, left ventricular; RV, right ventricular; VAD, ventricular assist device; VE, vented electric.

• **Complications related to single lung ventilation:**

Pulmonary Effects	Cardiovascular Effects	CO ₂ Insufflation Effects
Hypoxaemia	Hypotension	Hypotension
Hypercapnia	Hypertension	Bradycardia
Impaired HPV	Arrhythmias	Co ₂ air embolism
Pulmonary Edema	Mediastinal Shift	Subcutaneous Emphysema
Atelectasis		
Pneumonia		

Complications more with Rt Lung as Vascular supply is 10% more compared to the left side.

Table 60-17 Nonpulsatile Ventricular Assist Devices

Device	Flow Type	Length of Support	Position	Ventricular Support	Drive Mechanism
Levitronix CentriMag	Centrifugal	Short term	Extracorporeal	LV, RV, BV	Electric
Tandem Heart	Centrifugal	Short term	Extracorporeal	LV	Electric
Impella	Axial	Short term	Extracorporeal	LV	Electric
Jarvik 2000	Axial	Long term	Intracorporeal	LV	Impeller electrically driven
DeBakey LVAD	Axial	Long term	Intracorporeal	LV	Electric
HeartMate II	Axial	Short term	Intracorporeal	LV	Electric

BV, biventricular; LV, left ventricular; LVAD, left ventricular assist device; RV, right ventricular.

Table 60-20 The RIFLE* Classification Scheme for Acute Renal Failure

	GFR Criteria	Urine Output Criteria
Risk	Plasma creatinine increased 1.5× or GFR decrease >25%	<0.5 mL/ kg/h × 6 hr
Injury	Plasma creatinine increased 2× or GFR decrease >50%	<0.5 mL/kg/hr × 12 hr
Failure	Plasma creatinine increased 3×, acute plasma creatinine ≥350 μmol/L, or acute rise ≥44 μmol/L	<0.3 mL/kg/hr × 24 hr or anuria × 12 hr
Loss	Persistent acute renal failure = complete loss of kidney function >4 wk	
ESKD	End-stage kidney disease (>3 mo)	

*Acronym for risk, injury, failure, loss, and end-stage kidney disease (ESKD).

GFR, glomerular filtration rate.

From Kuitunen A, Vento A, Suojaranta-Ylinen R, Pettilä V: Acute renal failure after cardiac surgery: Evaluation of the RIFLE classification. *Ann Thorac Surg* 81:542-546, 2006.

Box 60-14 Vasodilators Available for the Treatment of Perioperative Hypertension

Adenosine

α_1 -Adrenergic antagonists

α_2 -Adrenergic agonists

Angiotensin-converting enzyme inhibitors (enalaprilat)

Angiotensin II antagonists

Atrial natriuretic peptide (nesiritide)

β_2 -Adrenergic agonists

Dihydropyridine-type calcium channel blockers*

Dopamine agonists

Hydralazine

Nitrovasodilators*

Phosphodiesterase enzyme inhibitors

Prostaglandins

*Intravenous vasoactive therapies in widespread use to treat perioperative hypertension.

From Levy JH: Management of systemic and pulmonary hypertension. *Tex Heart Inst J* 32:467-471, 2005.

Box 60-15 Aims of and Treatment Modalities to Reduce or Prevent the Development of Postoperative Renal Dysfunction

1. Maintain adequate oxygen delivery—by ensuring adequate cardiac output, adequate oxygen-carrying capacity, and proper hemoglobin saturation
2. Suppression of renovascular constriction—by ensuring adequate volume preload and use of infusions of mannitol, calcium entry blockers, and angiotensin-converting enzyme inhibitors
3. Renal vasodilation—by dopaminergic agents, prostaglandins, and atrial natriuretic peptide
4. Maintain renal tubular flow—by loop diuretics and mannitol (which may act to prevent tubular obstruction, which can cause cellular swelling, ischemia, and death)
5. Decrease oxygen demand—by the use of loop diuretics and mild cooling
6. Attenuate ischemic reperfusion injury—as a result of the release of oxygen free radicals and calcium ions

From Sear JW: Kidney dysfunction in the postoperative period. *Br J Anaesth* 95:20-32, 2005.

Box 62-1 Physiologic Changes with Aortic Cross-Clamping* and Therapeutic Interventions**Hemodynamic Changes**

- ↑ Arterial blood pressure above the clamp
- ↓ Arterial blood pressure below the clamp
- ↑ Segmental wall motion abnormalities
- ↑ Left ventricular wall tension
- ↓ Ejection fraction
- ↓ Cardiac output[†]
- ↓ Renal blood flow
- ↑ Pulmonary occlusion pressure
- ↑ Central venous pressure
- ↑ Coronary blood flow

Metabolic Changes

- ↓ Total-body oxygen consumption
- ↓ Total-body carbon dioxide production
- ↑ Mixed venous oxygen saturation
- ↓ Total-body oxygen extraction
- ↑ Epinephrine and norepinephrine
- Respiratory alkalosis[‡]
- Metabolic acidosis

Therapeutic Interventions

- Afterload reduction
 - Sodium nitroprusside
 - Inhaled anesthetics
 - Amrinone
 - Shunts and aorta-to-femoral bypass
- Preload reduction
 - Nitroglycerin
 - Controlled phlebotomy
 - Atrial-to-femoral bypass
- Renal protection
 - Fluid administration
 - Distal aortic perfusion techniques
 - Selective renal artery perfusion
 - Mannitol
 - Drugs to augment renal perfusion
- Other
 - Hypothermia
 - ↓ Minute ventilation
 - Sodium bicarbonate

*These changes are of greater significance with longer duration of cross-clamping and with more proximal cross-clamping.

[†]Cardiac output may increase with thoracic cross-clamping.

[‡]When ventilatory settings are unchanged from preclamp levels.

Box 62-3 Physiologic Changes with Aortic Unclamping* and Therapeutic Interventions**Hemodynamic Changes**

- ↓ Myocardial contractility
- ↓ Arterial blood pressure
- ↑ Pulmonary artery pressure
- ↓ Central venous pressure
- ↓ Venous return
- ↓ Cardiac output

Metabolic Changes

- ↑ Total-body oxygen consumption
- ↑ Lactate
- ↓ Mixed venous oxygen saturation
- ↑ Prostaglandins
- ↑ Activated complement
- ↑ Myocardial depressant factor(s)
- ↓ Temperature
- Metabolic acidosis

Therapeutic Interventions

- ↓ Inhaled anesthetics
- ↓ Vasodilators
- ↑ Fluid administration
- ↑ Vasoconstrictor drugs
- Reapply cross-clamp for severe hypotension
- Consider mannitol
- Consider sodium bicarbonate

*These changes are of greater significance with longer duration of cross-clamping and with more proximal cross-clamping.

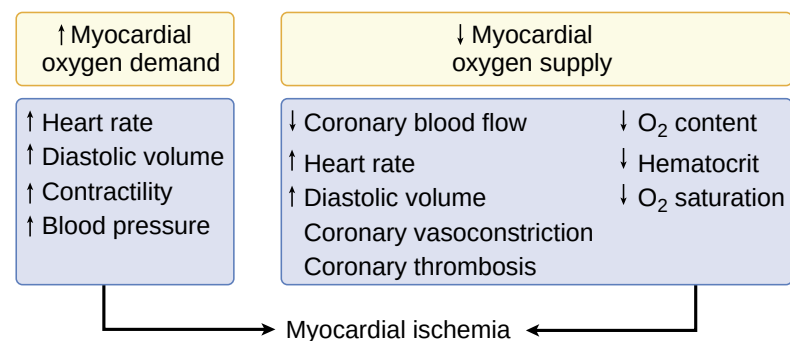


Figure 62-18 Determinants of myocardial oxygen supply and demand that lead to myocardial ischemia. During the perioperative period, virtually every determinant is altered by factors such as fluid shifts, blood loss, pain, catecholamines, altered coagulability, and ventilatory insufficiency. (Adapted from Beattie C, Fleisher LA: Perioperative myocardial ischemia and infarction. *Int Anesthesiol Clin* 30:1-17, 1992.)

Table 63-1 Intracranial Compartments and Techniques for Manipulation of Their Volume

Compartment	Volume Control Methods
Cells (including neurons, glia, tumors, and extravasated blood)	Surgical removal
Fluid (intracellular and extracellular)	Diuretics Steroids (principally tumors)
CSF	Drainage
Arterial blood	Decrease CBF
Venous blood	Improve cerebral venous drainage

CBF, cerebral blood flow; CSF, cerebrospinal fluid.

Table 63-2 Factors That Influence Cerebral Blood Flow*

Pao₂
 Paco₂
 Cerebral metabolic rate
 Arousal/pain
 Seizures
 Temperature
 Anesthetics
 Blood pressure/status of autoregulation
 Vasoactive agents
 Anesthetics
 Pressors
 Inotropes
 Vasodilators
 Blood viscosity
 Neurogenic pathways (intra-axial and extra-axial)

*See Chapter 13 for detailed discussion.

Table 63-4 Methods for Rapid Reduction of Intracranial Pressure and Brain Volume*

Further titration of $Paco_2$ (≥ 25 mm Hg)
 CSF drainage (ventriculostomy, brain needle)
 Diuresis (usually mannitol)
 CMR suppression (barbiturates, propofol)
 MAP reduction (if dysautoregulation)
 Surgical control (i.e., removal of bone flap or lobectomy)

*After review of the checklist in [Table 63-3](#).

CMR, cerebral metabolic rate; CSF, cerebrospinal fluid; MAP, mean arterial pressure.

Table 63-6 Management of an Acute Air Embolic Event

Prevent further air entry
 Notify surgeon (flood or pack surgical field)
 Jugular compression
 Lower the head
 Treat intravascular Air
 Aspirate right heart catheter
 Discontinue N_2O
 Fio_2 : 1.0
 Pressors/inotropes
 Chest compression

Table 63-7 Relative Indications for Intra-arterial Pressure Monitoring

Elevated ICP
 Ischemia or incipient ischemia of neurologic tissue
 Recent SAH
 Recent head injury
 Recent spinal cord injury
 Intended or potential temporary vessel occlusion
 Circulatory instability
 Trauma
 Spinal cord injury (spinal shock)
 Sitting position
 Possible barbiturate coma
 Possibility of induced hypotension
 Possibility of induced hypertension
 Anticipated/potential major blood loss
 Aneurysm clipping
 Arteriovenous malformations
 Vascular tumors
 Tumors involving major venous sinuses
 Craniofacial reconstruction
 Extensive craniostomy procedures
 Anticipated light anesthesia without paralysis
 Brainstem manipulation/compression/dissection
 Anticipated cranial nerve manipulation (especially CN V)
 Advantageous for postoperative intensive care
 Hypervolemic therapy
 Head injury
 Diabetes insipidus
 Incidental cardiac disease

CN, cranial nerve; ICP, intracranial pressure; SAH, subarachnoid hemorrhage.

Table 63-8 World Federation of Neurosurgeons (WFNS) Subarachnoid Hemorrhage Scale

WFNS Grade	GCS Score	Motor Deficit
I	15	Absent
II	14-13	Absent
III	14-13	Present
IV	12-7	Present or absent
V	6-3	Present or absent

GCS, Glasgow Coma Scale.

Table 63-9 Hunt-Hess Classification of Neurologic Status after Subarachnoid Hemorrhage

Grade	Criteria*
I	Asymptomatic, or minimal headache and slight nuchal rigidity
II	Moderate to severe headache, nuchal rigidity, no deficit other than cranial nerve palsy
III	Drowsiness, confusion, or mild focal deficit
IV	Stupor, moderate to severe hemiparesis, possibly early decerebrate rigidity and vegetative disturbances
V	Deep coma, decerebrate rigidity, moribund appearance

*Serious systemic diseases, such as hypertension, diabetes, severe arteriosclerosis, chronic pulmonary disease, and severe vasospasm seen on arteriography, result in placement of the patient in the next less favorable category.

Table 63-10 Glasgow Coma Scale

Eyes open	Never	1
	To pain	2
	To speech	3
	Spontaneously	4
Best verbal responses	None	1
	Garbled/incomprehensible sounds	2
	Inappropriate words	3
	Confused but converses	4
	Oriented	5
Best motor responses	None	1
	Extension (decerebrate rigidity)	2
	Abnormal flexion (decorticate rigidity)	3
	Withdrawal	4
	Localizes pain	5
	Obeys commands	6
Total		3-15

Table 65-7 Management Guidelines for Correction of Anemia of Chronic Renal Disease

Erythropoietin	
Starting dosage	50-150 U/kg/wk IV or SC (once, twice, or three times per week)
Target hemoglobin	11-12 g/dL
Optimal rate of correction	Increase hemoglobin by 1-2 g/dL over 4 wk
Darbepoetin alfa	
Starting dosage	0.45 µg/kg administered as single IV or SC injection once weekly 0.75 µg/kg administered as a single IV or SC injection once every 2 wk
Target hemoglobin	12 g/dL
Optimal rate of correction	Increase hemoglobin by 1-2 g/dL over 4-wk period
Iron Monitor iron stores by TSat and serum ferritin If patient is iron-deficient (TSat <20%; serum ferritin <100 g/L), administer iron, 50-100 mg IV twice per week for 5 wk; if iron indices are still low, repeat the same course If iron indices are normal but hemoglobin is still inadequate, administer IV iron as outlined above; monitor hemoglobin, TSat, and ferritin. Withhold iron therapy when TSat >50% or ferritin >800 ng/mL (>800 g/L)	

IV, intravenously; SC, subcutaneously; TSat, percent transferrin saturation.

Table 63-14 Common Pediatric Neurosurgical Disorders and Anesthetic Considerations

Age Group	Lesion	Pathogenesis	Anesthetic Considerations
Neonates	Intraventricular hemorrhage Depressed skull fracture	Subependymal vascular rupture Forceps injury	Associated problems of prematurity Associated cerebral edema
Infants	Hydrocephalus	Varied	Increased ICP, especially dangerous in shunt-dependent revisions
	Meningocele	Outpouching of meninges through skull defect	Large size creates airway management problems Prone or lateral position Substantial blood loss Repair may increase ICP
	Encephalocele	Outpouching of meninges through skull defect with brain tissue enclosed	
	Myelomeningocele	Protrusion of spinal meninges and roots through spina bifida	Prone or lateral position Substantial blood loss Respiratory restriction after covering large defects Brainstem compression with head flexion
	Arnold-Chiari malformation	Impaction of posterior fossa contents into foramen magnum With or without hydrocephalus	Increased ICP Latex allergy
	Craniosynostosis	With or without myelomeningocele	Postoperative respiratory depression
		Premature cranial suture fusion	Substantial blood loss Air embolism Supine or prone position
	Craniofacial dysostosis	Developmental abnormality	Lengthy procedures Substantial blood loss Brain retraction Air embolism Endotracheal tube damage
	Vascular malformations	Varied	Congestive heart failure Large blood loss Elective hypotension
	Subdural hematoma/effusion	Trauma	Associated injuries
Older children	Posterior fossa tumors	Ependymoma Astrocytoma	Malnutrition/dehydration Hydrocephalus Increased ICP Prone or sitting position Air embolism Brainstem compression Postoperative cranial nerve dysfunction or brainstem swelling or compression

ICP, intracranial pressure.

Table 64-2 Waist Circumference and Risk

Waist Circumference	Body Mass Index (kg/m ²)		
	Normal Weight	Overweight	Obese Class 1
<102 cm (♂) <88 cm (♀)	Least risk	Increased risk	High risk
≥102 cm (♂) ≥88 cm (♀)	Increased risk	High risk	Very high risk

Table 64-1 Levels of Risk Associated with Increasing Body Mass Index

Classification	BMI (kg/m ²)	Risk of Health Problems Developing
Underweight	<18.5	Increased
Normal weight	18.5-24.9	Least
Overweight	25.0-29.9	Increased
Obese		
Class 1	30.0-34.9	High
Class 2	35.0-39.9	Very high
Class 3	40.0-49.9	Extremely high
Superobese	≥50	Exceedingly high

Table 65-1 Pain Conduction Pathways and Spinal Segment Projection of Pain of the Genitourinary System

Organ	Sympathetics, Spinal Segments	Parasympathetics	Spinal Levels of Pain Conduction
Kidney	T8-L1	CN X (vagus)	T10-L1
Ureter	T10-L2	S2-4	T10-L2
Bladder	T11-L2	S2-4	T11-L2 (dome), S2-4 (neck)
Prostate	T11-L2	S2-4	T11-L2, S2-4
Penis	L1 and L2	S2-4	S2-4
Scrotum	NS	NS	S2-4
Testes	T10-L2	NS	T10-L1

NS, not significant for nociceptive function.

Table 65-4 Conditions Affecting Blood Urea Nitrogen (BUN) Independently of Glomerular Filtration Rate**Increased BUN**

Reduced effective circulating blood volume (prerenal azotemia)
 Catabolic states (gastrointestinal bleeding, corticosteroid use)
 High-protein diets
 Tetracycline

Decreased BUN

Liver disease
 Malnutrition
 Sickle cell anemia
 SIADH

SIADH, syndrome of inappropriate secretion of antidiuretic hormone.

Table 65-5 Conditions Affecting Serum Creatinine Independently of Glomerular Filtration Rate

Condition	Mechanism
Conditions Causing Elevation	
Ketoacidosis	Noncreatinine chromogen
Cephalothin, cefoxitin	Noncreatinine chromogen
Flucytosine	Noncreatinine chromogen
Other drugs—aspirin, cimetidine, probenecid, trimethoprim	Inhibition of tubular creatinine secretion
Conditions Causing Decrease	
Advanced age	Physiologic decrease in muscle mass
Cachexia	Pathologic decrease in muscle mass
Liver disease	Decreased hepatic creatine synthesis and cachexia

Table 65-8 Drugs Used or Encountered in Anesthesia Practice That Significantly Depend on Renal Elimination

Completely Dependent	Partially Dependent
Digoxin, inotropes (used frequently; monitoring of blood levels indicated in chronic renal failure)	IV anesthetics—barbiturates
Others—aminoglycosides, vancomycin, cephalosporins, and penicillins	Muscle relaxants—pancuronium Anticholinergics—atropine, glycopyrrolate Cholinesterase inhibitors—neostigmine, edrophonium Others—milrinone, hydralazine, cycloserine, sulfonamides, and chlorpropamide

Table 65-12 Perioperative Causes of Oliguria

Prerenal
Hypovolemia
Dehydration
Low cardiac output states
Aortic/renal artery clamping
Thromboembolic phenomena
Hemorrhage
Transfusion reaction
Renal
Ischemic injury from shock (cardiogenic, septic, hemorrhagic)
Nephrotoxins
Antibiotics
Chemotherapeutic agents
Radiocontrast dyes
Free hemoglobin and myoglobin
Cellular debris
Acute interstitial nephritis
Hypersensitivity reactions
Acute glomerulonephritis
Postrenal
Calculi
Tumors
Clot retention
Surgical ligation
Edema

From Pataño O: Acute renal failure and perioperative oliguria. In Malhotra V (ed): Anesthesia for Renal and Genitourinary Surgery. New York, McGraw-Hill, 1996, p 38.

Table 65-13 Urinary Composition of Oliguria

	Physiologic Oliguria	Prerenal Failure	Acute Tubular Necrosis
Urinary sodium	<10 mEq/L	<25 mEq/L	>35 mEq/L
Urinary specific gravity	>1.024	>1.015	1.010-1.015
Urinary osmolality	>700 mOsm/kg	>500 mOsm/kg	<350 mOsm/kg
Urinary/plasma osmolality	>2.5 : 1	>1.8 : 1	≤1.1 : 1
Urinary/plasma urea	>100 : 1	>20 : 1	3 : 1, rarely >10 : 1
Urinary/plasma creatinine	>60 : 1	>30 : 1, rarely <10 : 1	<10 : 1
Fractional sodium excretion	<1	>1	>0.5

Table 65-16 Changes on Immersion during Lithotripsy

Cardiovascular	Increased Increased Increased	Central blood volume Central venous pressure Pulmonary artery pressure
Respiratory	Increased Decreased Decreased Decreased Increased	Pulmonary blood flow Vital capacity Functional residual capacity Tidal volume Respiratory rate

Table 65-17 Anesthetic Implications of Radical Nephrectomy for Tumors

85%-90% are for renal cell cancer
5%-10% extension to the IVC and right atrium
Large-bore IV access, A-line, IJV line (preferably on left side if IVC is involved)
Paraneoplastic syndrome
Hypercalcemia, eosinophilia; increased prolactin, erythropoietin, and glucocorticoids
Men > women
Chronic smoking history usually associated
CAD, COPD
Renal failure

CAD, coronary artery disease; COPD, chronic obstructive pulmonary disease; IJV, internal jugular vein; IVC, inferior vena cava.

Table 65-18 Anesthetic Implications of Radical Prostatectomy

Disease of the elderly
CAD, COPD, and renal dysfunction
Significant blood loss
Wide-bore IV access and invasive monitoring
Acute normovolemic hemodilution versus autologous blood donation
Hyperextended position
Nerve injuries, soft tissue injury, joint dislocations
Venous air embolism
Anesthesia
Benefits of regional anesthesia versus general anesthesia debated
Not known to influence mortality
Epidural anesthesia with spontaneous ventilation decreases blood loss
General or combined anesthesia with IPPV increases blood loss

CAD, coronary artery disease; COPD, chronic obstructive pulmonary disease; IPPV, intermittent positive-pressure ventilation.

Table 66-1 Modified Child-Turcotte-Pugh Scoring System

Parameters	Modified Child-Turcotte-Pugh Score*		
	1	2	3
Albumin (g/dL)	>3.5	1.8-3.5	<2.8
Prothrombin time			
Seconds prolonged	<4	4-6	>6
International normalized ratio	<1.7	1.7-2.3	>2.3
Bilirubin (mg/dL) [†]	<2	2-3	>3
Ascites	Absent	Slight-moderate	Tense
Encephalopathy	None	Grade I-II	Grade III-IV

*Class A = 5.6 points, B = 7 to 9 points, and C = 10 to 15 points.

[†]For cholestatic diseases (e.g., primarily biliary cirrhosis), the bilirubin level is disproportionate to the impairment in hepatic function and an allowance should be made. For these conditions, assign 1 point for a bilirubin level less than 4 mg/dL, 2 points for a bilirubin level of 4 to 10 mg/dL, and 3 points for a bilirubin level over 10 mg/dL.

From Pugh RNH, Murray-Lyon IM, Dawson JL, et al: Transection of oesophagus for bleeding of oesophageal varices. Br J Surg 60:646-649, 1973.

Table 66-2 Etiology of Postoperative Jaundice by Time Course

Immediate Postoperative Jaundice (<3 wk)
Hemolysis
Anesthesia
Hypotension/hypovolemia
Drugs
Infection/sepsis
Bleeding/resorption of hematoma
Bile duct ligation/stricture/surgical injury
Hepatic artery ligation
Retained common duct stone
Postoperative pancreatitis/cholecystitis
Acute viral hepatitis
Gilbert's syndrome/Dubin-Johnson syndrome
Inflammatory bowel syndrome
Heart failure
Pulmonary postoperative jaundice
Blood transfusion
Delayed Postoperative Jaundice (>3 wk)
Drugs
Blood transfusion
Post-intestinal bypass status
Total parenteral nutrition

Table 66-3 Medications Associated with Hemolysis

Acetaminophen
 Cephalosporins
 Hydralazine
 Ibuprofen and other NSAIDs (e.g., diclofenac, tolmetin)
 Insulin
 Intravenous contrast media
 Penicillin and all derivatives (e.g., ampicillin, methicillin)
 Procainamide
 Ranitidine
 Sodium thiopental

Table 67-1 Brainstem Reflexes That Should Be Absent in Brain Death

Pupillary response to light
Corneal reflex
Oculocephalic reflex (doll's eye response)
Oculovestibular reflex (caloric response)
Gag and cough reflex
Facial motor response

Table 67-2 Immunosuppressive Drugs Used in Solid Organ Transplantation and Their Side Effects

Mechanism of Action	Side Effects
Inhibition of T-cell interaction	
Prednisolone	As with all steroids: osteoporosis, diabetes mellitus, glaucoma, infections
Muromonab-CD3 (Orthoclone OKT3)	Fever, lymphoproliferative disease, pulmonary edema, anaphylactic reaction, neoplasia
15-Deoxyspergualin	Bone marrow suppression, gastrointestinal syndromes, paresthesia
Inhibition of adhesion molecules	
Rabbit/horse antithymocyte globulin	Fever, nausea, anaphylactic reaction, higher incidence of cytomegalovirus and Epstein-Barr virus infection
Antilymphocyte globulin	Fever
Enlimomab	Fever, hypertension, chills, nausea, vomiting
OKT4A	Unknown
Inhibition of cytokine synthesis	
Cyclosporine	Nephrotoxicity, hepatotoxicity, neurotoxicity, hypertension, hyperlipidemia, hirsutism, tremor, gingival hyperplasia, diabetes
Tacrolimus (FK506)	Nephrotoxicity, neurotoxicity, hypertension, hyperlipidemia, hyperglycemia
Sirolimus (rapamycin)	Hyperlipidemia, myelosuppression, infections
SDZ-RAD (everolimus)	Hyperlipidemia, myelosuppression, infections
Inolimomab	Headache, leukopenia, thrombocytopenia
Basiliximab	No major adverse effects
Daclizumab	No major adverse effects
Inhibition of DNA synthesis	
Azathioprine	Myelosuppression, hepatotoxicity, development of malignancy
Mycophenolate mofetil	Leukopenia, gastrointestinal syndromes

Table 68-1 Management of Patients with Cardiac Disease for Laparoscopy**Preoperative Evaluation: Echocardiography**

If left ventricular ejection fraction < 30%:

Intraoperative monitoring

Intra-arterial line

Pulmonary artery catheter?

Transesophageal echocardiography

Continuous ST-segment analysis?

Gasless laparoscopy?

Laparotomy?

Intraoperative Management

Slow insufflation

Low intra-abdominal pressure

Hemodynamic optimization before pneumoperitoneum (preload augmentation)

Patient tilt after insufflation

Anesthesia: remifentanyl, vasodilating anesthetic and drugs (nicardipine, nitroglycerin), cardiotonic agents

Experienced surgeon

Postoperative Care

Slow recovery from anesthesia (benefit of clonidine)

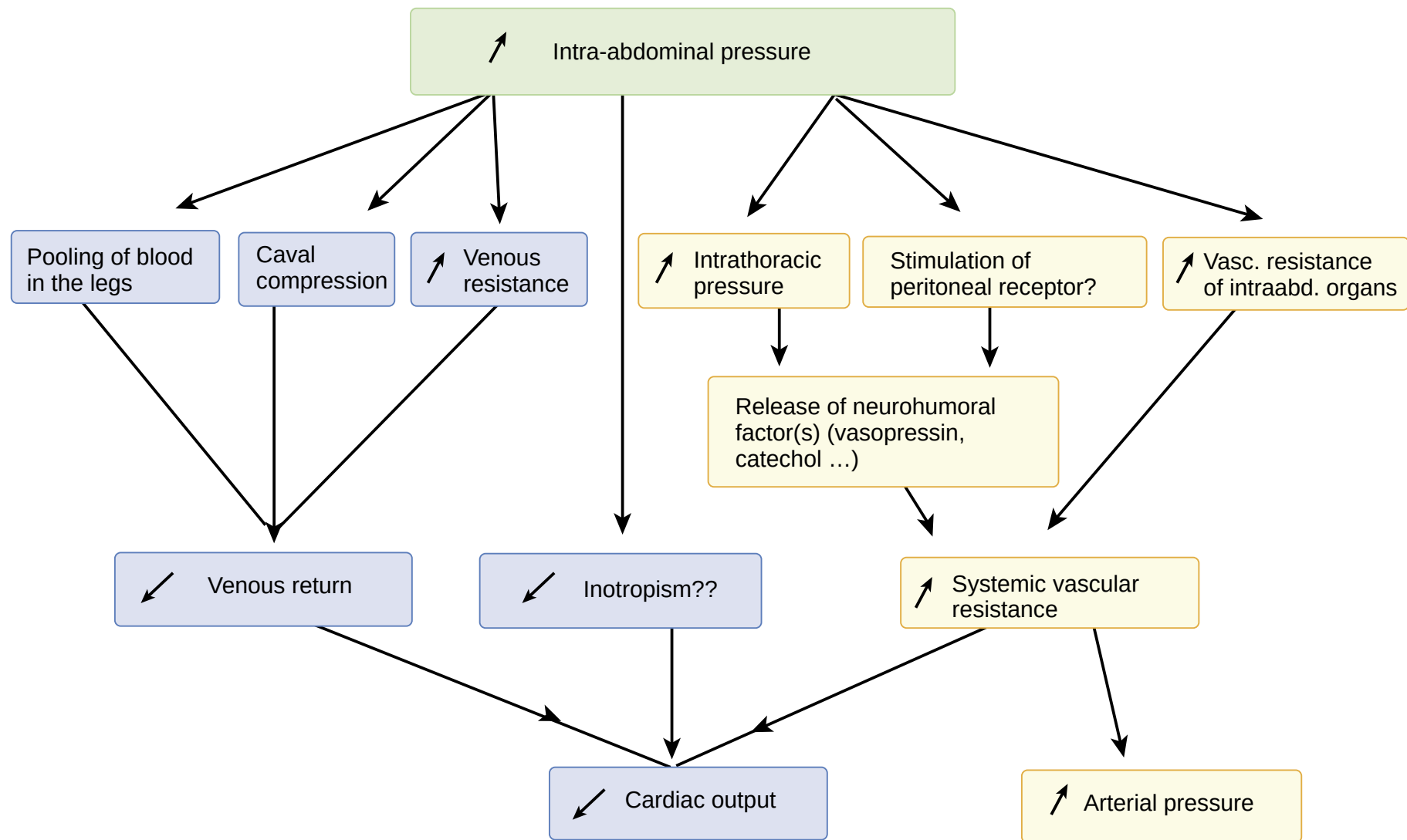


Figure 68-4 Schematic representation of the different mechanisms leading to decreased cardiac output during pneumoperitoneum for laparoscopy.

Table 69-1 Cardiovascular Changes in Pregnancy

Parameter	Change	Amount (%)
Heart rate	Increased	20-30
Stroke volume	Increased	20-50
Cardiac output	Increased	30-50
Contractility	Variable	±10
Central venous pressure	Unchanged	
Pulmonary capillary wedge pressure	Unchanged	
Systemic vascular resistance	Decreased	20
Systemic blood pressure	Slight decrease	Midtrimester 10-15 mm Hg, then rises
Pulmonary vascular resistance	Decreased	30
Pulmonary artery pressure	Slight decrease	

From Birnback DJ, Gatt SP, Datta S (eds): Textbook of Obstetric Anesthesia. New York, Churchill Livingstone, 2000, p 34.

Table 69-3 Coagulation Factors in Pregnancy

Factor	Change
II	Unchanged
VII	Increased +++
VIII, IX, X, XII	Increased
XI	Reduced
Fibrinogen	Increased +++
Platelets	Stable

Table 69-2 Effect of Pregnancy on Cardiovascular Investigations

Investigation	Findings
Chest radiography	Apparent cardiomegaly Enlarged left atrium (lateral views) Increased vascular markings Straightening of left-sided heart border Postpartum pleural effusion
Electrocardiography	Right-axis deviation Right bundle branch block ST-segment depression of 1 mm on left precordial leads Q waves in lead III T-wave inversion in leads III, V ₂ , and V ₃ Small decrease in PR and QT interval (heart rate dependent) Rotation ± 15 degrees (QRS axis)
Echocardiography	Trivial tricuspid regurgitation (up to 43%-93% at term) Pulmonary regurgitation (up to 94% at term) Increased left atrial size by 12%-14% Increased left ventricle end-diastolic dimensions by 6%-10% Inconsistent increase in left ventricle thickness Mitral regurgitation (28% at term) Pericardial effusion (40% postpartum)

Table 69-4 Values for Renal Function

Parameter	Pregnant	Nonpregnant
Creatinine clearance	140-160 mL/min	90-110 mL/min
Urea	2.0-4.5 mmol/L	6-7 mmol/L
Creatinine	25-75 µmol/L	100 µmol/L
Uric acid	0.2 mmol/L	0.35 mmol/L
pH	7.44	7.40
Bicarbonate	18-22 mmol/L	23-26 mmol/L

Table 69-8 Factors That Differentiate Mild from Severe Preeclampsia*

	Mild	Severe
Systolic arterial pressure	<160 mm Hg	≥160 mm Hg
Diastolic arterial pressure	<110 mm Hg	≥110 mm Hg
Urinary protein	<5 g/24 hr Dipstick + or 2+	≥5 g/24 hr Dipstick 3+ or 4+
Urine output	>500 mL/24 hr	≤500 mL/24 hr
Headache	No	Yes
Visual disturbances	No	Yes
Epigastric pain	No	Yes
Right upper quadrant abdominal pain	No	Yes
Pulmonary edema	No	Yes
Cyanosis	No	Yes
HELLP syndrome	No	Yes
Platelet count	>100,000/mm ³	<100,000/mm ³

HELLP, hemolysis, elevated liver enzymes, and low platelet count.

*Not all features need be present in the same patient.

From Birnback DJ, Gatt SP, Datta S (eds): Textbook of Obstetric Anesthesia. New York, Churchill Livingstone, 2000, p 543.

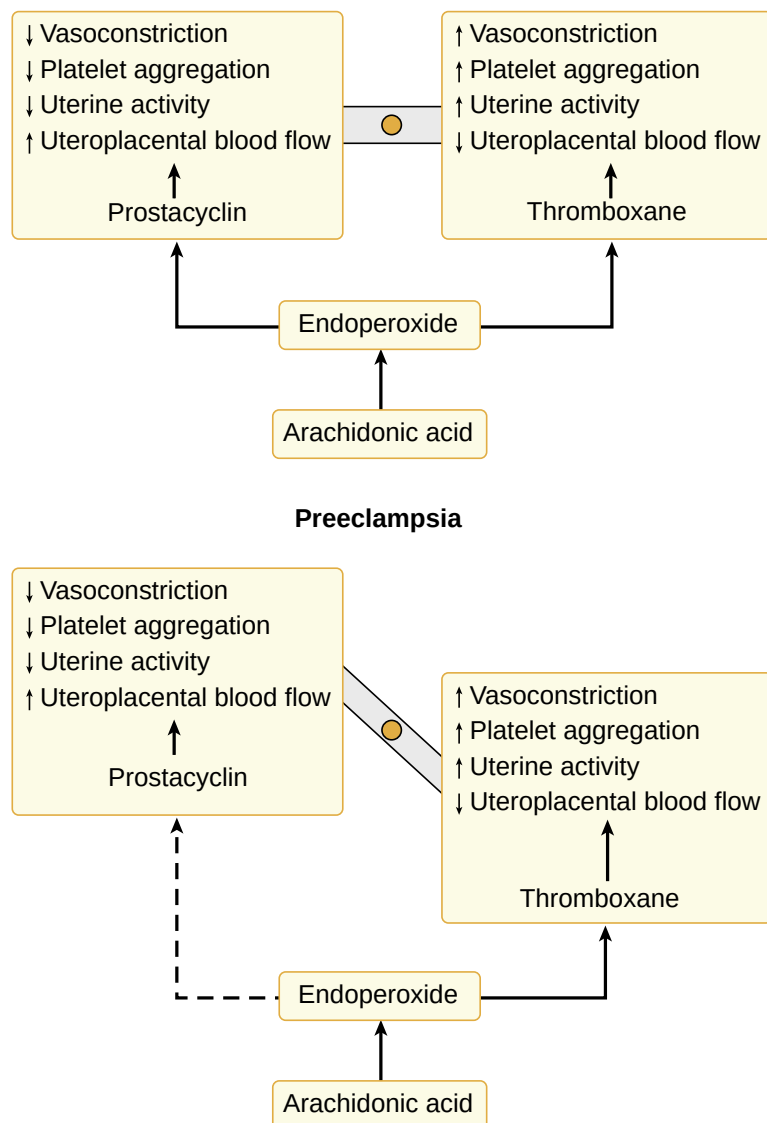


Figure 69-13 Comparison of the normal equilibrium and actions of thromboxane and prostacyclin in normal pregnancy with the dysequilibrium associated with preeclampsia. (Redrawn from Walsh SW: Preeclampsia: An imbalance in placental prostacyclin and thromboxane production. Am J Obstet Gynecol 152:335, 1985.)

Table 69-9 Comparison of Properties of Hydralazine and Labetalol for Treatment of Hypertension

Drug	Hydralazine	Labetalol
Mode of action	Vasodilator	α - and β -blocker (1 : 3)
Speed of onset	Gradual	Quick
Dose	5-10 mg IV slowly	10-20 mg IV slowly
Interval	Repeat after 20 min	Titrate to effect
Infusion rate	2 mg/hr up to 20 mg/hr	20 mg/hr to maximum of 160 mg/hr
Effect on heart rate	Compensatory tachycardia	No effect

Table 70-1 Gurd's Diagnosis of Fat Embolism Syndrome

Major Features (at least one)

Respiratory insufficiency
Cerebral involvement
Petechial rash

Minor Features (at least four)

Pyrexia
Tachycardia
Retinal changes
Jaundice
Renal changes

Laboratory Features

Fat microglobulinemia (required)
Anemia
Thrombocytopenia
High erythrocyte sedimentation rate

From Gurd AR, Wilson RI: The fat embolism syndrome. J Bone Joint Surg Br 56:408-416, 1974.

Table 70-2 Schonfeld Fat Embolism Syndrome Index*

Sign	Score
Petechial rash	5
Diffuse alveolar infiltrates	4
Hypoxemia $P_{aO_2} < 70$ mm Hg, F_{iO_2} 100%	3
Confusion	1
Fever $> 38^\circ\text{C}$ ($> 100.4^\circ\text{F}$)	1
Heart rate > 120 beats/min	1
Respiratory rate > 30	1

*Score > 5 required for diagnosis of fat embolism syndrome.

From Schonfeld SA, Ploysongsang Y, DiLisio R, et al: Fat embolism prophylaxis with corticosteroids: A prospective study in high risk patients. Ann Int Med 99:438-443, 1983.

Table 70-3 Anesthetic Considerations for Patients with Rheumatoid Arthritis

Airway	Limited TMJ movement Narrow glottic opening
Cervical spine	Atlantoaxial instability
Cardiac	Pericarditis Cardiac tamponade
Eyes	Sjögren's syndrome
Gastrointestinal	Gastric ulcers secondary to ASA, steroids
Pulmonary	Diffuse interstitial fibrosis
Renal	Renal insufficiency secondary to NSAIDs

ASA, acetylsalicylic acid; NSAIDs, nonsteroidal anti-inflammatory drugs; TMJ, temporomandibular joint.

Table 70-4 Anesthetic Considerations for Patients with Osteogenesis Imperfecta

Airway	Risk of fractures of the mandible, maxillary surface, and cervical spine
Bleeding	Platelet abnormalities
Cardiac	Congenital and valvular heart disease; cystic degeneration of proximal aorta
Eyes	Exophthalmos—prone positioning
Hyperthermia	Malignant hyperthermia; hydration; possible cooling
Positioning	Risk of repeated fractures
Pulmonary	Kyphoscoliosis—restrictive lung disease
Regional anesthesia	Fractures; intraosseous injections

Table 70-5 Patients Excluded for Single Operation for Bilateral Total Knee Arthroplasty

Age $\geq$ 75 yr
ASA class III
Active ischemic heart disease (positive stress test)
Poor ventricular function (LVEF $<$ 40%)
Oxygen-dependent pulmonary disease
Patients considered at increased risk for morbidity and mortality
IDDM
Renal insufficiency
Pulmonary hypertension
Steroid-dependent asthma
Morbid obesity (BMI $>$ 40)
Chronic liver disease
Cerebrovascular disease

ASA, American Society of Anesthesiologists; BMI, body mass index; IDDM, insulin-dependent diabetes mellitus; LVEF, left ventricular ejection fraction.

Table 70-6 Brachial Plexus Anesthesia

Site of Block	Simulated Brachial Plexus	Response
Interscalene	Roots; trunks	Shoulder to hand
Supraclavicular	Trunks (middle, inferior)	Upper arm to hand
Infraclavicular	Lateral cord	Forearm flexion
		Hand pronation
	Posterior cord	Wrist extension
Axillary	Medial cord	Fingers and thumb
	Median nerve	Forearm pronation
		Wrist flexion
	Ulnar nerve	Finger flexion
		Thumb opposition
	Radial nerve	Wrist extension

Table 70-9 Complications of the Prone Position*

Airway	ETT kinking, dislodgment Upper airway edema
Neck	Hyperextension or hyperflexion Cervical rotation—compromised blood flow to brain
Eyes	Orbital pressure—central retinal artery occlusion, supraorbital nerve Corneal abrasion
Abdomen	Pressure transmitted to epidural veins, increased bleeding
Upper extremity	Brachial plexus stretch—arms out Ulnar nerve compression—arms at the side
Lower extremity	Flexion of hips—occlusion of femoral vein, DVT, kinking of vascular grafts Pressure lateral to fibula—peroneal nerve palsy Pressure on iliac crest—lateral femoral cutaneous nerve

*See Chapter 36.

DVT, deep vein thrombosis; ETT, endotracheal tube.

Table 70-8 Predictors of a Difficult Endotracheal Intubation

Facial dyssymmetry (trauma)
Tracheal deviation
Cervical fractures
Small mouth opening (interincisor gap)
Inability to visualize the faucial pillars and uvula (Mallampati)
Limited neck range of motion
Prominent incisors
Distance between thyroid cartilage and mandible <6 cm
Narrow maxillary arch

Table 59-17 Fluid Management for Pulmonary Resection Surgery

1. Total positive fluid balance in the first 24-hour perioperative period should not exceed 20 mL/kg.
2. For an average adult patient, crystalloid administration should be limited to < 3 L in the first 24 hours.
3. There should be no fluid administration for third space fluid losses during pulmonary resection.
4. Urine output > 0.5 mL/kg/hr is unnecessary.
5. If increased tissue perfusion is needed postoperatively, it is preferable to use invasive monitoring and inotropes rather than to cause fluid overload.

Box 72-5 Goals for Early Resuscitation*

Maintain systolic blood pressure at 80 to 100 mm Hg
 Maintain hematocrit at 25% to 30%
 Maintain the prothrombin time and partial thromboplastin time in normal ranges
 Maintain the platelet count at greater than 50,000 per high-power field
 Maintain normal serum ionized calcium
 Maintain core temperature higher than 35°C
 Maintain function of the pulse oximeter
 Prevent an increase in serum lactate
 Prevent acidosis from worsening
 Achieve adequate anesthesia and analgesia
 *Fluid administration to limit hypoperfusion is balanced against an undesirable increase in blood pressure and thus bleeding.

Box 72-4 Risks Associated with Aggressive Volume Replacement during Early Resuscitation*

Increased blood pressure
 Decreased blood viscosity
 Decreased hematocrit
 Decreased clotting factor concentration
 Greater transfusion requirement
 Disruption of electrolyte balance
 Direct immune suppression
 Premature reperfusion
 Increased risk for hypothermia
 *Most complications of volume resuscitation arise from increased hemorrhage volume or excessive hemodilution.

Box 72-7 Goals of Late Resuscitation*

Maintain systolic blood pressure higher than 100 mm Hg
 Maintain hematocrit above individual transfusion threshold
 Normalize coagulation status
 Normalize electrolyte balance
 Normalize body temperature
 Restore normal urine output
 Maximize cardiac output by invasive or noninvasive measurement
 Reverse systemic acidosis
 Document decrease in lactate to normal range
 *Fluid administration should be continued until adequate systemic perfusion is restored.

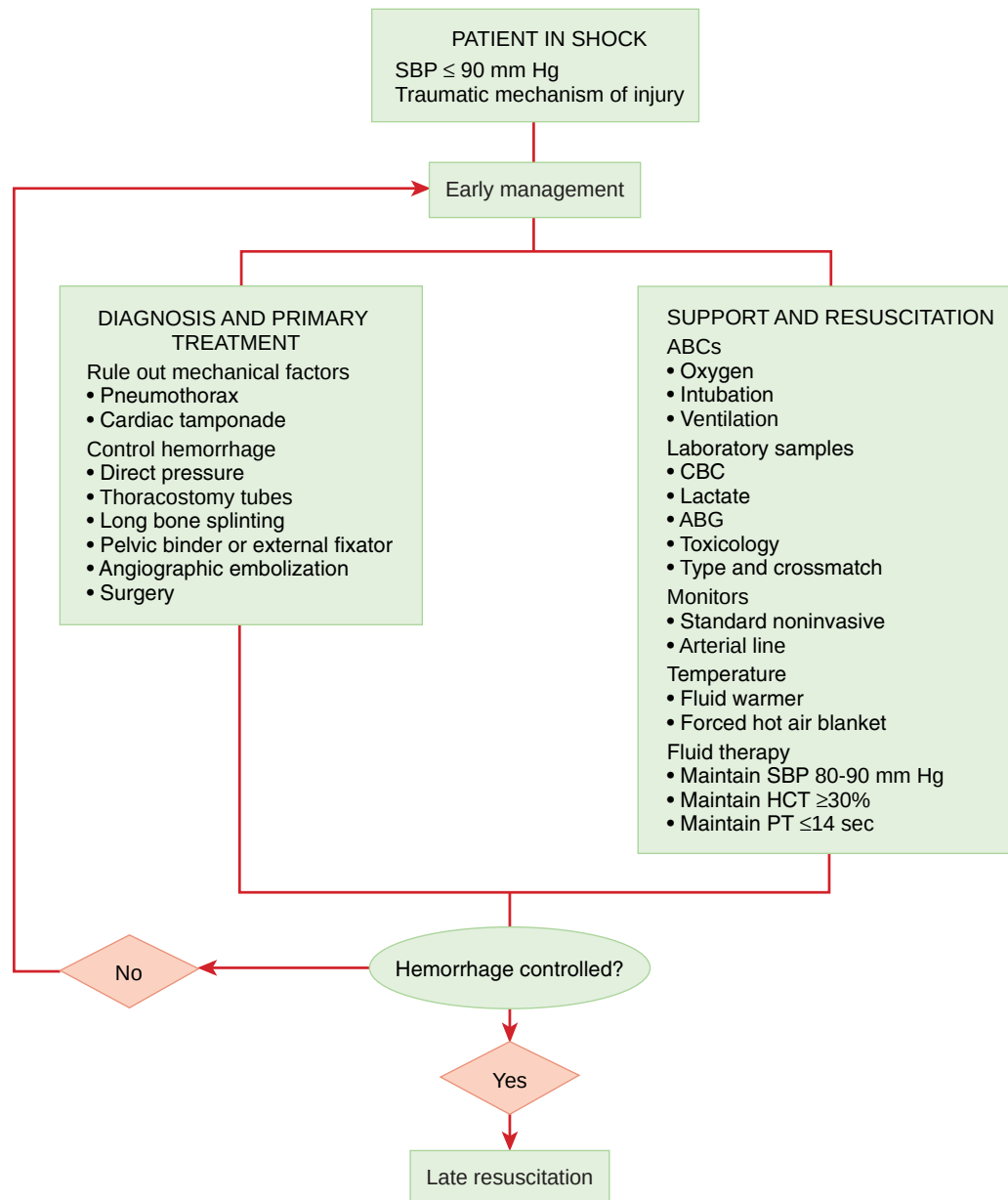


Figure 72-8 Algorithm for the management of early hemorrhagic shock. ABCs, airway, breathing, circulation; ABG, arterial blood gases; CBC, complete blood count; HCT, hematocrit; PT, prothrombin time; SBP, systolic blood pressure.

Table 72-5 Modalities for Assessment of Systemic Perfusion

Technique	Shortcomings
Vital signs	Will not indicate occult hypoperfusion
Urine output	May be confounded by intoxication, diuretic therapy, circadian variation, or renal injury
Systemic acid-base status	Confounded by respiratory status
Lactate clearance	Requires time to obtain laboratory result
Cardiac output	Requires placement of a pulmonary artery catheter or use of noninvasive technology
Mixed venous oxygenation	Difficult to obtain, but a very accurate marker
Gastric tonometry	Requires time to equilibrate, subject to artifact
Tissue oxygenation	Emerging technology, appears beneficial
Stroke volume variation	Emerging technology, requires an arterial line
Acoustic blood flow	Investigational technology, unproven

Box 72-8 Advantages and Disadvantages of Regional Anesthesia for Trauma Patients**Advantages**

Allows continued assessment of mental status
 Increased vascular flow
 Avoidance of airway instrumentation
 Improved postoperative mental status
 Decreased blood loss
 Decreased incidence of deep venous thrombosis
 Improved postoperative analgesia
 Better pulmonary toilet
 Earlier mobilization

Disadvantages

Peripheral nerve function difficult to assess
 Patient refusal common
 Requirement for sedation
 Hemodynamic instability with placement
 Longer time to achieve anesthesia
 Not suitable for multiple body regions
 May wear off before procedure(s) conclude

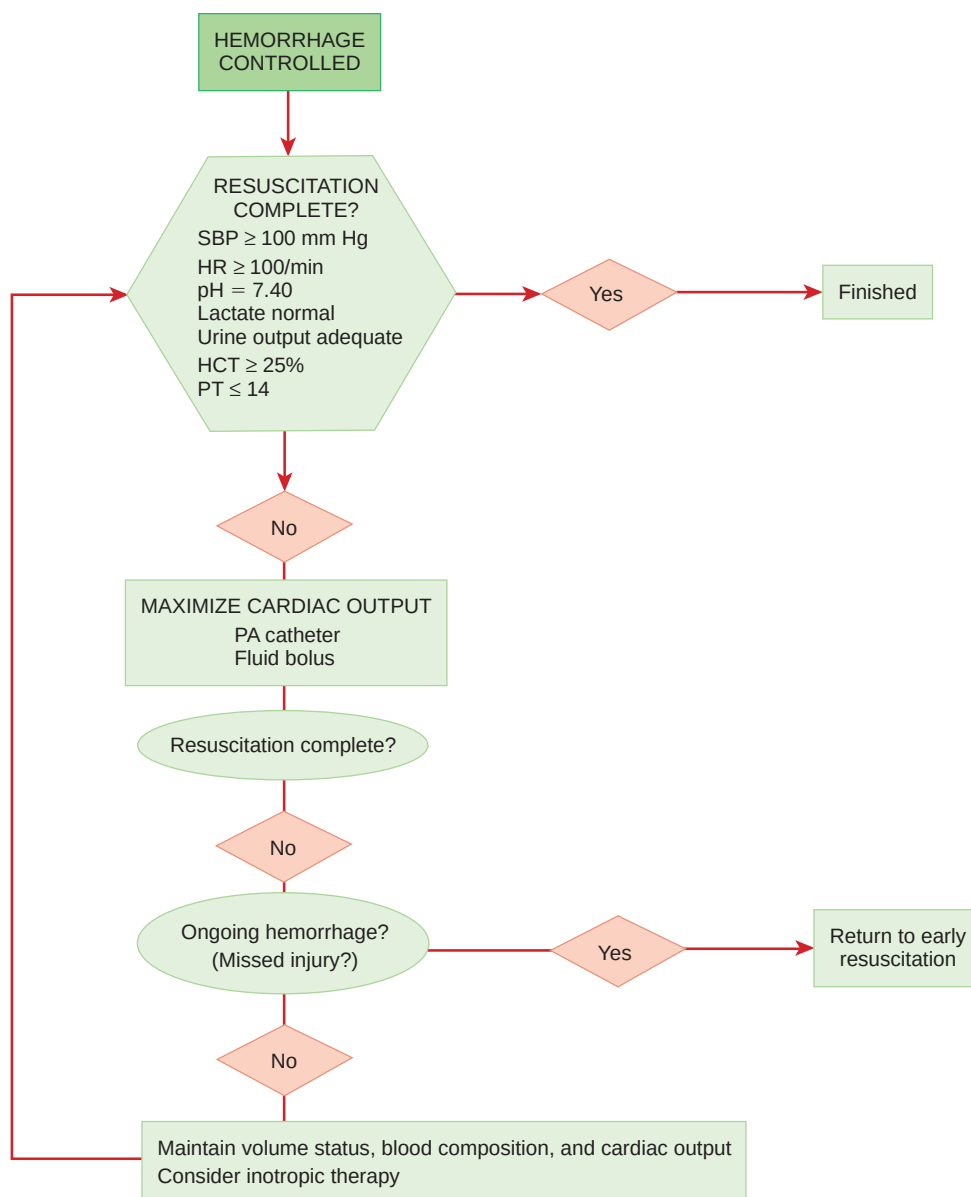


Figure 72-9 Algorithm for the management of late hemorrhagic shock. HCT, hematocrit; HR, heart rate; PT, prothrombin time; PA, pulmonary artery; SBP, systolic blood pressure.

Critical Pathway for Treatment of Cerebral Perfusion Pressure in Patients with Severe Traumatic Brain Injury

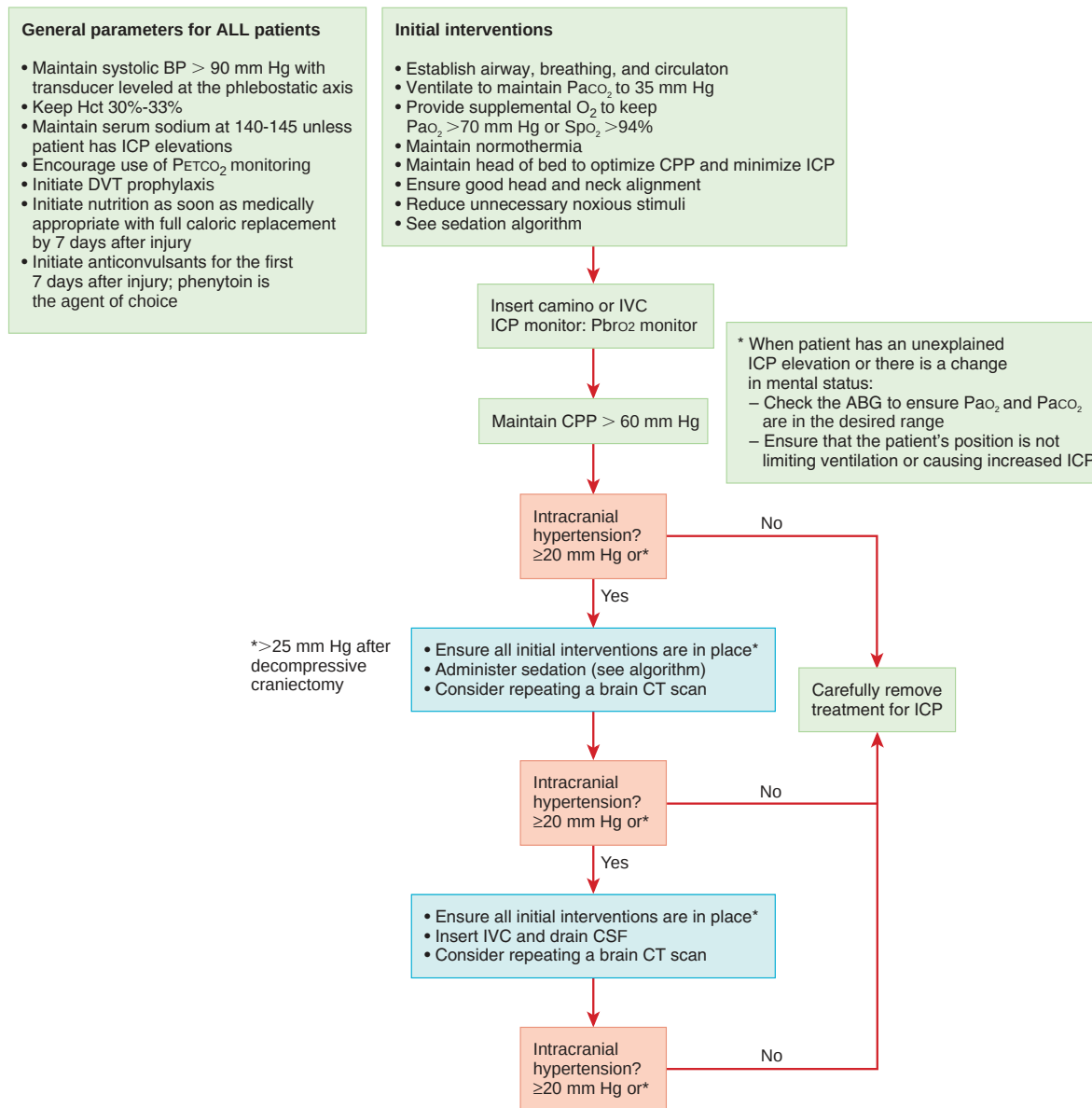


Figure 72-10 Clinical pathway for the management of severe traumatic brain injury. The goal of therapy is to maintain cerebral perfusion pressure above 60 mm Hg by support of the circulation and control of intracranial pressure. Progressively more intensive therapies are added until this goal is achieved. ABG, arterial blood gases; BP, blood pressure; CBF, cerebral blood flow; CPP, cerebral perfusion pressure; CSF, cerebrospinal fluid; CT, computed tomography; DVT, deep venous thrombosis; Hct, hematocrit; ICP, intracranial pressure; IVC, intraventricular catheter.

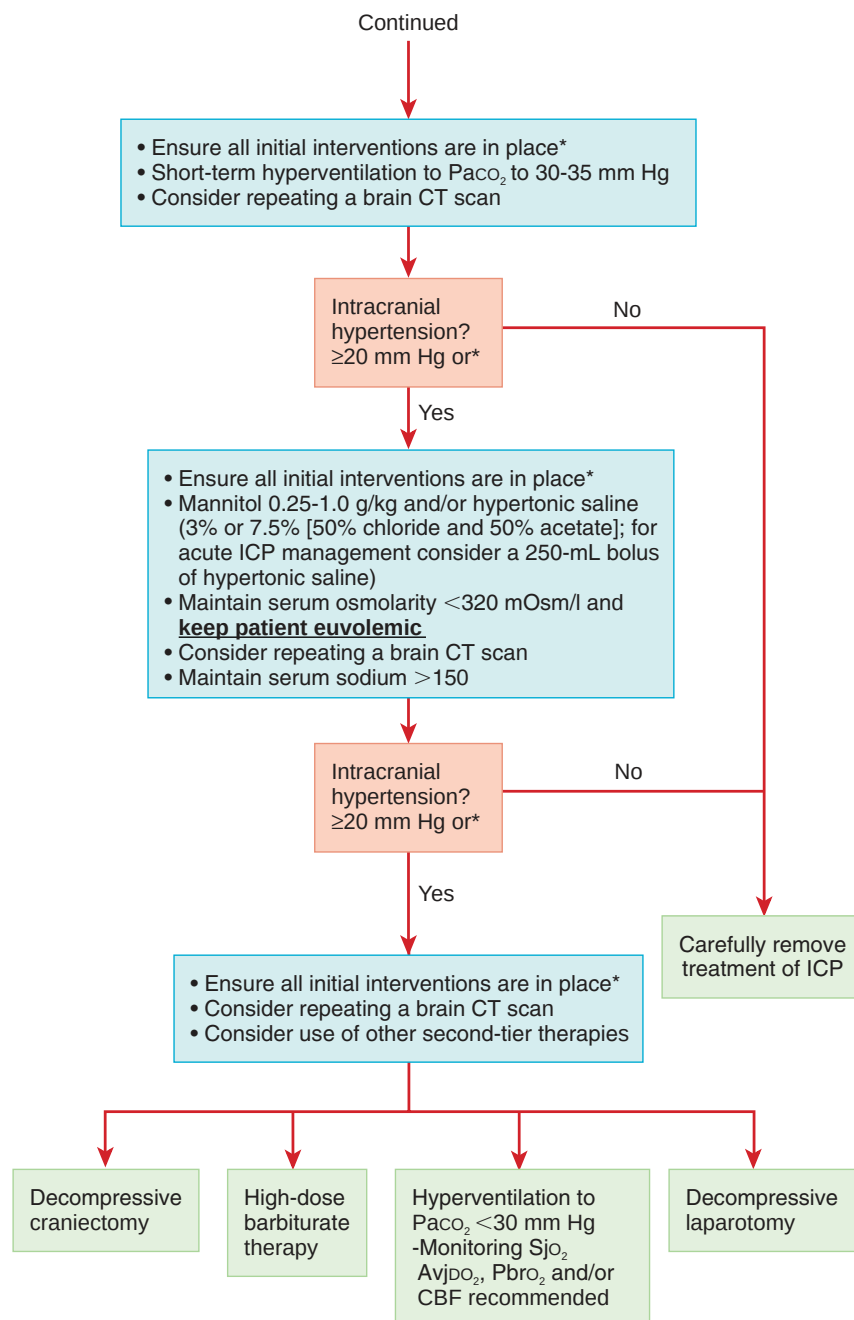


Figure 72-10, cont'd

Table 73-3 Common Nontraumatic Causes of Acute Shortness of Breath in Adults

Exacerbated chronic obstructive pulmonary disease
 Asthma
 Cardiogenic pulmonary edema (congestive heart failure)
 Noncardiogenic pulmonary edema (inhalational, sepsis-related)
 Pneumonia
 Pulmonary embolism
 Foreign body obstruction
 Aspiration

Table 73-4 Differential Diagnosis of Acute Chest Pain in Adults

Acute coronary syndrome (unstable angina, myocardial infarction)
 Pulmonary embolism
 Gastroesophageal reflux
 Peptic ulcer disease
 Gastritis or esophagitis
 Aortic dissection
 Pericarditis
 Pneumonia
 Pneumothorax
 Pleurisy
 Boerhaave's syndrome (esophageal rupture)
 Pancreatitis
 Musculoskeletal pain

Box 72-9 Advantages and Disadvantages of General Anesthesia for Trauma Patients**Advantages**

Speed of onset

Duration—can be maintained as long as needed

Allows multiple procedures for multiple injuries

Greater patient acceptance

Allows positive-pressure ventilation

Disadvantages

Impairment of global neurologic examination

Requirement for airway instrumentation

Hemodynamic management more complex

Increased potential for barotrauma

Table 73-2 On-Scene Triage Criteria for Major Trauma Necessitating Transport to Trauma Center**Mechanism of Injury**

Extrication time >20 minutes

High-speed crash with:

Intrusion into passenger compartment >20 cm

Speed >40 mph

Ejection

Death of another passenger in same car

Rollover

Pedestrian or bicyclist hit by motor vehicle

Motorcycle crash >20 mph or with separation of rider from motorcycle

Falls >3 m (9.8 ft)

Physiologic Criteria

Systolic blood pressure <90 mm Hg

Respiratory rate <10 or >29 breaths/min

Glasgow Coma Scale score <14

Pregnancy

Anatomic Criteria

Flail chest

Two or more proximal long bone fractures

Pelvic fracture

Penetrating injury to head, neck, torso, or extremities proximal to elbow or knee

Open or depressed skull fracture

Amputation proximal to wrist or ankle

Paralysis

Discretion of EMT

Per discretion of EMT, patients can be declared a major trauma patient. In addition, preexisting conditions of the patient can lead to an upgraded status, such as coronary artery disease, congestive heart failure, severe COPD, morbid obesity, or bleeding disorders.

COPD, chronic obstructive pulmonary disease.

From O'Connor RE: Trauma triage: Concepts in prehospital trauma care. Prehospital Emerg Care 10:307-310, 2006.

Table 73-5 Contraindications of Fibrinolytic Therapy in ST-Segment Elevation Myocardial Infarction**Absolute Contraindications**

Any prior intracranial hemorrhage
 Known structural cerebrovascular lesion (e.g., AVM)
 Known malignant intracranial neoplasm (primary or metastatic)
 Ischemic stroke within 3 months EXCEPT acute ischemic stroke within 3 hours
 Suspected aortic dissection
 Active bleeding or bleeding diathesis (excluding menses)
 Significant closed-head trauma or facial trauma within 3 months

Relative Contraindications

History of chronic, severe, poorly controlled hypertension
 Severe uncontrolled hypertension on presentation (SBP > 180 mm Hg or DBP > 110 mm Hg)*
 History of prior ischemic stroke < 3 months, dementia, or known intracranial pathologic process not covered in contraindications
 Traumatic or prolonged (>10 minutes) CPR or major surgery (<3 weeks)
 Recent (within 2 to 4 weeks) internal bleeding
 Noncompressible vascular punctures
 For streptokinase/anistreplase: prior exposure (<5 days ago) or prior allergic reaction to these agents
 Pregnancy
 Active peptic ulcer
 Current use of anticoagulants: the higher the INR, the higher the risk of bleeding

Table 73-6 Classification of Acute Hypertensive Episodes

Hypertensive emergency	Also termed <i>hypertensive crisis</i> ; hypertension with acute end-organ dysfunction
Hypertensive urgency	Hypertensive episode with high risk of imminent end-organ damage that has not yet occurred
Acute hypertensive episode	SBP > 180 mm Hg or DBP > 110 mm Hg without signs of evolving or imminent end-organ dysfunction
Transient hypertension	Related to anxiety or primary complaint

Table 73-7 Causes of Altered Level of Consciousness in Adults: AEIOU-TIPS

Alcohol and Airway (hypoxia)
Epilepsy, Electrolytes, Encephalopathy
Insulin—hypoglycemia
Opioids/Overdose
Urea (metabolic)
Trauma, Tumor
Infection
Psychiatric
Shock, Subarachnoid hemorrhage, Snake bite

Table 73-8 Definitions in Disaster Medicine

Term	Definition	Level of Prehospital Medical Care Possible	Examples
Emergency	Incident that demands urgent action; impact: minutes to hours	Individual medicine	Patient with bicycle accident, stroke, angina
Mass casualty	Large number of casualties in a short period that initially exceed local logistic support capabilities; impact: hours to days	Triage; increasing level of medical treatment dependent on resources	Airplane crash, train collision, mass poisoning
Disaster	Large number of casualties and destroyed infrastructure; exceeds local response capabilities. Supraregional (national) forces are needed to solve the problem; damage and interruption last long-term (weeks to years)	Triage; first aid; significant delay until professional medical help available usually only after temporary infrastructure was set up	Hurricane, earthquake, tsunami, nuclear disaster (e.g., Chernobyl)

Table 73-9 Triage Groups in Mass Casualty Incidents

With the aid of triage tags or colored flags the patients are sorted into four groups:		
Green/minor	Minor injuries with no immediate need of treatment; psychological support needed; patients can walk	Wounds, minor fractures
Yellow/delayed	Urgent treatment, no vital threat	Fractures, joint injuries, amputation, major blood loss, burns
Red/immediate	Immediate vital threat	Respiratory insufficiency, shock, brain trauma, burns with immediate vital threat, abdominal trauma
Black/expectant	Dead or moribund	Respiratory arrest, cardiac arrest, head injuries with dismal prognosis

Table 74-1 Physicochemical Properties of Nerve Agents

Properties*	Tabun (GA)		Sarin (GB)		Soman (GD)		VX		
Molecular weight (daltons)	162.3		140.1		182.18		267.36		
Specific gravity at 25°C	1.073		1.0887		1.022		1.0083		
Boiling point (°C)	246		147		167		300		
Melting point (°C)	−49		−56		−80		−20		
Vapour Pressure (VP) and Volatility (Vol) ^{†‡}	°C	VP (mm Hg)	Vol (mg m ^{−3})	VP (mm Hg)	Vol (mg m ^{−3})	VP (mm Hg)	Vol (mg m ^{−3})	VP (mm Hg)	Vol (mg m ^{−3})
	0	0.004	38	0.52	4279	0.044	470.9	—	—
	10	0.013	119.5	1.07	8494	0.11	1135.5	—	—
	20	0.036	319.8	2.10	16101	0.27	2692.1	0.00044	5.85 [§]
	25	0.07	611.3	2.9	21862	0.40	3921.4	0.0007	10.07
	30	0.094	807.4	3.93	29138	0.61	5881.4	—	—
	40	0.23	1912.4	7.1	60959	—	—	—	—
	50	0.56	4512.0	12.3	83548	2.60	23516.0	—	—

*At room temperature, GB is a comparatively volatile liquid. GD also is significantly volatile. GA is less volatile. GD may be thickened to increase its persistence. VX is relatively nonvolatile and is regarded as presenting little vapor hazard.

†Vapor pressure in mm Hg.

‡Volatility in mg • m^{−3}. Volatility = concentration of saturated vapor at specified temperature. Volatility calculated from PV = nRT.

§Some authorities quote values 0.1 to 1 mg/m^{−3}.

In the equation:

$$\text{Vol} = \frac{\text{VP} \times 101325 \times \text{MW}}{760 \times 8.3143 \times A} = \frac{\text{VP} \times \text{MW} \times 16.035}{A}$$

A = absolute temperature.

From Baker DJ: Anesthesia in extreme environmental conditions, part 2: Chemical and biologic warfare. *In* Grande CG (ed): Textbook of Trauma Anesthesia and Critical Care. St Louis, Mosby-Year Book, 1993, pp 1320-1354.

Table 74-2 Symptoms and Signs of Nerve Agent Poisoning by Type of Cholinergic Receptor and Target Organ

Receptor	Target Organ	Symptoms and Signs
Muscarinic	Iris muscle; ciliary muscle accommodation	Miosis; spasm leading to failure of pain in the eyes; nausea and vomiting; headache
	Conjunctival vessels	Vasodilation and hyperemia
	Nasal glands	Rhinorrhea and hyperemia
	Bronchial glands	Increased secretion
	Bronchial muscle	Bronchoconstriction; tightness in the chest; expiratory wheezing; dyspnea
	Gastrointestinal tract	Anorexia; nausea; vomiting; abdominal cramps; diarrhea; tenesmus; involuntary defecation
	Sweat glands	Increased activity
	Salivary glands	Increased activity
	Lacrimal glands	Lacrimation (not usually marked)
	Heart	Bradycardia; occasionally tachycardia
	Bladder	Frequency; involuntary micturition
Nicotinic	Skeletal muscle	Weakness; fatigue; fasciculations; cramps; flaccid paralysis (early effects on respiratory muscles may produce dyspnea)
	Autonomic ganglia	Pallor; occasional elevation of blood pressure
Muscarinic and nicotinic	Central nervous system	Anxiety; giddiness; restlessness; headache; withdrawal and depression; memory failure; impaired concentration; slurred speech; depression of respiratory and cardiovascular centers; Cheyne-Stokes respiration

Table 74-3 Properties and Toxic Exposure Levels of Phosgene**Properties**Chemical name: carbonyl chloride (COCl_2), chloroformyl chlorideBoiling point: 8.2°C Vapor pressure: 1215 mm Hg at 20°C Vapor density: $3.5 \times \text{air}$ Breakdown: slow aqueous hydrolysis to HCl and CO_2 **Toxic Exposure Levels**

1 ppm chronically—chronic lung disease

>25 ppm/min—acute lung effects

50-100 ppm/min—initial inflammatory response that may be followed by pulmonary edema

>150 ppm/min—clinically significant and life-threatening pulmonary edema

Lethal exposure in humans—800 ppm for 2 min

Table 74-4 Antidotes Used in the Treatment of Cyanide Poisoning

Antidote	Action	Route	Dose	Concurrent Drugs	Duration of Administration	Possible Side Effects
Oxygen	Increases arterial O ₂ content, potentiates activity of other antidotes	Inhalation via mask or ETT	High flow via mask or 100% via ETT	Oxygen used as primary antidote in all cases	≤24 hr	Unlikely—possible in patients with COPD
Amyl nitrite	Methemoglobin formation	Inhalation	Adult: 0.2 mL Pediatric: 0.2 mL; may need repeating	Oxygen (not simultaneously)	30 sec/min	Difficult to achieve effective antidotal levels without cardiovascular collapse
Sodium nitrite	Methemoglobin formation	Intravenous injection	Adult: 300 mg (10 mL of 30 mg/mL 3%) Pediatric: 0.13-0.33 mL/kg of 30 mg/mL (3%) solution (i.e., 4-10 mg/kg body weight)	Adult: sodium thiosulfate 25 mL of 500 mg/mL, (50%) solution and oxygen Pediatric: sodium thiosulfate 1.65 mL/kg body weight of 250 mg/mL (25%) solution (approximately 400 mg/kg body weight) and oxygen	≥5 min, ≤20 min	Methemoglobinemia, vasodilation and cardiovascular collapse
Dicobalt edetate	Binding of cyanide ions by dicobalt edetate and by free cyanide ions	Intravenous injection	Adult: 300 mg (20 mL of 15 mg/mL (15%)) Pediatric: 4-7.5 mg/kg (0.3-0.5 mL/kg of 15 mg/mL (15%))	50 mL dextrose (500 g/L) intravenously immediately after each dose and oxygen	1 min	Urticaria, edema of face and neck, chest pains, dyspnea, hypotension, convulsions
4-DMAP	Methemoglobin formation	Intravenous injection	Adult: 3.25 mg/kg Pediatric: 3.25 mg/kg	Oxygen and sodium thiosulfate	1 min	Methemoglobinemia, vasodilation and cardiovascular collapse, hemolysis, elevated bilirubin and iron (this is unlikely to be relevant to single-dose exposure)
Hydroxocobalamin	Binds cyanide ions	Intravenous injection	Adult: 5-10 g Pediatric: 70 mg/kg	5 g reconstituted in 100 mL 0.9% saline; oxygen	20 min	Reddish discoloration to skin and mucous membranes
Sodium thiosulfate	Sulfur donor for endogenous enzymatic conversion of cyanide to thiocyanate	Intravenous injection	Adult: sodium thiosulfate 25 mL of 500 mg/mL (50%) solution and oxygen Pediatric: sodium thiosulfate 1.65 mL/kg body weight of 250 mg/mL (25%) solution (approximately 400 mg/kg body weight) and oxygen	Oxygen and sodium nitrite or oxygen and DMAP	10 min	Excess administration may cause hyponatremia

COPD, chronic obstructive pulmonary disease; ETT, endotracheal tube.

Table 74-5 Characteristics of Common Toxins

Source	Toxin	Action
Bacteria		
<i>Bacillus anthracis</i>	Anthrax toxin	DNA toxin
<i>Staphylococcus aureus</i>	Staphylococcal enterotoxin B	Enterotoxin
<i>Vibrio cholerae</i>	Cholera toxin	Enterotoxin
<i>Clostridium botulinum</i>	Botulinum toxins	Prejunctional decrease in acetylcholine release
<i>Clostridium perfringens</i>	Perfringens toxin	Necrosis through phospholipase C
<i>Clostridium tetani</i>	Tetanus toxin	Neurotoxin increase in excitability of motor neurons
Protozoa		
<i>Gonyaulax catenella</i>	Saxitoxin	Depolarization block
Fungi		
<i>Fusarium</i> spp.	Tricothecenes	DNA toxin; hemorrhagic syndrome
Plants		
<i>Ricinus communis</i> (castor bean)	Ricin	DNA toxin; hepatorenal failure
Amphibia		
Colombian frog	Batrachotoxin	Irreversible; Na ⁺ channel blockade; depolarization block
Reptilia		
Asian cobra (<i>Naja naja oxiana</i>)	Cobra toxin	Postsynaptic neurotoxin via phospholipase A ₂
Taiwan banded krait (<i>Bungarus multicinctus</i>)	α-Bungarotoxin	Acetylcholine receptor blocker
Fish		
Deadly pufferfish	Tetrodotoxin	Depolarization block

Adapted from Baker DJ: Anesthesia in extreme environmental conditions, Part 2: Chemical and biologic warfare. *In* Grande CG (ed): Textbook of Trauma Anesthesia and Critical Care. St Louis, Mosby-Year Book, 1993, pp 1320-1354.)

Table 74-6 Treatment for Patients Exposed to Biological Warfare Agents

Agent	Infective Dose	Incubation Period	Effects (after Inhalation)	Staff Protection	Specific Treatment	Chemoprophylaxis	Vaccine	Mortality if Untreated
<i>Bacillus anthracis</i>	8000-15,000 spores	1-5 days	Mediastinitis, meningitis, MOF	Isolation, vaccination, universal precautions	Ciprofloxacin 400 mg IV tid; doxycycline 200 mg IV once, then 100 mg IV tid; penicillin 2 MU IV 2 hourly + streptomycin 30 mg/kg IM qd	Ciprofloxacin 500 mg PO bid × 4 wk + vaccinate; doxycycline 100 mg PO bid × 4 wk + vaccinate	Michigan at 0, 2, 4 wk, 6, 12, 18 mo, then annually	Pneumonic, 100%
<i>Yersinia pestis</i>	100-500 organisms	2-3 days	Pneumonia, septicemia, MOF	Isolation, universal precautions	Streptomycin 30 mg/kg IM qd × 10 days; doxycycline 200 mg IV, then 100 mg IV tid × 14 days (chloramphenicol)	Doxycycline 100 mg PO bid × 7 days; tetracycline 500 mg PO qd × 7 days	Greer at 1-3 mo + 3-6 mo	Pneumonic, 100%
Viral hemorrhagic fevers	1-10 organisms	4-21 days	Coagulopathy, edema, MOF	Isolation, HEPA masks, universal precautions	Ribavirin 30 mg/kg IV once, then 15 mg/kg IV qd × 4 days, then 7.5 mg/kg IV tid × 6 days (immunoglobulin)	NA	None	90% (Ebola-Zaire)
Viral encephalitides	10-100 organisms	2-6 days (VEE) 7-14 days (EEE/WEE)	Encephalitis, convulsions, coma, CNS damage	Universal precautions	Supportive, anticonvulsants	NA	Available for VEE, EEE, and WEE	75% (EEE)
<i>Francisella tularensis</i>	10-50 organisms	2-10 days	Pneumonia, pleural effusions	Universal precautions	Streptomycin 30 mg/kg IV qd × 10-14 days; gentamicin 3-5 mg/kg IV od	Doxycycline 100 mg PO bid × 14 days	Live attenuated	35%
Variola organisms	10-100	7-10 days	Rash, secondary pneumonia	Isolation, universal precautions	Cidofovir 5 mg/kg IV once every 2 wk	Vaccinia immunoglobulin	Wyeth	Unvaccinated 30%, vaccinated 3%
<i>Burkholderia mallei</i>	1-10 organisms	10-14 days	Septicemia, pneumonia, lymphadenopathy	Universal precautions	Co-amoxiclav 20 mg/kg IV tid	Tetracycline 500 mg PO qd × 14 days	None	Uncertain: >30% if septicemic
<i>Coxiella burnetii</i>	1-10 organisms	10-14 days	Myalgia, malaise, fever	Barrier nursing	Doxycycline 100 mg PO bid × 5-7 days after exposure × 5 days	Doxycycline 100 mg PO bid for 8-12 days	Q vax	<1%
<i>Brucella</i> spp.	10-100 organisms	5-60 days	Malaise + cough, sacroiliitis, pancytopenia	Barrier nursing	Doxycycline 100 mg PO bid + rifampicin 900 mg tid PO for 6 wk	Doxycycline and rifampicin for 3 wk	None	<5%
<i>Escherichia</i>	10-100 organisms	1-5 days	Vomiting + diarrhea, renal failure	Barrier nursing	Antibiotics not required	NA	None	<5% <i>E. coli</i> O157:H2

CNS, central nervous system; EEE, eastern equine encephalitis; HEPA, high-efficiency particulate air filter; MOF, multiple organ failure; NA, not applicable; VEE, Venezuelan equine encephalitis; WEE, western equine encephalitis. Adapted from White SM: Chemical and biological weapons: Implications for anaesthetic and intensive care. Br J Anaesth 89:306, 2002.

Table 74-8 Effects of Chemical and Biological Warfare Agents on Somatic Systems

System Affected	Agents
Epithelial	Vesicants (e.g., sulfur mustard), smallpox, ricin
Respiratory	
Upper, lower airway	Vesicants, phosgene
Respiratory control system	Nerve agents, ABO
Gaseous exchange	Pulmonary edemagens
Mechanics of breathing	Nerve agents, neurotoxins
Central nervous system	Nerve agents, cyanide, neuropeptides, agents of anesthetic origin (phencyclidines, BZ)
Peripheral nervous system	Nerve agents, neurotoxins (e.g., botulinum, saxitoxin)
Immune system	Vesicants, ABO (provocation of immune responses, inflammatory responses, organ failure)
Cardiovascular	Nerve agents, ABO
Alimentary, renal	Nerve agents, toxins, infectious agents

ABO, agents of biological origin.

Table 78-1 Key Elements of Perioperative Anesthetic Management for Facilitating a Fast-Track Recovery after Elective Ambulatory Surgery

Preoperative Period

- Stabilize coexisting diseases (e.g., hypertension, diabetes) and encourage prehabilitation exercise program and smoking cessation.
- Optimize patient comfort by minimizing anxiety and discomfort.
- Ensure adequate rehydration by replacing fluid deficits.
- Use appropriate prophylactic therapies to prevent postoperative complications (e.g., nausea, vomiting, pain, ileus).

Intraoperative Period

- Utilize anesthetic techniques that optimize surgical conditions, while ensuring a rapid recovery with minimal side effects.
- Administer local analgesia via peripheral nerve blocks, wound infiltration, and/or instillation.
- Apply multimodal analgesia and antiemetic prophylaxis (including use of glucocorticoid steroids).
- Minimize use of nasogastric tubes and surgical drains, and avoid excessive fluid administration.

Postoperative Period

- Allow patients who meet discharge criteria to be fast-tracked (i.e., discharged earlier from recovery units).
- Ensure adequate pain control in the postdischarge period utilizing nonopioid analgesics to minimize need for opioid-containing analgesics.
- Encourage early ambulation and resumption of normal activities of daily living.

Table 74-9 Effects of Toxic Agents on Respiration

Respiratory Component	Effect	Toxic Agent
Central nervous system	Depression of respiratory drive and convulsions leading to apnea	Nerve agents, cyanide, neuropeptides
Peripheral nervous system	Neuromuscular paralysis of respiratory muscles	Nerve agents, neurotoxins
Nasopharynx	May become blocked by excess secretions Prodromal rhinitis and rhinorrhea Sneezing	Lung-damaging agents, nerve agents Vesicants Early symptom of mustard
Larynx	Irritation, laryngeal spasm	Upper respiratory irritant, lung-damaging agents Riot-control agents, particularly CS and CR (tear gas)
Large airways	Blocked by secretions Blocked by inhaled vomitus Sloughing of walls of trachea and main bronchi, produces "pseudodiphtheritic" membrane, serious cause of large airway obstruction, leading to bronchopneumonia and death	Nerve agents (theoretical) Various agents Nerve agents
Small airways	Blocked by secretions Cholinergic innervation affected; bronchospasm (relieved by atropine) Chemical bronchiolitis, followed by serious bronchospasm	Nerve agents Mustard agents
Alveoli	Toxic pulmonary edema	Various agents, especially lung-damaging agents (latency 6-24 hr) Vesicant agents, particularly if inhaled at high ambient temperature*

*Data from Willems JL: Clinical management of mustard gas casualties. *Ann Med Mil Belg* 3(Suppl):1-61:47, 51, 1989.

From Baker DJ, Rustick JM: Anesthesia for casualties of chemical warfare agents. *In* Zaitchuk R, Grande C (eds): U.S. Army Textbook of Military Medicine, part IV, vol I. U.S. Department of the Army, Office of the Surgeon General Borden Institute, Washington, DC, 1995, pp 833-856.

Table 78-2 Benefits of Ambulatory Surgery

- Patient preference, especially children and the elderly
- Lack of dependence on the availability of hospital beds
- Greater flexibility in scheduling operations
- Low morbidity and mortality
- Lower incidence of infection
- Lower incidence of respiratory complications
- Higher volume of patients (greater efficiency)
- Shorter surgical waiting lists
- Lower overall procedural costs
- Less preoperative testing and postoperative medication

Table 78-12 Effects of Intravenous Anesthetic and Cardiovascular Drugs on the Duration of Electroconvulsive Therapy–Induced Seizure Activity

	Increased	No Change	Decreased
Anesthetic Drugs	Etomidate	Methohexital, [*] ketamine, alfentanil, [†] remifentanil [†]	Thiopental, thiamylal, lorazepam, midazolam, propofol
Cardiovascular Drugs	Aminophylline, caffeine	Clonidine, esmolol, labetalol, dexmedetomidine, nifedipine, nicardipine, nitroglycerin, trimethaphan, nitroprusside	Diltiazem, lidocaine

Table 78-5 Use of Anxiolytic-Sedative Drugs for Outpatient Premedication

	Dosage Range	Onset (min)	Key Points
Benzodiazepines			
Midazolam	7.5-15 mg PO	15-30	Large first-pass effect
	5-7 mg IM	15-30	Water soluble, nonirritating
	1-2 mg IV	1-53	Rapid onset, excellent amnesia
Diazepam	5-10 mg PO	45-90	Long-acting metabolites
Temazepam	15-30 mg PO	15-40	Comparable anxiolysis to midazolam
Triazolam	0.125-0.25 mg PO	15-30	Prominent sedation
Lorazepam	1-2 mg PO	45-90	Prolonged amnestic effect
α₂-Adrenergic Agonists			
Clonidine	0.1-0.3 mg PO	45-60	Prolonged sedative effect
Dexmedetomidine	50-70 μg IM	20-60	Bradycardia and hypotension
	50 μg IV	5-30	Reduced anesthetic/analgesic requirements

Table 78-6 Common Factors Associated with Nausea, Vomiting, and Retching during the Perioperative Period

Patient-Related Factors
Age
Gender
Preexisting diseases (e.g., diabetes)
History of motion sickness or postoperative nausea and vomiting
Smoking history
Level of anxiety
Intercurrent illness (e.g., viral infection, pancreatic disease)
Anesthesia-Related Factors
Premedication
Opioid analgesics
Induction and maintenance anesthetics
Reversal (antagonist) drugs
Gastric distention
Inadequate hydration
Residual sympathectomy
Surgery-Related Factors
Operative procedure
Length of surgery
Blood in the gastrointestinal tract
Forcing oral intake
Opioid analgesics
Premature ambulation (postural hypotension)
Pain

Data from Watcha MF, White PF: Postoperative nausea and vomiting: Its etiology, treatment, and prevention. *Anesthesiology* 77:162, 1992.

Table 78-7 Criteria Used to Determine Fast-Track Eligibility after Ambulatory Anesthesia

Criteria	Score
Level of Consciousness	
Awake and oriented	2
Arousable with minimal stimulation	1
Responsive only to tactile stimulation	0
Physical Activity	
Able to move all extremities on command	2
Some weakness in movement of the extremities	1
Unable to voluntarily move the extremities	0
Hemodynamic Stability	
Blood pressure < 15% of the baseline MAP value	2
Blood pressure between 15% and 30% of the baseline MAP value	1
Blood pressure > 30% below the baseline MAP value	0
Respiratory Stability	
Able to breathe deeply	2
Tachypnea with good cough	1
Dyspneic with weak cough	0
Oxygen Saturation Status	
Maintains value > 90% on room air	2
Requires supplemental oxygen (nasal prongs)	1
Saturation < 90% with supplemental oxygen	0
Postoperative Pain Assessment	
None or mild discomfort	2
Moderate to severe pain controlled with IV analgesics	1
Persistent severe pain	0
Postoperative Emetic Symptoms	
None or mild nausea with no active vomiting	2
Transient vomiting or retching	1
Persistent moderate to severe nausea and vomiting	0
Total score	14

A score over 12 with no individual score less than 1 is required for fast-tracking. MAP, mean arterial pressure.

From White PF, Song D: New criteria for fast-tracking after outpatient anesthesia: A comparison with the modified Aldrete's scoring system. *Anesth Analg* 88:1069, 1999.

Table 78-15 Modified Postanesthesia Discharge Scoring (PADS) System

Vital Signs	
2	Within 20% of the preoperative value
1	20%-40% of the preoperative value
0	40% of the preoperative value
Ambulation	
2	Steady gait/no dizziness
1	With assistance
0	No ambulation/dizziness
Nausea and Vomiting	
2	Minimal
1	Moderate
0	Severe
Pain	
2	Minimal
1	Moderate
0	Severe
Surgical Bleeding	
2	Minimal
1	Moderate
0	Severe

Table 80-1 Partial List of Conditions for Which Hyperbaric Oxygen Has Been Used

Gas-bubble disease *Air embolism ^{7,83} *Decompression sickness ^{83,291}
Poisoning *Carbon monoxide ^{7,41,48-50,55} Cyanide ⁴¹ Carbon tetrachloride ^{292,293} Hydrogen sulfide ⁴¹
Infections *Clostridial myonecrosis ^{7,99-101} *Other soft tissue necrotizing infections ^{7,97,98,101} *Refractory chronic osteomyelitis ^{7,90,294} *Intracranial abscess ^{7,295} Mucormycosis ^{296,297}
Acute ischemia *Crush injury ^{7,298} *Compromised skin flaps ^{7,299} Central retinal artery occlusion, central retinal vein occlusion ³⁰⁰
Chronic ischemia *Radiation necrosis (soft tissue, radiation cystitis, and osteoradionecrosis) ⁷ *Ischemic ulcers, including diabetic ulcers ^{7,11,301,302}
Acute hypoxia *Exceptional blood loss anemia (when transfusion delayed or unavailable) ⁷ Support of oxygenation during therapeutic lung lavage ^{103,104}
Thermal injury *Burns ^{7,303-306}
Envenomation Brown recluse spider bite ^{307,308}

*Approved by the Undersea and Hyperbaric Medical Society as an appropriate indication for HBO therapy.⁷

Table 80-3 Units of Pressure

Atmospheres Absolute (ATA)	Absolute Pressure (mm Hg)	Gauge Pressure (mm Hg)	Feet of Sea Water (fsw)	Meters of Sea Water (msw)
1	760	0	0	0
2	1520	760	33	10
3	2280	1520	66	20
6	4560	3800	165	50

Table 81-4 Commonly Used Additives and Recommended Doses in Pediatric Regional Anesthesia

Additive	Recommended Doses	Maximum Doses
Morphine		
Epidural	30 µg/kg	50 µg/kg
Intrathecal	10 µg/kg	20 µg/kg
Fentanyl (epidural)	1-1.5 µg/kg	2.5 µg/kg
Sufentanil (epidural)	0.25-0.5 µg/kg	0.75 µg/kg
Clonidine (epidural or along peripheral nerves)	1-1.5 µg/kg	2 µg/kg
Ketamine* (epidural or occasionally along peripheral nerves)	0.5 mg/kg	1 mg/kg

*Preservative free-ketamine (preferably preservative-free S-ketamine).

Table 81-9 Usual Doses and Infusion Regimens for Epidural Anesthesia in Pediatric Patients

Agent	Initial Dose	Continuous Infusion (Max. Doses)	Repeat Injections
Bupivacaine, levobupivacaine	<i>Solution:</i> 0.25% with 5 µg/mL (1/200,000) epinephrine <i>Dose:</i> <20 kg: 0.75 mL/kg 20-40 kg: 8-10 mL (or 0.1 mL/year/ number of metameres) >40 kg: same as for adults	<4 mo: 0.2 mg/kg/hr (0.15 mL/kg/hr of a 0.125% solution or 0.3 mL/kg/hr of a 0.0625% solution) 4-18 mo: 0.25 mg/kg/hr (0.2 mL/kg/hr of a 0.125% solution or 0.4 mL/kg/hr of a 0.0625% solution) >18 mo: 0.3-0.375 mg/kg/hr (0.3 mL/kg/hr of a 0.125% solution or 0.6 mL/kg/hr of a 0.0625% solution)	0.1 to 0.3 mL/kg every 6-12 hr of a 0.25% or 0.125% solution (according to pain scores)
Ropivacaine	<i>Solution:</i> 0.2% <i>Dose:</i> same regimen in mL/kg as for bupivacaine (see above)	Same age-related infusion rates in mg/kg/hr as for bupivacaine (usual concentration of ropivacaine: 0.1%, 0.15%, or 0.2%) Do not infuse for more than 36 hr in infants < 3 mo	0.1 to 0.3 mL/kg every 6-12 hr of a 0.15% or 0.2% solution (according to pain scores)
Adjuvants	Avoid in infants < 6 mo Fentanyl (1-2 µg/kg) or sufentanil (0.1-0.6 µg/kg) or clonidine (1-2 µg/kg)	<i>Select only one additive:</i> Fentanyl: 1-2 µg/mL Sufentanil: 0.25-0.5 µg/mL Morphine: 10 µg/mL Hydromorphone: 1-3 µg/mL Clonidine 0.3 at 1 µg/mL of solution	Morphine (without preservatives): 25-30 µg/kg every 8 hr

Table 81-10 Usual Doses of Local Anesthetics for Spinal Anesthesia in Neonates and Former Preterm Neonates Younger than 60 Weeks of Preconceptual Age (up to a Weight of 5 kg)

Local Anesthetic	Dose (mg/kg)	Volume (mL/kg)	Duration (min)
Tetracaine 1%	0.4-1.0	0.04-0.1	60-75
Tetracaine 1% with epinephrine	0.4-1.0	0.04-0.1	90-120
Bupivacaine 0.5% isobaric or hyperbaric	0.5-1.0	0.1-0.2	65-75
Levobupivacaine 0.5%	1.0	0.2	75-88
Ropivacaine 0.5%	1.08	0.22	51-68

Table 81-11 Usual Doses of Local Anesthetics for Spinal Anesthesia in Children and Adolescents

Local Anesthetic	Usual Dose(s)
0.5% Isobaric or hyperbaric bupivacaine	5 to 15 kg: 0.4 mg/kg (0.08 mL/kg) >15 kg: 0.3 mg/kg (0.06 mL/kg)
0.5% Isobaric or hyperbaric tetracaine	5 to 15 kg: 0.4 mg/kg (0.08 mL/kg) >15 kg: 0.3 mg/kg (0.06 mL/kg)
0.5% Isobaric levobupivacaine	5 to 15 kg: 0.4 mg/kg (0.08 mL/kg) 15-40 kg: 0.3 mg/kg (0.06 mL/kg) >40 kg: 0.25 mg/kg (0.05 mL/kg)
0.5% Isobaric ropivacaine	0.5 mg/kg (max 20 mg)

Table 82-4 Commonly Used Muscle Relaxants and Reversal Agents in Pediatrics

Drug	Average Intubation Dose (mg/kg)	Category	Approximate Duration
Muscle Relaxants*			
Pancuronium	0.1	Long acting	~45-60 min
Cisatracurium	0.1	Intermediate acting	~30 min
Vecuronium	0.1	Intermediate acting	~30 min
Rocuronium	0.3	Dose related: Short acting	~15-20 min
	0.6	Intermediate acting	~30-45 min
	1.2	Long acting	~45-75 min
Reversal Agents[†]			
Edrophonium	0.3-1.0 mg/kg + atropine, 0.02 mg/kg		
Neostigmine	0.02-0.06 mg/kg + atropine, 0.02 mg/kg		

*The response of preterm and term neonates (who may be more sensitive to the drugs) to muscle relaxants varies greatly from patient to patient. Therefore, all doses should be titrated to response. The recommended intubation doses may be reduced 30% to 50% in the presence of a potent inhaled agent.

[†]The dose of reversal agent given to antagonize nondepolarizing neuromuscular blockade should be determined by the degree of residual neuromuscular blockade (i.e., the dose should be titrated to clinical effect).

Table 82-5 Fasting Guidelines for Pediatric Patients

Age	Fasting Time (hr)*	
	Milk and Solids	Clear Liquids
<6 months	4	2
6-36 months	6	3
>36 months	8	3

*Note that the American Society of Anesthesiologists guideline allows a "light breakfast" (tea and plain toast) 6 hours before anesthesia; however, it is difficult to determine what a "light breakfast" is for a child.

Table 82-8 Laryngeal Mask Size versus Patient Weight*

Laryngeal Mask Size	Patient Weight
1	≤5 kg
1.5	5-10 kg
2	10-20 kg
2.5	20-30 kg
3	30-50 kg
4	50-70 kg
5	70-100 kg
6	>100 kg

*Manufacturer's recommendations (LMA North America).

Table 82-9 Calculation of Maintenance Fluid Requirements for Pediatric Patients

Weight (kg)	Hourly Fluid Requirements (mL)
<10	4 mL/kg
11-20	40 mL + 2 mL/kg for each kilogram above 10
>20	60 mL + 1 mL/kg for each kilogram above 20

Table 83-2 Classification of Congenital Heart Defects

Physiologic Classification	Pulmonary Blood Flow	Comments
Left-to-right shunts VSD ASD PDA AV canal	↑	Volume-overloaded ventricle Development of CHF
Right-to-left shunts Tetralogy of Fallot Pulmonary atresia/VSD Eisenmenger complex	↓	Pressure-overloaded ventricle Cyanotic Hypoxemia
Mixing lesions Transposition/VSD Tricuspid atresia Anomalous venous return Univentricular heart	Generally ↓ but variable $\dot{Q}_p/\dot{Q}_s$	Variable pressure versus volume loaded Usually cyanotic
Obstructive lesions Interrupted aortic arch Critical aortic stenosis Critical pulmonic stenosis Hypoplastic left heart syndrome Coarctation of the aorta Mitral stenosis		Ventricular dysfunction Pressure-overloaded ventricle Ductal dependence
Regurgitant lesions Ebstein's anomaly		Volume-overloaded ventricle
Other secondary causes		Development of CHF

ASD, atrial septal defect; AV canal, atrioventricular canal; CHF, congestive heart failure; PDA, patent ductus arteriosus; $\dot{Q}_p$, pulmonary blood flow; $\dot{Q}_s$, systemic blood flow; VSD, ventricular septal defect.

Table 83-6 Common Perioperative Cardiovascular Medications and Considerations

Cardiac Drug Class	Interactions	Considerations
Angiotensin-converting enzyme inhibitors	Hypotension with induction of general anesthesia	Consider withholding AM dose, or reducing dosage, in hypotensive patients; avoid fixed dose induction regimens with drugs having a profound vagomimetic effect
β -Blockers	Acute withdrawal can precipitate tachycardia and arrhythmias; can potentiate hypotension with volatile anesthesia; can decrease response to inotropic agents	Continue in the perioperative period
Calcium channel blockers	May augment the negative inotropic and chronotropic effects of volatile anesthesia	Continue in the perioperative period
Diuretics	Hypovolemia/hypokalemia; may augment effect of neuromuscular blocking agents	Discontinue preoperatively
Antiarrhythmics	Can be proarrhythmic with inotropes, electrolyte disturbances; high catecholaminergic states; can interact with other antiarrhythmics and precipitate bradycardia	Avoid electrolyte imbalance Avoid drugs that are proarrhythmic Monitor carefully
α_2 -Agonists	Reduces perioperative shivering, ischemia, anesthetic and analgesic requirements	Continue into the perioperative period with appropriate monitoring

Table 83-7 Drugs That May Cause Torsades de Pointes in Patients with Congenital Long QT Syndrome

Drug Category	Drug Name
Antiarrhythmics	Amiodarone
	Procainamide
	Disopyramide
	Ibutilide
	Quinidine
	Sotalol
Antipsychotics	Chlorpromazine
	Haloperidol
	Thioridazine
	Mesoridazine
Antimicrobials	Erythromycin
	Clarithromycin
Miscellaneous	Cisapride
	Arsenic
	Methadone
	Droperidol
	Domperidone
	Dolasetron
	Ondansetron
	Glycopyrrolate

Table 83-8 Monitoring of Organ Systems**Cardiopulmonary System**

Esophageal stethoscope
 Electrocardiogram
 Standard seven-lead system, ST-T wave analysis, esophageal electrocardiographic lead
 Pulse oximetry
 Automated oscillatory blood pressure
 Capnograph
 Ventilator parameters
 Indwelling arterial catheter
 Central venous pressure catheter
 Pulmonary artery catheter
 Transthoracic pressure catheter
 Left or right atrium, pulmonary artery
 Echocardiography with Doppler color flow imaging
 Epicardial or transesophageal

Central Nervous System

Peripheral nerve stimulator
 Processed electroencephalography
 Specialized
 Cerebral blood flow—xenon clearance methodology
 Cerebral metabolism—near-infrared spectroscopy, oxygen consumption measurements
 Transcranial Doppler
 Jugular venous bulb saturations

Temperature

Nasopharyngeal, rectal, esophageal, tympanic

Renal Function

Foley catheter

Table 83-10 Differences between Adult and Pediatric Cardiopulmonary Bypass

Parameter	Adult	Pediatric
Hypothermic temperature	Rarely below 25-30°C	Commonly 15-20°
Use of total circulatory arrest	Rare	Common
Pump prime		
Dilution effects on blood volume	25-33%	150-300%
Additional additives in pediatric primes		Blood, albumin
Perfusion pressures	50-80 mm Hg	20-50 mm Hg
Influence of α - vs. pH-stat management strategy	Minimal at moderate hypothermia	Marked at deep hypothermia
Measured P_{aCO_2} differences	30-45 mm Hg	20-80 mm Hg
Glucose regulation		
Hypoglycemia	Rare—requires significant hepatic injury	Common—reduced hepatic glycogen stores
Hyperglycemia	Frequent—generally easily controlled with insulin	Less common—rebound hypoglycemia may occur

Table 83-12 Sequelae of Pediatric Cardiopulmonary Bypass

End-Organ Injury	Etiology/Signs
Renal injury	Organ immaturity, preexisting renal disease Post-cardiopulmonary bypass low CO, use of DHCA Renal dysfunction characterized by reduced GFR and ATN
Pulmonary injury	Endothelial injury, increased capillary leak, complement activation, and leukocyte degranulation Pulmonary dysfunction characterized by reduced compliance, reduced FRC, and increased A-a gradient
Cerebral injury after DHCA	Loss of autoregulation, suppressed metabolism and cerebral blood flow, cellular acidosis, and cerebral vasoparesis CNS dysfunction characterized by seizures, reduced developmental quotients, choreoathetosis, learning disabilities, behavioral abnormalities

Table 83-13 Comparison of ECMO versus VAD

	ECMO	VAD
Bleeding at insertion	++	++
Sternotomy	Not required	Required
Left atrial venting	±	—
Blood product use	+++	+
Number of cannulae for biventricular support	2	4
Pulmonary support	+	—
Intravenous anticoagulation	+	±
Duration of support	Weeks	Months
Emergent support	Yes	No
Patient mobility	—	+

ECMO, extracorporeal membrane oxygenation; VAD, ventricular assist device.

Table 83-15 Infective Endocarditis Prophylaxis

Situation	Drug	Single Dose 30-60 min before Dental Procedure	
		Adults	Children
Oral	Amoxicillin	2 g	50 mg/kg
Unable to take oral medication	Ampicillin <i>or</i>	2 g IM/IV	50 mg/kg IM/IV
	Cefazolin/ceftriaxone	1 g IM/IV	50 mg/kg IM/IV
Allergic to penicillins/oral	Cephalexin <i>or</i>	2 g	50 mg/kg IM/IV
	Clindamycin <i>or</i>	600 mg	20 mg/kg IM/IV
	Azithromycin/clarithromycin	500 mg	15 mg/kg
Allergic to penicillins/unable to take oral medication	Cefazolin/ceftriaxone <i>or</i>	1 g IM/IV	50 mg/kg IM/IV
	Clindamycin	600 mg	20 mg/kg

Vancomycin is an alternative for patients who are unable to tolerate a β -lactam or when the infective agent is considered to be methicillin-resistant *Staphylococcus aureus*.

Table 84-3 Vasoactive and Inotropic Agents in Pediatrics

Drug	Effect	Dose*	Inotropy	Chronotropy	Vasodilation	Vasoconstriction
Epinephrine (Adrenalin)	α, β	0.05-2.0	++	++		++
Isoproterenol (Isuprel)	β_1, β_2	0.05-2.0	++	++	+	
Dopamine (Inotropin)	δ	1-3			+Renal splanchnic	
	$\beta > \alpha$	5-15	+	+		+/-
	β, α	>15	+	+		+
Milrinone		Bolus: 50 $\mu\text{g/kg}$ over 10-min period Infusion: 0.375-0.75	+		+	
Norepinephrine	$\alpha \gg \beta$	0.05-1.0	sl+	+		++
Nitroprusside		0.5-10			++ Arterial > venous	
Nitroglycerin		1-20			++	

*All infusions are $\mu\text{g/kg/min}$.
sl, slight.

Box 84-2 Causes of Severe Hypertension in Children**Renal**

Acute glomerulonephritis (e.g., poststreptococcal, Henoch-Schönlein purpura)

Hemolytic-uremic syndrome

Chronic glomerulonephritis (all types)

Acute and chronic pyelonephritis

Congenital malformations (dysplasia, hypoplasia, cystic diseases)

Tumors (e.g., Wilms', leukemic infiltrate)

Post-renal transplantation status; also rejection

Oliguric renal failure

Trauma

Obstructive uropathy

After genitourinary surgery

Blood transfusions in children with azotemia

Cardiovascular

Coarctation of the aorta

Renal artery abnormalities (e.g., stenosis, thrombosis)

Takayasu's disease

Endocrine

Pheochromocytoma

Neuroblastoma

Adrenogenital disease

Cushing's syndrome

Hyperaldosteronism

Hyperthyroidism

Hyperparathyroidism

Iatrogenic

Intravascular volume overload

Administration of sympathomimetic drugs (e.g., epinephrine, ephedrine)

Administration of corticosteroid

Rapid intravenous infusion of methyldopa

Miscellaneous

Immobilization (e.g., fractures, burns, Guillain-Barré syndrome)

Hypercalcemia (e.g., hypervitaminosis D, metastatic disease, sarcoidosis, some immobilized patients)

Hypernatremia

Stevens-Johnson syndrome

Increased intracranial pressure (any cause)

Dysautonomia

After resuscitation

Table 84-5 Classification of Causes of Neonatal Respiratory Failure

	Congenital Abnormalities	Developmental Immaturity	Specific Neonatal "Stress"
Impaired control of ventilation	Central nervous system dysgenesis Ondine's curse	Apnea of prematurity Intracranial hemorrhage	Drug intoxication (note maternal drugs) Sepsis Central nervous system infections Seizures
Neuromuscular disorders	Congenital myopathies		High cervical cord injuries
Structural impairment	Thoracic deformities Lung hypoplasia Diaphragmatic hernia Potter's syndrome Abdominal malfunction Gastroschisis Omphalocele		Severe abdominal distention Pneumothorax or other air leak
Airway obstruction Upper airway	Choanal atresia Pierre Robin syndrome Laryngeal web/cleft Congenital tracheal/laryngeal stenosis Recurrent laryngeal palsy Hemangioma Lymphangioma		Massive meconium aspiration Vocal cord paralysis secondary to myelodysplasia
Lower airway	Tracheoesophageal fistula Lobar emphysema		Meconium/blood aspiration
Alveolar disorders		Respiratory distress syndrome	Bronchopulmonary dysplasia
Cardiovascular disorders	Congenital cardiac malformations Congestive heart failure (critical coarctation or aortic stenosis) Total anomalous pulmonary venous return		Persistent pulmonary hypertension

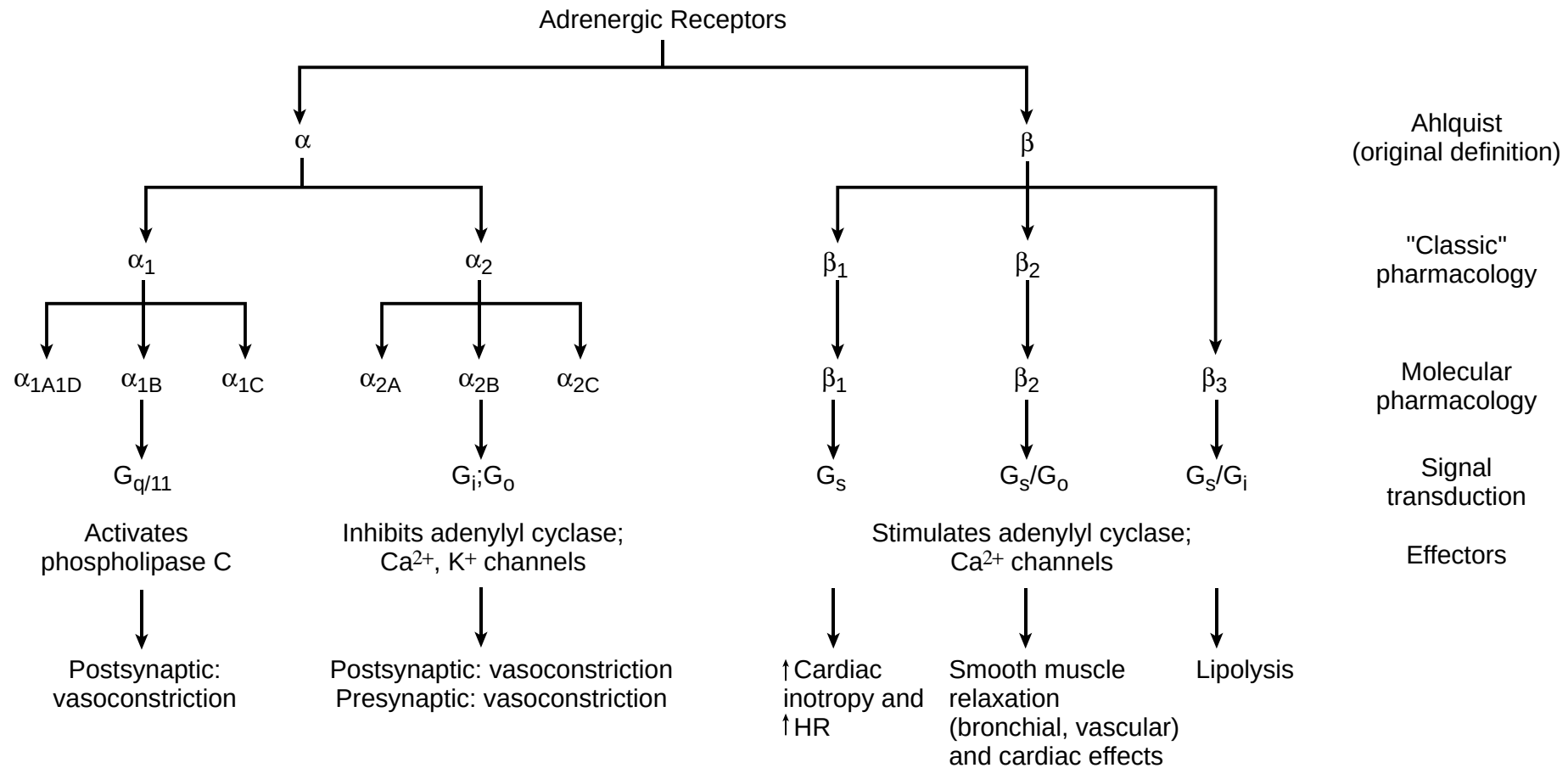


Figure 12-10 Classification of adrenergic receptors. HR, heart rate.

Table 12-2 Responses Elicited in Effector Organs by Stimulation of Sympathetic and Parasympathetic Nerves

Effector Organ	Adrenergic Response	Receptor Involved	Cholinergic Response	Dominant Response (A or C)
Heart				
Rate of contraction	Increase	β_1	Decrease	C
Force of contraction	Increase	β_1	Decrease	C
Blood vessels				
Arteries (most)	Vasoconstriction	α_1		A
Skeletal muscle	Vasodilation	β_2		A
Veins	Vasoconstriction	α_2		A
Bronchial tree	Bronchodilation	β_2	Bronchoconstriction	C
Splenic capsule	Contraction	α_1		A
Uterus	Contraction	α_1	Variable	A
Vas deferens	Contraction	α_1		A
Prostatic capsule	Contraction	α_1		A
Gastrointestinal tract	Relaxation	α_2	Contraction	C
Eye				
Radial muscle, iris	Contraction (mydriasis)	α_1		A
Circular muscle, iris			Contraction (miosis)	C
Ciliary muscle	Relaxation	β	Contraction (accommodation)	C
Kidney	Renin secretion	β_1		A
Urinary bladder				
Detrusor	Relaxation	β	Contraction	C
Trigone and sphincter	Contraction	α_1	Relaxation	A, C
Ureter	Contraction	α_1	Relaxation	A
Insulin release from pancreas	Decrease	α_2		A
Fat cells	Lipolysis	β_1		A
Liver glycogenolysis	Increase	α_1		A
Hair follicles, smooth muscle	Contraction (piloerection)	α_1		A
Nasal secretion			Increase	C
Salivary glands	Increase secretion	α_1	Increase secretion	C
Sweat glands	Increase secretion	α_1	Increase secretion	C

A, adrenergic; C, cholinergic.

From Ruffolo R: Physiology and biochemistry of the peripheral autonomic nervous system. In Wingard L, Brody T, Larner J, et al (eds): Human Pharmacology: Molecular to Clinical. St. Louis, Mosby-Year Book, 1991, p 77.

Table 12-5 Adrenergic Receptor Differentiation

Receptor	Stimulation	Inhibition
Alpha		
Heart		
Blood vessels	Vasoconstriction (skin, gut, kidney, liver, heart)	
Gastrointestinal tract	Sphincters	
Genitourinary tract	Sphincters	
Metabolic and endocrine effects		Insulin release
Beta		
Heart	(1) Rate, conduction contractility	
Blood vessels		(2) Vasodilation (skeletal muscle, heart, brain)
Respiration	(?) Respiratory center (2) Bronchodilation	
Gastrointestinal tract		(2) Smooth muscle
Genitourinary tract		(2) Ureteral and uterine muscle
Metabolic and endocrine effects	(2) Glycogenolysis (muscle, liver) (1) Lipolysis (2) Gluconeogenesis (1) Insulin release (?) Renin release (?) Antidiuretic hormone release	

(1), Mediated by β_1 -receptors; (2), mediated by β_2 -receptors; (?), controversial.

Table 12-7 Distribution of α - and β -Receptors

Receptor	Distribution	Response	Agonist	Antagonist
α_1	Smooth muscle	Constriction	Methoxamine Phenylephrine	Prazosin
α_2	Presynaptic	Inhibits norepinephrine release	Clonidine Dexmedetomidine	Yohimbine
β_1	Heart	Inotropy Chronotropy	Dobutamine	Metoprolol
β_2	Smooth muscle	Dilation Relaxation	Terbutaline	

Table 12-9 Dose-Dependent Actions of Inotropes and Chronotropes

Drug* (Proprietary Name)	Receptors	Usual Infusion Rate
Epinephrine (Adrenalin)	β_2 $\beta_1 + \beta_2$ α_1	1-2 $\mu\text{g}/\text{min}$ 2-10 $\mu\text{g}/\text{min}$ $\geq 10 \mu\text{g}/\text{min}^\dagger$ (bolus: 2-10 μg ; 0.5-1.0 $\text{mg}^\dagger$)
Norepinephrine (Levophed)	$\alpha_1, \beta_1, \gg \beta_2$	4-12 $\mu\text{g}/\text{min}^\dagger$
Dopamine (Intropin)	Dopaminergic β α	0-3 $\mu\text{g}/\text{kg}/\text{min}$ 3-10 $\mu\text{g}/\text{kg}/\text{min}$ >10 $\mu\text{g}/\text{kg}/\text{min}^\dagger$
Dobutamine (Dobutrex)	$\beta_1 \gg \beta_2, \alpha$	2.5-10 $\mu\text{g}/\text{kg}/\text{min}^\dagger$
Isoproterenol (Isuprel)	$\beta_1 > \beta_2$	0.5-10 $\mu\text{g}/\text{min}$
Amrinone (Inocor)	Increase cyclic adenosine monophosphate through inhibition of phosphodiesterase	0.75 mg/kg load over a period of 2-3 min 5- to 10- $\mu\text{g}/\text{kg}/\text{min}$ infusion

*All agents have elimination half-lives of a few minutes except amrinone ($t_{1/2}$, 3.6 hours; 5.8 hours in congestive heart failure).

† Much higher doses have been used in clinical practice.

‡ With anaphylaxis or cardiac arrest.

Data from Hoffman BB, Lefkowitz RJ: Catecholamines and sympathetic drugs. In Goodman A, Rall T, Nies A, et al (eds): Goodman and Gilman's the Pharmacological Basis of Therapeutics, 8th ed. New York, Pergamon Press, 1990, p 187.

Table 12-11 Muscarinic Anticholinergic Drugs

Drug	Duration	Central Nervous System*	Antisialagogue	Heart Rate
Atropine	Short	Stimulation	+	++
Glycopyrrolate	Long	0	++	+
Scopolamine	Short	Sedation	++	0/+

*The effects of atropine are limited with the usual clinical doses, but they can be significant in the elderly.

0, no effect; +, mild effect; ++, moderate effect.

Table 13-1 Normal Cerebral Physiologic Values

CBF	
Global	45-55 mL/100 g/min
Cortical (mostly gray matter)	75-80 mL/100 g/min
Subcortical (mostly white matter)	≈20 mL/100 g/min
CMRO ₂	3-3.5 mL/100 g/min
CVR	1.5-2.1 mm Hg/100 g/min/mL
Cerebral venous PO ₂	32-44 mm Hg
Cerebral venous SO ₂	55%-70%
ICP (supine)	8-12 mm Hg

CBF, cerebral blood flow; CMRO₂, cerebral metabolic rate of oxygen; CVR, cerebral vascular resistance; ICP, intracranial pressure.

Table 13-2 Factors Influencing Cerebral Blood Flow*

Factor	Comment
Chemical/Metabolic/Humoral	
CMR	CMR influence assumes intact flow-metabolism coupling, the mechanism of which is not fully understood
Anesthetics	
Temperature	
Arousal/seizures	
Paco ₂	
Pao ₂	
Vasoactive drugs	
Anesthetics	
Vasodilators	
Vasopressors	
Myogenic	
Autoregulation/MAP	The autoregulation mechanism is fragile, and in many pathologic states CBF is regionally pressure passive
Rheologic	
Blood viscosity	
Neurogenic	
Extracranial sympathetic and parasympathetic pathways	Contribution and clinical significance poorly defined
Intra-axial pathways	

*See text for discussion.

CBF, cerebral blood flow; CMR, cerebral metabolic rate; MAP, mean arterial pressure.

Box 15-1 Alveolar Gas Equations**Alveolar Oxygen Tension (P_{AO_2})**

$$P_{AO_2} = P_{IO_2} - \frac{P_{ACO_2}}{R} + \left[P_{ACO_2} \times F_{IO_2} \times \frac{1-R}{R} \right]$$

where P_{IO_2} is inspired oxygen tension, P_{ACO_2} is alveolar CO_2 tension (assumed to equal arterial PCO_2), R is the respiratory exchange ratio (normally in the range of 0.8 to 1.0), and F_{IO_2} is the inspired oxygen fraction.

The term within brackets compensates for the larger O_2 uptake than CO_2 elimination over the alveolar capillary membranes.

A simplified equation can be written without the compensation term:

$$P_{AO_2} = P_{IO_2} - \frac{P_{ACO_2}}{R}$$

Alveolar Ventilation

Alveolar ventilation ($\dot{V}_A$) can be expressed as

$$\dot{V}_A = f \times (V_T - V_{DS})$$

where f is breaths/min, V_T is tidal volume, and V_{DS} is physiologic dead space.

Alveolar ventilation can also be derived from

$$\dot{V}_{CO_2} = c \times \dot{V}_A \times F_{ACO_2}$$

where $\dot{V}_{CO_2}$ is CO_2 elimination, c is a conversion constant, and F_{ACO_2} is the alveolar CO_2 concentration.

If $\dot{V}_A$ is expressed in L/min, $\dot{V}_{CO_2}$ in mL/min, and F_{ACO_2} is replaced by P_{ACO_2} in mm Hg, $c = 0.863$. By rearranging,

$$\dot{V}_A = \frac{\dot{V}_{CO_2} \times 0.863}{P_{ACO_2}}$$

Table 15-1 Causes of Hypoxemia

Disturbance	P_{aO_2} (breathing air) at Rest	P_{aO_2} (breathing oxygen) at Rest	P_{aO_2} (breathing air) with Exercise (Versus Rest)	P_{aCO_2}
Hypoventilation	Reduced	Normal	No change or further decrease	Increased
$\dot{V}_A/\dot{Q}$ mismatch	Reduced	Normal	No or minor increase or decrease	Normal
Shunt	Reduced	Reduced	No change or further decrease	Normal
Diffusion impairment	Reduced	Normal	Small to large decrease	Normal

Box 15-2 Derivation of the Physiologic Dead Space Equation

$$F_E \times V_T = F_A(V_T - V_{DS}) + (F_I \times V_{DS})$$

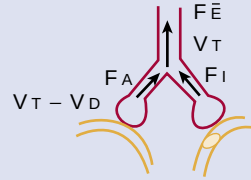
By rearranging,

$$\frac{V_{DS}}{V_T} = \frac{F_A - F_E}{F_A - F_I}$$

If $F_I = 0$, F replaced by P , and P_A replaced by P_a , for CO_2 ,

$$\frac{V_{DS}}{V_T} = \frac{P_{a\text{CO}_2} - P_{E\text{CO}_2}}{P_{a\text{CO}_2}}$$

where F_E , F_A , and F_I are mixed expired, alveolar, and inspired gas concentration, respectively, and V_T , V_{DS} , and V_A are tidal volume, dead space, and part of the tidal volume to perfused alveoli, respectively.

**Box 15-3** Derivation of the Venous Admixture ("Shunt") Equation

$$C_a \times \dot{Q}_T = (C_c' \times \dot{Q}_C) + (C_{\bar{v}} \times \dot{Q}_S) \quad (1)$$

$$\dot{Q}_C = \dot{Q}_T - \dot{Q}_S \quad (2)$$

By inserting Equation 2 into Equation 1,

$$C_a \times \dot{Q}_T = (C_c' \times [\dot{Q}_T - \dot{Q}_S]) + (C_{\bar{v}} \times \dot{Q}_S)$$

Rearranging,

$$\frac{\dot{Q}_S}{\dot{Q}_T} = \frac{C_c' - C_a}{C_c' - C_{\bar{v}}}$$

where C_c' , C_a , and $C_{\bar{v}}$ are oxygen content in pulmonary end-capillary, arterial, and mixed venous blood, respectively; $\dot{Q}_T$ is cardiac output; $\dot{Q}_C$ is capillary flow; and $\dot{Q}_S$ is shunt.

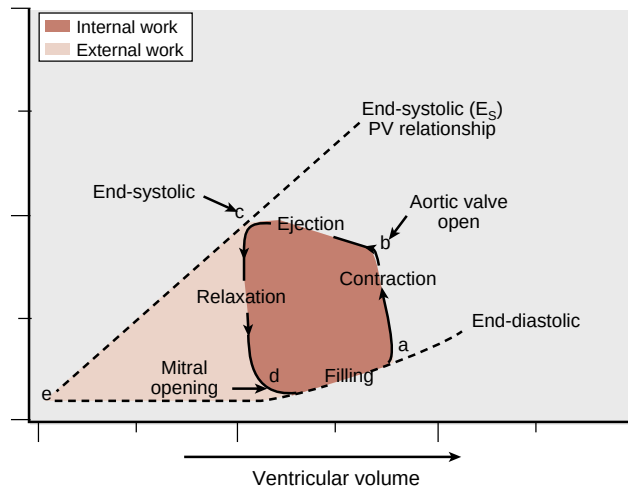
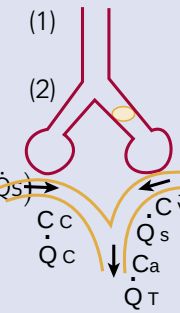


Figure 16-6 Pressure-volume loop. Point a depicts the start of isovolumic contraction. The aortic valve opens at point b, and ejection of blood follows (b→c). The mitral valve opens at d, and ventricular filling ensues. External work is defined by a, b, c, and d and internal work by e, d, and c. The pressure-volume area is the sum of external and internal work. (From Opie LH: Ventricular function. In *The Heart. Physiology from Cell to Circulation*, 4th ed. Philadelphia, Lippincott-Raven, 2004, pp 355-401.)

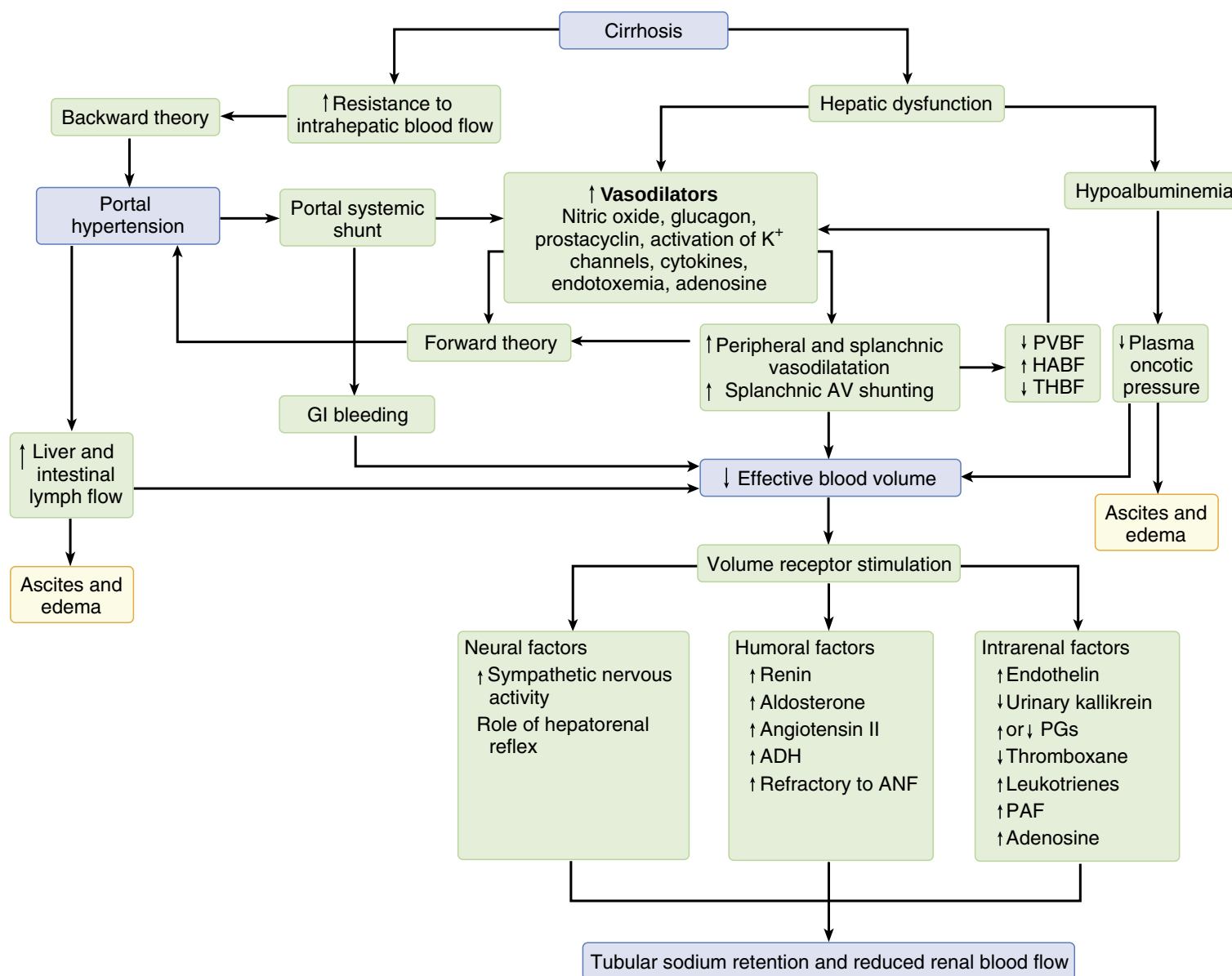


Figure 17-13 Schematic of pathways for cirrhosis-induced portal hypertension: the forward and backward theories. Cirrhosis and portal hypertension induce circulatory changes that decrease effective blood volume. This activates volume receptors and stimulates neurohumoral and intrarenal reflexes, which decreases renal blood flow and increases renal retention of sodium. ADH, antidiuretic hormone; ANF, atrial natriuretic factor; AV, arteriovenous; HABF, hepatic arterial blood flow; PAF, platelet-activating factor; PGs, prostaglandins; PVBF, portal venous blood flow; THBF, total hepatic blood flow. (Reprinted with permission from Mushlin PS, Gelman S: *Anesthesia and the liver*. In Barash PG, Cullen BF, Stoelting RK [eds]: *Clinical Anesthesia*, 4th ed. Philadelphia, Lippincott Williams & Wilkins, 2001, p 1088.)

Vasoconstrictor Systems	Vasodilator Systems
Sympathoadrenal system Renin-angiotensin system Aldosterone Antidiuretic hormone (ADH)	Prostaglandins Kinins Atrial natriuretic peptide (ANP)
↓ RBF ↓ GFR ↓ Urine flow ↓ Na excretion	↑ RBF ↑ GFR ↑ Urine flow ↑ Na excretion

Figure 18-17 Neurohormonal renal regulatory systems. GFR, glomerular filtration rate; Na, sodium; RBF, renal blood flow; ↓, decreased; ↑, increased. (Modified from Sladen RN: *Effect of anesthesia and surgery on renal function. Crit Care Clin* 3:380, 1987.)

Table 18-3 Adenosine Receptor Subtypes and Functions

Receptor	Agonist Function	Ischemic Injury
A ₁	Outer cortical vasoconstriction Decreased renin release Inhibition of diuresis and natriuresis	Highly protective
A _{2a}	Juxtamedullary vasodilation Increased renin release Promotion of diuresis and natriuresis	Highly protective
A _{2b}	Unknown	
A ₃	Unknown	Potentiates injury

From Fozard JR, Hannon JP: Adenosine receptor ligands: potential as therapeutic agents in asthma and COPD. *Pulm Pharmacol Ther* 12:111-114, 1999.

Table 18-2 Dopamine and its Analogs

Receptor	DA ₁	DA ₂	β ₁	β ₂	α ₁
Dopamine	+++	++	++	±	+++
Dobutamine	0	0	+++	++	±
Dopexamine	++	+	±	+++	0
Fenoldopam	++++	0	0	0	0

Dobutamine, dopexamine, and fenoldopam are all pharmacologic analogs of dopamine. However, dobutamine is devoid of dopaminergic activity; dopexamine has about one third of the dopaminergic activity of dopamine; and fenoldopam is a pure, selective dopamine₁-receptor agonist. α₁, alpha₁-receptor; β₁, beta₁-receptor; β₂, beta₂-receptor; DA₁, dopamine₁-receptor; DA₂, dopamine₂-receptor.

Renal Clearance

The kidneys clear drug from plasma by filtration at the glomerulus and direct transport into the tubules. Renal blood flow is inversely correlated with age, as is creatinine clearance, which can be predicted from age and weight according to the equation of Cockcroft and Gault⁵:

Men:

$$\text{Creatinine clearance (mL/min)} = \frac{(140 - \text{Age [yr]} \times \text{Weight [kg]})}{72 \times \text{Serum creatinine (mg\%)}} \quad (9)$$

Women:

85% of the above.

Table 19-2 Site of Action of Sedatives and Hypnotics

Anesthetic	GABA _A	Glycine	Neuronal nAChR	5-HT ₃	AMPA (GluR1-4)	NMDA
Barbiturates	+++	+/0	---	-/0	---	0
Chloroform	+++	+++	---	Unknown	---	0
Diethyl ether	+++	+++	---	+++	---	---
Enflurane	+++	+++	---	+++	---	-/0
Etomidate	+++	+/0	-/0	0	Unknown	Unknown
Halothane	+++	+++	---	+++	---	-/0
Isoflurane	+++	+++	---	+++	---	-/0
Ketamine	+/0	0	---	+/0	0	---
Methoxyflurane	+++	+++	---	+++	Unknown	Unknown
Nitrous oxide	+++	+/0	Unknown	Unknown	-/0	---
Propofol	+++	+++	-/0	0	-/0	-/0
Sevoflurane	+++	+++	---	Unknown	Unknown	Unknown
Steroidal anesthetics	+++	0	-/0	-/0	0	0
Xenon	0	Unknown	Unknown	Unknown	0	---

AMPA, α -amino-3-hydroxy-5-methyl-4-isoxazolepropionate; GABA, γ -aminobutyric acid; 5-HT, 5-hydroxytryptamine; nAChR, nicotinic acetylcholine receptor; NMDA, *N*-methyl-D-aspartate; +++, potentiation of the agonist; ---, inhibition of the agonist; +/0 and -/0, little potentiation or inhibition, except at concentrations well in excess of the clinical range; 0, no effect at any concentration.

From Krasowski MD, Harrison NL: General anaesthetic actions on ligand-gated ion channels. *Cell Mol Life Sci* 55:1278-1303, 1999.

Table 21-1 Partition Coefficients at 37°C

Anesthetic	Blood-Gas	Brain-Blood	Liver-Blood	Kidney-Blood	Muscle-Blood	Fat-Blood
Desflurane	0.45	1.3	1.4	1.0	2.0	27
Nitrous oxide	0.47	1.1	0.8	—	1.2	2.3
Sevoflurane	0.65	1.7	1.8	1.2	3.1	48
Isoflurane	1.4	1.6	1.8	1.2	2.9	45
Enflurane	1.8	1.4	2.1	—	1.7	36
Halothane	2.5	1.9	2.1	1.2	3.4	51
Diethyl ether	12	2.0	1.9	0.9	1.3	5
Methoxyflurane	15	1.4	2.0	0.9	1.6	38

Data from references 1 through 8.

Box 29-2 Reported Indications for the Use of Muscle Relaxants in the Intensive Care Unit

Facilitate mechanical ventilation

Facilitation of endotracheal intubation

Enable patients to tolerate mechanical ventilation

High pulmonary inflation pressures, e.g., acute respiratory distress syndrome

Hyperventilation for increased intracranial pressure

Facilitate therapeutic or diagnostic procedures

Tetanus

Status epilepticus

Reduce oxygen consumption

Abolish shivering

Reduce work of breathing

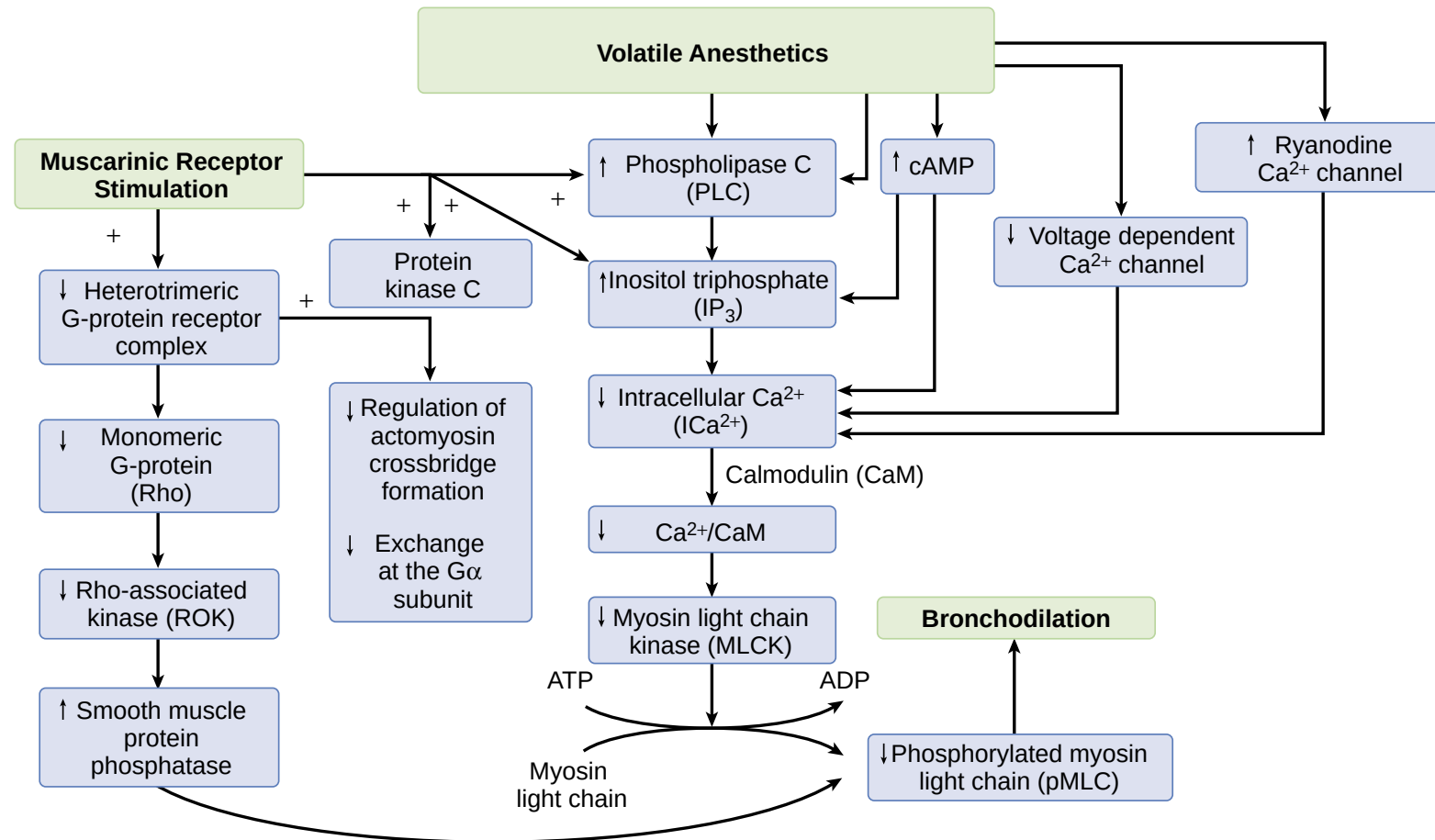


Figure 22-4 Proposed signaling pathways underlying volatile anesthetic (specifically, halothane)-induced bronchodilation or inhibition (or both) of muscarinic agonist-induced contraction of airway smooth muscle. +, excitatory action of muscarinic receptor agonist; ↑, activation or increase attributable to the volatile anesthetic; ↓, inhibition or decrease attributable to the volatile anesthetic. Signal transduction along pathway A is supported by work from Warner and coworkers on a role of halothane in decreasing Ca²⁺ sensitivity rather than a change in ICa²⁺ content. (Adapted from Pabelick CM, Prakash YS, Kannan MS, et al: Effects of halothane on sarcoplasmic reticulum calcium release channels in porcine airway smooth muscle cells. *Anesthesiology* 95:207, 2001; and Hanazaki M, Jones KA, Perkins WJ, et al: Halothane increases smooth muscle protein phosphatase in airway smooth muscle. *Anesthesiology* 94:129, 2001, with permission.)

Table 24-1 Anesthetic Properties of Xenon versus Other Anesthetics

	Xenon	Nitrous Oxide	Isoflurane	Desflurane	Sevoflurane
Oil-gas partition coefficient	1.9	1.4	90	18.7	53.4
Blood-gas partition coefficient	0.14	0.47	1.4	0.42	0.6
Minimum alveolar concentration (%)	71	≈105	1.15	6.0	1.71

Table 24-2 Clinical Features of Halothane Hepatitis

Mild Form	Fulminant Form
Incidence, 1 : 5	Incidence, 1 : 10,000
Repeat exposure not necessary	Multiple exposures
Mild elevation of ALT, AST	Marked elevation of ALT, AST, bilirubin, alkaline phosphatase
Focal necrosis	Massive hepatic necrosis
Self-limited	Mortality rate, 50%
	Antibodies to halothane-altered protein antigens

ALT, alanine aminotransferase; AST, aspartate aminotransferase.

Box 24-2 Risk Factors for Anesthetic-Induced Hepatitis

Gender
Age
Obesity
Enzyme induction
Prior anesthetic exposure
Genetics

Box 24-3 Investigation of Postoperative Liver Dysfunction

History and physical examination
Medications
Previous anesthetic exposure
Intraoperative course
Postoperative course
Clinical tests
Antibody screening

Table 26-3 Uses and Doses of Intravenous Propofol

Induction of general anesthesia	1-2.5 mg/kg IV dose reduced with increasing age
Maintenance of general anesthesia	50-150 µg/kg/min IV combined with N ₂ O or an opiate
Sedation	25-75 µg/kg/min IV
Antiemetic	10-20 mg IV, can repeat every 5-10 min or start infusion of 10 µg/kg/min

N₂O, nitrous oxide.

Table 26-4 Recommended Doses of Barbiturates for Anesthesia Induction and Maintenance

Drug	Induction Dose (mg/kg)*†	Onset (sec)	Intravenous Maintenance Infusion
Thiopental	3-4	10-30	50-100 mg every 10-12 min
Methohexital	1-1.5	10-30	20-40 mg every 4-7 min

*Adult and pediatric IV doses are roughly the same in milligrams per kilogram.

†Methohexital can be given rectally in pediatric patients as 20-25 mg/kg/dose.

Table 26-5 Physicochemical Characterization of Three Benzodiazepines

	Diazepam	Lorazepam	Midazolam
Molecular weight (D)	284.7*	321.2*	362*
pK _a	3.3 (20°)	11.5 (20°)	6.2 (20°)*
Water soluble	No*	Almost insoluble	Yes [†]
Lipid soluble	Yes*	Yes (less so, however)	Yes [†]
	Highly lipophilic	Relatively less lipophilic	Highly lipophilic

*Data from Moffet.⁵⁸¹[†]pH dependent: pH >4, lipid soluble; pH <4, water soluble.**Table 26-7** Uses and Doses of Intravenous Benzodiazepines

	Midazolam	Diazepam	Lorazepam
Induction	0.05-0.15 mg/kg	0.3-0.5 mg/kg	0.1 mg/kg
Maintenance	0.05 mg/kg prn 1 µg/kg/min	0.1 mg/kg prn	0.02 mg/kg prn
Sedation*	0.5-1 mg repeated 0.07 mg/kg IM	2 mg repeated	0.25 mg repeated

*Incremental doses given until desired degree of sedation is obtained.
prn, as required to keep patient hypnotic and amnestic.**Table 26-9** Uses and Doses of Ketamine

Induction of general anesthesia*	0.5-2 mg/kg IV 4-6 mg/kg IM
Maintenance of general anesthesia	0.5-1 mg/kg IV with N ₂ O 50% in O ₂ 15-45 µg/kg/min IV with N ₂ O 50-70% in O ₂ 30-90 µg/kg/min IV without N ₂ O
Sedation and analgesia	0.2-0.8 mg/kg IV over 2-3 min 2-4 mg/kg IM
Preemptive/preventive analgesia	0.15-0.25 mg/kg IV

*Lower doses are used if adjuvant drugs such as midazolam or thiopental also are given.

N₂O, nitrous oxide.**Table 26-10** Uses and Doses of Etomidate

Induction of general anesthesia	0.2-0.6 mg/kg IV
Maintenance of general anesthesia	10 µg/kg/min IV with N ₂ O and an opiate
Sedation and analgesia	Limited to periods of brief sedation because of inhibition of corticosteroid synthesis

N₂O, nitrous oxide.

Table 27-2 Pharmacologic Actions of Opioids and Opioid Receptors in Animal Models

	Actions of		
	Receptor	Agonists	Antagonists
Analgesia			
Supraspinal	μ, δ, κ	Analgesic	No effect
Spinal	μ, δ, κ	Analgesic	No effect
Respiratory function	μ	Decrease	No effect
Gastrointestinal tract	μ, κ	Decrease transit	No effect
Psychotomimesis	κ	Increase	No effect
Feeding	μ, δ, κ	Increase feeding	Decrease feeding
Sedation	μ, κ	Increase	No effect
Diuresis	κ	Increase	
Hormone secretion			
Prolactin	μ	Increase release	Decrease release
Growth hormone	μ and/or δ	Increase release	Decrease release
Neurotransmitter release			
Acetylcholine	μ	Inhibit	
Dopamine	δ	Inhibit	

Table 27-4 Potential Problems Associated with Opioid-Induced Rigidity

System	Problem
Hemodynamic	↑CVP, ↑ PAP, ↑ PVR
Respiratory	↓ Compliance, ↓ FRC, ↓ ventilation Hypercapnia Hypoxemia
Miscellaneous	↑ Oxygen consumption ↑ Intracranial pressure ↑ Fentanyl plasma levels

CVP, central venous pressure; FRC, functional residual capacity; PAP, pulmonary artery pressure; PVR, pulmonary vascular resistance.

Modified from Bailey PL, Egan TD, Stanley TH: Intravenous opioid anesthetics. *In* Miller RD (ed): Anesthesia, 5th ed. New York, Churchill Livingstone, 2000, p 291.

Table 27-7 Approximate Opioid Loading (Bolus) Doses, Maintenance Infusion Rates, and Additional Maintenance Doses for Total Intravenous Anesthesia

	Loading Dose (μg/kg)	Maintenance Infusion Rate	Additional Boluses
Alfentanil	25-100	0.5-2 μg/kg/min	5-10 μg/kg
Sufentanil	0.25-2	0.5-1.5 μg/kg/hr	2.5-10 μg
Fentanyl	4-20	2-10 μg/kg/hr	25-100 μg
Remifentanyl	1-2	0.1-1.0 μg/kg/min	0.1-1.0 μg/kg

From Bailey PL, Egan TD, Stanley TH: Intravenous opioid anesthetics. *In* Miller RD (ed): Anesthesia, 5th ed. New York, Churchill Livingstone, 2000, p 335.

Table 27-10 Hemodynamic Effects of Agonist-Antagonist Compounds Compared with Morphine

Drug	Cardiac Workload	Blood Pressure	Heart Rate	Pulmonary Artery Pressure
Morphine	↓	↓	=↓	=↓
Buprenorphine	↓	↓	↓	?
Butorphanol	↑	=↑	=	↑
Nalbuphine	↓	=	=↓	=
Pentazocine	↑	↑	↑	↑

From Zola EM, McLeod DC: Comparative effects of analgesic efficacy of the agonist-antagonist opioids. Drug Intell Clin Pharm 17:411, 1983.

Table 28-6 Manual Infusion Schemes*

Drug	Anesthesia		Sedation or Analgesia	
	Loading Dose (μg/kg)	Maintenance Infusion (μg/kg/min)	Loading Dose (μg/kg)	Maintenance Infusion (μg/kg/min)
Alfentanil	50-150	0.5-3	10-25	0.25-1
Fentanyl	5-15	0.03-0.1	1-3	0.01-0.03
Sufentanil	0.5-5	0.01-0.05	0.1-0.5	0.005-0.01
Remifentanyl	0.5-1.0	0.1-0.4	†	0.025-0.1
Ketamine	1500-2500	25-75	500-1000	10-20
Propofol	1000-2000	50-150	250-1000	10-50
Midazolam	50-150	0.25-1.5	25-100	0.25-1
Methohexital	1500-2500	50-150	250-1000	10-50
Dexmedetomidine‡			0.5-1 over 10 min	0.2-0.7

*After the loading dose, an initially high infusion rate to account for redistribution should be used and then titrated to the lowest infusion rate that will maintain adequate anesthesia or sedation. When using opiates as part of a nitrous-narcotic technique or for cardiac anesthesia, the dosing scheme listed under anesthesia is used. When the opiate is combined as part of balanced anesthesia, dosing listed for analgesia is needed.

†For analgesia or during sedation, an initial loading dose of remifentanyl should not be given because its very rapid onset may result in apnea or muscle rigidity.

Table 29-1 Relationship between Dibucaine Number and Duration of Succinylcholine or Mivacurium Neuromuscular Blockade

Type of Butyrylcholinesterase	Genotype	Incidence	Dibucaine Number*	Response to Succinylcholine or Mivacurium
Homozygous typical	E ₁ ^u E ₁ ^u	Normal	70-80	Normal
Heterozygous atypical	E ₁ ^u E ₁ ^a	1/480	50-60	Lengthened by 50%-100%
Homozygous atypical	E ₁ ^a E ₁ ^a	1/3200	20-30	Prolonged to 4-8 hr

*The dibucaine number indicates the percentage of enzyme inhibited.

Table 29-3 Classification of Nondepolarizing Neuromuscular Blockers According to Duration of Action (Time to T₁ = 25% of Control) after Twice the ED₉₅

Class of Blocker	Clinical Duration			
	Long-Acting (>50 min)	Intermediate-Acting (20-50 min)	Short-Acting (15-20 min)	Ultrashort-acting (<10-12 min)
Steroidal compounds	Pancuronium Pipcuronium	Vecuronium Rocuronium		
Benzylisoquinolinium compounds	<i>d</i> -Tubocurarine Metocurine Doxacurium	Atracurium Cisatracurium	Mivacurium	
Others				
Asymmetrical mixed-onium chlorofumarates				Gantacurium
Phenolic ether	Gallamine			
Diallyl derivative of toxiferine	Alcuronium			

A majority of nondepolarizing neuromuscular blockers are bisquaternary ammonium compounds. *d*-Tubocurarine, vecuronium, rocuronium, and rapacuronium are mono-quaternary compounds, and gallamine is a trisquaternary ammonium compound.

Table 29-4 Dose-Response Relationships of Nondepolarizing Neuromuscular Blocking Drugs in Human Subjects

	ED ₅₀ (mg/kg)	ED ₉₀ (mg/kg)	ED ₉₅ (mg/kg)	Reference(s)
Long-Acting				
Pancuronium	0.036 (0.022-0.042)	0.056 (0.044-0.070)	0.067 (0.059-0.080)	103, 107
<i>d</i> -Tubocurarine	0.23 (0.16-0.26)	0.41 (0.27-0.45)	0.48 (0.34-0.56)	107
Intermediate-Acting				
Rocuronium	0.147 (0.069-0.220)	0.268 (0.200-0.419)	0.305 (0.257-0.521)	103, 108-110
Vecuronium	0.027 (0.015-0.031)	0.042 (0.023-0.055)	0.043 (0.037-0.059)	107
Atracurium	0.12 (0.08-0.15)	0.18 (0.19-0.24)	0.21 (0.13-0.28)	107
Cisatracurium	0.026 (0.015-0.031)	—	0.04 (0.032-0.05)	11, 111, 112, 118
Short-Acting				
Mivacurium	0.039 (0.027-0.052)	—	0.067 (0.045-0.081)	9, 113-115
Ultrashort-Acting				
Gantacurium	0.09	—	0.19	106

Data are medians and ranges of reported values. ED₅₀, ED₉₀, and ED₉₅ are the doses of each drug that produce, respectively, a 50%, 90%, and 95% decrease in the force of contraction or amplitude of the electromyogram of the adductor pollicis muscle after ulnar nerve stimulation.

Table 29-6 Guide to Nondepolarizing Relaxant Dosage (mg/kg) with Different Anesthetic Techniques*

			Supplemental Dose after Intubation	Dosage for Relaxation	
ED ₉₅ under N ₂ O/O ₂		Dose for Intubation		N ₂ O	Anesthetic Vapors [†]
Long-Acting					
Pancuronium	0.07	0.08-0.12	0.02	0.05	0.03
d-Tubocurarine	0.5	0.5-0.6	0.1	0.3	0.15
Intermediate-Acting					
Vecuronium	0.05	0.1-0.2	0.02	0.05	0.03
Atracurium	0.23	0.5-0.6	0.1	0.3	0.15
Cisatracurium	0.05	0.15-0.2	0.02	0.05	0.04
Rocuronium	0.3	0.6-1.0	0.1	0.3	0.15
Short-Acting					
Mivacurium	0.08	0.2-0.25	0.05	0.1	0.08
Continuous Infusion Dosage (μg/kg/min) Required to Maintain 90%-95% Twitch Inhibition under N ₂ O/O ₂ with Intravenous Agents					
Mivacurium	3-15				
Atracurium	4-12				
Cisatracurium	1-2				
Vecuronium	0.8-1.0				
Rocuronium	9-12				

Box 29-3 Complications of Muscle Paralysis in the Intensive Care Unit

Short-term use

- Specific, known drug side effects
- Inadequate ventilation in the event of ventilator failure or circuit disconnection
- Inadequate analgesia and/or sedation

Long-term use

- Complications of immobility
 - Deep venous thrombosis and pulmonary embolism
 - Peripheral nerve injuries
 - Decubitus ulcers
- Inability to cough
 - Retention of secretions and atelectasis
 - Pulmonary infection

Dysregulation of nicotinic acetylcholine receptors

Prolonged paralysis after stopping relaxants

- Persistent neuromuscular blockade
- Critical illness myopathy
- Critical illness polyneuropathy
- Combination of the above

Unrecognized effects of drug or metabolites

- Succinylcholine and metabolic acidosis/hypovolemia
- 3-Desacetylvecuronium and neuromuscular blockade
- Laudanosine and cerebral excitation

Box 29-4 Causes of Generalized Neuromuscular Weakness in the Intensive Care Unit

Central nervous system

- Septic or toxic-metabolic encephalopathy
- Brainstem stroke
- Central pontine myelinolysis
- Anterior horn cell disorders (e.g., amyotrophic lateral sclerosis)

Peripheral neuropathies

- Critical illness polyneuropathy
- Guillain-Barré syndrome
- Porphyria
- Paraneoplasia
- Vasculitis
- Nutritional and toxic

Neuromuscular junction disorders

- Myasthenia gravis
- Lambert-Eaton myasthenic syndrome
- Botulism
- Prolonged neuromuscular junction blockade

Myopathies

- Critical illness myopathy
- Cachectic myopathy
- Rhabdomyolysis
- Inflammatory and infectious myopathies
- Muscular dystrophies
- Toxic
 - Acid maltase deficiency
 - Mitochondrial
 - Hypokalemia
 - Hypermetabolic syndromes with rhabdomyolysis (e.g., neuroleptic malignant syndrome)

From Lacomis D: Critical illness myopathy. Curr Rheumatol Rep 4:403-408, 2002.

Box 29-5 Recommendations for the Use of Neuromuscular Blockers in the Intensive Care Unit

Avoid the use of neuromuscular blockers by:

Maximal use of analgesics and sedatives

Manipulation of ventilatory parameters and modes

Minimize the dose of neuromuscular blocker:

Use a peripheral nerve stimulator with train-of-four monitoring.

Do not administer for more than 2 days continuously.

Administer by bolus rather than infusion.

Administer only when required and to achieve a well-defined goal.

Continually allow recovery from paralysis.

Consider alternative therapies:

Avoid vecuronium in female patients with renal failure.

Use isoflurane in place of muscle relaxants in patients with severe asthma.

Minimize the dose of steroid in patients with asthma.

Table 30-3 Classification of Peripheral Nerves According to Anatomy, Physiology, and Function

Fiber Class	Subclass	Myelin	Diameter (μm)	Conduction Velocity (msec)	Location	Function	Susceptibility to Local Anesthetic Block
A	α	+	6-22	30-120	Efferent to muscles	Motor	++
	β	+	6-22	30-120	Afferent from skin and joints	Tactile, proprioception	++
	γ	+	3-6	15-35	Efferent to muscle spindles	Muscle tone	++++
	δ	+	1-4	5-25	Afferent sensory nerves	Pain, cold temperature, touch	+++
B		+	<3	3-15	Preganglionic sympathetic	Various autonomic functions	++
C	sC	—	0.3-1.3	0.7-1.3	Postganglionic sympathetic	Various autonomic functions	++
	dC	—	0.4-1.2	0.1-2.0	Afferent sensory nerves	Various autonomic functions Pain, warm temperature, touch	+

Modified From Bonica JJ: Principles and Practice of Obstetric Anesthesia and Analgesia. Philadelphia, FA Davis, 1967.

Table 30-4 Infiltration Anesthesia

Drug	Plain Solution			Epinephrine-Containing Solution	
	Concentration (%)	Max Dose (mg)	Duration (min)	Max Dose (mg)	Duration (min)
Short Duration					
Procaine	1-2	500	20-30	600	30-45
Chlorprocaine	1-2	800	15-30	1000	30
Moderate Duration					
Lidocaine	0.5-1	300	30-60	500	120
Mepivacaine	0.5-1	300	45-90	500	120
Prilocaine	0.5-1	350	30-90	550	120
Long Duration					
Bupivacaine	0.25-0.5	175	120-240	200	180-240
Ropivacaine	0.2-0.5	200	120-240	250	180-240

Table 30-8 Spinal Anesthesia*

Drug	Usual Concentration (%)	Usual Volume (mL)	Total Dose (mg)	Baricity	Glucose Concentration (%)	Usual Duration (min)
Procaine	10.0	1-2	100-200	Hyperbaric	5.0	30-60
Lidocaine	1.5, 5.0	1-2	30-100	Hyperbaric	7.5	30-90
Mepivacaine	4	1-2	40-80	Hyperbaric	9.0	30-90
Tetracaine	0.25-1.0	1-4	5-20	Hyperbaric	5.0	90-200
	0.25	2-6	5-20	Hypobaric		90-200
	1.0	1-2	5-20	Isobaric		90-200
Dibucaine	0.25	1-2	2.5-5.0	Hyperbaric	5.0	90-200
	0.5	1-2	5-10	Isobaric		90-200
	0.06	5-20	3-12	Hypobaric		90-200
Bupivacaine	0.5	3-4	15-20	Isobaric		90-200
	0.75	2-3	15-20	Hyperbaric	8.25	90-200
Levobupivacaine	0.5	3-4	15-20	Isobaric		90-200
	0.75	2-3	15-20	Hyperbaric		90-200
Ropivacaine	0.5	3-4	15-20	Isobaric		90-200
	0.75	2-3	15-20	Hyperbaric		90-200

*Dosing refers to a 70-Kg adult. Dosing may be reduced in pregnancy (see Chapter 69) and with advancing age (see Chapter 71). Pediatric dosing is detailed in Chapter 81.

Table 34-1 American Society of Anesthesiologists Physical Status Classification

ASA 1	Healthy patient without organic, biochemical, or psychiatric disease
ASA 2	A patient with mild systemic disease, e.g., mild asthma or well-controlled hypertension. No significant impact on daily activity. Unlikely to have an impact on anesthesia and surgery
ASA 3	Significant or severe systemic disease that limits normal activity, e.g., renal failure on dialysis or class 2 congestive heart failure. Significant impact on daily activity. Probable impact on anesthesia and surgery
ASA 4	Severe disease that is a constant threat to life or requires intensive therapy, e.g., acute myocardial infarction, respiratory failure requiring mechanical ventilation. Serious limitation of daily activity. Major impact on anesthesia and surgery
ASA 5	Moribund patient who is equally likely to die in the next 24 hours with or without surgery
ASA 6	Brain-dead organ donor

"E" added to the classification indicates emergency surgery.
Available from www.asahq.org.

Box 34-1 Components of the Airway Examination

Length of the upper incisors
Condition of the teeth
Relationship of the upper (maxillary) incisors to the lower (mandibular) incisors
Ability to protrude or advance the lower (mandibular) incisors in front of the upper (maxillary) incisors
Interincisor or intergum (if edentulous) distance
Tongue size
Visibility of the uvula
Presence of heavy facial hair
Compliance of the mandibular space
Thyromental distance with the head in maximum extension
Length of the neck
Thickness or circumference of the neck
Range of motion of the head and neck

Table 34-2 Metabolic Equivalents of Functional Capacity

MET	Functional Levels of Exercise
1	Eating, working at a computer, dressing
2	Walking down stairs or in your house, cooking
3	Walking 1-2 blocks
4	Raking leaves, gardening
5	Climbing 1 flight of stairs, dancing, bicycling
6	Playing golf, carrying clubs
7	Playing singles tennis
8	Rapidly climbing stairs, jogging slowly
9	Jumping rope slowly, moderate cycling
10	Swimming quickly, running or jogging briskly
11	Skiing cross country, playing full-court basketball
12	Running rapidly for moderate to long distances

From Jetté M, Sidney K, Blumchen G: Metabolic equivalents (METs) in exercise testing, exercise prescription, and evaluation of functional capacity. *Clin Cardiol* 13:555-565, 1990.

MET, metabolic equivalent of the task. 1 MET = consumption of 3.5 mL O₂/min/kg of body weight.

Box 34-2 Revised Cardiac Risk Index

High-risk surgery (intraabdominal, intrathoracic, or suprainguinal vascular procedures)*
Ischemic heart disease (by any diagnostic criteria)
History of congestive heart failure
History of cerebrovascular disease
Diabetes mellitus requiring insulin†
Creatinine >2.0 mg/dL

*This risk factor is not considered a clinical predictor in the ACC/AHA 2007 guidelines for perioperative cardiac evaluation for noncardiac surgery.⁷

†This risk factor has been changed to simply diabetes mellitus in the ACC/AHA 2007 guidelines for perioperative cardiac evaluation for noncardiac surgery.⁷

From Lee TH, Marcantonio ER, Mangione CM, et al: Derivation and prospective validation of a simple index for prediction of cardiac risk of major noncardiac surgery. *Circulation* 100:1043-1049, 1999.

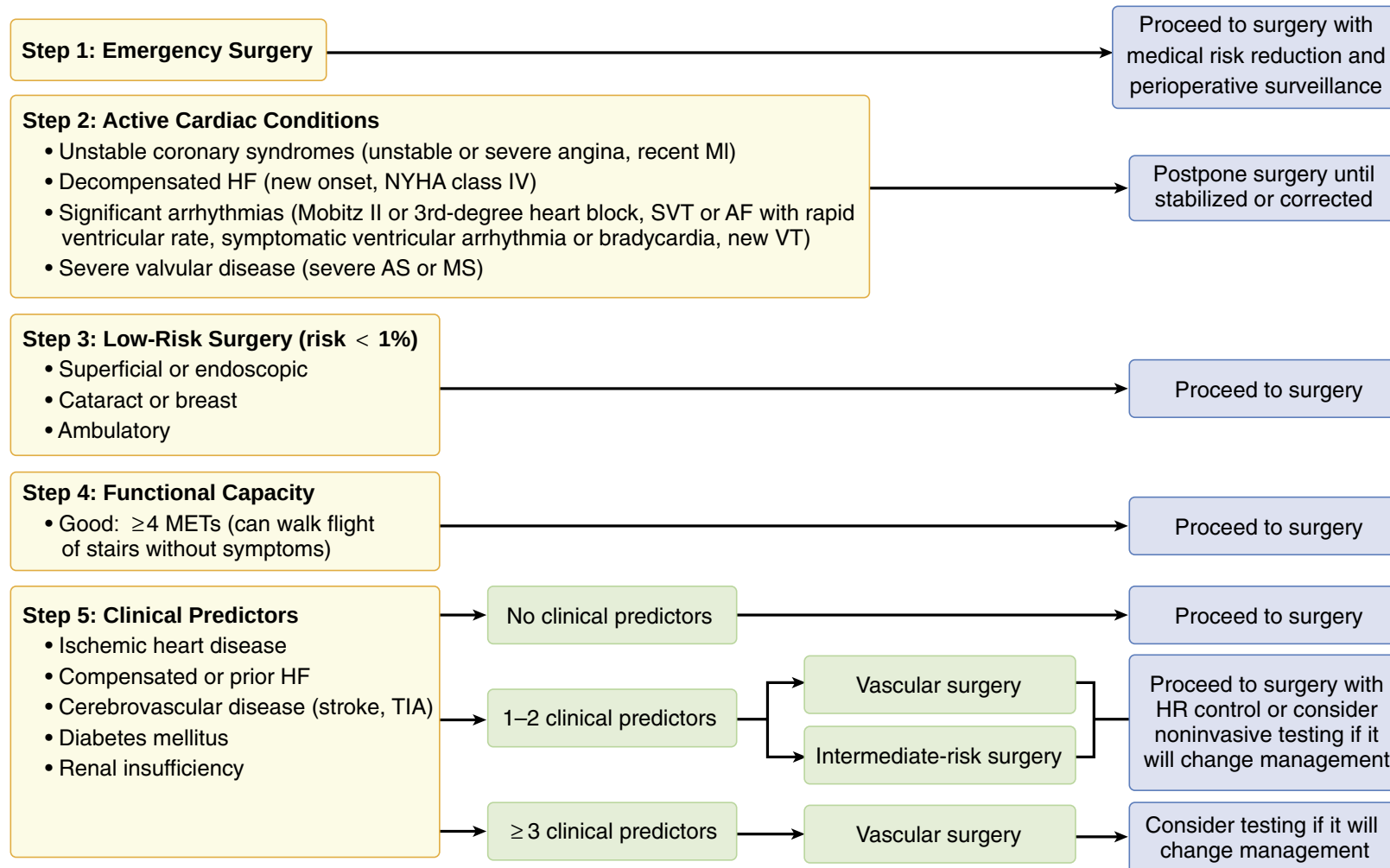


Figure 34-2 Simplified cardiac evaluation for noncardiac surgery. AF, atrial fibrillation; AS, aortic stenosis; HF, heart failure; HR, heart rate; METs, metabolic equivalents of the task; MI, myocardial infarction; MS, mitral stenosis; NYHA, New York Heart Association; SVT, supraventricular tachycardia; TIA, transient ischemic attack; VT, ventricular tachycardia. (From Fleisher LA, Beckman JA, Brown KA, et al: ACC/AHA 2007 guidelines on perioperative cardiovascular evaluation and care for noncardiac surgery. *J Am Coll Cardiol* 50:e159-e241, 2007. Available at <http://www.acc.org/qualityandscience/clinical/guidelines/Periop/>. Accessed September 28, 2007.)

Box 34-3 Recommendations for Preoperative Resting 12-Lead Electrocardiogram**Class I**

A preoperative resting 12-lead ECG is recommended for patients with at least one clinical risk factor* who are undergoing vascular surgical procedures

A preoperative resting 12-lead ECG is recommended for patients with known congestive heart failure, peripheral arterial disease, or cerebrovascular disease who are undergoing intermediate-risk surgical procedures

Class IIa

A preoperative resting 12-lead ECG is reasonable in persons with no clinical risk factors who are undergoing vascular surgical procedures

Class IIb

A preoperative resting 12-lead ECG may be reasonable in patients with at least one clinical risk factor who are undergoing intermediate-risk operative procedures

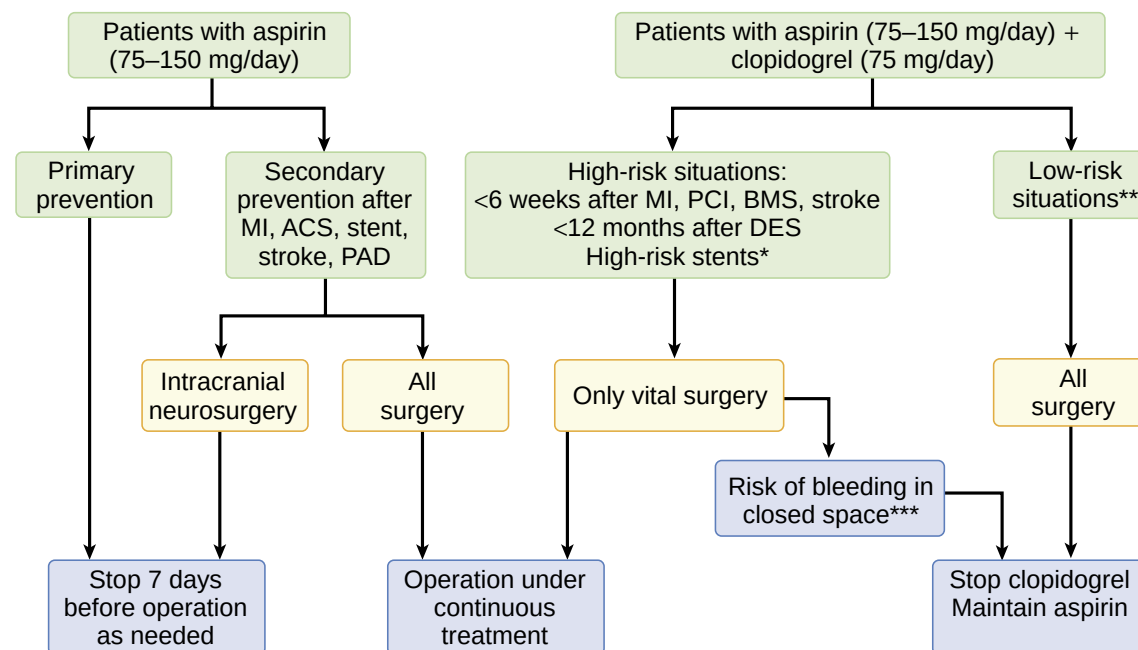
Class III

Preoperative and postoperative resting 12-lead ECGs are not indicated for asymptomatic persons undergoing low-risk surgical procedures

Class I recommendations: the procedure *should* be performed; class IIa: it is reasonable to perform the procedure; class IIb: the procedure may be considered; class III: the procedure *should not* be performed because it is not helpful.

*Clinical risk factors are listed in Box 34-2.

Adapted from Fleisher LA, Beckman JA, Brown KA, et al: ACC/AHA 2007 guidelines on perioperative cardiovascular evaluation and care for noncardiac surgery. J Am Coll Cardiol 50:e159-e241, 2007. Available at http://www.acc.org/qualityandscience/clinical/guidelines/Periop_Fulltext_2007.pdf Accessed September 28, 2007.



*High-risk stents: long (>36 mm), proximal, overlapping, or multiple stent implantation, stents for chronic total occlusions, stents in small vessels or bifurcated lesions.

**Examples of low-risk situations: >3 months after BMS, stroke, uncomplicated MI, PCI without stenting.

***Risk of bleeding in closed space: intracranial neurosurgery, intra-medullary canal surgery, posterior eye chamber ophthalmic surgery. In these situations, the risk/benefit ratio of upholding vs withdrawing aspirin must be evaluated for each case individually; in case of aspirin upholding, early postoperative re-institution is important.

Box 34-4 ACC/AHA Guideline Summary—Echocardiography in Asymptomatic Patients with Cardiac Murmurs*

Class I—There is evidence or general agreement (or both) that echocardiography is useful in asymptomatic patients with the following cardiac murmurs:

- Diastolic murmurs
- Continuous murmurs
- Late systolic murmurs
- Murmurs associated with ejection clicks
- Murmurs that radiate to the neck or back
- Grade 3 or louder systolic murmurs

Class IIa—The weight of evidence or opinion is in favor of the usefulness of echocardiography in asymptomatic patients with the following cardiac murmurs:

- Murmurs associated with other abnormal physical findings on cardiac examination
- Murmurs associated with an abnormal electrocardiogram or chest radiograph

Class III—There is evidence or general agreement (or both) that echocardiography is not useful in asymptomatic patients with the following murmurs:

- Grade 2 or softer midsystolic murmurs considered innocent or functional by an experienced observer

*These are indications for general evaluation but do not necessarily need to be performed preoperatively.

From Bonow, RO, Carabello, BA, Chatterjee, K, et al: ACC/AHA 2006 guidelines for the management of patients with valvular heart disease. A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to Revise the 1998 Guidelines for the Management of Patients with Valvular Heart Disease). J Am Coll Cardiol 48:e1, 2006.

Table 34-3 Descriptions of Murmurs Associated with Cardiac Abnormalities

Lesion	Location	Timing	Description
Aortic stenosis	Second parasternal interspaces	Midsystolic	Crescendo-decrescendo, radiates to the carotids; $\pm$ S ₃ , S ₄ ; Valsalva and sustained handgrip exercise decrease intensity
Aortic insufficiency	Third and fourth parasternal interspaces	Holodiastolic	Decrescendo, blowing, high pitched, radiates to the carotids; Austin-Flint rumble at the apex; squatting, handgrip exercise, and leaning forward increase intensity
Mitral stenosis	Apex	Mid-diastolic	Opening snap; low-pitched rumble radiates to the axilla; squatting and handgrip exercise increase intensity
Mitral regurgitation	Apex	Holosystolic	High pitched, blowing, radiates to the axilla; loud S ₃ ; standing decreases intensity; squatting and handgrip exercise increase intensity
Mitral valve prolapse	Apex	Late systolic	Crescendo, midsystolic click; Valsalva and standing increase intensity; squatting decreases intensity
Hypertrophic cardiomyopathy	Apex, lower left sternal border	Midsystolic	S ₄ , single S ₂ ; Valsalva and standing increase intensity; squatting, passive leg raising, and handgrip exercise decrease intensity

Table 34-4 Severity of Aortic Stenosis

Grade	Velocity of Aortic Jet (m/sec)	Mean Pressure Gradient (mm Hg)	Valve Area (cm ²)
Mild	<3	<25	≥1.5
Moderate	3-4	25-40	1.0-1.5
Severe	4-4.5	40-50	0.7-1.0
Critical	>4.5	>50	<0.7

Box 34-5 Indications for a Pacemaker**Class I Indications**

- Sinus bradycardia with symptoms clearly related to the bradycardia (usually with a heart rate <40 beats/min or frequent sinus pauses)
- Symptomatic chronotropic incompetence
- Complete (third-degree) AV block*
- Advanced second-degree AV block (block of ≥ 2 consecutive P waves)
- Symptomatic Mobitz I or II second-degree AV block
- Mobitz II second-degree AV block with a widened QRS or chronic bifascicular block, regardless of symptoms

Class II Indications

- Sinus bradycardia (heart rate <40 beats/min) with symptoms suggestive of bradycardia but without a clear association between bradycardia and symptoms
- Sinus node dysfunction with unexplained syncope
- Chronic heart rates <30 beats/min in an awake patient

Class I conditions are those in which permanent pacing is definitely beneficial and effective, provided that the condition is not due to a transient cause. Class II conditions are those in which permanent pacing may be indicated but there is conflicting evidence or divergence of opinion (or both).

*Controversy exists concerning complete atrioventricular (AV) block without symptoms. The current ACC/AHA guidelines classify asymptomatic third-degree AV block with average awake ventricular rates of 40 beats/min or greater as a class IIa indication, although others recommend definite pacemaker placement.

From Gregoratos, G, Abrams, J, Epstein, AE, et al: ACC/AHA/NASPE 2002 guideline update for implantation of cardiac pacemakers and antiarrhythmia devices: Summary article. A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (ACC/AHA/NASPE Committee to Update the 1998 Pacemaker Guidelines). Circulation 106:2145-2161, 2002.

Box 34-6 Classification of Pulmonary Hypertension**Pulmonary Arterial Hypertension**

Primary pulmonary hypertension

Sporadic

Familial

Associated with

Collagen vascular disease

Congenital shunts

Portal hypertension

Human immunodeficiency virus infection

Drugs/toxins

Persistent pulmonary hypertension of the newborn

Pulmonary Venous Hypertension

Left-sided heart disease

Extrinsic compression of central pulmonary veins

Pulmonary veno-occlusive disease

Pulmonary Hypertension Related to Lung Disease or Hypoxemia

Chronic obstructive pulmonary disease

Interstitial lung disease

Sleep-disordered breathing

Neonatal lung disease

Chronic exposure to high altitude

Pulmonary Hypertension Caused by Chronic Thromboembolic Disease

Pulmonary thrombosis or embolism

Sickle cell disease

Pulmonary Hypertension from Disorders Directly Affecting the Pulmonary Vasculature

Schistosomiasis

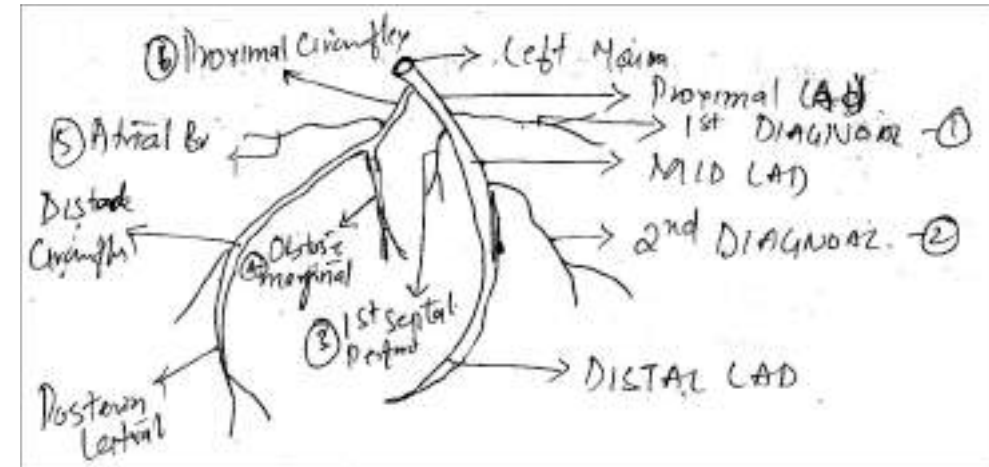
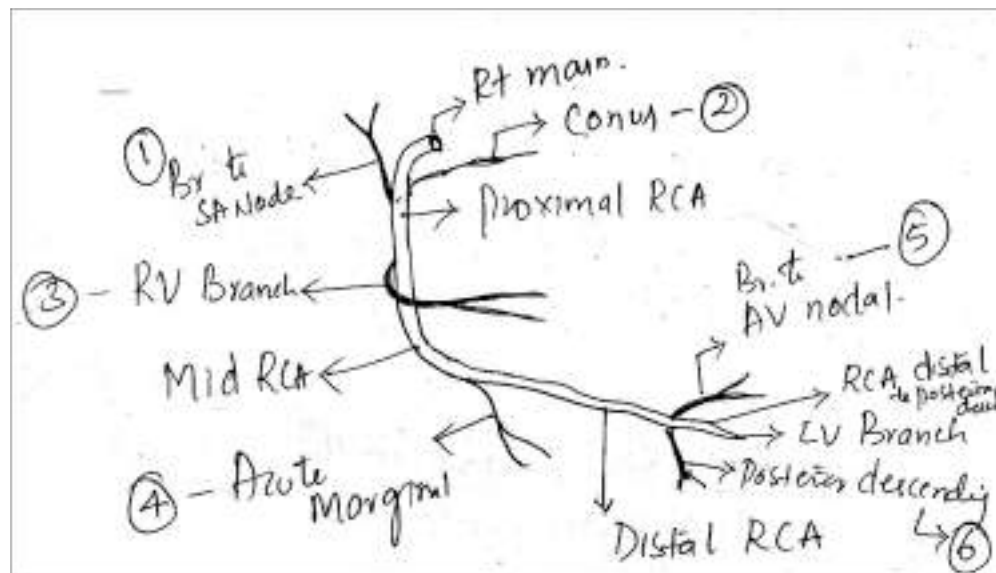
Sarcoidosis

Available from <http://www.who.int/ncd/cvd/pph.html>.

Table 34-5 Child-Turcotte-Pugh Classification

Parameter	Points (For Each with a Maximum of 15)		
	1	2	3
Ascites	Absent	Slight	Moderate
Bilirubin (mg/dL)	<2	2-3	>3
Albumin (g/dL)	>3.5	2.8-3.5	<2.8
Prothrombin time (seconds over control)	<4	4-6	>6
Encephalopathy	None	Grade 1-2	Grade 3-4

Class A: <7 points; Class B: 7-9 points; Class C: >9 points.

**Left Coronary Artery****Right Coronary Artery**

4. Classify cardiomyopathies. Describe management of 60 year old male with dilated cardiomyopathy scheduled for laparotomy

Classification of cardiomyopathies on morphologic and hemodynamic basis



Parameter	Dilated	Restrictive	Hypertrophic	Obliterative
Morphology	Biventricular dilation	Decreased ventricular compliance	Hypertrophy of left ventricle and usually interventricular septum	Thickened endocardium or mural thrombi
Ventricular volume	Marked increase	Normal to moderate increase	Normal to moderate decrease	Moderate decrease
Ejection fraction	Marked decrease	Normal to Moderate decrease	Marked increase	Normal to moderate decrease
Ventricular compliance	Normal to moderate decrease	Marked decrease	Marked decrease	Marked decrease
Ventricular filling pressure	Marked increase	Marked increase	Normal to moderate increase	Moderate increase
Stroke volume	Marked decrease	Normal to moderate decrease	Normal to moderate increase	Normal to moderate decrease

Definition

Cardiomyopathies are a heterogeneous group of diseases of the myocardium associated with mechanical and/or electrical dysfunction that usually (but not invariably) exhibit inappropriate ventricular hypertrophy or dilation and are due to a variety of causes that frequently are genetic.

Goldman's Cardiac Risk Index:

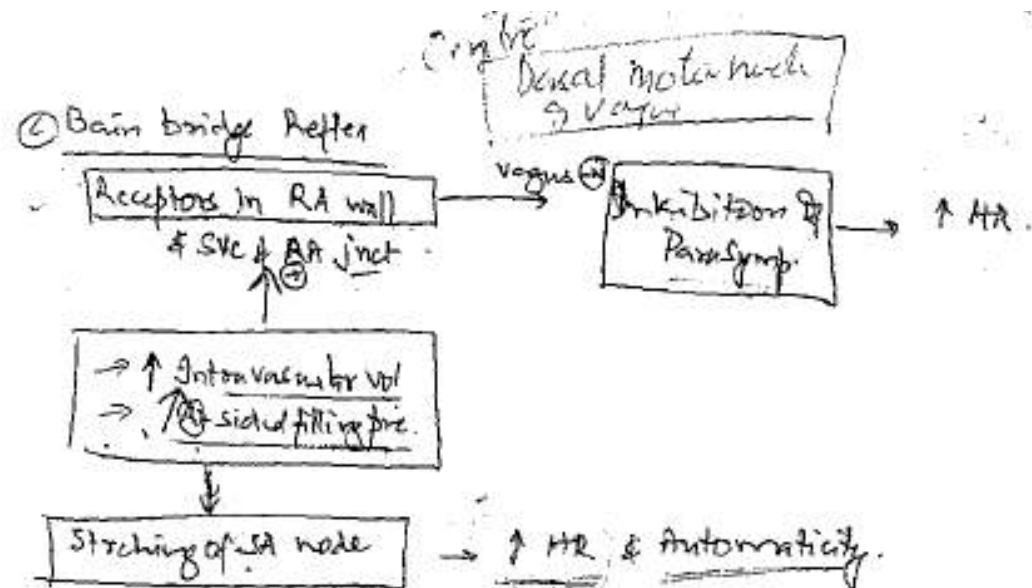
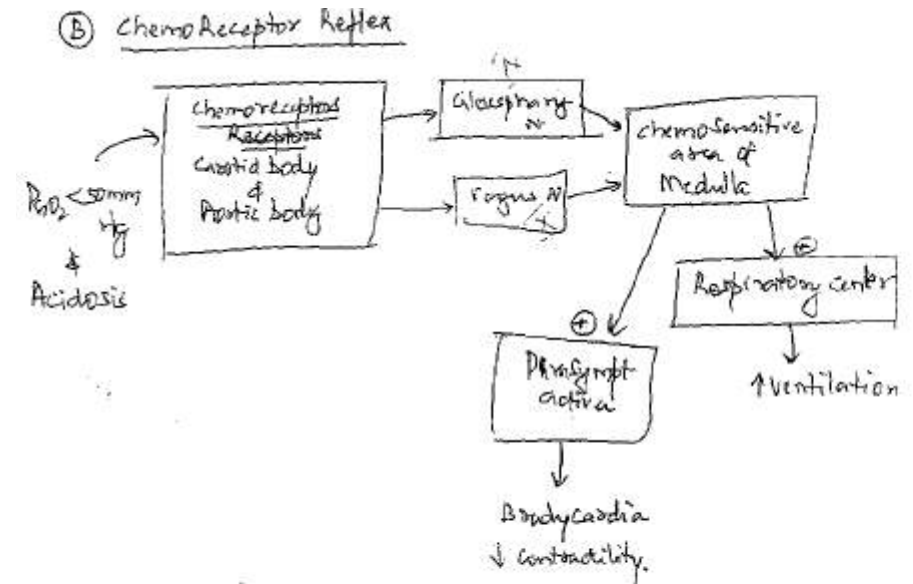
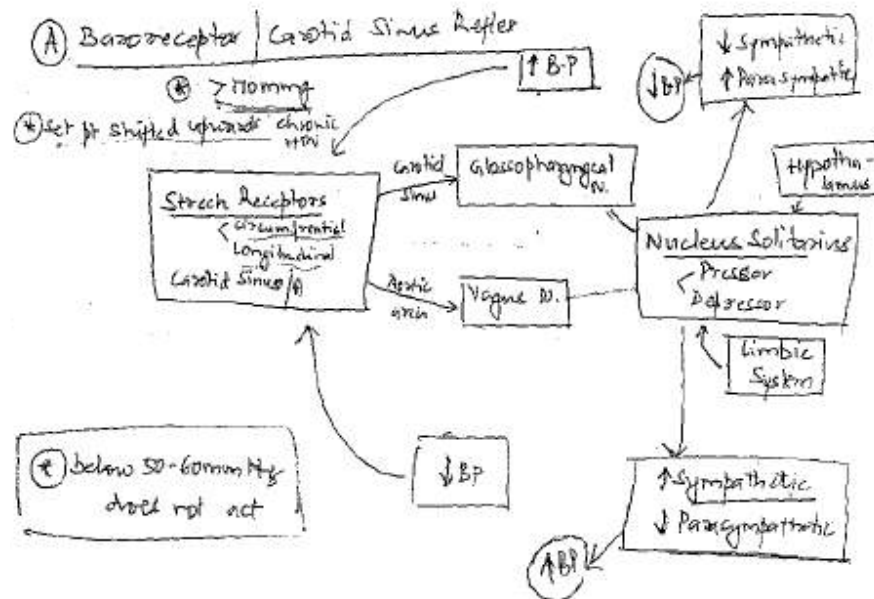
i.	Age > years	5
ii.	MI in previous 6 months	10
iii.	S3 gallop or ↑ JVP (0-3 cm)	11
iv.	Important aortic stenosis	3
v.	Rhythm other than sinus, Pac on last preoperative	7
vi.	>5 PVC/min at any time before	7
vii.	PaO ₂ , < 60 or PaCO ₂ > 50mm Hg, K<3.00 mEq/L or HCO ₃ < 20 mEq/l BUN > 50 or Cr> 3 mg/dl. Abnormal SGOT, signs of chronic liver disease, bed ridden from non-cardiac cause	3
viii.	Intraperitoneal, Intrathoracic, aortic surgery	3
ix.	Emergency operation	4
	Total	53

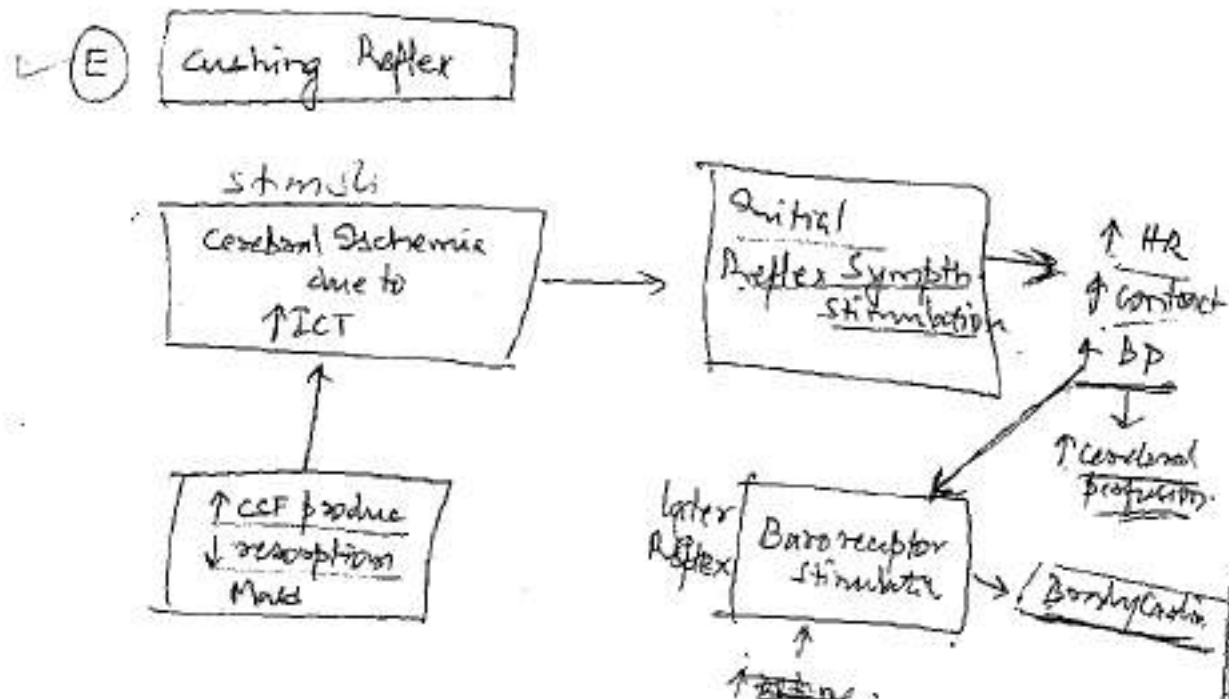
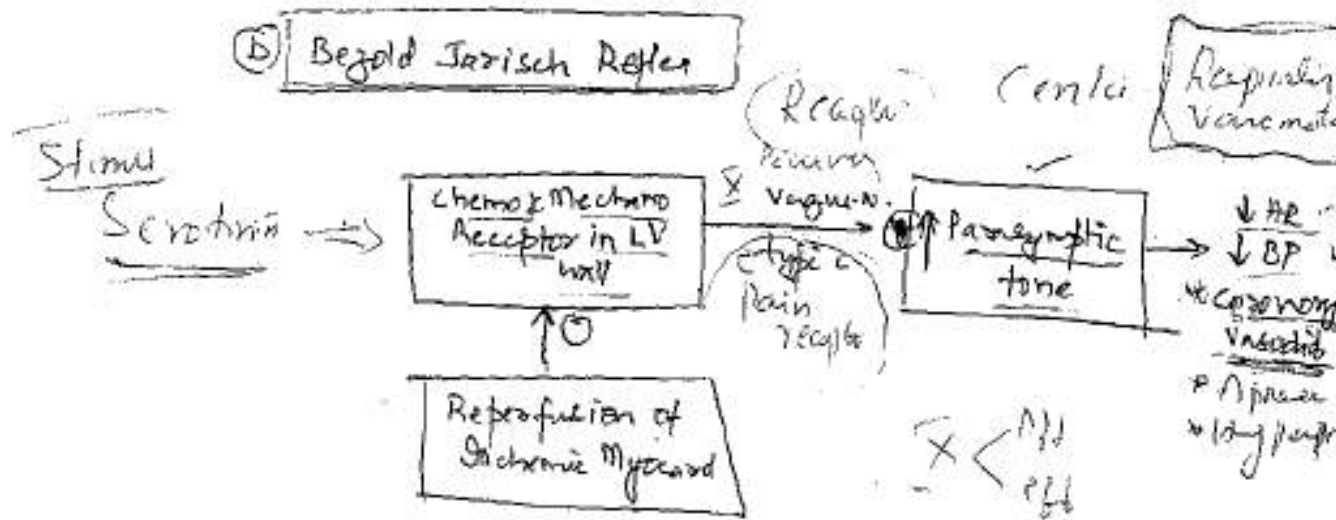
Detsky's Modified Cardiac Risk Index- 1986

i.	Age > 70 years	5
ii.	MI < 6 months	10
iii.	MI > 6 months	5
iv.	Unstable angina	10
v.	Pulmonary edema < 1 week	10
vi.	Pulmonary edema in the past	5
vii.	Non sinus rhythm PAC	5
viii.	CCVSA class III	10
ix.	CCVSA class IV	20
x.	Critical As	20
xi.	Emergency operation	5
xii.	Poor general status	5
	Total	120

	(Assign one point to each of the following variables)
i.	High risk type of surgery (intra-peritoneal, intra-thoracic or suprainguinal vascular procedures.
ii.	History of IHD (history of MI, positive exercise test, current complaint of ischemic chest pain or use of nitrate therapy of ECG with Q waves)
iii.	History of CHF
iv.	History of cerebro-vascular disease
v.	Insulin therapy for diabetes
vi.	Preoperative serum creatinine > 2.0 mg/gl Class I = 0 factors, Class II = 1 factor Class III = 2 factors Class IV = >2 factors

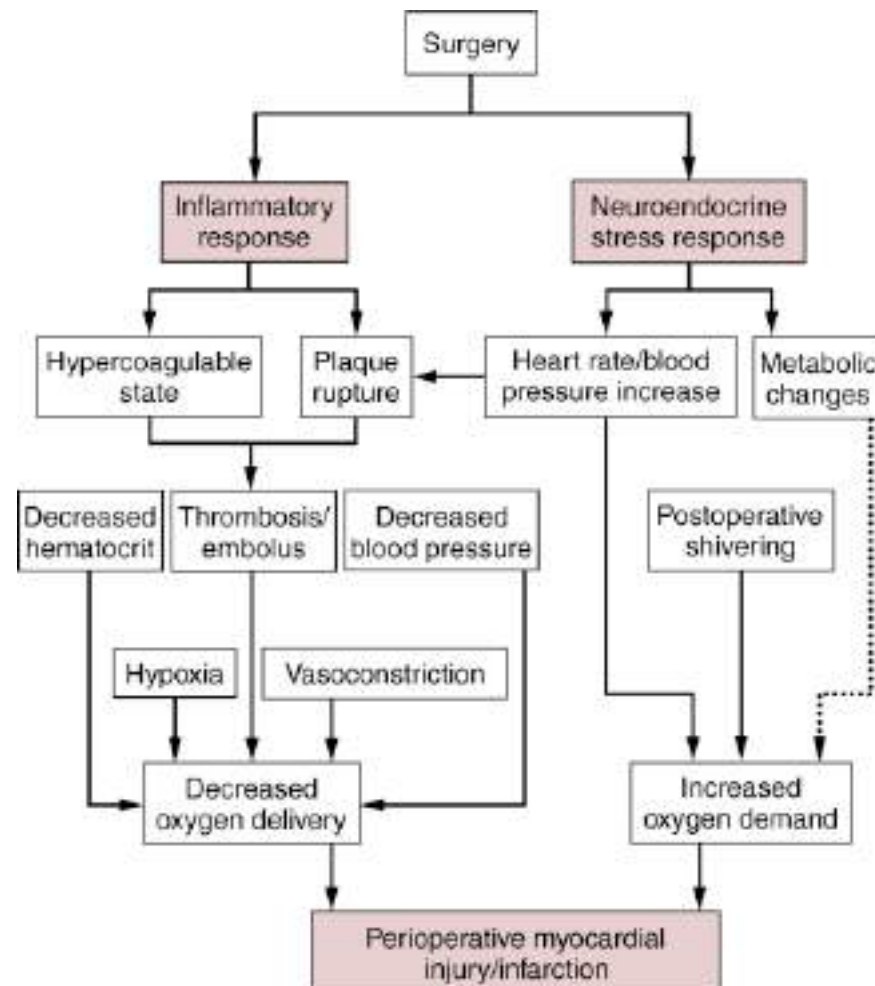
Lee's revised Cardiac Risk Index-- 1999.





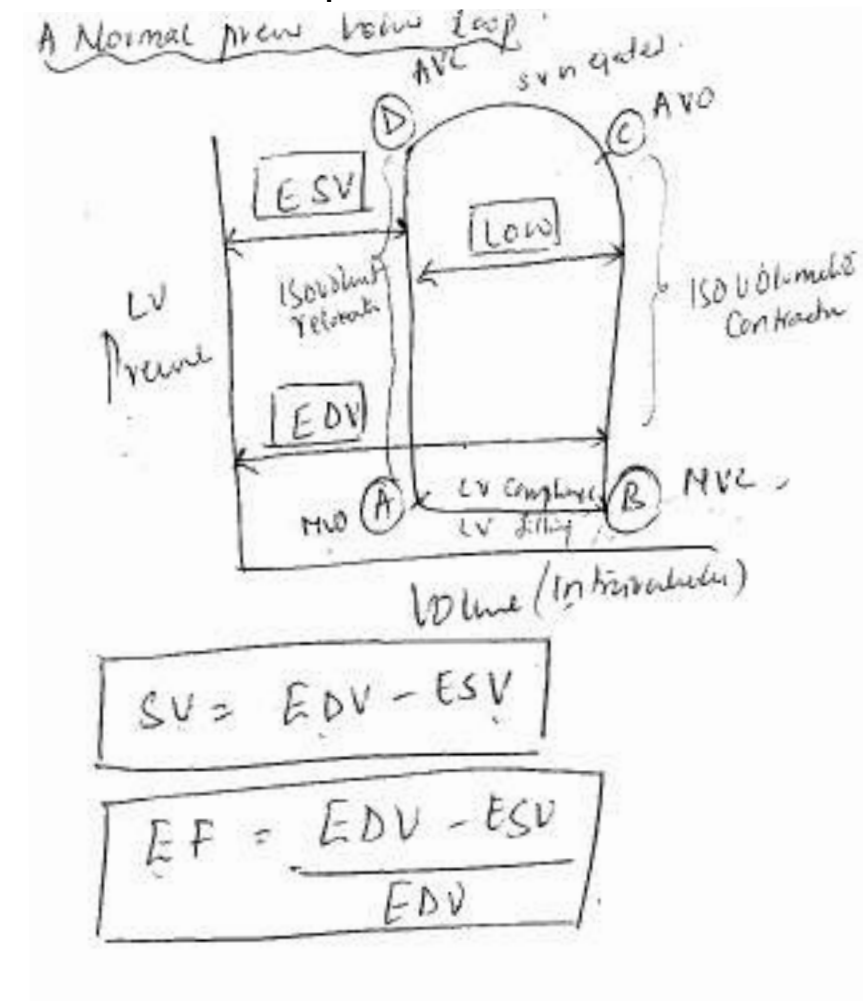
Reflex	Stimuli	Receptor	Afferents	Centre	Efferents	Response
Baroreceptor Reflex	Increase BP	Carotid Sinus & Aortic arch	IX Nerve(carotid Sinus) & X Nerve (Aortic Arch)	Nucleus of Tractus Solitarius in Medulla	IML Tract	Decrease in Blood pressure, Decreases Sympathetic stimulation, Heart Rate & SV
Chemoreceptor Reflex	pH pCO ₂ & pO ₂	Carotid & Aortic body	IX Nerve & X Nerve	Chemosensitive area of Medulla	X Nerve	Bradycardia, Increase Blood pressure & Hyperventilation
Bain Bridge Reflex	Increase filling of right atrium	Stretch receptor in right atrial wall	X Nerve	Dorsal motor nucleus of vagus	Vagal & sympathetic fibres	Increase in Heart Rate & Automaticity
Benzold Jarisch reflex	serotonin	Pain receptor C Fibre	X Nerve	Respiratory & Vasomotor Centre	X Nerve	Apnea, Hyperapnea & bradycardia, Coronary Vasodilation & Hypotension.
Oculo-cardiac Reflex	Pressure on the Eye, Traction on the muscles	Stretch receptor with in extraocular muscles.	Long & Short ciliary nerve & Ophthalmic division	Cillary ganglion & Gasserian ganglion	Vagal & Cardiodepressor fibre	Reflex Bradycardia

Diagrams & Flow Chart



Factors that can contribute to perioperative myocardial infarction.

Pressure Volume Loop:



Surgical Factors:

Major	Intermediate	Minor
Emergency Surgery	Carotid endarterectomy	cataract
Major vessel/surgery	Intrathoracic/Peritoneal surgery	Superficial surgery
Surgeries with major blood loss or fluid shifts	Head & Neck Surgery	Breast surgery
	Open prostatic surgery	

Clinical Predictors:**Patient Factors:**

Major	Intermediate	Minor
Acute MI (< 7days), Recent (7 to 30 Days)	Previous MI	Age > 65 Years
Decompensated CHF	Pathological Q-Waves	Poor functional state
Symptomatic arrhythmias	Anginal Class I & II	Uncontrolled HTN
Severe Valvular Heart disease.	Diabetes	
	CRF	

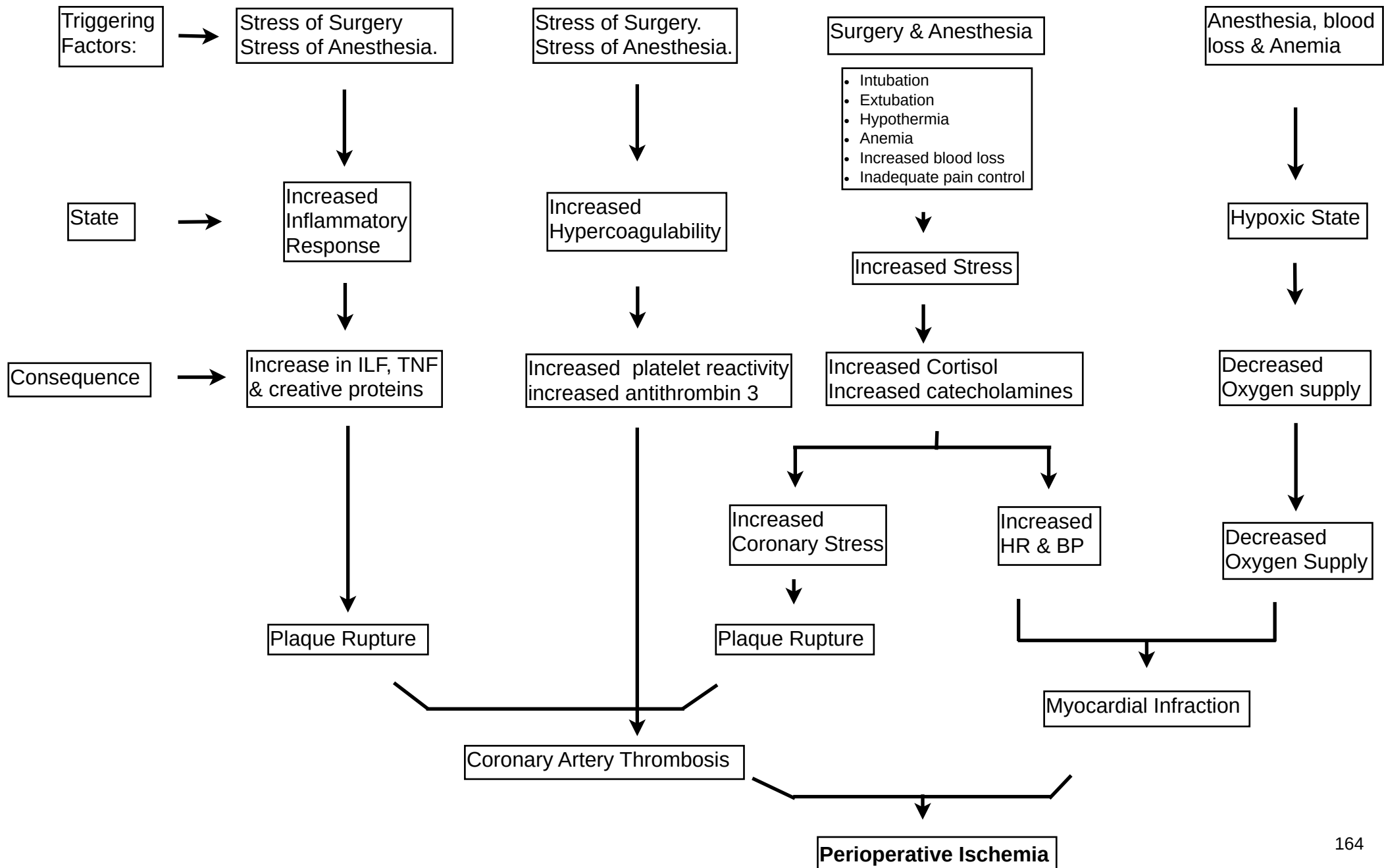
Cardioplegic solution:

A number of different solutions are described. Most common compounds are:

- Potassium = 15-40 mEq/l
- Sodium = 100-120 mEq/l
- Chloride = 110-120 mEq/l
- Calcium = 0.7 mEq/l
- Magnesium = 15 mEq/l
- Glucose = 28 mmol/l
- Bicarbonate = 27 mmol/l

St. Thomas hospital solution (supplied in 20ml ampoules):

- $\text{MgCl}_2 \cdot 6\text{H}_2\text{O}$ = 16 mmol
- KCl = 16 mmol
- Procaine 272.8mg
- Water to 20ml
- This ampoule is added to 1 liter of non-lactated Ringer's solution at 4°C and used.

Perioperative Myocardial Ischemia. Continuation:**Pathophysiology:**

Acute treatments for suspected intraoperative myocardial ischemia

	Associated Hemodynamic Finding	Therapy	Dosage
I	Hypertension, tachycardia +	Deepen anesthesia IV β - Blockade	Esmolol 2-100 mg, + 50-200 Metoprolol 0.5-2.5 mg Labetalol 2.5 – 10mg 33-330 μ g/min +
II	Normotension, tachycardia +	IV Nitroglycerin Assure adequate anesthesia change anesthetic regimen IV β - Blockade	As above
III	Hypertension, normal heart rate	Deepen anesthesia nicardipine	0-20 mg Nicardipine 1-5 mg, + 1-10 μ Phenylephrine 25-100 μ g
IV.	Hypotension, tachycardia +	IV α -agonist	Nor-epinephrine 2-4 μ g
V	Hypotension, bradycardia	Lighten anesthesia IV Ephedrine IV Epinephrine IV Atropine IV Nitroglycerin when normotensive	5-10 mg 4-8 μ g 0.3-0.6 mg As above
VI.	Hypotension, normal heart rate	IV α - Agonist IV Epinephrine alter anesthesia (eg, lighten) IV Nitroglycerin when normotensive	As above
VII.	No abnormality	IV Nitroglycerin IV Nicardipine	As above As above

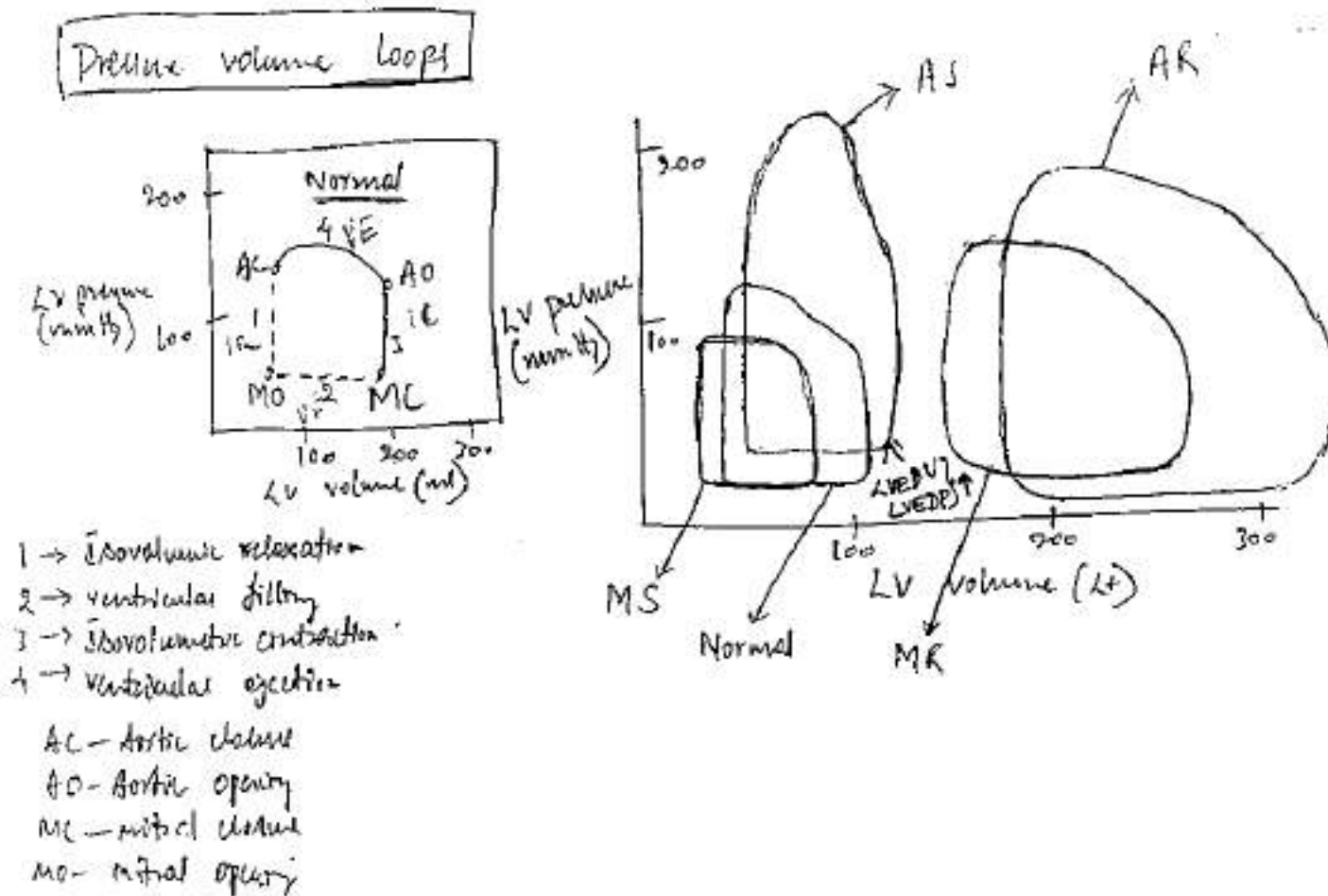


Table						
Drug	Site of Action	Route	Plasma half life	Excretion	Antidote	Stop Before procedure
Heparin	Ila / Xa	IV / SC	1.5 hrs	Hepatic	Protamine	6 hrs
LMWH	Xa	SC	4.5 hrs	Renal	Protamine (Partial reversal)	12 – 24 hr
Bivalirudin	Ila	IV	25 min	Hepatic	None	2- 3 hrs
Argatroban	Ila	IV	45 min	Hepatic	None	4 – 6 hrs
Warfarin	Vit –k	Oral	2 – 4 days	Hepatic	Vit – k, FFP	2 – 4 days

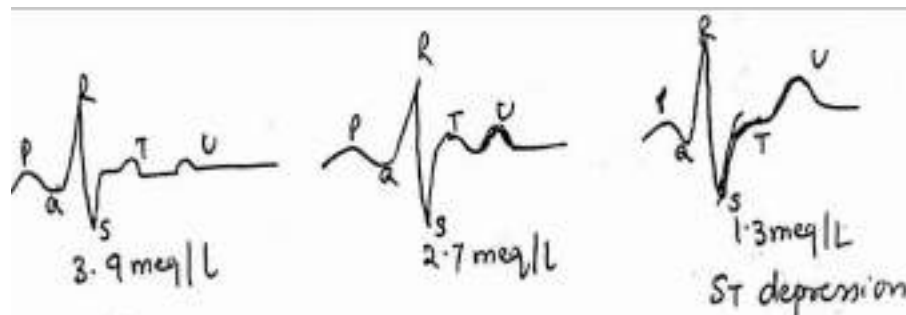

Hypokalemia

Table 7.1 Effects of the sympathetic and parasympathetic nervous systems on peripheral effector organs, and receptor subtypes mediating these functions (where known). All postganglionic parasympathetic fibres are muscarinic (M), but in many sites the subtype has not been identified.

Organ	Sympathetic		Parasympathetic	
	Receptor subtype	Effect	Receptor subtype	Effect
Heart	β_1 also β_2 , γ also α and DA_1	$\uparrow$ Heart rate $\uparrow$ Force of contraction $\uparrow$ Conduction velocity $\uparrow$ Automaticity (β_2) $\uparrow$ Excitability $\uparrow$ Force of contraction	M_2	$\downarrow$ Heart rate $\downarrow$ Force of contraction Slight $\downarrow$ conduction velocity
Arteries	β_1 β_2 α_1, α_2 DA_1, β_2	Coronary vasodilatation Vasodilatation (skeletal muscle) Vasoconstriction (coronary, pulmonary, renal and splanchnic circulations, skin and skeletal muscle) Splanchnic and renal vasodilatation	M^a	Vasodilatation in skin, skeletal muscle, pulmonary and coronary circulations
Veins	α_1 , also α_2 β_2	Vasoconstriction Vasodilatation		
Lung	β_2 α_1	Bronchodilatation Inhibition of secretions Bronchoconstriction	M_3	Bronchoconstriction Stimulation of secretions
GI tract	$\alpha_1, \alpha_2, \beta_2$ α_1, α_2	Decreased motility Contraction of sphincters Inhibition of secretions	M	Increased motility Relaxation of sphincters Stimulation of secretions
Pancreas	β_2 α_1, α_2	Increased insulin release Decreased insulin release		
Kidney	β	Renin secretion		
Liver	β_2, α β_2, γ	Glycogenolysis Gluconeogenesis	M	Glycogen synthesis
Bladder	β_2 α	Detrusor relaxation Sphincter contraction	M	Detrusor contraction Sphincter relaxation
Uterus	α_1 β_2	Myometrial contraction Myometrial relaxation		
Adipocytes	β_1	Lipolysis		
Eye	α_1	Mydriasis (radial muscle contraction) Ciliary muscle relaxation for far vision	M	Miosis Ciliary muscle contraction for near vision
Platelets	α_2	Promote platelet aggregation		
Sweat glands	M^b	Sweating		

^aMuscarinic receptors are present on vascular smooth muscle, but they are independent of parasympathetic innervation and have little or no physiological role in the control of vasomotor tone.

^bSympathetic cholinergic fibres supply sweat glands and arterioles in some sites.

Table 2E-1. Receptor Sites and Action

Receptor Site	Action
$\alpha-1$	Vasoconstricts vascular smooth muscle, GU contraction, GI relaxation, gluconeogenesis, glycogenolysis
$\alpha-2$	Decreased insulin secretion, causes platelet aggregation; decreased NE release, vasoconstriction of vascular smooth muscle
$\beta-1$	Increased cardiac contractility, heart rate, AV conduction; increased renin secretion; increased contractility and arrhythmias
$\beta-2$	Relaxation of vascular smooth muscle; bronchial relaxation, GI & GU relaxation, gluconeogenesis, glycogenolysis
D-1	Dilation of vascular smooth muscle (renal, mesentery, coronary; renal tubules (natriuresis, diuresis), juxtaglomerular cells (increased renin release)
D-2	Inhibits NE release, may constrict renal and mesenteric smooth muscle

Table 2E-2. Receptor Activity of Adrenergic Agonists

Drug	Direct	Indirect	$\alpha-1$ (Arterial)	$\alpha-1$ (Venous)	$\alpha-2$	$\beta-1$	$\beta-2$
Phenylephrine	+++	-	+++	+++++	+,0	0	0
Norepinephrine	+++	-	+++	---	+++++	+++	0
Epinephrine ₁	+++	-	+	+	+	+++	+++
Epinephrine ₂	+++	-	+++	+++	++	+++	+++
Epinephrine ₃	+++	-	+++++	+++++	+++	+++	+++
Ephedrine	+	+++	++	+++	?	+++	++
Dopamine ₁	+++	-	0	+++	Dopaminergic agonist		
Dopamine ₂	+++	-	+	+++	?	+++	+++
Dopamine ₃	+++	+	+++	+++	?	+++	+++
Dobutamine	+++	-	0	?	0	+++	++
Isoproterenol	+++	-	0	0	0	+++++	+++++

Fluoroquinolones

Antibiotic	Normal dose	Dose in renal failure (by GFR)		
		>50	10-50	<10
1st generation				
Nalidixic acid	1 g PO qid	n/a	n/a	n/a
2nd generation				
Ciprofloxacin	500-750 mg PO q12h 200-400 mg IV q12h	NC	250-500 mg q12h	250-500 mg q24h
Lomefloxacin	400 mg PO qd	NC	200-400 mg qd	200 mg qd
Norfloxacin	400 mg PO q12h	NC	400 mg q12-24h	400 mg q24h
Ofloxacin	200-400 mg PO/IV q12h	NC	400 mg q24h	200 mg q24h
3rd generation				
Levofloxacin	250-500 mg PO/IV q24h	NC	250 mg q24h	250 mg q48h
4th generation				
Gatifloxacin	400 mg PO/IV qd	NC	200 mg qd	200 mg qd
Gemifloxacin	320 mg PO qd	NC	160 mg qd	
Moxifloxacin	400 mg PO qd	NC	NC	NC

Tetracyclines

Antibiotic	Normal dose	Dose in renal failure (by GFR)		
		>50	10-50	<10
Doxycycline	100 mg PO/IV q12-24h	NC	NC	NC
Tigecycline	100 mg IV $\times$ 1 then 50 mg q12h	NC	NC	NC

Macrolides

Antibiotic	Normal dose	Dose in renal failure (by GFR)		
		>50	10-50	<10
Azithromycin	500 mg IV qd 500 mg PO on d 1, then 250 mg PO qd	NC	NC	NC
Clarithromycin	250-500 mg PO bid	?	?	?
Erythromycin	0.5-1 g IV q6h 250-500 mg PO qid	NC	NC	250-500 mg IV or 250 mg PO q6h
Telithromycin ^a	800 mg PO qd	NC	600 mg qd (if CrCl < 30)	

Cephalosporins				
Antibiotic	Normal dose	Dose in renal failure (by GFR)		
		>50	10-50	<10
1st generation				
Cefadroxil	0.5-1 g PO q12h	NC	0.5 g q12-24h	0.5 g q36h
Cefazolin	1 g IM/IV q8h	NC	1 g q12h	1 g q24h
Cephalexin	250-500 mg PO q6h	NC	NC	NC
2nd generation				
Cefaclor	250-500 mg PO q8h	NC	NC	NC
Cefotetan	1-2 g IM/IV q12h	NC	1-2 g q24h	1 g q24h
Cefoxitin	1-2 g IM/IV q4h	1-2 g q6h	1-2 g q8h	1 g q12h
Cefprozil	250-500 mg PO q12-24h	NC	NC	250 mg q12h
Cefuroxime	750-1500 mg IM/IV q6h	NC	750-1500 mg q8h	750 mg q24h
Loracarbef	200-400 mg PO q12h	NC	200 mg q12h	200 mg q3-5d
3rd generation				
Cefdinir	600 mg PO qd	NC	NC	300 mg qd
Cefditoren	200-400 mg PO bid	NC	200 mg bid	200 mg qd
Cefixime	400 mg PO q24h	NC	300 mg q24h	200 mg q24h
Cefoperazone	1-3 g IV q8h	NC	NC	NC
Cefotaxime	1-2 g IM/IV q6h	NC	NC	1-2 g q12h
Cefpodoxime	100-400 mg PO q12h	NC	NC	400 mg q24h
Ceftazidime	1-2 g IV q8h	NC	1-2 g q12h	1 g q24h
Ceftributen	400 mg PO qd	NC	200 mg qd	100 mg qd
Ceftizoxime	1-2 g IV q6h	NC	1 g q12h	0.5 g q12h
Ceftriaxone	1-2 g IM/IV q12-24h	NC	NC	NC
4th generation				
Cefepime	1-2 g IM/IV q12h	NC	1-2 g q16-24h	1-2 g q24-48h

Aminoglycosides				
Antibiotic	Normal dose	Dose in renal failure (by GFR)		
		>50	10-50	<10
Gentamicin		60-90% q8-12h	30-70% q12-18h	20-30% q24-48h
Tobramycin	1-1.7 mg/kg q8h	or -- 1-1.7 mg/kg q(8 ~ serum Cr)h		
Amikacin	5 mg/kg q8h	60-90% q8-12h	30-70% q12-18h	20-30% q24-48h
		or -- .5 mg/kg q(8 ~ serum Cr)h		

Properties of Compressed Gases in E Cylinders

Characteristic	Oxygen	Nitrous Oxide	Air
Color	Green	Blue	Yellow
Physical state	Gas	Liquid and gas	Gas
Volume (L)	660	1590	625
Pressure full (psi)	2200	750	1800

Macroshock

(current applied outside body)

- 1 milliamp - perception
- 10 milliamps - one would let go
- 100 milliamps - cause V-fib

Microshock

(current applied inside body)

- 10 microamps - max leakage current
- 100 microamps - cause V-fib

Causes of Increased Peak Inspiratory Airway Pressure (PIP)**↑ PIP & ↑ Plateau Pressure**

- Tidal volume
- Pulmonary compliance
- Mechanical causes:
 - Abdominal gas insufflation
 - Trendelenburg position
 - Abdominal packing
 - Endobronchial intubation
- Physiologic causes:
 - Pulmonary edema
 - Pleural effusion
 - Ascites
 - Tension pneumothorax

↑ PIP & Unchanged Plateau Pressure

- ↑ Inspiratory gas flow rate
- ↑ Airway resistance
- Mechanical causes:
 - Kinked endotracheal tube
 - Airway compression
 - Foreign body aspiration
- Physiologic causes:
 - Bronchospasm
 - Secretions

Independent Risk Factors for Difficult Mask Ventilation

- Presence of a beard
- Body mass index $>26 \text{ kg/m}^2$
- Lack of teeth
- Age >55
- History of snoring

Oral Tracheal Tube Sizing

Age	Internal Diameter (mm)	Tube Length at Lip (cm)
Full-term infant	3.5	12
Child	4 - age/4	14 + age/2
Adult: female	7.0-7.5	20
male	7.5-8.5	22

Practical Approach to Unanticipated Difficult Airway**Plan A**

- Standard laryngoscopy with blade of choice
- If unable to intubate → make 2nd attempt with a different blade
- Make no more than 2 attempts (avoid ↑ risk of oral bleeding, secretions & edema)

Plan B

- Direct laryngoscopy & insertion of bougie or intubating catheter
- Confirm placement by (1) using hand on anterior neck to palpate catheter advancement through glottis; (2) after this, 40 cm of catheter should reach carina & provide resistance (no resistance will be encountered if in esophagus); (3) if using intubating catheter, may attach to EtCO₂ monitor

Plan C

- Insertion of LMA (disposable, Fastrach™, Proseal™)
- 5.0 or 6.0 ETT will fit through disposable LMA (- fiberoptic assistance)

Plan D

- Terminate anesthetic & awaken patient
- Perform awake fiberoptic intubation
- Perform surgical airway (i.e., tracheostomy)

Source: Adapted from: Morgan GE, Mikhail MS, Murray MM. Clinical Anesthesiology, 4th ed.

Nerve Blocks to Anesthetize the Airway

Topical anesthesia for tongue/oropharynx

- Cetacaine spray (tetracaine/benzocaine combination)
 - benzocaine toxicity occurs at ~100 mg; can lead to methemoglobinemia (tx with methylene blue)
- Viscous lidocaine: 2–4 mL swish & swallow
- Nebulized lidocaine: 4%, 4 mL for 5–10 min (or atomizer)
- Lidocaine jelly: 2% on tongue blade, peaks in 5–10 min

Superior laryngeal nerve block (sensory innervation to epiglottis, arytenoids, & vocal cords)

- Laterally displace hyoid bone toward block side, direct 22-gauge needle to lateral portion of hyoid bone
- Withdraw slightly & walk off bone inferiorly (below each greater cornu)
- Advance through thyrohyoid membrane (may feel loss of resistance)
- Aspirate & inject 2 mL of 2% lidocaine superficial & deep to membrane

Translaryngeal block (recurrent laryngeal nerve)

- Penetrate cricothyroid membrane with a 22-gauge plastic up catheter and 10 mL syringe
- After aspirating air, remove needle & attach a syringe with 4 mL of 4% lidocaine
- Inject at end of expiration to anesthetize glottis & upper trachea

Recurrent laryngeal nerve block

- Aim for ipsilateral lesser cornu of thyroid cartilage in tracheoesophageal groove
- Insert needle perpendicular to pt directing it medially, contacting lesser cornu of thyroid cartilage
- Once reached, withdraw needle slightly & inject

Glossopharyngeal nerve block (posterior third of tongue)

- Inject 2 mL 1%–2% lidocaine in glossopharyngeal arch

Drug Class/ Drug	Adult Dose (mg)	Onset/Peak (min)	Notes
Sodium citrate (PO)	10–20 mL		
Gastric motility stimulator			
Metoclopramide (PO, IM, IV)	5–20		
Antiemetics			
Ondansetron (IV)	4–8 mcg/kg		
Granisetron (IV)	3 mcg/kg		

Drug Class/ Drug	Adult Dose (mg)	Onset/Peak (min)	Notes
Benzodiazepines —anxiolysis, sedation, & amnesia (no guarantee against recall, no analgesia)			
Diazepam (PO)	5–20	30–60 in adults, 15–30 in children	Crosses placenta; highly protein-bound (↑ potency in pt with ↓ albumin)
Lorazepam (IM)	3–7	30–40	Among benzos, lorazepam has most delayed onset & longest action (may cause prolonged sedation)
Lorazepam (IV)	Titrates 1–2.5 mg doses		
Midazolam (IM)	0.05–0.1 mg/kg	5–10, 30–60	Rapid onset & short duration; given within 1 hr of surgery
Midazolam (IV)	Titrates 1–2.5 mg	1–2	
Barbituates —main advantage: cost; primarily induces sedation; no analgesia, may cause disinhibition; little cardiorespiratory depression at usual premed doses			
Secobarbital (PO, IM)	50–200	60–90	Sedation for 4 hr; performance may be impaired for 10–22 hr
Pentobarbital (PO, IM)	50–200		Prolonged action; not appropriate for short procedures
Opioids —treat pain assoc. with preop experience (regional anesthesia, central lines); provide little anxiolysis, may cause dysphoria; consider supplemental O ₂			
Morphine (IM, IV)	5–15	15–30, 45–90	Lasts 4 hr
Meperidine (IM)	50–150	Unpredictable	Lasts 2–4 hr
Antihistamines			
Diphenhydramine (PO, IM)	25–75		Sedation; can use with cimetidine & steroids to protect against histamine release from allergic rxns
Anticholinergics —useful for drying oral secretions (oral surgery/fiberoptic intubation)			
Atropine (IM)	0.3–0.6		
Scopolamine (IM)	0.3–0.6		
Glycopyrrolate (IM)	0–0.3		Least sedating agent (does not cross blood–brain barrier)
H₂ antagonists			
Cimetidine (PO, IM, IV)	300		
Ranitidine (PO)	50–200		
Famotidine (PO)	20–40		
Antacids			

Continuum of Depth of Sedation (ASA Definition)

	Minimal Sedation (Anxiolysis)	Moderate Sedation/ Analgesia ("Conscious Sedation")	Deep Sedation/ Analgesia	General Anesthesia
Responsiveness	Normal response to verbal stimulation	Purposeful response to verbal or tactile stimulation	Purposeful response following repeated or painful stimulation	Unarousable even with painful stimulus
Airway	Unaffected	No intervention required	Intervention may be required	Intervention often required
Spontaneous Ventilation	Unaffected	Adequate	May be inadequate	Frequently inadequate
Cardiovascular Function	Unaffected	Usually maintained	Usually maintained	May be impaired

Reflex withdrawal from a painful stimulus is **NOT considered a purposeful response

ASA Physical Status Classification**Example**

I	No organic/physiologic/psychiatric problems	Healthy patient
II	Controlled medical conditions with mild systemic effects, no limitations in functional ability	Controlled HTN/ smoker/obesity
III	Medical conditions with severe systemic effects, limitations in functional ability	Controlled CHF/stable angina/morbid obesity/COPD/chronic renal insuff.
IV	Poorly controlled medical conditions associated with significant impairment in functional ability that is potential threat to life	Unstable angina/ symptomatic COPD or CHF
V	Critical condition, little chance of survival without surgical procedure	Ruptured AAA
VI	Brain dead, undergoing organ donation	
E	Emergency, trauma	Gunshot wound, GI perf.

Commonly Used Drugs for Conscious Sedation

Drug Name	Induction Bolus Dose	Maintenance Infusion Rate	Maintenance Intermittent Boluses	Notes
Benzodiazepines				
Midazolam	1-5 mg		1-2 mg	Rapid onset & short duration; often given alone or as anxiolytic adjunct to narcotic boluses, remifentanyl infusion and/or propofol infusion
Opioid analgesics				
Alfentanil	0.2-0.5 mg	0.5-2 mcg/hr	0.2-0.5 mg	
Fentanyl	25-50 mcg		25-50 mcg	
Remifentanyl		0.025-0.1 mcg/kg/min	25 mcg	Avoid large boluses (risk of chest wall rigidity); 1 dose when given with midazolam or propofol
Hypnotics				
Propofol	0.25-0.5 mg/kg	2-4 mg/kg/hr (30-70 mcg/kg/min)	0.3-0.5 mg/kg (300-500 mcg/kg)	Easily titratable, rapid recovery, antiemetic effects; pain on injection
Dexmedetomidine	1 mcg/kg over 10 min	0.2-0.7 mcg/kg/hr	-	Some analgesia with little respiratory depression; bradycardia & hypotension common side effects; may have prolonged sedation; small doses of midazolam & fentanyl initially may help reduce the initial bolus & prolonged sedation
Ketamine	0.7 mg/kg	2-4 mcg/kg/min	-	Maintenance of cardiovascular system & respiratory drive make ketamine appealing; avoid in pts with CAD, uncontrolled HTN, CHF and arterial aneurysms

Source: Adapted from: Hillier SC. "Chapter 47: Monitored Anesthesia Care." In Barash PG, Cullen BF, Stoelberg RK, eds. *Current Anesthesia*, 4th ed. Lippincott: Williams & Wilkins: Philadelphia, PA, 2001:1247.

Diagrams & Flow Chart

Recommended Antibiotic Regimen (30–60 min prior to surgery)		
	First-Line Antibiotic	Alternative Antibiotic (PCN-allergic pts)
Oral	Amoxicillin 2 g	Clindamycin 600 mg
	Cephalexin 1 g	Azithromycin 500 mg
IVIM	Ampicillin 2 g IVIM	Clarithromycin 500 mg
	Cefazolin 2 g IVIM	Clindamycin 600 mg IVIM
	Ceftriaxone 1 g IVIM	

Source: Adapted from Wham VK et al. Prevention of Infective Endocarditis. *Circulation* 2007;115(15):1736–1754.

Most Sensitive

Least Sensitive

Extraocular > Pharyngeal > Masseter > Adductor pollicis > Abdominal rectus > Orbicularis oculi > Diaphragm > Vocal Cord

Figure 2D-2 Speed of Onset & Recovery of Neuromuscular Blockade

Fastest Onset

Slowest Onset

Larynx > Diaphragm > Orbicularis oculi > Adductor pollicis

Fastest Recovery

Slowest Recovery

Larynx > Orbicularis oculi = Diaphragm > Adductor pollicis

Interpretation of Brain Function Monitor Number During GA

<60	Increased risk of awareness during GA
40–60	Appropriate anesthetic depth
>60	Excessive depth of anesthesia

Advantages of TIVA over Inhalation induction & Maintenance

- Smooth induction with minimal coughing/hiccups
- Easier control of anesthetic depth
- More rapid, predictable emergence
- Lower incidence of PONV
- Ideal operating conditions for neurologic surgery with reduced cerebral blood flow & cerebral metabolic rate; allows intraoperative neuromonitoring
- ↓ Organ toxicity & atmospheric pollution
- Avoids N₂O side effects (expansion of closed airspaces & bone marrow suppression)

Common Indications for TIVA

- Anesthesia for airway endoscopies, laryngeal & tracheal surgery
- Anesthesia in remote locations or during transport
- Malignant hyperthermia-susceptible patients
- History of significant PONV

Advantages of Continuous Infusions Compared with Intermittent Bolus Dosing

- Avoid oscillations in drug concentration
- Minimize relative over- or underdosing
- Provide stable depth of anesthesia
- Reduce incidence of side effects (hemodynamic instability)
- Shorten recovery times
- Total drug requirements by 25–30%

Diagrams & Flow Chart

Guidelines for Prevention and Management of Intraoperative Awareness	
Prevention	Management
- Check delivery of anesthetic agents to pt - Consider premedication with amnesics - Give adequate dose of induction agents - Avoid muscle paralysis unless it is needed; even then avoid total paralysis - Supplement N₂O & opioid anesthesia with 0.6 MAC of volatile agent - Administer 0.8 MAC when volatile agents are used alone - Use amnesics when light anesthesia is only regimen tolerated by pt - Inform pt about possibility of awareness - Consider brain function monitoring	- Perform detailed interview with patient - Verify patient's account - Sympathize & apologize - Explain what happened - Reassure about nonrepetition in future - Offer psychological support - Record interview in patient's chart - Inform patient's surgeon, nurse, & hospital lawyer - Visit patient daily during hospital stay & keep in contact by telephone after - Do not delay referral to a psychologist or psychiatrist

From Ghoneim MM, Weiskopf RB. Awareness during anesthesia. *Anesthesiology* 2000;92:557-602 With permission.

Summary of ASRA Regional Anesthesia & Anticoagulation Guidelines	
Medication Class	Recommendation
Aspirin, other NSAIDs	No contraindications
Clopidogre	Wait 7 d before needle insertion
Ticlopidine	Wait 14 d before needle insertion
GP IIb/IIIa inhibitors	Wait 6-48 hr before needle insertion
Low-molecular-weight heparin (High-dose enoxaparin 1 mg/kg q12h or 1.5 mg/kg qd; dalteparin 120 U/kg q2h or 200 U/kg qd)	Wait 10-12 hr (for normal dose) or 24 hr (for high dose) before needle insertion Postop bid dosing: Remove catheter 2 hr before 1st dose, which should be no earlier than 24 hr postop. Postop qd dosing: Remove catheter 10-12 hr after last dose & 2 hr before subsequent dose
Heparin SQ <4 days	Assess platelet count prior to removing catheter
Heparin intraop (vascular procedures)	Delay heparin 1 hr after insertion; remove catheter 2-4 hr after last dose; reheparinization OK after catheter removal
Warfarin	Document normal INR < 1.5 prior to needle insertion & catheter removal
Thrombolytics	No specified time interval; follow fibrinogen level

Source: Adapted from *Reg Anesth Pain Med* 2003;3:172-197

Typical Dosing Regimens for IV Agents Used for General Anesthetics

Drug	Induction Bolus	Maintenance Infusion Rate	Maintenance Intermittent Bolus
Thiopental	5-7 mg/kg	-	-
Etonidate	0.2-0.3 mg/kg	-	-
Propofol	2-3 mg/kg	6-10 mg/kg/hr (100-180 mcg/kg/min)	-
Fentanyl	50-100 mcg	0.5-4 mcg/kg/hr	25-50 mcg
Alfentanil	0.5-1.5 mcg	1-3 mcg/hr	0.2-0.5 mcg
Remifentanyl	1-2 mcg/kg	0.1-0.25 mcg/kg/min	-
Sufentanil	0.2 mcg/kg	0.2-0.4 mcg/kg/hr	-
Ketamine	0.1-0.2 mg/kg	5-10 mcg/kg/min	-

From Urban RD, Flapio FB. "Chapter 9: Anesthetic Agents: Which One?" *Manual of Office-Based Anesthesia Procedures*. Lippincott Williams & Wilkins: Philadelphia, PA, 2007:63

Factors Affecting Anesthetic Spread in Intrathecal Space

Baricity	Local solution density in relation to CSF; mixing medication with dextrose or sterile water results in hyperbaric or hypobaric solutions (respectively) - Isobaric (density = CSF)—get block at level where medication is injected - Hyperbaric (density > CSF)—get spread of medication with gravity in intrathecal space - Hypobaric (density < CSF)—get spread of medication against gravity in intrathecal space
Patient position	Gravity can help spread of medication within intrathecal space when hypo- or hyperbaric solutions used
Spinal curvature	Thoracic kyphosis at T4 prevents migration of spinal toward cervical region
Other factors: Dose, volume, temperature of medication injected, age, increased abdominal pressure, pregnancy, direction of needle bevel	
No effect: Weight, height, gender, barbotage	

Diagrams & Flow Chart

Level of Sensory Block Needed for Surgical Procedures		
Sensory Level	Type of Surgery	Local Anesthetic and Dose
T4 (nipple)	Upper abdominal surgery C-section	Tetracaine, bupivacaine, or ropivacaine 8–16 mg
T6-T7 (xiphoid)	Lower abdominal surgery Appendectomy Herniorrhaphy	Lidocaine 75–100 mg, bupivacaine or ropivacaine 10–14 mg
T10 (umbilicus)	Hip surgery TURP Vaginal delivery	Lidocaine 50–75 mg, tetracaine 6–10 mg, bupivacaine or ropivacaine 8–12 mg
L1 (inguinal ligament)	Lower extremity	Tetracaine, bupivacaine, or ropivacaine, 6 mg
L2-L3 (knee)	Foot surgery	Tetracaine, bupivacaine, or ropivacaine, 6 mg
S2-S5	Hemorrhoidectomy	Lidocaine 30–50 mg

Source: Table adapted from Stoelting RK, Miller RD: *Basics of Anesthesia*, 4th ed. Churchill Livingstone, New York, NY 2000

Doses of Epidural Opioids			
Drug	Dose (mg)	Onset (min)	Duration (hr)
Alfentanil	1	5	1
Sufentanil	0.005–0.010	3–5	2–4
Fentanyl	0.05–0.10	5–20	3–5
Mettazone	5–8	10–20	6–8
Hydromorphone	1	15–20	7–15
Meperidine	30–100	5–10	4–20
Morphine	3–5	10–60	12–24

Identifying the Epidural Space

Loss-of-resistance (LOR) technique

- Engage epidural needle in ligament
- Attach low friction syringe (filled with air/saline) to epidural needle
- Apply constant/intermittent pressure to syringe as needle is slowly advanced
- When needle passes through ligamentum flavum (into epidural space), plunger will be easily advanced (indicating a loss of resistance)

Hanging-drop technique

- Drop of fluid placed on hub of epidural needle (once engaged in ligamentum flavum)
- Advance needle until fluid sucked into needle hub (indicates epidural space)

Nerve Localization Techniques

Paresthesia

Block needle used to elicit paresthesia when it contacts nerve. Injection should cause transient enhanced paresthesia; intense searing pain indicates intraneural injection (stop immediately, withdraw needle, reassess)

Nerve stimulation

Insulated needle connected to nerve stimulator elicits muscle twitches in target nerve's pattern of innervation. Dial current down slowly from >1 mA to <0.5 mA while retaining muscle twitch

Ultrasound-guided

Ultrasound probe used to visualize target nerve, block needle, & local anesthetic

Infiltration/field blocks

Local anesthetic injected in close proximity to nerve to be blocked based on its constant relationship with anatomic landmarks

Figure 6-2 Upper extremity innervation. (From Dunn PD, et al. *Clinical Anesthetic Procedures of the AGA*. 7th ed. Lippincott Williams & Wilkins; Philadelphia, PA: 19-230. With permission.)

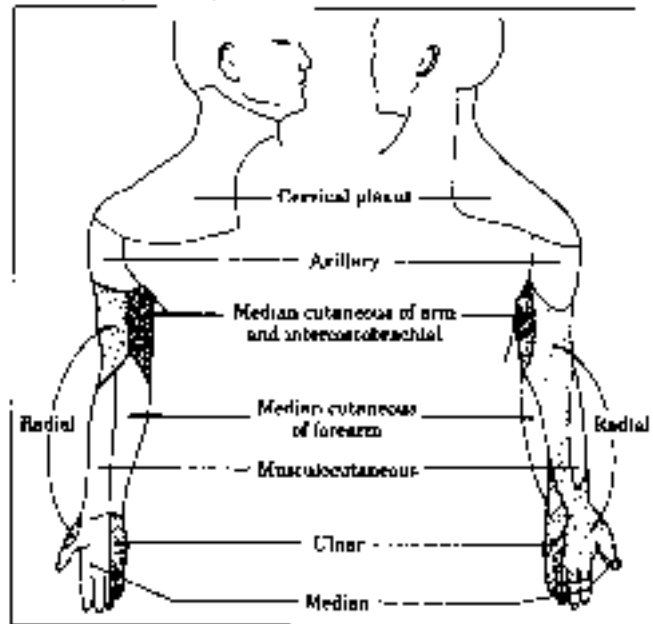
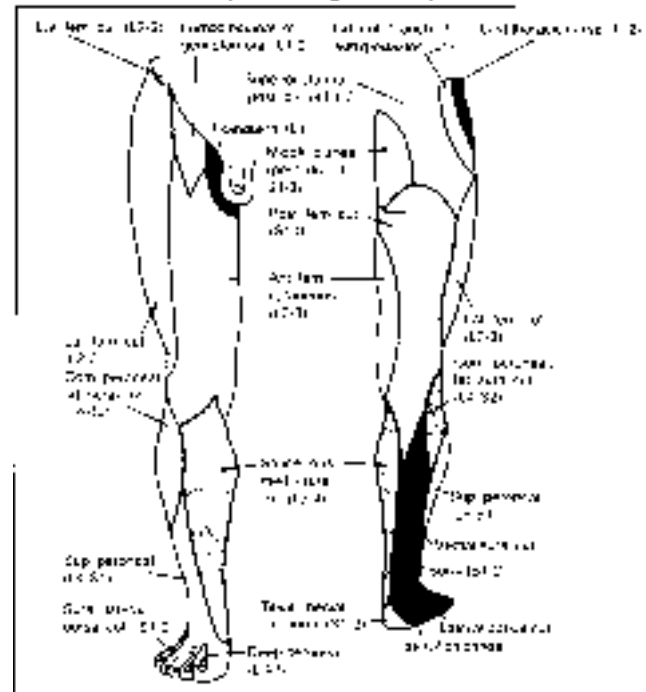


Figure 6-3 Cutaneous innervation of the lower extremity. Adapted from Gray H. *Anatomy of the Human Body*. Lea & Febiger; Philadelphia, PA, 1978.



Diagrams & Flow Chart

Figure 6-4: Innervation of the foot/ankle for ankle block. (Image courtesy of J. Ehrenfeld)

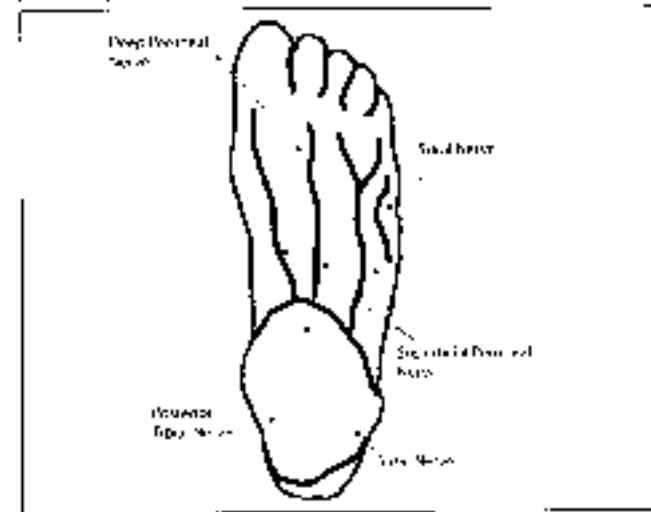
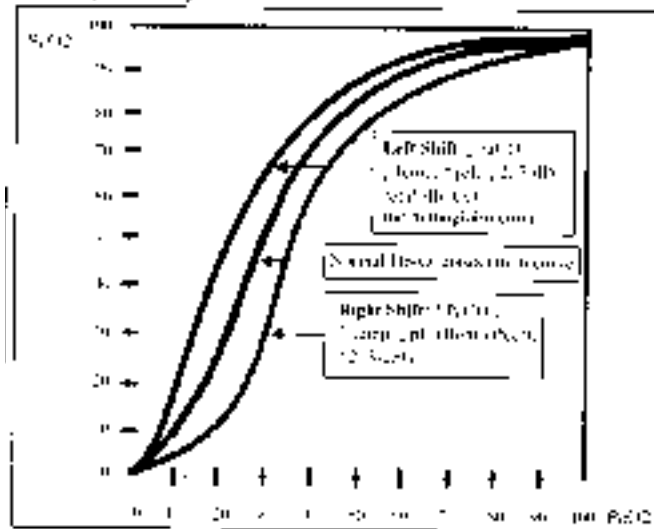


Figure 7-1: The hemoglobin-oxygen (Hb-O_2) dissociation curve (Courtesy of S. Shah, MD, with permission.)



Artifact	SpO ₂ reading	Remedy/Notes
Low perfusion (hypothermia, pressors, atherosclerosis)	Lowers	Need to restore perfusion & pulsatile flow; can passively warm (do not actively warm)
Movement	Raises	Move probe to ear
Nail polish	Lowers	Remove polish or turn probe 90°; black, purple, blue colors worst
Vasopressors	Lowers	Consider dobutamine ("extremity perfusion")
Carboxyhemoglobin (CO exposure)	Raises	↑ FiO ₂ ; Hyperbaric O ₂ ; use orange color co-oximetry
Methemoglobinemia (benzocaine)	Lowers	Administer methylene blue dye
Hemoglobinopathies	Lowers	Supportive care; transfusion
Anemia (severe)	Raises	Consider transfusion in anemic pts, 100% SpO ₂ may still mean significantly ↓ O ₂ delivery
Acidosis	Lowers	Correct; shifts HbO ₂ curve to right
Alkalosis	Lowers	Correct; shifts HbO ₂ curve to left
Cardiopulmonary bypass	Lowers/obliterates	Restore pulsatile flow following CPB

NORMAL CVP & CORRESPONDING ECG WAVEFORM (Fig 7-2)

Feature of the CVP Waveform		
Waveform	Results From	Remark
"a" wave	Venous distention from RA contraction	Biggest pulsation visible, especially during inspiration
"c" wave	Bulging of tricuspid valve into RA during RV isovolumic systole	Called the "c" wave because it coincides with (& is accentuated by) carotid pulse
"x" descent	(1) Atrial relaxation & (2) caudal displacement of tricuspid valve during RV systole	Occurs during systole, accentuated in constrictive pericarditis

Diagrams & Flow Chart

Features of the CVP Waveform (continued)		
Waveforms		
Feature	Results From	Remark
cV wave	Increasing volume of blood in the RA during ventricular systole when the TV is closed	Late systolic, accentuated with tricuspid regurgitation
Y descent	Opening of tricuspid valve & subsequent rapid inflow of blood into RV	Rapid & deep with TR; slow with TS or aortic myxoma, both of which cause slowing of outflow from RA.

Causes of Low Mixed Venous Low O ₂ Delivery		Causes of High Mixed Venous Low O ₂ Use	
• Hypoxia	• Fever	• CN poisoning	• Impaired O ₂ tissue delivery (sepsis, burns)
• Anemia	• ↑ Metabolic states	• Hypothermia	• Mitral regurg.
• ↓ Cardiac output	• Shivering		
• Alkalosis			
• Methemoglobinemia			

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• Anemia	• Fever	• Hypothermia	• Mitral regurg.
• ↓ Cardiac output	• ↑ Metabolic states		
• Alkalosis	• Shivering		
• Methemoglobinemia			

Cardiac Output Erroneously ↓	Cardiac Output Erroneously ↑
• Large volume injected	• Small volume injected
• R-to-L shunt	• L-to-R shunt
• Catheter tip too proximal	• Wedged catheter
	• Tricuspid regurgitation

Diagrams & Flow Chart

Common Reasons for PA Catheterization

- Assess RV pressure
- Diagnose & treat pulm HTN
- Assess hypotension (Sepsis vs cardiogenic shock on basis of CO & SVR)
- Manage severe organ dysfunction/severe sepsis
- Administer fluids judiciously (COPD/pulm HTN/burns/renal failure)
- Diagnose & treat intraoperative oliguria
- Manage MI with shock
- Construct a Starling curve of CO vs preload
- Manage valvular heart dz with appropriate preload & afterload adjustment
- Assess vasoactive therapy by serial CO & SVR measurements
- Manage pts with cerebral vasospasm after SAH (may involve induced hypertension, hemodilution, & hypervolemia under PA catheter monitoring)
- Manage cardiac patients after heart surgery

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Possible Contraindications to PA Catheterization

Previous/planned pneumothorax (consequence of PA rupture would likely be fatal)	Existing pacemaker lead
Tricuspid prosthesis, pulmonic prosthesis	Warfarin, heparin, antiplatelet therapy (e.g., clopidogrel or high dose aspirin)
Tricuspid stenosis, pulmonic stenosis	Endocarditis
R atrial or ventricular masses; documented mural thrombi or valvular growth	
Cyanotic heart dz or R-to-L shunt	Floating during CFB
Latex allergy (if PA components are latex)	Tight AS (until chest is open, as floating A line — loss of atrial kick — CV collapse)
Recent infarct or LBBB	Mitral regurgitation, which may make wedging impossible (predisposes to rupture)
Severe ventricular dysrhythmias	Atrial dysrhythmias (consider echo to rule out atrial thrombus)
Heparin-coated catheter in HIT ⁺ patient	

Possible Contraindications to PA Catheterization

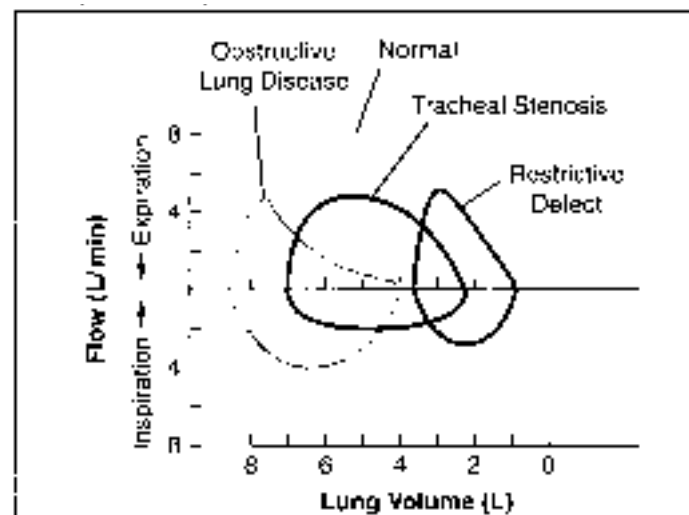
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Heparin-coated catheter in HIT ⁺ patient	

Uses and Indications for Capnography

- Detection of esophageal intubation
- Detection of disconnection from breathing circuit, inadvertent extubation, or insufficient ventilation
- Detection of hyperventilation
- Detection of high metabolic rates (i.e., Malignant hyperthermia or sepsis)
- Detection of low metabolic rates (i.e., Hypothermia, lowered cardiac output states)
- Detection of pulmonary embolism
- Detection of bronchospasm/small airway obstruction
- Calculation of Vd/Vt or dead space ventilation (need PaCO₂ & ET CO₂)

$$Vd/Vt = [(PaCO_2 - P_E-CO_2)/PaCO_2]; \text{normal } Vd/Vt = 0.30$$

Low-Flow Supplemental O ₂ : Typical Means of Administration							
Method of Low-Flow O ₂ Administration	Device	O ₂ Flow (L/min)	FiO ₂	Capnography Feasible	Humidified	Complications	Comment
Nasal cannula O ₂	Nasal cannula attached to standardized flowmeter	0	0.21	Yes with specially designed cannulas; attach to sampling port of capnograph	Yes	Dried nasopharynx; swallowing of air; bleeding, sinusitis	Pts may mouth-breathe or have nasal constriction, sinusitis, packing, nasogastric tubes, etc. (add 4% FiO ₂ for each L/min of O ₂)
		1	0.25				
		2	0.29				
		3	0.33				
		4	0.37				
		5	0.41				
		6	0.45				
		7	0.49				
Venturi face mask	Venturi diluters attached to tubing	10+	0.30–0.50	No	Yes		Fixed FiO ₂ diluters entrain room air
Simple face mask		10+	0.55	No	No	Drying of mucosa	
Nonrebreather face mask with reservoir bag		10+	0.80	No	No	Drying of mucosa	
Ambu (bag-valve-mask)	Has 15-mm adapter to attach standard mask or ET tube	10+	1.0	No	No	Spontaneous breaths difficult to see in bag	Spontaneous breathing or breaths from caretakers; PEEP feasible
Oxygen tent		10–15	0.50	Yes	Yes	Combustion risk	Used in pediatrics

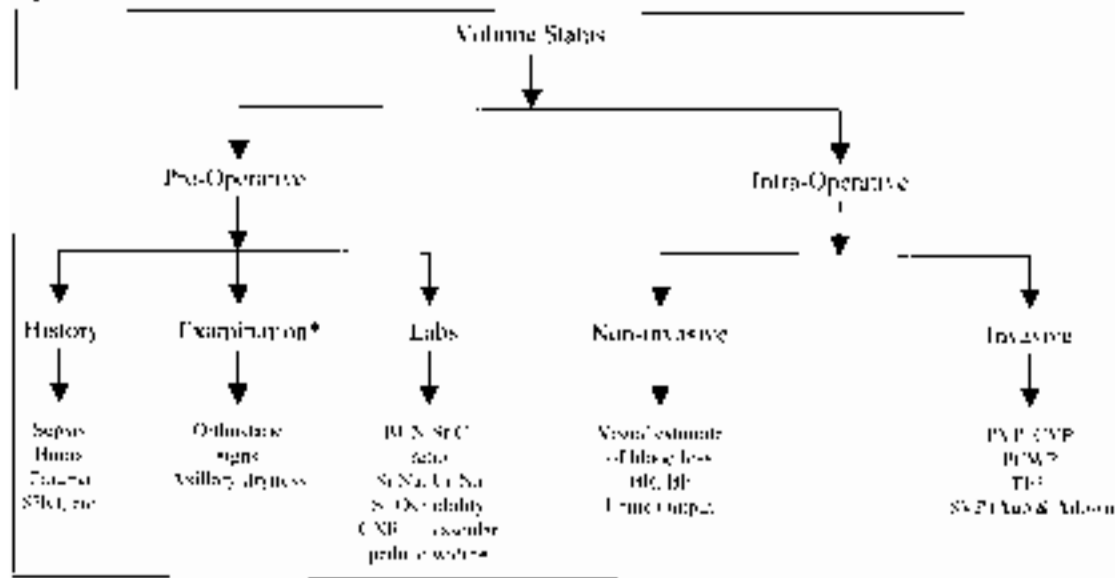


Diagrams & Flow Chart

Criteria for Extubation Used in Many ICUs	Criteria	Remarks
• Fully extended head lift (correlates with NIF of 50 cm H ₂ O) best indicator of upper airway strength		Conscious head lift to command not from triggering a spasm
• PaO ₂ /FiO ₂ > 120 or equivalent by PaO ₂ > 60 on FiO ₂ of 0.50		O ₂ requirement should be 50% or lower before extubation is considered FiO ₂ 40% is better
• PEEP support < 4 cm when adequate oxygenation		Sudden withdrawal of PEEP may cause fall in PaO ₂
• CNS intact (GCS > 13)		Use GCS Scale, Chapter 20
• Ability to maintain PaCO ₂ < 50 mm Hg and 7.30 < pH < 7.50		sepsis/hyperthermia/hypothermia on ↑ CO ₂ production; these causes may be addressed; metabolic alkalosis from GI losses or diuretics may cause CO ₂ retention; treat alkalosis with NaCl & KCl if > 4 fluid or other measures
• Ability to maintain spontaneous respiration without drug		Extubation of pts from PE of > 5 cm H ₂ O may cause them to tire after extubation
• Ability to clear secretions without plugging bronchies		Assess secretions prior to extubation, remember anticholinergics side effects of opioids
• No overt risk of pulm aspiration (no ongoing bowel obstruction or high NG/OG output)		Aspiration may occur with most airway reflexes if bowel obstruction is present or GI bleeding is present
• Adequate Hb, cardiac output, & cardiovascular stability		Hg > 10 allows adequate O ₂ carrying capacity (for ArDO ₂) & vessels open for maintaining SVR & lowering SP
• Pain is controlled adequately to allow spontaneous breathing without excessive splinting		Consider intercostal/intervertebral epidural or intrapleural non-inferiores or subarachnoid block to allow deep breathing and cough

Diagrams & Flow Chart

Figure 9-1 Assessment of volume status



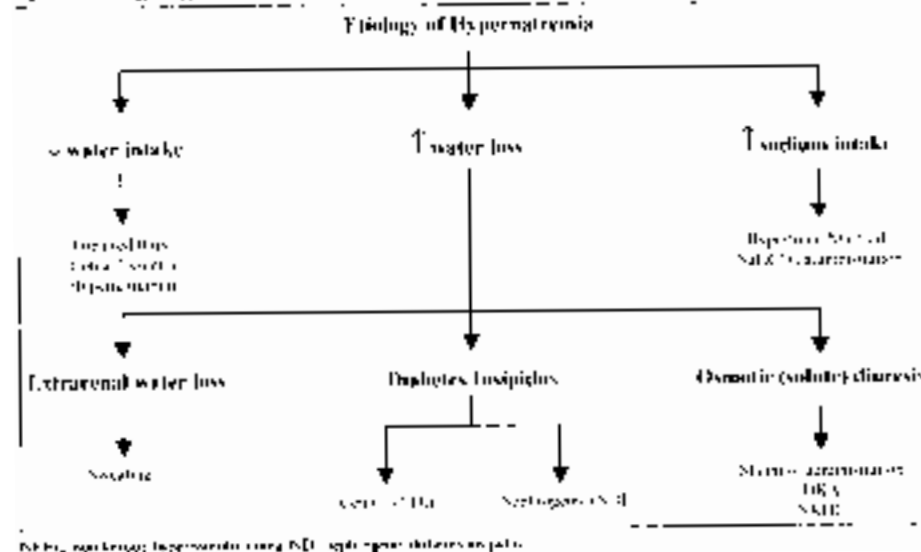
Daily Adult Electrolyte Requirements

Electrolyte	Daily Requirements
Na	60–150 mEq
K	70–150 mEq
Cl	60–150 mEq
Mg	8–24 mEq
Phos	7–10 mmol/1000 kcal

Compositions of Crystalloids & Colloids

Fluid	Na (mEq/mL)	K (mEq/mL)	Osm (mOsm/L)	pH	Other
Extracellular fluid	140	4.5	290	7.4	Cl = 108 Lactate = 5 mEq/L Glucose 50 g/L
D5W	0	0	252	4.5	Cl = 154
0.9 NS	154	0	308	6.0	Ca = 3 mEq/L Lactate = 28 mEq/L HCO ₃ = 27mEq/L Cl = 110
LR	130	4	273	6.5	COP = 32–35 mm Hg COP = 30 mm Hg
5% Albumin	145 ± 15	<2.5	330	7.4	
6% Hetastarch	154	0	310	5.9	
D5 1/4 NS	39	0	320	4.2	Cl = 39
Plasmalyte	140	5	294	7.4	Cl = 98
3% NS	513	0	1025	5.6	Cl = 513

Figure 9.3 Etiology of hypernatremia: DKA, diabetic ketoacidosis, hemoC, nonketotic hyperosmolar coma

**Treatment of Hyperkalemia**

Intervention	Dose	Onset	Comment
Calcium gluconate	1–2 amps IV	Few min	Transient effect
Calcium chloride			Stabilizes cell membrane
Insulin	Reg. insulin 10 U IV 1–2 amps D50W	15–30 min	Transient effect Drives K ⁺ into cells
Bicarbonate (NaHCO ₃)	1–3 amps IV	15–30 min	Transient effect Drives K ⁺ into cells in exchange for H ⁺
β ₂ agonists	Albuterol 10–20 mg inh or 0.5 mg IV	30–90 min	Transient effect Drives K ⁺ into cells
Kayexalate	30–90 g PO/PR	1–2 hr	↓ total body K ⁺ Exchanges Na ⁺ for K ⁺ in gut
Diuretics	Furosemide 40 mg IV	30 min	↓ total body K ⁺
Hemodialysis	For renal failure/life-threatening K ⁺		↓ total body K ⁺

Source: Adapted from Sabatine MS, et al. *Pocket Medicine*, 3rd ed. Lippincott Williams & Wilkins, Philadelphia, PA, 2008.

Available Blood Components				
Component	Content	Indications	Volume	Shelf Life
RBCs	RBCs and WBCs, platelet debris, plasma, fibrinogen	Red cell volume and plasma volume replacement	450 ± 50 mL	Heparin 48 hrs ADSOL 42 days ACD 21 days CPD 28 days CPDA-1 35 days same as whole blood
Packed RBCs	RBCs, WBCs, plasma, platelet debris	Red cell volume replacement	200 mL	
Frozen RBCs	No plasma, minimal WBCs & platelet debris	Red cell volume replacement in special circumstances	160–190 mL	Frozen: 3 years Thawed: 24 hours
Platelets	Platelets, low WBCs, some plasma	Platelet count less than 50,000–100,000, clinical signs of dilutional thrombocytopenia and/or platelet dysfunction	30–50 mL unit	Pheresis: 24 hours Room temperature 5 days, Frozen with DMSO: 3 years
Fresh frozen plasma	Plasma proteins, all coagulation factors	Bleeding from factor deficiencies, and thrombin III deficiency, massive transfusions, coumadin reversal	200–250 mL	Thawed 6–24 hours Frozen: 1 year
Cryoprecipitate	Factors VIII, XIII, fibrinogen, fibronectin, von Willebrand's Factor	Hemophilia A, von Willebrand's disease, fibrinogen deficiency	25 mL unit	Thawed: 4–6 hours Frozen: 1 year
Factor VIII concentrate	Factor VIII, fibrinogen, von Willebrand's Factor	Hemophilia A (classic hemophilia)	Lyophilized (requires reconstitution)	2–8°C: 1 year Room temp: 3 months
Factor IX concentrates (Konyne, Proplex)	Factor II, VII, IX, X	Hemophilia B (Christmas disease)	Lyophilized (requires reconstitution)	2–8°C: 1 year Room temp: 1 month
Albumin 25% (5%)	Albumin	Volume expansion, maintenance of intravascular oncotic pressure	250 or 500 mL (50 mL)	3–5 years
Plasma protein fraction	Albumin, alpha globulin, beta globulin	Volume expansion, maintenance of intravascular oncotic pressure	250 mL	3–5 years

Source: From Ringer DE, Sai SW, MA. Transfusion therapy, part I. *Prog Anesthesiol* 1989;3:1–14

Diagrams & Flow Chart

Suggested Platelet Transfusion Thresholds

- 10,000 Prophylaxis (based on studies in cancer pts *N Engl J Med* 337[15]:1870-1875)
- 20,000 Any bleeding or pre-procedure, pts with concurrent coagulation disorder/infection
- 50,000 Major bleeding or during surgery; prior to CNS or major eye surgery; bleeding with trauma; peritracheal bleeding; after prolonged cardiac-pulmonary bypass

Given classes for TP with life-threatening bleeding from GI/GU source or from CNS, massive transfusion bleeding usually precedes fatal hemorrhage

Indications for FFP Transfusion

- INR > 2.0 Prophylactic transfusion prior to invasive procedures or for actively bleeding pts
- INR 1.5–2.0 FFP may be of value in actively bleeding pts; uncertain benefit as pre-procedure prophylaxis; unlikely to correct INR value without massive no. of units of FFP (*Transfusion* 2006;46:1279)
- INR < 1.5 FFP not indicated

Treatment of a Suspected Hemolytic Transfusion Reaction

1. Stop transfusion
2. Treat hypotension with fluids and/or vasopressors
3. Maintain urine output (75–100 mL/hr) with fluids, mannitol, & furosemide
4. Alkalinize urine (give 40–70 mEq bicarb per 70-kg body wt) to prevent precipitation
5. Send unused blood & fresh pt sample to blood bank (for recrossmatch)
6. Send blood sample to lab for free Hb, haptoglobin, Coombs test, DIC screen
7. Consider corticosteroids

Complications of Massive Transfusion

- K^+ & Ca^{++} (citrate preservative binds Ca^{++})
- Distal renal thrombocytopenia (may need FFP & platelets)
- Metabolic alkalosis (due to citrate forming HCO_3^-)
- Hypothermia

Estimated allowable blood loss = $EBV \times (H_{pre} - H_{post})/H_{pre}$

H_{pre} = initial Hct

H_{post} = final lowest acceptable Hct

Estimated blood volume (EBV) = $weight (kg) \times average blood volume$

Average Blood Volume

Premature neonates	75 mL/kg
Full-term neonates	85 mL/kg
Infants	80 mL/kg
Adult men	75 mL/kg
Adult women	65 mL/kg

Estimating Blood Loss in Surgical Sponges

Sponge Type	Fluid Capacity
4 × 4 sponge	70 mL
Raptech 500 g	10–20 mL
Ulp sponge	100 mL

Diagnosis and Management of Transfusion Reactions				
Type	Notes	Symptoms/Signs	Cause	Treatment
Febrile nonhemolytic transfusion reaction	• Most common (1:200–500) • 15% will have a 2nd reaction	Fever (1°C above pretransfusion), chills, + mild dyspnea within 1–6 hr after transfusion	RBCs: Class I HLA Ab against donor leukocytes Plts: Storage-dependent cytokines	• Stop transfusion & rule out hemolytic reaction, severe infection • Give antipyretics, IM meperidine in pts with chills and rigors • Use leukoreduced blood products in the future
Simple allergic reaction	• 1:333–500	Hives + itching	Transfused allergens in plasma cause mast cell degranulation	• Pause transfusion • If only hives + itching, may continue same unit • Give antihistamines if pt symptomatic • Unlikely to recur in future, consider premedication with antihistamines and use washed cells if repeated reactions
Transfusion-related acute lung injury (TRALI)	• 1:5,000 • 1–6 hr after onset of transfusion • CVP is normal • Looks like ARDS • Death 10%	Acute respiratory distress, hypoxemia, hypotension, fever, pulmonary edema	Donor antibodies agglutinate host neutrophils to cause lung injury	• Stop transfusion • ABCs, O₂, mechanical ventilation, diuresis, ? steroids • If recovers, not at increased risk for recurrent episodes following transfusions from other donors
Acute hemolytic transfusion reaction	• 1:15,000, fatal in 1:250,000 to 1:600,000 • Results in DIC, shock, acute renal failure	Fever, chills, N/V, pink plasma, flank pain, pink, red or brown urine, or any combination of the above symptoms	Destruction of donor RBC by preformed recipient Abs. Usually secondary to ABO incompatibility	• Stop transfusion, leave IV attached for treatment • Start NS at 100–200 mL/hr • Lasix 40–80 mg IV initially, then titrate to UO₂ > 100 mL/hr for 24 hr • From other arm obtain direct antiglobulin test (will be positive), CBC, lytes, new blood bank sample • May require pressors, watch for hyperkalemia

Diagrams & Flow Chart

Primary Hemostasis	Secondary Hemostasis
• Constriction of injured vessels • Exposure of subendothelial collagen • Adhesion & aggregation of blood platelets on damaged surface • Formation of 1st hemostatic plug	• Formation of thrombin catalyzed by surface of activated platelets • Formation of thrombin via activation of factor VII by tissue factor • Conversion of fibrinogen → fibrin catalyzed by thrombin • Formation of fibrin clot & its stabilization

Screening Test Abnormalities in Inherited and Acquired Coagulopathies			
PT	PTT	Inherited	Acquired
↑	—	Factor VII deficiency	Vit K deficiency; Liver dz; factor VII inhibitors
—	↑	Hemophilias, vWD	Factor inhibitors; antophospholipid Ab
↑	↑	Deficiency: fibrinogen, factor II, factor V	DIC; liver dz; inhibition of fibrinogen, factors II, V or X

- Thromboelastogram (TEG)
 - Viscoelastic test that characterizes formation & strength of blood clot over entire period of clotting & fibrinolysis
 - Assesses coagulation system, platelet function, & fibrinolysis

Agent	Bleeding Time	Prothrombin Time	Activated Thromboplastin Time	Activated Clotting Time	Time to Peak Effect	Time to Normal Hemostasis Post-therapy	Comments
Aspirin	↑↑	—	—	—	Hours	1 week	Platelet function not accurately predicted by bleeding time
Other NSAIDs	↑↑	—	—	—	Hours	3–5 days	Platelet function not accurately predicted by bleeding time
Heparin, regular intravenous	—	—	↑↑	↑↑	Minutes	4–6 hours	Monitor activated clotting time or activated thromboplastin time
Heparin, regular subcutaneous	—	—	↑↓	↑↑	1 hour	4–6 hours	Activated thromboplastin time may remain normal; Monitor anti-Xa activity
Heparin, low molecular weight Subcutaneous	—	—	↓↑	↓↑	12 hours	1–2 days	Activated thromboplastin time may remain normal; Monitor anti-Xa activity
Thrombolytic agents	↑↑	—	↓	—	Minutes	1–2 days	Frequently administered along with intravenous heparin

↓: Clinically insignificant increase. ↓↓: Possibly clinically significant increase. ↑↑: Clinically significant increase

Diagrams & Flow Chart

Characteristics of Anticoagulants

	Warfarin	Unfractionated Heparin	Low-Molecular-Weight Heparin	Factor Xa Inhibitors	Direct Thrombin Inhibitors
No. Cascade targets	Many	Many	Few	Few	Few
Activity specificity	Nonspecific	Nonspecific	Specific	Specific	Specific
No. daily doses	1	2-3	1-2	1	1-2
Route	PO	IV, SC	SC	SC, PO	IV, PO, SC
Monitoring	INR	aPTT, plt count	Plt count, anti-Xa	None	aPTT, liver fx
Variability in response	High	High	Low	None	Low
Risk of HIT	None	2-5%	1-2%	None	None
Other notes	Inhibits factors II, VII, IX, X, protein C	B nds antithrombin III			

Properties and Antidotes for Anticoagulants & Fibrinolytics

Anticoag.	t _{1/2}	Labs	Rx for overdose w/ serious bleeding*
UFH	60-90' RES	↑ PTT	Protamine IV 1 mg/100 U unfractionated heparin (UFH) (max 50 mg). For infusions, dose to reverse 2x rate of UFH given per hr.
Bivalirudin	25', K	↑ PTT	Dialysis
Lepirudin	80', K	↑ PTT	Dialysis
Argatroban	45', L	↑ PTT	↑ Dialysis
Enoxaparin	8', K	{anti-Xa}	↑ Protamine (reversal incomplete)
Fondaparinux	24', K	{anti-Xa}	↑ Dialysis
Warfarin	36', L	↑ PT	No bleeding: INR > 5: vit. K 1-5 mg PO (superior to SC, IV at 24°; 2.5 mg for INR 6-10; 5 mg for INR >10 (Archives 2003:163:2469) Bleeding: vit. K 10 mg IV + FFP 2-4 units IV q 6-8°
Fibrinolytic	20-90' LK	- fbg ↑ FDP	Cryoprecipitate, FFP, ± aminocaproic acid

Heparin-Induced Thrombocytopenia (HIT)

Overview of Heparin-Induced Thrombocytopenia (HIT)

Feature	Type I	Type II
Mechanism	Direct effect of heparin	Immune (Ab)-mediated
Incidence	20%	1-3%
Onset	After 1-4 d of heparin	After 4-10 d; can occur early (< 24 hr) if prior exposure in last 100 d, can occur after heparin stopped
Platelet count	> 100,000/ μ L	30-70,000/ μ L or 50% decrease from baseline
Sequelae	None	Thrombotic events (HITT) in 30-50% Rare hemorrhagic problems
Management	Continue heparin; observe	Stop heparin; start alternative anticoagulation therapy

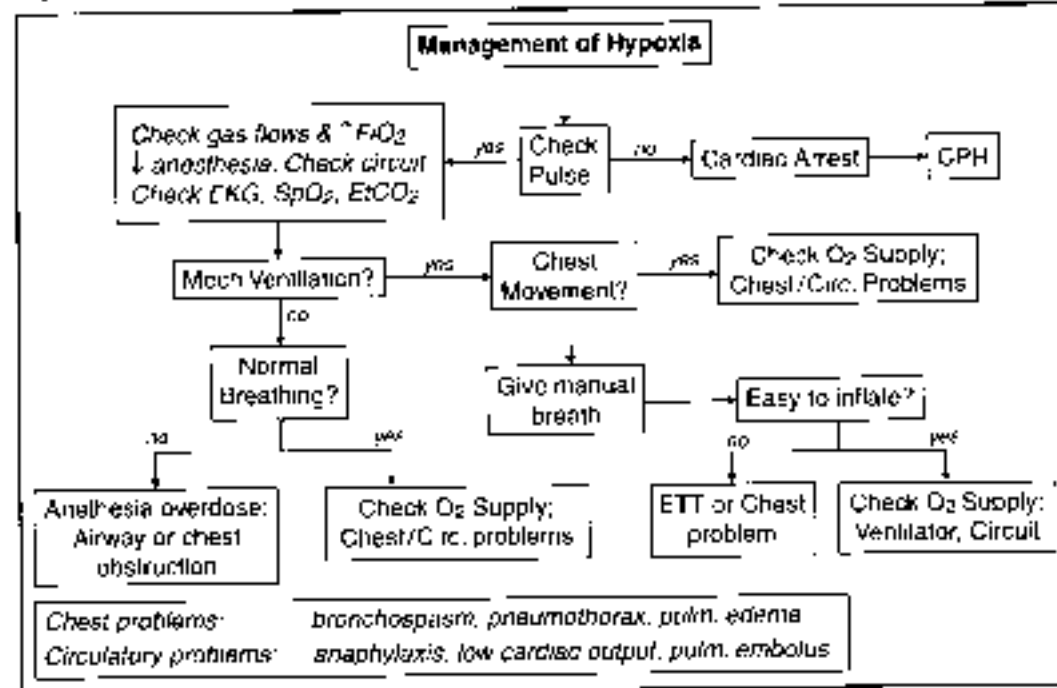
Causes of DIC

Acute	Chronic
• Sepsis, ARDS • Shock • Trauma • Obstetric (e.g., amniotic fluid embolism) • Hemolytic transfusion reaction • Extensive burns	• Malignancy • Liver disease • Retained dead fetus • Intra-aortic balloon pump • Peritoneovenous shunt • Aortic dissection/aneurysm

Diagrams & Flow Chart

Perioperative Management of Sickle Cell Disease

- Ensure adequate hydration
- Avoid factors that cause sickling (hypoxia, hypothermia, dehydration, acidosis, polycythemia, infection)
- Consider preop simple transfusion to HCT $\geq 30\%$
- Consider exchange transfusion to keep HbS $\leq 40\%$ (can also ↓ blood viscosity)

Figure 10-1 (Adapted from Murphy, Fale. Pocket Reference to Anesthesia, 2nd ed.)**Adverse Physiologic Effects of Uncontrolled Acute Pain**

Cardiac	HTN, tachycardia, dysrhythmias, MI
Pulmonary	Atelectasis, V/Q mismatch, pneumonia
Endocrine	Protein catabolism, hyperglycemia, fluid retention
Immune	Immune function impairment
Coagulation	Hypercoagulation, ↓ platelet adhesion
GI	Ileus
GU	Urinary retention

Changing From One Opioid to Another (example)

1. Calculate total 24-hr opioid dose (Morphine 15 mg q4h = 90 mg in 24 hr)
2. Find new opioid on equianalgesic table (Hydromorphone 7.5 mg = 30 mg morphine)
3. Solve equation for new dose (90 mg/30 mg $\times$ 7.5 = 22.5 mg)
4. Divide new 24-hr dose by # doses/day (22.5 mg/4 doses = 5.7 mg q6h)
5. ↑ Calc dose by 25–50%
6. Titrate to clinical effect (4 mg q6h)

Opioid Side Effects**Treatments**

Nausea/vomiting	Switch/lean agent, ondansetron, phenegan, prochlorperazine, haloperidol, metoclopramide
Allergic reaction	Switch agent, diphenhydramine or other antihistamines
Respiratory depression	Support airway, ↓ dose, consider naloxone
Pruritus	Switch agent, nalbuphine (antihistamines not very effective)
Delirium	Switch agent, ↓ dose, haloperidol, olanzapine
Constipation	Laxatives (senna, lactulose) + stool softeners (Colace)
Sedation	↓ Dose, hold anxiolytics, consider CNS stimulants if persistent

Diagrams & Flow Chart

Ultrasound-Guided Central Line Placement

Insertion of central lines with US guidance may provide enhanced safety

Advantages	Disadvantages
• ↓ Complication rate in pts with predicted difficulty of line insertion • ↓ Time & ↑ success in pts with predicted difficulty of line insertion	• ↑ Time required for setup • Potential ↑ risk of breaking sterility • No evidence of benefit in pts where no difficulty in line placement is predicted

Source: Espino A, Dunning J. Does ultrasound-guided central line insertion reduce complications and time to placement in elective patients undergoing cardiac surgery? *Intens Care Med* 2004; 3(3): 523-527

Common Non-narcotic Adjuncts			
	Route	Dose	Side Effects
Ketorolac	IV/IM	30 mg q8h (max 120 mg daily)	GI bleeding, decrease dose in renal impairment & elderly; contraindicated in active peptic ulcer dx.
Diclofenac	IV, IM, PO	50-100 mg	
Ibuprofen	PO	300-800 mg	
Naproxen	PO	250-500 mg	
Gabapentin	PO	300 mg PO tid titrated to 1800 mg/d	Leukopenia, thrombocytopenia
Ketamine	IV, IM, PO	IV: 0.2-0.8 mg/kg IM: 2-6 mg/kg PO: 6-10 mg/kg	Direct myocardial depressant General vasodilator Sympathetic nervous system outflow (↑ HR & CO)
Clonidine	PO	0.3-0.4 mg	Bradycardia
Lorazepam	IV, IM, PO	IV/IM: 0.02-0.08 mg/kg PO: 2-3 mg	Resp depression (if given with opioids)
Ondansetron	IV	IV: 4 mg Peds IV: 0.05-0.075 mg/kg	Headache, dizziness, sedation, shivers, LFT elevation
Benzadryl	IV, PO	IV: 10-50 mg q6-8h Peds IV: 5 mg/kg/d in 4 divided doses (max 300 mg)	Tachycardia, dizziness, seizures, urinary retention
Acetaminophen	PO, PR	0.5-2 g	Hepatotoxicity, GI upset
Celecoxib (Celebrex)	PO	100-200 mg	May ↑ risk of MI/stroke
Aspirin	PO	500-1000 mg	Reye's syndrome (do not use in children under 12 yr)

PCA Guidelines

Opioid	Adult Dose	Pedi Dose	Adult Lockout (min)	Pedi Lockout (min)	Adult Infusion (mg/hr)	Pedi Infusion
Morphine	1-3 mg	50-75 mcg/kg	5-20	q2-3h	0-10	15-20 mcg/kg/h
Fentanyl	0.015-0.05 mg	1 mcg/kg	3-15	q1-2h	0-0.1	2-4 mcg/kg/h
Sufentanil	0.003-0.015 mg		3-10		0-0.1	
Hydromorphone	0.1-0.3 mg	6-10 mcg/kg	10-20	q3-4h	0-0.5	3-4 mcg/kg/h
Meperidine	5-15 mg		5-15		0-30	
Methadone	10 mcg/kg	10 mcg/kg	10-15	q10-15h	20 mcg/kg	20 mcg/kg

PCEA General Guidelines

Opioid	Bolus Dose (mg)	Onset (min)	Peak (min)	Duration (hr)	Infusion Rate (mg/hr)	PCEA Dose (mg)	PCEA Lockout (min)
Morphine	5	15-30	30-90	4-24	0.3-0.9	0.2-0.3	30
Fentanyl	0.05-0.1	5-10	10-20	2-3	0.025-0.05	0.02-0.03	15
Hydromorphone	0.75-1	10-15	30	8-16	0.1-0.2	0.15	30
Sufentanil	0.03-0.05	7	20-30	2-3			
Methadone	100 mcg/kg	15-20		2-3 d	2 mcg/kg/h	1 mcg/kg	15

Diagrams & Flow Chart

Epidural Local Anesthetics	
Solution	Rate (mL/kg/h)
1/16% (0.0625%) Bupivacaine + 1 mcg/mL fentanyl	0.1–0.2
1/32% (0.3125%) Bupivacaine + 2 mcg/mL fentanyl + 2 mcg/mL epi	0.1–0.2
1/32% (0.3125%) Bupivacaine + 5 mcg/mL hydromorphone + 2 mcg/mL Epi	0.1–0.2
Fentanyl 2 mcg/mL + bupivacaine 1/32 % + 2 mcg/mL epinephrine	0.1–0.4
Clonidine 5 mcg/mL + ropivacaine 0.2 %	0.3–0.5
1/5% (0.2%) Bupivacaine + dilaudid 0.01 or 0.02 mg/mL	mcg/kg/h
1/8% (0.125%) Bupivacaine + dilaudid 0.01 or 0.02 mg/mL	4–12 mL/h
1/16% (0.0625%) Bupivacaine + dilaudid 0.01 or 0.02 mg/mL	

Treatment of Side Effects With Epidural Analgesia

Side Effect	Treatment
Pruritus	Nalbuphine 5–10 mg IV/IM Benadryl 25–50 mg IV Naloxone 40–80 mcg IV
Nausea/Vomiting	Metoclopramide 10 mg IV Nalbuphine 10 mg IV Naloxone 40–80 mcg IV
Respiratory depression	Naloxone 0.1 mg IV

Intrathecal Opioids

Opioid	Dose	Duration	Indications	Side Effects
Morphine	0.2–0.4 mg	24 hr	Good spread & long duration of action	Nausea, vomiting, resp depression, sedation, itching, urinary retention
Fentanyl	12.5–25 mcg	3–4 hr	For segmental spread	Nausea, resp depression
Sufentanil	5–10 mcg	2–4 hr	Very lipid soluble & has segmental spread	Nausea, resp depression
Meperidine	10 mg	3–6 hr	In addition to opioid effect has local anesthetic effect	Hypotension

Epidural Placement Levels

- Thoracotomy T4-T6
- Upper abdomen T8
- Lower abdomen T10-T12
- Lower extremity/pelvis L2-L4

Mixed Narcotic Agonists/Antagonists					
Analgesic Drug	Opioid Potency	Respiratory Withdrawal	Depression	Side Effects	Dose
Nalbuphine	None	Can cause abstinence syndrome	None	No sedation	
Nalbuphine	Equal to morphine	Abrupt discontinuation can cause opioid withdrawal	Less respiratory depression than morphine	Considerable sedation may cause psychological or physical dependence	10 mg IV q3–4hr
Buprenorphine	25–50 times more potent than morphine	Mild to moderate withdrawal effects	Ceiling in respiratory depression	Prolonged use can result in physical dependence	0.4 mg IV q4–6hr
Pentazocine	1/3 potency of morphine	Mild	Respiratory depression can be reversed by narcotic analgesics	Can cause tolerance and addiction	30 mg PO q4–6hr
Butorphanol	1/3 potency of morphine	Withdrawal effects similar to that caused by naloxone	Respiratory depression 5 times more than morphine	Sedation, Nausea, Unpleasant psychomimetic effects	0.5–2 mg IV q3–4hr

Diagrams & Flow Chart

HYPOTENSION

Common Causes of Hypotension in PACU

- Hypovolemia
- Bleeding
- Sepsis/ SVR
- Cardiac arrhythmias
- Drugs/anesthesia (spinal/epidural)
- Error in measurement (inappropriate cuff size, machine malfunction)
- Pulmonary embolism
- MI/ myocardial contractility
- Cardiac tamponade
- Congestive heart failure
- Anaphylaxis/anaphylactoid reaction
- Pneumothorax
- Adrenal insufficiency/severe hypothyroidism

Source: Adapted from Keel, RD, et al. Cardiovascular events in postanesthesia care unit. Contribution of risk factors. *Anesthesiology* 1994;81:777

HYPERTENSION

Common Causes of Hypertension in the PACU

- Pain
- Anxiety
- Respiratory insufficiency (hypoxia, hypercarbia)
- Hypothermia/shivering
- ↑ Sympathetic activity
- ↑ ICP
- Essential hypertension/missed medications
- Fluid overload
- Endocrine disease (thyroid storm) pheochromocytoma)
- Error in measurement (inappropriate cuff size, machine malfunction)

RESPIRATORY AND AIRWAY PROBLEMS

Common Causes of Respiratory Insufficiency in the PACU

- | | | |
|---|---|--|
| Hypoventilation <ul style="list-style-type: none"> • Residual anesthesia • Residual muscle relaxant • Postop narcotics • Splinting 2° to pain • Tight abdominal binder • Obstructive sleep apnea/obesity • Premature infants/neonates | Upper Airway Obstruction <ul style="list-style-type: none"> • Airway edema • Trauma • Vocal cord paralysis • Arytenoid dislocation • Secretions • Foreign body • Laryngospasm • Anxiety/Munchausen's stridor | Hypoxemia <ul style="list-style-type: none"> • Atelectasis • Asthma/COPD exacerbation • CHF/fluid overload • Pulmonary embolism • ALI/ARDS • Aspiration • Pneumo/hemothorax • pleural effusion • Diaphragmatic injury/paralysis • Pneumonia |
|---|---|--|

Source: Adapted from Keel, RD, et al. Critical Respiratory events in the postanesthesia care unit: patient, surgical, and anesthetic factors. *Anesthesiology* 1994;81:410.

Diagrams & Flow Chart

Treatment of Hypoventilation	
Suspected Cause	Therapy
Residual inhalational IV anesthesia	Arouse patient, be supportive
Residual muscle relaxant	Administer anticholinesterase inhibitor (neostigmine)
Postop narcotic administration	Administer naloxone
Splinting 2 nd to pain	Initiate pain control, consider PCA/regional analgesia
Tight abdominal binder	Release binder, consult surgeon
Obstructive sleep apnea/obesity	Reposition patient, consider BiPAP
Premature intubation/obesity	Supplemental O ₂ , consider acetaminophen/regional analgesia instead of narcotics

Causes of Delayed Awakening in PACU

Anesthesia related	Residual anesthetic
	Residual muscle relaxant, pseudocholinesterase deficiency
	Excessive narcotics
Metabolic	Hypothermia
	Hypoxemia
	Hypercarbia/hyponatremia/hypocalcemia/hypoglycemia
	Renal/hepatic failure
Intracranial event	Stroke/cerebrovascular accident (CVA)
	Seizure
	Intracranial hypertension

Modified Aldrete Scoring System

Activity: Able to move (voluntarily or on command)	
• Four extremities	2
• Two extremities	1
• No extremities	0
Respiration	
• Able to breathe deeply or cough freely	2
• Dyspnea, shallow, or limited breathing	1
• Apnea	0
Circulation	
• BP $\geq$ 20 mmHg of preop level	2
• BP $\geq$ 20–50 mmHg of preop level	1
• BP $<$ 50 mmHg of preop level	0
Consciousness	
• Fully awake	2
• Arousable on calling	1
• Unresponsive	0
Oxygen saturation	
• SpO ₂ $\geq$ 92%	2
• Needs supplement O ₂ to maintain SpO ₂ $\geq$ 90%	1
• SpO ₂ $<$ 90% with oxygen	0

Guidelines for Discharge From Phase 1

Mental status	Preop level of mental status, pain should be $\leq$ 4 out of 10 or tolerable
Vital signs	Stable & within acceptable limits
Surgical site	In appropriate condition, invasive lines & tubes patent/working
Regional anesthesia	Progressively $\pm$ sensory & motor block
Pts discharged from phase 1 to hospital room should have postanesthesia recovery score (modified Aldrete score) of $\geq$ 9	

Diagrams & Flow Chart

Summary of ASA Practice Advisory: Prevention of Peripheral Neuropathy	
Preop assessment	Helpful to ascertain that pts can comfortably tolerate anticipated operative position
Upper extremity positioning	- Limit arm abduction to 90° in supine pts. Prone pts may comfortably tolerate arm abduction >90° - Position arms to ↓ pressure on posterior/groove of humerus (ulnar groove); when arms tucked at side, neutral forearm position is recommended; when arms abducted on armboards, either supination or neutral forearm position acceptable - Avoid prolonged pressure on radial nerve in spiral groove of humerus - Extension of elbow beyond comfortable range may stretch median nerve
Lower extremity positioning	- Lithotomy positions that stretch hamstring muscle group beyond comfortable range may stretch sciatic nerve - Avoid prolonged pressure on peroneal nerve at fibular head - Neither extension nor flexion of hip ↑ risk of femoral neuropathy
Protective padding	- Padding armboards may ↓ risk of upper extremity neuropathy - Chenille rolls in laterally positioned pts may ↓ risk of upper extremity neuropathies - Padding at elbow & fibular head may ↓ risk of upper & lower extremity neuropathies, respectively
Equipment	- Properly functioning automated BP cuffs on upper arms do not affect risk of upper extremity neuropathies - Shoulder braces in steep head-down positions may ↑ risk of brachial plexus neuropathies
Postop assessment	Simple postop assessment of extremity nerve function may lead to early recognition of peripheral neuropathies
Documentation	Charting specific positioning actions during care of pts may result in improvements of care by helping practitioners focus attention on relevant aspects of patient positioning & providing information that continuous improvement processes lead to refinement in patient care

Source: American Society of Anesthesiologists Task Force on Prevention of Perioperative Peripheral Neuropathies. *Anesthesiology* 2006;104:1168-1177

Perioperative Blindness

Summary of ASA Practice Advisory: Perioperative Visual Loss & Spine Surgery

- A subset of spine surgery pts while prone have ↑ risk of perioperative visual loss including pts who are anticipated to have **substantial blood loss**, or **long surgeries**
- Inform high-risk pts of the small, unpredictable, risk of visual loss
- Deliberate hypotensive techniques not associated with visual loss
- Use colloids along with crystalloids to maintain volume in pts with significant blood loss
- No transfusion threshold known to eliminate risk of visual loss 2° to anemia
- High-risk pts should be positioned head level to or higher than heart
- Consider use of staged spine procedures in high-risk pts

Source: American Society for Anesthesiologists Task Force on Perioperative Blindness. Roth S, et al. Practice advisory for perioperative visual loss associated with spine surgery. *Anesthesiology* 2006;104:1319-1326

Significance of Spinal Cord Injury Site: (most common injury sites: C5–6, T12–L1)

C5	→ Impaired innervation of diaphragm/necessitates ventilatory support
C7	→ Reduced vital capacity/FEV ₁ (up to 70%)
T1	→ Quadriplegia
T4	→ Bradycardia
T7	→ Reduced strength of accessory muscles of breathing
L4	→ Paraplegia

Goals for Early Resuscitation of the Trauma Patient in Hemorrhagic Shock

Maintain SBP 80–100 or MAP 60–70 (goal may be higher if ICP is elevated)

Maintain Hb 7–9 (goal may be higher in brain injured patients/pts with comorbidities)

Maintain normal INR & PTT

Maintain platelets >50,000

Maintain normal serum ionized Ca²⁺

Maintain core temperature >35°C

Prevent acidosis from worsening

Diagrams & Flow Chart

Rhabdomyolysis

Definition	Acute disintegration of striated muscle
Causes	Trauma, crush injury, electrical shock, CPR, ischemia, arterial occlusion, compartment syndrome, DIC, burn injury, hypothermia, medications, & illicit drugs
Signs/symptoms	Acute myalgias/pigmenturia ↑ Serum ck, myoglobin, potassium, urea & phosphorus Arrhythmias (caused by excess potassium & hypocalcemia)
Consequence	Free myoglobin toxic to renal tubules → acute renal failure
Course	CK levels typically peak 2-5 days after initial insult Levels > 16,000 U/L more likely to cause renal failure Hypocalcemia (from influx/deposition of Ca^{2+} in damaged muscle) may occur
Therapy	Restore blood flow to ischemic areas IV fluids (maintain urine output 200 mL/hr until CK levels ↓) No evidence for mannitol/sodium bicarbonate Treat hypocalcemia if tetany/severe hyperkalemia develops Treat compartment syndrome if this develops Dialysis if fluid resuscitation fails to correct intractable hyperkalemia and/or acidosis

Criteria for SIRS (two or more must be present)

Fever > 38°	OR	Hypothermia < 36°
Tachypnea > 20 breaths/min		
Hypocarbia with $PaCO_2$ < 32 mmHg (with spontaneous breathing)	OR	Requirement for mechanical ventilation
Heart rate > 90		
Leukocytosis > 12K	OR	Leukopenia < 4K OR greater than 10% band forms

Criteria for Sepsis

Sepsis	SIRS + infection
Severe sepsis	Sepsis + hypotension/hypoperfusion & acute organ dysfunction
Septic shock	Severe sepsis + hypotension that does not respond to fluids
Refractory septic shock	Hypotension persists 1 hr despite fluid resuscitation

Necrotizing Fasciitis/Myonecrosis

Definitions	Deep infection that involves fascia & subcutaneous tissue, myonecrosis indicates muscle involvement
Signs/symptoms	Ce lulitis, ↑ temp, lethargy, subcutaneous tissue has hard, "wooden" feel, pain out of proportion to exam; gas gangrene - severe fulminant clostridial myonecrosis (may also see crepitus)
Pathology	Group A streptococcal species, Staph aureus, anaerobic streptococcal species & bowel flora
Course	Infections can spread rapidly/cause systemic toxicity; mortality is high
Treatment	Early surgical debridement & broad-spectrum antibiotics

Bioterrorism and Chemical Warfare Agents

Potential Agent	Effect	Treatment	Staff Protection
Botulinum	Paralysis (symmetric descending weakness)	Trivalent botulism antitoxin or botulism immune globulin	Universal precautions
Nerve agents (Sarin, VX)	Cholinergic crisis	Atropine repeated q5-10min and pralidoxime followed by infusion	Level C chemical protection suits & filtration breathing apparatus
Hydrogen cyanide	Inhibits aerobic respiration; metabolic acidosis	Na thiosulfate, Na nitrite, hydroxycobalamin	Level C chemical protection suits & filtration breathing apparatus
Bacillus anthracis	Mediastinitis, meningitis, multiorgan failure	Ciprofloxacin, doxycycline, penicillin, and streptomycin	Isolation, vaccination
Variola virus (smallpox)	Rash, pneumonia	Cidofovir	Isolation, vaccination with vaccinia immunoglobulin

Initial Resuscitation and Infection Issues Strength of recommendation and quality of evidence have been assessed using the GRADE criteria, presented in parentheses after each guideline. - Indicates a strong recommendation or "we recommend" - Indicates a weak recommendation, or "we suggest" <i>Initial resuscitation (first 6 hours)</i> - Begin resuscitation immediately in patients with hypotension or elevated serum lactate >4 mmol/L; do not delay pending ICU admission (1C) - Resuscitation goals: (1C) - CVP 8–12 mm Hg^a - Mean arterial pressure ≥ 65 mm Hg - Urine output ≥ 0.5 mL·kg⁻¹·hr⁻¹ - Central venous (superior vena cava) oxygen saturation $\geq 70\%$, or mixed venous $\geq 65\%$ - If venous oxygen saturation target is not achieved (2C) - Consider further fluid - Transfuse packed red blood cells if required to hematocrit of $\geq 30\%$ and/or - Start dobutamine infusion maximum 20 $\mu\text{g}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ <i>Diagnostics</i> - Obtain appropriate cultures before starting antibiotics provided this does not significantly delay antimicrobial administration (1C) - Obtain two or more BCs - One or more BCs should be percutaneous - One BC from each vascular access device in place <48 hrs - Culture other sites as clinically indicated - Perform imaging studies promptly to confirm and sample any source of infection, if safe to do so (1C) <i>Antibiotic therapy</i> - Begin intravenous antibiotics as early as possible and always within the first hour of recognizing severe sepsis (1D) and septic shock (1B) - Broad-spectrum: One or more agents active against likely bacterial/fungal pathogens and with good penetration into presumed source (1B) - Reassess antimicrobial regimen daily to optimize efficacy, prevent resistance, avoid toxicity & minimize costs (1C) - Consider combination therapy in Pseudomonas infections (2D) - Consider combination empiric therapy in neutropenic patients (2D) - Combination therapy ~ 3–5 days and de-escalation following susceptibilities (2D) - Duration of therapy typically limited to 7–10 days: Longer if response slow or there are unremovable foci of infection or immunologic deficiencies (1D) - Stop antimicrobial therapy if cause is found to be noninfectious (1D) <i>Source identification and control</i> - A specific anatomic site of infection should be established as rapidly as possible (1C) and within first 6 hrs of presentation (1D) - Formally evaluate patient for a focus of infection amenable to source control measures (e.g., abscess drainage, tissue debridement) (1C) - Implement source control measures as soon as possible following successful initial resuscitation (1C) (exception: infected pancreatic necrosis, where surgical intervention is best delayed) (2B) - Choose source control measure with maximum efficacy and minimal physiologic upset (1D) - Remove intravascular access devices if potentially infected (1C)
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Hemodynamic Support and Adjunctive Therapy

Strength of recommendation and quality of evidence have been assessed using the GRADE criteria, presented in parentheses after each guideline.

- Indicates a strong recommendation or "we recommend"
- Indicates a weak recommendation or "we suggest"

Fluid therapy

- Fluid-resuscitate using crystalloids or colloids (1B)
- Target a CVP of ≥ 8 mm Hg (≥ 12 mm Hg if mechanically ventilated) (1C)
- Use a fluid challenge technique while associated with a hemodynamic improvement (1D)
- Give fluid challenges of 1000 mL of crystalloids or 300–500 mL of colloids over 30 min. More rapid and larger volumes may be required in sepsis-induced tissue hypoperfusion (1D)
- Rate of fluid administration should be reduced if cardiac filling pressures increase without concurrent hemodynamic improvement (1D)

Vasopressors

- Maintain MAP ≥ 65 mm Hg (1C)
 - Norepinephrine and dopamine centrally administered are the initial vasopressors of choice (1C)
 - Epinephrine, phenylephrine, or vasopressin should not be administered as the initial vasopressor in septic shock (2C). Vasopressin 0.03 units/min may be subsequently added to norepinephrine with anticipation of an effect equivalent to norepinephrine alone.
 - Use epinephrine as the first alternative agent in septic shock when blood pressure is poorly responsive to norepinephrine or dopamine (2B)
 - Do not use low-dose dopamine for renal protection (1A)
 - In patients requiring vasopressors, insert an arterial catheter as soon as practical (1D)
- #### Inotropic therapy
- Use dobutamine in patients with myocardial dysfunction as supported by elevated cardiac filling pressures and low cardiac output (1C)
 - Do not increase cardiac index to predetermined supranormal levels (1B)

Steroids

- Consider intravenous hydrocortisone for adult septic shock when hypotension responds poorly to adequate fluid resuscitation and vasopressors (2C)
 - ACTH stimulation test is not recommended to identify the subset of adults with septic shock who should receive hydrocortisone (2B)
 - Hydrocortisone is preferred to dexamethasone (2B)
 - Fludrocortisone (50 μ g orally once a day) may be included if an alternative to hydrocortisone is being used that lacks significant mineralocorticoid activity. Fludrocortisone is optional if hydrocortisone is used (2C)
 - Steroid therapy may be weaned once vasopressors are no longer required (2D)
 - Hydrocortisone dose should be ≤ 300 mg/day (1A)
 - Do not use corticosteroids to treat sepsis in the absence of shock unless the patient's endocrine or corticosteroid history warrants it (1D)
- #### Recombinant human activated protein C
- Consider rhAPC in adult patients with sepsis-induced organ dysfunction with clinical assessment of high risk of death (typically APACHE II ≥ 25 or multiple organ failure) if there are no contraindications (2B, 2C for postoperative patients)
 - Adult patients with severe sepsis and low risk of death (typically, APACHE II < 20 or one organ failure) should not receive rhAPC (1A)

Other Supportive Therapy of Severe Sepsis

Strength of recommendation and quality of evidence have been assessed using the GRADE criteria presented in parentheses after each guideline.

- Indicates a strong recommendation, or "we recommend"
- Indicates a weak recommendation, or "we suggest"

Blood product administration

- Give red blood cells when hemoglobin decreases to <7.0 g/dL (<70 g/L) to target a hemoglobin of 7.0–9.0 g/dL in adults (1B). A higher hemoglobin level may be required in special circumstances (e.g., myocardial ischemia, severe hypoxemia, acute hemorrhage, cyanotic heart disease, or lactic acidosis)
- Do not use erythropoietin to treat sepsis-related anemia. Erythropoietin may be used for other accepted reasons (1B)
- Do not use fresh frozen plasma to correct laboratory clotting abnormalities unless there is bleeding or planned invasive procedures (2D)
- Do not use antithrombin therapy (1B)
- Administer platelets when (2D)
 - Counts are $<5000/\text{mm}^3$ ($5 \times 10^3/\text{L}$) regardless of bleeding
 - Counts are 5000–30,000/ mm^3 ($5-30 \times 10^3/\text{L}$) and there is significant bleeding risk.
 - Higher platelet counts ($>50,000/\text{mm}^3$ [$50 \times 10^3/\text{L}$]) are required for surgery or invasive procedures

Mechanical ventilation of sepsis-induced ALI/ARDS

- Target a tidal volume of 6 mL/kg (predicted) body weight in patients with ALI/ARDS (1B)
- Target an initial upper limit plateau pressure ≤ 30 cm H₂O. Consider chest wall compliance when assessing plateau pressure (1C)
- Allow PEEP to increase above normal, if needed, to minimize plateau pressures and tidal volumes (1C)
- Set PEEP to avoid extensive lung collapse at end-expiration (1C)
- Consider using the prone position for ARDS patients requiring potentially injurious levels of PEEP or plateau pressure, provided they are not put at risk from positional changes (2C)
- Maintain mechanically ventilated patients in a semirecumbent position (head of the bed raised to 45°) unless contraindicated (1B), between 30° and 45° (2C)
- Noninvasive ventilation may be considered in the minority of ALI/ARDS patients with mild to moderate hypoxemic respiratory failure. The patients need to be hemodynamically stable, comfortable, easily arousable, able to protect/clear their airway and expected to recover rapidly (2B)
- Use a weaning protocol and an SBT regularly to evaluate the potential for discontinuing mechanical ventilation (1A)
- SBT options include a low level of pressure support with continuous positive airway pressure 5 cm H₂O or a T piece
 - Before the SBT, patients should:
 - be arousable
 - be hemodynamically stable without vasopressors
 - have no new potentially serious conditions
 - have low ventilatory and end-expiratory pressure requirement
 - require FIO₂ levels that can be safely delivered with a face mask or nasal cannula
 - Do not use a pulmonary artery catheter for the routine monitoring of patients with ALI/ARDS (1A)
 - Use a conservative fluid strategy for patients with established ALI who do not have evidence of tissue hyperperfusion (1C)
- Sedation, analgesia, and neuromuscular blockade in sepsis
- Use sedation protocols with a sedation goal for critically ill mechanically ventilated patients (1B)
- Use either intermittent bolus sedation or continuous infusion sedation to predetermine end points (sedation scales), with daily interruption/lightening to produce awakening. Re-titrate if necessary (1B)
- Avoid neuromuscular blockers where possible. Monitor depth of block with train-of-four when using continuous infusions (1B)

Other Supportive Therapy of Severe Sepsis (Continued)*Glucose control*

- Use intravenous insulin to control hyperglycemia in patients with severe sepsis following stabilization in the ICU (1B)
- Aim to keep blood glucose < 150 mg/dL (8.3 mmol/L) using a validated protocol for insulin dose adjustment (2C)
- Provide a glucose calorie source and monitor blood glucose values every 1–2 hrs (4 hrs when stable) in patients receiving intravenous insulin (1C)
- Interpret with caution low glucose levels obtained with point of care testing, as these techniques may overestimate arterial blood or plasma glucose values (1B)

Renal replacement

- Intermittent hemodialysis and CVVH are considered equivalent (2B)
- CVVH offers easier management in hemodynamically unstable patients (2D)

Bicarbonate therapy

- Do not use bicarbonate therapy for the purpose of improving hemodynamics or reducing vasopressor requirements when treating hypoperfusion-induced lactic acidemia with pH ≥ 7.15 (1B)

Deep vein thrombosis prophylaxis

- Use either low-dose UFH or LMWH, unless contraindicated (1A)
- Use a mechanical prophylactic device, such as compression stockings or an intermittent compression device, when heparin is contraindicated (1A)
- Use a combination of pharmacologic and mechanical therapy for patients who are at very high risk for deep vein thrombosis (2C)
- In patients at very high risk, LMWH should be used rather than UFH (2C)

Stress ulcer prophylaxis

- Provide stress ulcer prophylaxis using H2 blocker (1A) or proton pump inhibitor (1B). Benefits of prevention of upper gastrointestinal bleed must be weighted against the potential for development of ventilator-acquired pneumonia

Consideration for limitation of support

- Discuss advance care planning with patients and families. Describe likely outcomes and set realistic expectations (1D)

NORMAL CARDIOVASCULAR PHYSIOLOGY**Coronary Artery Anatomy**

Major Vessel	Branch 1	Branch 2	Supply
Left main coronary artery	Left anterior descending (LAD) coronary artery	Septal branches	Anterior 2/3 of interventricular septum, apical anterior papillary muscle
	Circumflex artery	Diagonal branches Obtuse marginal branches	Anterior surface of left ventricle Lateral & posterior wall of left ventricle, anterolateral papillary muscle; Left dominant: gives rise to posterior descending artery (PDA) which supplies inferior & posterior ventricles, posterior 1/3 of interventricular septum
Right main coronary artery	Acute marginal branches AV nodal artery Usually SA nodal artery Posterior descending artery (Right dominant)		Right ventricle AV node SA node Inferior & posterior ventricles, posterior 1/3 of interventricular septum, posteromedial papillary muscle

ICU MEDICATIONS			
Drug	Class	Per kg	Dose Average
Pressors, Inotropes, and Chronotropes			
Phenylephrine	α_1	10–300 mcg/min	
Norepinephrine	α_1, β_1	1–40 mcg/min	
Vasopressin	V_1	0.01–0.1 U/min (usually 0.04)	
Epinephrine	$\alpha_1, \alpha_2, \beta_1, \beta_2$	2–20 mcg/min	
Isoproterenol	β_1, β_2	0.1–10 mcg/min	
Dopamine	D	0.5–2 mcg/kg/min 2–10 mcg/kg/min 10 mcg/kg/min	50–200 mcg/min 200–500 mcg/min 500–1000 mcg/min
Dobutamine	β_1, β_2	2–20 mcg/kg/min	30–1000 mcg/min
Milrinone	PDE inhibitors	50 mcg/kg over 10 min then 0.375–0.75 mcg/kg/min	3–4 mg over 10 min then 20–50 mcg/min
Inamrinone	PDE inhibitors	0.75 mg/kg over 3 min then 5–15 mcg/kg/min	40–50 mg over 3 min then 250–900 mcg/min
Vasodilators			
Nitroglycerin	NO	10–1000 mcg/min	
Nitroprusside	NO	0.1–10 mcg/kg/min	5–800 mcg/min
Nesiritide	BNP	2 mcg/kg IVB then 0.01 mcg/kg/min	
Labetalol	α_1, β_1 , and β_2 blocker	20 mg over 2 min then 20–80 mg q10min or 10–120 mg/h	
Fenoldopam	D	0.1–1.6 mcg/kg/min	10–120 mcg/min
Epoprostenol	Vasodilator	2–20 ng/kg/min	
Enalapril	ACE	0.625–2.5 mg over 5 min then 0.625–5 mg q6h	
Hydralazine	Vasodilator	5–20 mg q20–30min	
Antiarrhythmics			
Amiodarone	K (Class III)	150 mg over 10 min, then 1 mg/min × 6h, then 0.5 mg/min × 18h	
Lidocaine	Na channel (Class IB)	1–1.5 mg/kg then 1–4 mg/min	100 mg then 1–4 mg/min
Procainamide	Na channel (Class IA)	17 mg/kg over 60 min then 1–4 mg/min	1 g over 60 min then 1–4 mg/min
Ibutilide	K channel (Class III)	1 mg over 10 min, may repeat × 1	
Propranolol	β blocker	0.5–1 mg q5min then 1–10 mg/h	
Esmolol	β_1, β_2 blocker	500 mcg/kg then 25–300 mcg/kg/min	20–40 mg over 1 min then 2–20 mg/min
Verapamil	CCB	2.5–5 mg over 1–2 min repeat 5–10 mg in 15–30 min prn 5–20 mg/h	
Diltiazem	CCB	0.25 mg/kg over 2 min reload 0.35 mg/kg × 1 prn then 5–15 mg/h	20 mg over 2 min reload 25 mg × 1 prn then 5–15 mg/h
Adenosine	Purnergic	6 mg rapid push if no response 12 mg → 12–18 mg	

Diagrams & Flow Chart

To Calculate Variables of Cardiovascular Function		
Variable	Formula	Normal Value With Units
Cardiac index	$C.O./BSA$	2.8–4.2 L/min/m ²
SVR (FPR)	$[(MAP - CVP) \cdot 80]/C.O.$	1200–1500 dynes/cm ⁵
PVR	$[(MPAP - PCWP) \cdot 80]/C.O.$	100–300 dynes/cm ⁵
SV	$(C.O. \cdot 1000)/HR$	60–90 mL/beat
SI	SV/BSA	20–65 mL/beat/m ²
LV stroke work index	$0.0136 (MAP - PCWP) \cdot SI$	46–60 g · m/beat/m ²
RV stroke work index	$0.0136 (MPAP - CVP) \cdot SI$	30–65 g · m/beat/m ²
MAP	$DBP + (SBP - DBP)/3$	50–70 mmHg

Treatment of Coronary Artery Disease & Acute Coronary Syndromes (Continued)

STEMI	MONA morphine, O₂, nitroglycerine, ASA Medical therapy—thienopyridines (clopidogrel), heparin (UFH or LMWH), beta-adrenergic receptor blockers Adjunctive therapy: ACE inhibitors, ARBs, HMG-CoA reductase inhibitors (statins) If <12 hr since event If >12 hr since event Door to needle PCI & revascularization (fibrinolysis with tPA) goal within 48 hr goal within 30 min Door to balloon (PCI) goal within 90 min
PCIs	Persistent anginal episodes, significant stenosis of 1–2 coronary arteries or lower-risk pts with 3-vessel dz; favorable anatomy
CABG	>50% stenosis of left main coronary artery, 2–3 vessel dz with reduced LV contractile fx or diabetes

Anesthetic Goals in Valvular Disease

	Preload	Afterload	HR	SVR	Contractility
Tricuspid stenosis	High normal	High	Low	High	Maintain
Tricuspid regurg	High normal	Low	High normal	Low	Maintain
Pulm stenosis	High	High	Low	High	Maintain
Pulm insuff	High normal	Low	High normal	Low	Maintain
Mitral stenosis	High normal	High	Low	High	Maintain
Mitral regurg	High normal	Low	High normal	Low	Maintain
Aortic stenosis	High	High	Low	High	Maintain
Aortic insuff	High	Low	High normal	Low	High
CAD	Normal	High normal	Low	High	Normal
HCM	High	High	Low	High	Low
ICM	High	Low	High normal	High	High
Tamponade	High	Low	High	High	High

Indications & Contraindications for Intra-aortic Balloon Pump

Indications	Contraindications
Complications of myocardial ischemia • Hemodynamic, cardiogenic shock • Mechanical MR, VSD • Intractable dysrhythmias • Postinfarct angina Acute cardiac instability • Angina: Unstable, preinfarction • Cath lab mishap: Failed PTCA • Bridge to transplantation • Cardiac contusion • Septic shock	• Severe aortic insufficiency • Inability to insert • Irreversible cardiac dz (not transplant candidate) • Irreversible brain damage

Diagrams & Flow Chart

Generic Pacemaker Codes

Pacing Chamber	Sensing Chamber	Response to Sensing	Programmability	Multisite Pacing
O - none	O - none	O - none	O - none	O - none
A - atrium	A - atrium	I - inhibited	R - rate modulation	A - atrium
V - ventricle	V - ventricle	T - triggered		V - ventricle
D - dual (A-V)	D - dual (A-V)	D - dual (T-I)		D - dual (A-V)

Commonly Used Pacing Mode

Mode	Description	Function	Indication
AOO	Atrial asynchronous	A pacing	Bradycardias
VOO	Ventricular asynchronous	V pacing	Bradycardias
DOO	Atrial & vent. asynchronous	AV pacing	Bradycardias
VAT	Atrial sensed & vent triggered	V paced, A sensed, sensed A beat triggers V output	Complete heart block with normal SA node
VVI	Ventricular noncompetitive	V pacing on demand, sensed V beat inhibits paced V beat	Sinus node dysfunction, chronic Afib, complete block
DDD	Universal	A paced, V paced, AV sensed, sensed A inhibits A output, sensed A triggers V output, sensed V inhibits V output	Sinus brady, sinus tachy, complete block, 2° AV block

Heparin

- Mechanism: binds to antithrombin III → potentiates inhibition of factor X and thrombin
- CPB dose: 300–400 units/kg → check ACT (goal 400)
- Heparin resistance: antithrombin III deficiency, prior heparin therapy, oral contraceptive use, age
- Management of heparin resistance: Give additional dose of heparin, FFP (↑ antithrombin levels)
- Complications: Bleeding, thrombocytopenia (HIT) — heparin alternatives: Dabigatran, argatroban, bivalirudin, epoprostenol sodium

Protamine

- Mechanism: Base that ionically binds heparin (acid) by forming a stable/inactive complex
- CPB dose: 1 mg for 100 units of heparin; give small test dose first to check for allergic response (signs: Hypotension, anaphylaxis, anaphylactoid reaction, bronchospasm, pulm htn)
- Goal ACT postbypass: 120–130
- Increased risk of protamine reaction: Prior exposure, NPH/PZI insulin, rapid infusion, seafood allergy
- **Note: Never give protamine while still on bypass!**

Cardiac Pharmacology

Heart Failure Drugs			
Name	Mechanism of Action	Dosing	Comment
Milrinone	Phosphodiesterase inhibitor	50 mg/kg over 10 minute loading dose followed by 0.375–0.75 mg/kg/min. Max 1.13 mg/kg/day	Increased inotropy and vasodilation
Dobutamine	β ₁ -adrenergic stimulating drug that stabilizes the troponin molecule in cardiac muscle	12–24 mg/kg loading dose followed by 0.1–0.3 mg/kg/min for 24 hr	Increases inotropy and vasodilation. May be associated with hepatic dysfunction
Nitroglycerin (nitrate)	Recombinant form of human brain natriuretic peptide	2 mg/kg loading dose followed by 0.1 mg/kg/min for 24 to 48 hr	Venous and arterial vasodilation. Diuresis. It does not affect cardiac contractility

Diagrams & Flow Chart

Actions of Adrenergic Agonists						Dose	Comment
Sympathomimetics	$\alpha 1$	$\alpha 2$	$\beta 1$	$\beta 2$	DA 1		
Phenylephrine	+++	?	+	0	0	IV push: 50–100 mcg Drip: 0.15 mcg/kg/min	Primarily vasoconstriction
Norepinephrine	+++	---	++	0	0	Drip: 0.01–0.1 mcg/kg/min	$\beta 2$ effect present but not seen clinically
Epinephrine	+++	++	---	+	0	IV push: 0.3–0.5 mg Drip: 0.01–0.3 mcg/kg/min	High doses: α Low doses: β
Ephedrine	+	?	+	+	0	IV push: 5–10 mg	
Dopamine	- to ++	?	++	++	---	Drip: 0.05–10 mcg/kg/min	DA: 0.5–4 mcg/kg/min β : 5–10 mcg/kg/min α : >10 mcg/kg/min
Dobutamine	0 to +	+	++	++	0	Drip: 2–30 mcg/kg/min	Inotropism greater than chronotropism
Dopexamine	0	0	+	+++	+	Drip: 1 to 5 μ g/kg/min	Moderate inotropy with intense arterial vasodilation. Synthetic analog of dopamine. May protect hepatosplanchnic circulation
Isoproterenol	0	0	+++	+++	0	IV push: 0.004 mg Drip: 0.015–0.15 mcg/kg/min	

Source: Derived from Barash PG. Clinical Anesthesia, 6th ed. Lippincott Williams & Wilkins, Philadelphia, PA: 2006

Antifibrinolytics¹

Drug & Class	Mechanism	Advantages	Disadvantages	Dose
ϵ-aminocaproic acid: Lysine analog	Binds to lysine binding sites on plasminogen and fibrinogen, thereby inhibiting plasminogen activation and preventing plasmin release	• Minimal bleeding • May ↓ transfusion requirements	• No effect on resp rate • End organ safety not well established • May cause thrombosis	• Load: 100–150 mg/kg followed by infusion of 10–15 mg/kg/hr • Load: 10 g + 3: Baseline, CPB, and protoplasmin
Tranexamic acid: Lysine analog	Same as above	Ten times the potency of ϵ -aminocaproic acid	• Dosing rate not well standardized in literature • Same as above	• Low dose: Load: 10–15 mg/kg followed by infusion of 1–1.5 mg/kg/hr • High dose: Load: 100–150 mg/kg followed by infusion rate: 10 mg/kg/hr
Aprotinin: Nonspecific protease inhibitor NOTE: THIS DRUG HAS BEEN REMOVED FROM THE MARKET	Inhibits proteases kallikrein, plasmin and others leading to inhibition of intrinsic coagulation cascade, complement activation, fibrinolysis, and bradykinin formation	• Most evidence for decreased postoperative bleeding and transfusion requirements • Reduces coagulation for bleeding • May reduce stroke rate	• May be associated with renal dysfunction and transient elevated creatinine • Allergy potential IgG formation because it is derived from compound extracted from beef lung • Association with mortality not well defined • High cost	• 8-mL test dose: wait 10 min, to assess for possible anaphylactic response "high-dose regimen" • 2 × 10⁶ KIU (200 mg) over 20 min, followed by an infusion of 5 × 10⁶ KIU (70 mg) per hour • Half-dose regime is half of the high-dose above

¹NOTE: Administration of antifibrinolytics is contraindicated in patients that experience or are at risk for thrombotic complications, such as DVT, PE, MI, stroke, etc.

Technique Double-Lumen Tube Placement

1. Perform laryngoscopy to optimize glottic view
2. Insert bronchial cuff just below cords with tube bend pointed anteriorly (aids initial insertion)
3. Remove stylet
4. Advance DLT while rotating tube to midline (90° left for left DLT)
5. Inflate tracheal cuff & confirm tracheal placement
 - Breath sounds should be equal on both sides
 - No leak should be present around tracheal cuff
6. Inflate bronchial cuff CAREFULLY (rarely >2 mL air)
7. Recheck breath sounds
8. Selectively clamp & recheck R/L breath sounds
 - Changes may be difficult to detect if soft baseline breath sounds (e.g., COPD)
9. Malposition often missed on auscultation alone
10. Verify position with fiberscope
 - Verify bronchial cuff down proper mainstem
 - Airway rings most prominent anteriorly
 - Identify right-upper-lobe bronchus open
 - Ensure no bronchial cuff herniation over carina upon inflation
 - DLT often migrates with lateral/neck repositioning

Troubleshooting Double-Lumen Tubes: Both cuffs inflated & one lumen clamped

	Tracheal Side	Bronchial Side	Problem
	Ventilated	Ventilated	
Breath sounds	Clear or absent	Clear on one side, ↑ airway pressures	DLT too deep
	Absent	Both sides	DLT too shallow
	Absent or on wrong side	Wrong side only	DLT on wrong side

Diagrams & Flow Chart

Troubleshooting Double-Lumen Tubes

1. Recheck adequate balloon inflation if air leak
2. Consider returning to 2-lung ventilation (if hypoxemic/inadequate ventilation)
3. Recheck position with fibroscope
4. If large $\uparrow$ P_{AW} or no TV
 - Consider tube too shallow (bron cuffs in trachea if ventilating tracheal lumen)
 - Consider tube too deep
 - Cuff at 2nd carina, TV delivered to lobe
 - Risks: Barotrauma, pneumothorax
5. If right lung will not fully deflate, recheck patency of R upper lobe bronchus with fibroscope
6. Consider full repositioning
 - Insert fibroscope through bronchial lumen into airway
 - Deflate cuffs & pull tube back so bronchial cuff is below cords but above carina
 - Advance fibroscope down desired mainstem
 - Advance tube over fibroscope
 - Replace fibroscope into tracheal lumen to recheck tube position

Systemic Manifestations of Liver Disease

Cardiovascular	Cardiomyopathy, $\uparrow$ C.O., $\uparrow$ intravasc volume, $\uparrow$ SVR
Respiratory	Hypoxemia from intrapulmonary shunts, $\downarrow$ FRC, concurrent COPD/pneumonia, pleural effusions, resp. alkalosis
Gastrointestinal	Ascites, portal HTN, GI bleeding, $\downarrow$ gastric emptying
Renal	Renal insufficiency, hepatorenal syndrome
Hematologic	Anemia, coagulopathy, thrombocytopenia
Neurologic	Encephalopathy, neuropathy
Metabolic	Hypoglycemia, $\downarrow$ K, $\downarrow$ Na, $\downarrow$ albumin

Level of Epidural Catheter Insertion in Relation to Type of Surgery

Surgical Location	Level of Epidural Catheter Insertion
Pancreas, spleen, esophagus, stomach, liver, gallbladder, ileal loop	T7-T10
Adrenals, small intestine, colon, kidney, ureters, uterus, ovaries, & testes	T8-L1
Prostate, urethra, & rectum	L3-L4

Physical Signs of Hypovolemia

Sign	Fluid Loss (% of body weight)		
	5%	10%	15%
Sensorium	Normal	Lethargic	Obrunded
Heart rate	Normal or $\uparrow$	$\uparrow$ >100 bpm	Markedly $\uparrow$ >120 bpm
Blood pressure	Normal	Mildly $\downarrow$ with resp variation	.
Orthostatic changes in HR & BP	Absent	Present	Marked
Mucous membranes	Dry	Very dry	Parched
Urine output	Mildly $\downarrow$	$\downarrow$	Markedly $\downarrow$

Child-Pugh Classification of Severity of Liver Disease			
	Points Assigned		
Parameter	1	2	3
Ascites	Absent	Slight	Moderate
Bilirubin mg/dL	<2	2–3	>3
Albumin g/dL	>3.5	2.8–3.5	<2.8
Prothrombin time (seconds over control)	<4	4–6	>6
INR	<1.7	1.7–2.3	>2.3
Encephalopathy	None	Grade 1–2	Grade 3–4
Grade A: Total score – 5–6	→ 1- & 2-year survival = 100% & 85%		
Grade B: Total score – 7–9	→ 1- & 2-year survival = 80% & 60%		
Grade C: Total score = 10–15	→ 1- & 2-year survival = 45% & 35%		

Multisystem Involvement in Alcoholic Abuse

Cardiovascular	Dilated cardiomyopathy, hypertension
Respiratory	COPD (20% of alcohol abuse patients)
Neurologic	Cerebellar degeneration, polyneuropathy Nutritional disorders (Wernicke–Korsakoff syndrome) Delirium tremens (alcohol withdrawal)
Gastrointestinal	Esophagitis, gastritis, pancreatitis, hepatic cirrhosis
Hematologic	Anemia, thrombocytopenia
Endocrine	↓ Gluconeogenesis (hypoglycemia), hypomagnesemia

Drug-Dose Adjustment in Obesity

Some drugs have established dosing adjustments for obese pts;
 → however, it is unknown if dosing adjustments needed for most drugs

Dosage by Total Body Weight (TBW) Dosage by Ideal Body Weight (IBW)

- | | |
|--|---|
| <ul style="list-style-type: none"> • Benzodiazepine (loading dose) • Thiopental • Propofol • Opioids (loading dose) • Succinylcholine | <ul style="list-style-type: none"> • Benzodiazepine (maintenance dose) • Muscle relaxants • Opioids (maintenance dose) |
|--|---|

IBW calculation:

Men = $50 \text{ kg} + 2.3 \text{ kg} \times (\text{height [in]} - 60)$

Women = $45.5 \text{ kg} + 2.3 \text{ kg} \times (\text{height [in]} - 60)$

Obese pts typically have ↑ cardiac output, ↑ volume of distribution

Physiologic Changes During Laparoscopy

Physiologic Change	Mechanism
Respiratory	
↓ Lung compliance	Trendelenburg position, ↑ intra-abd pressure
↑ Ventilation/perfusion mismatch	↓ FRC
↑ Inspiratory pressures	Trendelenburg position, pneumoperitoneum
↑ PaCO ₂ and ↓ pH	↓ Pulm perfusion, ↓ alveolar ventilation
Cardiovascular	
↑ Systemic vascular resistance, ↑ pulm vascular resistance, ↑ mean arterial pressure	Hypercapnia, ↑ intra-abd pressure, ↑ catecholamine release
↓ Venous return	Vena cava compression
↓ Cardiac output	↓ preload, ↑ afterload
Neurologic	
* Intracranial pressure	Trendelenburg position, ↑ cerebral blood flow due to hypercapnia
Renal	
↓ Urine output	↓ Renal blood flow; * ADH secretion

Diagrams & Flow Chart

Causes of Hypotension During Laparoscopy

- Reverse Trendelenburg position
- Bleeding & hypovolemia
- High insufflation pressures
- Arrhythmias
- Myocardial ischemia
- Venous gas embolism
- Tension pneumothorax
- Tension pneumoperitoneum
- Pericardial tamponade

Complications of Carotid Endarterectomy

- HTN: Damage to (or local anesthetic at) carotid sinus; ↑ risk for neuro deficits compared to pts with normal BP (due to hyperperfusion); more likely with GA (vs regional)
- Hypotension: Removal of plaque → inc stimulation of baroreceptors; more likely with regional
- MI: Most frequent cause of morbidity/mortality
- Stroke: Usually embolic
- Bleeding: Can lead to airway obstruction from hematoma or edema
- CNS injury: 10% pts; most common nerves—hypoglossal, vagus, recurrent laryngeal, accessory
- Carotid body damage: ↓ Ventilatory response to hypoxemia/hypercapnia; esp. imp if 2nd side CEA

Glasgow Coma Scale (3–15 points)

Best Eye Opening

- Spontaneous 4
- To speech 3
- To pain 2
- None = 1

Best Verbal Response

- Oriented - 5
- Confused - 4
- Inappropriate - 3
- Incomprehensible - 2
- None - 1

Best Motor Response

- Obeys commands - 6
- Localizes pain - 5
- Withdrawals - 4
- Flexion to pain - 3
- Extension to pain - 2
- None - 1

Score of ≥ 8 indicates coma & requires intubation

Age-adjusted normal scores:

- 0–6 mo → 9
- 6–12 mo → 11
- 1–2 yr → 12
- 2–5 yr → 13
- >5 yr → 14

Diagrams & Flow Chart

Cerebral Blood Flow—Metabolic Rate Coupling (CBF & CMR)

- PaCO_2 (normal) = 20–80 mm Hg:
 - Hypercarbia **CBF ↑, CMR ↔** Hypocarbia **CBF ↓, CMR ↔**
 - CO_2 response improved by inhaled potent agents, preserved by IV agents
 - CO_2 response curve flattened in presence of chronic hypercapnia
- Profound hypoxia (PaO_2 = 50 mm Hg): **CBF ↑, CMR ↔**
- Temp: **CMR ↓ (5–7%°C), CBF ↓**
- Potent inhalational agents: **CMR ↓, CBF ↑** ("uncouple" metabolic link to CBF)
 - Effects may be potentiated by ↓ PaCO_2 or IV agents
 - Agents also impair blood flow autoregulation
- Nitrous oxide: **CBF ↑, CMR ↑ (regional, not global)**
 - Effect on CMR may be attenuated by combination with other agents
- IV agents (barbiturates/propofol/etomidate/benzodiazepines)
 - CMR ↓, CBF ↓** (changes small with barbitates & narcotics)
 - Generally preserve autoregulation and CO_2 responsiveness
 - Ketamine – unusual exception: **CBF ↑, CMR ↔** (may ↑ if used alone)
- Narcotics: Generally minimal effect on CBF and CMR
- Traumatic brain injury: heterogeneous response, **CMR ↓, CBF ↑ (luxury perfusion)**
 - Luxury perfusion: CBF exceeds metabolic demand, usually after infarct
 - Basal metabolic rate: 3.5 mL O_2 /100 g/min
 - Avg brain: 1400 g; Avg CaO_2 : 20 mL O_2 /100 mL blood
 - Global blood flow = 50 mL/100 g/min; ischemic blood flow = 15 mL/100 g/min

Management of increased ICP

- Hyperventilation**
 - Vasoconstriction of cerebral vasculature ↓ inflow
 - Intracerebral H^+ conc promotes flow out of brain tissue
 - Equilibration over a period of hrs will negate long-term benefits
 - O_2 delivery → brain volume at PaCO_2 26 mm Hg
 - Rapid normalization of PaCO_2 → brain edema & ↑ ICP
- Head position**
 - Head-up position (15–30°) for drainage from jugular venous system
 - Often one of most effective interventions to ↓ brain volume
 - Avoid extremes of neck rotation & related venous engorgement
- Direct drainage:** ventriculostomy, lumbar drain
- Blood pressure control:** avoid hypotension (& extreme hypotension)
- Pharmacologic agents**
 - Avoid high-dose inhaled potent agents (↓ O_2 MAC)
 - Propofol, thiopental—↑ cerebral blood flow and CMR
 - Diuretics – IV furosemide (0.1–1.0 mg/kg)
 - Potentiate effects of osmolar agents (block Na^+ reuptake → diuresis)
- Osmotic therapy**
 - Relies on intact blood-brain barrier
 - Mannitol (0.5–2 g/kg total dose) or hypertonic saline (HTS)
 - Comparative efficacy remains controversial
 - Hetastarch may be better choice in trauma resuscitation with hypovolemia/shock
 - Caution in setting of impaired cardiac fx (transient ↑ in extracellular volume) or unstable aneurysm
 - Maintain osmolar gap (mannitol) = 20 or serum osmolality = 320 mOsm/kg
 - Mannitol—↑ viscosity & ability to scavenge radicals

Potential Complications of Hyperosmolar Therapy

- Leakage of hyperosmolar soln into brain tissue with resulting ↑ in brain edema (late)
- Hypernatremia (with neurologic effects): hypokalemia
- Rapid overcorrection of a preexisting hyponatremia → central pontine myelinolysis
- Renal failure (from osmotic load to impaired kidneys)
- Pulmonary edema (due to intravascular fluid overload with impaired cardiac fx)
- Hypovolemia (from mannitol/furosemide diuresis + concomitant blood loss)

EEG Frequency Ranges

Delta rhythm (0–3 Hz)	Deep sleep, deep anesthesia, or pathologic states (e.g. brain tumors, hypoxia, metabolic encephalopathy)
Theta rhythm (4–7 Hz)	Sleep and anesthesia in adults, hyperventilation in awake children and young adults
Alpha rhythm (8–13 Hz)	Resting, awake adult with eyes closed, predominantly seen in occipital leads
Beta rhythm (>13 Hz)	Mental activity, light anesthesia

Diagrams & Flow Chart

CRANIOTOMY

Anesthetic Management

Evoked Potentials

Types of potentials:

1. EEG—depth of anesthesia, burst-suppression, isoelectric EEG, ischemia
 - Aneurysm clipping, AVM resection, carotid end-arterectomy
2. Somatosensory evoked potentials (SSEPs)—dorsal tracts & somatosensory cortex
 - Generated by peripheral nerve stimulation & central detection
 - Reliable but may not detect loss of motor function
 - Used in hinge of spine & intracranial procedures
 - Measurement compatible with most anesthetics
(Inhaled potent agents suppress more than IV agents)
3. Brainstem auditory evoked potentials (BAEPs)—depth of anesthesia & VIII
 - Posterior fossa craniotomy, acoustic neuroma resection
 - Easy to monitor via sounds directly applied to ear
 - Difficult to suppress
4. Visual evoked potentials (VEP)—monitor visual cortex/optic nerve
 - Most sensitive to suppression, rarely used
5. Motor evoked potentials (MEPs)—ventral motor pathways in the spine
 - Widely used in spine surgery, brainstem lesion resection
 - Craniotomy near motor cortex or descending tracts
 - Need to avoid long-acting muscle relaxation
6. Transcranial motor evoked potentials—motor function from cortex to endplate
 - Strict avoidance of inhalational agents
 - Etomidate/ketamine may enhance amplitude
 - Need to avoid profound muscle relaxation
7. EMG—motor potentials from spinal cord/nerves
 - Minimal effects of anesthetic agents

Sensitivity to anesthetic agents:

Cortical > deep brain > spine

Visual > cortical motor > deep motor > spinal motor > SSEPs > auditory evoked

Suppressant effects of anesthetic agents:

1. Inhalational agents—propofol > etomidate > ketamine > opioids
2. N₂O generally tolerated up to 50% when conc **STABLE** during monitoring
3. Consider TIVA (propofol/etomidate > narcotic) for easily suppressed potentials
4. Benzos in usual doses—minimal effects on monitored potentials

Awake Craniotomy

- Used for resection of tumors (motor or speech cortex) & epileptic foci
- Team approach with emphasis on communication, patience, & experience
- Critical to set pt & surgeon expectations
- Awake with variable sedation versus "asleep → awake → asleep" with LMA/ETT
 - Preference varies by center & experience
 - Simplest method is to avoid airway instrumentation
 - Only period of intense stimulation is opening/drilling/dural incision
- Patient comfort
 - Positioning with padding/pillows for optimal comfort
 - Vasoactive substances (nitroglycerin) may cause profound headache
 - Warming blanket as needed for patient comfort
 - Placement of Mayo stand over head to lift drapes off of the awake patient
- Preparation
 - IV access, monitors, & arterial line prior to blocks
 - Consider bilateral nasal trumpets (28–34 Fr) connected to O₂ for airway management
 - Topical anesthetic & vasoconstrictors to nares prior to placement
 - Consider peripheral nerve blocks
 - CN V₁—supraorbital, supraorbital
 - CN V₂—auriculotemporal, zygomaticotemporal
 - Cervical branches—posterior auricular, greater & lesser occipital n
 - Remifentanyl infusion for analgesia during block placement
 - Choice of local. Use long acting ropivacaine 0.375% with 1:200,000 epi
 - Bupivacaine in large volume may have increased risk of cardiac toxicity
 - Allow for additional local to be infiltrated by surgeons
 - Metoprolol prior to block to blunt tachycardia
- Maintenance
 - Deeper sedation only during drilling & opening of bone flap
 - Minimize sedation just before dural incision
 - Monitor CO₂—hypercarbia can contribute to brain swelling
 - Propofol/remifentanyl with spontaneous ventilation
 - Dexmedetomidine infusion is a useful adjunct
 - Minimal resp. depression can cause hypotension/brochospasm
 - Pretreat with atropine
 - Load 1 mcg/kg over 15 min & infuse 0.3–0.7 mcg/kg/hr
 - Neuromonitoring
 - Map functional cortex—usually speech area
 - Requires continuous patient feedback, communication, contact

Diagrams & Flow Chart

Sitting Craniotomy: Lounge chair, back elevated 60°; hips & knees flexed

Advantages

- Operative exposure with ↓ brain swelling/volume, used for:
 - Posterior fossa lesions/cerebellar tumors
 - Pituitary gland tumor resection
- Avoidance of prone position & improved ventilation dynamics

Disadvantages

- Risk of air embolus & paradoxical air embolus (PEO/septal defect)
- Signs: Drop in ET/CO₂, hypertension, tachycardia, ↑ ETN;
- Monitoring: precordial Doppler or continuous TEE
- Access: Place central venous access (for air extraction)
 - May be placed via antecubital vein
 - Multicatheter catheter preferred (more effective)
- Quadriplegia from extreme neck flexion & ↓ perfusion
- Hypotension from venous pooling & head elevation may result in brain ischemia (within apparently acceptable BP limits)
 - Consider transduction of arterial BP at level of tragus (middle of ear)
 - Maintain BP ± 20% baseline MAP
 - Consider support stockings to ↓ venous pooling
- Nerve injury due to positioning (e.g., arms, neck/brachial plexus)

Risk Factors for Postoperative Respiratory Failure

Coexisting lung dz: Vital capacity <2.9 L

Disease duration >6 years

Pyridostigmine dose >750 mg/day

Poorly controlled disease

TIVA or Potent Agent for ENT Procedures

Advantages of total intravenous anesthesia

1. May ↓ bleeding in operative field
2. May ↓ operative time spent clearing field to achieve adequate visualization
3. May ↓ emergence coughing & avoid disruption of surgical homeostasis/dura repair
4. May ↓ postop nausea & vomiting if propofol used

Disadvantages of total intravenous anesthesia

1. Depth monitoring: Greater interindividual pharmacodynamic & pharmacokinetic variability (compared with potent agents)
2. May ↑ risk of awareness if muscle relaxation employed
3. Processed EEG monitoring difficult (interference from forehead CT image sensors)

(continued)

TIVA or Potent Agent for ENT Procedures (Continued)

4. Undetected IV infiltration → potential for awareness & soft tissue necrosis
5. ↑ Cost of infusion medications

One approach

- Propofol (50–150 mcg/kg/min) + remifentanyl (0.1–0.3 mcg/kg/min) infusions
- Low dose potent-agent (e.g., 0.3 MAC desflurane) as amnestic adjunct
- Agents to support BP (e.g., phenylephrine infusion) as needed
- Muscle relaxation may be indicated by surgical need/movement concerns

Awake Tracheostomy**Indications**

- Acute stridor/upper airway obstruction
- Severe airway trauma
- Obstructing glottic tumor
- Severe tracheomalacia

Blocks (see Chapter 4, airway)

- Superficial cervical plexus block
- Transtracheal block
- Superior laryngeal nerve block
- Local field block

Key Points

- Psychological preparation/counseling
- Pt must be able to cooperate
- Head-up position
- Supplemental O₂ via face mask
- Sedative meds for anxiolysis only
- Maintain spontaneous vent.
- May induce GA after trach in place
- Asleep with flexible LMA & spont vent may be an option

Adjunctive Agents

Dexmedetomidine, remifentanyl, droperidol, ketamine, midazolam

Laser Surgery and Airway Fires**Airway Fire Algorithm**

- Detect fire or smoke in airway
- **Cease ventilation & extubate**
- Mask ventilation with O₂
- Inspection airway for injury
- Reintubate
- Perform fiberoptic inspection of airway
- Consider trach if significant burn injury
- Measure carboxyhemoglobin level in blood

Precautions

- Goggles—including pt
- Laser sign on door
- Use of laser-resistant tube
- • O₂ flows—FiO₂ <30% optimal
- N₂O may support combustion
- Surgeon communication
- Moist pledgers/packing at laser site
- Monitor laser power/duration

Lasers (Light Amplification Stimulated Emission of Radiation)

- Types of Laser: Nd:YAG; CO₂; KTP laser : power/penetration varies
- Laser use: Laryngeal, endometrial, endobronchial mass ablations; skin resurfacing, ophthalmologic procedures; coagulation
- Risks: Retinal damage, airway fire, vaporized infectious/tumor particles
- Advantages: highly focused with limited collateral tissue damage

Introduction to Jet Ventilation

- Used frequently in complex airway surgery
- Jet ventilation catheter eliminates need for ETT
- Subglottic & supraglottic approaches (depends on procedure & anatomy)
- Safe jet ventilation requires open airway for entrained air to escape during exhalation
- Jet ventilation can achieve effective oxygenation & ventilation
 - Brief low-volume, high-frequency pulses of O_2 exiting the jet catheter entrain ambient air → deliver larger tidal volume
- Advanced devices (e.g., Monsoon/Mistral)
 - Set FiO_2 , humidity, driving pressure (DP), frequency (f), & inspiratory time (IT)
 - Example settings: FiO_2 100%, hum 40%; DP - 22: psi - 120 bpm; IT - 40%
 - Peak airway pressure can be measured & alarms set

Introduction to Jet Ventilation (Continued)

Applications	Potential Complications	Devices
Suspension laryngoscopy	Barotrauma	Mistral
Tracheal resection	Hypercarbia	Monsoon
Limb motion in field (thoracic)	Airway desiccation	Bird
Difficult airway	Hypoxemia	Manual/Hand
Strategies		
Trans-LMA		
Supraglottic		
Sub-glottic		

Blocks for Ophthalmologic Surgery

- **Contraindications (regional):** uncooperative pt or pt with severe medical comorbidities that prevent positioning/immobility, trauma to eye, blindness in nonoperative eye, glaucoma, anticoagulation (relative)
- Advantages of local blocks over GA
 - Avoids complications/side effects of GA (e.g., decreased hemodynamic effects)
 - Useful for day surgery/office procedures (fast recovery)
 - Produce good eye akinesia & surgical analgesia
 - Minimal effect on IOP
- Disadvantages of local blocks:
 - Not suitable for all pts (children, language barrier, mentally handicapped)
 - Depends on skills/experience of anesthesiologist/ophthalmologist performing block
 - Not suitable for all types of surgery (open-eye surgery)
 - Complications (see below)
- Choice of technique varies with surgeon
- Most blocks currently performed by ophthalmologists

Block	Agent	Complications
Superficial application	Lidocaine 2%	Toxicity (high levels): Seizure/cardiac effects Epinephrine: Tachycardia
Subtenon's Block	Lidocaine 1–2% w/ Epinephrine (1:400,000)	Subarachnoid injection: Apnea
Peribulbar Block	Bupivacaine	Intravascular injection: Seizure
Retrobulbar Block (highest risk)	0.375–0.75% w/ Epinephrine (1:400,000)	Globe rupture: Proptosis/agitation Intraocular: Optic nerve damage/ blindness Chemosis Oculocardiac Reflex Vessel injury—hemorrhage/ecchymosis ↑ Intraorbital pressure/proptosis Central retinal artery occlusion Extraocular muscle injury

Oculocardiac Reflex

Cardiac reflex (bradycardia, sinus arrest, arrhythmia) with multiple triggers:
(1) ocular pressure, (2) ocular muscle stretch, (3) intense stimulation of empty orbit
extremely common in pediatric strabismus surgery

Mechanism: Afferent-trigeminal
Efferent-vagus

Treatment/Prevention: Anticholinergic (pre)-treatment (e.g., glyco/atropine).
Use of regional block with local anesthetics
Release ocular pressure/stop stimulation
Increase anesthetic depth

$$\text{Estimated CrCl} = \frac{(140 - \text{age}) (\text{body weight in kg}) (0.85 \text{ in women})}{(\text{serum Cr}) (72)}$$

Renal Failure**Acute Renal Failure**

• ↑ of Cr by ≥ 0.5 mg/dL or ↑ of Cr by $\geq 20\%$ over 2 weeks

	Prerenal	Intrinsic	Postrenal
Urine Na^+	< 10 mEq/L	> 20 mEq/L	> 20 mEq/L
Urine osmolality	500	< 350	350
FE_{Na}	1%	2%	$> 2\%$
BUN/Cr	< 20	$> 10-15$	$> 10-15$
Urine Cr/Serum Cr	40	< 20	20

Stages of CRF

Stage	Degree of Impairment	GFR
1	Normal	> 90
2	Mild	60–89
3	Moderate	30–59
4	Severe	15–29
5	Renal failure	< 15

Clinical Features of CRF

System	Manifestations
Neurologic	Peripheral/autonomic neuropathy, encephalopathy
Cardiovascular	Hypervolemia, HTN, CHF, uremic pericarditis, pericardial effusion, accelerated atherosclerosis
Pulmonary	Pulmonary edema
Gastrointestinal	Delayed gastric emptying
Hematologic	Anemia, platelet [*] /leukocyte dysfunction
Metabolic	Hyperkalemia, hypermagnesemia, hyperphosphatemia, hypocalcemia, hypoalbuminemia, metabolic acidosis
Endocrine	Glucose intolerance, hypertriglyceridemia
Musculoskeletal	Osteoporosis, osteomalacia

Commonly Used Diuretics		
Class	Site of Action	Side Effects
Osmotic (mannitol)	Proximal convoluted tubule, loop of Henle, & collecting tubule	Hypovolemia Hyperosmolality
Loop diuretics (furosemide, bumetanide, ethacrynic acid)	Thick ascending loop	Hypokalemia Metabolic alkalosis Volume contraction Ototoxicity
Thiazides (HCTZ, dyazide, metolazone)	Distal convoluted tubule	Hypokalemia Hyponatremia Metabolic alkalosis Volume contraction
Carbonic anhydrase inhibitors (acetazolamide)	Proximal convoluted tubule	Hyperchloremic metabolic acidosis
Potassium-sparing diuretics (triamterene, amiloride, aldactone)	Collecting tubule	Hyperkalemia Metabolic acidosis

GA vs Regional Simplified	
Speed	GA > regional
Recovery	Regional (peripheral nerve blocks) > GA
Amnesia	GA > regional unless adjuncts used
Cost	Variable
Reliability	GA > regional anesthesia
Long term analgesia	Regional > GA
Mortality	No difference in non-OB population
DVT incidence	GA > regional

Effect of IV Agents on SSEPs		
↓ Amplitude	No Change	↑ Amplitude
Barbiturates (+ latency)	Propofol	Ketamine
Magnesium	Narcotics	Etomidate
Ca ²⁺ antagonists	Midazolam	
	Droperidol	
	Clonidine	
	Dexmedetomidine	
Local anesthetics inhibit SSEPs (contraindicated if monitoring)		

Treatment of Thyroid Storm
• Iodine: 5 drops of 10% KI PO tid or 10 drops of Lugol's PO tid; or 1 g Nal slowly by IV drip over 24 h • Propylthiouracil: 600 mg PO given before iodine, then 400 mg q6h • Propranolol: 40 mg PO qid; or 1 mg slowly IV q4h under close monitoring; do not exceed 1 mg/min; repeat 1-mg dose may be given after 2 min. or esmolol infusion • IV dextrose solutions • Correction of dehydration & electrolyte imbalance • Cooling blanket for hyperthermia • Antiarrhythmic drugs (e.g., Ca channel blockers, adenosine, β-blockers) if necessary for atrial fibrillation • Treatment of underlying dz. such as infection • Corticosteroids: Hydrocortisone 100 mg IV q8h or dexamethasone 8 mg IV qd

THYROID GLAND

Laboratory Evaluation of Thyroid Function in Various Clinical Situations				
Physiologic State	Serum TSH	Serum Free T ₄	Serum T ₃	24-h Radiiodine Uptake
Hyperthyroidism, untreated	↓	↑	↑	*
Hyperthyroidism, T ₃ toxicosis	↓	Normal	↑	Normal or *
1° hypothyroidism, untreated	↑	↓	↓ Or normal	↓ Or normal
Hypothyroidism 2° to pituitary dz	↓ Or normal	↓	↓ Or normal	↓ Or normal
Euthyroid, on iodine	Normal	Normal	Normal	↓
Euthyroid, on exogenous thyroid hormone	Normal	Normal on T ₄ , ↓ on T ₃	↑ On T ₃ , normal on T ₄	↓
Euthyroid, on estrogen	Normal	Normal	*	Normal
Euthyroid sick syndrome	Normal, ↓, or ↑	Normal or ↓	Low	Normal

Hypothyroidism**1° Causes**

↓ Production of thyroid hormones, Hashimoto's thyroiditis (may be assoc. with DM or Addison's), congenital causes, iodine deficiency, thyroidectomy, radioactive thyroid ablation, antithyroid med overuse, amiodarone side effect

Clinical Features

Cold intolerance, joint pains, leg swelling (due to CHF), bradycardia, depressed hypoxic & hypercapnic vent responses, abnl baroreceptor fx, hypovolemia, coarse dry skin, puffy eyes, muscle weakness, constipation, hair loss

2° Causes

↓ TSH due to isolated deficiency in TSH production by the anterior pituitary, resistance to thyroid hormones, destruction of anterior pituitary (tumor/surgery)

Diagnosis

Based on history, physical exam, TFTs

Treatment

Replacement therapy (levothyroxine)

Hyperthyroidism

Causes	Clinical Features
- Grave's dz (diffuse toxic goiter) - Toxic nodular goiter (Plummer's dz) - Substernal & multinodular goiter - Thyroiditis	- Graves' triad (visible neck mass, clinical thyrotoxicosis, exophthalmos) - Hypermetabolism (sweating, wt loss, heat intolerance, thirst) - Cardiovascular signs (high-output cardiac failure, CHF, peripheral edema, arrhythmias (v. tach & a. fib))
Diagnosis	Treatment
- History & physical exam - TFTs - Imaging studies (MRI, US)	Surgical: Total & subtotal thyroid resection (including near-total or subtotal thyroidectomy) Nonsurgical (see table below)

Medical Treatment of Hyperthyroidism

Drug	Doses	Mechanism of Action	Comments
Antithyroid drugs			
Propylthiouracil	Initial dose: 100–150 mg PO tid	Inhibit thyroid hormone synthesis; in large doses PTU blocks peripheral conversion of T_4 to T_3	Resulting ↓ in serum T_3 level reported within 4–8 h after a single 200-mg PO dose of PTU
Methimazole	Initial dose: 20–40 mg/d PO Maintenance dose: 2.5–15 mg/d PO		
Beta blockers			
Propranolol	20–80 mg PO tid; 1–2 mg IV q4–8h	Ameliorate action of T_3 & T_4 in tissues.	Control cardiac arrhythmias &
Atenolol	50–100 mg/d PO	inhibit adrenergic effects	psychomotor manifestations within minutes

(continued)

Medical Treatment of Hyperthyroidism (Continued)

Drug	Doses	Mechanism of Action	Comments
Iodides			
10% KI Lugol's solution	Lugol's soln: 3–5 gtt in water PO tid KI: 1–2 gtt in water PO bid	Blocks thyroid hormone release, inhibit T_3 , T_4 formation, ↓ thyroid gland size	Iodides reserved for treatment of thyroid storm, or for 10–14 d prior to surgery, including thyroidectomy
Steroids			
Prednisone	40–60 mg PO qd for 4–6 wk	Inhibit conversion of T_4 to T_3 ; ↓ TSH; immunosuppressive action	Can relieve symptoms of hyperthyroidism & restore serum T_3 conc. to normal within 7 d
Dexamethasone	2 mg IV q6h		
Radioactive iodine (iodine-131)			
	5–15 mCi	Destruction of thyroid gland by radiation	Max effect usually seen in 3 mo

Myxedema Coma**Clinical Signs**

- Stupor/coma
- Hypothermia
- Bradycardia
- CHF
- + Na
- ↓ BP
- Precipitated by surgery, trauma, infection, stress

Treatment

- Support ventilation & BP
- IV fluids
- Correct + Na & blood sugar
- Levothyroxine (100–500 mcg IV slowly, then 75–100 mcg IV daily) or liothyronine (synthetic T_3 —short-acting, quick dose adjustments) at 25–50 mcg slow IV infusion, then 65–100 mcg/d divided tid/qid

PARATHYROID GLANDS**Hyperparathyroidism**

1° Causes	2° Causes
Inappropriate secretion of PTH (usually parathyroid adenoma)	Secretion of PTH as response to low Ca (from other causes)
Ectopic hyperparathyroidism (pseudoparathyroidism) (PTHrP) secretion by tumor	PTH related protein
Clinical Features Muscle weakness, altered mental status, coma, CHF, HTN, compression vertebral fractures, osteoporosis, kidney stones, pancreatitis & peptic ulcers	Diagnosis 1° hyperparathyroidism: ↑ serum PTH, serum Ca >5.5 mEq/L, ionized Ca >2.5 mEq/L Imaging studies (osteoporosis, kidney stones)
Treatment Treatment: surgical; no effective medical treatment for 1° hyperparathyroidism (acute treatment goal – control elevated blood Ca; consider hemodialysis in severe cases)	

Hypoparathyroidism

Causes of Hypoparathyroidism Absence/low PTH secretion (2° to parathyroidectomy); hypoplasia/aplasia of parathyroid glands (DiGeorge syndrome); resistance of peripheral tissues to PTH action	
Clinical Features (results from ↓ Ca) Neurologic: Neuromuscular irritability, laryngospasm, inspiratory stridor, tetany, seizures, perioral paresthesia, dementia, Chvostek's sign (nip facial n → painful facial muscle twitches), Trousseau's sign (3 min tourniquet application → wrist spasm/hypocentil)	
Cardiovascular: Hypotension, prolonged QT interval, CHF Musculoskeletal: Muscle cramps & weakness	
Diagnosis Serum Ca conc <4.5 mEq/L, ionized Ca conc <2.0 mEq/L + serum PTH level	
Treatment Maintain normal serum & ionized Ca level; treat acute hypocalcemia with 100–300 mg elemental Ca = 10–30 mL of 10% Ca gluconate in 150 mL D5W IV over 10 min; initial rate is 0.3–2 mg/kg/h; chronic therapy: (1) oral Ca preparations, (2) ergocalciferol, dihydroxycholesterol (forms of vitamin D), (3) phosphate binders (zincium hydroxide)	

Medical Treatment of Hyperparathyroidism			
Agent	Mode of Action	Indications	Precautions
Normal saline 2–4 L IV daily for 1–3 d	Enhances filtration and excretion of Ca ²⁺	Severe Ca ²⁺ >14 mg/dL, 13.5 mmol/L; moderate Ca ²⁺ with symptoms	May exacerbate CHF; lowers Ca ²⁺ by 1–3 mg/dL (0.25–0.75 mmol/L)
Furosemide 10 to 20 mg IV as necessary	Inhibits calcium reabsorption in distal renal tubule	Following aggressive rehydration	Loss of K ⁺ ; dehydration if used before intravascular volume restored
Bisphosphonates —pamidronate, 60–90 mg IV over 4 h; zoledronic acid, 4 mg IV over 15 min	Inhibits osteoclast action and bone resorption	Hypercalcemia of malignancy	Nephrotoxicity; rebound Ca ²⁺ in hyperparathyroidism; Max effects at 72 hr
Calcitonin 4 to 8 U per kg IM or SQ q6 hr for 24 hr	Inhibits bone resorption; augments Ca ²⁺ excretion	Intral treatment (after rehydration) in severe Ca ²⁺	Rebound Ca ²⁺ after 24 h; vomiting, cramps, flushing
Glucocorticoids hydrocortisone, 200 mg IV daily for 3 d	Inhibits vitamin D conversion to calcitriol	Vit D intoxication, hematologic malignancies, granulomatous dx	Immune suppression, myopathy
Mithramycin , 25 mg/kg/d IV over 6 hr for 3–8 doses	Cytotoxic to osteoclasts	Rarely used in severe hypercalcemia	Marrow, hepatic, renal toxicity

Pheochromocytoma—Causes, Features, & Treatment

Causes	Etiology unknown: 25% familial, 75% sporadic (male:female ratio 2:1) Tumor arising from chromaffine tissue in medulla of adrenal gland & sympathetic ganglia
Clinical features	History of resistant HTN & diabetes Idiopathic dilated cardiomyopathy Hyperadrenergic attacks (self-limited episodes of headache, anxiety, diaphoresis, pallor)
Laboratory features	Measurement of fractionated metabolites of catecholamines, in 24-h urine: normetanephrine >900 mcg; metanephrine >400 mcg Clonidine suppression test: clonidine normally suppresses release of catecholamines from neurons but not from pheochromocytoma
Treatment	Surgical excision; pts should be stabilized preop with α- & β-blockers, IV fluids

Types of Diabetes**Diabetes Mellitus Types 1A & 1B**

- 10% of diabetics
- Insulin-dependent
- Develops early in life
- ↑ Familial risk
- Type 1A—antibodies to islet cells in >90% cases
- Type 1B—no antibodies found
- Insulin production virtually ceases

Diabetes Mellitus Type II

- 90% of diabetics
- Non-insulin-dependent
- Develops at older age
- ↑ Familial risk
- Antibodies to islet cells in 0.1–1%
- Decreased insulin production

Pharmacology of Insulin

Insulin	Onset (h)	Peak (h)	Duration (h)
Lispro, Aspart	within 15 min	1	3–4
Regular	• 1	2–4	6–8
NPH	2–2	4–8	12–16
Lente	1–3	6–14	20–
Ultralente	6	14–18	18–24
Glargine	1.5	No distinct peak	24

American Diabetes Association Recommendations for Target Inpatient Blood Glucose Concentrations

Patient Population	Blood Glucose Target	Rationale
General medical/surgical	Fasting: 90–126 mg/dL Random: < 180 mg/dL	Better outcomes, ↓ infection rates
Cardiac surgery	150 mg/dL	↓ Mortality, ↓ risk of sternal wound infections
Critically ill	80–110 mg/dL	↓ Mortality, morbidity (SICU); ↓ morbidity, ↓ length of stay (MICU)
Acute neurologic disorders	< 110 mg/dL	↓ Mortality if admission blood glucose > 110 mg/dL

Protocol for Perioperative Insulin Infusion

1. Mix 50 U regular insulin in 500 mL NS (1.0 U/h = 10 mL/h)
2. Initiate infusion at 0.5–1.0 U/h
3. Check blood glucose level q1–2h & adjust infusion rate as needed
4. Insulin infusion (U/h) = blood glucose (mg/dL)/150

Blood Glucose Conc**Infusion**

< 80 mg/dL	Turn insulin infusion off for 30 min; give 25 mL of 50% glucose; recheck level in 30 min
80–120 mg/dL	↓ Insulin infusion rate by 0.3 U/h
120–180 mg/dL	No change in insulin infusion rate
180–220 mg/dL	↑ Insulin infusion rate by 0.3 U/h
> 220 mg/dL	↑ Insulin infusion rate by 0.5 U/h

Physiologic Changes of Pregnancy

Metabolism and respiration	1. Oxygen consumption ↑ by 30-60% 2. $\dot{V}_E$ ↑ 45% and RR ↑ 10% 3. $\dot{V}_O_2$ ↑ by 55% 4. FRC, RV and ERV ↓ by 20% at term 5. $\dot{V}_E$, ERV and RV, but TLC same 6. $\dot{V}_O_2$ ↑ by 45% (progesterone effect) 7. In short $\dot{V}_O_2$ can ↑ to 300% 8. P_{aO_2} ↓ to 28-32 mm Hg — incompletely compensated respiratory alkalosis 9. $\downarrow$ O_2 content (secondary to anemia)
Circulation	1. CO ↑ by 40% blood volume ↑ 45% 2. Uterine perfusion ↑ to 700-950 mL/min (20% cardiac output) 3. BP ↓ Decrease in systolic and diastolic pressures, SVR ↓ 20%, HR ↑ 15% 4. Aortocaval compression (the gravid uterus compresses the vena cava and aorta in the supine position) this can ↓ CO by 25%, increase in uterine venous pressure, uteroplacental insufficiency and possibly fetal distress 5. Hemodynamics postpartum: CO ↑ 75%, return to prebor values by 48 h (pregnancy values by 12-24 wk) 6. ↑ volume of distribution
Hematology and coagulation	1. Blood volume ↑ 50%, increase in plasma volume increase in RBC mass — relative anemia 2. Plasma cohesiveness conc ↓ by 25% 3. Hypercoagulable state in pregnancy: ↑ platelet function, clotting and fibrinolysis 4. ↑ 2,3 DPG — right shift of oxygenoglobin curve — ↑ O_2 delivery
G.I.	1. Uterine enlargement — distortion of gastroesophageal junction and distortion of pyloric angle interfere with gastric emptying 2. ↑ LES tone, ↑ intra-abdominal pressure leads to ↑ risk of aspiration 3. Labor and narcotics — gastric emptying
CNS	1. MAC ↓ by 70-80% 2. Vasopressor response 3. Requirement for epidural & spinal loc anesthetics

Stages of Labor

Stage of labor	Events	Duration
1st	Onset of regular contractions to 10 cm cervical dilation	T ¹ 0-11
2nd	Complete dilation to delivery of infant	52-54
3rd	Delivery of infant to delivery of placenta	52-54

Common Medications Used in Obstetric Anesthesiology for Systemic Analgesia

Name	Class	Common Dosage	Duration
Morphine	Opioid	2-4 mg IV	3-4 h
Hydromorphone	Opioid	1-2 mg IM/IV	1-2 h
Meperidine	Opioid	25-50 mg IV	2-3 h
Fentanyl	Opioid	25-50 mcg IV	30-60 min
Nalbuphine	Mixed agonist/antagonist	10 mg IV	3-6 h
Butorphanol	Mixed agonist/antagonist	1-2 mg IV	3-4 h

Choices for Initiation of Labor Neuraxial Analgesia

	Advantages	Disadvantages
Combined spinal epidural (CSE)	1. Rapid onset compared with epidural, important for multiparous women in active labor 2. Minimal motor blockade 3. Improved overall maternal satisfaction compared with epidural analgesia 4. Lower probability of failed epidural catheters	1. Cannot diagnose a non-functional catheter until spinal recedes 2. Increased pruritus compared with epidural analgesia
Epidural	1. Ability to slowly titrate level 2. Ability to diagnose a functional catheter after placement (important for parturients with bad airways, morbid obesity)	1. Slower onset than CSE 2. High concentrations of local anesthetic may lead to motor blockade
Continuous spinal catheter	1. Ability to titrate level 2. Rapid onset	Increased risk of postdural puncture headache compared to CSE or epidural

Anesthesia for Cesarean Delivery		
Situation	Choices for Anesthesia	Advantages
Urgent cesarean delivery	1. Spinal/CSE 2. Epidural 3. Extending anesthesia with indwelling epidural catheter or spinal catheter 4. GA	For urgent cesarean deliveries: • If patient has an epidural or spinal catheter in situ, it may be dosed for surgery • If the patient does not have an indwelling catheter, a spinal may be performed unless there is a contraindication to regional anesthesia, in which case a general anesthetic should be performed
Emergency cesarean delivery	1. Spinal 2. Extending anesthesia with indwelling epidural or spinal catheter 3. GA 4. Local anesthesia block by surgeon (in case of inability to perform #1-3)	In emergency cesarean deliveries, decision regarding mode of anesthesia depends on maternal & fetal status: • If there is an indwelling epidural catheter, it may be dosed with a fast-onset local anesthetic such as chloroprocaine • If there is severe fetal distress and there is not an indwelling epidural catheter, there may not be time to perform a regional technique; therefore general anesthesia should be performed • If patient and fetus are stable, a regional technique should be performed

Contraindications to Regional Anesthesia

Absolute	- Patient refusal - Severe coagulopathy - Sepsis or infection at puncture site - Severe hypovolemia - Increased intracranial pressure
Relative	- Valvular cardiac lesions (severe AS or MS) - Thrombocytopenia (see below)
Controversial	- Previous back surgery - Thrombocytopenia (thresholds vary by institution: Beilin in a retrospective review found no incidence of neurologic complications in women with platelet counts of 69,000–80,000 at time of neuraxial analgesia placement (Anesth Analg 1997;85:385–388))

Morbidity and Mortality of GA in Obstetric Population

1. Anesthesia-related complications are 6th leading cause of pregnancy-related deaths in U.S.
2. Incidence of failed intubation is 1:280 (8 × higher than nonpregnant population)
3. 73% of anesthesia-related maternal deaths occurred during general anesthesia
4. Majority of women who died from complications of general anesthesia primarily died from airway management problems, including aspiration, induction, or intubation problems; inadequate ventilation; and respiratory failure

Source: Adapted from Anesthesiology 1997;86:277–284.

General Anesthesia: Anesthetic Implications of Maternal Physiologic Changes

Endotracheal intubation

- Smaller endotracheal tubes required (may have airway edema)
- Increased risk of trauma with nasotracheal intubation
- Increased risk of failed intubation

Maternal oxygenation

- Increased physiologic shunt when supine
- Increased rate of denitrogenation
- Increased rate of decline of PaO₂ during apnea

Maternal ventilation

- Increased minute ventilation required

From: Shnider SM, Levinson G. Anesthesia for Cesarean Section. In: Anesthesia for Obstetrics (3rd ed; Shnider SM, Levinson G eds.) 1993:211–246.

Common Medications Used in Neuraxial Analgesia & Anesthesia	
Scenario	Common Regimens
Labor analgesia initiated by CSE technique	• 25 mcg fentanyl or 10 mcg sufentanil • 2.5 mg bupivacaine with 15 mcg fentanyl
Labor analgesia initiated with epidural technique	Common initial epidural dosing regimens include local anesthetic ± fentanyl 50–100 mcg: • Bupivacaine 0.125–0.25% (15 mL) • Ropivacaine 0.1–0.2% (6–10 mL) • Lidocaine 1% (6–10 mL)
Common test dose regimens	• Lidocaine 1.5% + epi 1:200,000 (3 mL) • Fentanyl 100 mcg • Air (1 mL) while monitoring precordial Doppler • 3% 2-chloroprocaine (3 mL)
Maintenance infusion regimens for epidural analgesia	• Bupivacaine 0.04–0.125% with fentanyl 1–2 mcg/mL at 8–15 mL/h • Bupivacaine 0.125% plain at 8–15 mL/h
Epidural bolus	• Bupivacaine 0.25–0.5% — 5–10 mL • Bupivacaine 0.06–0.125% — 8–15 mL • Add fentanyl 1–3 mcg/mL or sufentanil 0.1–0.5 mcg/mL
Maintenance infusion regimen with spinal catheter	Bupivacaine 0.06% at 1–2 mL/h

Common Medications Used in Neuraxial Analgesia & Anesthesia (Continued)

Scenario	Common Regimens
Spinal for cesarean delivery	Local anesthetic: bupivacaine 8–12 mg or lidocaine 50–70 mg with fentanyl 15 mcg, † morphine 150 mcg, † epinephrine 100–200 mcg
Epidural for cesarean delivery	3% 2-chloroprocaine or 2% lidocaine with bicarbonate & epinephrine (15–20 mL total)
Epidural morphine for postoperative pain	3–4 mg

Labor Neuraxial Analgesia Management

Typical Sequence of Events	Remarks
1. Request for analgesia from obstetrician	No evidence to support delay of epidural placement until an arbitrary cervical dilation
2. Perform preanalgesia evaluation, including physical exam, obtain consent	Per ASA practice guidelines for OB anesthesia: a focused H&P may be associated with ↓ maternal, fetal & neonatal complications
3. Administer antacid prophylaxis	Patients should receive a nonparticulate antacid
4. Wash your hands, use aseptic technique	
5. Place BP cuff and pulse oximeter; take baseline BP; consider fluid bolus	Measure the blood pressure every 2–3 min during procedure
6. Position the patient	Sitting or lateral position are acceptable Sitting may facilitate landmark identification in obese patients
7. If initiating with CSE, administer intrathecal medications	See table above on (Common Medications Used in Neuraxial Analgesia and Anesthesia) for common dosing regimens
8. Place epidural catheter, administer test dose to assess for intravascular/ intrathecal catheter	
9. If initiating using epidural technique, after a negative test dose, bolus local anesthetic through the epidural catheter	
10. Start epidural infusion	Maintenance regimens include intermittent bolus dosing of epidural catheter or continuous infusion of medications = PCEA See table above (Common Medications Used in Neuraxial Analgesia and Anesthesia)
11. Monitor BP for 15–20 min after injection	Both ephedrine and phenylephrine are acceptable vasopressors for use in L&D
12. Follow up until delivery	Monitor maternal vital signs, motor blockade & level of analgesia every 2–3 hr; if inadequate analgesia, administer bolus dose of epidural medication

Source: Adapted from *N Engl J Med* 2005;352:655–665 & *Anesthesiology* 2007;106:843–863

Regional Anesthesia: Anesthetic Implications of Maternal Physiologic Changes

Technical considerations

- Lumbar lordosis increased
- CSF return is unaltered
- Reduced sensitivity of "hanging drop" technique

Hydration

- Increased fluid requirements to prevent hypotension*

Local anesthetic dose requirements¹

- Subarachnoid dose reduced 20% to 33%

General Anesthesia for Emergency Cesarean Delivery

Typical Sequence of Events

1. CS called by obstetrician; maximize placental perfusion & oxygenation en route to OR
2. Discuss urgency of procedure with obstetricians
3. Focused history and physical, including an airway examination
4. Preoxygenate well, prepare for a rapid sequence induction
5. Induce general anesthesia after confirmation of surgical team readiness
6. Before delivery, maintain anesthesia with N₂O 50% or O₂ 100%, 1 MAC halogenated agent
7. After delivery, increase N₂O concentration to 66%, decrease halogenated agent to 0.5 MAC
8. Extubate once patient is awake & meets extubation criteria

Remarks

- Ensure LUD on transport & in OR
- Administer O₂ on arrival in OR
- Administer a nonparticulate antacid ASAP
- Based on urgency, decide if there is time for a regional anesthetic
- Thiopental, propofol, etomidate and ketamine all suitable for induction. Succinylcholine or high-dose of NMBD if there is a contraindication to succinylcholine
- Opioids & benzodiazepines may be administered after delivery of the infant

Anesthesia for Cesarean Delivery

Typical Sequence of Events

1. Set up OR
2. Perform preanesthetic evaluation, including physical examination, obtain consent; assess need for lab work & blood availability
3. Administer antacid prophylaxis (regardless of the planned mode of anesthesia)
4. Prehydrate patient
5. ASA standard monitors, position patient for spinal or epidural
6. Wash your hands, use aseptic technique
7. Place spinal or epidural
8. If the patient has an indwelling epidural catheter, dose the epidural catheter
9. Consider epidural morphine after delivery of the infant
10. Place patient in left uterine displacement position (LUD)
11. Ascertain sensory level & document
12. Treat hypotension
13. Administer prophylactic antibiotics if appropriate
14. After the umbilical cord is clamped, administer oxytocin

Remarks

- Per ASA guidelines for OB anesthesia, a focused H&P may be associated with maternal, fetal, & neonatal complications
- Patients should receive an H₂ receptor antagonist or proton pump inhibitor, metoclopramide, and 1 nonparticulate antacid
- 10–15 mL/kg crystalloid should be administered 0–15 min before initiation of regional blockade
- Sitting or lateral position both acceptable. Sitting may facilitate landmark identification in obese patients
- See table (page 25-3) Common Medications Used in Neuraxial Analgesia and Anesthesia
- Choice of local anesthetic depends on urgency of delivery: 3% CP has a faster onset than 2% lidocaine, therefore may be used for emergency cesarean deliveries
- Epidural morphine may be used for postoperative analgesia (has an assoc risk of postpartum resp. depression, especially if combined with other systemic analgesics)
- Use a wedge or a towel
- T4–T6 sensory level should be obtained prior to surgical incision
- Ephedrine 5–10 mg or phenylephrine 100 mcg
- 10–20 U oxytocin in 500 mL LR

General Anesthesia: Pharmacology During Pregnancy***Inhalation anesthetics**

- Minimum alveolar concentration reduced 20% to 40%
- Rate of induction increased

Induction agents

- ED₅₀ of thiopental reduced 35%
- Elimination half-life of thiopental prolonged
- Elimination half-life of propofol unaltered

Meperidine

- Elimination half-life unaltered

Succinylcholine

- Duration of blockade unaltered (or decreased)
- Sensitivity reduced

Nondepolarizing muscle relaxants

- ED₅₀ of vecuronium reduced
- Elimination half-life of vecuronium and pancuronium shortened
- Duration of blockade of atracurium unaltered

Chronotropic agents

- Response diminished

Pressors

- Response variable

Anesthesia for Cervical Cerclage Placement

Anesthesia for cerclage placement similar to anesthesia for cesarean delivery

Common intrathecal regimens include

- Bupivacaine 0.5–0.75% 12.5 mg ± fentanyl 15–25 mcg
- Lidocaine 5% 50–75 mg – fentanyl 15–25 mcg

Anesthesia for Multiple Gestations

Exaggerated physiologic & anatomic changes of pregnancy compared with singleton pregnancy

- ↓ TLC, ↓ FRC, ↑ maternal metabolic rate → more rapid development of hypoxemia
- ↑ Weight gain
- ↑ Risk of aspiration (↓ LES tone)
- Blood volume ↑s an additional 500 mL
- ↑ C.O.

↑ Maternal complications

- ↑ Risk of preterm labor, PROM
- ↑ Risk of preeclampsia
- ↑ Perineal trauma
- ↑ Risk of uterine atony, antepartum & postpartum hemorrhage

Anesthetic management for vaginal delivery

- Epidural analgesia for labor
- Uterine or cervical relaxation may be necessary for delivery of twin B

Anesthetic management for cesarean delivery

- Regional anesthetic preferred technique
- If GA, adequate preoxygenation is required
- Adequate IV access, T&S

Anesthetic Implications of Breech Delivery

Choice of anesthesia depends on mode of delivery

Most breech infants are delivered by cesarean section, usual anesthetic is a spinal blockade

For breech vaginal deliveries, epidural analgesia may be initiated

Cord prolapse

If cord prolapse occurs, anesthesia must be immediately initiated (either epidural or GA)

Head entrapment

Fetal head may become entrapped in cervix; IV nitro or GA can be used to facilitate smooth muscle relaxation, followed by high-conc volatile anesthetic

Post-Dural Puncture Headache

Symptoms	Onset & Duration	Treatment
Positional headaches (worse when sitting/standing, improved when supine) Frontal, occipital, or generalized headaches Associated symptoms include diplopia/photophobia, nausea, vomiting, neck stiffness, tinnitus	Onset of headache typically 24–48 h after dural puncture Duration usually 7–14 d	Conservative management includes: 1. ↑ Fluid intake 2. ↑ Caffeine intake. 3. Oral analgesics such as Fioricet (contains acetaminophen, butalbital, caffeine). NSAIDs Gold standard: Epidural blood patch: 15–30 mL blood sterily injected into epidural space; relief often immediate, can take 24 hr

Implications for Vaginal Birth After Cesarean Section (VBAC)

1. 1% risk of uterine rupture—signs include fetal distress, abdominal pain, uterine tenderness, cessation of uterine contractions, palpable fetal parts in abdomen
2. Continuous FHR monitoring, consider intrauterine pressure monitoring
3. IV access
4. Type & screen or cross-match blood available
5. Epidural analgesia **not** contraindicated; breakthrough pain that is not relieved between uterine contractions may be indicative of uterine rupture

Common Obstetric Drugs			
Drug	Indication	Mechanism	Side Effects
Ephedrine	Hypotension	α - and β -agonist (indirect)	HTN, arrhythmias, myocardial ischemia, $\downarrow$ uterine blood flow
Phenylephrine	Hypotension	α -agonist (direct)	HTN, reflex bradycardia, inc. uterine vascular resistance
Oxytocin	Stimulate uterine contractions	Activates myometrial oxytocin receptors, increases sodium permeability	Hypotension, tachycardia, flushing, antidiuretic effect
Prostaglandins (15-methyl $\text{PGF}_{2\alpha}$)	Cervical ripening, uterine atony	Activates uterine smooth muscle contractions	Bronchoconstriction, vasoconstriction, HTN, nausea, vomiting, diarrhea
Ergot alkaloids (methylergonovine)	Uterine atony	Direct activation of uterine smooth muscle	Arterial and venous vasoconstriction, HTN, coronary vasoconstriction, bradycardia
Terbutaline	Inhibit uterine contractions (tocolysis)	β -2 receptor agonist	HTN, arrhythmias, myocardial ischemia, pulmonary edema

Uteroplacental Blood Flow

$$\text{Uterine blood flow (UBF)} = \frac{\text{uterine arterial pressure (UAP)} - \text{uterine venous pressure (UVP)}}{\text{uterine vascular resistance (UVR)}}$$

Factors That Decrease UBF**Decrease in UAP**

- Any factor that causes maternal hypotension will cause $\downarrow$ in UAP
- UAP is directly proportional to maternal MAP

Increase in UVP**Increase in UVR**

1. Aortocaval compression
2. Hypovolemia 2° to hemorrhage
3. Sympathectomy
1. Vena cava compression
2. Uterine contractions/uterine hypertonus
3. Skeletal muscle hypertonus (Valsalva)
1. Catecholamine release
2. Vasopressin release 2° to hypovolemia
3. Vasopressors (ephedrine)

Drugs That Do Not Cross the Placenta

Muscle relaxants	NMBD's cannot cross owing to high molecular weight & ionization
Anticoagulants	Heparin & LMWH cannot cross owing to high molecular weight
Anticholinergics	Glycopyrrolate does not cross easily; atropine does
Insulin	Insulin cannot cross placenta owing to high molecular weight

Antihypertensive Drugs Used to Prevent or Treat Hypertension During General Anesthesia					
Drug	Administration and Dose	Onset and Duration of Action	Effect on Uterine and Placental Blood Flow	Special Properties	
				Advantages	Disadvantages
Hydralazine (arteriolar vasodilator)	IV bolus, 5–10 mg	Maximum effect requires 20–30 min after IV administration. Duration about 2 hr	Originally thought to improve flow, this has now been questioned	1. Easy to administer; no special equipment or monitoring required 2. Maintains maternal cardiac output 3. Long history of safe use in obstetrics	1. Slow, unreliable onset 2. Maternal tachycardia 3. Decreased placental blood flow; fetal distress 4. Neonatal thrombocytopenia 5. Maternal nausea
Labetalol (α_1 and β blocker) (β_2 agonist)	IV boluses: 10–20 mg up to total of 1–3 mg/kg	IV onset, 1–2 min; Duration, 2–3 hr	Improves uterine and placental blood flow	1. Easy to administer; no special equipment or monitoring 2. Little risk of overshoot 3. No associated fetal bradycardia or distress 4. Improved placental blood flow 5. Few maternal side effects 6. Rapid onset 7. Becoming widely used by obstetricians and anesthesiologists	1. Large variation in effective dose 2. Alone, may not effectively $\downarrow$ BP 3. Use with caution in patients with asthma, COPD, or compromised ventricular function
Nitroglycerin (venodilator)	Constant IV infusion: 5–50 μ g/min infusion; 25–50 μ g/500 ml (50–100 μ g/ml)	Onset less than 2 min; duration only a few minutes	Questionable, depends on state of maternal hydration; has been associated with fetal deterioration	1. Rapid onset and dissipation	1. Need IV pump to administer 2. Should make up in glass 3. May need arterial line 4. Great variation in response

Antihypertensive Drugs Used to Prevent or Treat Hypertension during General Anesthesia (Continued)					
Drug	Administration and Dose	Onset and Duration of Action	Effect on Uterine and Placental Blood Flow	Advantages	Special Properties Disadvantages
Trimethaphan Ganglionic blocker	IV boluses 1–4 mg IV infusion: 0.3–5 mg/min (1 mg/ml)	IV onset less than 1 min; duration less 5 min	Minimal, if no severe maternal hypotension	1. Reliable, rapid acting 2. Large molecular weight limits total transfer 3. Rapid offset	1. May require arterial line for BP monitoring 2. Interferes with action of pseudo-cholinesterase resulting in prolonged duration of succinylcholine 3. Histamine release (?) 4. May cause mydriasis
Nitroprusside Direct-acting arterial vasodilator	Constant IV Infusion: 0.15–10 µg/kg/min Infusion: 50 mg/500 ml 5% dextrose in water (100 µg/ml)	IV onset less than 1 min; duration only a few minutes	Dilates uterine artery in vitro; no ill effects unless severe hypotension present	1. Rapid onset and offset 2. Potent reliable antihypertensive 3. No ill effects on fetus	1. Unstable solution, protect from light 2. Easy to overshoot, need arterial line 3. Difficult to titrate 4. Increased intracranial pressure 5. Cyanide toxicity: not a problem with short-term use and infusion • 3 µg/kg/min 6. Tachyphylaxis

Assessment of the Newborn: Apgar Score			
Score	0	1	2
Color	Pale or white	Pink body, peripheral acrocyanosis	Pink
HR	Absent	<100	>100
Response to stimulation	None	Grimace	Cough, sneeze
Muscle tone	Flaccid	Some movement	Moving
Respiration	None	Weak, irregular	Crying, regular

Interpretation of Apgar Score

Normal: 7–10

Moderate impairment: 4–6

Needs resuscitation: 0–3

Antepartum Hemorrhage

Placenta previa
(placenta located over internal os)

- Risk factors: Multiparity, age, previous CS, previous previa
- Presentation: Painless vaginal bleeding in 2nd or 3rd trimester
- Diagnosis: US versus MRI
- Anesthetic mgmt: Large-bore IV access, assess volume status, T&C blood
- Choice of anesthesia: GA vs regional, consider 1 risk of accreta if h/o prev CS
- Risk factors: HTN, age, tobacco use, cocaine use, trauma, PROM, h/o previous abortion
- Presentation: Painful vaginal bleeding, 1 uterine activity, uterine tenderness
- Anesthesia mgmt: Large-bore IV access, assess volume status, assess coagulation status as placental abruption is assoc. with consumptive coagulopathy, T&C blood
- Choice of anesthesia: GA vs regional
- Placenta accreta: Abnormally adherent placenta
- 3 subtypes:
 1. Placenta accreta: Adherence to myometrium, not invading muscle
 2. Placenta increta: Placenta invades uterine muscle
 3. Placenta percreta: Placenta invades uterine serosa or other pelvic structures
- Risk factors: Previous CS, placenta previa
- Diagnosis: Antepartum dx can be made with US/MRI intrapartum dx with difficulty in separating placenta or at laparotomy
- Anesthesia mgmt: Large-bore IV access, assess volume status, assess coagulation status, T&C blood
- Choice of anesthesia: GA vs regional
- Incidence: <1% of pts with previous uterine surgery
- Presentation: Vaginal bleeding, hypotension, fetal distress, $\pm$ pain
- Risk factors: Previous uterine surgery, uterine trauma
- Treatment: Emergent CS
- Anesthetic mgmt: Large bore IV access, assess volume status, assess coagulation status, T&C blood, GA or extension of epidural anesthesia if pt is hemodynamically stable
- Risk factors: Placenta previa, multiple gestation, abnormal placenta, IVF pregnancy
- Obstetric management: immediate cesarean delivery
- Choice of anesthesia: GA vs regional depending on urgency of CS

Placental abruption
(separation of the placenta from the site of implantation)

Abnormal placentation
(abnormally adherent placenta)

Uterine rupture

Vasa previa
(fetal vessels transverse membranes ahead of fetal presenting part)

Postpartum Hemorrhage	
Cause	Comments
Uterine atony (risk factors: Multiple gestation, polyhydramnios, chorioamnionitis, precipitous labor, high conc of oxytocin use in labor, high conc of halogenated agents)	- Consider uterine massage - Consider uterotonic agents (oxytocin, cytotec, ergot alkaloids, or prostaglandin)
Retained placenta	- Treatment is manual removal of placenta - GA for unstable pts. otherwise consider epidural - Additional uterine relaxation may be obtained with 50–250 mcg of nitroglycerin or 1 conc of volatile agent
Abnormal placentation	See discussion above
Genital trauma (vaginal, vulvar, & cervical lacerations)	Neuraxial analgesia may be extended for repair if pt is hemodynamically stable
Uterine inversion	- Epidural/spinal anesthesia if pt hemodynamically stable - Uterine relaxation may be necessary for replacement of inverted uterus (consider iv nitroglycerin or volatile anesthetics)

Hypertensive Disorders of Pregnancy

Differential Diagnosis for Hypertension in Pregnancy

	Time of Diagnosis	Resolution After Pregnancy	Proteinuria	Comments
Gestational hypertension (gHTN)	<20 weeks' gestation	Yes	No	25% of women with gHTN may develop preeclampsia
Chronic hypertension	<20 weeks' gestation	No	No	
Pregnancy-induced hypertension	>20 weeks' gestation	Yes	No	
Preeclampsia	>20 weeks' gestation	Yes	Yes (>300 mg on 24-h urine sample or a random urine protein of 30 mg/dL)	- Seizures indicate progression to eclampsia - HELLP syndrome (see below)

Management of HELLP Syndrome

Diagnosis	HELLP (hemolysis, elevated LFTs, low platelets) severe form of preeclampsia; resolution of hemolysis/thrombocytopenia may require 24–72 h
Anesthetic management	Delivery = only treatment for HELLP syndrome—follow management for preeclampsia If parturient is thrombocytopenic, use neuraxial analgesia/anesthesia with caution

Severity of Preeclampsia			
	Blood Pressure Criteria	Proteinuria	Comments
Mild preeclampsia	sBP $\geq$ 140 or 30 mm Hg above normal dBP $\geq$ 90 or 15 mm Hg above normal	$\geq$ 5 g/24 h	Mild preeclampsia may progress to severe preeclampsia; may lead to peripheral edema
Severe preeclampsia	sBP $\geq$ 160 or dBP $\geq$ 110	$\geq$ 5 g/24 h	End-organ symptoms may include HA, visual changes, RUQ pain, lab abnormalities ($\uparrow$ LFTs, thrombocytopenia), oliguria, or IUGR

Anesthetic Management of Preeclampsia		
Intrapartum management of HTN	Hemodynamic goals include prevention of severe HTN (risk of cerebral hemorrhage) while still maintaining adequate placental perfusion	Hydralazine, labetalol, methyldopa, sodium nitroprusside, or nitroglycerin
Seizure prophylaxis	Mg (4-g bolus, followed by an infusion). Mg $\uparrow$ sensitivity to NMBD & $\downarrow$ response to vasopressors	Serum Mg ²⁺ conc: 4–6 mEq/L $\rightarrow$ therapeutic 5–10 mEq/L $\rightarrow$ QRS widening 10 mEq/L $\rightarrow$ $\downarrow$ deep tendon reflexes 15 mEq/L arrhythmias, respiratory weakness 25 mEq/L cardiac arrest Treatment for Mg toxicity = calcium chloride
Coagulation status	Preeclamptics may become thrombocytopenic due to platelet aggregation & consumption	Check platelet count, check coagulation status if elevated LFTs If pt is not thrombocytopenic or coagulopathic, may use regional anesthesia/analgesia
Monitoring	Consider arterial BP monitoring in pts with severe HTN, or CVP monitoring if volume status unclear or pt is oliguric	

Anesthetic Considerations for Postpartum Tubal Ligation

NPO status	NPO guidelines apply (see Chapter 1, on Preop Evaluation) Gastric emptying ↓ if opioids have been used
Risk of postpartum hemorrhage (PPH)	Pts at risk for PPH for 24 h Consider uterine bleeding if pt is hemodynamically unstable intraoperatively
Local anesthetic requirements	↓ Requirement; returns to prepregnancy value after 12–36 h postpartum
Regional vs general anesthesia	Regional preferred (spinal or epidural if in situ) Risk of epidural catheter failure ↑ if delivery to surgery interval >8–10 h

Anesthetic Goals for Nonobstetric Surgery in the Pregnant Patient*

1. Maintain placental perfusion	Avoid maternal hypotension, hypocarbia, hypoxemia, catecholamines
2. Avoid preterm labor	
3. Avoid possible teratogens	Highest risk in 3rd trimester; benzodiazepines, N ₂ O, avoid ketamine in 1st trimester (although no evidence that anesthetic drugs cause problems with organogenesis)
4. Antacid prophylaxis	
5. Choice of anesthesia (regional vs GA)	Use regional when possible; if GA, RSI
6. FHR monitoring	Monitor pre- & postop after '16 weeks' gestation

Anesthetic Considerations for Gynecologic Surgery			
Procedure	Indication	Positioning	Choice of Anesthesia
1. Hysteroscopy	1. Eval abnormal uterine bleeding 2. Eval cause of infertility	Lithotomy	MAC, GA (LMA or ETT), or neuraxial anesthesia
2. Dilation and curettage (D&C)	1. Eval of uterine bleeding 2. Incomplete or missed abortion	Lithotomy	Paracervical block, MAC, spinal, or GA (LMA or ETT)
3. Dilation and evacuation (D&E)	1. 2nd-trimester abortion (for fetal or maternal indications)	Lithotomy	Paracervical block, MAC, spinal, GA (LMA or ETT)
4. Conization of the cervix (LEEP procedure)	1. Diagnosis & treatment of cervical dysplasia	Lithotomy	MAC, GA (LMA or ETT), spinal
5. Total abdominal hysterectomy (TAH) ± BSO	1. Uterine or cervical cancer 2. Fibroids unresponsive to hormone therapy 3. Endometriosis	Supine	GA (ETT) = epidural for postop pain relief, or neuraxial anesthesia (epidural)
6. Vaginal hysterectomy		Lithotomy	
7. Robotic-assisted laparoscopic hysterectomy		Steep Trendelenburg	GA (ETT)
Anesthetic Considerations			1. Procedure generally short 2. Consider antiemetic prophylaxis 3. Potential for fluid overload (uterus often distended with a viscous fluid) 1. Consider antiemetic prophylaxis 2. Rare risk of uterine perforation or hemorrhage 1. Consider antiemetic prophylaxis 2. Rare risk of uterine perforation or hemorrhage 1. Often short procedures; do not require anesthesia 1. If hx of chemo, consider cardiac & pulm implications 2. Consider invasive monitoring 3. Potential for blood loss—T&S or cross-match Same considerations as for TAH & laparoscopic surgery

Anesthetic Considerations for Gynecologic Surgery (Continued)				
Procedure	Indication	Positioning	Choice of Anesthesia	Anesthetic Considerations
8. Laparoscopic surgery	May be used for many surgeries (e.g., ectopic pregnancy, hysterectomy, postpartum tubal ligation)	Trendelenburg	GA with ETT	1. Possible resp compromise from CO₂ insufflation (usually 15 mm Hg) 2. ↑ Min ventilation required if insufflation with CO₂ 3. Consider antiemetic prophylaxis
9. Vulvectomy or other oncologic surgeries	Pelvic malignancy	Supine/lithotomy	GA with ETT ± epidural for postop pain relief	1. If hx of chemo, consider cardiac & pulm implications 2. Consider invasive monitoring 3. Potential for blood loss—T&S or crossmatch

Age-Based Guidelines for Interacting With Pediatric Patients

Neonates & infants <9 months	Do not typically fear strangers & separation is usually uncomplicated; preop sedation usually unnecessary & may prolong emergence
Toddlers	Aware of their environment but limited reasoning ability & reality testing Benefit most from preop sedation & parental participation at induction
School-age children	Often do not deal well with loss of control; may admit to fear of waking up during surgery; may be reluctant to ask questions Sedation options should be discussed with parents
Adolescents	Often focus on cosmetic side effects & altered body image after surgery Preop sedation may extend beyond drugs to include meditation or music

Suggested Preoperative Sedation Drugs and Dosing

Drug	Route	Dose (mg/kg)	Onset Time (min)
Midazolam	IV	0.01–0.03	<5
	Oral	0.5–0.75	15–30
	Nasal	0.1–0.2	15–30
	IM	0.05	5–10
	PR	1–3	10
Fentanyl	IV	0.001–0.005	<5
	Oral ("Actiq")	0.010–0.020	15–20
Ketamine	IV	1–2 mg/kg	1–2
	Oral	5 mg/kg	20–45
	IM	2–3 mg/kg	5–10
Methohexital	PR	20–30 mg/kg	5–10
Chloral hydrate	PO, PR	30–100 mg/kg	30–60

Nonnarcotic Analgesics for Children

Drug	Dose	Interval	Route
Ibuprofen	4–10 mg/kg	q6–8h	PO
Naproxen	5–7 mg/kg	q6–8h	PO
Tolmetin	5–7 mg/kg	q6–8h	PO
Choline magnesium salicylate	10–15 mg/kg	q8h	PO
Ketorolac	First dose: 1 mg/kg Repeat dose: 0.5 mg/kg	q6h	IV or IM
Acetaminophen	10–15 mg/kg	q4h	PO
	15–20 mg/kg	q4h	PR

Preoperative Evaluation of Pediatric Patients With a Systems Focus

History	Important Questions and Pertinent Findings
Prenatal care & delivery	Gestational age; Apgar score at birth; duration of intubation & ventilatory support; assoc congenital conditions (BPD, cyanotic heart dz); freq of hospitalizations; review of growth curves (failure to thrive); persistence of apnea/bradycardia

Preoperative Evaluation of Pediatric Patients With a Systems Focus (Continued)

History	Important Questions and Pertinent Findings
Airway	Dysmorphic features (e.g., Pierre-Robin = assoc with difficult airway); micrognathia, loose teeth; advanced caries
Respiratory	Symptoms of acute/recent URI; asthma; sick contacts; 2nd-hand smoke exposure; presence of wheezing, stridor, nasal flaring, cyanosis; sleep apnea
Cardiac	Murmurs assoc with PFO, PDA, congenital heart dz; freq/duration of cyanotic spells; tachypnea; poor feeding tolerance
Gastrointestinal	Repetitive vomiting; delayed meconium passage; abd distention
Hematologic	Bruising; pallor; family history of sickle cell/thalassemia
Neurologic	Patterns of seizure activity; developmental delay; motor weakness; hypotonia; evidence of ↑ ICP

Estimated Pediatric Vital Sign Parameters

Age	RR	HR	SBP	DBP
Preterm	55–60	120–180	45–60	20–45
Neonate	40–55	100–160	55–75	20–60
Infant (<6 months)	30–50	80–140	85–105	55–65
1 year	30–35	80–120	90–105	55–65
6 years	20–30	75–110	95–105	50–70
10 years	20–30	80–100	95–110	55–70
16 years	15–20	60–80	110–125	65–80

OR Equipment and Setup

- Oral ETT size = (age/4) + 4; depth = (ETT internal diameter × 3)

Suggested ETT Size Selection & Appropriate Insertion Depths

Age/Weight	Internal Diameter (mm)	Depth (oral, cm)	Depth (nasal, cm)
<1.5 kg	2.5	9.0–10.0	12.0–13.0
1.5–3.5 kg	3.0	9.5–11.0	13.0–14.0
Term	3.5	10.0–11.5	13.5–14.5
3–12 months	4.0	11.0–12.0	14.5–15.0
12–24 months	4.5	12.0–13.5	14.5–16.0

Suggested Doses of Common Emergency Drugs

Drug	IV	IM/(SQ)
Atropine	0.01–0.02 mg/kg	0.02 mg/kg
Succinylcholine	1–2 mg/kg	3–4 mg/kg
Ephedrine	0.1–0.2 mg/kg	—
Epinephrine	10 mcg/kg	(10 mcg/kg)

Recommended Laryngoscopic Blade & LMA Sizes

Age	Blade	Weight	LMA Size
Premature	Miller 0	<5 kg	1
Neonate	Miller 0	5–10 kg	1.5
1–4 years	Miller 1	10–20 kg	2
4–10 years	Miller 2, Mac 2	20–30 kg	2.5
Adolescent	Miller 2, Mac 3	>30 kg	3
Normal/large adults	Miller 2, Mac 3–4	60–90 kg	3–5
Large adults	Miller 2–3, Mac 3–4	>90 kg	5

Techniques**Induction****Comparison of Pediatric Induction Methods and Commonly Used Drugs**

Technique	Advantages	Disadvantages
Mask induction (sevoflurane)	Brief onset (2–3 min) Avoids awake IV Spontaneous respiration Parental participation Facilitates IV start via vasodilation	Breath-holding/laryngospasm Contraindicated for full stomach/MH Unprotected airway Gases are cold & dry Requires good seal
Intravenous (propofol)	Rapid onset (<30 sec) Minimizes duration of unprotected airway	Anxiety about "shots" Pain upon injection Malfunction Extravasation
Intramuscular (ketamine)	Brief (2–4 min) Can inject at multiple sites Does not require cooperation	Pain upon injection Difficulties with obese children Secretions with ketamine Unprotected airway Nerve injury
Rectal (methohexital)	Rapid onset (1–2 min) Quick clearance	Useful only in young children No prepackaged delivery device Unprotected airway

Prematurity—Perioperative Concerns

- ↑ Risk of hypothermia
- Unable to regulate glucose control
- ↑ Risk of postop apnea (esp. if <50 weeks postconceptual age)
- Retinopathy of prematurity (esp. if <44 week postconceptual age)
- Pulmonary dysfunction

Pediatric Maintenance Fluid Calculations

Weight	Rate
<10 kg	4 mL/kg/hr
10–20 kg	40 mL/hr + 2 mL/kg/hr for each kg > 10 kg
>20 kg	60 mL/hr + 1 mL/kg/hr for each kg > 20 kg

Estimated HCT and EBV

Age	HCT (%)	EBV (mL/kg)
Premature	45–60	90–100
Neonate	45–60	80–90
3–6 months	30–33	70–80
6 months–1 year	32–35	70–80
1–12 years	35–40	70–75
Adult	38–45	60–70

Epiglottitis and Croup

	Epiglottitis	Croup
Etiology	H. flu (bacterial)	Viral
Age	1–8 yr	6 mo–6 yr
Timing	Fast onset	Gradual onset
X-ray findings	Swollen epiglottis	"Steeple" sign (edematous narrowing of subglottic inlet)
Signs & symptoms	High fever, stridor, drooling	Mild fever, "barking" cough, cyanotic
Anesthetic management	Surgery on standby for emergent surgical airway, usually inhalational induction (sitting position) while pt breathing spontaneously; no awake laryngoscopy (risk of laryngospasm)	Cool mist, racemic epi (nebulized), steroids If needed, perform intubation in OR, consider mask induction
	Use smaller-than-normal ETT (due to edema)	

Foreign Body Aspiration

- Airway manipulation (even minor) → can convert partial into complete obstruction
- Supraglottic foreign body: Careful inhalational induction & gentle upper airway endoscopy to remove foreign body
- Subglottic foreign body: RSI or inhalational induction → followed by rigid bronchoscopy or ETT + flex bronch
- Good communication between surgeon & anesthesiologist essential

Tetralogy of Fallot

Definition	Pulmonary stenosis, VSD, overriding aorta, RVH
TET spell	↑ Ventricular outflow tract obstruction & PVR → inc R-to-L shunt
Management	through VSD → cyanosis; Hyperventilation to ↓ PVR, phenylephrine to ↑ SVR (> than PVR) → causes L-R shunt; also can give fluids, propranolol
Anesthetic Management	Goals: ↓ R-to-L shunt (avoid ↑ PVR, ↓ SVR, ↑ heart contractility) Techniques: Hydration, continuation of β-blockade, avoidance of crying Induction: Ketamine (maintain SVR) vs inhalational induction (risk of ↑ PVR due to hypoxia & hypercarbia)
SVR/PVR Management	↓ SVR: Potent volatile agent, histamine-releasing drugs, α-blockers ↑ PVR: Acidosis, hypercarbia, hypoxia, PPV/PEEP, N ₂ O

Comparison of Gastroschisis and Omphalocele

	Gastroschisis	Omphalocele
Etiology	Omphalomesenteric artery occlusion	Failed gut migration from yolk sac to abdomen
Incidence	1:15,000	1:6000
Presentation	Lateral to umbilicus	Midline
Hernial sac	Absent	Present
Bowel function	Abnormal	Normal
Associated anomalies	Prematurity	Beckwith-Wiedemann syndrome; congenital heart disease; bladder extrophy

Syndromes and Their Anesthetic Implications		
Name	Description	Anesthetic Implications
Adrenogenital syndrome	Inability to synthesize hydrocortisone. Virilization of female.	All need hydrocortisone even if not salt-losing. Check electrolytes.
Alpert's syndrome	Craniofacial abnormalities, syndactyly and potential developmental delay.	Possible hydrocephalus and elevated intracranial pressures. Anticipate potential difficult airway.
Ataxia-telangiectasia	Cerebellar ataxia. Skin and conjunctival telangiectasia. Decreased serum IgA and reticuloendothelial malignancy.	Defective immunity; recurrent chest and sinus infections. Bronchiectasis.
Beckwith syndrome (infantile gigantism)	Birth weight >4000 g. Macroglossia and omphalocele.	Persistent severe neonatal hypoglycemia. Airway problems.
Cherubism	Tumorous lesion of mandibles and maxillae with intraoral masses. May cause respiratory distress.	Intubation may be extremely difficult. May require tracheostomy.
Cretinism (congenital hypothyroidism)	Absent thyroid tissue or defective synthesis thyroxine and goiter.	Airway problems; large tongue, goiter. Respiratory center very sensitive to depression. CO ₂ retention common. Hypoglycemia, hyponatremia, hypotension. Low cardiac output. Transfusion poorly tolerated.
Cri-du-chat syndrome	Chromosome 5-P abnormal. Abnormal cry, microcephaly. Micrognathia. Congenital heart disease.	Airway problems; stridor, laryngomalacia. Possibly difficult intubation.

Syndromes and Their Anesthetic Implications (Continued)		
Name	Description	Anesthetic Implications
Cayser's syndrome (micrognathia)	Micrognathia. Small nasopharynx. Hypochoch. 60% have congenital heart disease. Duodenal atresia in some. Cervical spine abnormalities.	Difficult airway, large tongue, small mouth. Risk of laryngeal edema usually on extubation. Problems of cardiac anomalies.
Duchenne muscular dystrophy	Muscular dystrophy with frequent cardiac muscle involvement. Usually die in 2nd decade. Amount of skeletal muscle involvement and cardiac involvement unrelated.	As for hypochoch congenital plus cardiac involvement. Minimal drug dosage. Avoid respiratory depressants, muscle relaxants. Facilitate ventilatory support may be required. Possible difficult intubation. Care with generally expensive drugs.
Edward's syndrome (trisomy 18)	Congenital heart disease in 90%. Micrognathia in 80%. Renal malformations 50-80%. Usually die in infancy.	Possible difficult intubation. Care with generally expensive drugs.
Ehlers-Danlos syndrome	Collagen abnormality with hyperextensibility and fragile tissues. Dissecting aneurysm of aorta. Fragility of other blood vessels. Bleeding diathesis.	CVC—spontaneous rupture of vessels. Angiogram 1% mortality. ECG conduction abnormalities. IV difficult to maintain. Hematoma. Poor tissue and clotting defects lead to hemorrhage especially GI tract. Spontaneous pneumothorax.
Fanconi's syndrome	Muscle disease. Hypokalemia attacks of quadriplegia.	Monitor serum K ⁺ . Limit use of diuretics. Monitor ECG. Avoid relaxants.
Fanconi syndrome (renal tubular acidosis)	Usually 2 to other disease. Proximal tubular defect. Acidosis, K ⁺ loss. Dehydration.	Impaired renal function. Treat with citrate and acid-base abnormalities. Look for 1 disease (galactosemia, cystinosis, etc.).
Hemochromatosis	Inborn error of metabolism. Thromboembolic phenomenon due to normal thickening blood cells. Osteoarthritis. Kypheoscoliosis.	Decrease 50 to reduce viscosity and platelet adhesiveness, increase peripheral perfusion. Angiography may detect thromboses, especially coronary.
Kernicterus syndrome	Overexposure to bilirubin and bronchiectasis. Abnormal immunity.	Chronic respiratory infections.
Klippel-Feil syndrome	Congenital fusion of two or more cervical vertebrae, leading to neck rigidity.	Difficult airway and intubation.
Median cleft face syndrome	Varying degrees of cleft lip. Frontal lipomas. Dermoids.	Cleft, nose, lip, and palate may cause intubation difficulties.
Marfan's syndrome (marfanocyst)	Connective tissue disorder. Dilated aortic root leads to Aortic dissection or abdominal aneurysm. Pulmonary artery, aorta valve involved. Kyphoscoliosis, pectus excavatum lung cysts, joint instability and dislocation.	Care with myocardial depressant drugs. Beware possible dissection of aorta long function seen. Possible perimembrane. Care in positioning easily dislocated joints.

Syndromes and Their Anesthetic Implications (Continued)		
Name	Description	Anesthetic Implications
Myasthenia congenita	Like adult myasthenia gravis.	Avoid respiratory depressants, muscle relaxants. May require postop IPPV. Problems with anticholinesterase therapy pre- and postop if necessary. Halothane may cause postop shivering and myotonia. Pulmonary complications due to poor cough.
McArdle disease	Glycogen storage disease V.	Muscles affected including cardiac muscle: Care with cardiac depressant drugs.
Pierre Robin syndrome	Cleft palate, micrognathia, glossoprosis. Associated congenital heart disease may occur	Anticipate difficult airway. Micrognathia & glossoprosis may lead to resp distress, requiring trach or tongue suture to relieve postoperative laryngeal obstruction.
Porphyria	Intermittent porphyria = most common autosomal dominant form. Usual latent before puberty. Abdominal pain, neurological dysfunction, electrolyte imbalances & psychiatric disturbances characterize acute episodes	Avoid barbiturates (thiopental, methohexital). Ketamine, etomidate & propofol appear safe. Maintenance of anesthesia with narcotics. Volatile agents & non-depolarizing muscle relaxants is recommended. Avoid regional anesthesia in presence of existing neurologic deficit.
Prader-Willi syndrome	Neonate: hypotonia, poor feeding, absent reflexes. Second phase—hyperactive, uncontrollable polyphagia, mental retardation	Obesity of extreme proportions leading to cardiopulmonary failure.
Scleroderma	Diffuse cutaneous swelling. Plastic surgery required for contractures and constrictions.	Scarring face and mouth; difficult airway and intubation. Chest resuscitation: poor compliance. Diffuse pulmonary fibrosis, hypoxia. Veins often irritable and palpable. Cardiac fibrosis or cor pulmonale. History of steroid therapy.
Stevens-Johnson syndrome	Erythema multiforme, urticarial lesions and erosions of mouth, eyes, and genitalia. Possibly hypersensitivity to exogenous agents (e.g., drugs).	Oral lesions: avoid intubation and esophageal stethoscope. Monitoring difficult because of skin lesions but essential. ECG—fibrillation, myocarditis, pericarditis occur. Temperature control—febrile episodes. Intravenous access—essential but avoid cut-down because of infection. Ketamine probably best anesthetic. Pleural blebs and pneumothorax may occur.
Tay Sachs disease	Gangliosidosis. Blindness and progressive dementia and degeneration of central nervous system.	No described anesthetic hazard. Progressive neurologic loss leads to respiratory complications. Supportive measures only treatment.

Syndromes and Their Anesthetic Implications (Continued)		
Name	Description	Anesthetic Implications
Treacher Collins syndrome (mandibulofacial dysostosis)	Micrognathia and aplastic zygomatic arches. Microstomia, choanal atresia. Congenital heart disease may occur.	Possible airway and intubation difficulties. Less severe than Pierre Robin deformity.
von-Hippel-Lindau syndrome	Retinal or CNS hemangioblastoma. (Posterior fossa or spinal cord.) Associated with pheochromocytoma & renal, pancreatic, or hepatic cysts.	Problems due to associated pheochromocytoma, renal and hepatic pathology.
Von Recklinghausen disease (neurofibromatosis)	Cafe-au-lait spots. Tumors in all parts of CNS. Peripheral tumors associated with nerve trunks. Increased incidence pheochromocytoma. Honeycomb cystic lung changes. Renal artery dysplasia and hypertension. Decreased ceruloplasmin causes abnormal copper deposits, especially in liver and CNS motor nuclei. Renal tubular acidosis.	Screen for pheochromocytoma (urinary VMA). Should be investigated for lung function. Tumors may occur in the larynx and right ventricular outflow tract. Care with renally excreted drugs if kidneys involved.
Wilson's disease (hepatolenticular degeneration)		Hepatic failure 2 ^o to fibrosis. IV induction (propofol, ketamine, etomidate) acceptable. Apnea uncommon with succinylcholine administration, despite pseudocholinesterase deficiency. Consider reduced dosing of renally-excreted drugs.
Wolff-Parkinson-White syndrome	ECG abnormality—short PR, prolonged QRS with phasic variation in 40%. Associated with many cardiac defects. Anomalous conduction path between atria and ventricles. Delta wave may be present on ECG.	Scopolamine preferred to atropine as drying agent. Tachycardia due to atropine or apprehension may change ECG and suggest infarction, with ST-segment depression. Paroxysmal SVT on induction of anesthesia or during cardiac surgery has been reported. Should be treated with digitalis, propranolol, pacemaker if necessary. Neostigmine may accentuate W-P-V pattern.

Risk Factors for PONV

Patient Factors	Anesthetic Factors	Surgical Factors
Female	Volatile anesthetics	Surgical Procedure
Nonsmokers	Nitrous oxide	(ophthalmologic, gynecologic, breast, abdominal, laparoscopy)
History of PONV or motion sickness	(controversial) Opioids	Surgical duration: Each additional 30 min raises PONV risk by 60%

Simplified Risk Score for PONV

Risk Factors	Points
Female	1
History of PONV/ motion sickness	1
Nonsmoker	1
Perioperative opioids	1

Prediction and Prophylaxis of PONV

No. of Points	% Risk of PONV	Prophylaxis
0	10% (low risk)	
1	21% (low risk)	No prophylaxis
2	39% (mod risk)	Monotherapy
3	61% (high risk)	Multimodal
4	79% (high risk)	therapy/consider TIVA

Antiemetic Drugs

Class	Drug	IV Dose	Timing	Side Effects
5HT ₃ Antagonists	Ondansetron	4 mg	End of surgery	Headaches
	Granisetron	0.35–1.5 mg		
	Dolasetron	12.5 mg		
	Tropisetron	2 mg		
Steroid	Dexamethasone	4–6 mg	At induction	
Butyrophenones	Droperidol	0.625–1.25 mg	End of surgery	QT prolongation, drowsiness, extrapyramidal effects
	Halopridol	0.5–2 mg		
Phenothiazines	Promethazine	6.25–25 mg	End of surgery	Drowsiness, agitation, extrapyramidal effects
	Prochlorperazine	5–10 mg		

(continued)

Antiemetic Drugs (Continued)				
Class	Drug	IV Dose	Timing	Side Effects
Antihistamine	Dimenhydrinate	1 mg/kg		Sedation
Other	Propofol	20 mg	In PACU	Resp depression
	Scopolamine	Trans patch	4 hr before end of surgery	Sedation, confusion dry mouth
	Ephedrine	0.5 mg/kg IM	End of surgery	
	Metoclopramide	10–20 mg	1 hr before end of surgery	Extrapyramidal effects

Modified Postanesthetic Discharge Scoring System (Score ≥ 9 for discharge)	
VITAL SIGNS	
Within 20% of preop value	2
20–40% of preop value	1
40% of preop value	0
AMBULATION	
Steady gait/no dizziness	2
With assistance	1
None/dizziness	0
NAUSEA/VOMITING	
Minimal	2
Moderate	1
Severe	0
PAIN	
Minimal	2
Moderate	1
Severe	0
SURGICAL BLEEDING	
Minimal	2
Moderate	1
Severe	0

Multimodal Pain Management Strategy

Step 3: Severe Postop Pain

Step 1 and 2 strategies
AND
 Local anesthetic peripheral neural blockade
 (with or without catheter)
AND
 Use of sustained-release opioid analgesics

Step 2: Moderate Postop Pain

Step 1 strategy
AND
 Intermittent doses of opioid analgesics

Step 1: Mild Postop Pain

Nonopioid analgesic
 Acetaminophen, NSAIDs, COX-2 selective inhibitors
AND
 Local anesthetic infiltration

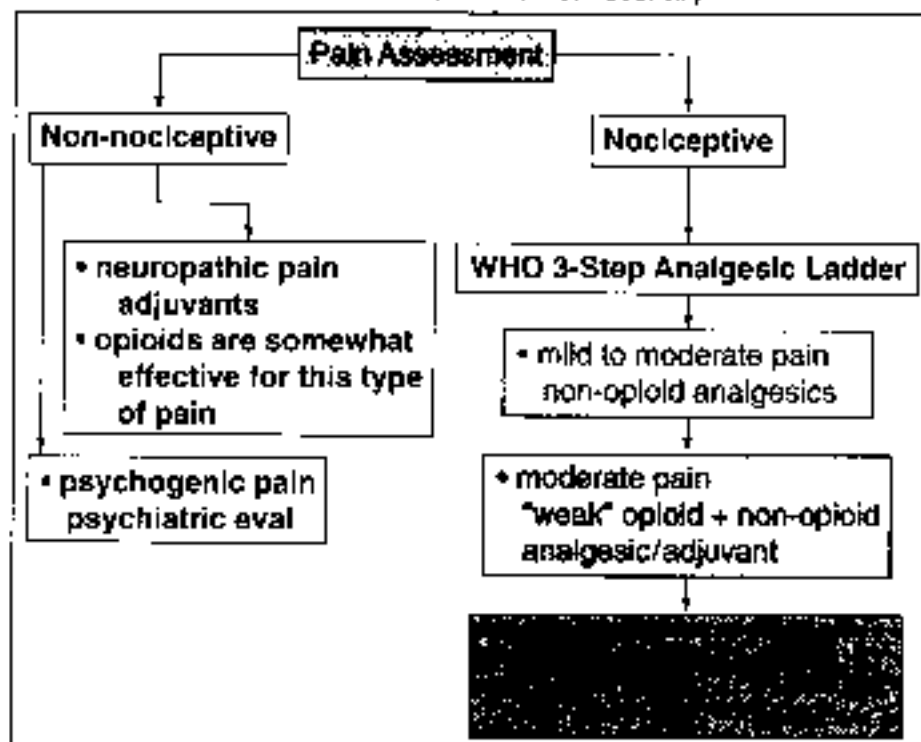
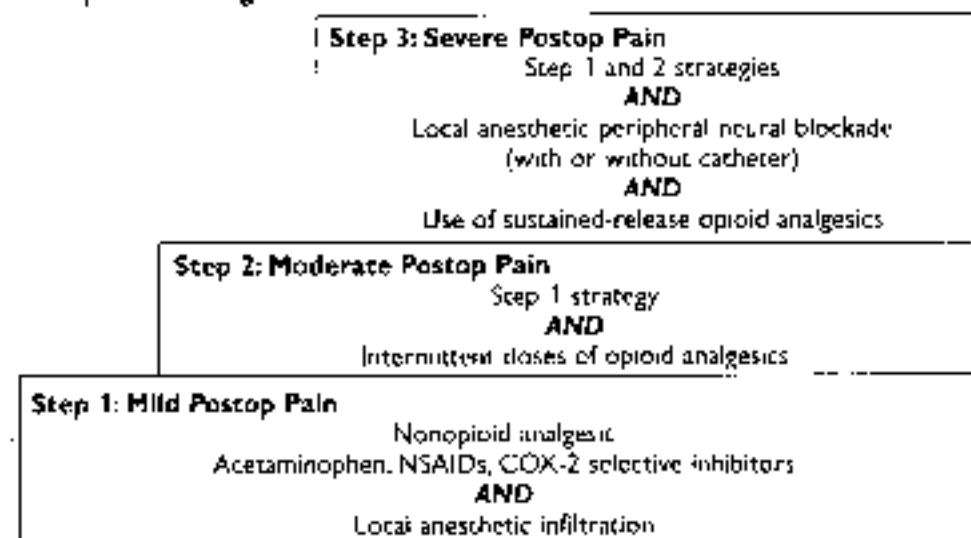
Summary of Options for Postop Pain Management		
Route	Therapy Class	Medication/Dose
INTRAVENOUS/ INTRAMUSCULAR	OPIOIDS	Fentanyl 25–100 mcg/kg q30–60 min Hydromorphone 0.2–2 mg q4–6h Meperidine 25–50 mg q3–4h Morphine 1–10 mg q2–6h
	NSAIDs MIXED AGONISTS/ ANTAGONISTS	Ketorolac 30 mg Butorphanol 20 mcg/kg Nalbuphine 0.25 mcg/kg
ORAL	NSAIDs	Ibuprofen 400–800 mg q4–6h Ketorolac 10–20 mg q4–6h Naproxen 500 mg q6–8h
	COX-2 INHIBITORS	Celecoxib 200–400 mg q12h Acetaminophen/propoxyphene napsylate (Darvocet) q4–6h Acetaminophen/oxycodone (Percocet) q4–6h
	OPIOID/ NONOPIOID COMBINATIONS	Acetaminophen/codeine (Tylenol with codeine) q4–6h Acetaminophen/hydrocodone (Vicodin) q4–6h
TRANSDERMAL	OPIOIDS	Hydrocodone 5–10 mg q4–6h Morphine 10–30 mg q3–4h Oxycodone 5 mg q3–6h
	OTHERS	Acetaminophen 325–1000 mg q4–6h
INTRANAŒAL	OPIOIDS	Fentanyl patch 25–100 mcg/h q72h Fentanyl Meperidine Butorphanol
	OPIOIDS	
LOCAL ANESTHETICS		Neuraxial anesthesia Regional nerve block Local infiltration by surgeon Continuous subcutaneous catheter
NON PHARMALOGICAL METHODS		Heat/cold therapy, massage, TENS relaxation, hypnosis, acupuncture, biofeedback

ASA Guidelines for Non-OR Anesthetizing Locations

1. Reliable O_2 source, including backup supply
2. Adequate & reliable suction
3. Adequate & reliable scavenging system if anesthetic gases are to be used
4. Self-inflating resuscitator bag capable of delivering 0.9 FiO_2 ; adequate drugs, supplies, & equipment for the planned activity
5. Adequate monitoring equipment to adhere to ASA Monitoring Standards
6. Sufficient electrical outlets
7. Sufficient site for equipment
8. Immediate availability of emergency cart with defibrillator & emergency drugs
9. Reliable 2-way communication
10. Observation of all applicable building & safety codes and facility standards

Chronic pain - persistence of pain >3 months, without any biological value

Figure 29-1 Algorithm for pharmacologic chronic pain management. (Adapted from The Texas Cancer Pain Initiative, Texas Cancer Council.)

**3-Step WHO Analgesic Ladder**

Cervical and Lumbar Disk Herniation Patterns					
Disc	Root	Pain:	Sensory Loss	Motor loss	Reflex loss
C4-C5	C5	Neck, shoulder, upper arm	Shoulder	Deconv. biceps, infraspinatus	Suprap
C5-C6	C6	Neck, shoulder, lat. arm radial forearm, thumb & index finger	Lat arm, ulnar forearm, thumb & index finger	Biceps, brachioradialis	Biceps, brachioradialis, supinator
C6-C7	C7	Neck, lat. arm, ring & index fingers	Radial forearm, index & middle fingers	Triceps, extensor carpi ulnaris	Triceps, supinator
C7-T1	C8	Ulnar forearm and hand	Ulnar half of ring finger, little finger	Intrinsic hand muscles, wrist extensors, flexor dig profundus	Finger flexion
L3-L4	L4	Anterior thigh, inner shin	Anteromedial thigh and shin, inner foot	Quadriceps	Patella
L4-L5	L5	Lat thigh and calf, dorsum of foot, great toe	Lat calf and great toe	Extensor hallucis longus, fibular dorsiflexion, eversion & inversion	None
L5-S1	S1	Back of thigh, lateral posterior calf, lat. foot	Posterior calf, lat. and sole of foot, smaller toes	Gastrocnemius, foot eversion	Achilles

Definition of Pain Terms

Allodynia	Painful response to nonpainful stimulus
Dysesthesia	Unpleasant abnormal sensation (spontaneous or evoked)
Hyperalgesia	↑ Sensitivity to stimulation
Hyperpathia	Painful syndrome with abnormally painful response to stimulus
Hypoalgesia	↓ Pain in response to normally painful stimulus
Hypoesthesia	↓ Sensitivity to stimulation
Paresthesia	Abnormal sensation (spontaneous or evoked)

Headache

Primary Headache Syndromes

- Tension: Associated with muscle contraction in neck or lower head
- Migraine: See below
- Cluster: Periodic, paroxysmal, brief, sharp, orbital headache that may awaken from sleep ± lacrimation, rhinorrhea, conjunctival injection, or unilateral Horner's synd.

Secondary Causes of Headaches

- Vascular: Stroke, intracerebral hemorrhage, SAH, subdural hematoma, AVM, unruptured aneurysm, arterial hypertension, venous thrombosis
- Infection: Meningitis, encephalitis, abscess
- Brain tumor
- CSF disorder: ↑ (hydrocephalus) or ↓ (sp LP)
- Trigeminal neuralgia
- Extracranial: Sinusitis, TMJ syndrome, temporal arteritis

Clinical Evaluation (JAMA 2006;296:1274)

- History: Quality, severity, location, duration, time of onset, precipitants, relieving factors
- Associated symptoms (visual Δs, nausea, vomiting, photophobia)
- Focal neurologic symptoms
- Head or neck trauma, constitutional symptoms
- Medications, substance abuse
- General and neurologic examination
 - Warning signs that should prompt neuroimaging:
 - Worst ever, worsening over days, wakes from sleep
 - Vomiting, aggravated by exertion or Valsalva
 - Fever, abnl neurologic exam, aura, cluster-type headache

Headache (Continued)**Migraine****Epidemiology**

- Affects 15% of women and 6% of men; onset usually by 30 y/o.

Clinical Manifestations (Lancet 2004;363:381; JAMA 2006;296:1274)

- Unilateral or bilateral, retro-orbital, throbbing or pulsatile headache; lasts 4–72 h
- Often accompanied by nausea, vomiting, photophobia
- "POUNDing": Pulsatile; duration 4–72 h; Unilateral; Nausea & vomiting; Disabling
- Likelihood ratio (LR) 3.5 if 3 criteria are met, LR 24 if ≥4 criteria are met
- Classic (18%) – visual aura (scotomata with jagged or colored edge) precede headache
- Common (64%) – headache without aura
- Complicated – accompanied by stereotypical neurologic deficit that may last hrs
- Precipitants: Stress, hunger, foods (cheese, chocolate) and food additives (MSG), fatigue, alcohol, menstruation, exercise

Treatment (NEJM 2002;346:257)

- Eliminate precipitants
- Prophylaxis: TCA, β -blockers, CCB, valproic acid, topiramate (JAMA 2004;291:965)
- Abortive therapy:
 - ASA, acetaminophen, caffeine, high-dose NSAIDs
 - Metoclopramide IV, prochlorperazine IM or IV
 - 5-HT₁ agonists ("triptans"); contraindic. in pts w/ complicated migraine, CAD, prior stroke
 - Combination of triptan + NSAID more efficacious than either single agent alone (JAMA 2007;297:1443)
 - Ergotamine, dihydroergotamine; use with caution in pts with CAD

Stellate Ganglion Block—Technique

- Point of entry: Between trachea & carotid sheath at level of cricoid cartilage & Classignac's tubercle (C6) (stellate ganglion lies at C7, but enter at C6 to prevent damage to pleura)
- After contacting C6, withdraw needle 1–2 mm (gets needle out of longus colli muscle)
- Ensure neg. aspiration, then give 1st test dose (0.5 mL of 0.25% bupr or 2% lido)
- 2nd test dose should be 3 mL of local with 1:200,000 (to rule out IV placement)
- Inject 12–15 mL 0.25% bupr in 3-mL aliquots with intermittent aspiration
- Place pt into sitting positn (facilitates spread onto stellate ganglion) for 10 min.
- Pt should be observed for 1 hr after the block

Trigger-Point Injection—Local Anesthetic or Steroids

- Raise small skin wheal with 25-ga needle & local anesthetic
- Inject 1–3 mL local (0.25–0.5% bupr or 2% lido) with 22 ga needle into trigger point (after negative aspiration)
- For corticosteroid injection, use larger needle (18–21 ga)
 - Triamcinolone 40 mg/mL
 - Methylprednisolone 40 mg/mL
 - Dexamethasone 4 mg/mL
- If 3 or more trigger point injections at same site fail (spaced 2 wk apart) Consider injection of botulinum toxin A (Botox)

Stellate Ganglion Block—Complications

- | | |
|--|--|
| • Infection | • Epidural/intrathecal block |
| • Vascular injury (carotid artery/internal jugular vein) | • Brachial plexus anesthesia |
| • Neural injury (vagus, brachial plexus) | • Recurrent laryngeal n. block (with resultant hoarseness) |
| • Pneumothorax | • Phrenic n. paralysis (with elevated hemidiaphragm) |
| • Hemothorax | • Chylothorax |
| • Intravascular injection (vertebral artery) | |

Technique of Celiac Plexus Block

- Performed at L1; pt usually in prone position
- 22-ga Quincke spinal or Chiba needle placed 5–7 cm lateral to midline → insert to depth of 7–9 cm
- Inject contrast dye to ensure correct placement
- Inject 15–20 mL of 0.5% bupiv, or 10–15 mL of 2% lido, or 10–15 mL of 0.5% ropiv
- Inject neurolytic agent (10–15 mL of 50% alcohol or 7–12% phenol)

Cryotherapy

- Definition: Extreme cold to freeze & destroy nerves & tissue
- Mechanism: Cell injury by necrosis (resulting from freezing & thawing)
- Technique: Liquid nitrogen most commonly used (argon & N₂O possible)
(desired temp usu -50 to -60°C); usually one or two 5- to 30-s freeze-thaw cycles performed q4–6wk

Radiofrequency (RF) neurolysis

- Destroys nerve conduction for 3–18 mo
- Indications: Denervate facet joints, splanchnic nerve (for abd pain), cranial nerve ganglion (trigeminal ganglion)
- Only pulsed RF is recommended for peripheral nerves
 - Usually done at 42–45°C for 120–180 s
 - Forms a lesion if neural temp exceeds 45°C

Facet Blocks

- Pain relief for neck & back pain (pain elicited by neck/back extension)
- Blocks medial branch of posterior ramus of spinal nerve
 - For severe & prolonged pain, consider denervation of facet joints by RF
- High temp 80°C applied for 90 s at each level

Contraindications to Solid Organ Transplantation

- | Absolute Contraindications | Relative Contraindications |
|---|-----------------------------|
| • Active uncontrolled infection | • Noncompliance |
| • Severe cardiopulmonary/medical condition (pt unfit for surgery) | • History of drug abuse |
| • Inability to tolerate immunosuppression (AIDS) | • Advanced age |
| • Continued drug/alcohol abuse | • Psychological instability |
| • Brain death | • HIV infection |
| • Extrahepatic malignancy | |
| • Inability to comply with medical regimen | |
| • Lack of psychosocial support | |

Extrahepatic Manifestations of Liver Disease

- | | |
|----------|---|
| • Pulm: | Portopulmonary HTN, hepatopulmonary syndrome, pleural effusions |
| • CV: | Hyperdynamic circulation (* cardiac output & ↓ SVR) |
| • GI: | Portal HTN, esophageal varices, ascites |
| • CNS: | Encephalopathy, ↑ ICP (with fulminant hepatic failure) |
| • Heme: | Thrombocytopenia (↓ thrombopoietin & hypersplenism), ↓ clotting factors (↓ decreased synthetic fx, DIC, fibrinolysis) |
| • Renal: | Oliguria, renal insufficiency, hepatorenal syndrome |

Phases of a Liver Transplant Operation**The Preanhepatic (Dissection) Phase**

- Primary purpose: Dissection of porta hepatis & mobilization of native liver
- Hypertension: Surgical bleeding, ascites/effusion drainage, clamping/pressure on abd veins
- Bleeding risks: Portal HTN, coagulopathy & prior abdominal surgery
- Metabolic alterations: $\uparrow K^+$, metabolic acidosis, $\downarrow Ca^{2+}$ (from citrate toxicity)
- Coagulopathy: Underlying factor deficiencies, thrombocytopenia & dilutional coagulopathies
- Hypothermia (must be corrected, or will worsen coagulopathy)
- Ensure adequate urine output & euvolemia

The Anhepatic Phase

- Begin - clamping hepatic artery & portal vein; end - reperfusion of donor liver
- Vessels perfusing liver are clamped & old liver removed
- Warm ischemic time: Begins when donor liver removed from ice, end when reper-fused, should limit warm ischemia time to between 30 & 60 min
- Portal vein, inferior vena cava (IVC) & hepatic artery usu. clamped during this phase
- Venovenous bypass occasionally required for pts who do not tolerate cross clamping
 - Blood diverted from portal vein & IVC into SVC, usu. via axillary vein
 - Advantages: Avoids renal/splanchnic engorgement, maintains preload, $\uparrow$ renal perfu-sion, may $\downarrow$ blood loss, $\downarrow$ transfusion requirements
 - Disadvantages: 1 Risk of air embolus/DVT, seroma, nerve injury, wound lymphocele

Phases of a Liver Transplant Operation (Continued)

- Piggyback technique may also obviate need for IVC clamping
 - Native hepatic veins fashioned into a cuff (serves as a receptacle for suprahepatic IVC of the donor liver); cava to cuff anastomosis performed & native IVC need not be clamped
- Preparation for reperfusion: $\uparrow K^+$ & acidosis prevalent in absence of functioning liver
- Aggressively treat with furosemide, albuterol, $\uparrow$ ventilation, insulin(0.50 and/or bicarb

The Neohepatic Phase

- Begins with allograft reperfusion; end with completion of biliary anastomosis
- Allograft flushed of air, debris & residual preservative solution
- Undamping of donor liver $\rightarrow$ embolic debris, $\uparrow K^+$, metabolic acidosis, $\downarrow Ca^{2+}$, hypother-mia, hypotension, hypovolemia, release of cytokines & other destabilizing agents
- **Postreperfusion syndrome (PRS)** — $\downarrow$ MAP of 30% from baseline, lasts ≤ 1 min, within 5 min of reperfusion
 - Can see arrhythmias, $\downarrow$ SVR, $\downarrow$ cardiac output, vasodilation, $\uparrow$ L ventricular filling pres-sure, R ventricular dysfx
 - Contributing factors: $\uparrow K^+$, $\downarrow Ca^{2+}$, acidosis, & blood loss
 - Air embolus & thromboembolus may be seen via echo or inferred via PA-line
 - Treatment: Minimize $\uparrow K^+$, $\downarrow Ca^{2+}$, acidosis; usually resolves within 30 min
- **Postreperfusion coagulopathy** — may also follow graft reperfusion
 - Due to (1) release of heparin or (2) tissue plasminogen activator (tPA) $\rightarrow$ $\uparrow$ fibrinolysis
 - Heparin effect reversible with heparinase on thromboelastography (TEG)
 - Cryoprecipitate, FFP & antifibrinolytics (epsilon aminocaproic acid, tranexamic acid) may treat $\uparrow$ fibrinolysis
 - Protamine will treat heparin effect
 - Refractory coagulopathy may indicate graft failure
- Indicators of good graft function: Resolution of coagulopathy & metabolic acidosis, return of normoglycemia & bile production renal dose dopamine may offer some renal protection

Possible Contraindications to Heart Transplantation

Severe $\uparrow$ pulm vascular resistance (>6 Wood units) (orthotopic procedure)	Age > 55 years
Continued illicit drug/tobacco use, noncompliance	Uncontrolled malignancy
Significant irreversible renal, hepatic, or pulm dysfunction	Obesity
Coexisting systemic illness with a poor prognosis	Previous malignancy
Diabetes mellitus with end-organ damage	Osteoporosis
Active infective process (hepatitis B & C)	Active peptic ulcer disease
Amyloidosis (cardiac dz may recur)	

ECG Lead Placement & Utility

- 12 leads
 - Limb leads: I, II, III, aVR, aVL, aVF
 - II, III, aVF—inferior
 - I, aVL—lateral
 - Preordial chest leads: V1, V2, V3, V4, V5, V6
 - V1, V2—septal
 - V3, V4—anterior
 - V5, V6—lateral
- Coronary arteries
 - LAD: V1, V6, anterior, septal, & inferior
 - Circumflex: I, aVL, V5, V6, lateral & posterior (inferolateral)
 - RCA: II, III, aVF, inferior & posterior (inferolateral)
- Intraoperative monitoring
 - II – best for detecting arrhythmias
 - V – best for detecting ischemia

Steps for ECG Interpretation

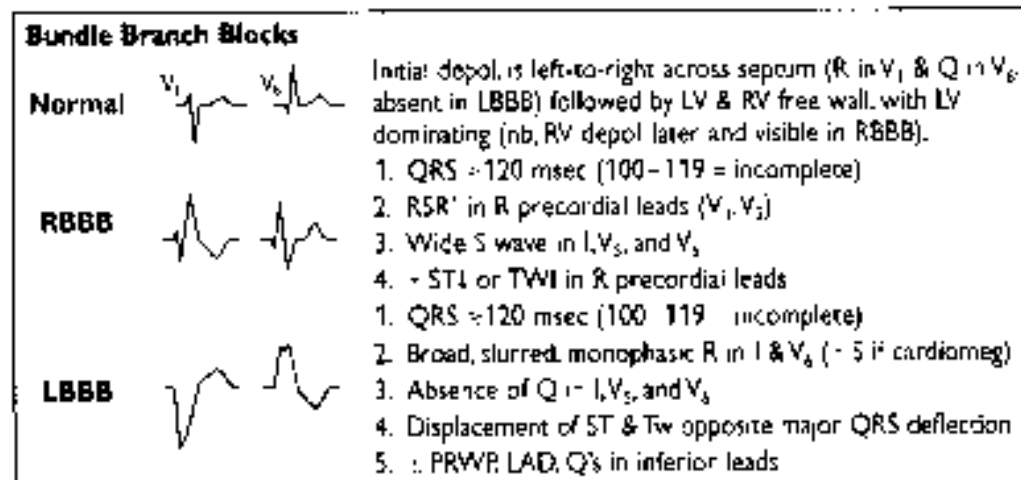
- Rhythm
 - Check for P before every QRS
 - Check PR intervals to assess for AV block
 - Check QRS intervals to assess for bundle branch blocks
- Rate
 - Count large boxes between R waves on ECG (each large box = 0.2 s)
 - 1 large box = 300 bpm
 - 2 boxes = 150 bpm
 - 3 boxes = 100 bpm
 - 4 boxes = 75 bpm
 - 5 boxes = 60 bpm
 - 6 boxes = 50 bpm
- Examine axis: ST segments: T, Q, and U waves; QRS width & progression, hypertrophy (see below)

Axis – direction of vector which represents heart's overall wave of depolarization

Interpretation of Axis in Vertical Plane

Lead I	Lead aVF	Axis
Positive	Positive	Normal
Positive	Negative	Left axis deviation
Negative	Positive	Right axis deviation
Negative	Negative	Extreme right axis deviation

- Atrioventricular Conduction System
 - **1st-degree AV block** PR interval increased >0.2 s
 - **2nd-degree AV block**
 - **Mobitz type I (Wenkebach)** AV delay (PR interval) increases with each beat, until QRS is dropped after P wave
 - Treatment—only if symptomatic: Atropine, isoproterenol, permanent pace
 - **Mobitz type II**—sudden unpredictable dropped QRS not associated with progressive PR interval prolongation
 - Caution: May progress to 3rd degree heart block
 - Treatment—permanent pacemaker
 - **3rd degree AV block** (complete heart block)
 - No relationship between P wave & QRS—"AV dissociation"
 - Treatment—permanent pacemaker
 - **Bundle-branch block**



Bifascicular block: RBBB + LAHB/LPHB, Trifascicular: 1° AVB + RBBB + LAHB/LPHB

- **Right bundle-branch block (RBBB)**
 - Examine QRS in V₁ & V₂
 - Right ventricular depolarization delayed
 - LBBB makes it difficult to determine infarction on ECG
- **Left bundle-branch block (LBBB)**
 - Examine QRS in V₅ or V₆
 - Left ventricular depolarization delayed
 - Difficult to determine infarction on ECG
- **Atrial flutter**
 - Regular atrial activity; 180–350 bpm; ventricular rate 150 bpm (2:1 AV block)
 - ECG "F waves," "sawtooth" pattern, flutter waves
 - Treatment
 - Unstable → immediate electrical cardioversion
 - Burst pacing (temporary or permanent pacemaker)
 - Medical therapy (β-blockers, Ca²⁺-channel blockers)
 - Radiofrequency catheter ablation (RFA)
- **Atrial fibrillation**
 - Irregular atrial activity at 350–600 bpm, ventricular rate 160
 - ECG: Wavy baseline, absent P waves
 - Treatment
 - Unstable — immediate electrical cardioversion
 - Chemical cardioversion (Class IA, IV, III antiarrhythmics)
 - Antiarrhythmic drugs
 - Anticoagulation
 - Rate control: β- or Ca²⁺-channel blockers, digoxin
 - Maze procedure
- **Paroxysmal SVT**
 - Ventricular rate 140–250 bpm
 - ECG: Narrow complex, P waves hidden in QRS complexes (QRS may be slightly widened, not more than 0.14 s)
 - Treatment: Vagal maneuvers, β- or Ca²⁺-channel blockers, radiofrequency ablation
- **AV Reentrant Tachycardia**
- **Wolff Parkinson-White**
 - PR interval shortened, delta wave, wide QRS
 - Treatment: β- or Ca²⁺-channel blockers, radiofrequency ablation
- **Ventricular Arrhythmias**
 - **Premature ventricular beats**
 - Widened QRS
 - **Couplet**—two in a row; **Bigeminy**—every other beat is PVC
 - **Ventricular tachycardia**—3 or more PVCs in row, 100–200 bpm
 - Nonsustained VT (NSVT)—persists for <30 s
 - Sustained VT—persists for >30 s
 - Treatment
 - Symptomatic: Electrical cardioversion followed by antiarrhythmic drugs; follow ACLS protocol
 - Asymptomatic NSVT: β-blockers, implantable cardioverter-defibrillator (ICD) in pts at high risk
 - Unstable: Defibrillation as if ventricular fibrillation

- **Torsades de pointes**
 - Polymorphic VT with varying amplitudes of QRS twisting about the baseline
 - Treatment: Magnesium 1–2 g IV followed by infusion
- **Ventricular fibrillation**
 - Chaotic irregular appearance without discrete QRS waveforms
 - Treatment: See ACLS protocol; ICD if arrhythmia not associated with acute MI

Hypertrophy

- **Right atrial hypertrophy**
 - Large, biphasic P wave with tall initial component
- **Left atrial hypertrophy**
 - Large, biphasic P wave with wide terminal component
- **Ventricular hypertrophy**
 - **Right ventricular hypertrophy**
 - R wave $\downarrow$ S in V1 (R wave becomes progressively smaller from V1 to V6)
 - S wave persists in V5 & V6
 - Right axis deviation with slightly widened QRS
 - Rightward rotation in horizontal plane
 - **Left ventricular hypertrophy**
 - S wave in V1 $\uparrow$ R wave in V5 ≥ 35 mm
 - Left axis deviation with slightly widened QRS
 - Leftward rotation in horizontal plane
 - Inverted T wave that slants downward gradually but upward quickly

Electrolyte Imbalances

- Hypokalemia
 - Flattened T wave
 - U waves
- Hyperkalemia
 - Peaked T waves
 - Wide or flat P wave
 - Wide QRS
- Hyper/hypocalcemia
 - Hypertalemia—shortened QT
 - Hypocalcemia—prolonged QT

Drug Effects

- Digitalis toxicity
 - Inverted or flattened T waves
 - Shortened QT interval

Pulmonary Embolism

- Right axis deviation
- Acute RBBB
- Inverted T waves in V1 to V4 from right ventricular overload
- Wide S in I large Q and Inverted T in III

Pericarditis

- Diffuse ST-segment elevation (looks similar to acute MI, usually more universal in nature)
- May see subsequent inverted T waves (similar to acute MI)

Hypothermia

- J wave or Osborne wave

Informed Consent

- Should be read & signed by patient prior to administering sedatives
- Principle of patient autonomy (patient may accept/refuse treatment)
- Contains description of procedure, potential risks and benefits
- Incapacitated pts (under influence of meds, altered consciousness, incompetent, disabled) → next of kin/health-care proxy/court-appointed guardian should provide consent
- Telephone consent acceptable, preferably cosigned by a witness
- Consent may be waived in an emergency situation
- Resuscitation efforts generally do not require informed consent because they are considered emergency interventions and consent is implied
- For non-English speakers, use an official interpreter (not family or care team member) whenever possible
- In addition to standard language of the consent form, document additional potential complications & procedures as discussed with patient

Comparative pharmacology of oral Anticoagulants:

	<i>Time to peak effect (hrs)</i>	<i>Duration after discontinuation (days)</i>	<i>Initial Adult dose (mg)</i>	<i>Maintenance adult dose (mg)</i>
1 Warfarin	36-72	2-5	15, 1 st day 10, second 10, 3 rd day	2.5-10
2 Dicumarol	36-48	2-6	200-300 1 st day	25-200
3 Phenindione	18-24	1-2	300, 1 st day, 200 second day	25-200

Brain Death Criteria—Adults & Children

Coma	Absence of gag reflex
Absence of motor responses	Absence of coughing in response to tracheal suctioning
Absence of pupillary responses to light	Absence of sucking and rooting reflexes
Absence of corneal reflexes	Absence of respiratory effort at PaCO ₂ of 60 mm Hg or 20 mm Hg > pts nl value
Absence of caloric (vestibulo-ocular) reflexes	

Required Interval Between Two Evaluations for Brain Death Determination

Term to 2 mo	48 hr
2 mo to 1 yr old	24 hr
>18 yr old	2 evals not required

Required Confirmatory* Testing

Term to 2 mo	2 confirmatory tests
2 mo to 1 yr old	1 confirmatory test
>1 yr old	Optional

*Confirmatory tests: Cerebral angio, EEG, transcranial Doppler ultrasound, cerebral scintigraphy

CARDIOVERSION	MEDICATIONS
<u>Asystolic equipment</u> Pulse oximeter Suction IV access Intubator equipment	adenosine: 6 mg rapid IVP then 20 cc NS bolus. 12 mg IVP ≤ 2 min $\times 2$ if needed
<u>Pre-prepare</u> midazolam 1-5 mg fentanyl 100-300 μ g titrate to effect	amiodarone: 150 mg IV over 10 min diltiazem: 15-20 mg IV over 2 min, 20-25 mg 15 min later if needed. 5-15 mg/h
<u>Synchronized cardioversion on</u> 100, 200, 300, 360 J or bichasoc equivalent	ibutilide: 1 mg over 10 min, repeat $\times 1$ if needed lidocaine: 1-1.5 mg/kg IVP repeat in 5-10 min metoprolol 5 mg IV q5 min $\times 3$ procainamide 17 mg/kg at 50 mg/min (avoid if EF low) verapamil 2.5-5 mg IV over 2 min, 5-10 mg 15-30 min later if needed

Fick Cardiac Output

Oxygen consumption (L/min) = CO (L/min) $\times$ arteriovenous (AV) oxygen difference

CO = oxygen consumption / AV oxygen difference

Oxygen consumption must be measured (can estimate w/ 125 ml/min/m², but inaccurate)

AV oxygen difference = Hb (g/dl) $\times 10$ (dl/L) $\times 1.36$ (ml O₂/g of Hb) $\times (S_aO_2 - S_vO_2)$

S_aO₂ is measured in any arterial sample (usually 93-98%)

S_vO₂ (mixed venous O₂) is measured in RA, RV, or PA (assuming no shunt) (normal 75%)

$$\text{Cardiac output (L/min)} = \frac{\text{Oxygen consumption}}{\text{Hb (g/dl)} \times 1.36 \times (S_aO_2 - S_vO_2)}$$

Shunts

$$Q_p = \frac{\text{Oxygen consumption}}{S_{aO_2} - \text{Pulm. vein } O_2 \text{ sat}} \quad \text{(if no R } \rightarrow \text{ L shunt, PV } O_2 \text{ sat} = S_{vO_2})$$

$$Q_t = \frac{\text{Oxygen consumption}}{S_aO_2 - \text{mixed venous } O_2 \text{ sat}} \quad \text{(MVO}_2 \text{ drawn proximal to potential L } \rightarrow \text{ R shunt)}$$

$$\frac{Q_s}{Q_t} = \frac{S_aO_2 - \text{MV } O_2 \text{ sat}}{\text{PV } O_2 \text{ sat} - \text{PA } O_2 \text{ sat}} = \frac{S_aO_2 - \text{MV } O_2 \text{ sat}}{S_vO_2 - \text{PA } O_2 \text{ sat}} \quad \text{(if only L } \rightarrow \text{ R and no R } \rightarrow \text{ L shunt)}$$

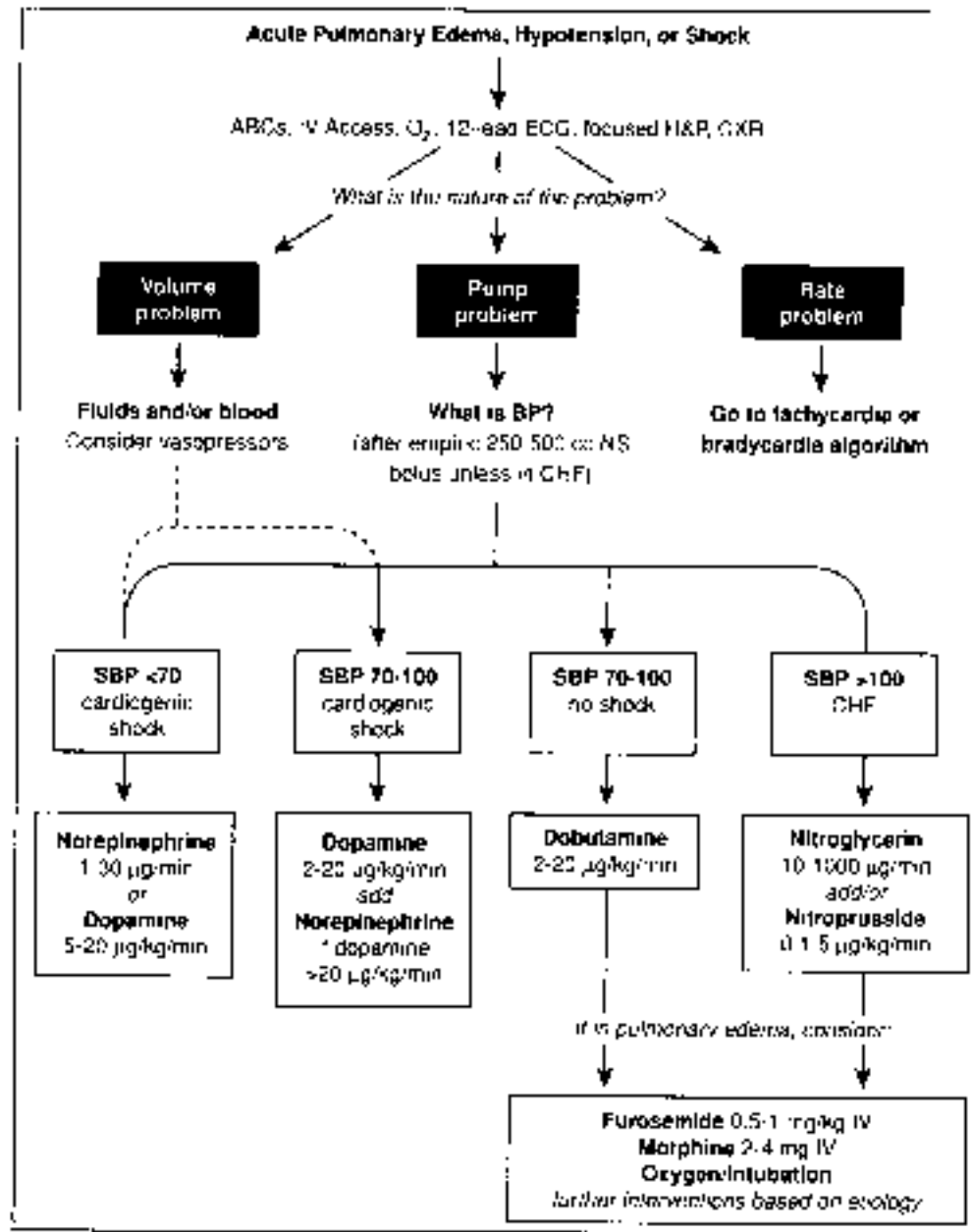
CARDIOLOGY

Hemodynamic Parameters	Normal Value
Mean arterial pressure (MAP) = $\frac{SBP + (DBP \times 2)}{3}$	70-100 mmHg
Heart rate (HR)	60-100 bpm
Right atrial pressure (RA)	0-6 mmHg
Right ventricular (RV)	Systolic 15-30 mmHg Diastolic 1-8 mmHg
Pulmonary artery (PA)	Systolic 15-30 mmHg Mean 9-18 mmHg Diastolic 6-12 mmHg
Pulmonary capillary wedge pressure (PCWP)	0-12 mmHg
Cardiac output (CO)	4-8 L/min
Cardiac index (CI) = $\frac{CO}{BSA}$	2.6 $\pm$ 0.2 L/min/m ²
Stroke volume (SV) = $\frac{CO}{HR}$	60-120 ml/contraction
Stroke volume index (SVI) = $\frac{CI}{HR}$	40-50 ml/contraction/m ²
Systemic vascular resistance (SVR) MAP - mean RA CO $\times 80$	800-1200 dynes $\times$ sec/cm ⁵
Pulmonary vascular resistance (PVR) mean PA - mean PCWP CO $\times 80$	120-250 dynes $\times$ sec/cm ⁵

("Rule of 6s" for PA catheter-measured pressures: RA = 6, RV = 30-6, PA = 30-12, WVP = 12)

(1 mmHg = 1.36 cm water or blood)

Diagram & Flow Charts



Anti-Hypertensive Therapy: In PIH

Drugs	Daily Dose	Adverse effects & comments
Methyldopa (Centrally acting alpha agonist)	500 - 3000 mg in 2 - 4 divided doses	safety of mother & fetus (after the first trimester) Postural Hypotension, drowsiness, fluid retention
Labetalol (Alpha & beta adrenergic blocker)	200 - 2400 mg in 2 - 3 divided doses	Safety appears equal to methyldopa. Headache, dry mouth, tremors, heart block. Avoid in bronchial asthma & CCF use with caution in Diabetes
Nifedipine (calcium channel blocker)	30 - 120 mg in 1 - 4 divided doses depending on preparation used.	May inhibit labour; may have synergistic action with MgSO ₄
Acute severe hypertension in Pregnancy		
Hydralazine onset 10 - 20 min Peak 60 min DOA - 4 - 6 hrs	5 - 10 mg IV, if no response repeat at 20 min interval. total of 30 mg	Tachycardia & increase C.O
Labetalol	10 - 20 mg IV Push every 10 - 20 min max - 300 mg	Decreases SVR Avoid in Asthmas, CCF heart blocks
NTG	5 - 50 µg/min	Increases ICP

Adult Drug Dosages			IV Dose	ETT Dose
Drug	Indication			
Adenosine	SVT		6 mg, repeat dose with 12 mg	
Amiodarone	SVT, VT/VF, AF/flutter Pulseless VF/VT		150 mg over 10 min, then 1 mg/min 300 mg, repeat with 150 mg	
Atropine	Asystole		1 mg q3-5min, max 0.04 mg/kg note: <0.5 mg can lead to paradoxical bradycardia	1 mg/10 mL dilution, 2-2.5 x IV dose
Calcium	Bradycardia		0.5-1.0 mg q5min, max 0.04 mg/kg	
	Hypocalcemia, hyperkalemia, hypomagnesemia		Chloride: 5-10 mg/kg Gluconate: 12-30 mg/kg	
Diltiazem	AF with RVR, reentrant tachycardia		0.25 mg/kg bolus, may repeat bolus 0.35 mg/kg; 5-15 mg/hr infusion	
Dobutamine	Systolic heart failure		2-20 mcg/kg/min	
Dopamine	Oliguria		1-5 mcg/kg/min	
	Hypotension, CHF		5-20 mcg/kg/min	
Epinephrine	Pulseless VT/VF, asystole		1 mg q3-5min	2-2.5 mg
	Hypotension		0.1-1 mcg/kg/min	
	Bronchospasm, anaphylaxis		0.1-0.25 mg	
Glucose/ Dextrose	Hypoglycemia		25-50 g	
Lidocaine	Refractory VT, PVCs		1-1.5 mg/kg IV, repeat 0.5-0.75 mg/kg q5-10 min, max 3 mg/kg; 15-50 mcg/kg/min infusion	2-2.5 x IV dose
			1-2 g	
Magnesium	Hypomagnesemia, Torsades de pointes			
Naloxone	Narcotic overdose		0.4-2 mg, titrated q2-3 min in 0.2 mg increments	Least desirable route; supported only by anecdotal evidence
Procainamide	Atrial and ventricular arrhythmias		Load: 20 mg/min until toxicity or up to 17 mg/kg; maintenance: 1-4 mg/min	
Sodium bicarbonate	Cardiac arrest		1 mEq/kg (after established ventilation), as per ABG	
	Metabolic acidosis		Base deficit x wt (kg) x 0.2	
Vasopressin	Pulseless VT/VF		40 U x 1 dose	2-2.5 x IV dose
Verapamil	SVT, AF/flutter, WPW		2.5-5 mg over 2 min, repeat 5-10 mg; max total 20 mg	

Pediatric Drug Dosages		
Drug	Indications	ETT dose
Adenosine	SVT	IV dose • 50 kg: 0.05–0.1 mg/kg, repeat dose by increasing 0.05–0.1 mg/kg q1–2min, max 0.3 mg/kg • 50 kg: Adult dose 5 mg/kg; up to 15 mg/kg or 300 mg 0.02 mg/kg q5min, max total 1 mg child, 2 mg adolescent Note: <0.1 mg can lead to paradoxical bradycardia 20 mg/kg
Amiodarone	Pulseless VT/VF	0.03 mg/kg ETT
Atropine	Bradycardia	
Calcium chloride	Hypocalcemia, hyperkalemia, hypermagnesium	
Dobutamine	Systolic heart failure	2.5–15 mcg/kg/min
Dopamine	Hypotension	1 20 mcg/kg/min 0.01 mg/kg (0.1 mL/kg 1 10,000) q3–5min, max 1 mg
Epinephrine	Cardiac arrest	0.1 mg/kg (0.1 mL/kg 1:1000) in 1–2 mL diluent, max 10 mg
Glucose/ Dextrose	Hypotension Anaphylaxis Hypoglycemia	0.1 mg/kg (0.1 mL/kg 1:1000) in 1–2 mL diluent, max 10 mg 0.01 mg/kg q20min 0.25 g/kg 6–9 mg/kg/min

(continued)

Pediatric Drug Dosages (Continued)		
Drug	Indications	ETT dose
Lidocaine	Refractory VT, PVCs	IV dose 1 mg/kg, max 100 mg 20–50 mcg/kg/min ETT dose 2–3 mg/kg
Magnesium	Hypomagnesemia, Torsades de pointes	25–50 mg/kg over 10–20 min, max 2 g
Naloxone	Narcotic overdose	0.005–0.01 mg/kg q3–5min Least desirable route; supported only by anecdotal evidence
Procainamide	Atrial and ventricular arrhythmias	Load: 3–6 mg/kg, max 100 mg; Maintenance: 20–80 mcg/kg/min
Sodium bicarbonate	Cardiac arrest	1 mEq/kg (after established ventilation)
Verapamil	Metabolic acidosis SVT	Base deficit $\times$ wt (kg) $\times$ 0.3 • 1 yr old: 0.1–0.2 mg/kg over 2 min q30min • 1–15 yr old: 0.1–0.3 mg/kg, max 5 mg, repeat 15 min, max 10 mg

Valve area

Gorlin equation: Valve area = $\frac{CO \text{ (DEP or SEP)} \times HR}{44.3 \times \text{constant} \times \sqrt{\Delta P}}$ (constant = 1 for AS, 0.85 for MS)

Hakki equation: Valve area = $\frac{CO}{\sqrt{\Delta P}}$

Coronary Artery Anatomy

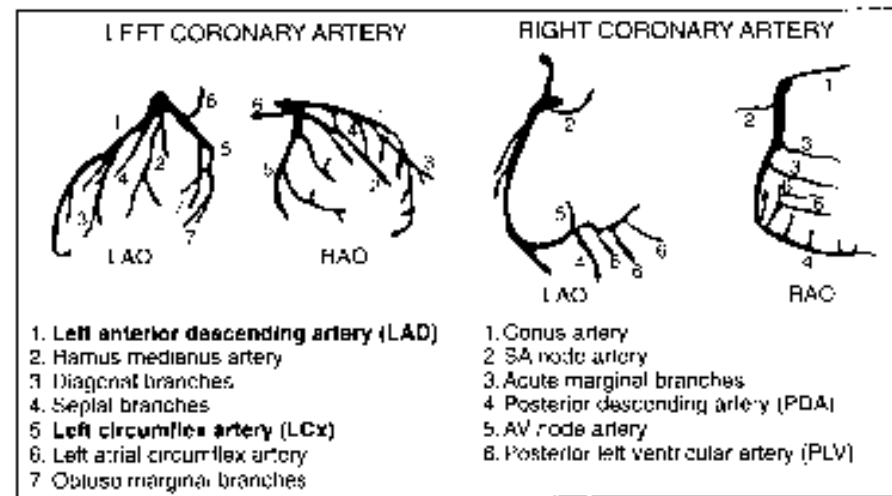


Figure App.-1 Coronary arteries.

(From Grossman WG. Cardiac Catheterization and Angiography, 4th ed. Philadelphia: Lea & Febiger, 1991, with permission.)

PULMONARY

Dead space – lung units that are ventilated but not perfused

Intrapulmonary shunt – lung units that are perfused but not ventilated

Alveolar gas equation: $P_AO_2 = F_iO_2 \times (760 - 47) - \frac{P_aCO_2}{R}$ (where R = 0.8)

$P_AO_2 = 150 - \frac{P_aCO_2}{0.8}$ (on room air)

A-a gradient = $P_AO_2 - P_aO_2$ (normal A-a gradient = $4 + (\text{age}/4)$)

Minute ventilation ($\dot{V}_E$) = tidal volume (V_T) $\times$ respiratory rate (RR) (normal 4–6 L/min)

Tidal volume (V_T) = alveolar space (V_A) + dead space (V_D)

Fraction of tidal volume that is dead space $\left(\frac{V_D}{V_T}\right) = \frac{P_ACO_2 - P_{\text{expired}}CO_2}{P_ACO_2}$

$P_aCO_2 = k \times \frac{CO_2 \text{ Production}}{\text{alveolar ventilation}} = k \times \frac{V_{CO_2}}{RR \times V_T \times \left(1 - \frac{V_D}{V_T}\right)}$

NEPHROLOGY

Anion gap (AG) = $Na - (Cl + HCO_3)$ (normal = $[alb] \times 2.5$; typically 12 ± 2 mEq)

Delta-delta ($\Delta\Delta$) = ΔAG (i.e., calc. AG - expected) / ΔHCO_3 (i.e., $24 - \text{measured } HCO_3$)

Urine anion gap (UAG) = $(U_{Na} + U_K) - U_{Cl}$

Calculated osmoles = $(2 \times Na) + \left(\frac{glc}{18}\right) + \left(\frac{BUN}{2.8}\right) + \left(\frac{EtOH}{4.6}\right)$

Osmolal gap (OG) = measured osmoles - calculated osmoles (normal = 10)

Estimated creatinine clearance = $\frac{140 - \text{age (yrs)} \times \text{wt (kg)}}{\text{serum Cr (mg/dl)} \times 72}$ ($\times 0.85$ in women)

Fractional excretion of Na ($FE_{Na}, \%$) = $\left[\frac{\frac{U_{Na}(\text{mEq/L})}{P_{Na}(\text{mEq/L})} \times 100\%}{\frac{U_{Cr}(\text{mg/ml})}{P_{Cr}(\text{mg/dl})} \times 100 (\text{ml/dl})} \right] = \frac{U_{Na}}{P_{Na}} \times \frac{P_{Cr}}{U_{Cr}}$

Corrected Na in hyperglycemia

$$\text{estimate in all Pts: corrected Na} = \text{measured Na} + \left[2.4 \times \frac{(\text{measured glc} - 100)}{100} \right]$$

however, Δ in Na depends on glc (Acad Med 1998; 73:359)

Δ is 1.6 mEq per each 100 mg/dl $\uparrow$ in glc ranging from 100-440

Δ is 4 mEq per each 100 mg/dl $\uparrow$ in glc beyond 440

Total body water (TBW) = 0.60 $\times$ IBW ($\times$ 0.85 if female and $\times$ 0.85 if elderly)

$$\text{Free H}_2\text{O deficit} = \text{TBW} \times \left(\frac{\text{Na}_{\text{current}} - 140}{140} \right) \times \left(\frac{\text{Na}_{\text{desired}} - 140}{3} \right) \quad (\text{in 70 kg Pt})$$

$$\text{Trans-tubular potassium gradient (TTKG)} = \frac{U_K}{P_K} \div \frac{U_{Osm}}{P_{Osm}}$$

HEMATOLOGY

HEMATOLOGY**Heparin for Thromboembolism**

80 U/kg bolus
18 U/kg/h

PTT	Adjustment
< 40	Bolus 5000 U, $\uparrow$ rate 300 U/h
40-49	Bolus 3000 U, $\uparrow$ rate 200 U/h
50-59	$\uparrow$ Rate 100 U/h
60-85	no Δ
86-95	$\downarrow$ Rate 100 U/h
96-120	Hold 30 min, $\downarrow$ rate 150 U/h
> 120	Hold 60 min, $\downarrow$ rate 200 U/h

(Circ 2001; 103:2994)

Heparin for ACS**STEMI w/ fibrinolysis**

60 U/kg bolus (max 4000 U)
12 U/kg/h (max 1000 U/h)

UA/NSTEMI

60-75 U/kg bolus (max 5000 U)
12-15 U/kg/h (max 1000 U/h)

PTT	Adjustment
< 40	Bolus 3000 U, $\uparrow$ rate 100 U/h
40-49	$\uparrow$ Rate 50 U/h
50-70	no Δ
71-85	$\downarrow$ Rate 50 U/h
86-100	Hold 30 min, $\downarrow$ rate 100 U/h
101-150	Hold 60 min, $\downarrow$ rate 150 U/h
> 150	Hold 60 min, $\downarrow$ rate 300 U/h

(ACC/AHA 2004 Guideline for STEMI)

- ✓ PTT q6h after every change (half-life of heparin is ~90 min)
- ✓ PTT qd or bid once PTT is therapeutic
- ✓ CBC qd (to ensure Hct and plt counts are stable)

OTHER

Ideal body weight (IBW) = [50 kg (men) or 45.5 kg (women)] + 2.3 kg/inch over 5 ft

$$\text{Body surface area (BSA, m}^2\text{)} = \sqrt{\frac{\text{height (cm)} \times \text{weight (kg)}}{3600}}$$

Test	Disease	
	present	absent
	a (true +)	b (false +)
	c (false -)	d (true -)

$$\text{Prevalence} = \frac{\text{all diseased}}{\text{all patients}} = \frac{a + b}{a + b + c + d}$$

$$\text{Sensitivity} = \frac{\text{true positives}}{\text{all diseased}} = \frac{a}{a + c} \quad \text{Specificity} = \frac{\text{true negatives}}{\text{all healthy}} = \frac{d}{b + d}$$

$$\oplus \text{ Predictive value} = \frac{\text{true positives}}{\text{all positives}} = \frac{a}{a + b}$$

$$\ominus \text{ Predictive value} = \frac{\text{true negatives}}{\text{all negatives}} = \frac{d}{c + d}$$

$$\text{Accuracy} = \frac{\text{true positives} + \text{true negatives}}{\text{all patients}} = \frac{a + d}{a + b + c + d}$$

$$\oplus \text{ Likelihood ratio} = \frac{\text{true positive rate}}{\text{false positive rate}} = \frac{\text{Se}}{1 - \text{Sp}}$$

$$\ominus \text{ Likelihood ratio} = \frac{\text{false negative rate}}{\text{true negative rate}} = \frac{1 - \text{Se}}{\text{Sp}}$$

$$\text{Odds} = \frac{\text{probability}}{1 - \text{probability}} \quad \text{Probability} = \frac{\text{odds}}{\text{odds} + 1}$$

$$\text{Posttest odds} = \text{pretest odds} \times \text{LR}$$

APPENDIX C: SETTING UP THE OR FOR AN ANESTHESIA CASE

Adapted from "The CA-1 Survival Guide, 2008" Brigham and Women's Hospital

MNEMONIC: "SOAP M"

S: Suction

Make sure suction is on, plugged in tightly to the canister, on at full blast

O: Oxygen

Check that pipeline supply is between 50–55 psi and connected to the wall

Check that the cylinder in the back of the machine has adequate oxygen

(1000 psi) in case of emergency

Calibrate O₂ sensor

Make sure an Ambu Bag is available

A: Airway

Check all laryngoscopes and handles

Choose appropriate ETT and check cuff for leaks. Have an ETT stylette available

Make sure an LMA is available

Oral airway, bite block, and tape

Stethoscope

P: Pharmacy

Draw up rescue drugs needed for the case (including a sedative, induction agent, paralytics)

Make sure emergency drugs (epinephrine, atropine, extra succinylcholine) are available

Make sure vaporizers are adequately filled

Make sure appropriate antibiotics are available

Set up appropriate drips and have a functioning drug infusion pump

M: Machine/Monitors

Check the machine (see Anesthesia Machine checkout recommendations, Appendix B)

Check the monitors

Check that the BP cuff is present and of appropriate size

Check that you have a pulse oximeter, EKG cables and electrodes

Check that the nerve stimulator is present and functioning

Fluid warmer and patient warmer devices

Arm straps

A-line, CVP, PA, and EEG monitors as indicated

EMERGENCY THERAPY FOR MALIGNANT HYPERTHERMIA

MH Hotline 1-800-644-9737

Outside the US: 1-315-464-7079

Diagnosis vs Associated Problems

Signs of MH:

- Increasing ETCO₂
- Trunk or total body rigidity
- Masseter spasm or trismus
- Tachycardia/tachypnea
- Mixed respiratory and metabolic acidosis
- Increased temperature (may be late sign)
- Myoglobinuria

Sudden/Unexpected Cardiac Arrest in Young Patients:

- Presume hyperkalemia and initiate treatment (see #6)
- Measure CK, myoglobin, ABGs, until normalized
- Consider dantrolene
- Usually secondary to occult myopathy (e.g., muscular dystrophy)
- Resuscitation may be difficult and prolonged

Trismus or Masseter Spasm with Succinylcholine

- Early sign of MH in many patients
- If limb muscle rigidity, begin treatment with dantrolene.
- For emergent procedures, continue with non-triggering agents, evaluate and monitor the patient, and consider dantrolene treatment.
- Follow CK and urine myoglobin for 36 hours
- Check CK immediately and at 6 hour intervals until returning to normal. Observe for dark or cola-colored urine. If present, liberalize fluid intake and test for myoglobin.
- Observe in PACU or ICU for at least 12 hours.

Acute Phase Treatment**1. GET HELP. GET DANTROLENE – Notify Surgeon**

- Discontinue volatile agents and succinylcholine.
- Hyperventilate with 100% oxygen at flows of 10 L/min, or more.
- Halt the procedure as soon as possible; if emergent, continue with non-triggering anesthetic technique.
- Don't waste time changing the circle system and CO₂ absorbant.

2. Dantrolene 2.5 mg/kg IV rapidly, through large-bore IV if possible

To convert kg to lb, for amount of dantrolene, give patients 1 mg/lb (2.5 mg/kg approximates 1 mg/lb).

- Dissolve the 20 mg in each vial with at least 60 ml sterile, preservative-free water for injection. Prewarming (not to exceed 39°C) the sterile water may expedite solubilization of dantrolene. However, to date, there is no evidence that such warming improves clinical outcome.
- Repeat until signs of MH are reversed.
- Sometimes more than 10 mg/kg (up to 30 mg/kg) is necessary.
- Each 20 mg bottle has 3 gm mannitol for isotonicity. The pH of the solution is 9.

3. Bicarbonate for metabolic acidosis

- 1–2 mEq/kg if blood gas values are not yet available.

4. Cool the patient with core temperature >39°C. Lavage open body cavities, stomach, bladder, or rectum. Apply ice to surface. Infuse cold saline intravenously. Stop cooling if temp. <38°C and falling to prevent drift <36°C.**5. Dysrhythmias usually respond to treatment of acidosis and hyperkalemia.**

- Use standard drug therapy **except calcium channel blockers, which may cause hyperkalemia or cardiac arrest in the presence of dantrolene.**

6. Hyperkalemia—Treat with hyperventilation, bicarbonate, glucose/insulin, calcium.

- Bicarbonate: 1–2 mEq/kg IV.
- Insulin: For **pediatric**, 0.1 units insulin/kg and 1 ml/kg 50% glucose or for **adult**, 10 units regular insulin IV and 50 ml 50% glucose.

- Calcium chloride 10 mg/kg or calcium gluconate 10–30 mg/kg for life-threatening hyperkalemia.

- Check glucose levels hourly

- 7. Follow ETCO₂, electrolytes, blood gases, CK, core temperature, urine output and color, coagulation studies.** If CK and/or K⁺ rise more than transiently or urine output falls to less than 0.5 ml/kg/hr, induce diuresis to >1 ml/kg/hr and give bicarbonate to alkalinize urine to prevent myoglobinuria-induced renal failure. (See D below.)
- Venous blood gas (e.g., femoral vein) values may document hypermetabolism better than arterial values
 - Central venous or PA monitoring as needed and record minute ventilation.
 - Place Foley catheter and monitor urine output.

Post Acute Phase**A. Observe the patient in an ICU for at least 24 hours, due to the risk of recrudescence.****B. Dantrolene 1 mg/kg q4–6h or 0.25 mg/kg/hr by infusion for at least 24 hours. Further doses may be indicated.****C. Follow vitals and labs as above (see #7 above)**

- Frequent ABG as per clinical signs
- CK every 8–12 hours; less often as the values trend downward

D. Follow urine myoglobin and institute therapy to prevent myoglobin precipitation in renal tubules and the subsequent development of Acute Renal Failure. CK levels above 10,000 IU/L is a presumptive sign of rhabdomyolysis and myoglobinuria. Follow standard intensive care therapy for acute rhabdomyolysis and myoglobinuria (urine output >2 ml/kg/hr by hydration and diuretics along with alkalinization of urine with Na-bicarbonate infusion with careful attention to both urine and serum pH values).**E. Counsel the patient and family regarding MH and further precautions: refer them to MHAUS. Fill out and send in the Adverse Metabolic Reaction to Anesthesia (AMRA) form (www.mhreg.org) and send a letter to the patient and her/his physician. Refer patient to the nearest Biopsy Center for follow-up**

How will you grade severity of mitral stenosis?

Severity	MVA, cm ²	Gradient, mm Hg	PAP	Symptoms	Signs
Mild	>1.8	2-4	Normal	Usually absent	S ₁ -OS >120 ms; normal P ₂
Moderate	1.2-1.6	4-9	Normal	Class II	S ₁ -OS 100-120 ms, normal P ₂
Moderate to severe	1.0-1.2	10-15	Mild pulmonary HTN	Class II-III	S ₁ -OS 80-100 ms; P ₂ increase
Severity	MVA, cm ²	Gradient, mm Hg	PAP	Symptoms	Signs
Severe (Tight)	<1.0	>15	Mild to severe pulmonary Hypertension	Class II-IV	S ₁ -P ₂ increase; OS <80 ms, RV lift Sx if R heart fails

Cormier's grading of mitral valve anatomy

Echocardiographic group	Mitral valve anatomy
Group 1	Pliable non-calcified anterior mitral leaflet and mild subvalvular disease (i.e. thin chordae >= 10 mm long)
Group 2	Pliable non-calcified anterior mitral leaflet and severe subvalvular disease (i.e. thickened chordae < 10 mm long)
Group 3	Calcification of mitral valve of any extent, as assessed by fluoroscopy, whatever the state of subvalvular apparatus

Drug	Indication	Fetal S/E
Furosemide	To decrease congestion associated with valvular heart disease levels	Increased urinary sodium and potassium levels
Beta-blockers	Hypertension, supraventricular arrhythmias, to control heart rate in women with clinically significant mitral stenosis	Possible decreased heart rate, Possible lower birth weight
Warfarin	For anticoagulation of mechanical heart valves, valvular heart disease with associated atrial fibrillation during (12–36 wk) of pregnancy	Hemorrhage, developmental abnormalities when used between (wk 6 and 12) of gestation
Unfractionated heparin	For anticoagulation of mechanical heart valves, valvular heart disease with associated atrial fibrillation during (wk 6–12) and (after wk 36) of pregnancy	Hemorrhage, no congenital defects
Low-molecular-weight heparin	Use during pregnancy is controversial	Hemorrhage
Aspirin	Low-dose aspirin (81 mg/day) occasionally used as an adjunct in patients with previous embolic events or prosthetic-valve thrombosis	Hemorrhage, prolongation of labour, low birth weight (when taken in high doses)
Digoxin	For suppression of supraventricular arrhythmias	No major adverse effects
Adenosine	For immediate conversion of supraventricular arrhythmias	No major adverse effects
Amiodarone	May be used to suppress atrial or ventricular arrhythmias in high-risk patients	Hypothyroidism, intrauterine growth retardation, premature birth. Rarely used during pregnancy because of side effects

Risk assessment	Goldman 1977	Detsky 1986	Ungle 1989	Lee 1999
Age > 70 years	5	5	-	-
Emergency surgery	4	10	-	-
Intraoperative intubation or other surgery	3	-	-	-
Canadian Cardiovascular Society Class I angina	-	10	-	-
Canadian Cardiovascular Society Class II angina	-	20	-	-
Unstable angina in previous 6 months	-	10	-	-
Any angina	-	-	1	-
Myocardial infarction in previous 6 months	10	10	-	-
Myocardial infarction more than 6 months ago	-	5	-	-
Q wave on ECG	-	-	-	-
Third heart sound or jugular venous distension	11	-	-	-
Pulmonary edema in previous 7 days	-	10	-	-
Any history of pulmonary edema	-	5	-	-
Significant aortic stenosis (aortic gradient > 50 mmHg)	2	20	-	-

	Goldman 1977	Detsky 1986	Eagle 1989	Lee 1999
ECG rhythm other than sinus with or without APBs	1	5	-	-
1°/2° PVBs/min documented on ECG before surgery	1	5	-	-
History of ventricular ectopy	-	-	1	-
Diabetes mellitus	-	-	1	-
Preoperative medical status ^a	3	5	-	-
High risk surgery	-	-	-	1
History of ischaemic heart disease	-	-	-	1
History of congestive cardiac failure	-	-	-	1
History of cerebrovascular disease	-	-	-	1
Preoperative treatment with insulin	-	-	-	1
Preoperative serum creatinine > 177 µmol/l	-	-	-	1
Total possible points	53	120	5	6
Risk Class I	0-5 (1%)	0-15 (0.4%)	0-10 (3%)	0-4 (0.5%)
Risk Class II	6-12 (6-6%)	20-30 (4-33%)	1-2 (1-16%)	1-3 (9-17%)
Risk Class III	13-25 (13-8%)	> 30 (17-60%)	> 3 (25-50%)	2-4 (7-17%)
Risk Class IV	> 26 (48%)	-	-	> 4 (5-17%)

Post-anesthesia Recovery Score (Modified Aldrete Score)	Post-anesthesia Discharge Scoring System
Activity	Vital signs (BP and Pulse)
2 = Moves all extremities voluntarily/on command	2 = Within 20% of preoperative baseline
1 = Moves two extremities	1 = 20%–40% of preoperative baseline
0 = Unable to move extremities	0 = >40% of preoperative baseline
Respiration	Activity
2 = Breathes deeply and coughs freely	2 = Steady gait, no dizziness
1 = Dyspneic, shallow or limited breathing	1 = Requires assistance
0 = Apneic	0 = Unable to ambulate
Consciousness	Pain
2 = Fully awake	Acceptable to patient; control with PO medications
1 = Arousable on calling	2=Yes
0 = Not responding	1 = No

Oxygen Saturation	Surgical Bleeding
2 = SpO ₂ >92% on room air	2 = Minimal: no dressing change required
1 = Supplemental O ₂ req. to maintain SpO ₂ >90%	1 = Moderate: up to 2 dressing changes
0 = SpO ₂ <92% with O ₂ supplementation	0 = Severe: more than 3 dressing changes
10 = Total score	10 = Maximum score
Score >9 required for discharge	Score >9 required for discharge

Table: Causes of imbalance in myocardial oxygen supply and demand

Decreased Oxygen Supply	Increased Oxygen Demand
Decreased coronary blood flow	Tachycardia
Tachycardia (low diastolic perfusion time)	Increased wall tension
Hypotension (especially diastolic)	Increased preload
	Increased afterload (hypertension)
Increased preload (low perfusion pressure)	Increased myocardial contractility
Hypocapnia (coronary vasoconstriction)	
Coronary artery spasm	
Decreased oxygen content and availability	
Anemia	
Hypoxemia	
Reduced oxygen release from hemoglobin (e.g., pH, 2,3-DPG, temperature)	

Define hypertension.

Category	Systolic Blood Pressure	And	Diastolic Blood Pressure
Optimal	< 120	And	< 80
Normal	< 130 and	And	< 85
High normal	130-139	Or	85-89
Mild hypertension	140-159	Or	90-99
Moderate hypertension	160-179	Or	100-109
Severe hypertension	≥ 180	And	≥ 110
Isolated systolic hypertension	≥ 140	And	< 90

Factors which increase pacing threshold	Factors which decrease pacing threshold
1-4 weeks after implantation	Increased catecholamines
Myocardial ischaemia/infarction	Stress, anxiety
Hypothermia, hypothyroidism	Sympathomimetic drugs
Hyperkalaemia, acidosis/alkalosis	Anticholinergics
Antiarrhythmics (class Ic,3)	Glucocorticoids
Antiarrhythmics (class IA/B,2)	Hyperthyroidism
Severe hypoxia/hypoglycaemia	Hypermetabolic status
Inhalation-local anaesthetics	

Agent	MAVC
Nitrous Oxide	105
Halothane	0.75
Isoflurane	1.2
Desflurane	6.0
Sevoflurane	2.0
Enflurane	1.7

There are various positions for postural drainage depending on the site of the affected lobe/lobes



What is the spirometric classification of COPD?

According to Global initiative for chronic Obstructive Lung Disease (www.goldcopd.com), COPD severity can be graded as follows:

Stage	Severity	Spirometry
Stage 0	None	At risk, cough or sputum present but lung function normal*
Stage I	Mild	$FEV_1/FVC < 0.70$, $FEV_1 > 80\%$ predicted
Stage II	Moderate	$FEV_1/FVC < 0.70$, $50\% < FEV_1 < 80\%$ predicted
Stage III	Severe	$FEV_1/FVC < 0.70$, $30\% < FEV_1 < 50\%$ predicted
Stage IV	Very Severe	$FEV_1/FVC < 0.70$, or $FEV_1 < 30\%$ predicted OR $FEV_1 < 50\%$ predicted plus chronic respiratory failure

Maximum Safe Dose (mg/kg)

- Bupivacaine 2.0
- Levobupivacaine 2.5-3.0
- Articaine 7.0
- Lignocaine 4.0
- with epinephrine 7.0
- Mepivacaine 7.0
- Prilocaine 6.0
- Ropivacaine 3.0-4.0

Table 3. Safe doses of common LAs

	Mild	Severe
Systolic BP	< 100 mmHg	110 mmHg or higher
Proteinuria	Trace or 1 +	Persistent 2 + or more
Headache	Absent	Present
Visual disturbances	Absent	Present
Upper abdominal pain	Absent	Present
Oliguria	Absent	Present
Convulsion	Absent	Present (eclampsia)
Serum creatinine	Normal	Elevated
Thrombocytopenia	Absent	Present
Liver enzyme elevation	Minimal	Marked
Fetal growth restriction	Absent	Obvious
Pulmonary edema	Absent	Present

Changes in Coagulation system

Coagulation factor/ Parameter	Changes
Fibrinogen	Increased from 2.5g/l to 5g/l
Factor II	Slightly Increased
Factor V	Slightly Increased
Factor VII	Increased 10 folds
Factor VIII	Increased 2 fold
Factor IX and X	Increased
Factor XI	Decreased 70%
Factor XII	Increased 40%
Factor XIII	Decreased 40%
Bleeding Time	WNL
PT	WNL
PTT	WNL

Changes in Anemia:

Severity of anaemia: Anaemia is defined as a qualitative or quantitative deficiency of-- hemoglobin. It can be classified as follows:

- Mild anemia Hemoglobin 9-11 gm %
- Moderate anaemia Hemoglobin 7-9 gms %
- Severe anaemia Hemoglobin 4-7 gm %
- Very severe anaemia Hemoglobin <4gm %

Historical overview of various burn resuscitation formulas

1942	Harkins formula	Any patient with atleast 10% burn: administer 1000cm ³ plasma for each 10% TBSA burn over first 24 hr
1947	Body-weight Burn budget	First 24 h: 1-4 lit RL + 1200 ml 0.5 NS + 7-5% body weight colloid + 1.5-5 lit 5% D. Second 24 h: same formulation except change colloid to 2.5% body weight
1952	Evans formula	First 24 h: NS at 1ml/kg/% burn + colloids at 1ml/kg/% burn + 2lit glucose in water. Second 24 h: one half the first 24 h crystalloid & colloid requirement + the same amount of glucose in water as in the first 24h.
1953	Brooke formula	First 24 h: RL at 1.5ml/kg/% TBSA burn + colloid at 0.5ml/kg/% TBSA burn. Second 24 h: 2 lit 5% D
1968	Parkland Formula	First 24 h: RL at 4ml/kg/% TBSA, give half in first 8 h & the remaining over next 16 h. Second 24 h: colloid at 20-60% of calculated plasma volume to maintain adequate urinary output
1974	Muir & Barclay	Colloids (mls) = % TBSA burns x body weight in kg / 2. Time blocks: 4hrly for 1 st 12 hrs, 6 hrly for next 12 hrs, after 12 hrs. A total of 36 hrs post injury. Crystalloid: (1.5ml x body weight) ml/hr. Blood: 50ml/% BSA

1979 Modified Brooke

First 24 h: RL at 2 ml/kg/% TBSA burn, one half in the first 8 h & half in the remaining 16 h. Second 24 h: colloid at 0.3-0.5 ml/kg/% TBSA burn + 5% D to maintain urine output.

1984 Monafio formula

First 24 h: saline with 250 mEq Na + 150 mEq lactate + 100 mEq Cl. Rate adjusted as per urine output. Second 24 h: one third of isotonic salt administered orally

Etiology of halothane Hepatotoxicity:

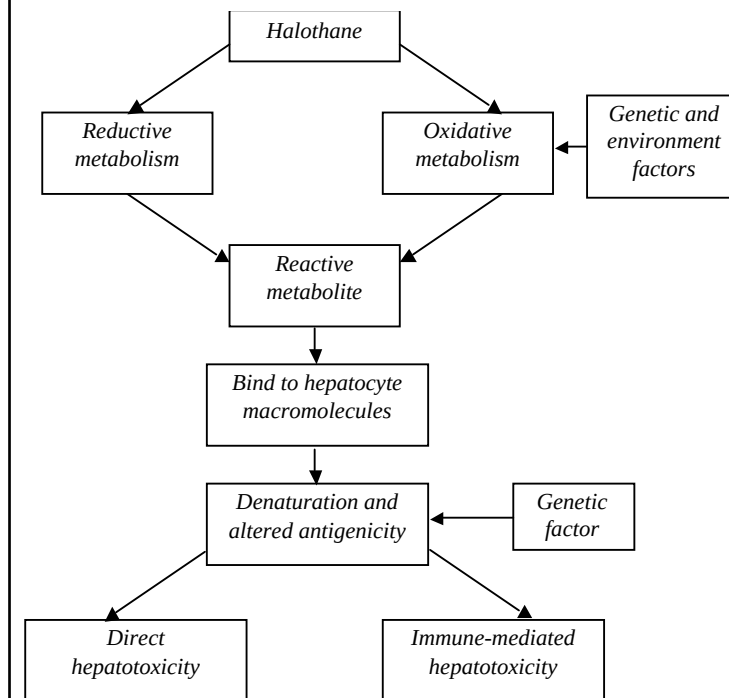


Table 1 Other syndromes associated with Cleft lip and palate and their anaesthetic implications

Syndrome	Associated anomalies	Anaesthetic implications
Pierre Robin Sequence	80% associated with cleft palate Micrognathia Glossoposis Underlying syndrome/anomalies	Difficult airway Postoperative respiratory obstruction Usually easier to intubate with increasing age
Treacher Collins Syndrome	28% associated with cleft palate Micrognathia and maxillary hypoplasia Choanal atresia Eye and ear malformations	Intubation may become more difficult with age Postoperative respiratory obstruction
Hemifacial Microsomia (Goldenhar syndrome)	Hemifacial and mandibular hypoplasia Cervical spine abnormalities Ear and eye abnormalities	Intubation may become more difficult with age
Velocardiofacial Syndrome	Microcephaly and microstomia Flat nasal bridge, small ears, short stature Immune deficiency, congenital cardiac disease, Laryngeal and tracheal anomalies Velopharyngeal incompetence with/ without cleft palate 22q 11 deletion (FISH test)	Difficult airway Antibiotics for congenital heart disease
Stickler Syndrome	Progressive connective tissue disorder Micrognathia and flat face Eye, ear and joint abnormalities Congenital cardiac disease	Difficult airway – both mask ventilation and intubation can be very difficult
Down Syndrome	Microstomia and relative macroglossia Epicanthic folds, simian crease Congenital cardiac disease Atlantoaxial subluxation and instability	Difficult intubation Caution with neck positioning Antibiotics for congenital heart disease Prone to airway obstruction

Syndrome	Associated anomalies	Anaesthetic implications
12° Alcohol Syndrome	Smooth philtrum, thin vermillion Small palpebral fissures Growth retardation CNS abnormalities	Difficult airway Antibiotics for congenital heart disease
Klippel Feil syndrome	15% associated with cleft palate Short, webbed neck and fused cervical vertebrae Congenital cardiac disease Renal anomalies Scoliosis	Difficult intubation Antibiotics for congenital heart disease

American Society of Anesthesiologists Fasting Guidelines.

Clear liquids (i.e. include but are not limited to water, fruit juices without pulp, carbonated beverages, clear tea and black coffee. Does not include alcohol)	2 hr
Breast milk	4 hr
Infant formula	6 hr
Non-human milk	6 hr
Light meal (Toast without butter and clear liquid)	6 hr
Full meal (Fried fatty meal)	> 8 hr

Risk of post-operative MI for non-cardiac surgery (Goldman's Criteria)

i) Patients without previous MI	0.13 %
ii) Patients with previous MI more than 6 months back	4.6 %
iii) Patients with MI 1-6 Months prior to surgery	38.0 %

S. Sodium levels	CNS effects	ECG effects
120 meq/L	Confusion, restlessness	Wide QRS
115 meq/L	Somnolence, nausea	Wide QRS, ST elevation
110 meq/L	Seizures, coma	Vent. Tachycardia / Fibrillation

Sodium in TURP.

Body-mass index (kg/m²)

< 18.5	Underweight
18.5-24.99	Normal
25-26.9	Overweight
27-30	Mild Obesity
> 30	Moderate Obesity
>35	Severe Obesity
>40	Morbid Obesity
> 50	Super Obesity

Table 1 Differences between Haemolytic, Obstructive and Hepatocellular Jaundice

Features	Haemolytic	Obstructive	Hepatocellular
Serum Bilirubin	Modest rise, mainly unconjugated	High, mostly conjugated	High both conjugated and unconjugated
Urine Urobilinogen	High	Absent	Present (absent in predominant cholestatic phase)
Bilirubin, Bile salts & pigments	Absent	High	High
Stool stercobilins	Absent	Present	Present
Stool stercobilins	High	Absent	Present (absent in predominant cholestatic phase)
Liver Enzymes	Normal	Alkaline phosphatase, gGT raised	Elevated transaminases
Coagulopathy	Absent		Present / absent
Response to vit K	NA	Yes	Yes / No

Table 2. Types and differences in Hepatitis viruses

Hepatitis	A Virus	B Virus (non A- non B)	C Virus (delta body)	D virus	E Virus
Spread	Faecal-oral Contaminated food & water Crab/Anal sexual contact	Blood & body fluid contact Sex Needles Mother to baby Human bite	Blood & body fluid contact Needles Mother to baby Sex (minimal)	Co-infection or super infection with HBV	Faecal-oral Contaminated food & water
Incubation	30 (15-50) days	60-90 (45-120) days	6-7 weeks (months)	60-90 (30-150) days	40 (14-60)
Symptoms of Initial infection	Asymptomatic (especially HCV) jaundice, fever, myalgia, Nausea, vomiting, Anorexia, Fatigue Dark urine & pale bowel movements				
Chronic Infection (infection for >6m)	None	1-10% Chronic	85% Chronic	Common	No chronic disease
Prevention	Hep A IgG up to 2 weeks after exposure	Hepatitis B IgG for PEP Recombinant vaccine			
Treatment	Sell-limiting	Interferon Lamivudine Adefovir Pegylated interferon Entecavir Telbivudine	Pegylated interferon plus ribavirin	Interferon?	None
Vaccine	Yes	Yes	No	HBV vaccine	No

Table 4. Portosystemic anastomoses

Site of PS anastomosis	Portal component	Systemic component	Clinical effect
Lower oesophagus	Oesophageal branches of left gastric vein	Azygous veins	Submucosal gastroesophageal varices
Upper anal canal	superior rectal vein	middle inferior rectal veins	May be confused with hemorrhoids
Umbilical (anterior abdominal wall)	Veins of ligamentum teres	Superior/inferior epigastric veins	Caput medusae
Bare area of liver	Hepatic/portal veins	Inferior phrenic veins	
Patent ductus arteriosus (rare)	left branch of portal vein	Inferior vena cava	
Retroperitoneal	Colonic veins	Body wall veins	usually none

Table 5. Grades of Hepatic Encephalopathy

Grade	Level of consciousness	Intellectual function	Neurological findings	EEG
1	Lack of awareness Personality change Day/night reversal	Short attention	Incoordination Mild asterixis	Slowing (5-6 cps) Triphasic
2	Lethargic Inappropriate behavior	Disoriented	Asterixis Abnormal reflexes	Slowing Triphasic
3	Asleep Rousable	Loss of meaningful communication	Asterixis Abnormal reflexes	Slowing Triphasic
4	Unrousable	Absent	Decerebrate	Very slow (2-3 cps), delta

What are the clinical signs of liver cell failure?

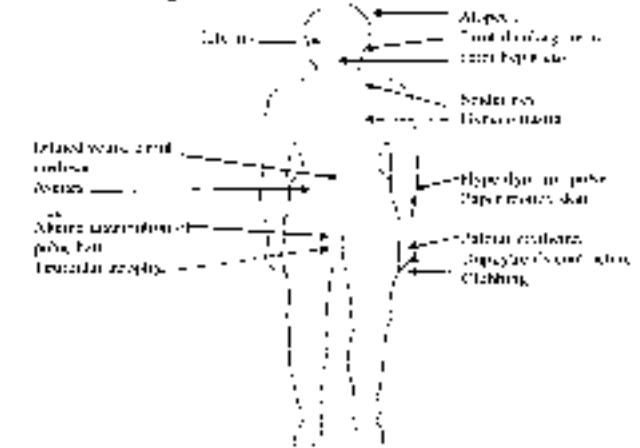


Table 6. Child-Turcotte-Pugh Classification of Cirrhosis (Score range 3-15)

Factor	1	2	3
Serum bilirubin	<2.0 mg/dL	2.0-3.0 mg/dL	>3.0 mg/dL
Serum albumin	>3.5 g/dL	3.0-3.5 g/dL	<3.0 g/dL
Prothrombin time Prolongation in secs or INR	0-4	4-6	>6
Ascites	None	Easily controlled	Poorly controlled
Hepatic encephalopathy	None	Minimal	Advanced

Discuss complications associated with prone position. Discuss postoperative visual loss seen after prone position.

Central nervous system complications of prone position are summarized in the table below.

CNS Complications			
Mechanism	Anatomy	Caused by	Clinical consequence
Arterial occlusion	internal carotid artery, vertebral artery	rotation, unrecognized cervical extension	stroke, hemiplegia, quadriplegia, lateral medullary syndrome, brainstem & cerebellar infarcts
Venous occlusion	abnormal veins	poor positioning	hemipareses, paraparesis, quadriplegia/bilateral venous infarcts
Air entrainment		pneumothorax (air in spinal canal)	transient quadriplegia

(Contd...)

(Contd...)

Mechanism	Anatomy	Caused by	Clinical consequence
Cervical spine injury	Excessive neck flexion	overstretching of cervical cord consequent ischemia	transsection below C5/6
	Excessive neck extension		prolapsed intervertebral disc
Undiagnosed space-occupying lesions		Altered CSF flow dynamics epidural venous engorgement	Previously undiagnosed, lesions became symptomatic

	Ischemic Optic Neuropathy (ION)	Central Retinal Artery Occlusion (CRAO)
Etiology	Intraoperative hypotension, prolonged surgery, excessive blood loss, Large amounts crystalloid infusion	Direct external pressure Emboli
Mechanism	Ischemia (due to factors above) Orbital edema leading to stretching/compression of optic nerve	Decreased ocular perfusion pressure
Clinical Features	Painless, bilateral, decreased light perception and visual fields	Painless, unilateral, periorbital swelling or ecchymosis

CONDITION:	Normal	Hyperthyroidism	Hypothyroidism Primary	Hypothyroidism Secondary
TSH	Normal	Low	High	Low
T4	Normal	High	Low	Low

Diagnostic criteria and typical total body deficits of water and electrolytes in DKA and HHS (Reproduced from ref 10)

Diagnostic criteria	Mild DKA	Moderate DKA	Severe DKA	HHS
Plasma glucose (mg/dl)	>250	>250	>250	>600
Arterial pH	>7.25-7.35	7.0 to < 7.25	<7.0	>7.3
S. Bicarbonate (mEq/L)	15-18	10 to <15	<10	>15
Urine Ketone	+	+	+	small
Serum Ketone	+	+	+	small
Serum osmolality	variable	variable	variable	>320mosm/kg
Anion gap	>10	>12	>12	<12
Mental status	Alert	Alert/drowsy	Stupor/coma	Stupor/coma
Water deficit (ml/kg)	100			100-200
Na deficit (meq/kg)	7-10			5-14
Cl deficit (meq/kg)	3-5			5-15
K deficit (meq/kg)	3-5			4-6
PO ₄ deficit (meq/kg)	5-7			3-7
Mg deficit (meq/kg)	1-2			1-2
Ca deficit (meq/kg)	1-2			1-2

Cockcroft–Gault equation (eC_{cr} = estimated creatinine clearance)

$$eC_{cr} = \frac{(140 - \text{Age}) \times \text{Mass (in kilograms)} \times [0.85 \text{ if female}]}{72 \times \text{Serum Creatinine (in mg/dL)}}$$

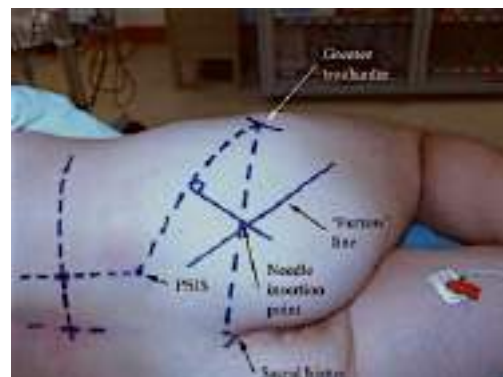
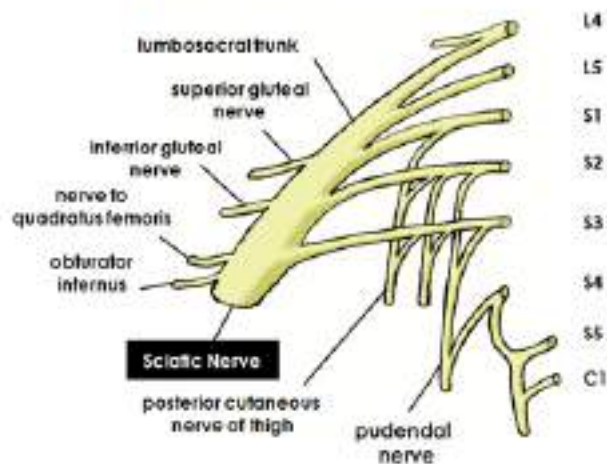
The RIFLE grading system for acute renal dysfunction

Grade	Glomerular filtration rate criteria	Urine output (UO) criteria
R, Risk	Serum creatinine increase: 1.5-fold, GFR decrease: >25%	UO < 0.5 ml/kg/h for 6 h
I, Injury	Serum creatinine increase: 2-fold, GFR decrease: >50%	UO < 0.5 ml/kg/h for 12 h
F, Failure	Serum creatinine increase: 3-fold; GFR decrease: >75%; Serum creatinine decrease: >350 $\mu\text{mol/litre}$ (4 mg/dl) with acute increase > 44 $\mu\text{mol/litre}$ (0.5 mg/dl)	UO < 0.3 ml/kg/h for 24 h or anuria for 12 h
L, Loss	Persistent ARF = complete loss of renal function for > 4 weeks	
E, End stage	ESRD = complete loss of renal function for > 3 months	

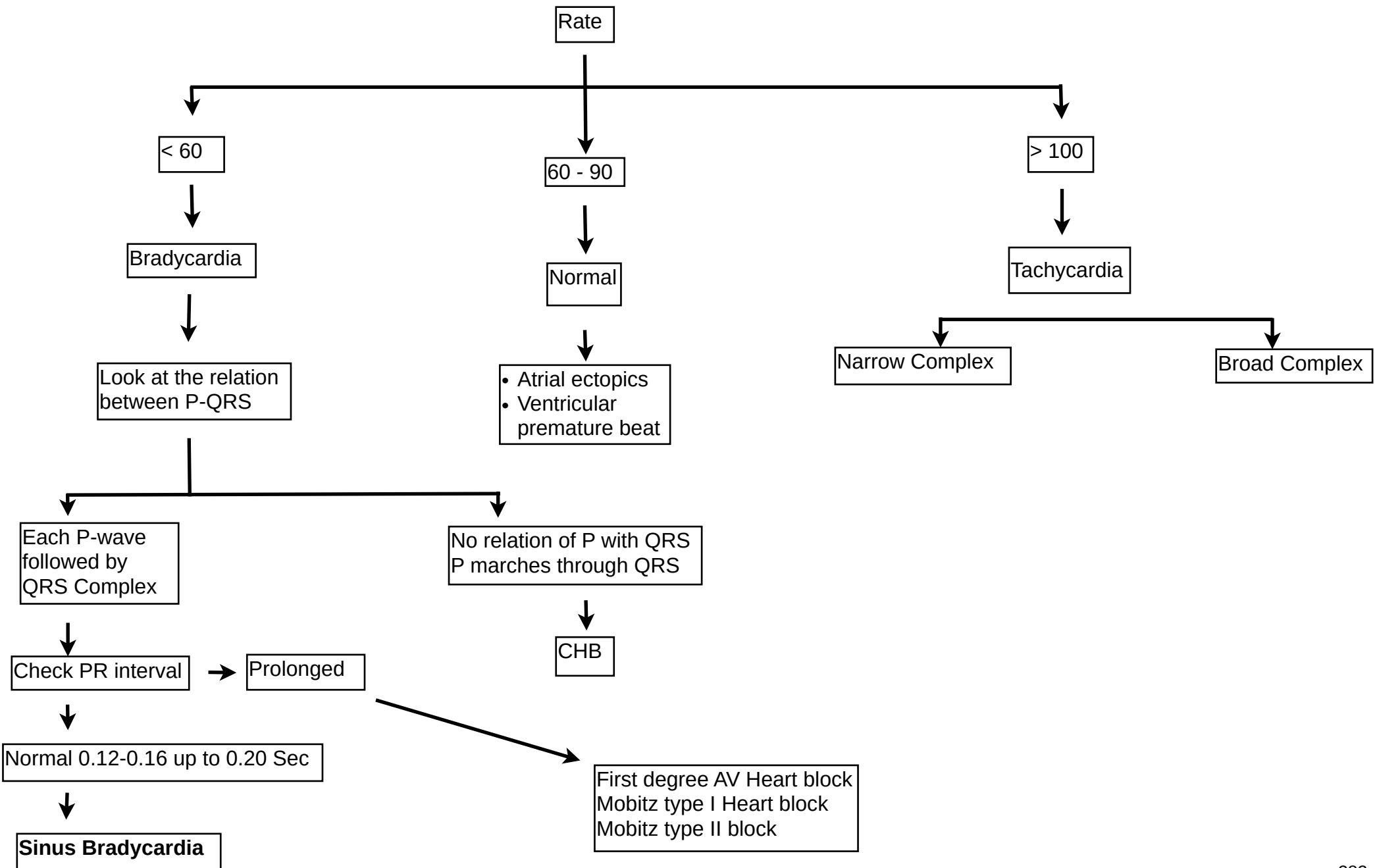
		Oxygen	Nitrous oxide	Air	Nitrogen	Carbon dioxide
Size D	Capacity in litres	400	940	375	370	940
	Pressure in psig (kPa)	1900 (13200)	745 (4400)	1900 (13200)	1900 (13200)	838 (5000)
Size E	Capacity in litres	660	1590	625	610	1590
	Pressure in psig (kPa)	1900 (13200)	745 (4400)	1900 (13200)	1900 (13200)	838 (5000)
Size H	Capacity in litres	6000	15800	6350	6000	
	Pressure in psig (kPa)	2200 (4400)	745 (4400)	2200 (4400)	2200 (4400)	

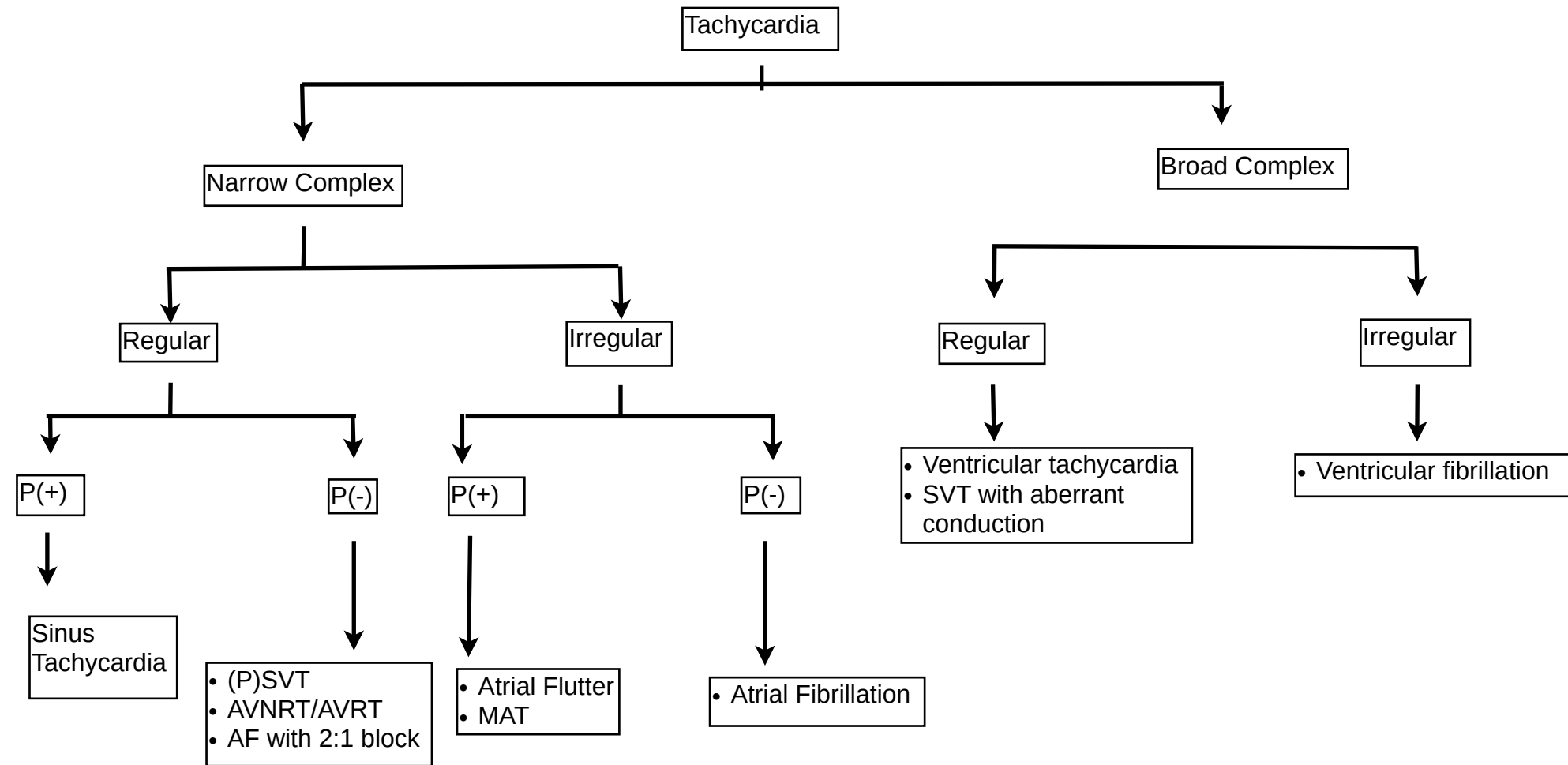
Gas	International	United states
Oxygen	Black body & white shoulders	Green
Nitrous oxide	Blue	Blue
Carbon dioxide	Grey	Grey
Air	Black body & shoulders with black & white quarters	Yellow
Helium	Brown	Brown
Entonox	Blue body & shoulders with blue & white quarters	
Cyflupropene	Orange	Orange

Branches of the Lumbosacral Plexus



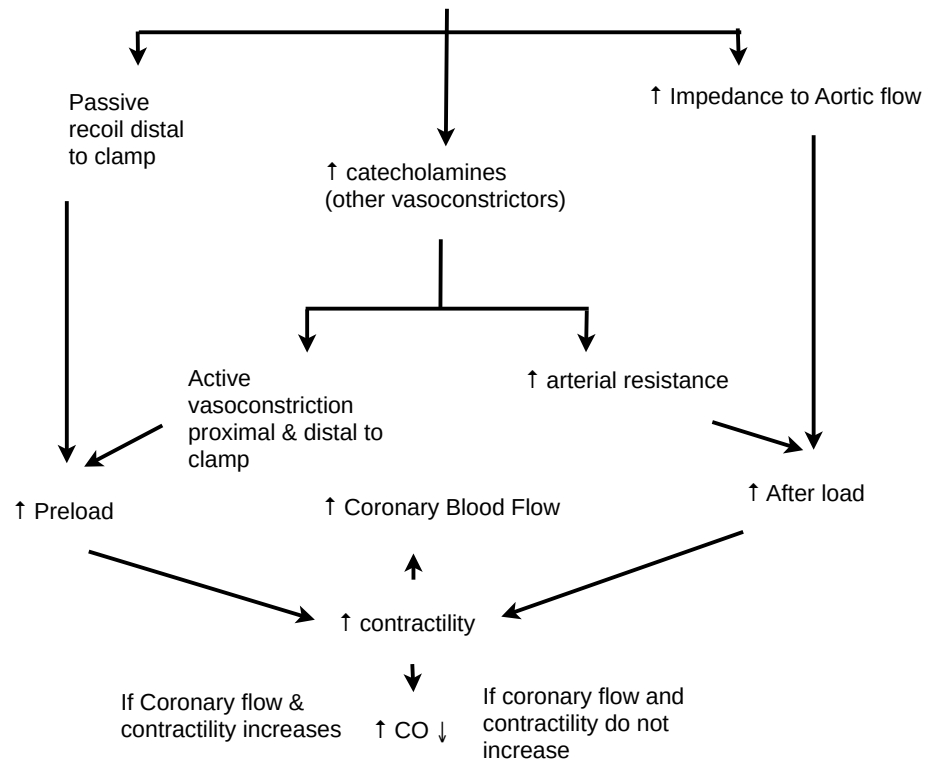
Indications	Drugs	Dose	Onset	Duration
For patient with good LV function ↑ HR CI in bronchospastic disease	<i>Esmolol IV</i>	<i>0.5mg/kg over /min</i> <i>50-300µg/kg/min</i>	<i>1min</i>	<i>12-20min</i>
	<i>Labetalol</i>	<i>5-20mg</i>	<i>1-2min</i>	<i>4-8hr</i>
	<i>Propanolol</i>	<i>1-3mg</i>	<i>1-2min</i>	<i>4-6hr</i>
Ca+2 channel blockers Myocardial ischemia ← reflex tachycardia	<i>Nicardipine</i>	<i>0.25-0.5mg</i>	<i>1-5min</i>	<i>3-4 hr</i>
	<i>Nifedipine (S/L)</i>	<i>10mg</i>	<i>5-10min</i>	<i>4h</i>
Mod to severe HTN – most rapid	<i>Nitroprusside</i>	<i>0.5-10µg/kg/min</i>	<i>30-60 sec</i>	<i>1-5min</i>
	<i>Nitroglycerine</i>	<i>0.5-10µg/kg/min</i>	<i>1min</i>	<i>3-5min</i>
Sustained control of BP ← delayed onset reflex tachycardia ACE Inhibitors	<i>Hydralazine</i>	<i>5-20mg</i>	<i>5-20min</i>	<i>4-8hr</i>
	<i>Phentolamine</i>	<i>1-mg</i>	<i>1-10min</i>	<i>20-40mm</i>
	<i>Enalaprilat</i>	<i>0.625-1mg</i>	<i>6-14min</i>	<i>4-6h</i>
	<i>Fenoldopam</i>	<i>0.1-0.6 µg/kg/min</i>	<i>5 min</i>	<i>5 min</i>

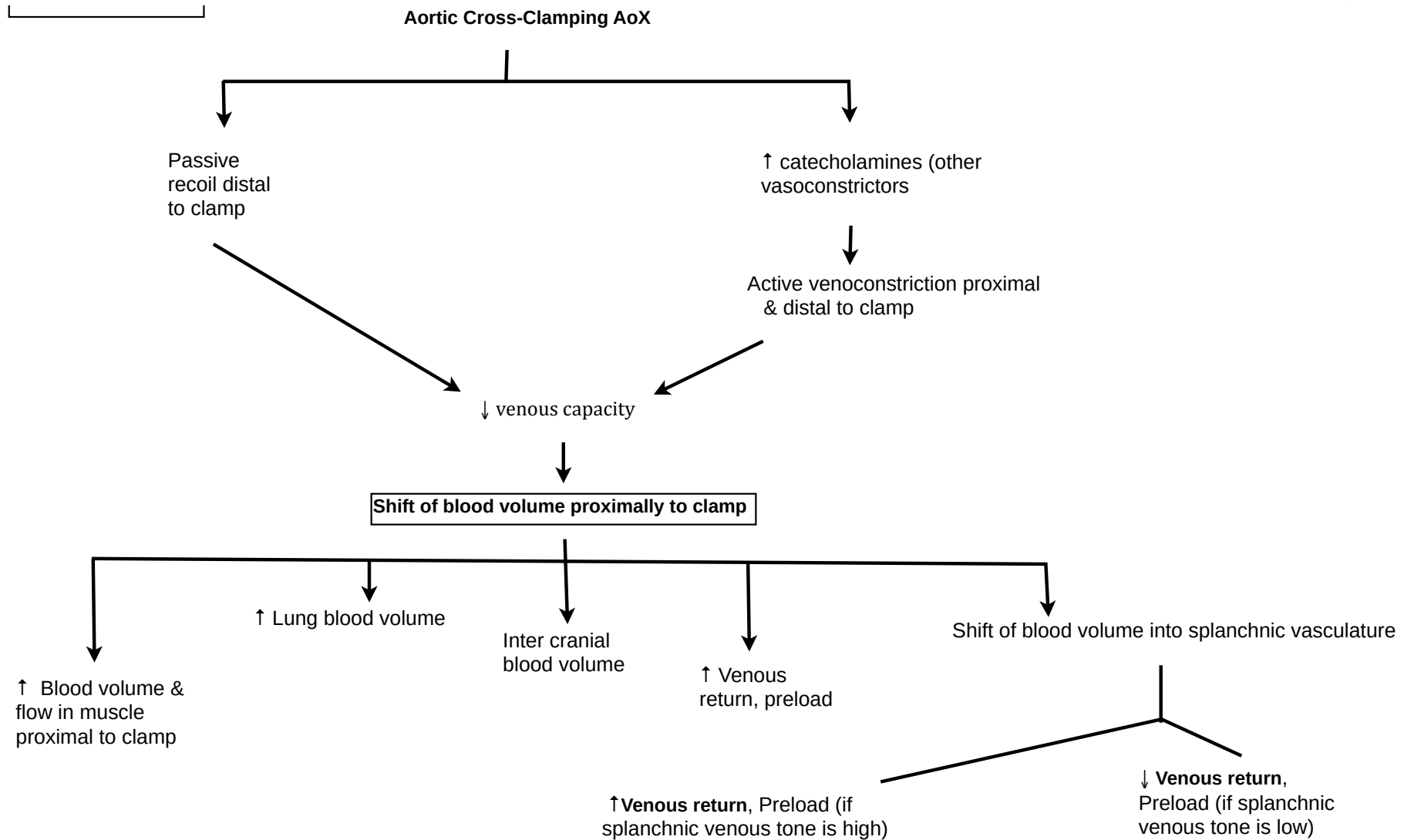


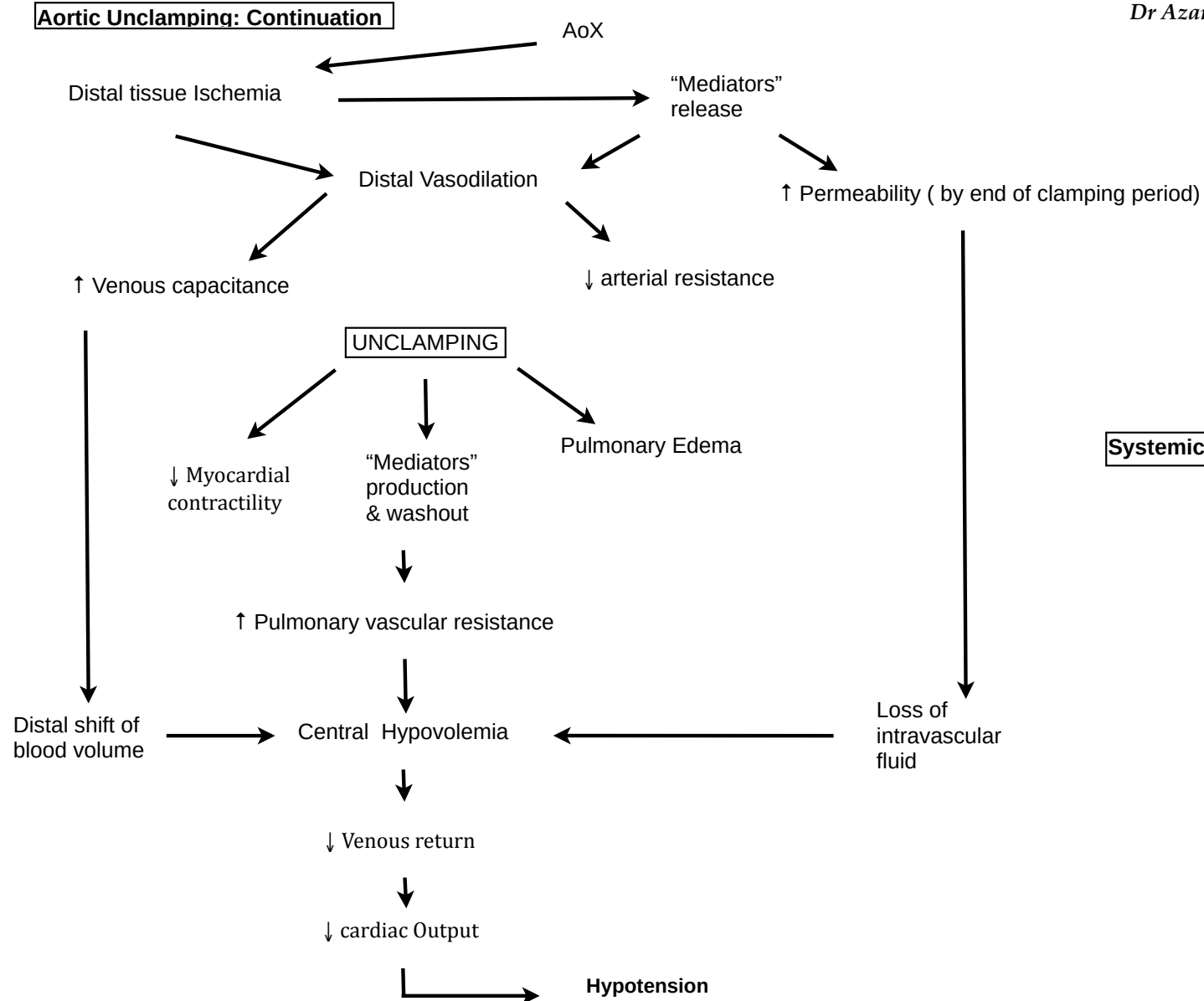


HEMODYNAMIC CHANGES:

Aortic Cross-Clamping AoX







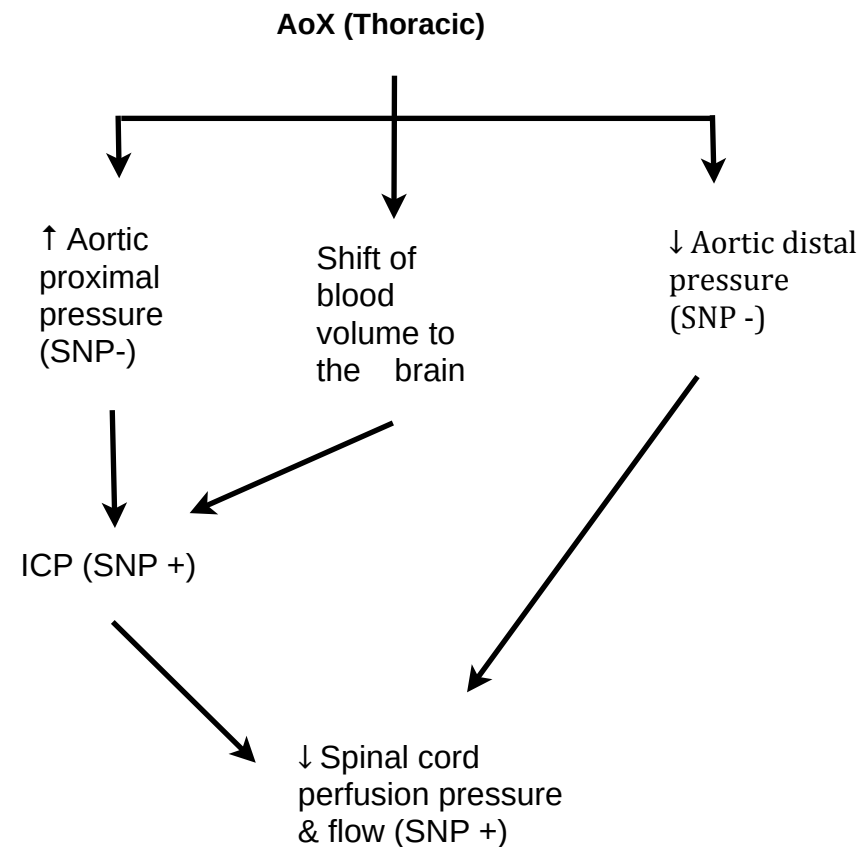
Surgical Factors:

Major	Intermediate	Minor
Emergency Surgery	Carotid endarterectomy	cataract
Major vessel/surgery	Intrathoracic/Peritoneal surgery	Superficial surgery
Surgeries with major blood loss or fluid shifts	Head & Neck Surgery	Breast surgery
	Open prostatic surgery	

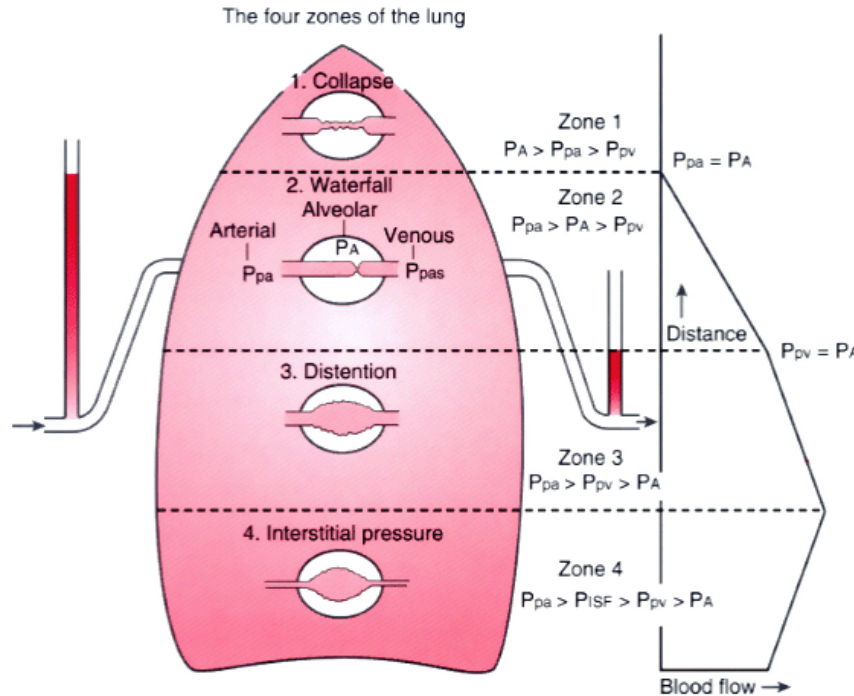
Reflex	Stimuli	Receptor	Afferents	Centre	Efferents	Response
Baroreceptor Reflex	Increase BP	Carotid Sinus & Aortic arch	IX Nerve(carotid Sinus) & X Nerve (Aortic Arch)	Nucleus of Tractus Solitarius in Medulla	IML Tract	Decrease in Blood pressure, Decreases Sympathetic stimulation, Heart Rate & SV
Chemoreceptor Reflex	pH pCO ₂ & pO ₂	Carotid & Aortic body	IX Nerve & X Nerve	Chemosensitive area of Medulla	X Nerve	Bradycardia, Increase Blood pressure & Hyperventilation
Bain Bridge Reflex	Increase filling of right atrium	Stretch receptor in right atrial wall	X Nerve	Dorsal motor nucleus of vagus	Vagal & sympathetic fibres	Increase in Heart Rate & Automaticity
Benzold Jarisch reflex	serotonin	Pain receptor C Fibre	X Nerve	Respiratory & Vasomotor Centre	X Nerve	Apnea, Hyperapnea & bradycardia, Coronary Vasodilation & Hypotension.
Oculo-cardiac Reflex	Pressure on the Eye, Traction on the muscles	Stretch receptor with in extraocular muscles.	Long & Short ciliary nerve & Ophthalmic division	Ciliary ganglion & Gasserian ganglion	Vagal & Cardiodepressor fibre	Reflex Bradycardia

Cardiac Reflexes:

Indications of IABP	Contraindications of IABP
1. Cardiogenic shock Myocardial infarction Myocarditis Cardiomyopathy Pharmacologic	1. Aortic valvular insufficiency
2. Failure to separate from CPB	2. Aortic disease Aortic dissection Aortic aneurysm
3. Stabilization of preoperative patient Ventricular septal defect Mitral regurgitation	3. Severe peripheral vascular disease
4. Stabilization of non-cardiac surgical preoperative patient	4. Severe non-cardiac systemic disease
5. Procedural support during coronary angiography	5. Massive trauma
6. Bridge to transplantation	6. DNR patients



Spinal cord perfusion pressure during thoracic aortic occlusion, with or without SNP



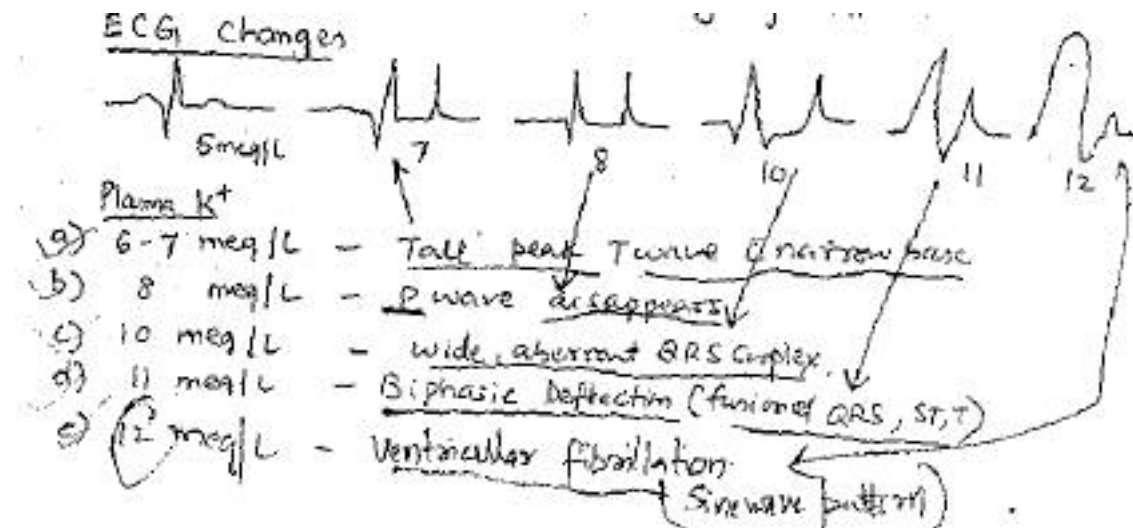
SLUDGE	DUMBELS
Salivation	Diarrhea
Lacrimation	Urinary incontinence
Urinary incontinence	Miosis, muscle fasciculations
Diarrhea	Bronchorrhea, bronchospasm, bradycardia
Gastrointestinal distress	Emesis
Emesis	Lacrimation
	Salivation

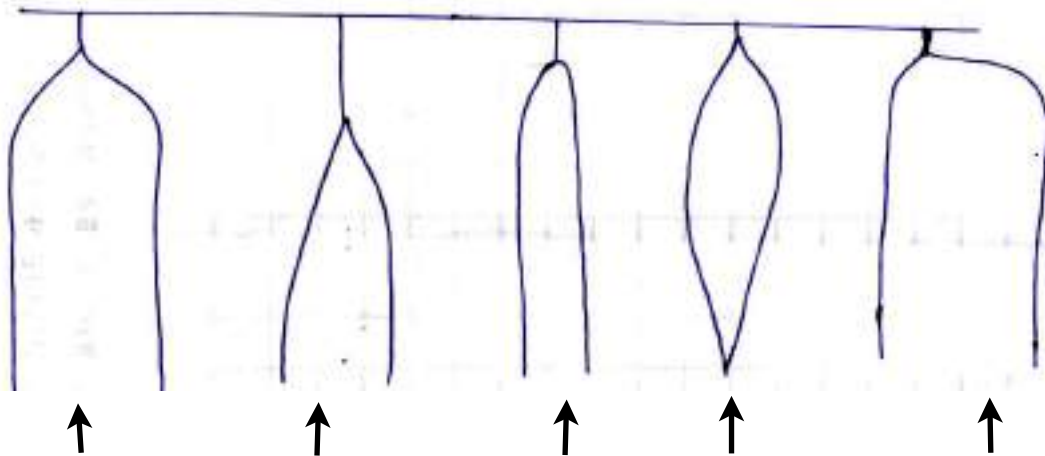
Carbon monoxide	Nicotine
200 times more affinity for HB than O ₂	15 to 50 microgram / ml in smokers
Carboxy Hb, Half life is 6 hours	1/2 life of one cigarette is 30min. (nicotine)
ODC - Left Shift Absolute decrease in O ₂ content	It stimulate sympatho adrenal axis.

Effects of smoking:

ECG changes due to Hyperkalemia:

ECG changes that occur with a raised K⁺ concentration are non-specific and may affect any part of the ECG



THROMBO ELASTOGRAPHY: Continuation:

Normal	Hemophilic	Thrombocytopenia	Fibrinolysis	Hypercoagulability
	1) Prolonged R	1) Prolonged R	1) ↓ MA	1) Shortened R
	2) ↓ α^0	2) ↓ α^0	2) ↓ α^0	2) ↑ MA
	3) ↓ MA	3) ↓ MA	3) ↓ F	4) Prolonged F

Relationship between BIS & patients Anesthetic status:

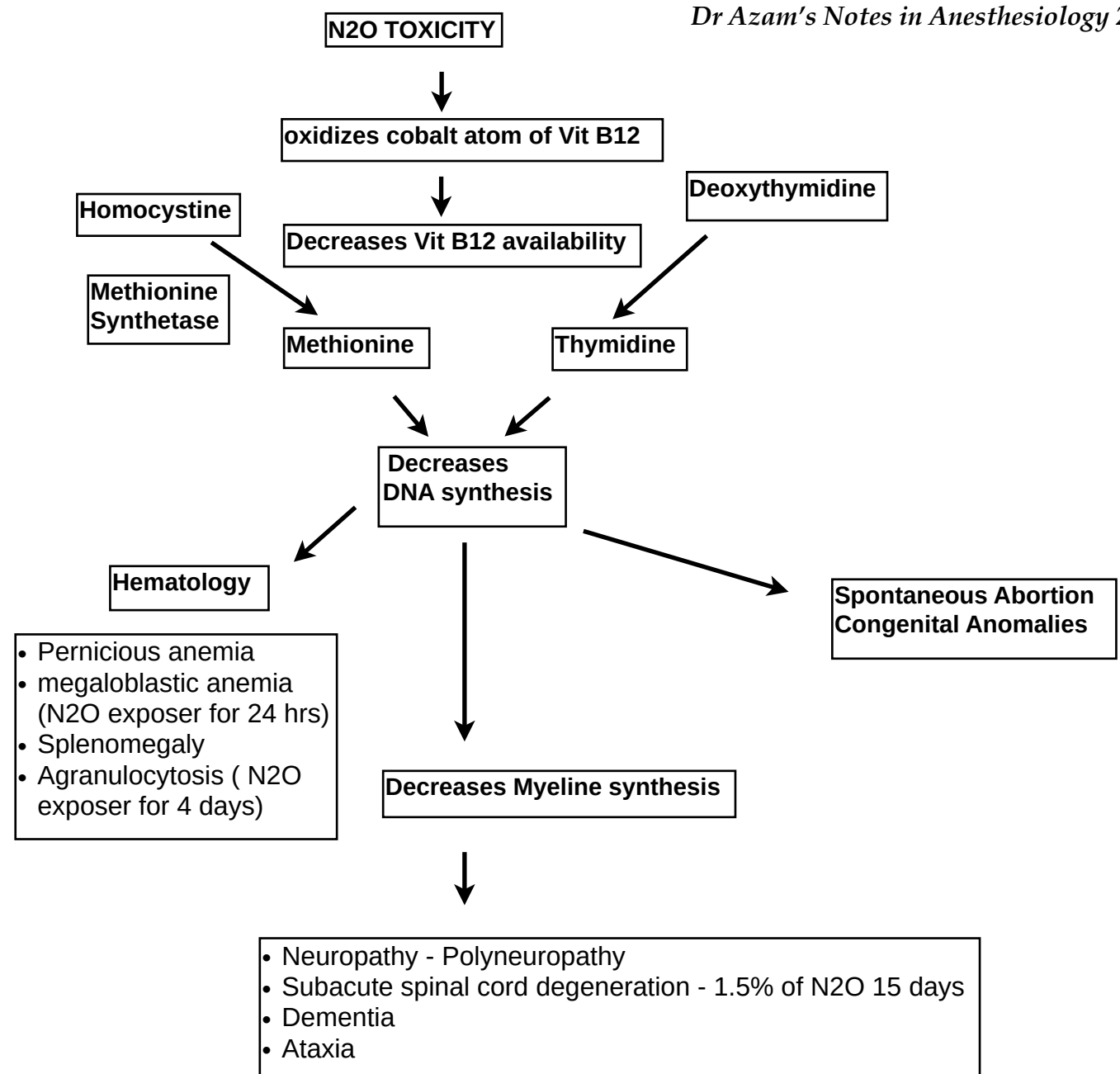
BIS Value	Patients Status	EEG
100	Awake & alert	High frequency activity
60	Moderate hypnotic levels	Low frequency activity
40	Deep Hypnotic level	Burst suppression begins delta activity
0	Profound anesthetic state	Isoelectric EEG

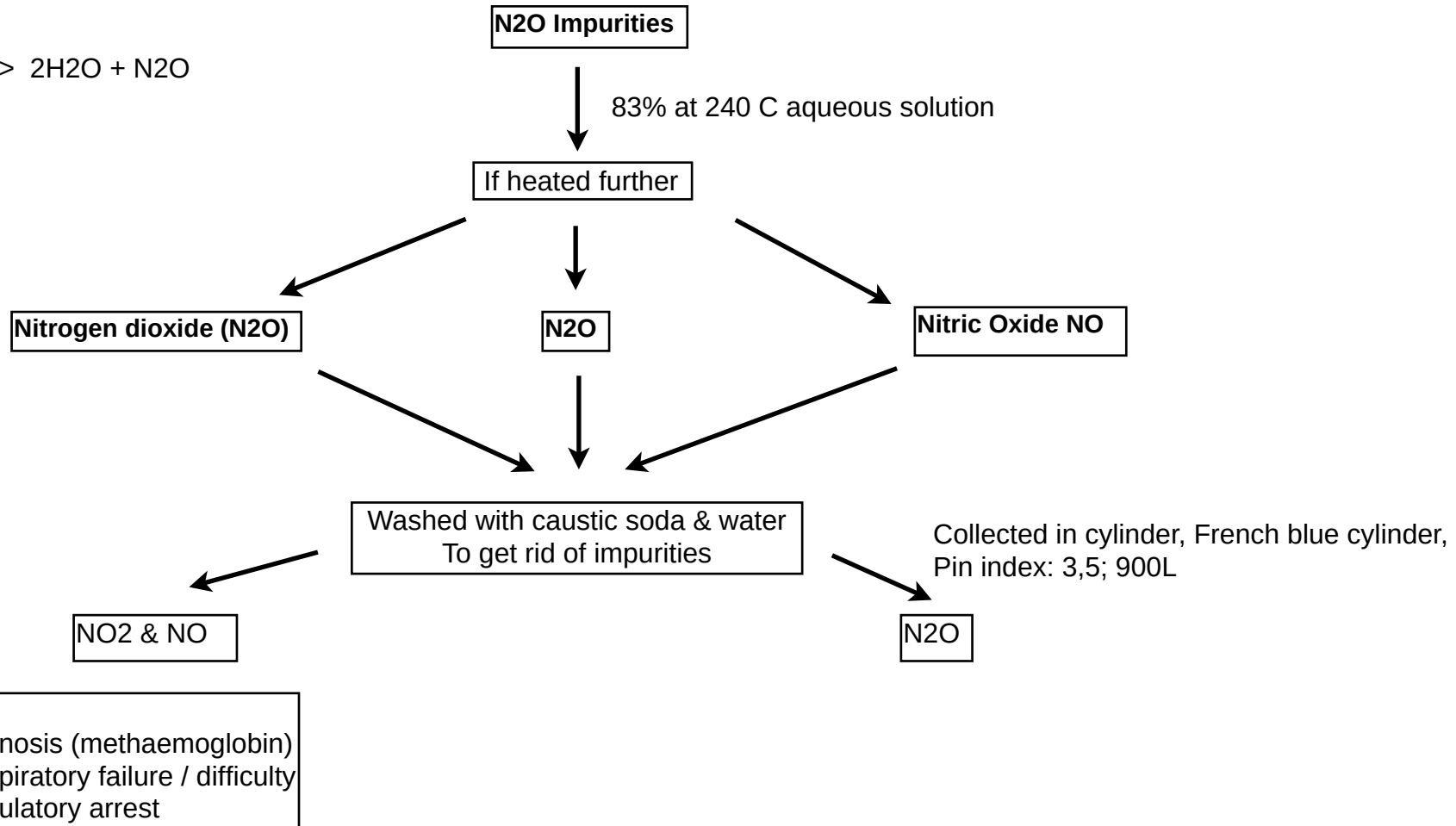
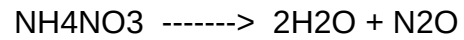
Magnesium Therapy:

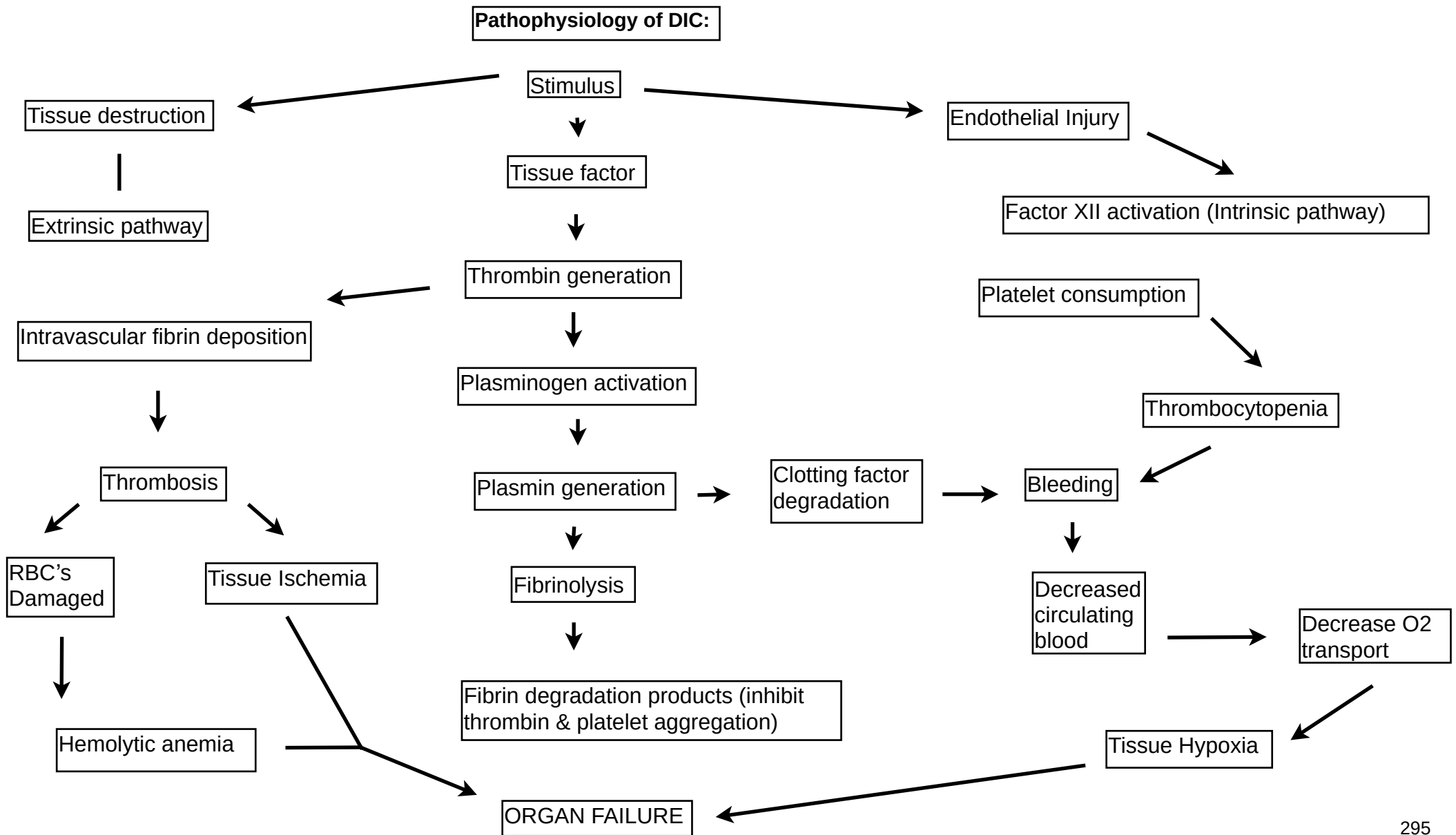
MgSO ₄ Regimens	Loading Dose	Maintainance
• Zuspan Regimens:	4 gm over 5 - 10 min IV	1 - 2 gm/hr IV
Pritchard	4 gm over 3- 5 min IV, 10 gm IM	5 gm IM every 4th hour in alternate buttock
Sibai	6 gm over 20 min IV	2 gm/hr IV

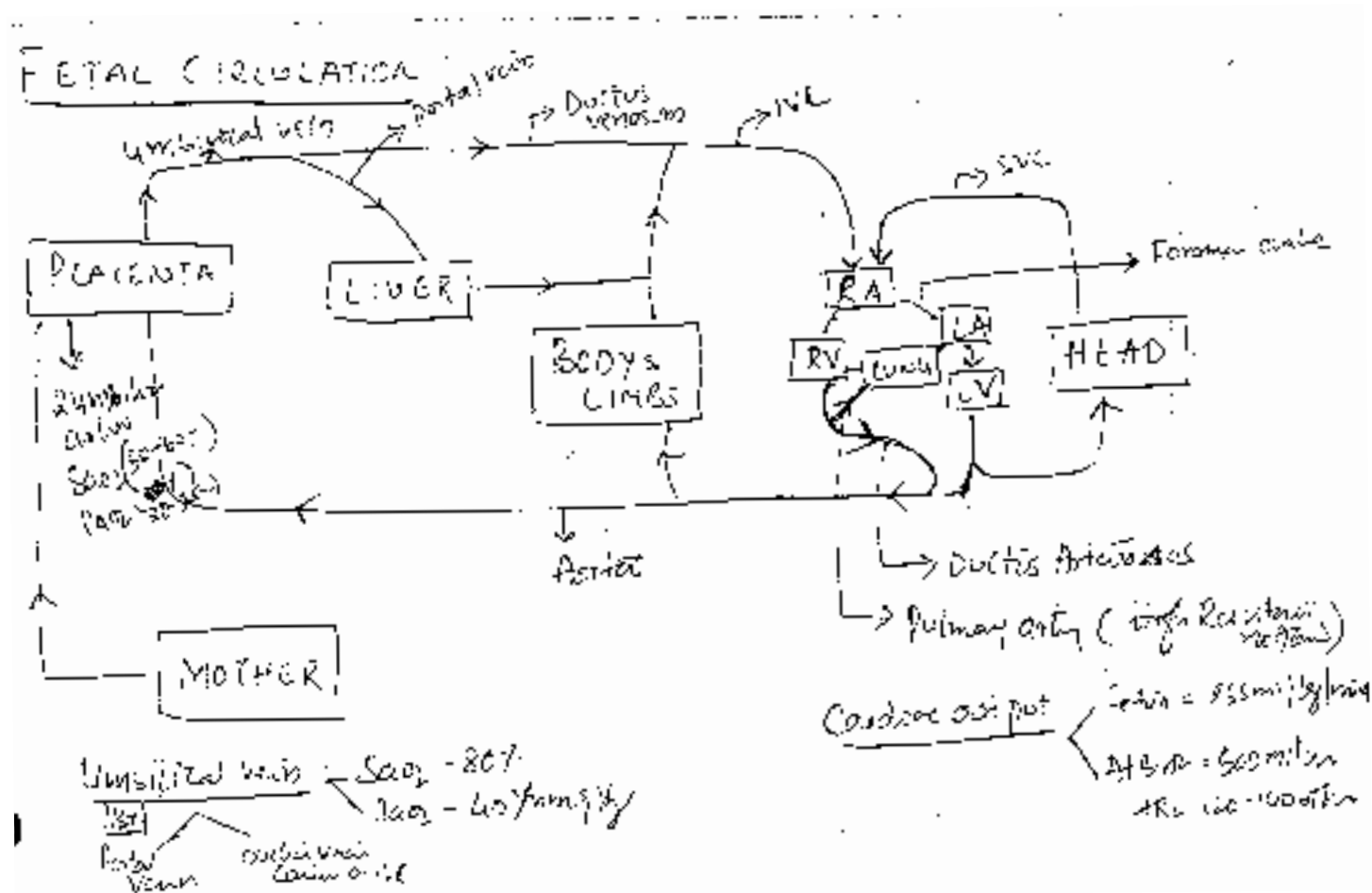
Effects of Increasing MgSO₄ levels:

Plasma Mg (mEq/lit)	Effects
1.5 - 2	Normal plasma level
4 - 8.0	Therapeutic range
5 - 10	ECG changes, (P-Q interval prolonged, QRS Complex widens)
10	Loss of deep tendon reflex
15	Sinoatrial & atrioventricular block
15	Respiratory arrest
25	Cardiac arrest

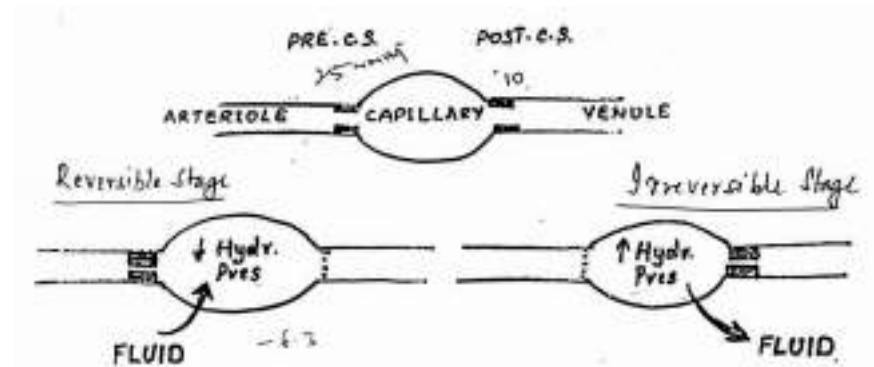
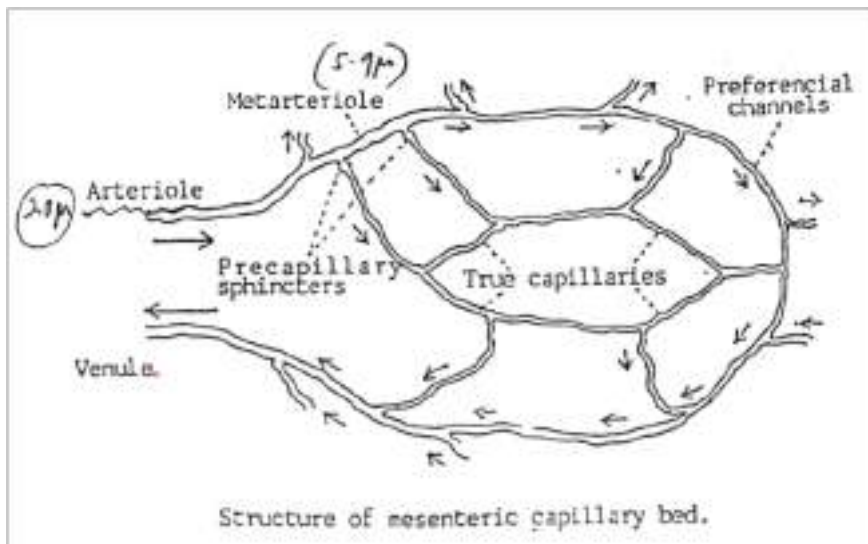








Meth-Hemoglobin Concentration %	Clinical sign & symptoms
10 to 20 %	central cyanosis of trunk & limbs usually asymptomatic
20 to 45%	CNS depression (headache, dizziness, fatigue, lethargy, syncope)
45 - 55%	Coma, arrhythmia, shock & convulsions
> 70%	High risk of Death



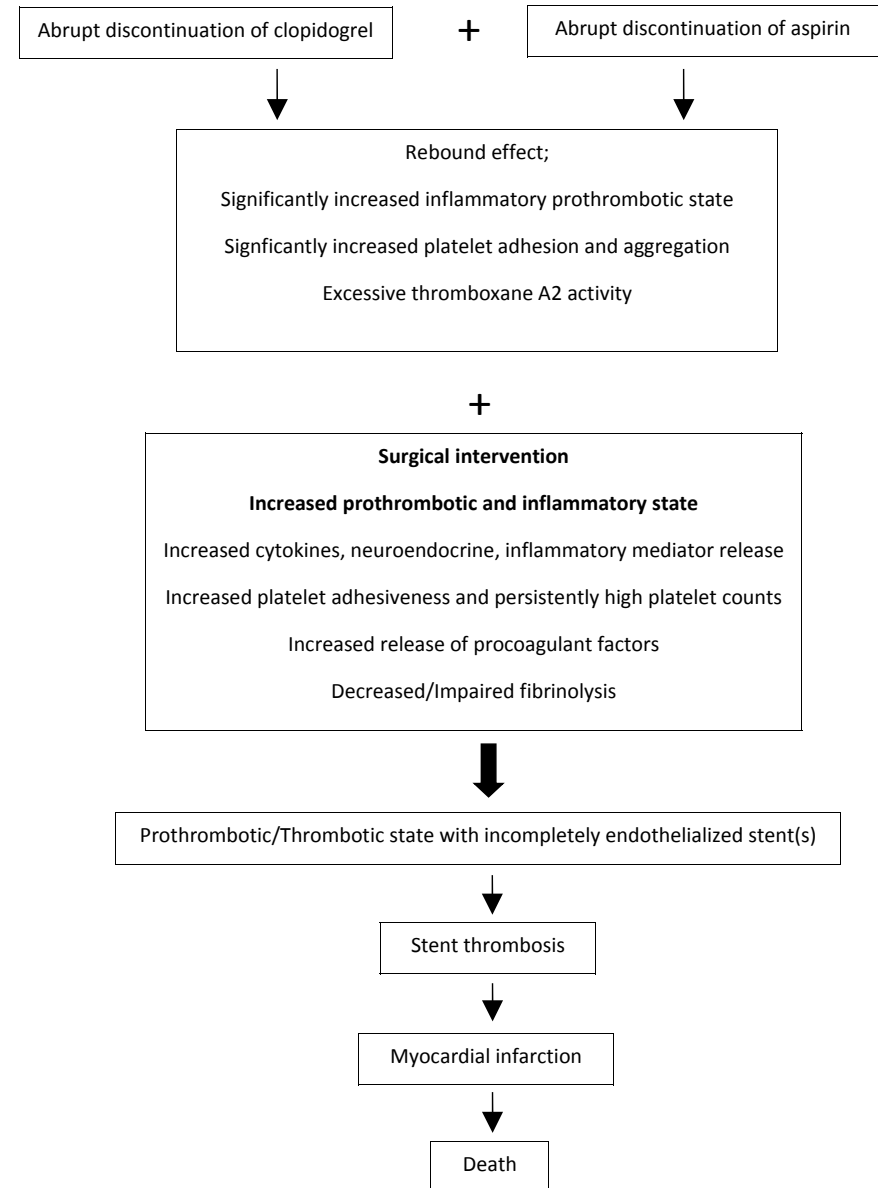
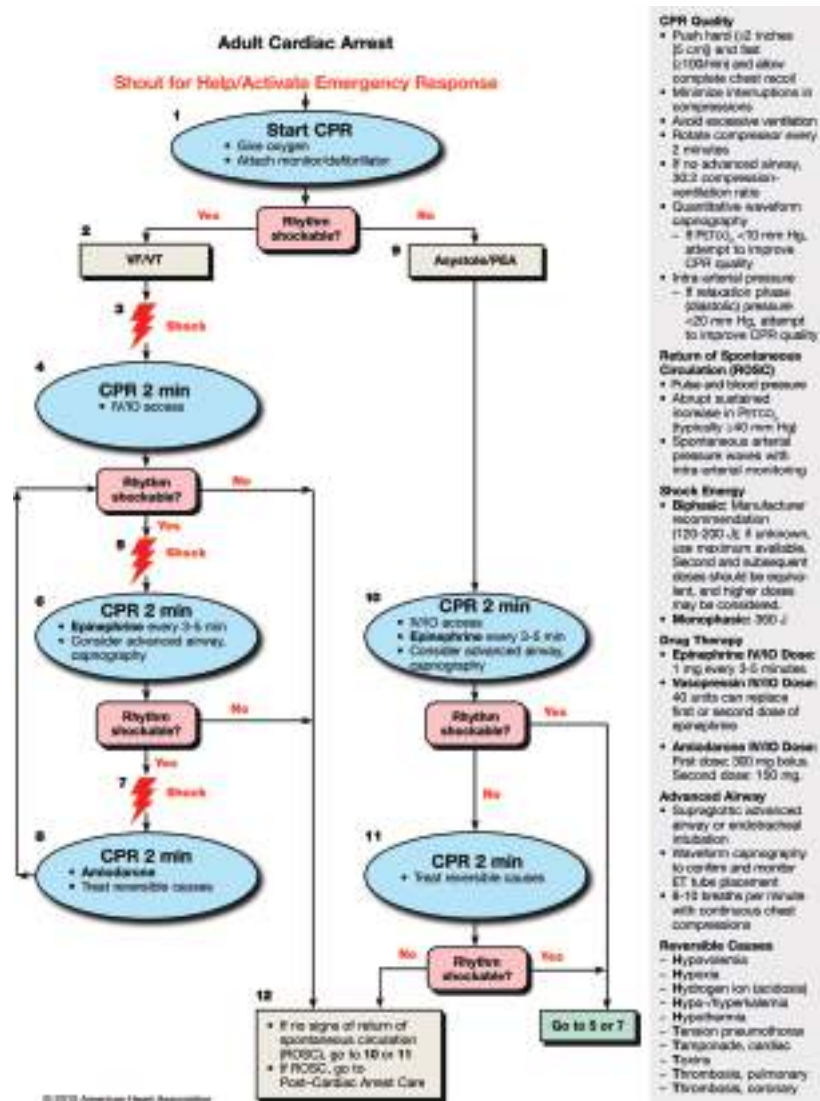


Fig.1. Diagram of the pathophysiology of acute perioperative stent thrombosis

Coronary Stents:**Bare Metal Stents (BMS)**

- BMS implantation effectively eliminates acute vessel closure.
- Stent thrombosis rates are reduced by dual antiplatelet therapy with clopidogrel & aspirin.
- Re-stenosis still remains high (20 to 25% within 6 months) re-stenosis occurs as a result of neointimal hyperplasia.

Therapy:

- Patients on BMS insertion dual antiplatelet should be continued for atleast 4 - 6 weeks with life long aspirin.

Drug Eluting Stents (DES)

- DES release drugs that inhibit neointimal hyperplasia.
- Re-stenosis rates are very low.
- Reason for DES thrombosis is - incomplete endothelialization of the stent.

Factors which increases risk of thrombosis after DES are:

- ACS
- DM
- Renal impairment
- Advanced age
- Ejection fraction less than 30 %
- Premature discontinuation of antiplatelet
- Resistance to antiplatelet therapy

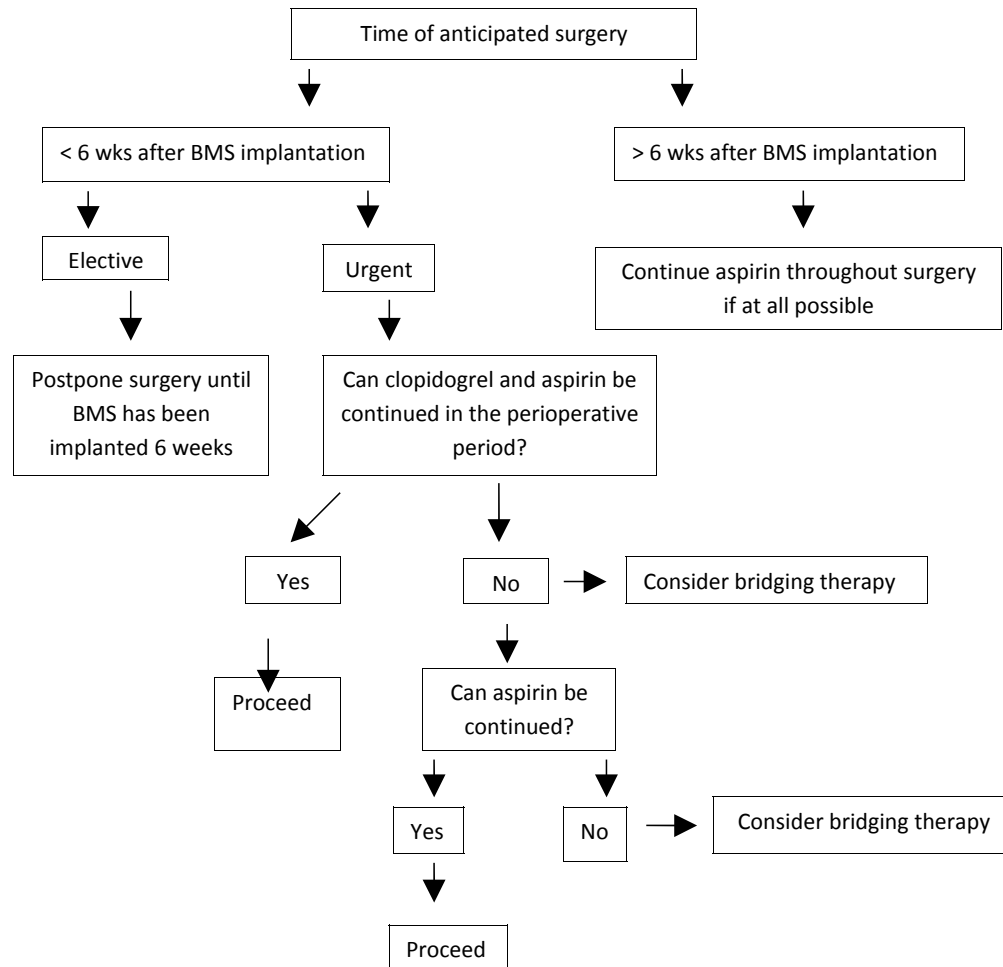
Therapy:

- After DES implantation 12 months dual antiplatelet therapy with clopidogrel & aspirin and life long aspirin therapy

Stent thrombosis is classified into:

- Acute (within 24 hours)
- Sub acute (24 - 30 days)
- Late (30 - 1 year)
- Very late (> 1 year)

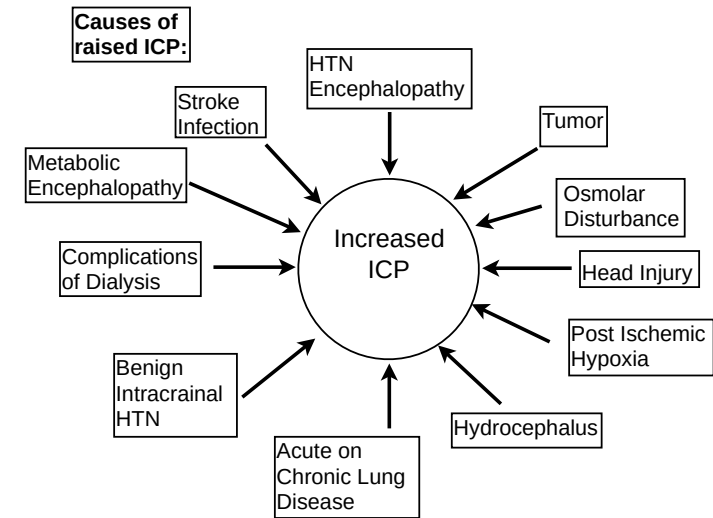
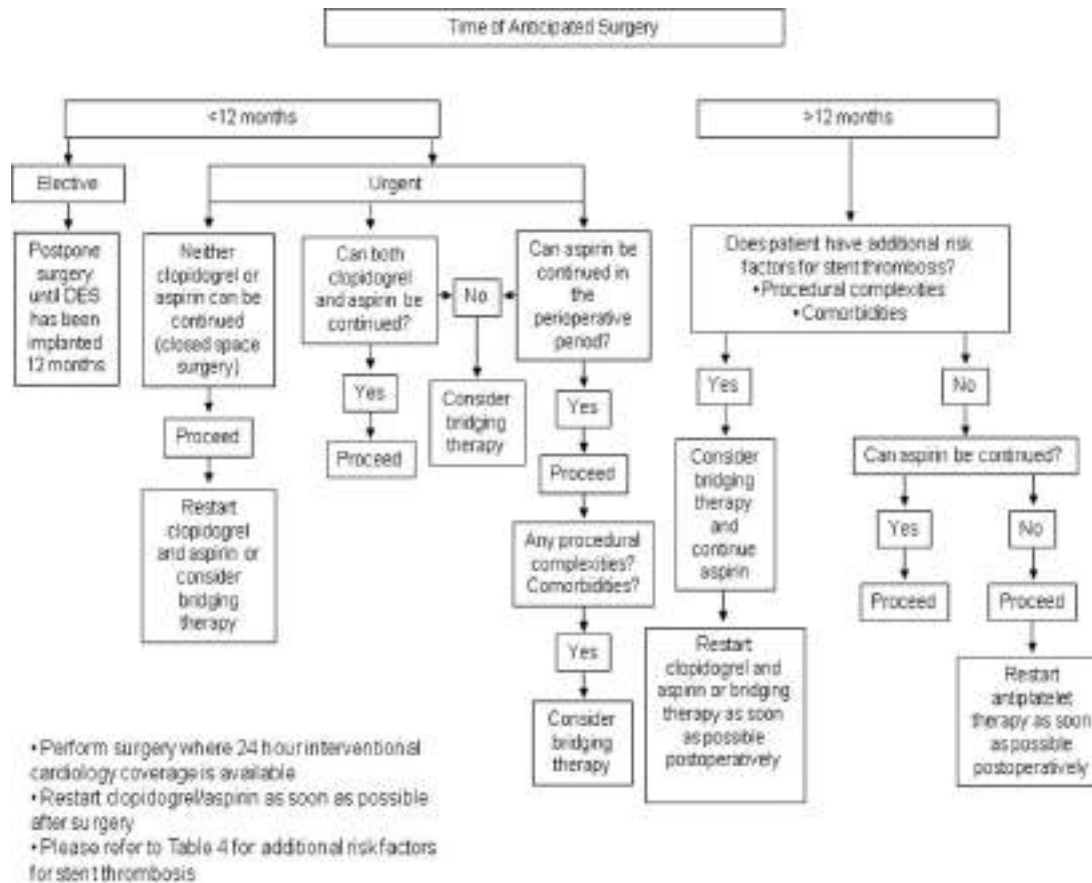
Fig.2. Algorithm for perioperative management of patients with bare metal stents



LMA is available in various sizes and depends on the body weight of the patient.

Appropriate LMA size	Body Weight (kg)	Air volume (ml)
1	Upto 5	4
1.5	5-10	7
2	10-20	10
2.5	20-30	14
3	30-35	20
4	35-50	30
5	50-100	40
6	>100	50

Fig.3. Algorithm for perioperative management of patients with drug eluting stents



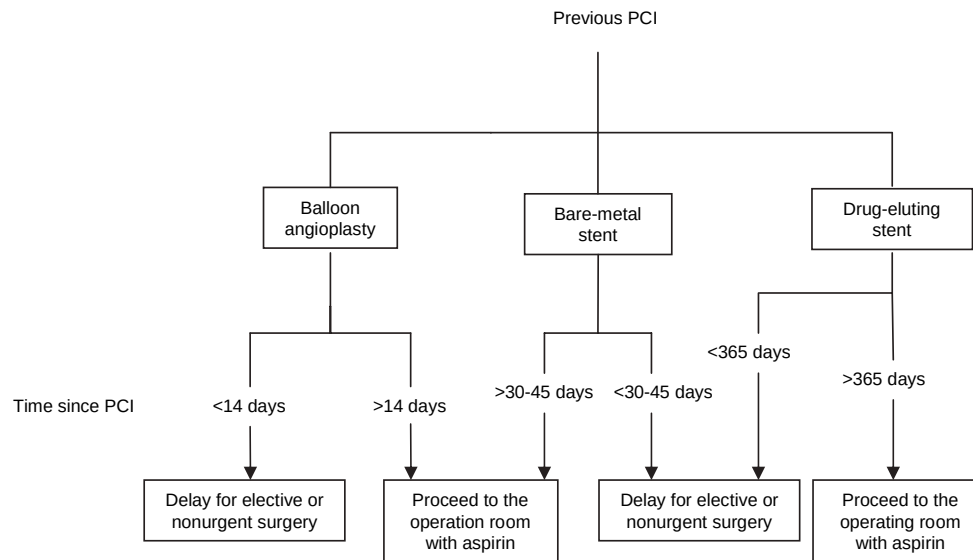
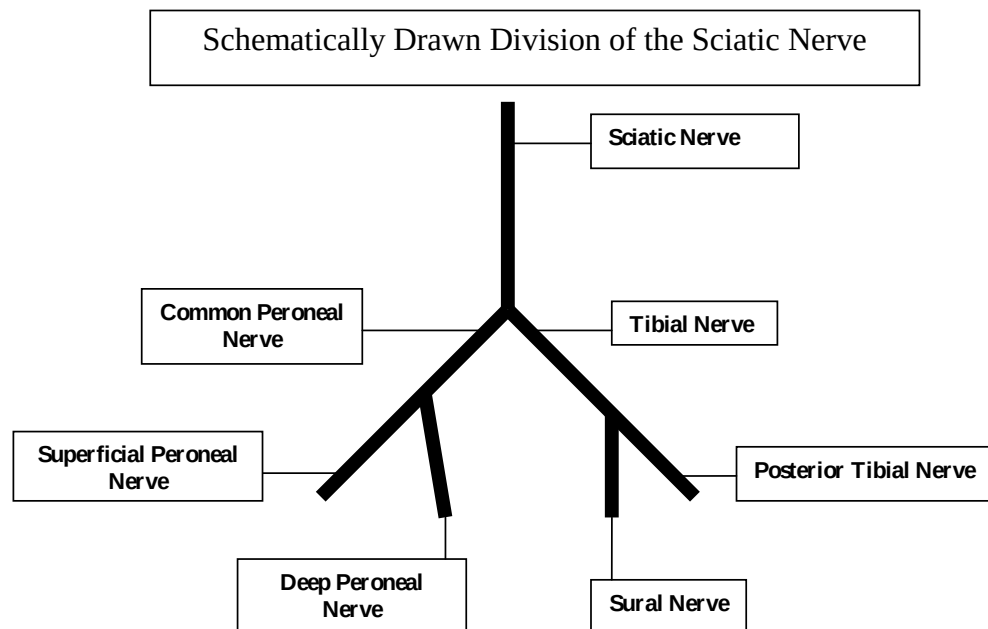
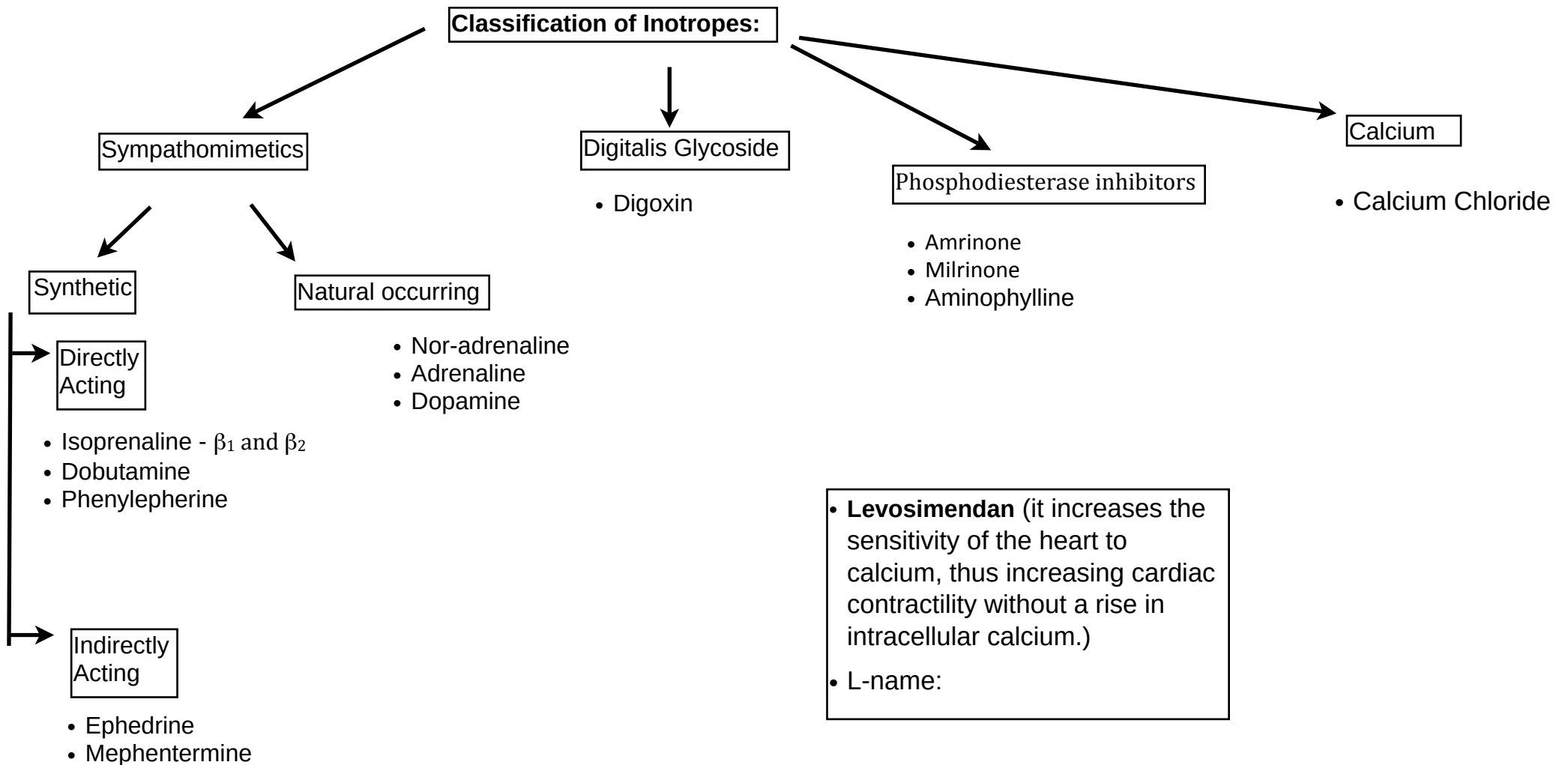


Figure 2. Proposed approach to the management of patients with previous percutaneous coronary intervention (PCI) who require non-cardiac surgery, based on expert opinion.



Flow Charts & Diagrams



Dopamine	Isoprenaline	Dobutamine
1) Naturally occurring Catecholamine	Synthetic	Synthetic derivative of isoprenaline
2) Site of action - α_1 , β_1 and dopaminergic (DA_1 DA_2) receptors ($D > B > \alpha$)	Only β_1 β_2 stimulant ($\beta_1 > \beta_2$)	β_1 β_2 , α_1 ($\beta_1 \gg \beta_2$)
3) CVS effects: can cause both relaxation and constriction of vascular smooth muscle Dose dependent Causes vasodilatation in renal, mesenteric and coronaries Increases contractility SV, CO (inotropic) In high doses stimulates α $\rightarrow$ increase PVR $\rightarrow$ $\downarrow$ RBF (hence raises BP)	CVS effects: Stimulates β_1 receptors increasing H.R, automaticity and contractility $\rightarrow$ $\uparrow$ SBP Stimulates also β_2 receptors hence vasodilation in skeletal muscle ($\downarrow$ PVR), also renal and mesenteric vasodilatation $\rightarrow$ $\downarrow$ DBP. Therefore $\downarrow$ MAP. Hence (was) used in complete heart block; cardiac failure with valvular disease who benefit with $\uparrow$ HR and $\downarrow$ venous capacitance filling	CVS effects Stimulates $\beta_1 \rightarrow \uparrow$ myocardial contractility without effect on PVR and H.R. i.e. inotropic > chronotropic β_2 and receptors are only slightly stimulated hence mild vasodilatation effect
4) Renal effects	RS effects:	Renal effects
Improves blood flow to renals by vasodialator effects on kidneys and hence increases RBF, GFR and increases Na excretion.	β_2 stimulation Marked bronchodilation	Does not stimulate renal dopaminergic receptors. Hence no renal vasodilation

Dopamine	Isoprenaline	Dobutamine
B) 2-10 µg/kg/ml cause β_1 stimulation $\rightarrow$ $\uparrow$ cardiac contractility and C.O. i.e. at doses > 5 µg/kg/ml stimulates $\uparrow$ release of endogenous NE.	Hence useful in bronchial asthma and drug induced bronchoconstriction (aerosol form) bronchoconstriction due to pulmonary embolism	Unlike dopamine it does not release endogenous NE.
C) 10-20 µg/kg/ml α and β_1 stimulated, but α vasoconstriction effect predominates (renal perfusion may be lost)		
Dosage	Dosage	Dosage
short-half life hence no loading dose only infusion Renal dose $\rightarrow$ 2-5 µg/kg/ml Cardiac $\rightarrow$ 5-10 µg/kg/ml Vasopressor $\rightarrow$ 10-20 µg/kg/ml	Infusion µg/kg/ml (1mg in 250ml of fluid gives soln of 4 µg/ml)	2-10 µg/kg/ml
Uses	Uses	Uses
1) Initial agent in treatment of shock particularly in vasodilated state like septic shock and hypovolema	1) To treatment bradycardia or heart block resistant to atropine	1) Treatment in low cardiac output failure
2) To protect kidney and aid diuresis in oliguric patients (renal transplant)	2) To treatment cardiac failures especially with valvular heart disease	2) Hypotension 2 to $\downarrow$ M.contractility

Dopamine	Isoprenaline	Dobutamine
3) In refractive cardiac failure	3) To treatment bronchoconstriction - replaced now by other newer drugs	3) Emergency from CP bypass 4) SVR is little effected and filling pressures Dopamine + Dobutamine is used to limit increase in LV filling pressure induced by dopamine alone.
Side effects	Side effects	Side effects
Before administration ensure patients has adequate B.P and treatment hypovolemia Nausea, vomiting, Tachy, arrhythmia, dyspnoea, angina, headache, vasoconstriction → ↑ SBP Extravasation causes sloughing and necrosis in tissues, so better given via central line.	Tachycardia Ventricular arrhythmia Hypotension Isoprenaline used in aerosols to treatment B. asthma caused increased mortality due to 1) ↑ stimulation of β_1 race → V.arrth 2) Hypoxia due to V/Q mismatch inspite of bronchodilation 3) Toxicity of fluorinated hypo carbons used in propellants	Nausea, headache, angina, palpitation, arrhythmia, dyspnoea Tachycardia and V.dysrhythmia are less common than popamine
Contraindication: Phaeochromocytoma Arrhythmias		

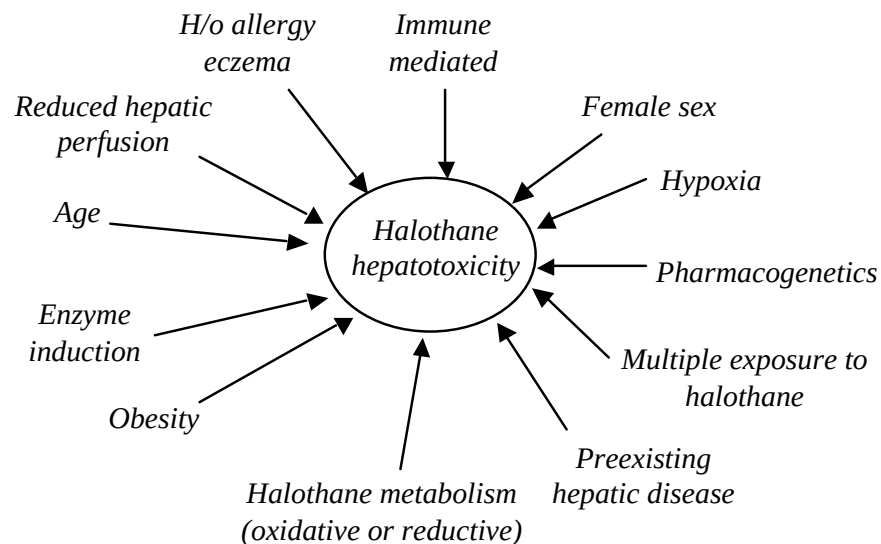
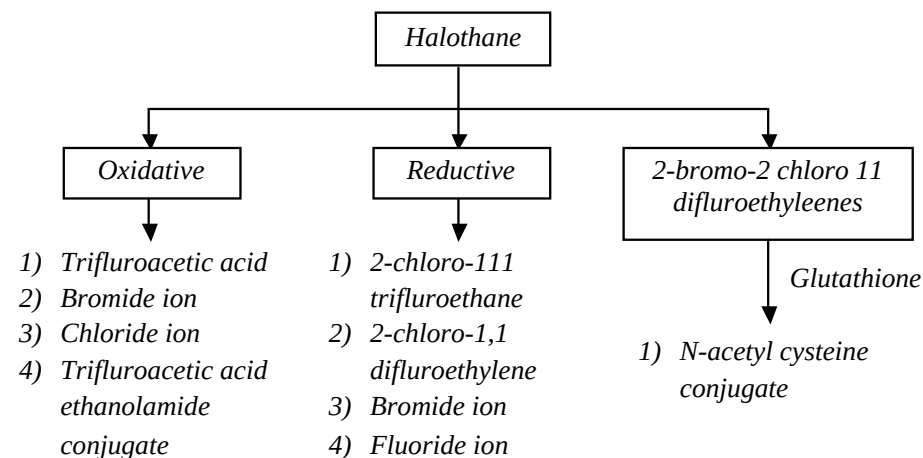
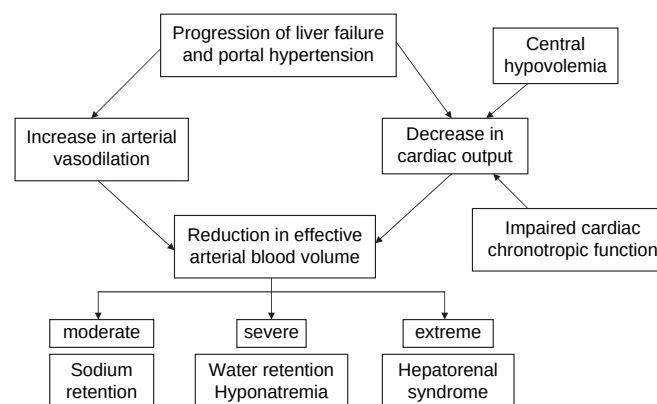
Factors affecting halothane hepatotoxicity:**Pathway for halothane biotransformation:****Pathophysiology of Hepatorenal Syndrome:**

Fig. 2. Pathophysiology of the hepatorenal syndrome. (From Arroyo V, Terra C, Ginès P. New treatments of hepatorenal syndrome. Semin Liver Dis 2006;26:254–64; with permission.)

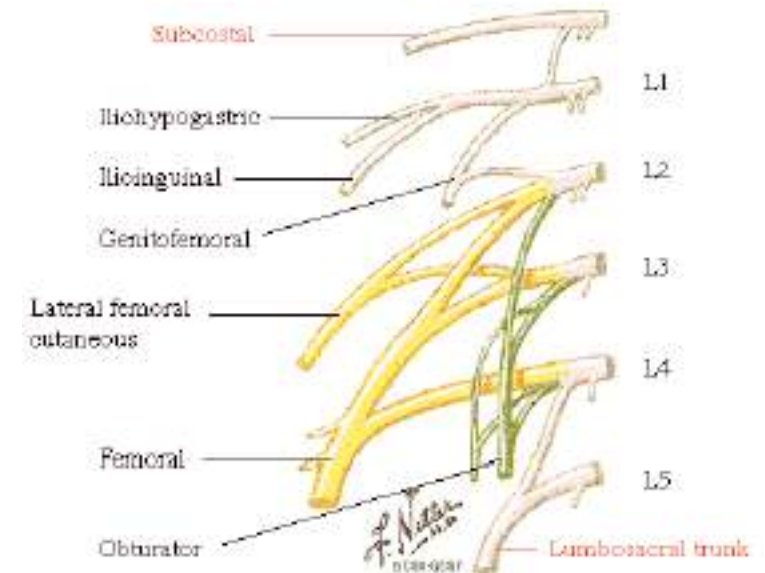
Use of Blood Substitutes: Two types of blood substitutes are available:

Perfluorocarbons:

- They are biologically inert volatile fluids with high dissolving capacity for O₂ & CO₂
- They transport & deliver O₂ by simple physical dissolution
- Not water soluble
- their O₂ carrying capacity depends on the concentration of perfluorocarbons which varies between products.
 - H⁺ atoms are replaced by fluorines
 - One unit of perfluorocarbons carry 3 times O₂ carried by similar volume of blood
 - Fluosol - DA: 20 % emulsion of 2 different compounds. Dose = 20ml/kg
 - Polyfluro-octobromide: O₂ solubility is more

Hb based O₂ carriers:

- These are based on natural or recombinant human Hb or bovine Hb
- produced by purification, encapsulation in synthetic phospholipid.
- Side effect:
 - Increased Blood pressure due to increased systemic vasoconstriction due to scavenging of nitric oxide & oxidation which generated Meth Hb.
- Short intravascular half life
- hence used in emergency situation.



Age	Milk & Solids	Liquid
< 6 months	4 hours	2 hours
6 - 36 months	6 hours	3 hours
> 36 months	8 hours	3 hours

Pin Index Cylinders

- 1 - 3 = **Ethylene**
- 1 - 4 = **Nitrogen**
- 1 - 5 = **Air**
- 1 - 6 = **O₂ + CO₂ (CO₂ > 7.5%)**
- 2 - 4 = **O₂ + He (He < 80.5%)**
- 2 - 5 = **O₂**
- 2 - 6 = **O₂ + CO₂ (CO₂ < 7.5%)**
- 3 - 5 = **N₂O**
- 3 - 6 = **Cyclopropane**
- 4 - 6 = **O₂ + He (He > 80.5%)**
- 7 = **Entonox**

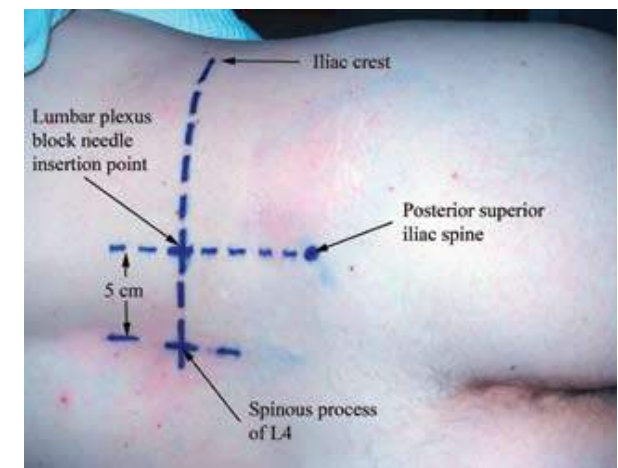
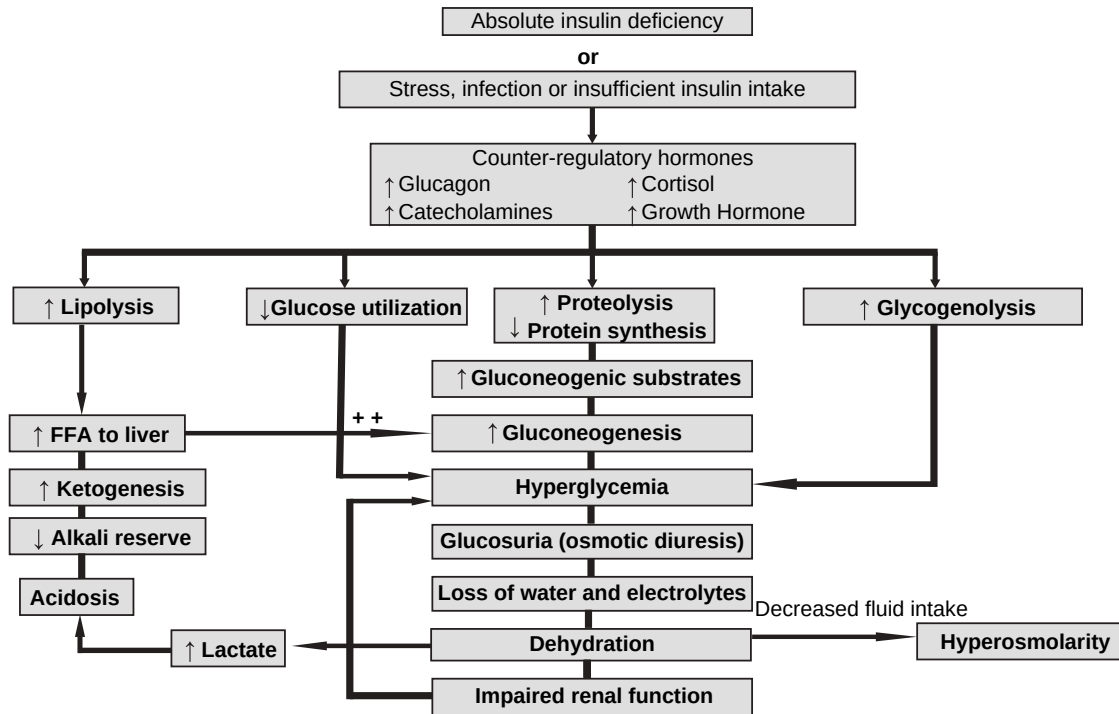


Figure 14-4. Lumbar plexus block landmarks

Diabetic Ketoacidosis: Continuation

Pathophysiology of Diabetic Ketoacidosis (DKA)

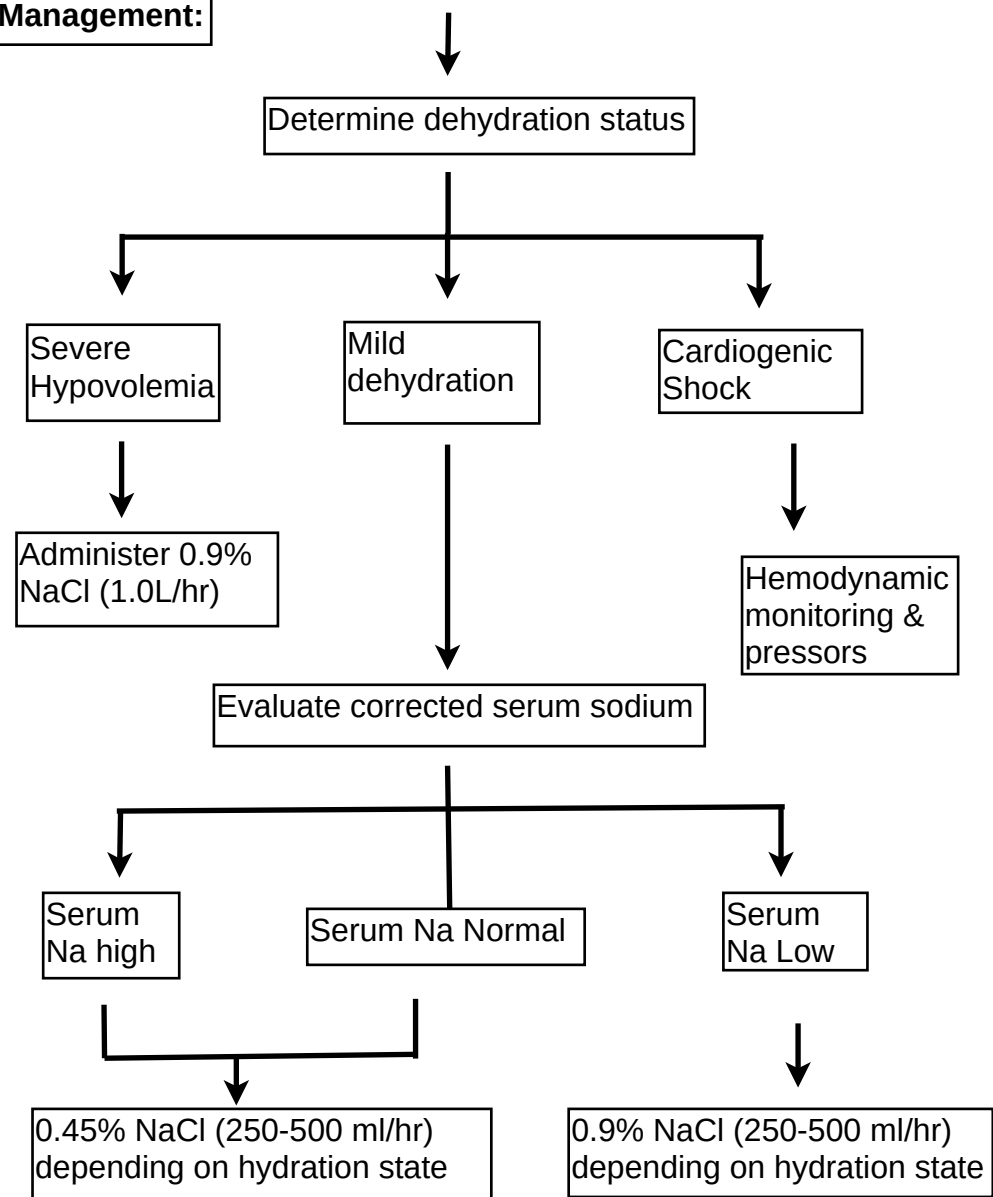


Check electrolytes, BUN, venous pH, creatinine & glucose every 2 - 4 hrly until stable. After resolution of DKA & when patient is able to eat, Initiate SC multi-dose insulin regimen. continue IV infusion for 1-2 hrs after SC insulin to ensure adequate plasma insulin levels. Look for precipitating cause.

When Serum glucose reaches 200 mg/dl, change to 5 % Dextrose with 0.45% NaCl at 150 - 250 ml/hr

Management:

I. Dehydration: Fluids



Diabetic Ketoacidosis: Continuation

III. Electrolytes - Potassium

Establish adequate renal function & urine output (at least 50ml/hr)

If serum K+ is < 3.3 mEq/L, hold insulin & give 40 mEq, K+ per hour (2/3 KCL & 1/3 KPO4) until K > 3.3 mEq/L

If serum K+ > 5.5 mEq/L, but check K+ every 2 hour.

If serum K+ > 3.3 but < 5.5 mEq/L, give 20 - 30 mEq/L in each liter of IV fluid (2/3 KCL & 1/3 KPO4) to keep serum K+ at 4-5 mEq/L

Check electrolytes, BUN, venous pH, creatinine & glucose every 2 - 4 hrly until stable. After resolution of DKA & when patient is able to eat, Initiate SC multi-dose insulin regimen. continue IV infusion for 1-2 hrs after SC insulin to ensure adequate plasma insulin levels. Look for precipitating cause.

II. Lack of Insulin - Insulin

IV Route

Insulin: Regular
0.1 U/Kg B.wt as
IV bolus

0.1U/kg/hr
continuous
insulin infusion

Uncomplicated
DKA - SC route

Rapid-acting Insulin:
0.3 U/Kg then 0.2 U/Kg
one hr later

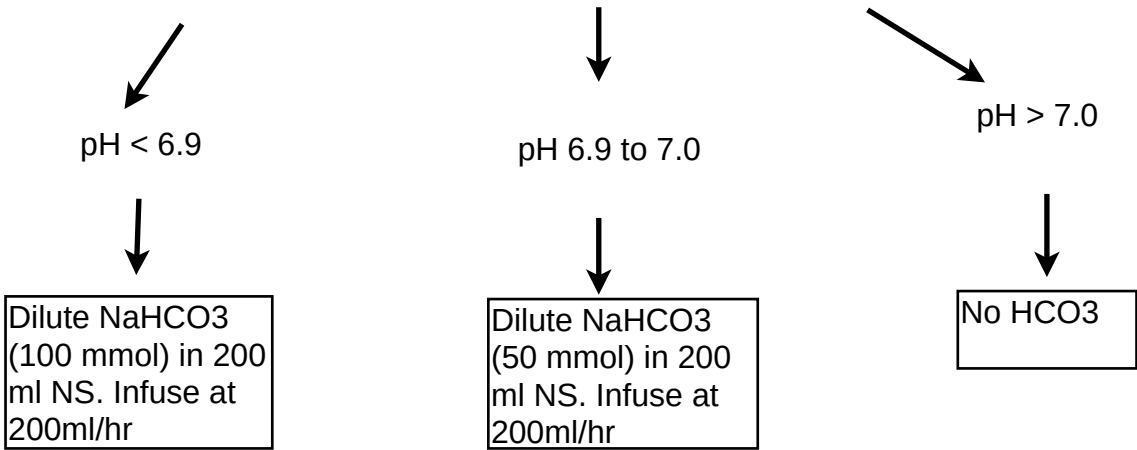
Rapid-acting
Insulin: 0.2 U/kg
every 2 hours

If serum glucose does not fall by
50 - 70 mg/dl in first hour, double
IV or SC insulin bolus

When serum glucose reaches
200mg/dl, reduce regular insulin
infusion to 0.05 - 0.1 u/kg/hr IV, or
give rapid-acting insulin at 0.1U/kg
SC every two hrs. Keep serum
glucose between 150-200 mg/dl until
resolution of DKA

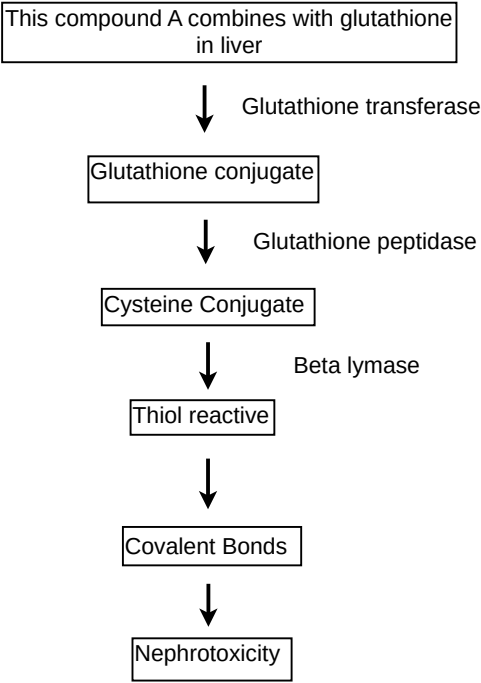
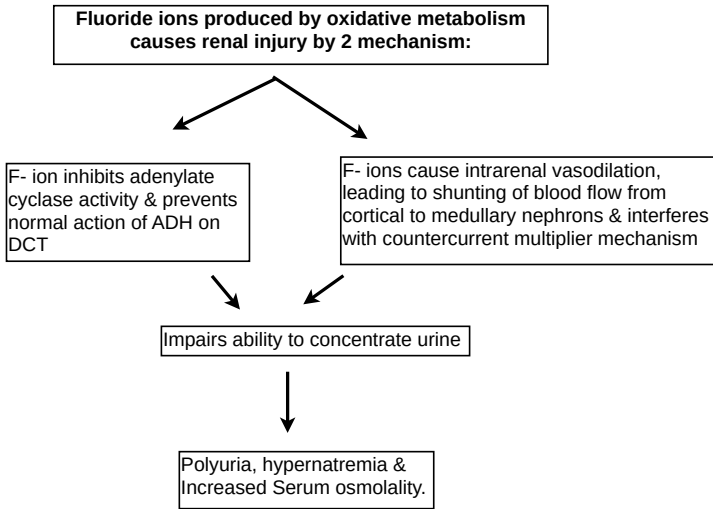
When Serum glucose reaches 200
mg/dl, change to 5 % Dextrose with
0.45% NaCl at 150 - 250 ml/hr

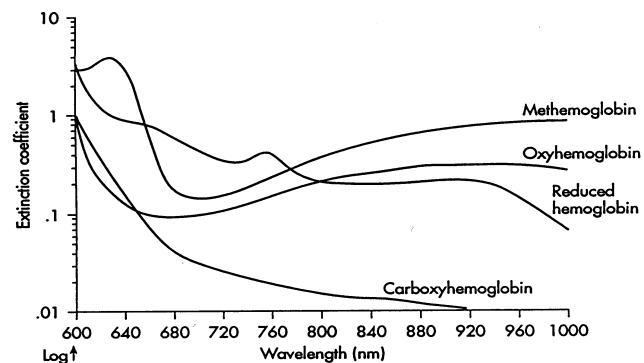
IV. Acidosis - Assess the need of Bicarbonate



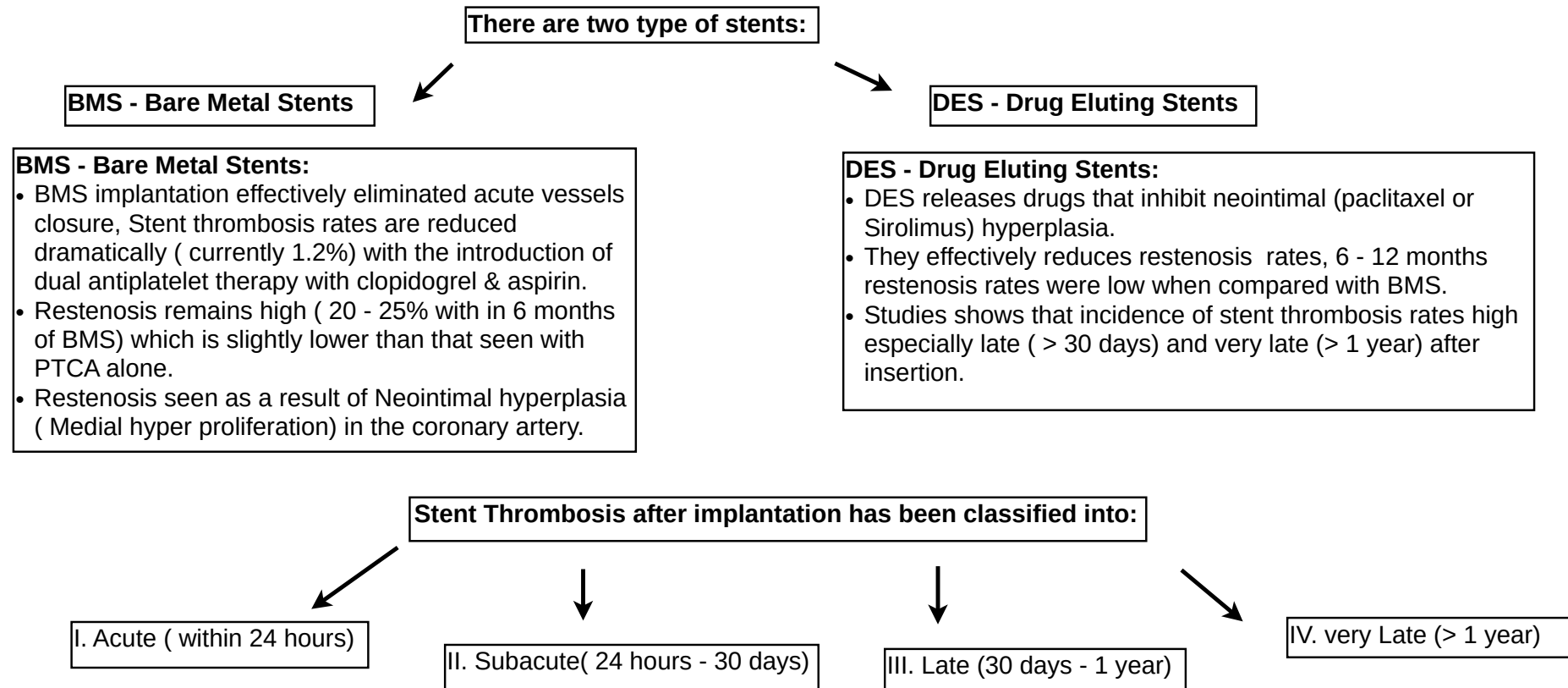
- Normal Fluoride level is 50 - 60 microm/liter
- 50 - 80 microm/ltr - Mild renal injury
 - 80 - 120 microm/ltr - Moderate renal injury
 - > 120 microm/ltr - Severe injury

Fluoride toxicity in Renal failure:

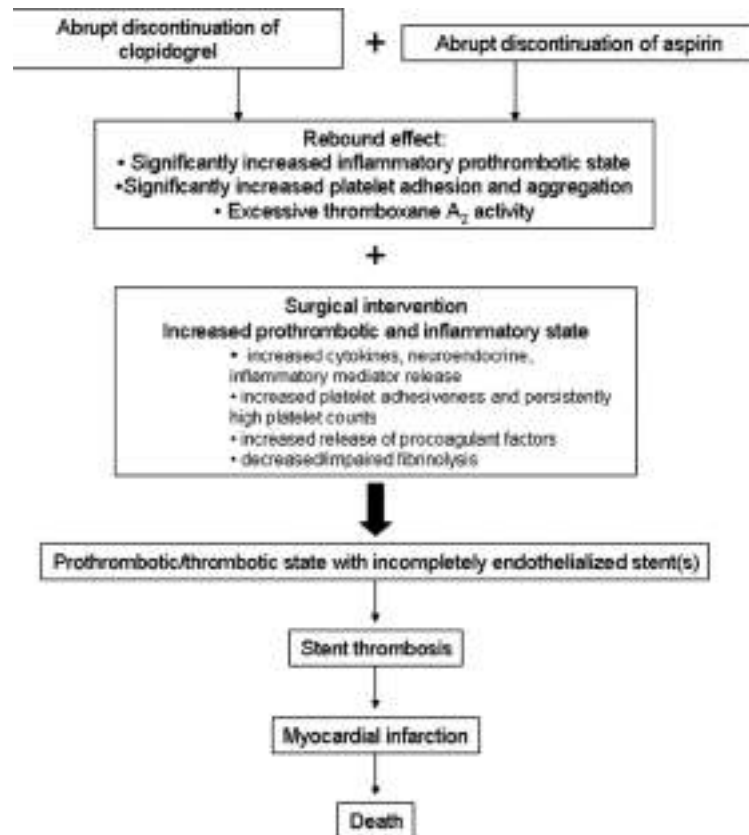




	Halothane	Isoflurane	Sevoflurane	Desflurane	Enflurane
Blood Pressure	↓↓↓	↓	↓	↓	↓↓
SVR	No Change or Very less	Potent decreases in SVR	Potent decreases in SVR	Potent decreases in SVR	More than halothane ↓ in SVR
Contractility	↓↓↓ Effect on Ca channels	No much effect	No much effect	No much effect	↓↓
Heart Rate	Normal or ↓	↑	N/↑	↑↑↑ (if > MAC)	↑
Sympathetic activity	Sensitizes heart to catecholamines	↑↑	N/↑	↑↑↑	↑
Coronary Vasodilation		Coronary Steal	Causes coronary vasodilatation		



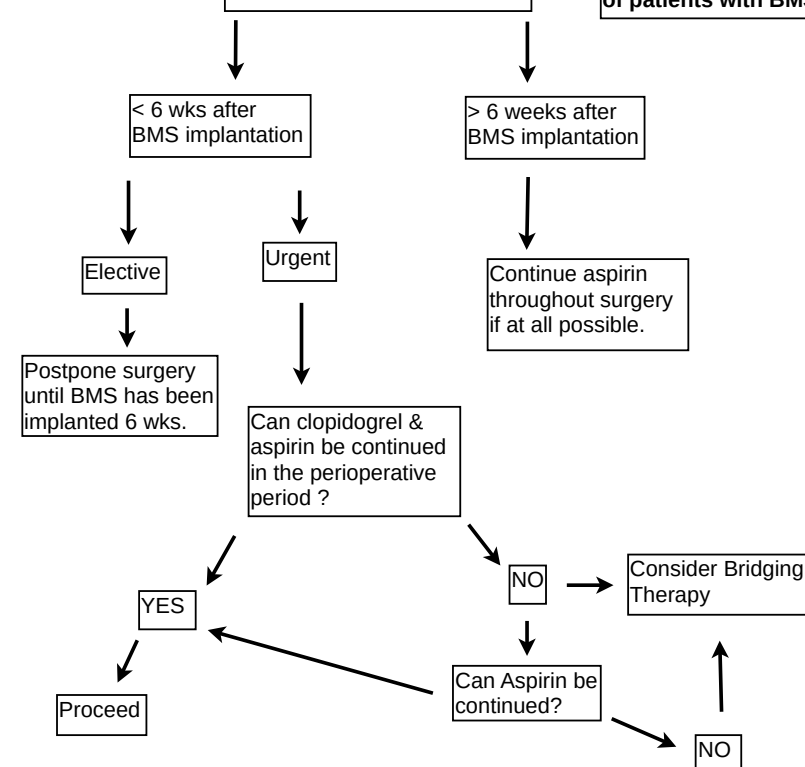
Pathophysiology of Acute perioperative Stent thrombosis:

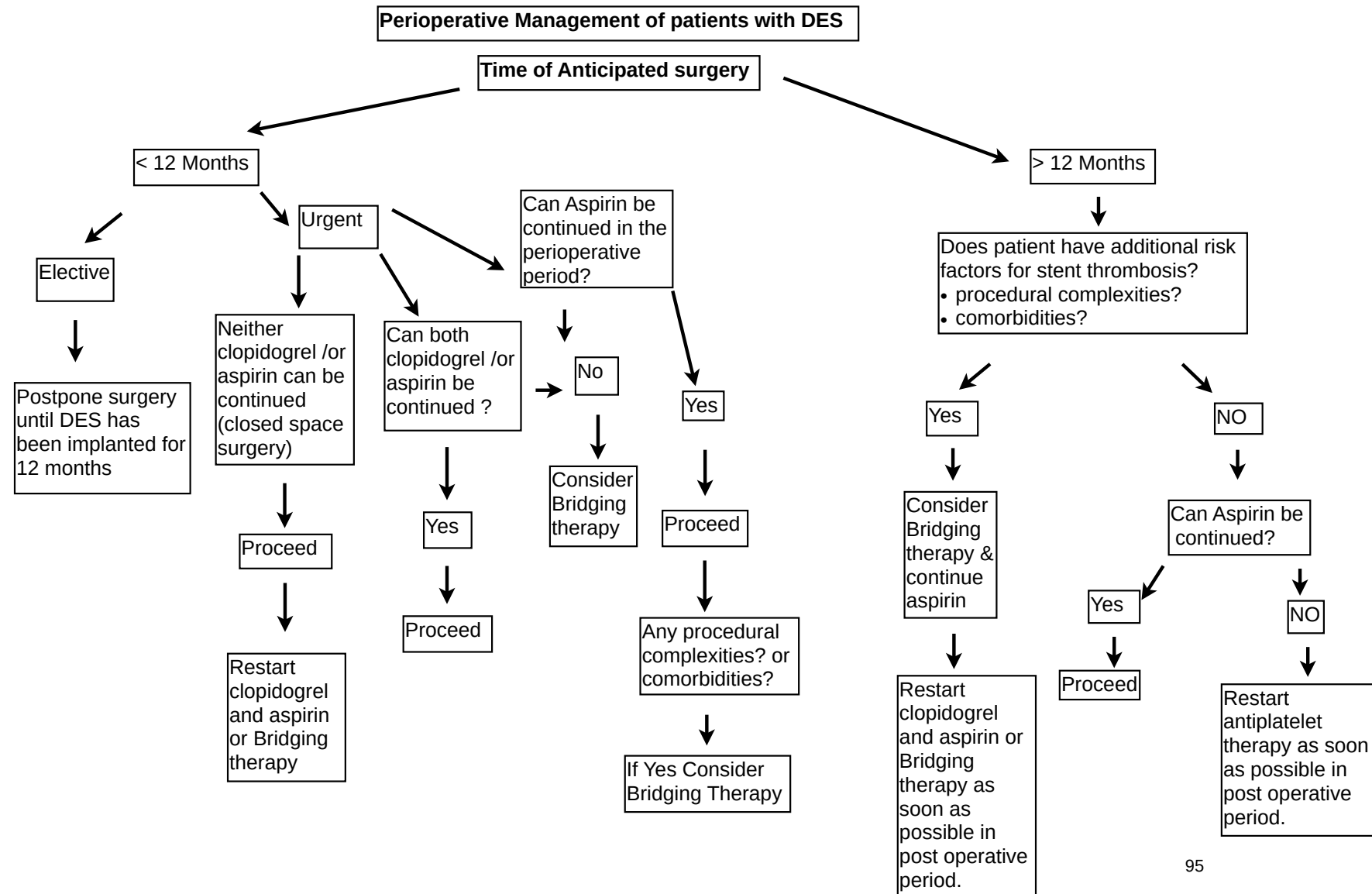


αα

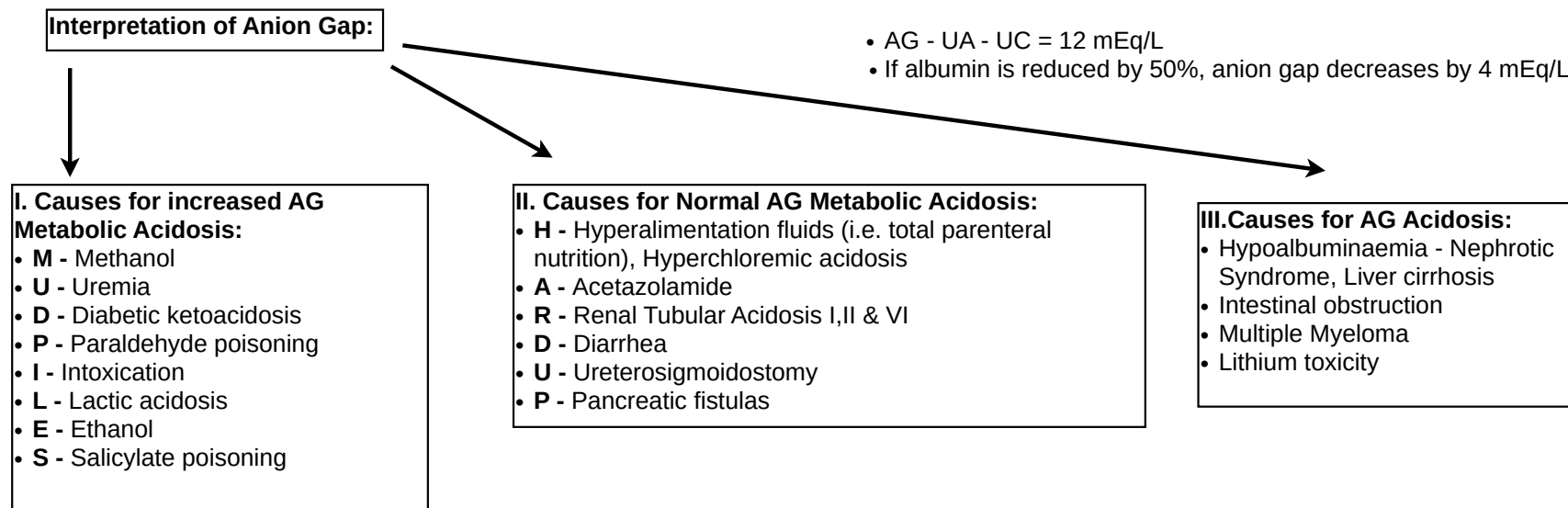
Time of Anticipated surgery

Preoperative Management of patients with BMS





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**Urinary Anionic Gap:**

- If Normal Anionic Gap Acidosis
 - Urinary AG = (Na + K) – Cl
 (Per-requisites: No Ketosis, Carbenicillin)
- If UAG is negative => GI or Iatrogenic
 - Positive UAG (> 20 to 30 mEq/l) = > RTA – (I,II,IV)
 - Look at Urine pH: > 6 - Distal (Type I) RTA
 - Look at Urine pH: <5.5 - Proximal Type II /Type IV
- Look at Serum K⁺
 - Hypokalemia - Proximal Type II
 - Hyperkalemia - Aldosterone Deficiency Type IV

ANION GAP: EXPECTED & ACTUAL AG

- Calculated AG = (Na – Cl + HCO₃)
- Corrected Expected Anion Gap (S.Proteins /pH)
 - For every 1gm % increase or decrease of S. Albumin the AG increase or decrease by 2 mEq/l
 - E.g.: In Patients with Nephrotic Syndrome / Cirrhosis
S. Albumin 2 gm % so expected AG = 12 – 4 = 8
- In Acidemia State Normal AG decrease by 2 mEq/L
- In Alkalemia State Normal AG increase by 4 mEq/L

Fluosol - DA	Polyfluro-octobromide
A 20% emulsion of 2 different perfluorocompounds which has oxygen carrying capacity at 37° C of about 40% of that of red cells.	Its a new Perfluorocompound (radio-opaque)
Dose: 20 ml/kg	It has 2 Advantages: O ₂ is more soluble in perfluorocarbon • Higher concentration of compound can be administered, therefore 100% emulsion with phospholipids has a sufficiently low viscosity to be infused without dilution. • It may carry as much as oxygen as Hb solution at a concentration of 7 gm/dl
Side effects: Pulmonary reactions, cytotoxicity, complement activation, Vulnerability to O ₂ toxicity	

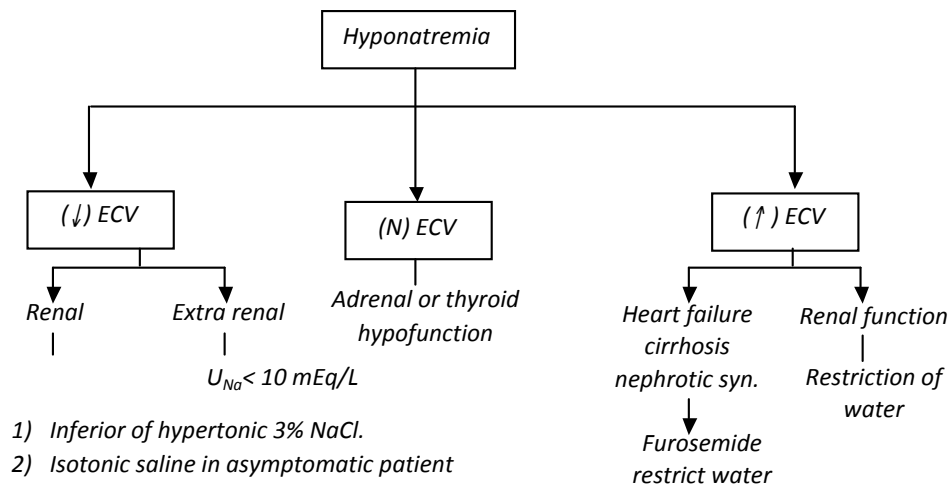
Treatment of Cardiac arrest with Lipid emulsion: 70 Kg patient

- Start oxygen & secure Airway & CPR - ACLS
- Give IV bolus of Intralipid 20% - 1.5 ml/kg over one min (100 ml)
- Continue CPR
- Start IV infusion of Intralipid 20% - 0.25 ml/kg/min (400 ml over 20 min)
- Repeat bolus every 3-5 minutes up to 3 mL/kg total dose until circulation is restored (100 ml at 5 min interval X 2 times)
- Continue infusion until hemodynamic stability is restored. Increase the rate to 0.5 mL/kg/min if BP declines (400 ml over 20 min)

A maximum total dose of 8 mL/kg is recommended

Estimated Fluid requirements - 4:2:1 Formula:

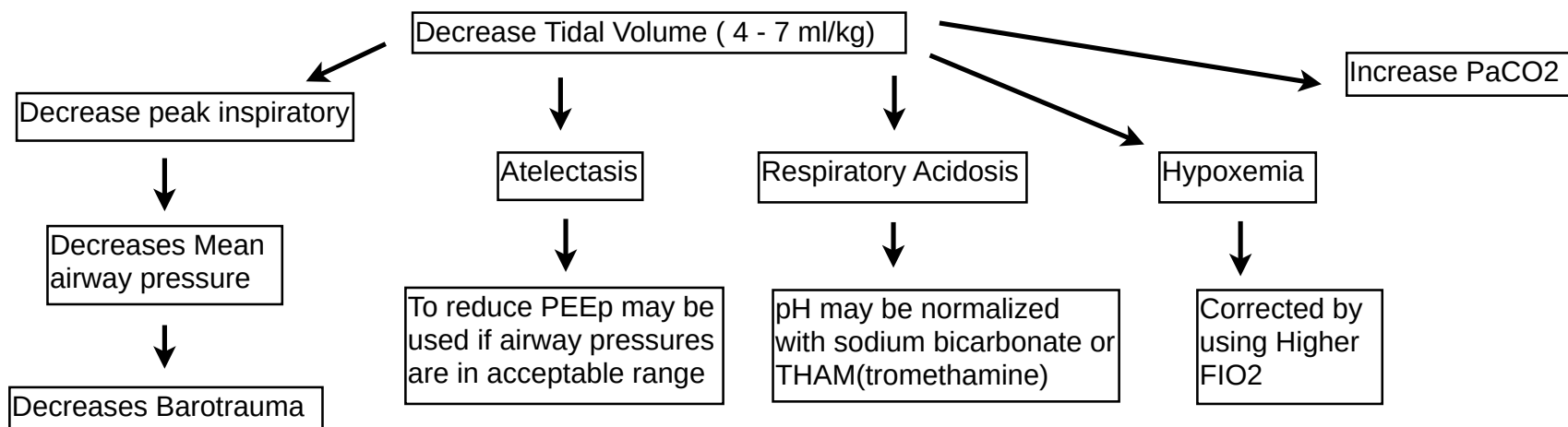
Body Weight	Fluid
First 10 kg	4ml/kg/hr
Second 10 kg	2 ml/kg/hr
Subsequent	1 ml/kg/hr



Third Space Loss:

Degree of Blood loss or Tissue Trauma	Additional fluid requirements
Minimal (e.g. herniorrhaphy)	0 - 2 ml/kg
Moderate (e.g. Appendicitis)	2 - 4 ml/kg
Severe (e.g. Bowel resection)	4 - 8 ml/kg

Mechanism & physiologic changes in permissive Hypercapnia

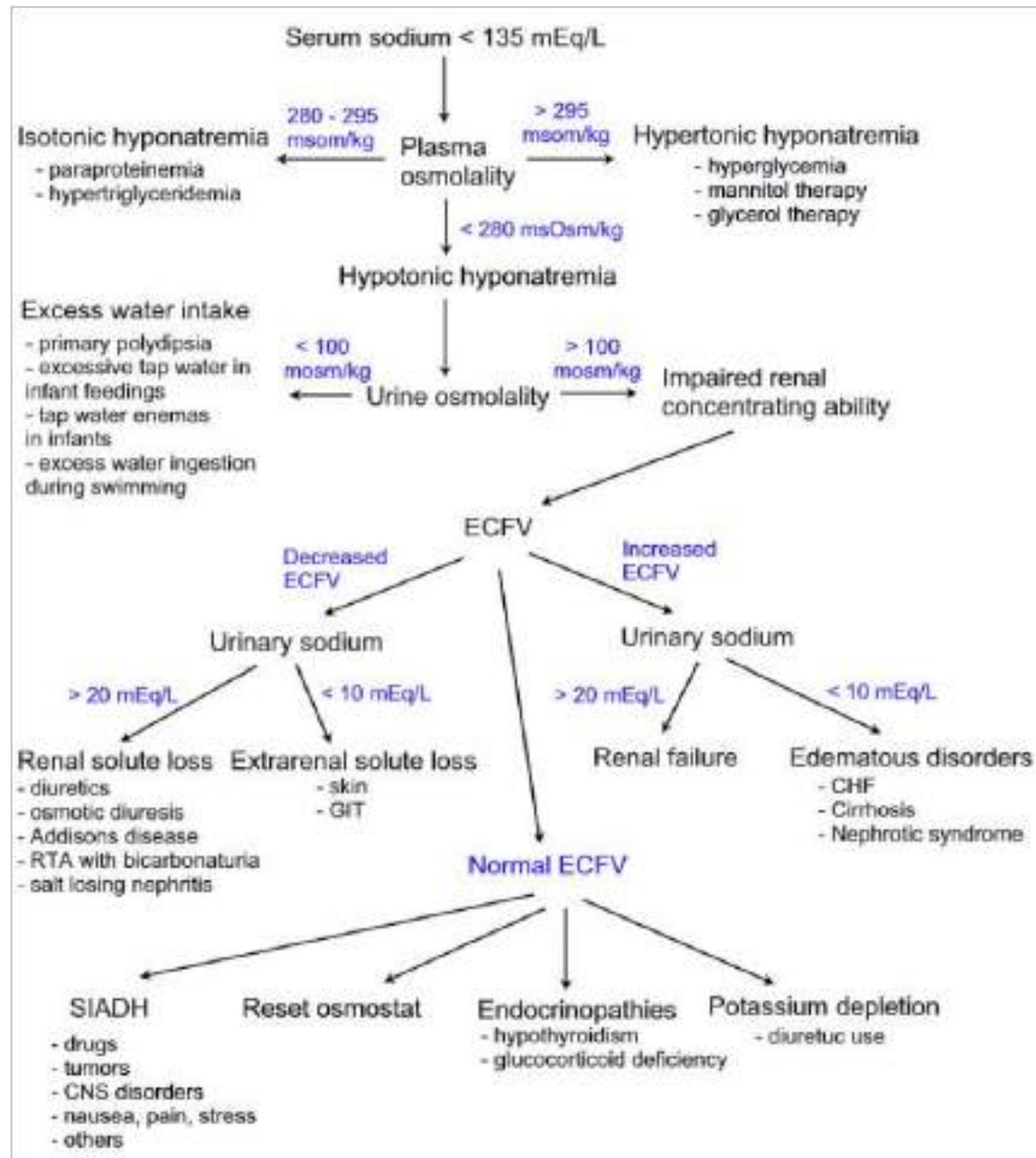


Solutions	Elemental Ca	Unit Vol.
10% CaCl ₂	27 mg/ml (1.3 mEq)	10 ml amp
10% Ca gluconate	9 mg/mL (0.4 mEq)	10 ml amp

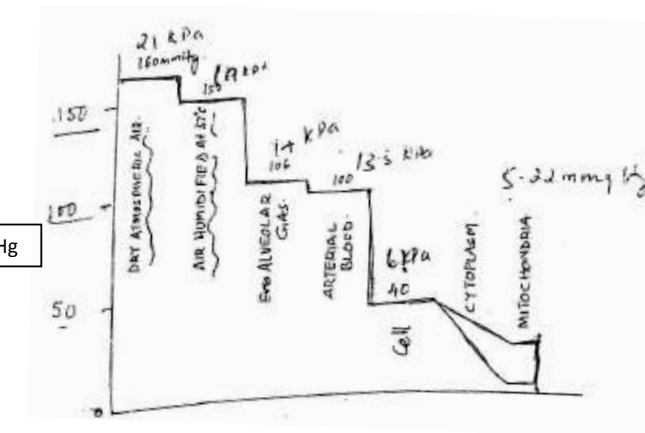
	Epidural space (mm)	Thickness of dura (mm)
Cervical	1.0 – 1.5	2.0 – 1.5
Upper thoracic	2.5 – 3.00	1.0
Lower thoracic	4.00 – 5.00	1.0
Lumbar	5.00 – 6.00	0.66 – 0.33

Liquid Ventilation - 2 Forms

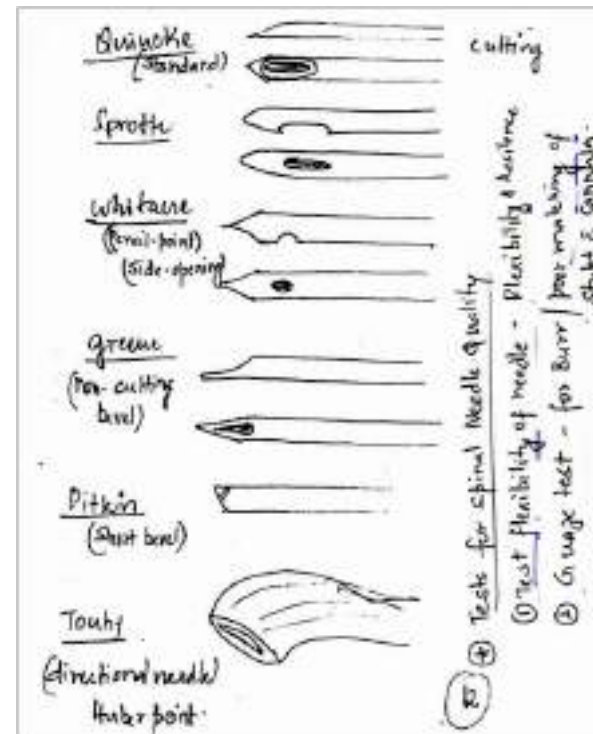
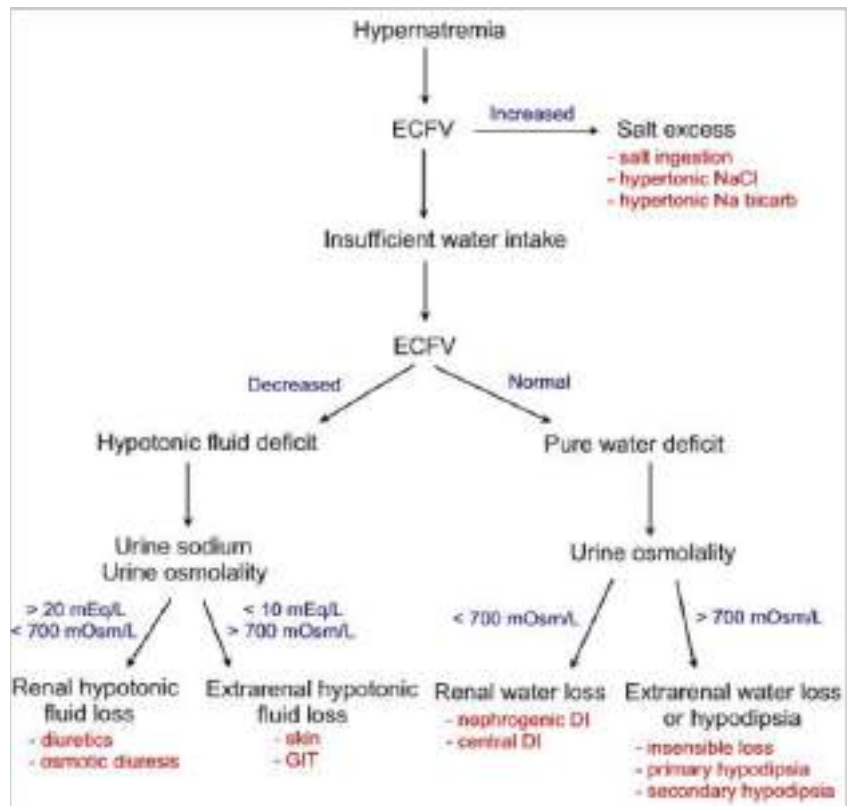
- Total or tidal liquid ventilation (TLV)
- PFC liquid equal to FRC is instilled into ETT.
- Similar volumes are cycled to facilitate gas exchange
- Partial liquid ventilation (PFC associated gas exchange PAGE or PLV.
- PFC liquid equal to FRC is put into ETT.
- Ventilate via convential ventilator



	HFPPV	HFJV	HFOV
RR	60-100	100-400	400-2400
Flow pattern	Square wave	Square wave	Sine wave
Gas extraction	No	Yes	No
Flow characteristics	Turbulent	Turbulent	Turbulent
FiO ₂	Controlled	Controlled	Controlled
Baro trauma	Yes	No	Yes
Flow generation	Pneumatic	Solenoid pneumatic	Piston
Expiration	Passive	Passive	Active
Psi	60	60	60

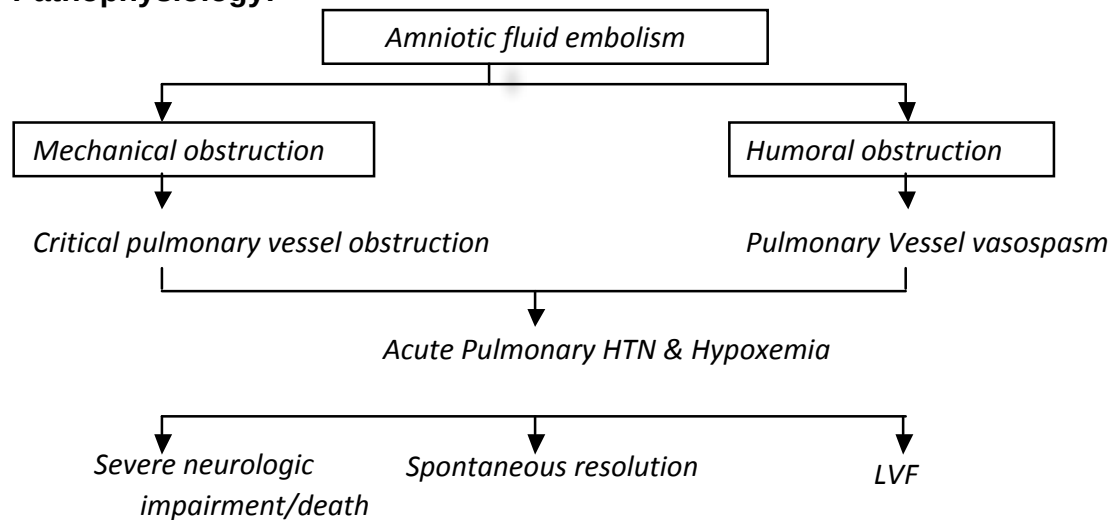


Oxygen Cascade:



TYPES:

Pathophysiology:



Classification:

<i>Hepatic porphyrias</i>	<i>Erythropoietic porphyrias</i>
Acute intermittent porphyria	Erythropoietic uroporphyrria
Variegate porphyria (AD)	Erythropoietic proto porphyria
Hereditary coproporphyria	
Porphyria cutania tarda(AD)	

Muscles of Larynx:

S.

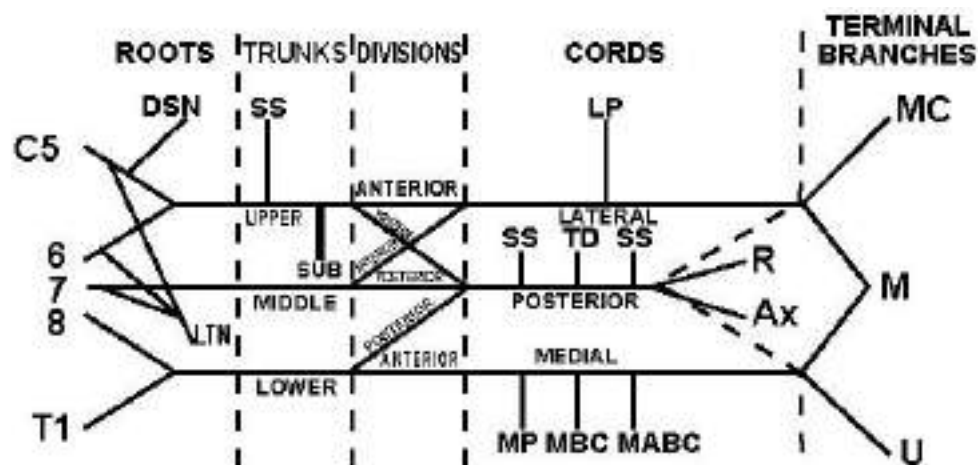
Muscles of Larynx:

Intrinsic Muscles:

- **Abductors:**
 - Posterior cricoarytenoid
- **Adductors:**
 - Lateral cricoarytenoid
 - Transverse arytenoid
 - Cricothyroid*
 - Thyroarytenoid *
- **Relaxors:**
 - Thyroarytenoid*
 - Vocalis
- **Tensors:**
 - Cricothyroid *

Extrinsic Muscles:

- Sternothyroid
- Thyrohyoid
- Inferior constrictor
- Stylopharyngeus
- Palatopharyngeus



Ligaments:

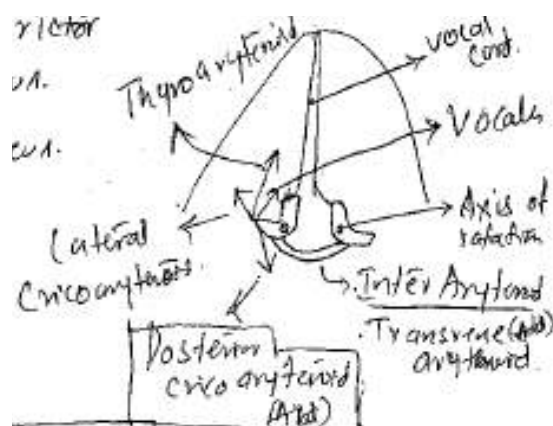
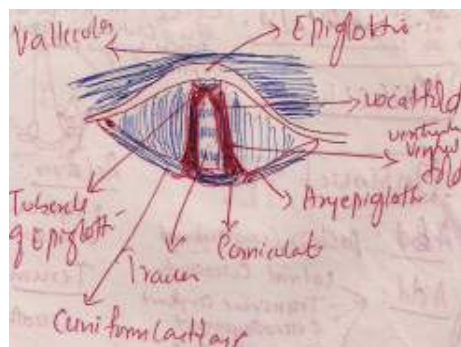
Intrinsic Ligaments:

- Cricovocal membrane
- Quadrangular membrane
- Cricothyroid membrane

Extrinsic Ligaments:

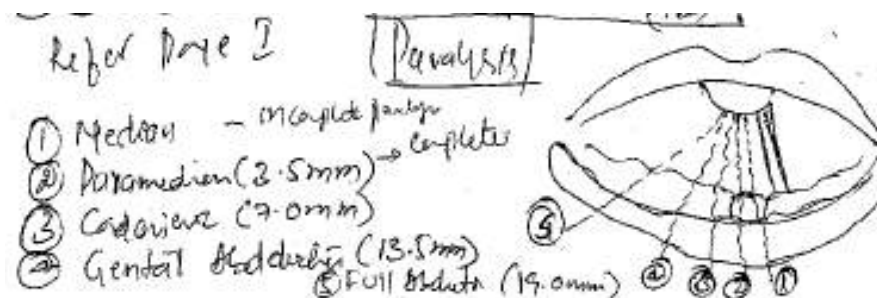
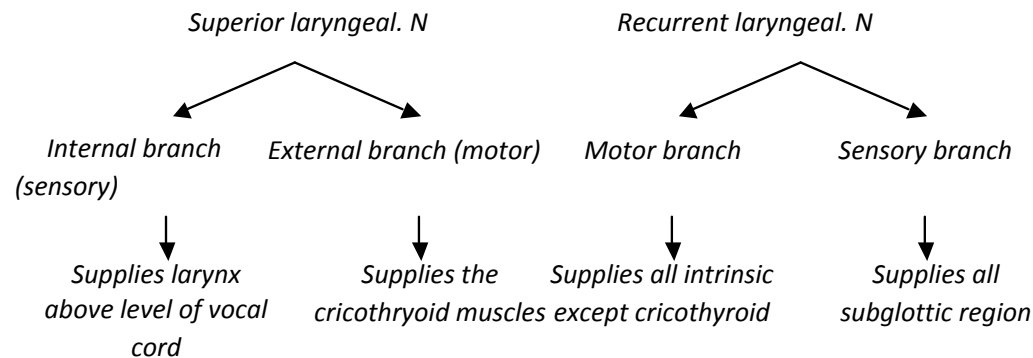
- Thyrohyoid membrane
- Cricotracheal membrane
- Hypoepiglottic ligament

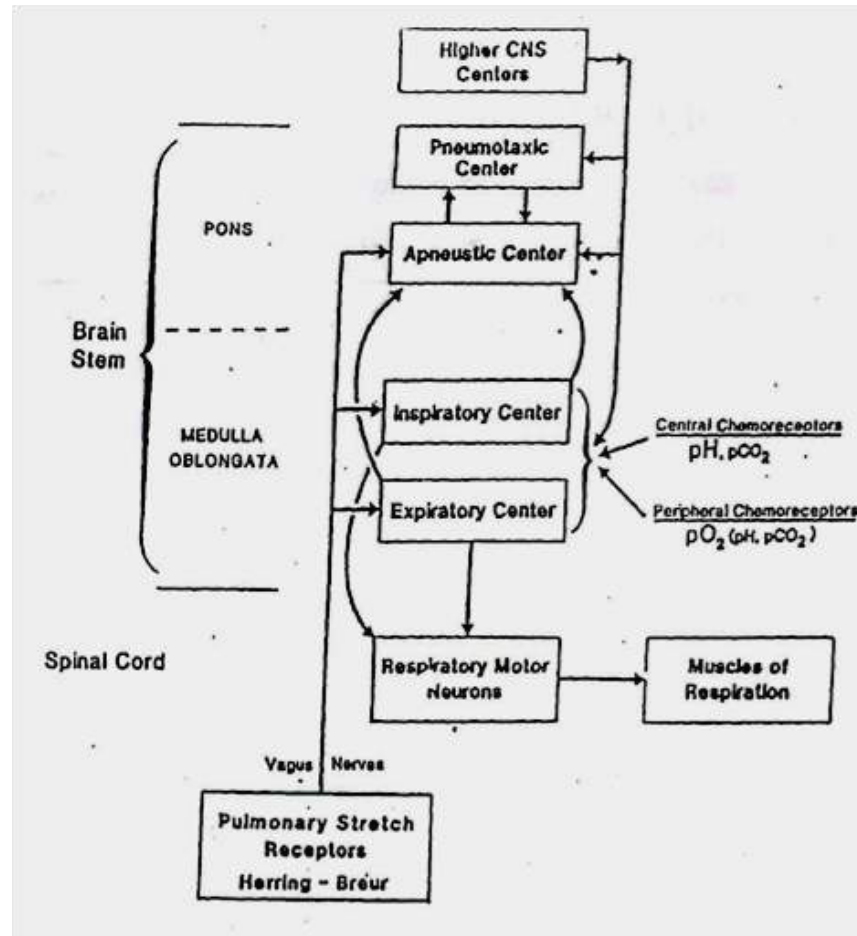
Ligaments of larynx:



Nerve Supply:

VAGUS NERVE





Control Of Breathing:

Intraoperative bronchospasm: Identification & Management:

opioids via epidu

To R/O other causes of high airway pressure & wheezing:

- ET - Tube Kinking
- Thick secretions
- Pulmonary edema
- tension pneumothorax
- Aspiration Pneumonitis
- Pulmonary edema
- Endobronchial intubation
- bucking

Deepen Anesthesia:

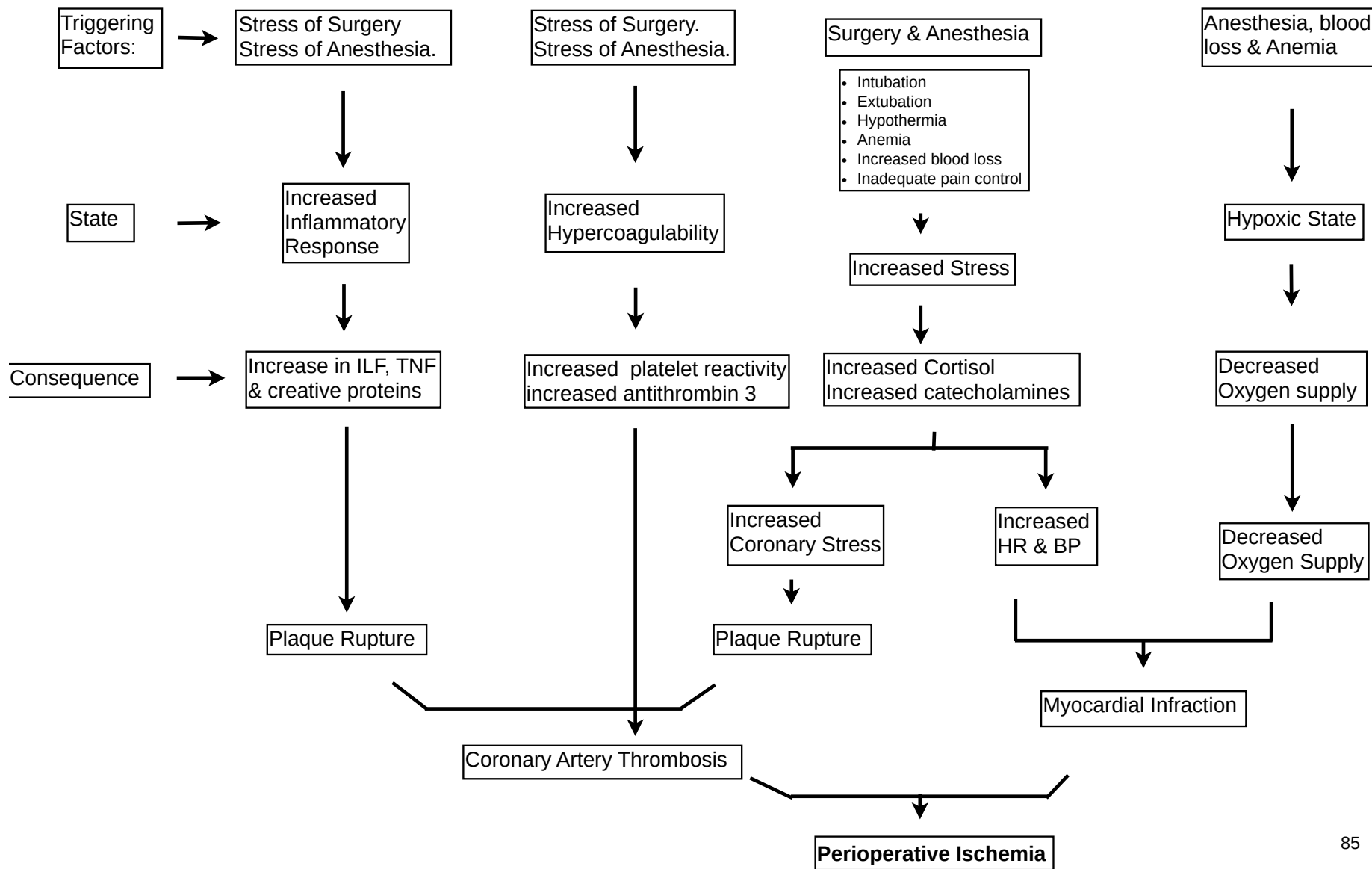
- Give 100% oxygen
- Inhalation Anesthetic agents if BP is normal.
- Low BP - Ketamine

Beta2 Agonist:

- Rapid acting albuterol through meter dose inhaler directly in breathing circuit.
- Aminophylline 0.5mg/kg - tachyarrhythmias
- Terbutaline S/C 0.25mg every 20 min for 3 doses.
- Epinephrine: 0.1 - 0.5 ml of 1:1000 S/C may be the last resort.
- Steroids: hydrocortisone 4mg/kg followed by 3mg/kg.

Flow Charts & Diagram.

Pathophysiology:

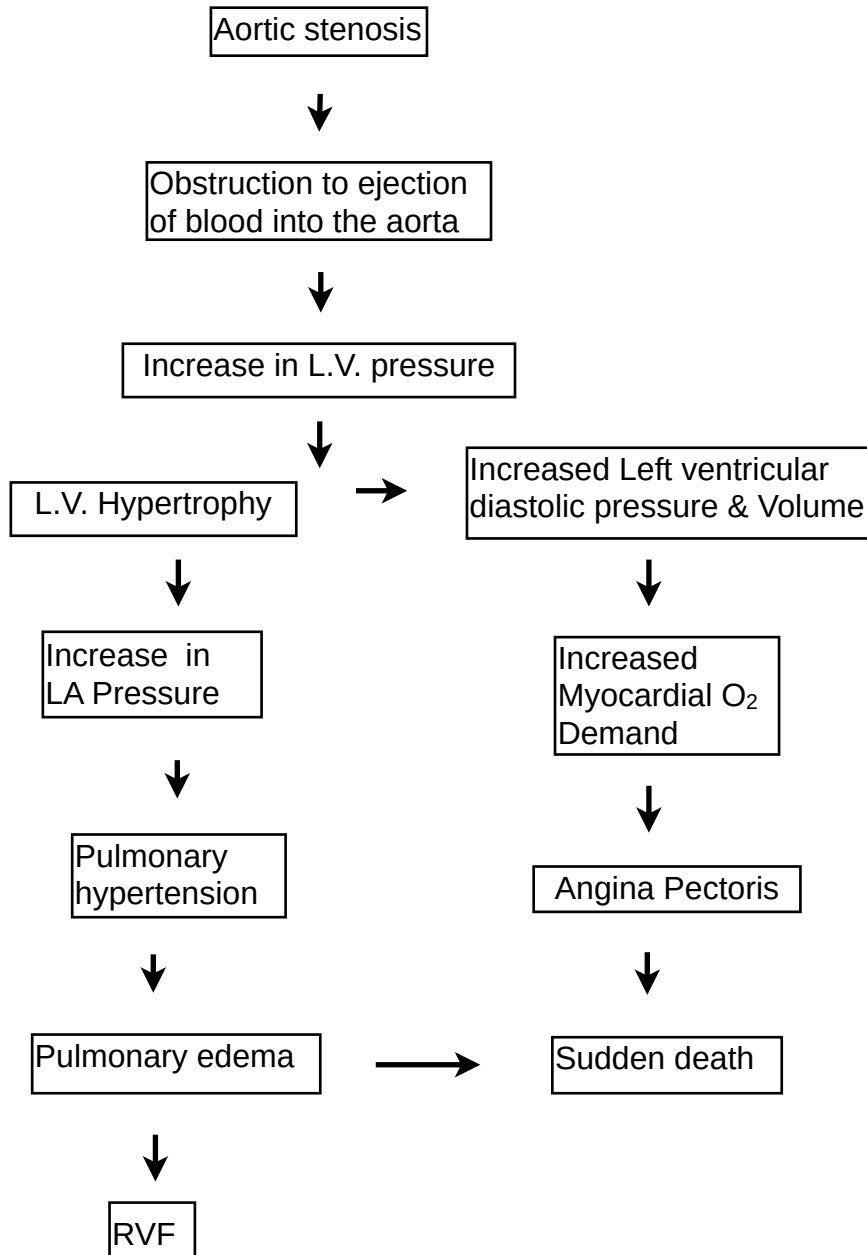


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Perioperative MI:

Acute treatments for suspected intraoperative myocardial ischemia

	Associated Hemodynamic Finding	Therapy	Dosage
I	Hypertension, tachycardia +	Deepen anesthesia IV β - Blockade	Esmolol 2-100 mg, + 50-200 Metoprolol 0.5-2.5 mg Labetalol 2.5 – 10mg 33-330 μ g/min +
II	Normotension, tachycardia +	IV Nitroglycerin Assure adequate anesthesia change anesthetic regimen IV β - Blockade	As above
III	Hypertension, normal heart rate	Deepen anesthesia nicardipine	0-20 mg Nicardipine 1-5 mg, + 1-10 μ Phenylephrine 25-100 μ g
IV.	Hypotension, tachycardia +	IV α -agonist	Nor-epinephrine 2-4 μ g
V	Hypotension, bradycardia	Lighten anesthesia IV Ephedrine IV Epinephrine IV Atropine IV Nitroglycerin when normotensive	5-10 mg 4-8 μ g 0.3-0.6 mg As above
VI.	Hypotension, normal heart rate	IV α - Agonist IV Epinephrine alter anesthesia (eg, lighten) IV Nitroglycerin when normotensive	As above
VII.	No abnormality	IV Nitroglycerin IV Nicardipine	As above As above

Aortic Stenosis. Continuation:**Pathophysiology:****Calculation of Stenosis:****Gorlin's modification of standard hydraulic formula**

$$\frac{(\text{Cardiac output} / \text{systolic ejection period}) \times \text{H.R.}}{1}$$

$$\text{Aortic valve orifice (cm}^2\text{)} = 1 \times 44.5 \times \text{mean aortic pressure gradient}$$

Where 1 = Aortic orifice constant

44.5 – hydraulic constant Simplified version of Gorlin's formula

Aortic valve area = Cardiac out put (lt/min) / Mean aortic pressure gradient

Normal : Aortic valve area 2.5 – 3.5 cm²

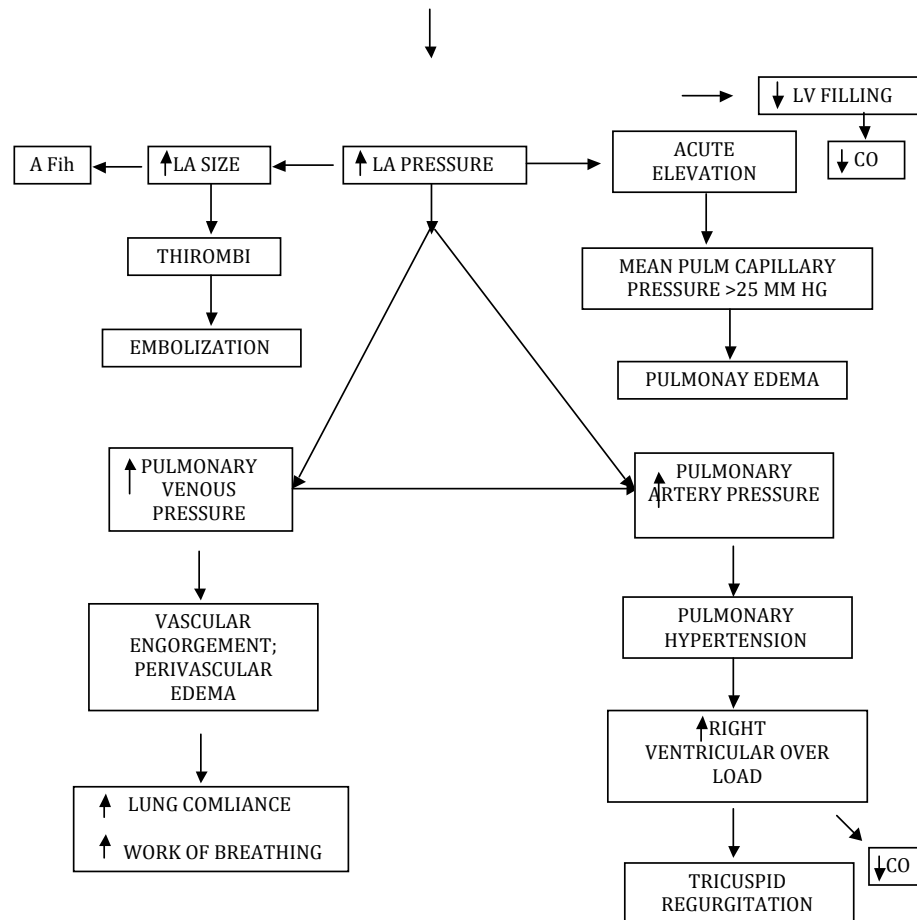
Aortic valve area reduction

- Normal - 2.6 – 3.5 cm²
- Mild - 1.2 – 1.8 cm²
- Mod - 0.8 – 1.2 cm²
- Significant - 0.6 – 0.8 cm²
- Critical - < 0.6 cm²

Mitral Stenosis. Continuation:

Pathophysiology: Downward to upward disease or failure.

OBSTRUCTION TO LA EMPTYING



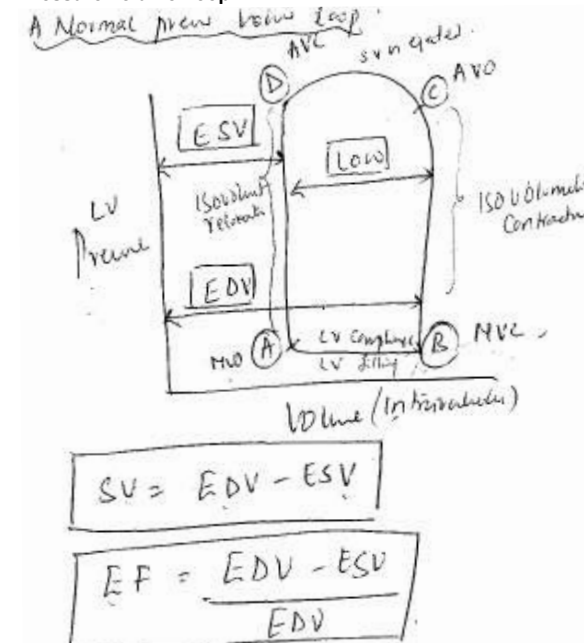
Mitral valve Stenosis:

Grades	MVA
Mild	1.5 – 2.5 cm ²
Moderate	1.1 – 1.5 cm ²
Severe	0.6 – 1 cm ²
Critical	Less than 0.6 cm ²

Mitral Stenosis – hemodynamic goals:

Preload	Maintain, preload, avoid hypovolemia
After load	Prevent pulmonary vasoconstriction (hypoxia, hypercarbia) ionotropes may be required for systemic hypotension.
Contractility	RV dysfunction may be a problem with long standing pulmonary hypertension.
Rate	Low end of normal. Avoid tachycardia
Rhythm	Maintained

Pressure Volume Loop:



Lee's revised CRI- 1999.

	(Assign one point to each of the following variables)
i.	High risk type of surgery (intra-peritoneal, intra-thoracic or supra inguinal vascular procedures.
ii.	History of IHD (history of MI, positive exercise test, current complaint of ischemic chest pain or use of nitrate therapy of ECG with Q waves)
iii.	History of CHF
iv.	History of cerebro-vascular disease
v.	Insulin therapy for diabetes
vi.	Preoperative serum creatinine > 2.0 mg/gl Class I = 0 factors, Class II = 1 factor Class III = 2 factors Class IV = >2 factors

High (reported cardiac risk often >5%)

- Emergent major operations, particularly in the elderly.
- Aortic and other major vascular
- Peripheral vascular
- Anticipated prolonged surgical procedures associated with large fluids shifts and/or blood loss.

Intermediate (risk generally <5%)

- Carotid Endarterectomy
- Head and neck
- Intraperitoneal and Intrathoracic
- Orthopedic
- Prostate

Low (Cardiac risk generally <1%)

- Endoscopic procedures
- Superficial procedures
- Cataract
- Breast

Goldman's CRI - 1977.

i.	Age > years	5
ii.	MI in previous 6 months	10
iii.	S3 gallop or ↑ JVP (0-3 cm)	11
iv.	Important aortic stenosis	3
v.	Rhythm other than sinus, Pac on last preoperative	7
vi.	>5 PVC/min at any time before	7
vii.	PaO ₂ , < 60 or PaCO ₂ > 50mm Hg, K<3.00 mEq/L or HCO ₃ < 20 mEq/l BUN > 50 or Cr> 3 mg/dl. Abnormal SGOT, signs of chronic liver disease, bed ridden from non-cardiac cause	3
viii.	Intraperitoneal, Intrathoracic, aortic surgery	3
ix.	Emergency operation	4
	Total	53
	Class I	0-5 points (low risk)
	Class II	6-12 points (intermediate risk)
	Class III	13-25 points (high risk)
	Class IV	≥ 26 points (very high)

Detsky's Modified CRI - 1986

i.	Age > 70 years	5
ii.	MI < 6 months	10
iii.	MI > 6 months	5
iv.	Unstable angina	10
v.	Pulmonary edema < 1 week	10
vi.	Pulmonary edema in the past	5
vii.	Non sinus rhythm PAC	5
viii.	CCVSA class III	10
ix.	CCVSA class IV	20
x.	Critical As	20
xi.	Emergency operation	5
xii.	Poor general status	5
	Total	120

Risk of Perioperative re-infarction in patients suffered with recent MI:

Duration Since Last MI	Re-infarct Rate
With in 3 months	30%
3 - 6 months	15%
> 6 months	6%

COMMONLY USED HYPOTENSIVE AGENTS

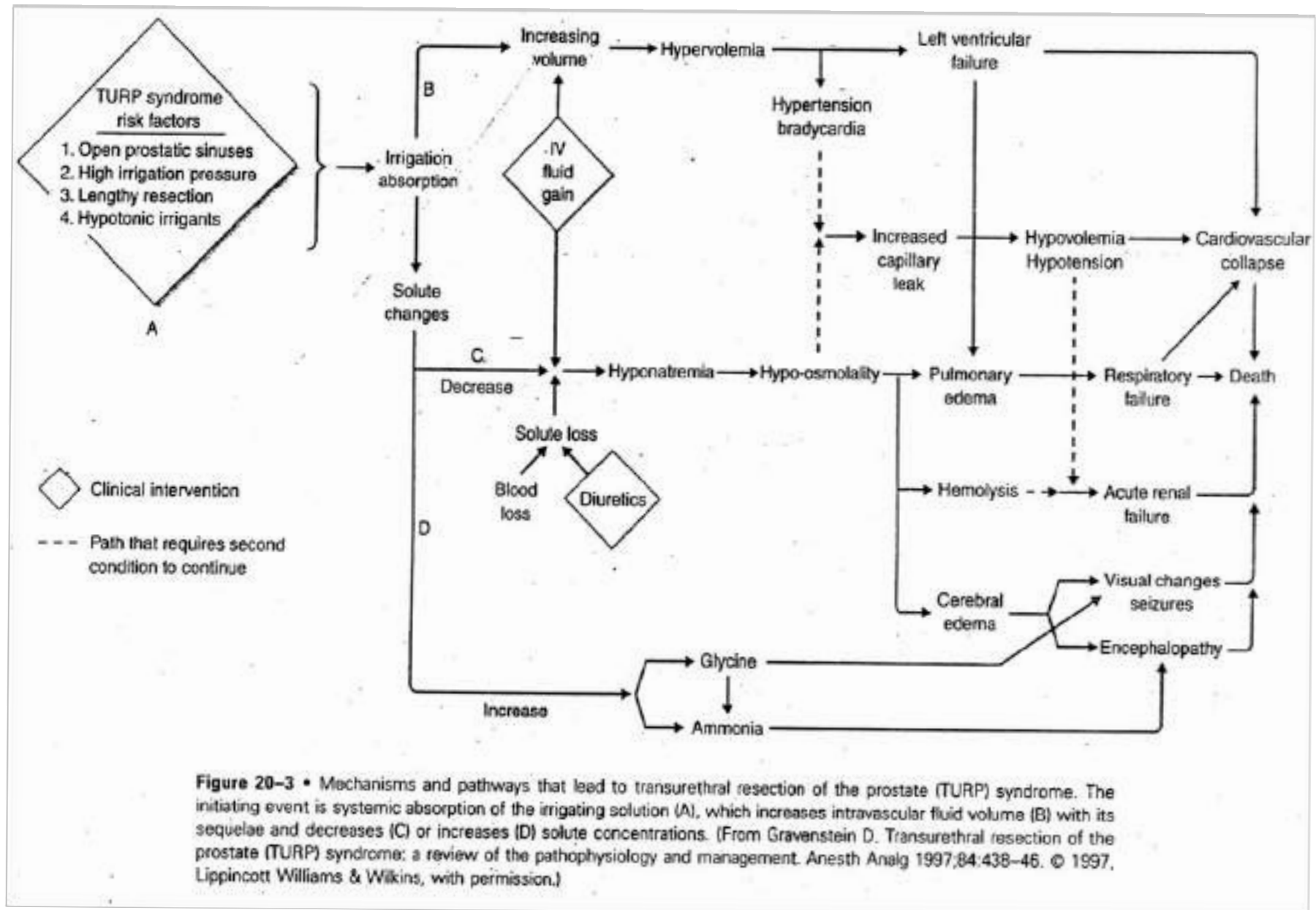
Drugs	Dosage	Mechanism of action	Advantage	Disadvantage
1. Sodium nitroprusside	0.5 to 10µg/kg/iv	Nitric oxide mediated direct vasodilator	Rapid onset and offset	Cyanide toxicity, ↑ ILP rebound HTN, ↑ pulmonary shunt coagulates abnormal
2. NTG nitroglycerine	1 to 10 µ/kg/iv	Same of nitric oxide	Rapid onset and offset	Cyanide toxicity, ↑ ILP rebound HTN, ↑ pulmonary shunt coagulates abnormal
3. Trimethaphan	1 to 5mg/iv	Ganglion blockade	Rapid onset and offset	Histamine release, ↓ pseudocholinesterase cerebral compression below MAP 55mmHg
4. Esmolol	0.2 to 5mg/kg/iv loading, 50-200µg/kg/iv	β-adrenergic blockade	Rapid onset and offset	Cardiac depress and bronchospasm
5. Labetalol	20mg tab 0.5 to 2mg iv, total 300mg	α and β - adrenergic blocked	↓ probability of adverse effects	Bronchospasm
6. PGE1	0.1 to 0.65 µg/kg/iv	Direct vasodilation	Rapid onset, decreases reflex tachycardia and stable CBF	Slow offset, bradycardia hypotension
7. Nicardipine	Begin 5mg/hr max 15mg/hr	Coronary and peripheral vasodilator	rapid onset, decreases reflex tachycardia and stable CBF	Slow off, resists antihypotensive therapy. ↑ pulmonary. shunt
8. Inhalation anesthetics	Titrate by inspired conc.	Vasodilation and myocardial depression	Provides surgical therapy	↑ ICP, ↑ cerebral edema, ↓ vital organ blood flow

Factors affecting myocardial O2 supply and demand balance.

DECREASE O2 SUPPLY	INCREASE O2 DEMAND
Decrease coronary blood flow	Tachycardia
Tachycardia	Increase wall tension (↑preload & after load)
Hypotension (diastolic)	Increase myocardial contractility
Increase preload	Hypertrophy
Hypocapnia (coronary vasoconstriction)	Shivering
Coronary artery spasm	HTN
II) DECREASE O2 CONTENT ± AVAILABILITY	Increase metabolic status
Anemia	Increase stress (surgery)
Hypoxemia	Exercise
Decrease O2 release from Hb	
Increase pH	
Decrease, DPG	
Decrease temp	

24. What are the side effects of different irrigation solutions?

Solution	Osmolality (MOSM/L)	Advantages	Disadvantages
Distilled water	0.0	Improved visibility	Hemolysis, Hemoglobinemia, Hemoglobinuria Hyponatremia
Glycine (1.5%)	220	Less likelihood of Transurethral Resection syndrome	Transient postoperative visual syndrome, Hyperammonemia Hyperoxaluria
Sorbitol (3.3%)	165	Same as Glycine	Hyperglycemia, possible Lactic acidosis Osmotic diuresis
Mannitol (5%)	275	Isosmolar solution it's Not metabolized	Osmotic diuresis possibility of acute intravascular volume expansion.
Normal Saline	308 (ISo)		Ionized, cannot be used with cautery
Urea 1%	167		Free passage into extra-vascular space, ↑ Blood urea
Glycine (1.2%)	175	Less likelihood of TURP syndrome	Transient visual impairment (blindness) Hyperammonemia, Hyperoxaluria
Glucose 2.4 - 5%	139		Sticky
Cytal (sorbitol 2.7% + Mannitol (.54%))	178		Expensive Not easily available



What are the systemic effects of Glycine?

Volume of Glycine absorbed	Signs and symptoms
>500 ml	Visual disturbances, headache
>1000 ml	Depressed consciousness, Pain abdomen
>3000 ml	Diarrhea
Plasma conc. of Glycine	Signs and symptoms
5-8 mmol/l	Visual disturbances
>10 mmol/l	Transient blindness, N&V
>20 mmol/l	Fatal

What is the formula used to calculate change in Serum sodium?

Adroque Formula:

$$\text{Change in serum Na}^+ = \frac{(\text{infusate Na}^+ + \text{infusate K}^+) - \text{serum Na}^+}{\text{total body water} + 1}$$

What are the manifestation of Hyponatremia?

Serum Sodium (mEq/L)	ECG Changes	CNS	CVS Manifestations
120	Widened QRS	Restlessness and confusion	hypotension & Bradycardia
115	Widened QRs/elevated ST segment	Nausea and / or semi coma	Cardiac depression
110	Ventricular tachycardia/fibrillation	Seizure and coma	CHF

What is the strength of sodium in various solutions?

Infusate	Infusate Na ⁺ (mmol/L)
5% NaCl	855
3% NaCl	513
0.9% NaCl (NS)	154
Lactate Ringer's	130
0.45% NaCl (½ NS)	77
0.2% NaCl (¼ NS)	34
5% Dextrose in water (D5W)	0

Formula to calculate the sodium deficit?

- **Sodium deficit (mEq) = Weight X Normal TBW X (130 - Current Na)**
- The normal TBW (in liters) is 60% of the lean body weight (in kg) in men, and 50% of the lean body weight in women. Thus, for a 60 kg woman with a plasma sodium of 120 mEq/L, the sodium deficit will be $0.5 \times 60 \times (130 - 120) = 300$ mEq.
- E.g. Because 3% sodium chloride contains 513 mEq of sodium per liter, the volume of hypertonic saline needed to correct a sodium deficit of 300 mEq will be $300/513 = 585$ mL. Using a maximum rate of rise of 0.5 mEq/L per hour for the plasma sodium (to limit the risk of a demyelinating encephalopathy), the sodium concentration deficit of 10 mEq/L in the previous example should be corrected over at least 20 hours. Thus, the maximum rate of hypertonic fluid administration will be $585/20 = 29$ mL/hour. If isotonic saline is used for sodium replacement, the replacement volume will be 3.3 times the replacement volume of the hypertonic 3% saline solution.

Table 5.2 Minimum alveolar concentration (MAC) values.

Agent	MAC
Desflurane	6.0%
Sevoflurane	2.05%
Isoflurane	1.15%
Halothane	0.75%
Nitrous Oxide	105%

Table 5.3 Factors affecting MAC.

Increased MAC	Decreased MAC
Children (from infancy to adolescence)	Premature infants and the elderly
Hypernatremia	Hypothermia
Cocaine or amphetamine intoxication	Pregnancy
Chronic alcohol use	Acute alcohol intoxication
MAO inhibitors	Opiates, benzodiazepines, barbiturates, clonidine, dexmedetomidine
Tricyclic antidepressants	

Table 6.1 Pain fiber types.

Fiber type	Local anesthetic sensitivity	Size	Myelination
A	+	Large	Yes
B	++	Medium	Yes
C	+++	Small	No

Table 2.1 Historical dates of note.

1500 BC	The use of opium-like preparations in anesthesia recorded in the Ebers Papyrus
1275	Ether discovered by Spanish chemist Raymundus Lullius
1540	The synthesis of ether was described by German scientist Valerius Cordus
1665	First intravenous injection of an opiate through a quill
1773	Joseph Priestly introduces nitrous oxide
1842	Crawford W. Long successfully uses ether during neck tumor excision in Jefferson, Georgia
1845	Horace Wells publicly demonstrates the use of nitrous oxide in Boston – however, it is labeled a “failure”
1846	William T.G. Morton shows first successful public demonstration of ether anesthesia at Massachusetts General Hospital, Boston.
1847	James Young Simpson uses Chloroform for labor pain
1853	John Snow administers chloroform to Queen Victoria during childbirth
1878	William MacEwan introduces oro-tracheal intubation with a flexible brass tube
1884	Carl Koller discovers local anesthetic properties of cocaine
1889	August Bier describes spinal anesthesia for surgery
1894	Anesthetic charts introduced
1905	Long Island Society of Anesthetists founded
1921	Fidel Pagés describes a lumbar approach to epidural anesthesia
1932	First barbiturate, hexobarbital, used clinically
1941	Robert Miller and Sir Robert Macintosh introduce “Miller” and “Macintosh” blade concepts, respectively
1942	Harold Griffith uses curare for the first time during an appendectomy
1956	Michael Johnstone introduces halothane, a halogenated inhalational agent
1960s	Fentanyl, ketamine and etomidate synthesized
1977	Propofol is synthesized
1970s	Pulse oximeter is developed and becomes widely available for use in 1980s
1980s	Halothane gradually replaced by enflurane and isoflurane
1983	Archie I.J. Brain introduces Laryngeal Mask Airway (LMA)
1986	ASA House of Delegates passes “Standards for Basic Anesthetic Monitoring” resolution
1990s	Sevoflurane and Desflurane introduced into clinical practice
2000s	Anesthesia Information Management Systems (AIMs) come into widespread use

Table 4.3 Recommended drug dosages for common IV agents.^a

<i>Benzodiazepines^b</i>	<i>Induction agents</i>
Midazolam 1–4 mg	Propofol 2–2.5 mg/kg (induction), 25–200 mcg/kg/min (infusion)
Diazepam 2.5–10 mg	Thiopental 3–5 mg/kg
Lorazepam 1–4 mg	Etomidate 0.2–0.5 mg/kg
<i>Opioids (Bolus)^b</i>	Ketamine 1–2 mg/kg IV, 3–4 mg/kg IM (induction or bolus), 1–2 mg/kg/h infusion
Morphine 1–5 mg	<i>Neuromuscular blockers^c</i>
Hydromorphone 0.2–0.5 mg	Succinylcholine 1–2 mg/kg, 20 mg bolus for laryngospasm
Fentanyl 25–100 mcg	Rocuronium 0.6 mg/kg
Meperidine 25–50 mg	Vecuronium 0.1 mg/kg
	Cisatracurium 0.15 mg/kg
	Pancuronium 0.1 mg/kg

^a Adult dosages: Always start at low end of range and titrate up.

^b Titration ranges for premedication or intraop/post op bolus dosing.

^c Intubating dosages: Divide by 3 for ED95, divide by 5 for maintenance bolus dosing.

Table 4.5 Contraindications to succinylcholine use.

Elevated serum potassium levels (>5.5 meq/L)
History of burn injury
History of denervation injury
Known or suspected myopathy
Known or suspected risk for Malignant Hyperthermia
Known pseudocholinesterase deficiency

Table 6.2 Properties of common local anesthetic agents.

	Agent	Onset of action	pK _a (36°C)	Max dose (mg/kg)*	Duration of action (h)
Amides	Lidocaine	Rapid	7.8	4.5 (7 with epi)	1–2
	Mepivacaine	Moderate	7.7	5 (7 with epi)	1.5–3
	Prilocaine	Slow	8.0	6 (9 with epi)	1–2
	Ropivacaine	Slow	8.1	2.5 (3 with epi)	4–8
	Bupivacaine	Slow	8.1	2.5 (3 with epi)	4–8
Esters	2-Chloroprocaine	Very Rapid	9.1	9 (15 with epi)	0.5–1
	Procaine	Rapid	8.9	7 (10 with epi)	0.75–1
	Tetracaine	Slow	8.4	1.5 (2.5 with epi)	3
	Cocaine	Rapid	8.7	1.5	0.5

* Maximum dose for a single subcutaneous injection.

Table 7.1 Actions of vasoactive receptor sites.

Receptor	Receptor site action
α-1	Glycogenolysis, gluconeogenesis, constricts vascular smooth muscle, relaxes GI tract
α-2	Constricts vascular smooth muscle, decreases insulin secretion and norepinephrine release
β-1	Increases heart rate and contractility, secretion of renin
β-2	Glycogenolysis, gluconeogenesis, relaxes vascular smooth muscle and bronchioles
Δ-1	Increases renin release, dilates vascular smooth muscle
Δ-2	Constricts smooth muscle, inhibits norepinephrine release

Table 7.2 Receptor actions of commonly used vasopressors.

Drug	Direct	Indirect	Site of action
Ephedrine	+	++	α, β
Phenylephrine	+		α
Norepinephrine	+		α, β
Dopamine	++	+	α, β, D (dopamine receptor)

Table 7.3 Vasopressor dosing.

Ephedrine	2.5–10 mg IV bolus
Phenylephrine	40–100 mcg IV bolus or 20–150 mcg/min infusion
Norepinephrine	0.01–0.1 mcg/kg/min infusion
Dopamine	2–20 mcg/kg/min infusion

Table 7.4 Commonly used antiemetics.^a

Ondansetron (Zofran)	4 mg IV, may repeat × 1 (0.1mg/kg up to 4mg in children)
Dolasetron (Anzemet)	12.5 mg IV, may repeat × 1
Granisetron (Kytril)	0.5–1 mg IV
Promethazine (Phenergan)	12.5–25 mg IV, may repeat × 1
Dexamethasone (Decadron)	4–8 mg IV, best given early during intraoperative period
Droperidol (Inapsine) ^b	0.625 mg IV, may repeat q 10 min × 3

^aAll dosages for adults unless noted.

^bMust monitor ECG for 2 h post administration.

Factors Affecting Mask Ventilation

- Presence of a beard
- BMI > 26 kg/m²
- Missing teeth
- Age > 55
- History of snoring

Adapted from Langeron, O. Prediction of difficult mask ventilation. *Anesthesiology* 200; 92:1229.

Table 8.1 Energy requirements for various activities.

1 MET	Eating, getting dressed, working at a desk
2 METS	Showering, walking down eight steps
3 METS	Walking on a flat surface for one or two blocks
4 METS	Raking leaves, weeding or pushing a power mower
5 METS	Walking four miles per hour, social dancing, washing car
6 METS	Nine holes of golf carrying clubs, heavy carpentry, using push mower
7 METS	Digging, spading soil, singles tennis, carrying 60 pounds
8 METS	Moving heavy furniture, jogging slowly, rapidly climbing stairs, carrying 20 pounds upstairs
9 METS	Bicycling at a moderate pace, sawing wood, slow jumping rope
10 METS	Brisk swimming, bicycling uphill, walking briskly uphill, jogging at 6 MPH
11 METS	Cross-country skiing, full court basketball
12 METS	Running continuously at 8 MPH

Adapted with permission from Brigham and Women's Hospital Preoperative Assessment Form.

Clinical Risk Factors for Increased Perioperative Cardiac Risk

History of heart disease (MI, Q waves on ECG, known CAD)

History of compensated or prior heart failure

History of cerebrovascular disease

Diabetes mellitus

Renal insufficiency

# Clinical risk factors	Surgical Risk	Action
0	Any	Proceed with surgery
1–2	Intermediate risk surgery Vascular surgery	Proceed with surgery with heart rate control or
3+	Intermediate risk surgery	Consider cardiac testing if it will change management
	Vascular surgery	Consider cardiac testing if it will change management

Source: ACC/AHA 2007 Guidelines on Perioperative Cardiovascular Evaluation and Care for Noncardiac Surgery: Executive Summary. *JACC* Vol. 50, No. 17, 2007.

Table 8.2 Risk reduction strategies for pulmonary complications.

Preoperative

- Smoking cessation (for at least 8 weeks)
- Treat airflow obstruction (patients with COPD or asthma)
- Give antibiotics / postpone surgery in presence of respiratory infection
- Educate patients about lung-expansion maneuvers

Intraoperative

- Limit surgical duration <3 h
- Avoid pancuronium
- Consider laparoscopic surgical approach

Postoperative

- Encourage incentive spirometry or deep breathing exercises
- Initiate CPAP (continuous positive airway pressure)
- Consider epidural analgesia / intercostal nerve blocks

Adapted from Smetana et al. Preoperative pulmonary evaluation. NEJM. Vol 340: 2008.

Table 9.1 Components of the preoperative airway physical examination.

Component	Nonreassuring finding
Length of upper incisors	Relatively long
Relation of maxillary and mandibular incisors during normal jaw closure	Prominent "overbite" (maxillary incisors anterior to mandibular incisors)
Relation of maxillary and mandibular incisors during voluntary protrusion of the jaw	Patient's mandibular incisors anterior to (in front of) maxillary incisors
Inter-incisor distance (mouth opening)	<3 cm
Visibility of uvula	Not visible when tongue is protruded with patient in sitting position (e.g., Mallampati class >II)
Shape of palate	Highly arched or narrow
Compliance of submandibular space	Stiff, indurated, occupied by mass, or non-resilient
Thyromental distance	<3 finger breadths or 6–7 cm
Length of neck	Short
Thickness of neck	Thick (neck size > 17 inches)
Range of motion of head and neck	Patient cannot touch tip of chin to chest or cannot extend neck

Reproduced with permission from Caplan RA, Benumof JA, Berry FA (2003) Practice guidelines for the management of the difficult airway: an updated report by the American Society of Anesthesiologist's Task Force on management of the difficult airway. Anesthesiology 98:1269–1277.

Table 10.1 Basic anesthesia machine components.

- Source of gases (O₂, N₂O, Air)
- Flowmeters
- Vaporizers
- Scavenging system

Note: the breathing circuit and CO₂ absorber are separate from the machine.

Table 10.2 Components of the high and low pressure systems.

High pressure system:

- Pipelines and cylinders for the gases delivered
- Fail-safe valve
- Pressure regulator
- Oxygen flush valve

Low pressure system:

- Flowmeters
- Vaporizers
- Flow control valve
- Check valve
- Common gas outlet

Table 12.1 M.S.M.A.I.D.S. mnemonic.

M → Machine

S → Suction

M → Monitors

A → Airway

I → IV

D → Drugs

S → Special

In preparing a room, most anesthesiologists will use the mnemonic M.S.M.A.I.D.S.

Table 12.2 Stages of general anesthesia.

Stage 1 – Amnesia	Patients should follow commands; Respiration pattern typically regular
Stage 2 – Delirium	Period of uninhibited excitation; Patients at risk for laryngospasm; Pupils often divergent; Respirations often irregular
Stage 3 – Surgical anesthesia	Target depth for anesthesia during surgery; Respiration pattern typically regular
Stage 4 – Overdosage	Patients at risk for hypotension and cardiovascular collapse

Table 13.1 Risks of neuraxial anesthesia.

Bleeding

Infection

Nerve injury

Post-dural puncture headache

Failure of block to provide adequate anesthesia

Table 13.2 Contraindications to neuraxial anesthesia.

Absolute contraindications	Relative contraindications
Patient refusal	Bacteremia
Infection in the area of needle puncture	Pre-existing neurologic disease (e.g. multiple sclerosis)
Elevated intracranial pressure	Cardiac disease
Uncontrolled bleeding	Abnormal coagulation studies

Table 12.3 Action sequence of a general anesthetic.

Air plane analogy	Anesthesia tasks	Important points
<i>Preflight check</i>	– Operating room setup – Preoperative patient evaluation – Preoperative medications	– M.S.M.A.I.D.S – Assessment of medical history – Confirm NPO status – Obtain informed consent – Obtain I.V. access – Administer appropriate preoperative medications and/or anxiolysis
<i>Takeoff</i>	– Patient monitoring – Induction of anesthesia – Airway management	– Place and confirm appropriate monitors – Position patient and pad pressure points – Preoxygenate – Administer induction agent – Place endotracheal tube or other advanced airway device
<i>Cruising altitude</i>	– Maintenance of anesthesia – Maintenance of homeostasis	– Protect patient eyes – Monitor vital signs and maintain appropriate blood pressure – Ensure amnesia and anesthesia – Monitor blood loss and administer appropriate fluids
<i>Landing</i>	– Antagonism of neuromuscular blockade – Emergence/extubation	– “Reversal” of neuromuscular blockade – Turn off anesthetic agents – Ensure patient is awake, following commands, protecting airway and can ventilate and oxygenate adequately prior to extubation – Confirm stable vital signs
<i>Taxi to the terminal</i>	– Safe transfer to PACU – PACU orders and discharge	– Monitor airway – Maintain oxygenation – Confirm stable vital signs – Write appropriate order to treat pain, nausea, vomiting and hyper or hypotension – Give report to PACU staff

Table 13.3 Summary of upper extremity nerve blocks.

Type of nerve block	Indications	Anatomical landmarks	Average needle depth	Potential complications
<i>Interscalene</i>	Shoulder; upper arm	Between middle and anterior scalene muscles at the level of C6 (cricoid cartilage)	1–2 cm	Hemi-diaphragmatic paralysis; Horner's Syndrome; epidural spread; intravascular injection; ulnar nerve sparing
<i>Supraclavicular</i>	Upper and lower arm; hand	Between the middle and anterior scalene muscles, 1 cm above the clavicle	2–3 cm	Pneumothorax (1% incidence); intravascular injection
<i>Infravascular</i>	Lower arm; hand	2–3 cm caudad from the midpoint of the clavicle	6–8 cm	Pneumothorax (much lower than with supraclavicular); intravascular injection
<i>Axillary</i>	Lower arm; hand		4–6 cm	Intravascular injection; prolonged set-up time; miss the musculocutaneous nerve

Table 14.4 Example fluid replacement calculation.

Patient and procedure:

An 80 kg male patient undergoes a 1-h tonsillectomy at 8:00 am after being made NPO at midnight.

Blood Loss:

Estimated blood loss is ultimately 250 ml

Crystalloid vs. colloid choice:

Crystalloid is adequate, no colloid needed for this small volume blood loss. Lactated Ringers is optimal though saline could be used.

Replacement:

(Calculated from the four parts of Table 14.3.)

Total crystalloid administered is:

120 ml (maintenance for the 1 h duration)

+960 ml (for the NPO deficit)

+750 ml (for the blood loss)

±250 ml (for the third space loss, estimated at 2 ml/kg/h)

=2080 of NS or LR over the 2 h perioperative period.

Postoperative maintenance:

May be 120 ml per hour with adjustments made based on vital signs and urine output.

Table 13.4 Summary of lower extremity nerve blocks.

Type of nerve block	Indications	Anatomical landmarks	Average needle depth	Potential complications
<i>Femoral</i>	Anterior thigh; knee; medial aspect of lower leg (saphenous nerve)	1 cm lateral to palpation of femoral artery along the inguinal ligament	3–5 cm	Intravascular injection; miss obturator and lateral femoral cutaneous nerves
<i>Lumbar plexus (Psoas)</i>	Hip; anterior thigh; knee; medial aspect of lower leg (saphenous nerve)	5 cm lateral from the spinous process (midline) at the level of iliac crest	6–8 cm	Epidural spread; intravascular injection; retroperitoneal hematoma; needle trauma to kidney
<i>Sciatic (posterior approach)</i>	Posterior thigh; below the knee surgery (except saphenous nerve distribution)	Posterior superior iliac spine (PSIS); sacral hiatus; greater trochanter. A line bisecting the midpoint of the PSIS and greater trochanter to intersect with a line drawn from greater trochanter to sacral hiatus	5–7 cm	Intravascular injection; nerve injury
<i>Sciatic (lateral approach)</i>	Below the knee surgery (except saphenous nerve distribution)	Between vastus lateralis and biceps femoris muscles, contact femur, then change needle angle approx. 45°	6–8 cm	Intravascular injection, nerve injury, will miss posterior thigh (tourniquet pain)

Allowable Blood Loss (ABL) Formula

$$EBV \times (HCT_{\text{initial}} - HCT_{\text{final}}) / HCT_{\text{initial}}$$

where HCT_{final} = lowest acceptable hematocrit

Figure 14.2 Allowable Blood Loss (ABL) Formula.

Estimated Blood Volume (EBV) Formula

$$\text{EBV} = \text{weight (kg)} \times \text{average blood volume}$$

Note: Avg. blood volume in adult male = 75 ml/kg

Avg. blood volume in adult female = 65 ml/kg

Figure 14.3 Estimated Blood Volume (EBV) Formula.

Table 16.1 Common intraoperative problems and differential diagnoses.

Problem	Differential diagnosis	Remarks
Hypoxemia	Low inspired oxygen (FiO ₂)	Always start by increasing the FiO₂, and then continue to look for other causes.
	Hypoventilation	May be from opioids, benzodiazepines, or muscle relaxants, which decrease respiratory drive and muscle strength. If a patient is being ventilated, consider increasing respiratory rate or tidal volumes.
	Disconnection of breathing circuit	The most common cause of serious hypoxemic accidents.
	Atelectasis	Often a result of positive pressure ventilation, intubation and/or hypoventilation. Consider alveolar recruitment maneuvers.
	Bronchospasm	Consider administering albuterol
	Mucus plugging	Perform suction and alveolar recruitment maneuvers.
	Right-mainstem bronchial intubation	Maximum depth for tracheal tubes measured at teeth: females 21 cm; males 23 cm. May visualize with bronchoscope or auscultate with stethoscope.
	Pulmonary thromboembolism (PE)	This may be diagnosed by a sudden drop in end-tidal CO ₂ , and hypoxemia that does not improve with 100% FiO ₂ . Often accompanied by tachycardia and hypotension.
	Pulmonary edema	May be from fluid overload, diastolic or systolic dysfunction, or myocardial infarction.
	Venous air embolism	Also causes drop in end-tidal CO ₂ , and an increase in end-tidal N ₂ .
Hypotension	Hypovolemia	Common causes include blood loss or dehydration from preoperative fasting.
	Relative anesthetic overdose	Minimal or a decrease in surgical stimulation can lead to relative anesthetic overdose and hypotension.
	Vasodilatation from medication	Opioids, sedatives, and most anesthetics reduce central sympathetic outflow and cause vasodilatation. Virtually every anesthetic induction or heavy sedation will be accompanied by this finding. Usually treated with phenylephrine, 40–100 mcg.
	Low cardiac output	Many anesthetics decrease cardiac output. Other causes include congestive heart failure, myocardial infarction, or tamponade.
	Severe bradycardia	This may cause low cardiac output (CO = HR × SV), leading to hypotension.
	Severe tachycardia or arrhythmias	If atrial fibrillation or flutter becomes too fast, hypotension may result.

Hypertension	Pneumothorax	Uncommon, but may spontaneously arise during positive pressure ventilation.
	Anaphylactic reaction	Most commonly from reaction to muscle relaxants or antibiotics. Give epinephrine to treat.
	Sepsis	Sudden decreases in blood pressure may result from sepsis.
	Pain from surgical stimulus	Always consider "light" or insufficient anesthesia.
	Essential hypertension	These patients may be adequately anesthetized, but still markedly hypertensive.
	Tourniquet pain	A tourniquet can produce a hard, recalcitrant kind of hypertension called "cuff hypertension."
	Light anesthesia	Check for empty vaporizers or medication infusors
Failure to ventilate	Hypervolemia	Fluid overload from intravenous fluid or blood products; may lead to pulmonary edema and congestive heart failure in patients with heart disease.
	Kinked endotracheal tube	Most likely during ENT or thoracic surgery.
	Biting on endotracheal tube	May occur during "light" anesthesia or emergence; Can cause negative pressure pulmonary edema.
	Disconnection of endotracheal tube from circuit or adapter	The most common cause.
High airway pressures	Complete endotracheal tube obstruction from mucus or tissue	Can occur rapidly in infants/children whose ETT are narrow (especially when no humidification is used). Suction or replace tube.
	Hole in endotracheal tube or a punctured cuff	Most often during laser airway surgery or tracheostomy. Both surgeries also have risk of airway fire!
	Bronchospasm	(1) deepen anesthesia, (2) consider neuromuscular blockade, (3) administer beta-agonists, inhaled or intravenous corticosteroids, theophylline or epinephrine
	Kinked endotracheal tube	May require the use of an armored (metal spring reinforced) tube to prevent kinking.
	Biting on endotracheal tube	Consider placing a bite block or mouth gag.
	Mucus plugging	Common in patients with COPD, asthma and cystic fibrosis. This may require replacement of the ETT.
	Stacking or auto-PEEP of mechanical breaths	Occurs when the expiratory phase isn't long enough to allow exhalation. Decrease the respiratory rate.
	Dynamic airway obstruction	May be from an airway tumor or mediastinal mass, especially after change in patient position.

Table 16.2 Intraoperative hypoxemia: corrective actions.

1. Assure 100% FiO₂ delivery
2. Hand ventilate to assess compliance and chest excursion
3. Look for any disconnection: circuit, endotracheal tube, connectors
4. Check expired gases for ETCO₂
5. Inform the surgical team and operating room nurse if severe
6. Examine the capnograph waveform
7. Inspect the endotracheal tube and circuit for kinking, biting, obstruction
8. Inspect the endotracheal tube for excessively deep insertion (should be 21 cm in females, 23 cm in males)
9. Suction the endotracheal tracheal tube to remove secretions and assess patency or obstruction
10. Perform direct laryngoscopy to verify tube placement in the trachea
11. Apply recruitment maneuvers to open up alveoli

Table 16.1 (continued)

Problem	Differential diagnosis	Remarks
Hypocarbica	Obesity or chest wall rigidity	May be difficult to manage. High opioid dose can cause a "rigid chest syndrome."
	Acute Respiratory Distress Syndrome (ARDS)	A common cause of high mean pressures, especially in the ICU.
	Hyperventilation	May see in anxious (awake) or mechanically hyperventilated (anesthetized) patients.
	Leak of CO ₂ in sampling tubing	This may also cause an abnormality in the capnograph waveform or envelope.
	Massive pulmonary embolus	Can impair gas exchange, manifesting as a sudden drop in expired CO ₂ .
Hypercarbica	Hypothermia	Most evident in severe hypothermia as during cardiopulmonary bypass.
	Cardiac Arrest	Impaired circulation and CO ₂ elimination.
	Hypoventilation	Often from opioids, residual neuromuscular blockade, or low respiratory rate/ventilator tidal volumes.
	CO ₂ insufflation during laparoscopy	May need to increase minute ventilation to overcome hypercarbica.
Hypothermia	Malignant hyperthermia	Uncoupling of calcium metabolism in mitochondria from a rare (1:15,000) genetic defect in the ryanodine receptor of the calcium channel
	Hyperthermia	Metabolic rate increases 15% for every degree centigrade.
	Convective, conductive, radiative, evaporative losses	Convective losses are the #1 cause of heat loss in the OR (skin to air). Heat loss also occurs from wet drapes and sheets, exposed skin or body cavities, non-heated breathing circuits.
	Anesthetic effects on hypothalamus	Anesthetics cause impaired central thermoregulation due to effects on the hypothalamus.
	Administration of unwarmed fluid or blood products	Fluids should be warmed by an FDA-approved device.
Hyperthermia	Massive blood loss	Difficult to keep patients warm after ≥ 1 blood volume has been lost.
	Excessive warming	Use the air-warming blanket at a room-temperature setting to cool the patient.
	Fever from sepsis or transfusion reaction	Give acetaminophen or ibuprofen in addition to a cooling blanket.
	Stroke	Sudden extreme hyperthermia (>105°F) may be from a stroke to the hypothalamus.
	Neuroleptic malignant syndrome	Uncommon side effect of antipsychotic medications (chlorpromazine, haloperidol, olanzapine).
	Malignant hyperthermia	(1) stop anesthetic, (2) give iv dantrolene, (3) call for help (see Appendix B, Malignant Hyperthermia)

Table 17.3 Ischemic cardiac disease.

Disease	Definition
Coronary artery disease	Narrowing of coronary arteries from atherosclerosis
Ischemic heart disease	Myocardial O ₂ demand not met by coronary blood flow
Acute coronary syndrome / myocardial ischemia	Term that includes any of the life threatening conditions below which represent acute myocardial ischemia
Angina pectoris	Myocardial ischemia with chest discomfort
Stable angina	Exercise induced chronic angina pectoris
Unstable angina	Myocardial ischemia at rest or with minimal exertion
Variant angina	Discomfort from coronary artery vasospasm
Non-ST-segment elevation myocardial infarction (NSTEMI)	Myocardial ischemia from a partially occlusive coronary thrombus
ST-segment elevation myocardial infarction (STEMI)	Myocardial ischemia from a totally occlusive coronary thrombus

Table 17.4 Etiology of Ischemia and Treatments.

Etiology	Treatments
Hypotension	– phenylephrine
Hypoxia	– increase the FiO ₂
Tachycardia	– β-blockers
Vasospasm	– nitroglycerin
Anemia	– transfusion
Thrombosis	– heparin
Hypovolemia	– fluid administration

Key Features of a Neuroanesthetic

- (1) Neuroprotection
 - (a) optimization of CBF/CMR balance
 - (b) control of ICP
 - (c) temperature regulation (avoid hyperthermia)
- (2) Provision of optimal operating conditions, including neuromonitoring and “relaxed” brain
- (3) Maintenance of normal glucose and electrolyte balance
- (4) Prompt emergence from anesthesia to facilitate neurologic assessment

Table 19.2 Apgar score.

	0 Points	1 Point	2 Points
Appearance	completely blue	extremities blue	Pink
Pulse	absent	<100 bpm	>100 bpm
Grimace	no response to stimulation	grimaces when stimulated	pulls away when stimulated
Activity	none	some flexion	moving actively
Respiration	none	weak	good

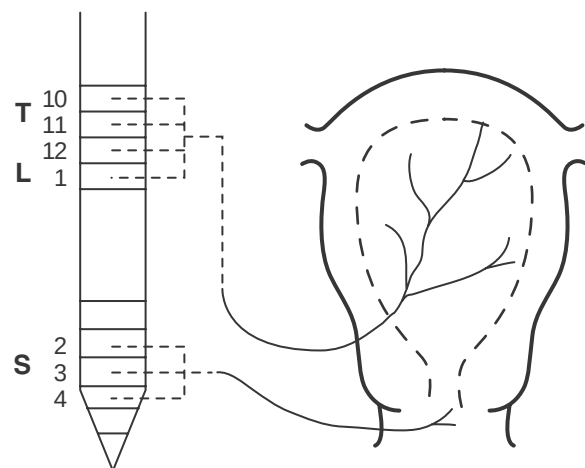


Figure 19.5 Pain pathways for the first and second stages of labor. (From Datta, S. *Obstetric Anesthesia Handbook*, 4th ed. Springer, 2006).

Table 20.1 Classification of obesity.

Obesity class	BMI	Health risk
Class I (overweight)	25–30	Low
Class II (obese)	30–35	Moderate
Class III (severely obese)	35–40	High
Class IV (morbidly obese)	>40	Very high

Table 20.2 Epidural catheter placement level for postoperative pain control.

Type of surgery	Usual incision	Epidural catheter placement
Liver, pancreas, spleen, stomach	Chevron or upper midline	Low thoracic
Kidney and ureter	Oblique flank	Upper lumbar
Colorectal	Low midline	Upper lumbar
Bladder, uterus surgery	Low transverse or low midline	Low lumbar
Hernia	Inguinal	Low lumbar
Hemorrhoid	Anorectal	Caudal

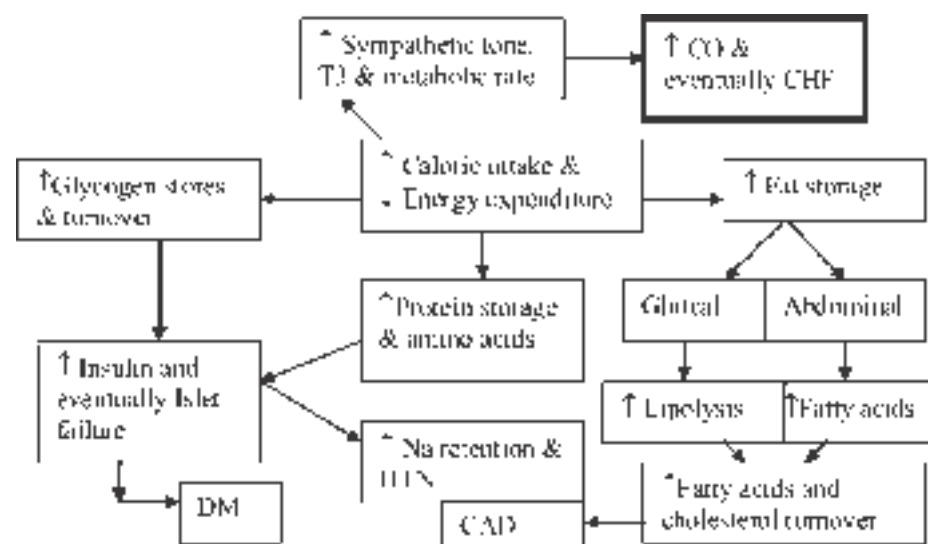


Figure 20.1 Systemic manifestations of obesity. CO = cardiac output; CHF = congestive heart failure; DM = diabetes mellitus; CAD = coronary artery disease; HTN = hypertension.

Table 21.1 Spinal pain segments for the genitourinary system.

Organ	Sympathetics	Pain pathways
Kidney	T8-L1	T10-L1
Ureter	T10-L2	T10-L2
Bladder	T11-L2	T11-L2 (bladder dome) S2–4 (bladder neck)
Prostate	T11-L2	T11-L2S2–4
Penis	L1 and L2	S2–4
Scrotum		S2–4
Testes	T10-L2	T10-L1

Table 23.1 Renal changes in the elderly.

Decrease in renal blood flow
 Decline in glomerular filtration rate
 Decline in ADH response
 Decrease in total body water
 Decreased ability to conserve sodium
 Diminished urine concentrating ability
 Decline in renin-aldosterone levels
 Decreased thirst perception

Table 21.2 Commonly used irrigating solutions.

Irrigating solution	Relative osmolality	Advantages	Disadvantages
Distilled water	Very hypoosmolar	↑ Visibility	Hemolysis, hemoglobinemia, hemoglobinuria, hyponatremia
Glycine	Hypoosmolar	↓ TURP syndrome incidence	Transient postoperative visual syndrome
Sorbitol	Hypoosmolar	↓ TURP syndrome incidence	Hyperglycemia, Osmotic diuresis
Mannitol	Isosmolar	Not metabolized	Osmotic diuresis, may cause intravascular volume expansion

Table 24.1 Patient selection factors for ambulatory surgery.

- Caregiver available for transport home and postoperative evaluation
- Patient willingness to go home the day of surgery
- Co-morbidities: obesity, obstructive sleep apnea, poorly compensated cardiopulmonary disease, chronic pain, renal failure, urinary retention, significant neurologic disease (myasthenia, Parkinson's, dementia)
- Prior anesthetic problems: difficult airway, PONV, postoperative cognitive dysfunction, malignant hyperthermia, poor pain-control

Table 21.3 Symptoms of TURP Syndrome.

Cardiovascular	Neurologic	Other
Hypertension	Confusion/disorientation	Hemolysis
Arrhythmias	Seizures	Hyponatremia
Congestive heart failure	Unresponsive	Hyperglycemia
Pulmonary edema	Visual problems or blindness	Hyperammonemia
Hypoxemia		
Myocardial ischemia		

Table 24.2 Procedure-related considerations for ambulatory surgery.

- Duration of surgery (no absolute cutoff)
- Intraoperative fluid shifts and bleeding
- Risk of serious postoperative complications (bleeding, infection, airway compromise)
- Extent of postoperative pain and analgesic needs
- Need for intravenous medications or inability to tolerate oral intake

Table 24.3 Common ambulatory procedures.

- Local lesion removal (cyst, melanoma, breast biopsy/part, mastectomy)
- Orthopedic procedures not involving major fractures
- Basic ENT procedures (sinus/tonsillectomy/tympanoplasty, cochlear surgery, single lobe thyroidectomy)
- Limited plastics procedures (blepharoplasty, scar revision)
- Limited urologic procedures (cystoscopy, biopsy, vasectomy, circumcision)
- Ophthalmologic procedures (excluding vitrectomy and enucleation)
- Limited GYN procedures (hysteroscopy, D&C/D&E, cone biopsy, tubal ligation)

Table 24.5 Unique aspects of Out-of-OR-anesthesia.

1. Limited anesthesia role in patient selection and preoperative optimization
2. Unfamiliar procedures and proceduralists
3. Remote locations with limited equipment availability
4. Enhanced role of nonanesthesia support staff
5. Greater potential for miscommunication
6. High expectations for patient satisfaction, clinical efficiency, and accommodation

Table 24.4 Procedure exclusions for outpatient management.

- Requires drain or nasogastric drainage tube to be placed
 - Hysterectomy, bowel resection, neck dissection
- Oral medications inadequate for postoperative pain control
 - Joint replacement, mastectomy, major abdominal surgery
- May require postoperative bladder catheterization
 - Ventral hernia repair, bladder tumor resection, ureteral stent
- Frequently requires intraoperative or postoperative transfusion
 - Hysterectomy, ORIF femur
- Expectation of postoperative electrolyte shifts
 - Parathyroidectomy, pituitary resection
- Requires hourly patient assessment
 - Free-flap, craniotomy, patients with severe sleep apnea

Table 24.6 Anesthetic options for GI endoscopy procedures.**Option A:**

1. Nasal cannula oxygen 2–4 L
2. Midazolam (1–4 mg) IVP
3. Glycopyrrolate 0.1–0.3 mg IVP
4. Propofol/ketamine (1 mg/mL) infusion @ 80–150 mcg/kg/min propofol after 0.8–1.4 mg/kg bolus at induction

Comment: Low likelihood of apnea, limited hemodynamic effects, modest analgesia, and possible ketamine side effects

Option B:

1. Nasal cannula oxygen 2–4 L
2. Propofol infusion (120–200 mcg/kg/min) after 1–2 mg/kg induction bolus
3. Consider addition of fentanyl 25–100 mcg for highly stimulating procedures

Comment: high doses of propofol as sole agent may induce apnea when bolused, no analgesic properties, sole agent provides clean anesthetic, and rapid emergence

Option C:

1. Preoxygenate with 100% O₂ via facemask
2. Deliver high FiO₂ via Mapleson circuit attached to patient with lubricated nasal trumpet
3. Propofol/remifentanyl (2 mcg/mL) infusion at 60–120 mcg/kg/min propofol after 0.6–1 mg/kg bolus

Comment: high likelihood of intermittent apnea, profound analgesia and blunting of reflexes, may be best suited to chronic opioid or benzodiazepine users or patients undergoing painful procedures with high anesthetic requirements, and greater hemodynamic side effects than A or B

Table 25.3 Trauma blood protocol.

- O-neg × 2 units, then
- O-pos if
 - o Male
 - o Obviously non-child bearing age
- Continue with O-neg

Table 25.4 Massive transfusion protocol.

- Thaw 4 units FFP and 1 unit Cryoprecipitate
- Crossmatch 6 units PRBC and 1 unit Platelets
- Deliver to OR
 - Cooler #1: 4 units PRBC
 - Cooler #2: 2 units PRBC + 3 units FFP
 - Bucket: Cryoprecipitate + Platelets
- Continue to replenish coolers/bucket until told to stop

Table 26.1 Classification of peripheral nerves.

Fiber class	Diameter (μm)	Myelin	Conduction velocity (m/s)	Innervation	Function
A alpha	12–20	+++	75–120	Afferent to skeletal muscle	Motor and reflexes
A beta	5–12	+++	30–75	Afferent from cutaneous mechanoreceptors	Vibration, light touch and pressure
A gamma	3–6	++	12–35	Efferent to muscle spindles	Muscle tone
A delta	1–5	++	5–30	Afferent pain and thermoreceptors	“Fast”, sharp, intense, lancinating pain, touch and temperature
B	<3	+	3–15	Preganglionic sympathetic efferent	Autonomic function
C	0.2–1.5	–	0.4–2.0	Afferent pain and thermoreceptors	“Slow”, dull, burning, aching pain, touch, pressure, temperature, postganglionic autonomic

Abnormal Pain Descriptor Definitions

Allodynia	The perception of pain by a stimulus that is not normally painful
Hyperalgesia	The enhanced perception of pain by a normally painful stimulus
Dysesthesia	Abnormal sensations experienced in the absence of stimulation
Paresthesia	An abnormal sensation (e.g. burning, pricking, tickling, or tingling)

Table 26.3 Common oral opioid pharmacodynamics and dosing.

Opioids	Half-life	Duration (h)	Equianalgesic oral doses (mg)	Initial dose (mg)	Dosing interval (h)
Codeine	3	3–4	80	30–60	4
Hydromorphone	2–3	2–3	2	2–4	4
Hydrocodone	1–3	3–6	10	5–7.5	4–6
Oxycodone	2–3	3–6	7	5–10	6
Methadone	15–30	4–6	10–20	20	6–8
Morphine	2–3.5	3–4	10	10–30	3–4
Propoxyphene	6–12	3–6	43–45	100	6
Tramadol	6–7	3–6	40	50	4–6

Table 27.3 Suggested medical therapies for hypertension.

Mild to moderate hypertension	• Beta blockers (labetalol, esmolol, metoprolol) • Calcium channel blockers (nicardipine) • Nitro paste • Hydralazine
Severe or refractory hypertension (consider intra-arterial BP monitoring)	• IV antihypertensive infusions – Nicardipine – Nitroglycerine – Nitroprusside

Table 27.4 Causes of postoperative tachycardia.

Noxious stimuli	• Pain, anxiety • Endotracheal tube • Distended bladder
Physiologic derangements	• Acidosis • Hypoxemia • Hypotension and hypovolemia • Hypoglycemia • Increased intracranial pressure • Myocardial ischemia
Medications	• Beta adrenergic vasopressors • Dopamine • Dobutamine • Bronchodilators
Anesthetics	• Ketamine • Isoflurane

Table 27.5 Common causes of postoperative bradycardia.

Medications	• Neostigmine, edrophonium • Phenylephrine, norepinephrine • Opioids • Succinylcholine • Beta blockers • Local anesthetics • Ganglionic blockers • High spinal/epidural anesthesia
Physical causes	• Carotid sinus massage • Valsalva maneuver • Gagging • Rectal exam • Increased ocular pressure • Distended bladder • Stimulation of pharynx
Metabolic derangements	• Severe acidemia • Hypoxemia

Table 27.6 Causes of postoperative mental status changes.

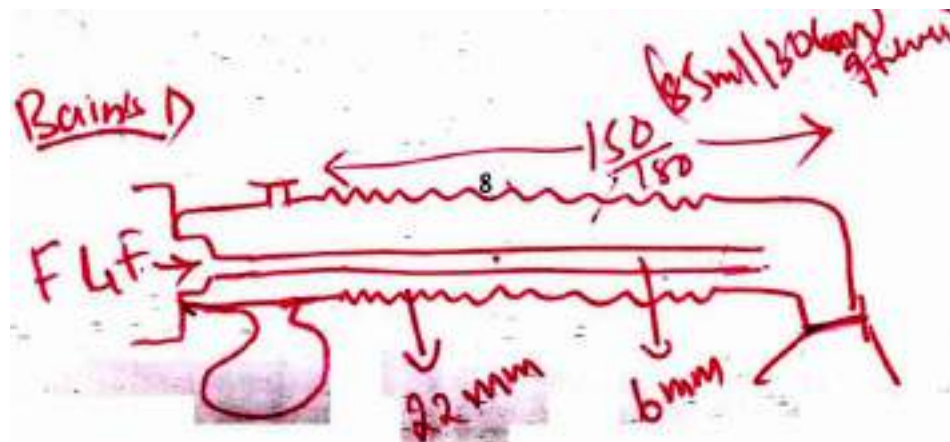
Hypoxemia
Metabolic derangements
Cerebral hypoperfusion
Extremes of age
Emotionally significant operations
Presence of intraoperative recall
Scopolamine or atropine
Substance abuse
Pain, nausea, pruritus

Characteristic	Type of Fluid
Most Physiological	RL
Rich in Na	NS,DNS
Rich in K	Isolyte P,M
Correct Acidosis	RL, Isolyte-P
Corrects Alkalosis	Isolyte G
Avoided in Liver Failure	RL, Isolyte G
Cautious use in RF	RL, Isolyte P,G,E
Glucose Free	NS,RL
Sodium Free	Dextrose 5% 10%
Potassium Free	NS, DNS, Dextrose

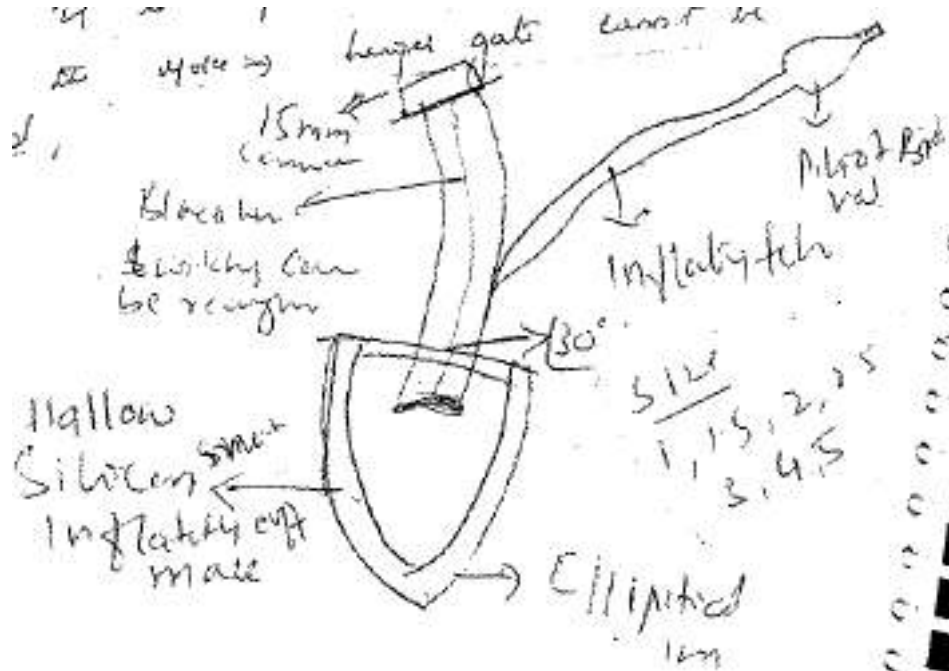
Rapid Assessment of Airway: FUN - M - MAPing with MOANS, RODS, BANGS, Assessment of risk factors for aspiration & difficulties for removal of airway maintenance device.

Assessment	Test
Face	Unusual or deformed facies, any obvious abnormality in face, beard, Syndrome - Pierre-robin Syndrome, klippel-Feil, Goldenhar and Fetal Alcohol.
Upper Incisors	Absent, Loose Protruding
Nose	Nasal Patency
MMAP"ing	Mallampati Class III & IV.
	Measurements: 3-3-1; •Thyromental span = < 3 fingers breath, Mouth Opening = < 3, Jaw Protrusion = < 1 cm or upper lip bite test.
	A-O extension (in absence of C-Spine precautions), Inability to adequately to flex & extend the atlanto-occipital joint.
	Pathology in the mouth & Upper airway. Swellings in and around the airway H/O snoring & stridor.
MOANS (Specific for BMV)	Mask Seal, Obesity/Obstruction, Age > 55, No teeth, Stiff lungs
RODS (Specific for supraglottic devices)	Restricted mouth opening, Obstruction in upper airway, Disrupted upper airway e.g trauma, intra-oral burns, Stiff lungs (Poor compliance)
BANG (specific for surgical airway)	Bleeding tendency, Agitated patient, Neck scarring and flexion deformity, Growth or vascular abnormalities in the region of the surgical airway
Factors for higher risk of aspiration.	NPO status & Full stomach, GE Reflex, Pregnancy, Obesity Hiatus Hernia, presence of Ryle's tube.
Difficulties during and after removal of airway maintenance device.	Any cause encountered as above

Bain's Circuit:



Classic LMA:



Intubating LMA:

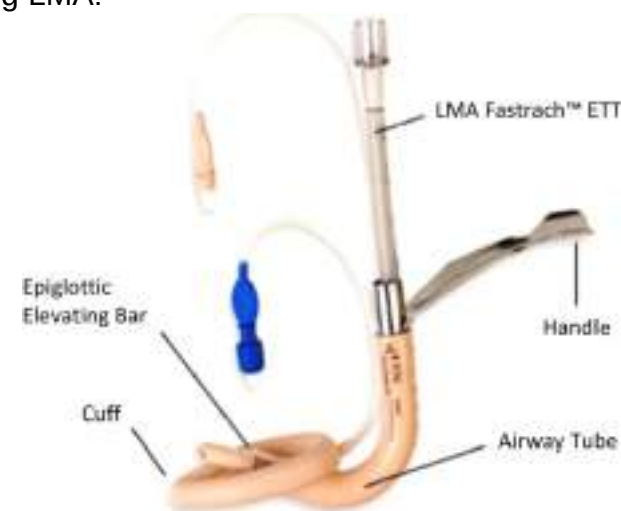
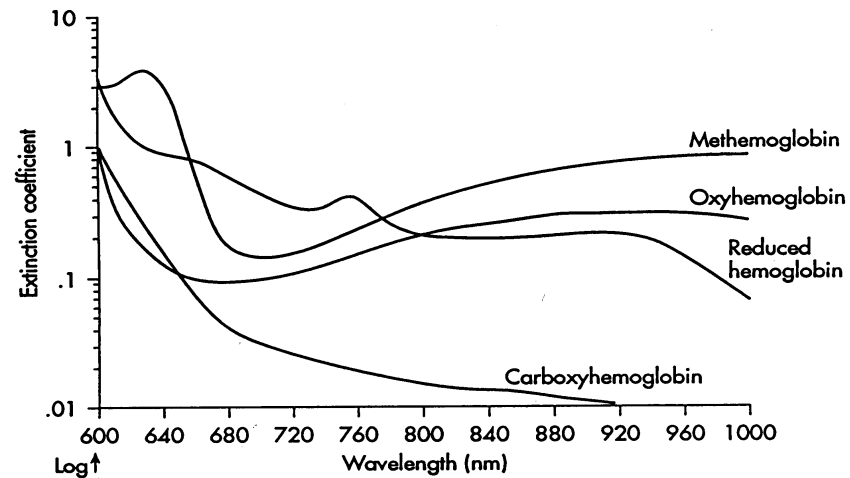
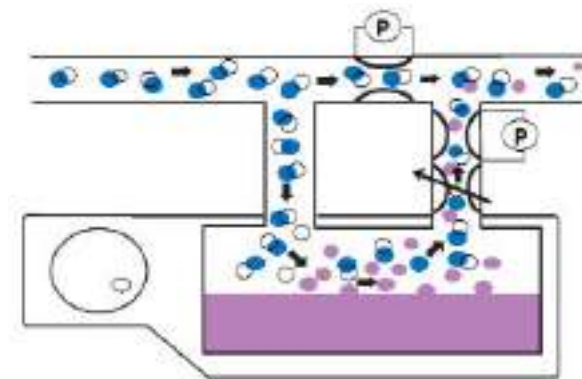
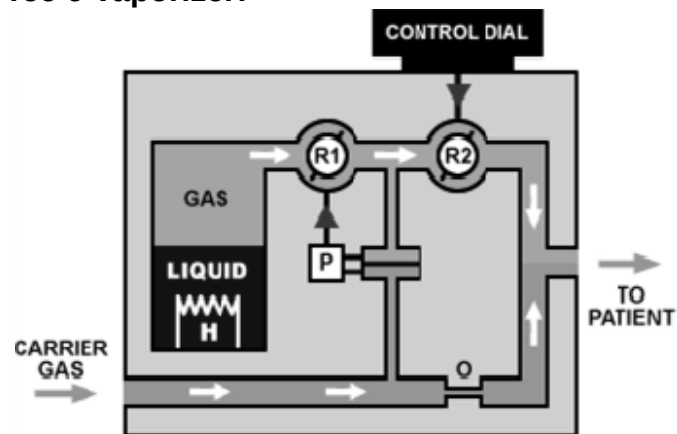


Fig. 8. Laryngeal mask airway LMA Fastrach. LMA ET Tube is placed in the airway tube. The tip of the ET tube protrudes under the epiglottic elevating bar. (Courtesy of LMA North America, Inc; with permission.)

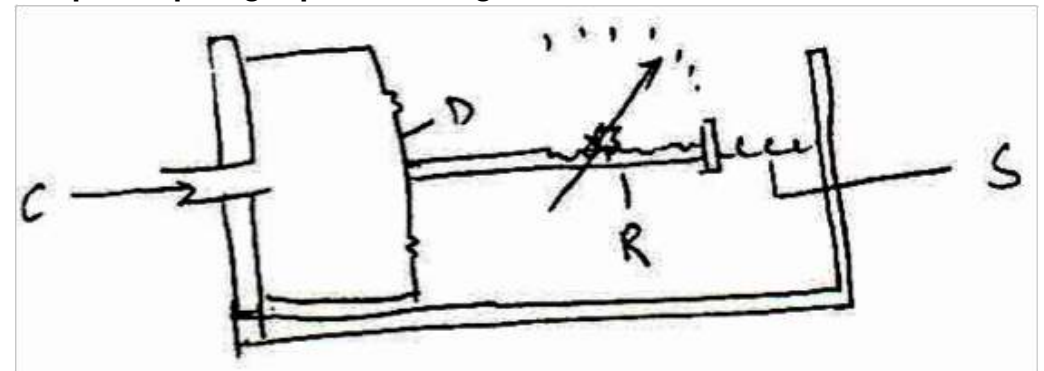
iGel:



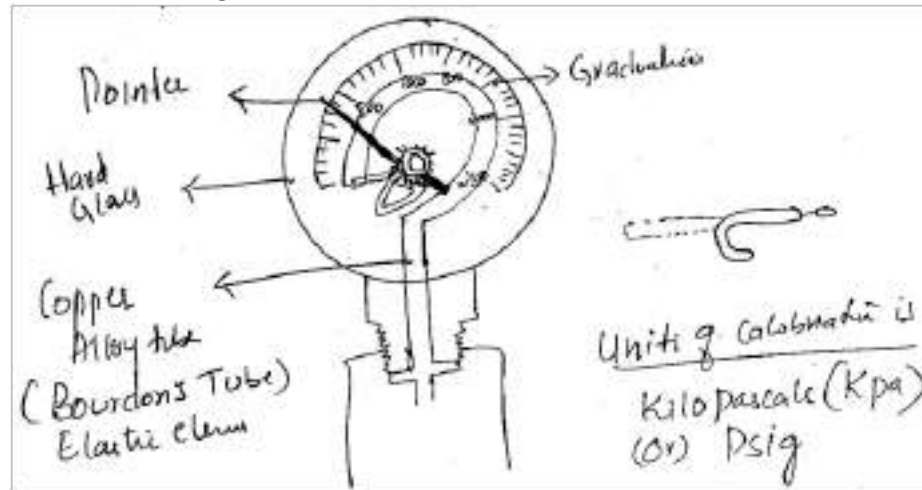
Fig. 19. i-gel supraglottic airway device. (Courtesy of i-gel Intersurgical Ltd, Wokingham, Berkshire, UK; with permission.)

Pulse Oximeter Graph:**Aladin Cassette:****Tec 6 Vaporizer:**

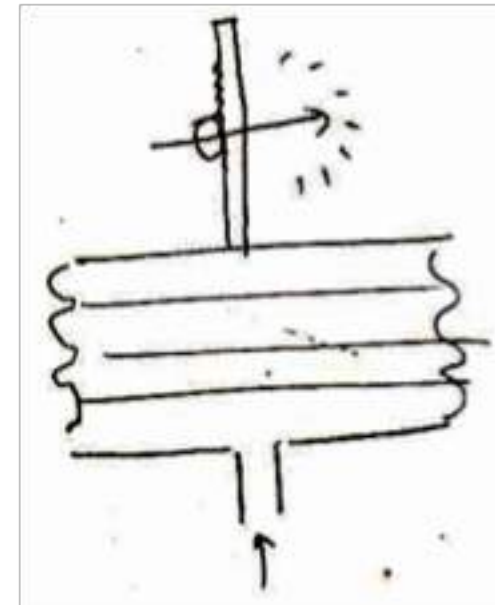
- H-Heating element
- O-Orifice
- P-Differential Pressure Transducer
- R1-Resistor
- R2-2nd Resistor

Simple diaphragm pressure regulator:

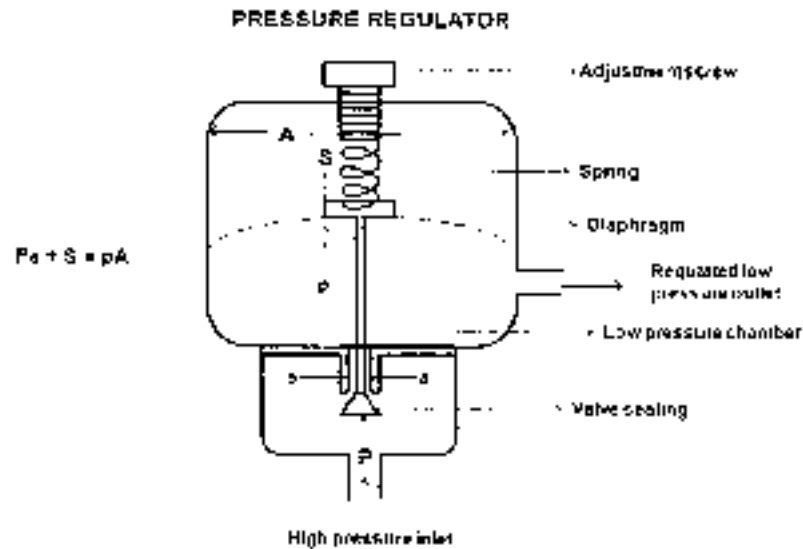
Bourdon Gauge:



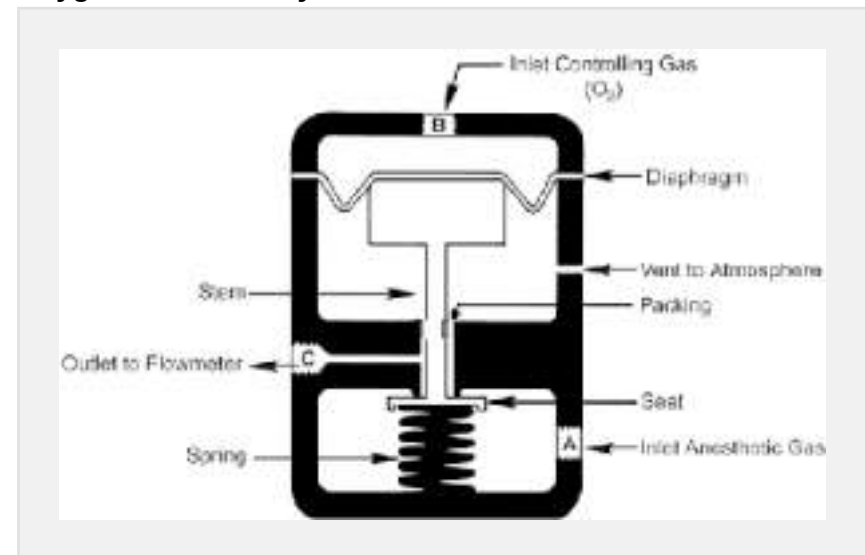
Low Pressure Aneroid Gauge:

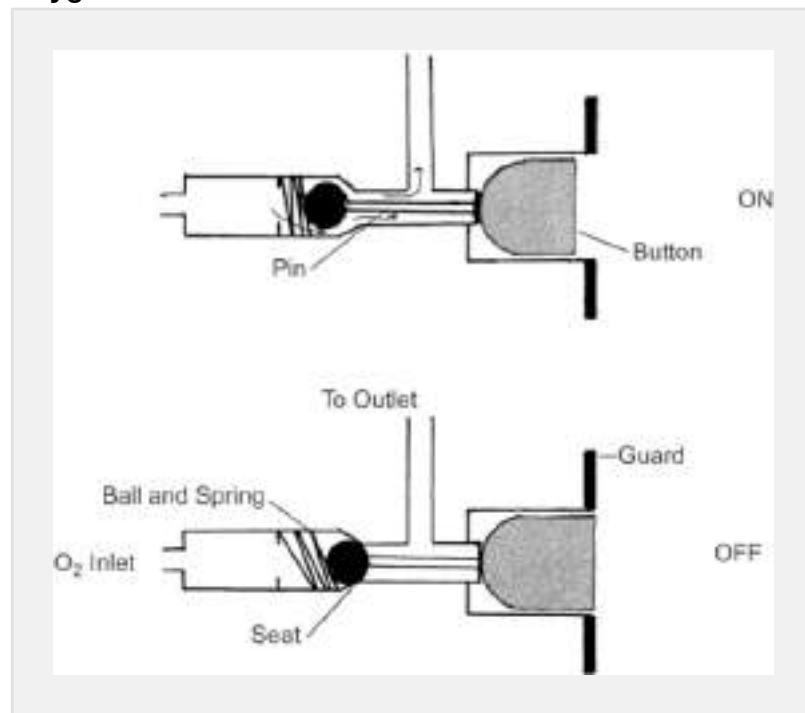


Pressure Regulator:

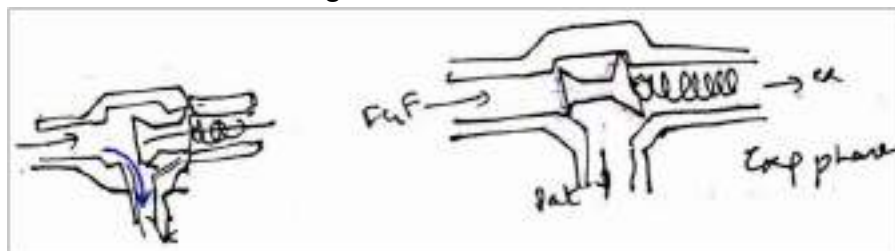
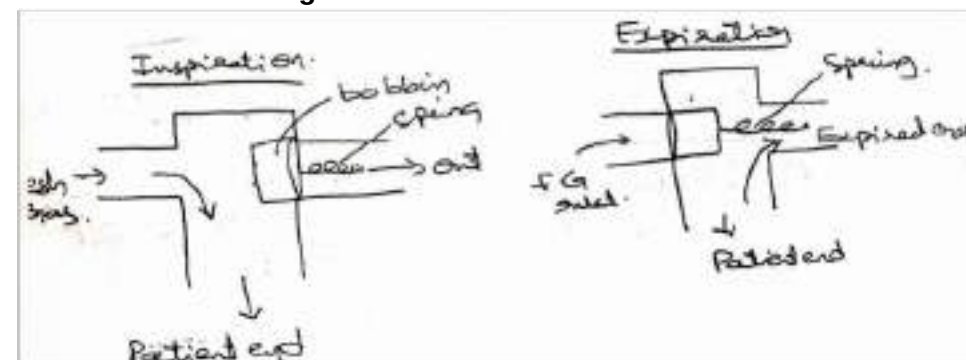


Oxygen failure safety device:

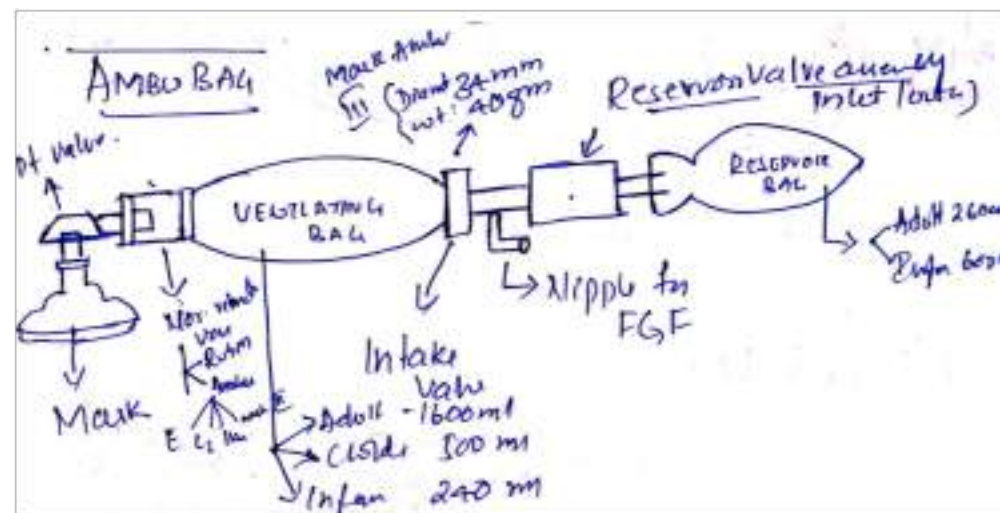


Oxygen Flush:

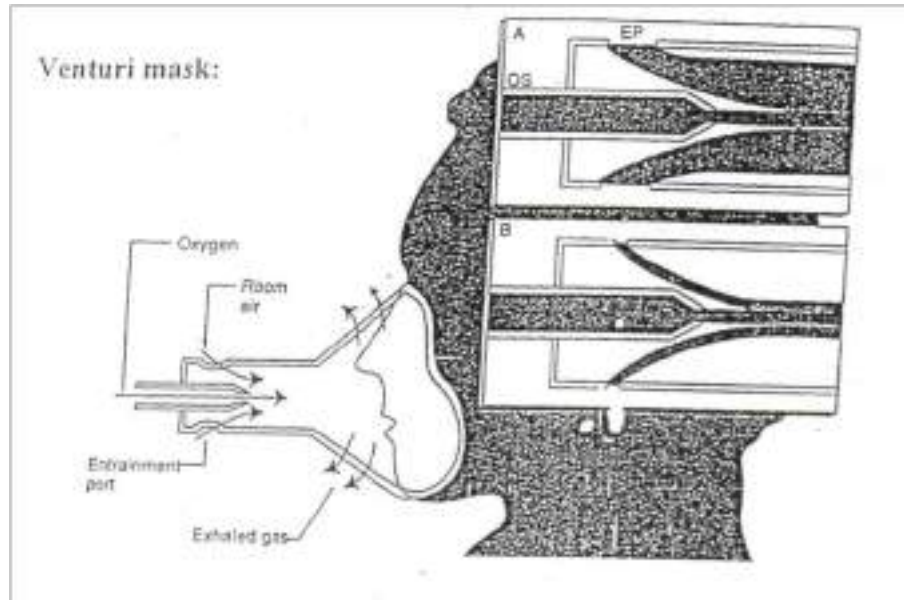
Flow will be 35-75 L/min.
Pressure of 45 to 55 psi.

Ruben Non Rebreathing valve:**Ruben Non-breathing valve:**

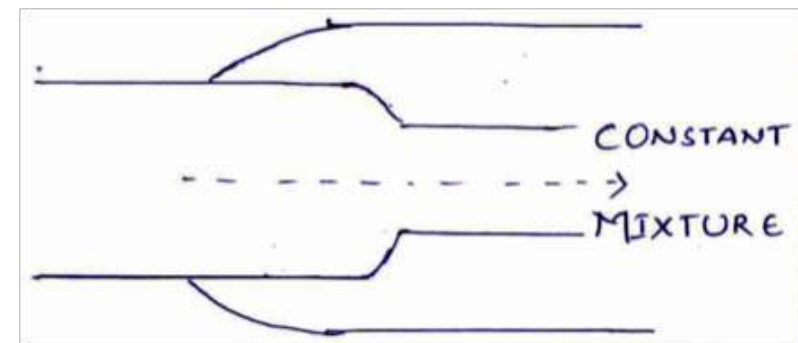
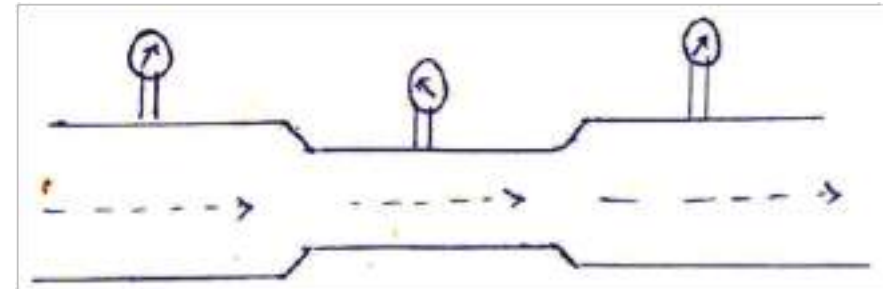
- Ruben Non rebreathing valve
- Inspiration: 0.8 cm of H₂O Inspiratory resistance
- Expiration: 1 cm of water expiratory resistance

AMBU BAG:

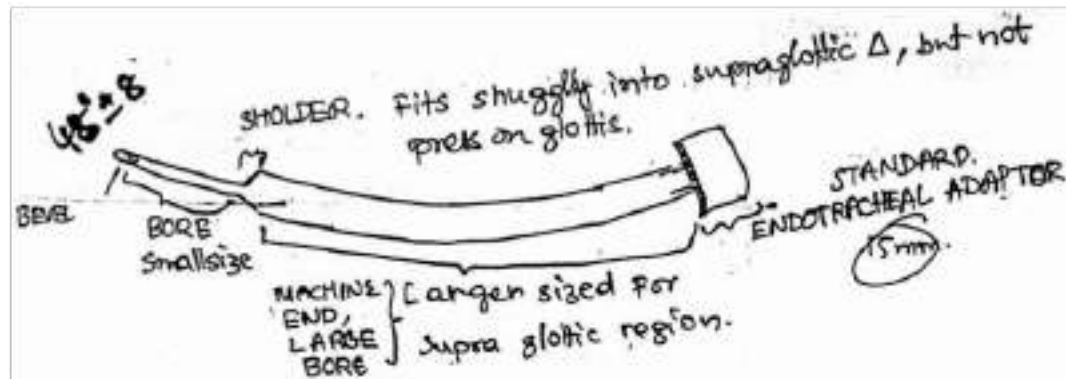
Venturi Mask:

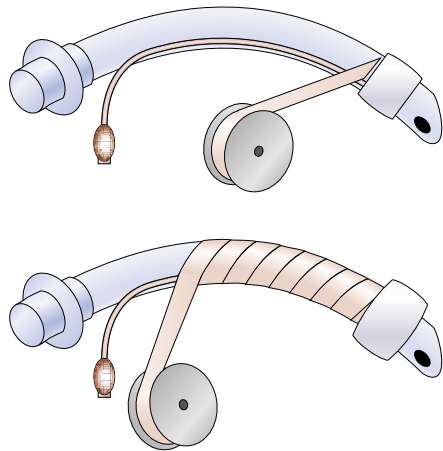


Bernoulli's Principle:



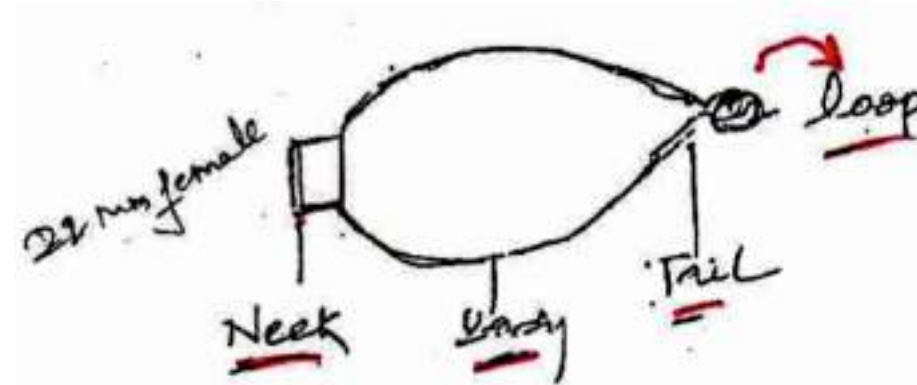
Parts:



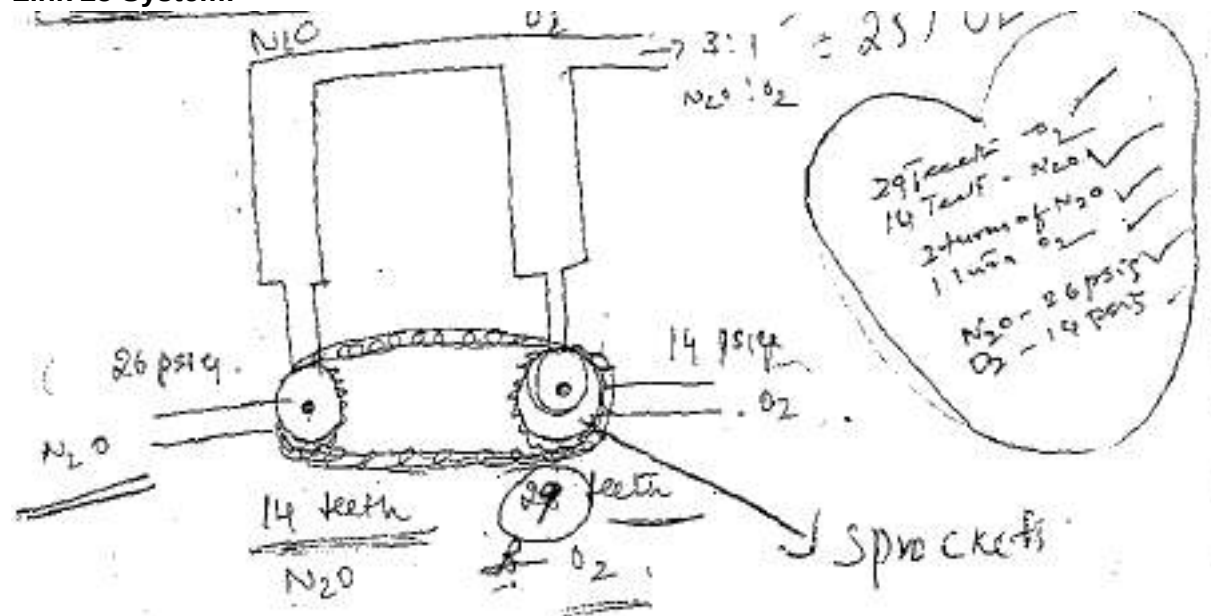


Cuff wrapping technique

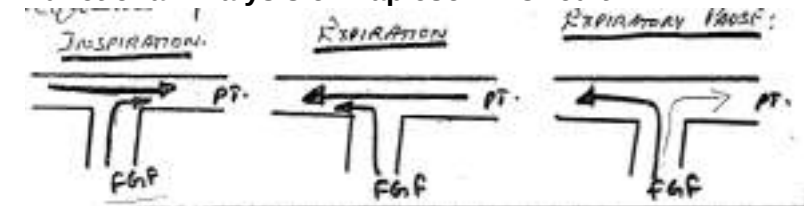
Reservoir Bag:



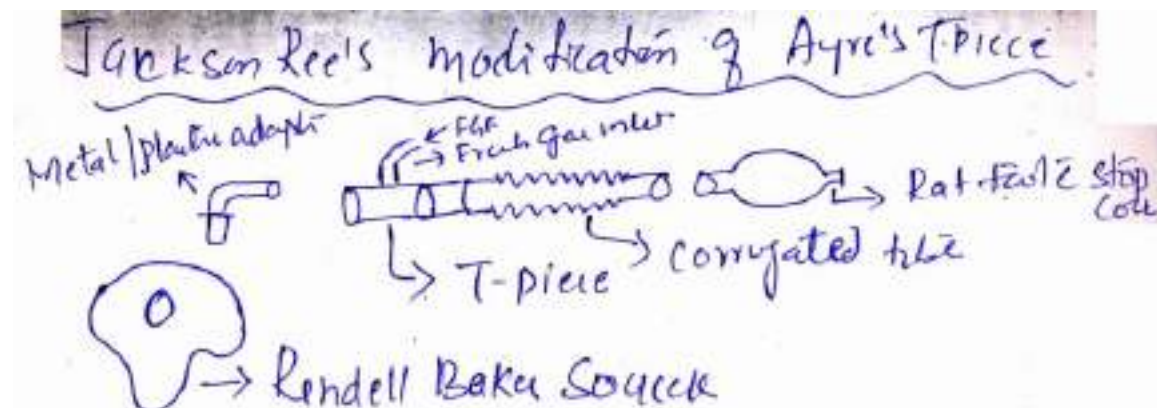
Link 25 System:



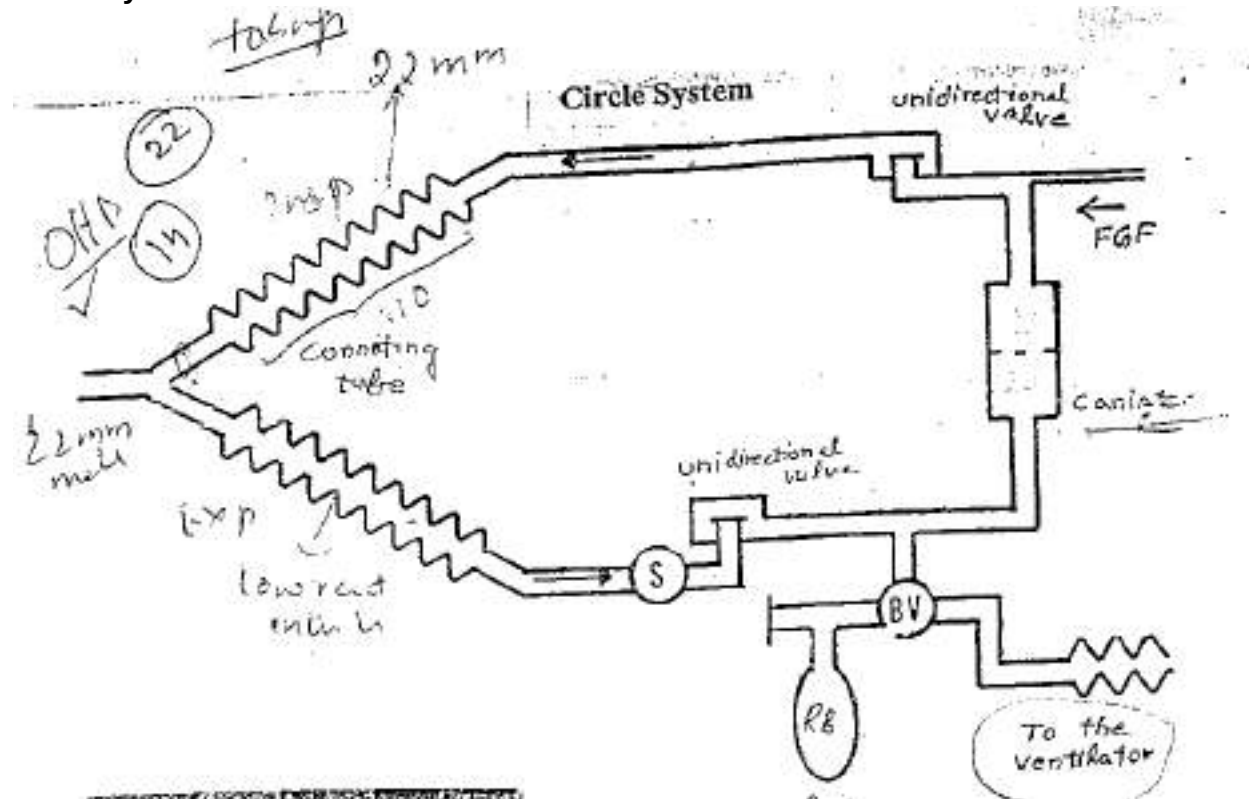
Functional Analysis of Mapleson E Circuit:



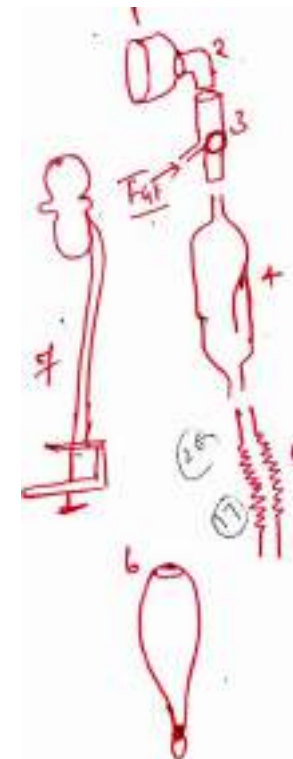
Jackson Rees Circuit:



Circle System:



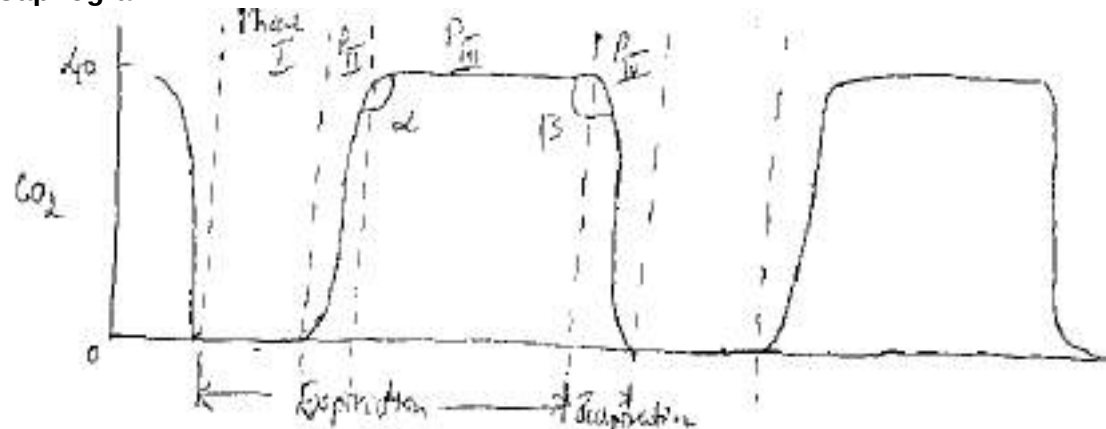
Waters To & Fro:



Components of the circle system include:

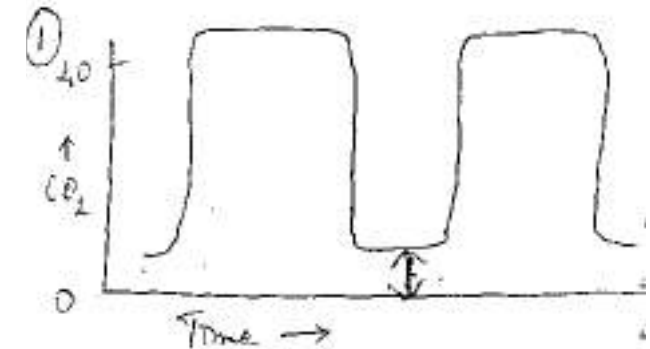
- Soda lime canister,
- Unidirectional valves,
- Fresh gas entry,
- Y-piece.
- Reservoir bag.
- Relief valve,
- Low resistance interconnecting tubing.

Capnogram:



Clinical Application:

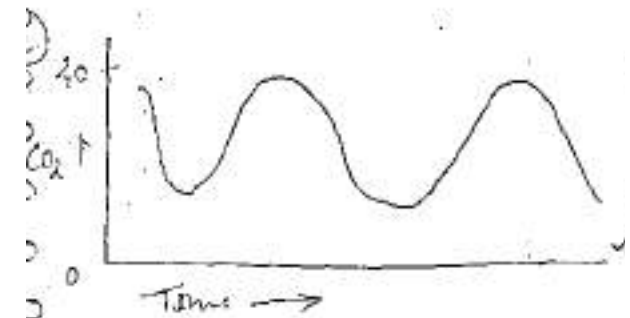
I. Elevated Base Line:



Seen in:

- Rebreathing
- Incomplete expiratory valve
- Exhausted absorbant
- Insufficient fresh gas flow.
- Deliberate addition of FGF.

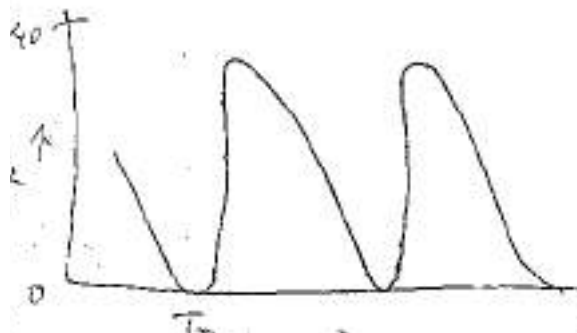
II. Elevated base & low rounded peak:



Seen in:

- Too low rates of sampling with a side stream.

III. Absence of Plateau:



Seen in:

- Contamination of the expired sample gas by FGF by placing the sampling unit near the fresh gas inlet.
- Too high rate of sampling.

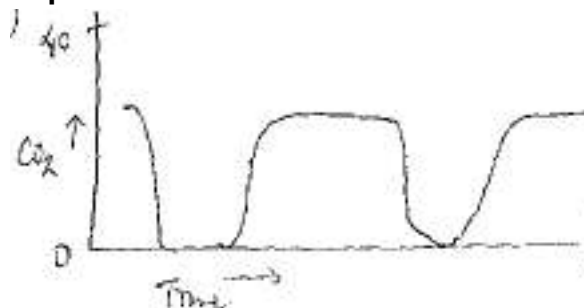
IV. Slanting & prolongation of the expiratory upstroke:



Seen in:

- Obstruction to the ET-tube
- Airway obstruction - E.g. COPD, bronchospasm
- Acute Asthma

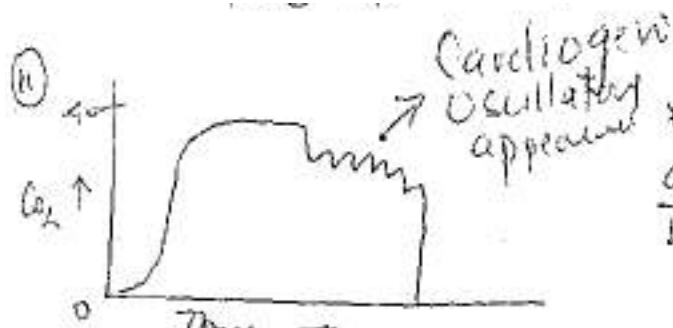
V. Low end tidal CO₂ with good alveolar plateau:



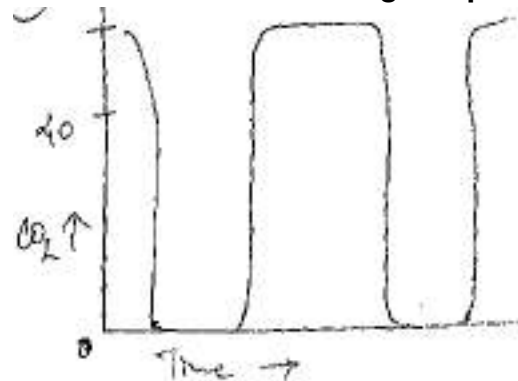
Seen in:

- Hyperventilation
- Increase in dead space ventilation
- Decrease temperature
- Increased muscle relaxation
- Increased depth of anesthesia
- Decreased blood flow:
 - Decreased CO
 - PE

XI. Cardiogenic Oscillations:

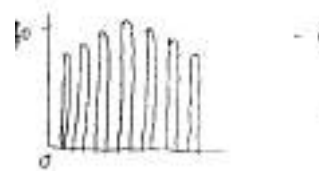


VI. Elevated EtCO₂ with good plateau:

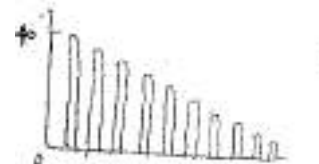


Seen in:

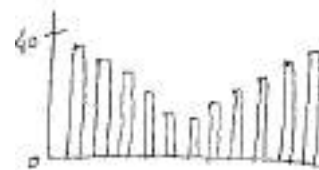
- Hypoventilation
- Increased metabolism
- Increased temperature
- Shivering & convulsions
- Excess of catecholamines



Release of Tourniquet/
Unclamping of a major
muscle.



Exponential decrease in
EtCO₂: Sudden
hypotension, Circulatory
arrest & PE

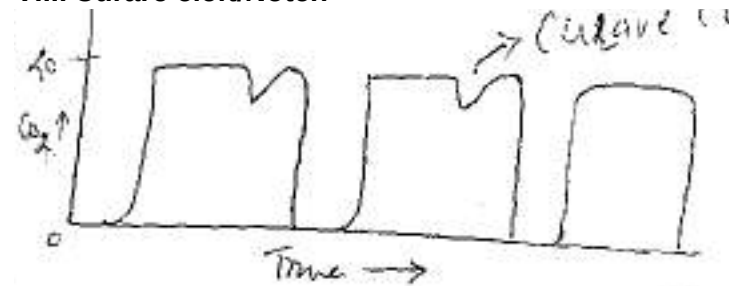


Small air embolus.

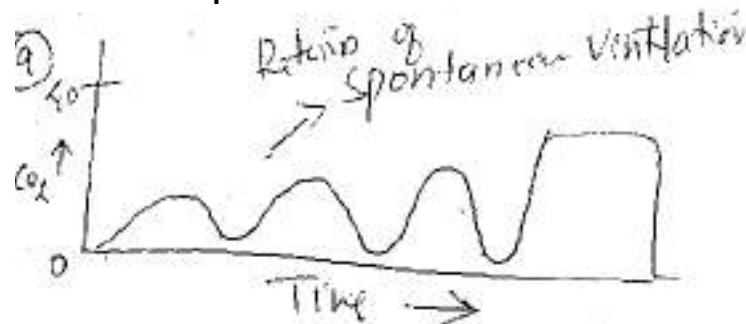
VII. Spontaneous respiratory efforts during mechanical ventilation:



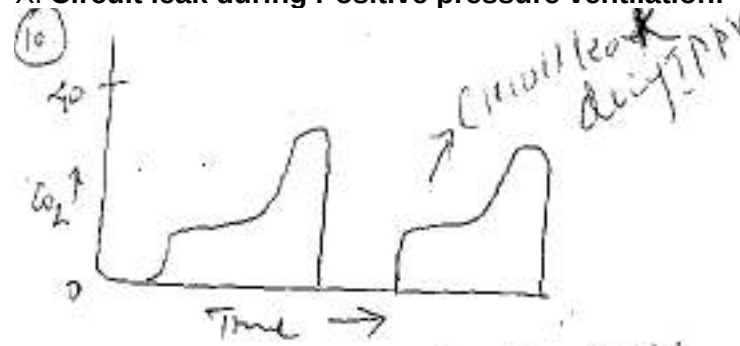
VIII. Curare cleft/Notch



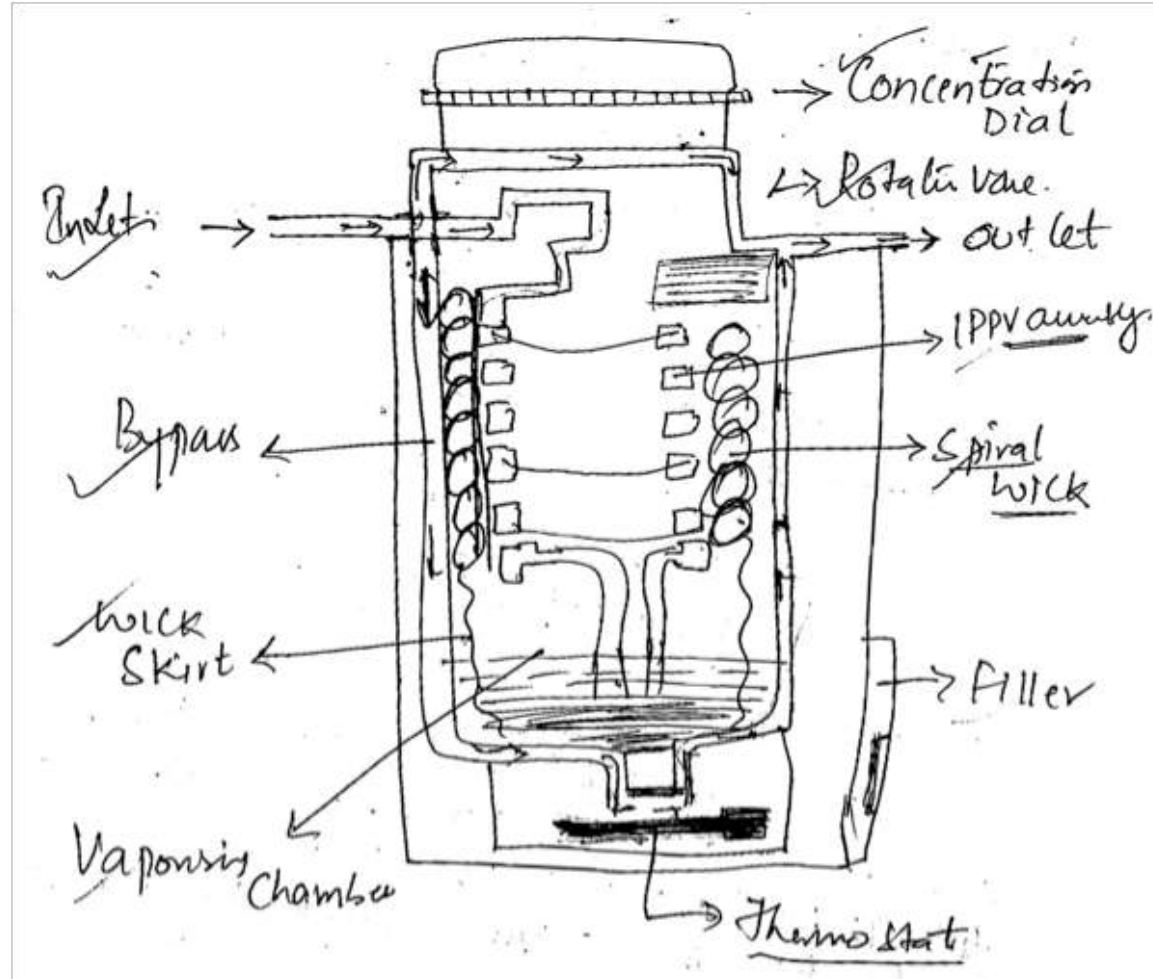
IX. Return of spontaneous ventilation:



X. Circuit leak during Positive pressure ventilation:



TEC 5 Vaporizer:



Waters To & Fro:

