ISA
Indian Society of Anaesthesiologists
Delhi Branch

Eternal Vigilance

ISA DELHI

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INDEX

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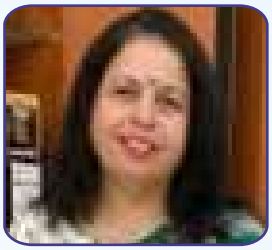

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Dear Delhi ISAians,

Warm Greetings & Wish you a very Happy New Year!

December is a month of two shades, one gives hope of new year and other holds the memories from. ISA Delhi branch performed stupendously throughout the year 2024, winning several accolades at all levels. With beautiful memories of 2024 as we prepare to enter into the new year 2025, lets get together again to weave another success story with monthly clinical meetings, YUVACON, ISACON Delhi 2025. We must all encourage our residents, faculty, consultants to actively prepare and participate for all these events. We also plan to organize various events related to awareness about our speciality amongst general public. As responsible citizens we should also work towards making our operation theatres "greener" and contribute towards minimizing our carbon footprints and consequent global warming. I request all institutions to actively participate in all these forth coming events.

Once again wishing everyone a Very Happy, Healthy and Prosperous New Year!

Long Live ISA.

Dr Munisha Agarwal
President Delhi ISA

Vice President (ISA Delhi)

Dear Delhi ISAians,

Warm greetings and wish you a very happy and prosperous 2025.

Delhi ISA has had an incredible 2024 with several academic, social awareness, cultural and sports activities under its banner. Many congratulations to the ISA Delhi Office bearers under the presidency of Dr Lokesh Kashyap for a very successful tenure over the past one year. It was very heartening and encouraging to see Delhi branch and its members receive various awards and recognition at the 71st Annual National Conference ISACON 2024 at Patna.

The new governing council ISA Delhi has planned various activities for the coming year with the guidance of the ISA National. I invite all members to the ISA monthly clinical meet to be held on 23rd Dec 2024 at Aakash Healthcare Dwarka, New Delhi

Best wishes,

Long live ISA

Dr Sonia Wadhawan
Vice President, Delhi ISA

Honorary Secretary (ISA Delhi)

Dear DELHI ISAians,

Greetings from ISA Delhi Headquarters!

The year 2024 is soon coming to an end, I am sure you all must have made amazing memories with your loved ones and attained new heights. It was a remarkable year for ISA Delhi as well as our team had left no stone unturned to glorify the branch with many new initiatives.

My vision as Honorary Secretary of ISA Delhi is very clear. My team took charge in 2023 with the concept of " Unified ISA Delhi" where there should be equal opportunities for ISAians from medical colleges, private practitioners, free lancers and YUVA rising stars. ISA Delhi YUVACON 2024 and ISACON Delhi 2024 were the true reflections of the same.

ISACON Delhi 2024 comprised of 8 well crafted workshops on 27th September at different institutions of Delhi and conference on 28th and 29th September which was largely attended by nearly 500 anaesthesiologists. It was a true amalgamation of academics and socialisation. I thank my organising team, governing council members and seniors for making it a grand success.

I congratulate Dr Munisha Agarwal and Dr Sonia Wadhawan for taking over as President and Vice President of ISA Delhi during a plush ceremony on the auspicious occasion of World Anaesthesia Day on 16th October 2024. I am sure their vision and dedication will add more wings to the journey.

I humbly request all senior anaesthesiologists, teachers and colleagues to keep guiding us with the valuable inputs so as to take ISA Delhi to further heights.

Long Live ISA
Long Live ISA Delhi

Dr Amit Kohli
Honorary Secretary ISA Delhi

Honorary Treasurer (ISA Delhi)

Dear ISA Delhi members,

Greetings from the treasurer's desk.

As we close this year on a high note, I want to extend my heartfelt gratitude to each one of you for making the activities of ISA Delhi this entire year a resounding success.

The ISACON Delhi 2024, held from 27th to 29th September 2024, was a true testament to the collective efforts and dedication of our Governing council. From the engaging sessions to the impactful collaborations, the event showcased the best of what we can achieve together. It is still receiving many accolades from all over India and the ISA Delhi branch has been conferred the award of second-best metro city branch by ISA National during ISACON 2024 in Patna. Your participation, enthusiasm, and contributions have been invaluable in bringing our vision to life.

Looking back at this year, it has been nothing short of eventful. We have navigated challenges, celebrated milestones, and built a strong foundation for the future. As treasurer, I am proud to report that we have managed our resources responsibly, ensuring the sustainability and growth of our initiatives.

On behalf of the ISA Delhi branch, my heartfelt gratitude goes out to all those who have attended the ISA monthly clinical meets in massive numbers. Please keep the spirits high and participate in the forthcoming ISA Delhi activities with similar ardor.

Before I conclude, I would like to remind you that the ISA Delhi branch has its own YouTube channel where you can revisit the recorded versions of newly launched academic series, and soon, we will be expanding to other social media platforms as well. Please subscribe and maximize your engagement there as well.

Thank you all for being valuable members of ISA Delhi.

Long live ISA.
Jai Hind.
With regards,

Dr Abhijit Kumar
Honorary Treasurer

Editor (ISA Delhi)

Dear ISA Delhi Members,

Warm greetings and wish you a very happy and prosperous 2025. It is with immense pleasure that we present the December issue of our ISA Delhi monthly newsletter. It is focused on postgraduate topics with rapid reviews of various important aspects.

We invite all the readers of various hospitals to submit case reports, review articles and other articles for potential inclusion in the newsletter. This year we plan to have a theme-based newsletter. The theme for the month would be communicated and we would encourage active participation from all the members to diversify the content.

We would active suggestions for continuous improvement in our newsletter. We would thank the previous editorial board led by Dr Punnet Khnana and would try to continue the academic vigour in the newsletter.

Best wishes,

Long live ISA

Dr Nishkarsh Gupta
Professor, Department of Onco-Anesthesiology and Palliative Medicine
Editor (ISA Delhi Branch), Delhi ISA

GLIMPSES OF ISACON DELHI 2024

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WORLD ANAESTHESIA DAY ACTIVITIES

ISA Delhi branch celebrated World Anaesthesia Day on October 16th, 2024 at the AIIMS Gymkhana Club in Delhi. The event kicked off around 7 PM with a vibrant cultural program featuring performances by residents, followed by an official handover ceremony. The enthusiasm of the residents truly enhanced the evening.

The cultural segment included a variety of acts such as solo songs, group dances, a skit, and a fashion show, all skillfully anchored by Dr Michell Gulabani and overall coordinated by Dr Geetanjali T Chilkoti. The cultural event commenced with a captivating performance by Dr Ragi Jain, who set the tone for the evening by playing an instrumental song on sarod. Adding to the excitement was a lively foot-tapping song sung by Dr Sravana, whose infectious enthusiasm drew everyone in. His dynamic stage presence and engaging performance had the audience clapping and singing along, creating an atmosphere of joy and celebration.

Two scintillating dance performances by the residents of MAMC and UCMS truly stole the show, captivating the audience with their energy and creativity. The vibrant choreography showcased the talent and dedication of the performers, making it an unforgettable experience. Another highlight of the evening was undoubtedly the skit performed by UCMS residents, which depicted the remarkable journey of Anaesthesiology as a distinguished discipline, earning appreciation from all attendees. The idea for the skit was conceived by Dr Anita Blessy, whose creative vision brought a unique narrative to life.

The fashion show was conceived by Dr Khushboo, whose artistic flair and vision transformed the event into a stunning showcase of creativity and style. The fashion show, themed around a solidarity walk for women's safety, was the show-stopping performance of the evening. All participants donned striking black outfits, making a powerful statement that resonated with the audience. This impactful segment highlighted the importance of women's safety and empowerment, adding a meaningful touch to the celebration. The highlight of the evening was the mesmerizing Bollywood mashup played on guitar by Dr Deepanshu and Dr Faizan. The intricate arrangements and skilled execution left the audience in awe.

WORLD ANAESTHESIA DAY ACTIVITIES

The extempore song performances by the senior teachers like Dr Rakesh Kumar, Dr Anil Misra, Dr Sonia Wadhawan, Dr Sunil, Dr Maitree Pandey were an added highlight of the evening. Their spontaneous musical displays not only showcased their talent but also brought a sense of camaraderie and joy to the celebration. It was a truly memorable part of the event!

Following the cultural program, the official handover ceremony commenced, honoring Dr Munisha Agarwal and Dr Sonia Wadhawan as the President and Vice-President for the year 2024-25. This was followed by the felicitations of senior eminent and emeritus teachers, recognizing their contributions to the field. They both took prestigious ISA Oath.

The World Anaesthesia Day celebration concluded with a festive cake-cutting ceremony and a gala dinner, allowing everyone to come together in a spirit of joy and celebration. It was a fitting end to a wonderful evening!

Overall, the event was a beautiful showcase of talent, collaboration, and community spirit, leaving a lasting impression on all who had attended.

WORLD ANAESTHESIA DAY ACTIVITIES

Evidence-based management of physiologically difficult airway in critically ill patients

Dr Soumya Sarkar
Assistant Professor, Anesthesiology, AIIMS, Kalyani

Critically ill patients in the intensive care unit (ICU) have unique anatomical and physiological concerns that make standard airway management techniques more challenging, collectively referred to as a "physiologically difficult airway". It usually occurs with underlying physiological derangements such as hypoxemia, hypotension, acidosis, or right ventricular failure. It has increased the risk of adverse outcomes, including cardiac arrest, during or immediately after airway management. Effective management requires thorough pre-intubation preparation for a meticulous patient assessment, adequate preoxygenation, hemodynamic support, and deliberate pharmacological selections during induction to ensure the safest possible intubation and minimize adverse outcomes, including hypoxemia and cardiac arrest.

Hypoxemia is one of the most common dreaded challenges in critically ill patients due to diminished oxygen reserves, reduced functional residual capacity (FRC) and increased oxygen consumption due to underlying conditions such as acute respiratory distress syndrome (ARDS), sepsis, or pneumonia. It leads to rapid desaturation during intubation when the ventilation is temporarily interrupted.

In this context, preoxygenation utilizing high-flow nasal oxygen (HFNO), noninvasive positive pressure ventilation (NIPPV), or a non-rebreathing mask for at least three minutes prior to induction and continuous apneic oxygenation through a nasal cannula during the intubation process plays a critical role in mitigating the risk of rapid desaturation.

Another common issue in ICU patients is haemodynamic instability. They are generally volume-depleted, have poor cardiac function, or are on vasopressors, making them particularly susceptible to post-intubation hypotension with administration of sedatives and induction agents, even with positive pressure ventilation. Fluid resuscitation, the use of vasoactive agents such as norepinephrine, and careful titration of induction agents are crucial to maintaining adequate blood pressure during intubation. Ketamine with sympathomimetic effects possesses an advantage over other sedatives. However, etomidate may be considered in patients with myocardial ischemia or elevated intracranial pressure. The etomidate role is limited in patients with adrenal insufficiency or sepsis. Propofol, commonly used in the operating room, should be avoided in critically ill patients with hemodynamic instability.

Acidosis is another physiological challenge. Patients with diabetic ketoacidosis (DKA), septic shock, or respiratory failure have limited physiological reserves and are at high risk for decompensation during intubation. Rapid sequence induction (RSI) is usually practised to minimize the risk of aspiration, which can also worsen the acidosis if ventilation is delayed or inadequate.

Patients with pulmonary hypertension or right ventricular (RV) failure represent a distinct subset of high peri-intubation risks. Positive pressure ventilation with high tidal volumes can exacerbate RV failure by increasing afterload, thereby precipitating cardiovascular collapse.

Evidence-based management of physiologically difficult airway in critically ill patients

Dr Soumya Sarkar
Assistant Professor, Anesthesiology, AIIMS, Kalyani

For alleviating this risk avoidance of high airway pressures during intubation, using lung-protective ventilation strategies with low tidal volumes(6 mL/kg of ideal body weight) with permissive hypercapnia, use of dobutamine or milrinone to maintain or improve RV contractility may be beneficial to preserve cardiac function during intubation.

Post-intubation care, in terms of intense monitoring of hemodynamics, oxygenation, and ventilation parameters immediately after intubation, is crucial to prevent complications. As discussed earlier, early initiation of lung-protective ventilation strategies is essential for patients with preexisting lung injury. The role of continuing optimum sedation and analgesia in critically ill patients to ensure comfort and prevent ventilator dyssynchrony is paramount. Analgesia-first sedation strategies with the application of opioids such as fentanyl or morphine may reduce the requirement of high doses of sedatives and prevent complications.

Managing the physiologically difficult airway in critically ill patients requires a multifaceted approach that addresses the underlying physiological derangements present in these patients with multiple overlapping physiological derangements, resulting in difficulty prioritizing management strategies. Significant challenges persist despite advancements in understanding newer strategies, including optimizing preoxygenation and apneic oxygenation, providing hemodynamic resuscitation, selecting appropriate induction and paralytic agents, and implementing lung-protective ventilation strategies. Developing algorithms and protocols to integrate these considerations to provide a more comprehensive approach to airway management in the ICU is the need of the hour.

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Rapid review physiology for anesthesiologists

Dr Prakash Gondode

Professor, Anesthesiology, Pain and Critical care, AIIMS

- **Red Blood Cells and Haemoglobin:** RBCs are produced in the bone marrow and stimulated by erythropoietin in response to hypoxia, living for about 120 days before macrophages in the spleen and liver remove them. Haemoglobin (Hb) carries four oxygen molecules, significantly enhancing blood's oxygen capacity. When broken down, Hb splits into heme (iron converts to bilirubin) and globin (amino acids).

- **Oxygen-Haemoglobin Dissociation Curve:** The curve has a sigmoid shape due to cooperative binding; as one oxygen binds, the affinity for more increases. A right shift (oxygen release) occurs with decreased pH, increased CO2, and higher temperature, while a left shift occurs under opposite conditions. The Bohr effect shows that increased CO2 and H+ levels facilitate oxygen release, with a P50 value around 3.5 kPa indicating 50% saturation of Hb.

- **Hypoxia:** Hypoxia can be classified as hypoxic (low arterial PO2 from altitude or lung disease), anaemic (normal PO2 but insufficient Hb), stagnant (reduced tissue perfusion in shock), or histotoxic (cells unable to use oxygen, e.g., cyanide poisoning).

- **Oxygen Transport:** Oxygen transport consists of 98.5% bound to haemoglobin and 1.5% dissolved in plasma, with the content formula: $CaO2 = [Hb \times 1.34 \times SaO2] + [PaO2 \times 0.0225]$. The oxygen cascade describes the PO2 decline from inspired air to mitochondria, with the A-a gradient (normally <2 kPa) reflecting oxygen transfer efficiency.

- **Carbon Dioxide Transport:** CO2 is transported as 90% bicarbonate ($HCO3^-$), 5% dissolved in plasma, and 5% bound to proteins. The chloride shift maintains electrical neutrality in RBCs. The Haldane effect states that deoxygenated haemoglobin carries more CO2, aiding removal in the lungs. The CO2 dissociation curve is more linear than the O2 curve.

- **Alveolar Gas Equation:** The formula is $PAO2 = PiO2 - (PaCO2 / R)$, with R ~0.8. The A-a gradient increases in conditions like V/Q mismatch. Factors affecting PiO2 include barometric pressure and altitude, which reduces PiO2, aiding in diagnosing hypoxemia causes.

- **Ventilation-Perfusion (V/Q) Mismatch and Shunt:** The normal V/Q ratio is ~0.8; high ratios indicate good ventilation but poor perfusion, while low ratios indicate the opposite. Shunting is perfusion without ventilation ($V/Q = 0$), and dead space is ventilation without perfusion ($V/Q = \infty$).

- **Respiratory Dead Space:** Anatomical dead space comprises conducting airways (2 mL/kg), while alveolar dead space includes ventilated but unperfused alveoli, increasing in disease. Physiological dead space is the sum of both types.

- **Lung Volumes:** Lung volumes include tidal volume (TV, ~500 mL), residual volume (RV, ~1200 mL), vital capacity (VC, max air exhaled after full inspiration), and functional residual capacity (FRC, volume remaining after normal expiration).

Rapid review physiology for anesthesiologists

Dr Prakash Gondode

Professor, Anesthesiology, Pain and Critical care, AIIMS

- **Lung Compliance:** Compliance indicates the lung's ability to stretch; it decreases in fibrosis and increases in emphysema. Influencing factors include surface tension (reduced by surfactant) and lung elasticity, with clinical relevance in ventilation and pathology.

- **Control of Respiration:** Medullary centres manage inspiration and expiration, while pontine centres adjust the timing of breaths. Central chemoreceptors respond to CO2 and H+, and peripheral chemoreceptors respond to O2 and pH. The Hering-Breuer reflex prevents lung overinflation.

- **Altitude and Diving:** Altitude causes hypoxia due to reduced pressure, with acclimatization increasing ventilation and RBC production. In diving, increased pressure leads to nitrogen absorption, and rapid ascent can cause decompression sickness.

- **Lung Function Measurement:** Spirometry measures lung volumes (e.g., FEV1, FVC), with FEV1/FVC <70% indicating obstructive disease. DLCO assesses gas transfer ability, PEFR measures maximum expiration speed, and body plethysmography measures volumes like residual volume.

- **Effects of Anesthesia on Lung Function:** Anesthesia reduces FRC, leading to atelectasis, and increases V/Q mismatch. Opioids and anaesthetics depress respiratory centre responsiveness to CO2, while mechanical ventilation can increase dead space and worsen V/Q mismatch.

- **Baroreceptors and Control of Blood Pressure:** Baroreceptors in carotid sinuses and aortic arch respond to arterial pressure changes. Increased pressure leads to parasympathetic activation (lowering heart rate), while decreased pressure triggers sympathetic activation (increasing heart rate). Long-term control involves renal blood volume regulation through RAAS and ADH.

- **Cardiac Cycle:** The cardiac cycle consists of atrial systole, isovolumetric contraction, ventricular ejection, isovolumetric relaxation, and ventricular filling. Stroke volume (SV) is the blood volume pumped per beat (SV = EDV - ESV), influenced by preload, afterload, and contractility. Heart sounds include S1 (AV valve closure) and S2 (semilunar valve closure), with a normal ejection fraction (EF) of 60-70%.

- **Coronary Circulation:** Coronary arteries supply blood to the heart muscle mainly during diastole. Blood flow is regulated by local metabolic factors and the autonomic nervous system. Ischemia occurs with reduced coronary blood flow, while coronary steal redistributes blood flow away from ischemic areas.

- **Exercise:** During exercise, heart rate, stroke volume, and cardiac output increase to meet metabolic demands. Minute ventilation rises to expel CO2 and supply O2, with enhanced oxygen delivery due to improved cardiac output and O2 extraction. Lactic acid is produced in anaerobic conditions, contributing to muscle fatigue.

- **Carbohydrate Metabolism:** Glycolysis converts glucose to pyruvate, yielding 2 ATP. Glycogenolysis breaks down glycogen for glucose in the liver and muscles, while gluconeogenesis synthesizes glucose from non-carbohydrate sources in the liver. Insulin lowers blood glucose, whereas glucagon raises it.

- **Starvation:** Starvation progresses through three phases: Phase 1 (0-24 hours) depletes glycogen for brain glucose; Phase 2 (2-3 days) mobilizes fat stores and induces ketosis; Phase 3 (3+ days) increases protein catabolism for gluconeogenesis, causing muscle wasting. Ketosis provides ketone bodies for brain energy, with hormonal changes promoting gluconeogenesis and lipolysis.

- **Nausea and Vomiting:** The vomiting centre in the medulla responds to signals from the chemoreceptor trigger zone (CTZ), vagal afferents, and higher brain centres. The CTZ is sensitive to drugs, toxins, and metabolic disturbances. Common triggers include motion sickness and gastroenteritis. Antiemetics target receptors in the CTZ and vomiting centre.

- **Liver Physiology:** The liver performs metabolism (carbohydrates, fats, proteins), detoxification, bile production, and storage of vitamins/minerals. It stores glycogen and releases glucose through glycogenolysis and gluconeogenesis. The liver synthesizes plasma proteins and converts ammonia to urea, while bile aids in fat digestion and absorption.

- **Gastric Regulation:** Gastric secretion occurs in three phases: the cephalic phase (stimulated by food cues), the gastric phase (triggered by stomach distension), and the intestinal phase (inhibiting gastric secretions). Gastrin increases acid production and gastric motility, with mucus and bicarbonate protecting the stomach lining from acid.

- **Total Parenteral Nutrition (TPN):** TPN is indicated when the gastrointestinal tract cannot be used. It includes carbohydrates, proteins, fats, electrolytes, vitamins, and trace elements. Complications may arise, such as hyperglycaemia and infections, requiring monitoring of blood glucose, electrolytes, liver function, and triglycerides.

- **Acid-Base Balance:** Normal values are pH 7.35-7.45, $PaCO_2$ 35-45 mmHg, and HCO_3^- 22-28 mEq/L. Acidosis can be respiratory (hypoventilation) or metabolic (HCO_3^- loss), while alkalosis can be respiratory (hyperventilation) or metabolic (HCO_3^- retention). Compensatory mechanisms involve lungs for metabolic disorders and kidneys for respiratory disorders.

- **Buffers:** The bicarbonate buffer system is the primary extracellular buffer, maintaining pH by balancing HCO_3^- and H^+. The phosphate buffer system is important in the kidneys, while protein buffers include haemoglobin and plasma proteins. Respiratory compensation controls CO_2, and renal compensation regulates HCO_3^-.

- **Renal Blood Flow:** Renal blood flow is primarily autoregulated, maintaining a constant flow despite systemic blood pressure changes. The glomerular filtration rate (GFR) is about 120 mL/min and is affected by blood flow and filtration pressure. The renin-angiotensin-aldosterone system responds to low blood pressure, while prostaglandins help maintain blood flow during stress.

- **Glomerular Filtration Rate (GFR):** GFR is best measured by inulin clearance but estimated using creatinine clearance. Factors affecting GFR include renal perfusion pressure and oncotic pressure. The filtration barrier prevents large proteins from entering the filtrate. Pathological conditions like acute kidney injury reduce GFR.

- **Renal Handling of Glucose, Sodium, and Inulin:** Glucose is fully reabsorbed in the proximal tubule until a threshold (~180 mg/dL). Sodium reabsorption is regulated by aldosterone and atrial natriuretic peptide. Inulin measures GFR as it is freely filtered and neither reabsorbed nor secreted. Water reabsorption is regulated by ADH.

- **Fluid Compartments:** Total body water is ~60% of body weight, with two-thirds intracellular and one-third extracellular. Plasma osmolality is mainly determined by sodium concentration (~285-295 mOsm/kg). Starling forces regulate fluid exchange between capillaries and interstitial fluid, with oedema arising from various imbalances.

- **Osmoregulation:** Osmoregulation involves osmoreceptors in the hypothalamus regulating ADH release, which promotes water reabsorption in the kidneys. Increased plasma osmolality triggers thirst and ADH secretion, while diabetes insipidus can result from ADH deficiency or insensitivity.

- **Action Potentials:** Action potentials begin with a resting membrane potential of -70 mV, created by the sodium-potassium pump. Depolarization occurs with sodium influx, followed by repolarization with potassium efflux. There are absolute and relative refractory periods, determining the timing of subsequent action potentials.

- **Cerebral Blood Flow:** Cerebral blood flow (CBF) is autoregulated between a MAP of 50-150 mmHg and is influenced by CO2 and O2 levels. Increased CO2 leads to vasodilation, while the blood-brain barrier protects the brain from harmful substances. Cushing's reflex occurs in response to increased ICP.

- **Cerebrospinal Fluid (CSF):** CSF is produced by the choroid plexus, circulates through ventricles and subarachnoid space, and is reabsorbed by arachnoid villi. It provides cushioning and waste removal; hydrocephalus results from excess CSF.

- **Autonomic Nervous System (ANS):** The ANS consists of the sympathetic (SNS) and parasympathetic (PNS) systems. The SNS prepares the body for "fight or flight," while the PNS promotes "rest and digest." Key neurotransmitters include ACh for both systems and norepinephrine for the SNS.

Rapid review physiology for anesthesiologists

Dr Prakash Gondode

Professor, Anesthesiology, Pain and Critical care, AIIMS

- **Child vs. Adult Physiology:** Children have smaller airways, higher respiratory rates, and proportionally larger tongues than adults, increasing the risk of obstruction. They also have a greater surface area-to-weight ratio, leading to higher heat loss.

- **Pregnancy:** Pregnancy causes increased blood volume, cardiac output, and respiratory rate, alongside reduced systemic vascular resistance. Renal function improves, leading to increased GFR and urinary tract infection risk.

- **Placental Transfer:** Gases and nutrients transfer across the placenta via simple diffusion. Lipophilic and low molecular weight substances cross more easily, with the placenta serving as a barrier for harmful substances.

- **Foetal Circulation:** Foetal circulation features the ductus venosus, foramen ovale, and ductus arteriosus, allowing blood to bypass the lungs. At birth, these shunts close, and pulmonary blood flow increases.

- **Aging:** Aging affects the cardiovascular system with increased arterial stiffness and decreased cardiac output. Respiratory function declines with reduced lung elasticity, and renal function decreases with age, affecting drug clearance.

- **Adrenal Gland:** The adrenal cortex produces corticosteroids, including aldosterone for fluid balance and cortisol for the stress response. The adrenal medulla secretes catecholamines (epinephrine and norepinephrine) during stress.

- **Thyroid Gland:** The thyroid gland produces T3 and T4, regulating metabolism and growth. TSH from the anterior pituitary stimulates hormone production, with iodine being essential for synthesis.

- **Eye Physiology:** Aqueous Humor maintains intraocular pressure and drains through the trabecular meshwork. Accommodation for focusing involves ciliary muscles, and phototransduction occurs in the retina's photoreceptors.

- **Endothelium:** The endothelium regulates vasodilation and barrier function, producing nitric oxide (NO) and endothelin. Endothelial dysfunction contributes to diseases like hypertension and atherosclerosis.

- **Portal Circulations:** The hepatic portal system transports nutrient-rich blood from the GI tract to the liver. The hypophyseal portal system connects the hypothalamus and anterior pituitary, influencing hormone regulation.

- **Immune Mechanisms:** Innate immunity is the non-specific first line of defence, involving physical barriers, phagocytes, natural killer cells, and complement proteins. Adaptive immunity is specific and long-lasting, mediated by lymphocytes. B cells produce antibodies, while T cells are involved in cell-mediated immunity. Inflammation is an immune response characterized by increased blood flow and immune cell migration. Cytokines are signalling molecules that regulate immunity and inflammation.

Rapid review physiology for anesthesiologists

Dr Prakash Gondode

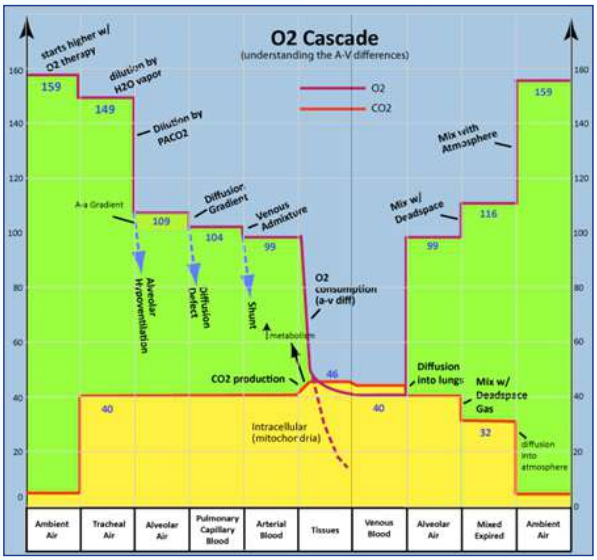
Professor, Anesthesiology, Pain and Critical care, AIIMS

- **Pain Pathways:** Nociceptors are sensory receptors that detect harmful stimuli and transmit pain signals via A-delta fibres for sharp pain and C fibres for dull pain. These signals ascend through the spinal cord's dorsal horn via the spinothalamic tract to the thalamus and cortex. Pain modulation occurs through descending pathways from the brainstem, releasing endogenous opioids to inhibit pain transmission.

- **Muscle Electrophysiology:** Muscle cells maintain a resting membrane potential similar to neurons. Action potentials are triggered by sodium influx during depolarization, followed by potassium efflux during repolarization. At the neuromuscular junction, acetylcholine binds to receptors on muscle membranes, initiating contraction. Excitation-contraction coupling occurs when an action potential releases calcium from the sarcoplasmic reticulum, facilitating interaction between actin and myosin.

- **Reflexes:** The monosynaptic reflex arc involves a direct connection between a sensory neuron and a motor neuron, such as in the patellar reflex. The polysynaptic reflex arc includes one or more interneurons, as seen in the withdrawal reflex. The stretch reflex protects against overstretching, while the Golgi tendon reflex inhibits muscle contraction to prevent excessive tension.

Figures:



1. Oxygen cascade

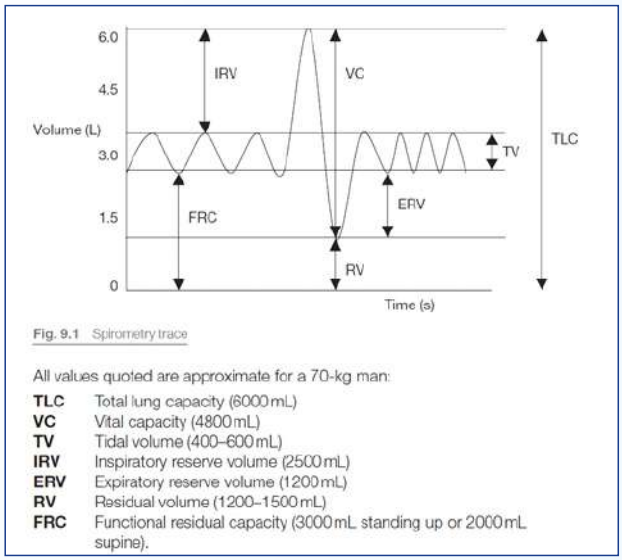


Fig. 9.1 Spirometry trace

All values quoted are approximate for a 70-kg man:

TLC	Total lung capacity (6000 mL)
VC	Vital capacity (4800 mL)
TV	Tidal volume (400–600 mL)
IRV	Inspiratory reserve volume (2500 mL)
ERV	Expiratory reserve volume (1200 mL)
RV	Residual volume (1200–1500 mL)
FRC	Functional residual capacity (3000 mL standing up or 2000 mL supine).

2. Spirometry trace

Rapid review physiology for anesthesiologists

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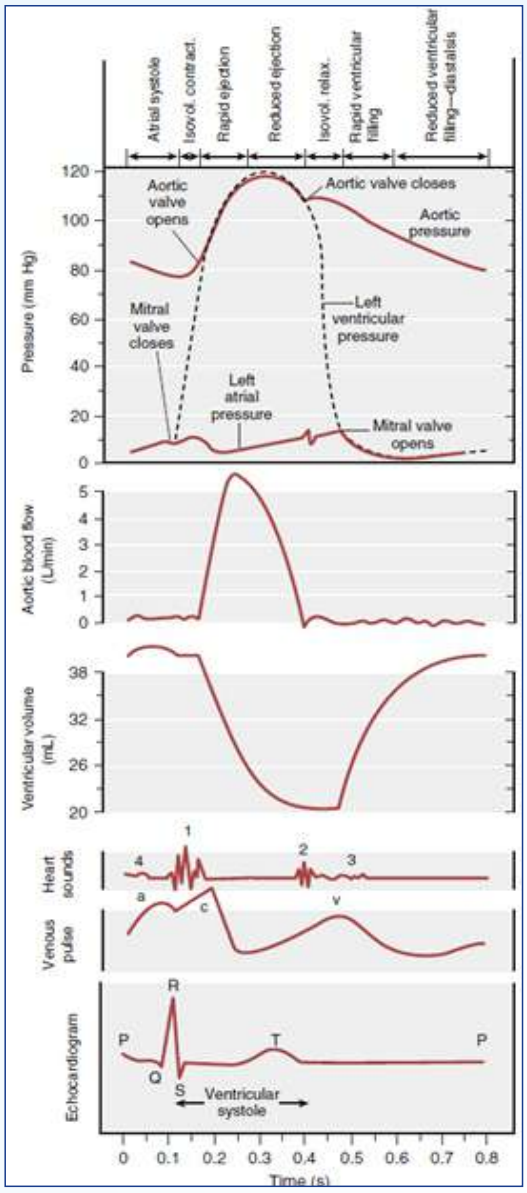
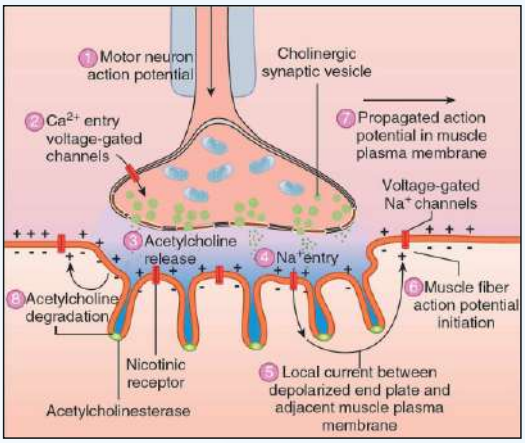
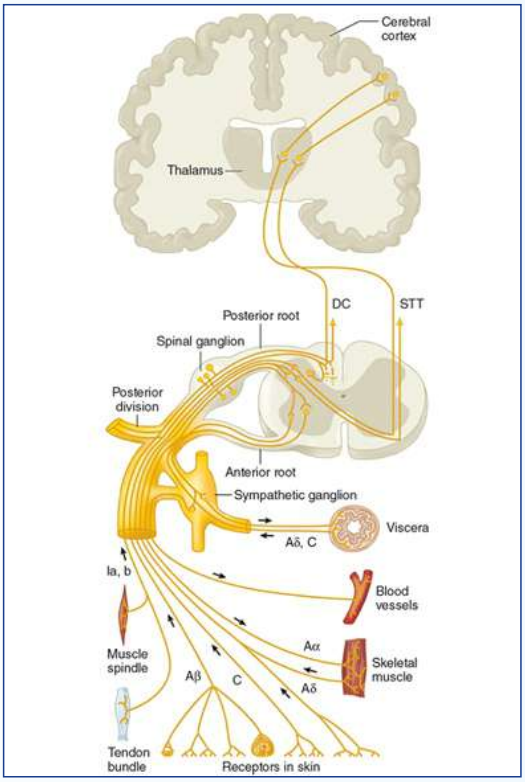


3. Cardiac cycle

4. Major activities of different parts of the renal tubule



5. Neuromuscular junction



6. Pain Pathway

Table: Primary changes and compensatory mechanisms in acid-base disorders

Primary disturbance	Initial imbalance	Compensatory response	Compensatory mechanism	Expected level of compensation
Metabolic acidosis	↓ HCO_3^-	↓ PCO_2	Hyperventilation	1.2 mmHg decrease in PCO_2 for each 1 mmol/L decrease in HCO_3^- (minimum PCO_2 of 1.3–1.9 kPa in compensation)
Metabolic alkalosis	↑ HCO_3^-	↑ PCO_2	Hypoventilation	0.7 mmHg increase in PCO_2 for each 1 mmol/L increase in HCO_3^- (PCO_2 should not rise above 7–8 kPa in compensation)
Respiratory acidosis	↑ PCO_2	↑ HCO_3^-		
• **Acute**			Intracellular buffering	1–2 mmol/L increase in HCO_3^- for every 10 mmHg increase in PCO_2
• **Chronic**			Renal: generation of bicarbonate via excretion of ammonium	3–4 mmol/L increase in HCO_3^- for every 10 mmHg increase in PCO_2
Respiratory alkalosis	↓ PCO_2	↓ HCO_3^-		
• **Acute**			Intracellular buffering	1–2 mmol/L decrease in HCO_3^- for every 10 mmHg decrease in PCO_2
• **Chronic**			Renal: decreased reabsorption of HCO_3^-, decreased excretion of ammonium	4–5 mmol/L decrease in HCO_3^- for every 10 mmHg decrease in PCO_2

Must Do's before we practice Obstetric Anaesthesia

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Obstetric anaesthesia refers to peripartum anaesthetic and analgesic activities performed during

- Labor
- Vaginal delivery
- Caesarean delivery
- Removal of retained placenta
- Postpartum tubal ligation.

The intended patient population includes, but is not limited, to intrapartum and postpartum patients with uncomplicated pregnancies or with common obstetric problems.

Physiological Changes in Pregnancy and its Anaesthetic Implications

Systems	Changes in pregnancy	Implications
Nervous system	↓MAC of inhalational anaesthetic	Modify dose of anaesthetics
	↓Local anaesthetic requirement	Pharmacological sympathectomy is detrimental
	Dependence on the sympathetic system for maintaining haemodynamic stability	
Cardiovascular system	↑HR, SV, CO	Difficulty in estimating blood loss
	↓SVR	Left uterine displacement
	Aortocaval compression	
Haematological system	Disproportionate increase in plasma volume	Physiological anaemia
	Hypercoagulability	Transfusion trigger and blood volume replacement
		Thromboembolic events
Airway and respiratory system	Increased mucosal vascularity and oedema	Difficult intubation
	↑Alveolar ventilation and oxygen consumption	Raised ICT during intubation
	Respiratory alkalosis	Epistaxis
	↓FRC	↑Oxygen requirement
		Rapid desaturation
		Hyperventilation not tolerated
		Decreased reserve for gas exchange
Gastrointestinal and hepatobiliary	↑Intragastric pressure and ↓tone of LOS	RSI
	↑Placental ALP	Aspiration prophylaxis
	↑Gall bladder volume and ↓contractility	Mimics obstructive pathology
		↑Chances of gall stone disease

Perianesthetic Evaluation and Preparation

Perianesthetic evaluation and preparation include

1. A focused history and a physical examination,
2. An intrapartum platelet count
3. A blood type and screen
4. Perianesthetic recording of fetal heart rate patterns.

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Aspiration Prevention

Aspiration prevention includes (1) clear liquids, (2) solids, and (3) antacids, H2-receptor antagonists, and metoclopramide.

Clear Liquids.

The uncomplicated patient undergoing elective surgery (e.g., scheduled cesarean delivery or postpartum tubal ligation) may have moderate amounts of clear liquids up to 2 h before induction of anesthesia.

Solids.

The patient undergoing elective surgery (e.g., scheduled cesarean delivery or postpartum tubal ligation) should undergo a fasting period for solids of 6 to 8 h depending on the type of food ingested (e.g., fat content)

Antacids, H2-receptor Antagonists, and Metoclopramide.

Before surgical procedures (e.g., cesarean delivery or postpartum tubal ligation), timely administration of nonparticulate antacids, H2-receptor antagonists, and/or metoclopramide for aspiration prophylaxis should be done.

Neuraxial Analgesia and Trial of Labor after Prior Cesarean Delivery.

Analgesia/Anesthetic Techniques:

Considerations for analgesic/anesthetic techniques include (1) early insertion of a neuraxial (i.e., spinal or epidural) catheter (2) continuous infusion epidural (CIE) analgesia, (3) epidural local anesthetics combined with opioids, (4) higher versus lower concentrations of local anesthetics, (5) single-injection spinal opioids with or without local anesthetics, (6) pencil-point spinal needles, (7) CSE analgesia, and (8) patient-controlled epidural analgesia (PCEA).

Recommendations for Anaesthetic Care for Labor and Vaginal Delivery

- The option of neuraxial analgesia should be provided in early labor (i.e., less than 5 cm dilation). Consider early placement of a neuraxial catheter that can be used later for labor analgesia or for anesthesia in the event of operative delivery.
- When a continuous epidural infusion of local anesthetic is selected, an opioid may be added to reduce the concentration of local anesthetic, this improves the quality of analgesia, and minimize the motor block.
- CSE techniques may be used to provide effective and rapid onset of analgesia for labor.
- PCEA may be used with or without a background infusion.

Removal of Retained Placenta

Techniques for removal of retained placenta include

1. Anesthetic techniques for removal of retained placenta and
2. Nitroglycerin for uterine relaxation.

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Nitroglycerin may be used as an alternative to terbutaline sulfate or general endotracheal anesthesia with halogenated agents for uterine relaxation during removal of retained placental tissue.

- If an epidural catheter is in place and the patient is hemodynamically stable, epidural anesthesia can be used
- Hemodynamic status should be assessed before administering neuraxial anesthesia.
- Aspiration prophylaxis
- Sedation/analgesia should be carefully titrated due to the potential risks of respiratory depression and pulmonary aspiration during the immediate postpartum period.
- In cases involving major maternal hemorrhage with hemodynamic instability, GA with an endotracheal tube may be considered in preference to neuraxial anesthesia.

Anaesthetic Care for Cesarean Delivery

General, Epidural, Spinal, or CSE Anesthesia.

- The decision to use a particular anesthetic technique for cesarean delivery should be individualized, based on anesthetic, obstetric, or fetal risk factors (e.g., elective vs. emergency)the preferences of the patient, and the judgment of the anesthesiologist.
- Uterine displacement (usually left displacement) should be maintained until delivery regardless of the anesthetic technique used
- Consider selecting neuraxial techniques in preference to GA for most cesarean deliveries
- If spinal anesthesia is chosen, use pencil-point spinal needles instead of cutting-bevel spinal needles.
- For urgent cesarean delivery, an indwelling epidural catheter may be used as an alternative to initiation of spinal anesthesia.
- GA may be the most appropriate choice in some circumstances (e.g., profound fetal bradycardia, ruptured uterus, severe hemorrhage, severe placental abruption, umbilical cord prolapse, and preterm footling breech).

IV Fluid Preloading or Coloading.

- IV fluid preloading or coloading may be used to reduce the frequency of maternal hypotension after spinal anesthesia for cesarean delivery.

Ephedrine or Phenylephrine.

- Either IV ephedrine or phenylephrine may be used for treating hypotension during neuraxial anesthesia.
- In the absence of maternal bradycardia, consider selecting phenylephrine because of improved fetal acid–base status .

Must Do's before we practice Obstetric Anaesthesia

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SUMMARY OF ANAESTHETIC TECHNIQUES FOR CASEREAN DELIVERY

Anaesthetic technique	Benefit	Risk
General anaesthetic	• Generally considered to be faster option for foetal delivery	• Increased maternal mortality and morbidity
	• Suitable if neuraxial block contraindicated, e.g. the presence of coagulopathy	• Risks associated with airway management (*increased risk of difficult intubation/high risk of pulmonary aspiration of gastric contents*)
	• May be easier to manage an asleep patient in some emergency situations, e.g. major haemorrhage	• Risk of awareness
	• Can modify drugs used for rapid sequence induction if haemodynamic instability present	• Uterine atony with volatile anaesthetic agents
	• Not contraindicated in systemic sepsis	• Maternal transfer of drugs with risk of foetal sedation and respiratory depression
		• Lack of parental presence at delivery
		• Does not provide post-operative analgesia
Spinal	• Generally considered to be the fastest option for neuraxial blockade	• Least suitable for lengthy procedures
	• Low incidence of maternal morbidity including infection and nerve damage	• May require conversion to general anaesthesia if technical failure
	• Avoids risks of general anaesthesia	
	• Can maintain patient in lateral position if situations such as cord prolapse present	
	• Patient remains awake for birth of child	
Epidural extension of labour analgesia	• Relatively fast onset	• Generally considered to take longer than general anaesthesia or spinal techniques
	• Avoids risk of technical failure (e.g. with spinal) in high-risk situation	• Requires adequately working epidural
CSE	• Can be used to provide a more stable	• Higher maternal morbidity than

Recommendations for Postpartum Tubal Ligation

- Before a postpartum tubal ligation, the patient should have no oral intake of solid foods within 6 to 8 h of the surgery, depending on the type of food ingested (e.g., fat content).
- Aspiration prophylaxis.
- Both the timing of the procedure and the decision to use a particular anesthetic technique (i.e., neuraxial vs. general) should be individualized, based on anesthetic and obstetric risk factors (e.g., blood loss), and patient preferences.
- Consider selecting neuraxial techniques in preference to GA for most postpartum tubal ligations.

Cardiopulmonary Resuscitation.

- Basic and advanced life-support equipment should be immediately available in the operative area of labor and delivery units.
- If cardiac arrest occurs, initiate standard resuscitative measures.
  - Uterine displacement (usually left displacement) should be maintained.
  - If maternal circulation is not restored within 4 min, cesarean delivery should be performed by the obstetrics team.

Must Do's before we practice Obstetric Anaesthesia

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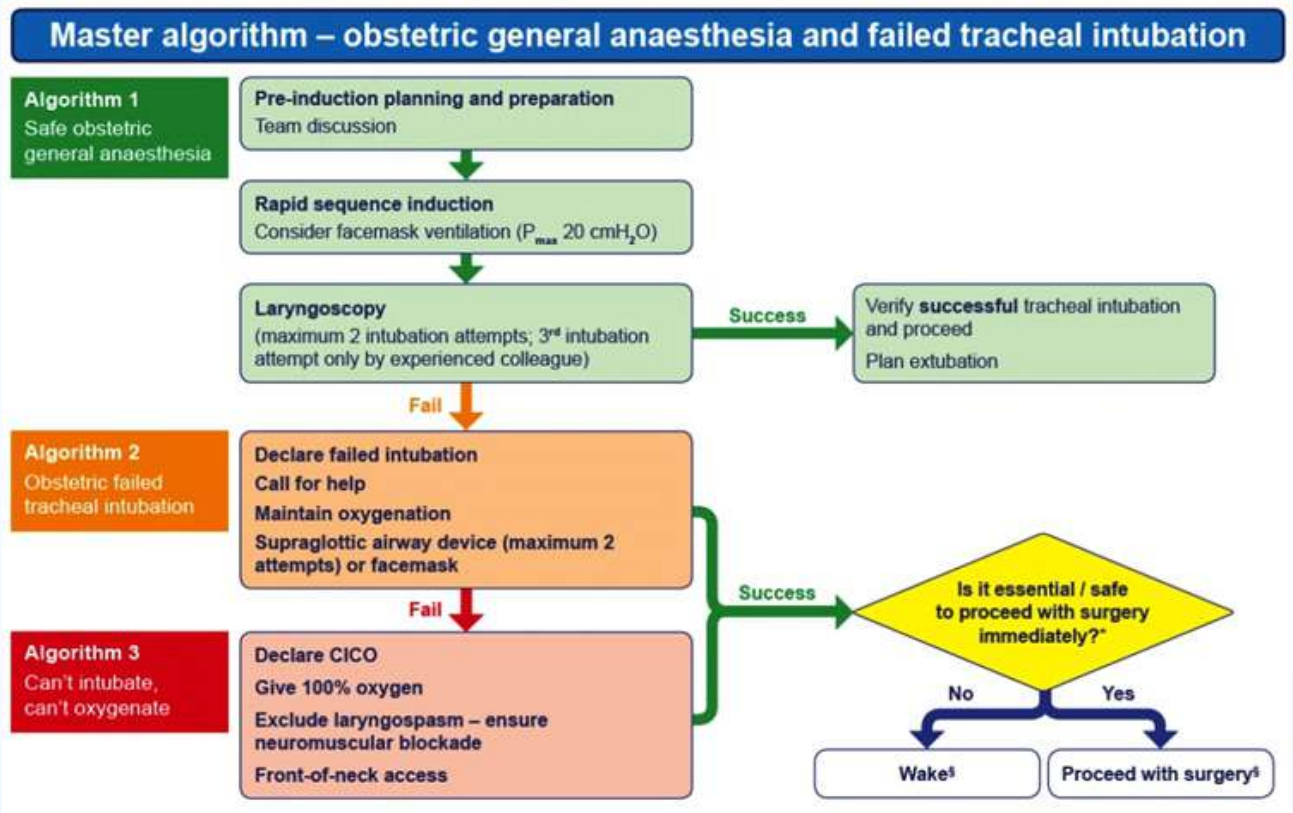
Management of Airway Emergencies.

- Labor and delivery units should have personnel and equipment readily available to manage airway emergencies consistent with the ASA Practice Guidelines for Management of the Difficult Airway to include a pulse oximeter and carbon dioxide detector.
  - Basic airway management equipment should be immediately available during the provision of neuraxial analgesia .
  - Equipment for difficult airway management should be readily available in the operative area of labor and delivery units .
  - A preformulated strategy for intubation of the difficult airway should be in place.
  - When tracheal intubation has failed, consider ventilation with mask and cricoid pressure or with a supraglottic airway device (e.g., laryngeal mask airway, intubating laryngeal mask airway, or laryngeal tube) for maintaining an airway and ventilating the lungs.
  - If it is not possible to ventilate or awaken the patient, a surgical airway should be performed.



Obstretic Anesthetics association/ Difficult Airway Society (2015)

Rapid review of anatomy for anaesthesia

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A strong foundation in basic sciences, such as anatomy, physiology, and pharmacology, is indispensable for anesthesiologists. The interventional nature of anesthesia practice, spanning various body systems like the airway, respiratory, cardiovascular, central and peripheral nervous, necessitates a deep understanding of anatomical structures. This review aims to highlight the critical role of anatomical knowledge in ensuring safe and effective anesthesia practice. It will provide a concise and focused overview of the applied anatomy relevant to anesthesiologists, equipping them with the necessary information to navigate complex clinical scenarios, help them in making informed decisions, anticipate potential complications, and deliver safe and effective anesthetic care. We propose to cover a quick guide of the anatomical details of major body systems relevant to the practice of anaesthesiology.

Airway and Respiratory System

Management of airway and respiratory dynamics is an indispensable task for providing anaesthesia. The respiratory system can be functionally divided into the conducting zone, which transports inhaled air to the respiratory zone, where gas exchange occurs. Anatomically, it's divided into the upper respiratory tract (nose, pharynx, larynx) and the lower respiratory tract (trachea, bronchi, bronchioles, alveolar ducts, alveoli).

The upper airway form the first encounter for providing anaesthesia to any patient. The facial features an often predictable estimation of the ease or difficulty in airway management. The degree of mouth opening in centimetres, upper lip bite test, presence of facial deformity like midface depression, hollow sunken cheeks, protrusion of mandible or retrognathia, all provide useful information about need for preparation for a difficult airway. The assessment of pharynx, is equally crucial for anesthesia providers. It is divided into three sections: the nasopharynx, oropharynx, and laryngopharynx. The assessment of parapharengeal space on using the Modified Mallampatti grading is often used to anticipate difficult intubation scenarios.(Figure 1.) Common causes of upper airway obstruction include enlarged tonsils, adenoids, or a retrognathic mandible. Muscle weakness or excessive soft tissue in the pharynx as in obese patients or with Obstructive sleep apnoea, can passively obstruct an unsupervised airway during sedation or anesthesia.

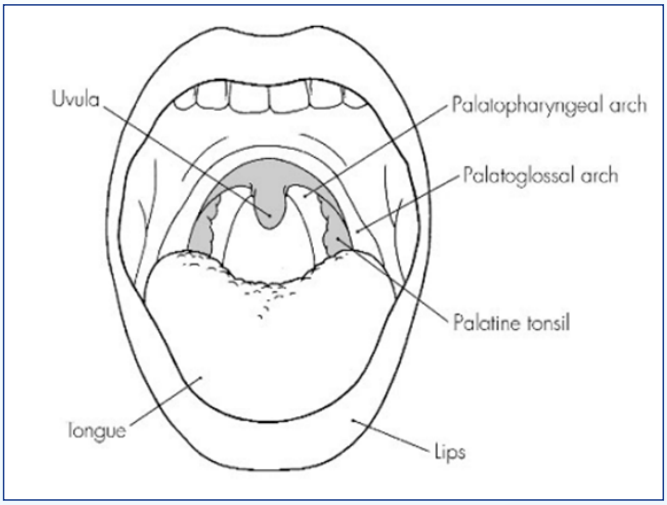

Figure 1. Mouth and oropharyngeal structures

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The larynx, a complex structure composed of cartilage, muscles, and ligaments, plays a vital role in airway management. Key structures include the thyroid cartilage, cricoid cartilage, arytenoid cartilages, epiglottis, and vocal cord. Intubation through the glottic opening or vocal cords is a key component of providing general anaesthesia. (Figure 2.) An array of potential complications during airway management like laryngeal edema, laryngospasm, or vocal cord injury need caution during airway handling during anaesthesia understanding the relationship between the larynx and surrounding structures, such as the thyroid gland and nerves, is crucial for avoiding inadvertent damage during surgical procedures.

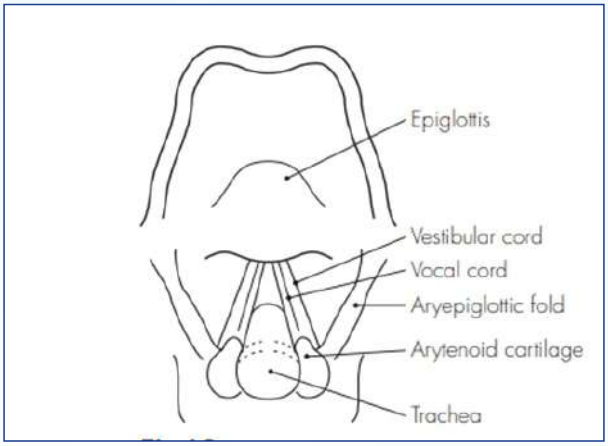

Figure 2. The laryngeal view during intubation

The lower respiratory tract, composed of the trachea, bronchi, bronchioles, alveolar ducts, and alveoli, is crucial for gas exchange during anesthesia. The trachea, a tube-shaped structure, carries air from the larynx to the lungs. It divides into the right and left main bronchi, which further branch into smaller bronchioles. The terminal bronchioles lead to the respiratory bronchioles and alveoli, where gas exchange occurs.(Figure 3) Anaesthetists must be aware of anatomical variations, such as tracheal stenosis or bronchial abnormalities, that can affect airway patency. Additionally, understanding the relationship between the lower respiratory tract and surrounding structures, such as the heart and great vessels, is essential for avoiding inadvertent damage during surgical procedures.

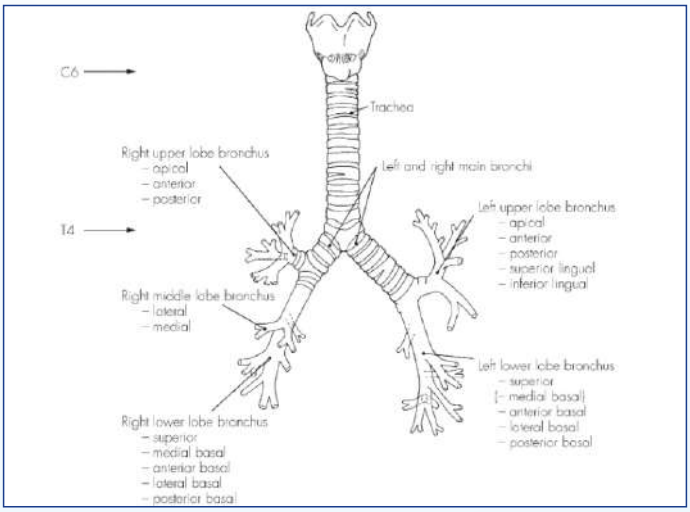

Figure 3. The tracheobronchial tree

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Cardiovascular system

The cardiovascular system, comprising majorly of the heart and the vascular system is vital for maintaining life. Anaesthetists must have a thorough understanding of its anatomy to ensure safe and effective perioperative management. The intravenous mode of instillation of most anaesthetic agents as well as perfusion dynamics of all organs under the effect of anaesthesia mandates a thorough knowledge of cardiovascular anatomy for anaesthesiologists. The heart, a muscular pump, is divided into four chambers. The right atrium receives deoxygenated blood, which is pumped into the right ventricle and then expelled into the pulmonary artery for oxygenation. Oxygenated blood returns to the left atrium via the pulmonary veins and is pumped into the left ventricle, which propels it into the aorta for distribution to the body's tissues. The coronary arteries supply blood to the heart muscle, while the coronary veins return deoxygenated blood to the right atrium. (Figure 4) The circulation of blood in this format helps in understanding any deviation in its flow in the presence of physiological effects of high ventilatory pressures during general anaesthesia as well as the deranged physiology in the presence of congenital heart disorders. A sound knowledge of the heart's electrical conduction system, including the sinoatrial (SA) node, atrioventricular (AV) node, Bundle of His, and bundle branches, is also essential for interpreting electrocardiograms and managing cardiac arrhythmias.

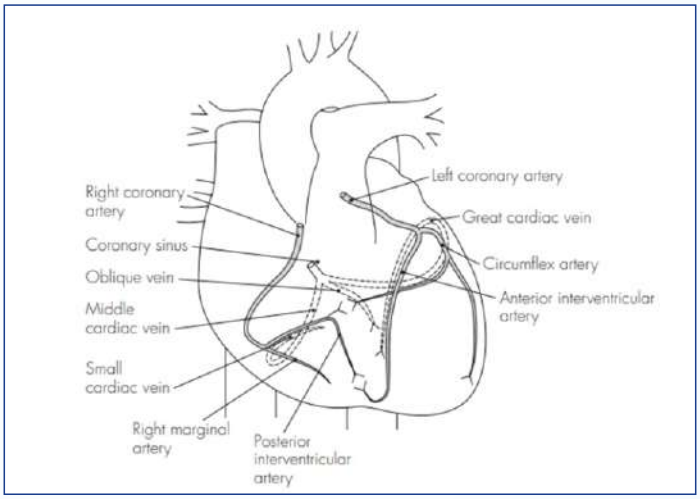

Figure 4. The four chambered heart and its blood supply

The blood vessels are divided into arteries, veins, and capillaries. Arteries carry blood away from the heart, while veins return blood to the heart. Capillaries facilitate the exchange of oxygen, nutrients, and waste products between the blood and tissues. Cannulation of peripheral veins is almost the most mandatory step for initiation of both general and regional anesthesia, as well as under monitored anaesthesia care protocols. Anaesthetists must be familiar with the major blood vessels, including the aorta, pulmonary arteries, pulmonary veins, superior vena cava, and inferior vena cava. (Figure 5) These vena cava are often cannulised for central venous cannulation for monitoring as well as interventional needs during conduct of anaesthesia. Understanding the anatomical relationships between these vessels and other organs is crucial for identifying potential vascular complications and avoiding inadvertent damage during surgical procedures.

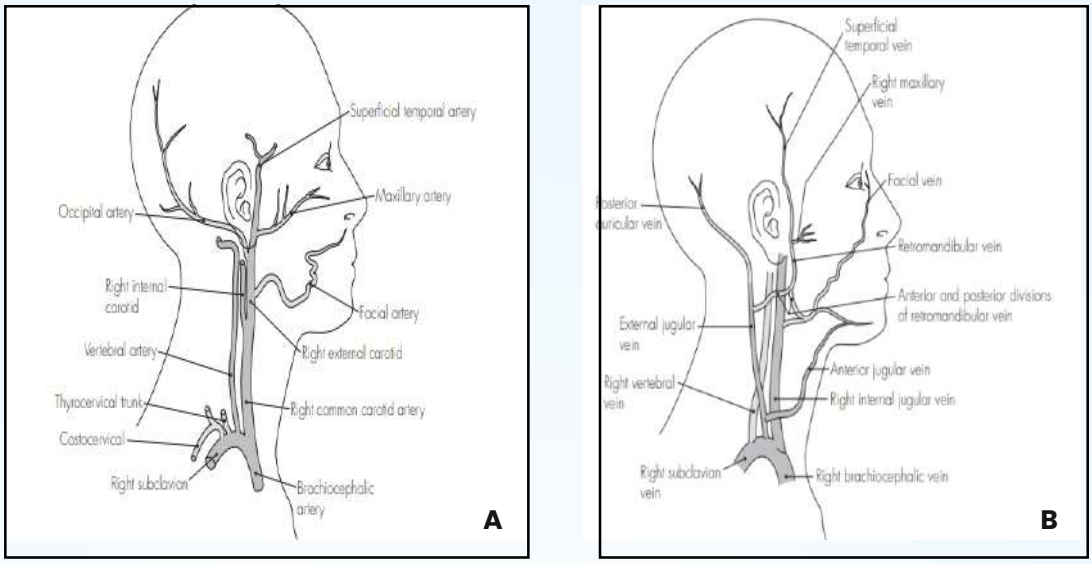

Figure 5(A) The major arteries and (B) veins of head and neck

Central Nervous system

The central nervous system (CNS) is composed of the brain and spinal cord, which are protected by the meninges. The brain is divided into three main parts: the forebrain, midbrain, and hindbrain. The forebrain consists of the cerebral hemispheres and diencephalon, while the hindbrain includes the pons, medulla oblongata, and cerebellum. The brain receives blood supply from the internal carotid and vertebral arteries, forming the circle of Willis. (Figure 6)Venous drainage is primarily through the dural venous sinuses.

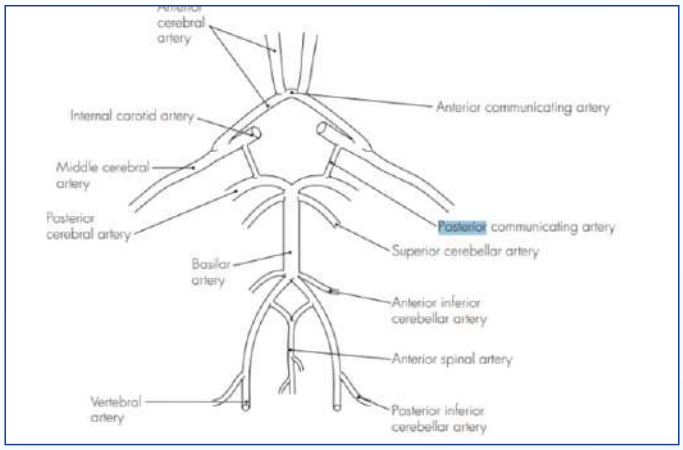

Figure 6: Circle of Willis

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The spinal cord extends from the medulla oblongata to the lumbar region. It is protected by the vertebral column and is responsible for transmitting sensory information to the brain and motor commands from the brain to the body. The spinal cord is divided into cervical, thoracic, lumbar, sacral, and coccygeal segments, each with corresponding spinal nerves. The spinal cord has a gray matter core, shaped like an "H," surrounded by white matter. The grey matter contains nerve cell bodies, while the white matter is composed of nerve fibres that carry sensory and motor information. The spinal cord is supplied by the anterior and posterior spinal arteries, which are reinforced by radicular arteries. Venous drainage occurs through segmental veins.(Figure 7)

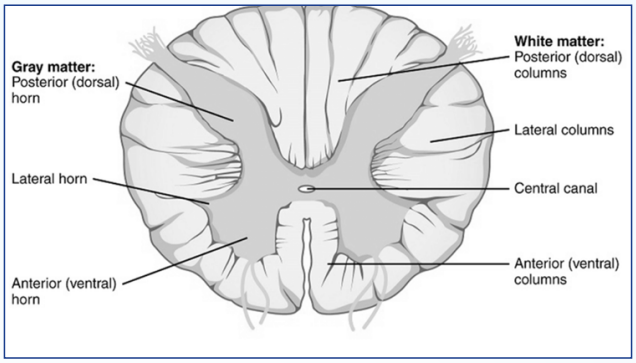

Figure 7. Cross section of Spinal Cord

The meninges, consisting of the duramater, arachnoid mater, and piamater, protect and support the neural tissue. The spaces between these layers, including the subdural and subarachnoid spaces, contain cerebrospinal fluid (CSF), which plays a vital role in maintaining intracranial pressure and providing nutrients to the CNS.

Anaesthetists must have a thorough understanding of CNS anatomy to manage the changes in intracranial pressures and its effects due to anaesthesia. Knowledge of blood supply, cerebrospinal fluid circulation, and the relationship between the CNS and surrounding structures is essential for preventing complications and ensuring patient safety during anesthesia. The conduct of regional anaesthesia and analgesia techniques like spinal and the epidural routes require a keen understanding of the anatomical landmarks encasing the central nervous system components

Spinal Nerves and major nerve plexus

The spinal nerves, 31 pairs in total, are formed from the fusion of ventral motor roots and dorsal sensory roots. They exit the vertebral canal through the intervertebral foramina and divide into dorsal and ventral rami. The dorsal rami innervate the back, while the ventral rami supply the limbs and anterior/lateral torso.The ventral rami of certain spinal nerves unite to form complex nerve plexuses in the cervical, brachial, lumbar, and sacrococcygeal regions. These plexuses are responsible for innervating specific areas of the body.(Figure 8)

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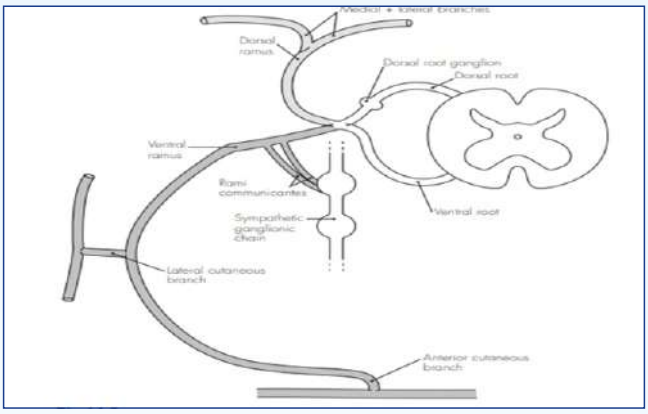

Figure 8. The course of a spinal nerve

The cervical plexus is formed by the ventral rami of C1-C4 and innervates the neck, head, and diaphragm. The brachial plexus is formed by the ventral rami of C5-T1 and innervates the arm. The lumbar plexus is formed by the ventral rami of L1-L4 and innervates the lower abdomen, hip, and proximal lower limb. The sacrococcygeal plexus is formed by the ventral rami of L4-S5 and the coccygeal nerve and innervates the pelvis, perineum, and lower limb.

The upper limb is innervated by the brachial plexus, which is formed by the ventral rami of C5-T1.(Figure 9)The radial nerve is the largest branch of the brachial plexus and supplies the posterior arm, forearm, and hand. The musculocutaneous nerve supplies the anterior arm and forearm. The median nerve supplies the anterior forearm and hand, while the ulnar nerve supplies the medial forearm and hand.

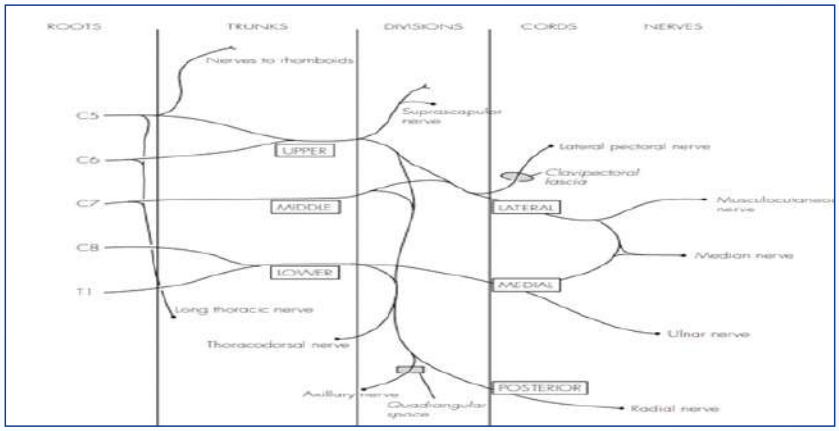

Figure 9. Brachial Plexus

The lower limb is innervated by the lumbar plexus, which is formed by the ventral rami of L1-L4, and the sacral plexus, which is formed by the ventral rami of L4-S5 and the coccygeal nerve. (Figure 10) The femoral nerve is the largest nerve in the lower limb and supplies the quadriceps femoris, sartorius, and skin of the thigh. The obturator nerve supplies the adductor muscles of the thigh. The sciatic nerve is the largest nerve in the body and supplies the hamstrings, calf muscles, and foot.

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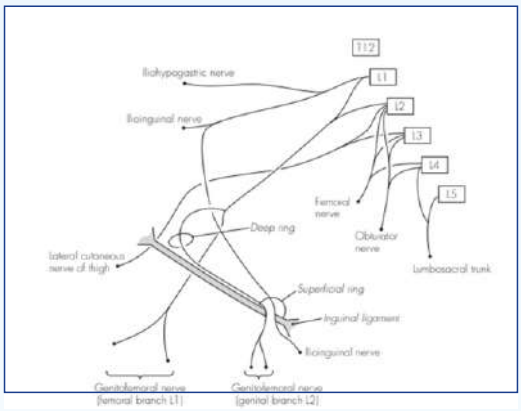

Figure 10. Lumbar plexus

A thorough understanding of spinal nerve and plexus anatomy to perform regional anesthesia techniques as well as knowledge of the distribution of spinal nerves and their corresponding plexuses is essential for identifying appropriate nerve blocks for specific surgical procedures and managing potential complications associated with these procedures.

Autonomic nervous system

The autonomic nervous system (ANS) is a complex network of nerves that regulates involuntary bodily functions, including heart rate, blood pressure, respiration, digestion, and body temperature. It is divided into two main branches: the sympathetic nervous system and the parasympathetic nervous system. The sympathetic nervous system includes the preganglionic neurons that originate in the thoracic and lumbar spinal cord, specifically in the lateral horn of the gray matter. They continue into the sympathetic chain located on either side of the vertebral column. Preganglionic fibres synapse in these ganglia. They further lead into the postganglionic neurons that further distribute to various organs and tissues, including the heart, blood vessels, lungs, and digestive system. The major function of the sympathetic nervous System is the Fight-or-Flight Response, which increases the heart rate, blood pressure, and breathing rate. Dilates pupils. Shunts blood to essential organs

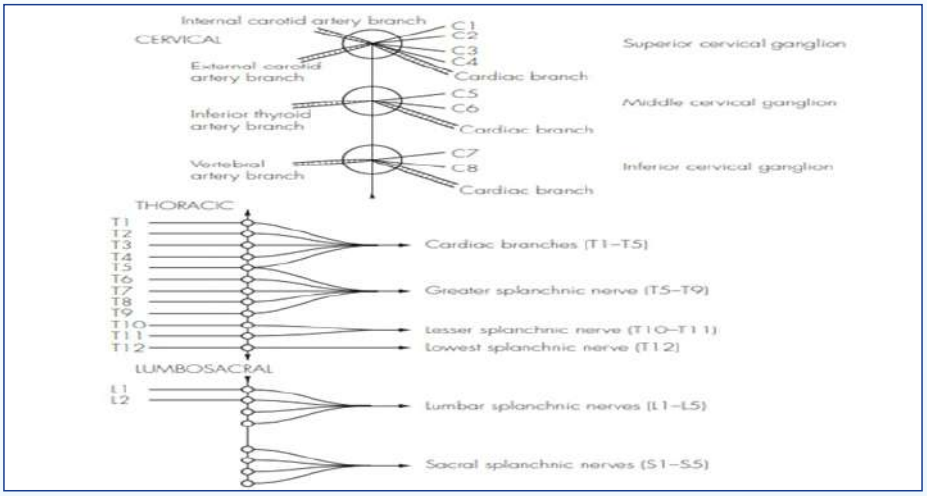

Figure 11. The sympathetic nervous system

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The parasympathetic nervous system has a cranial outflow of preganglionic neurons that originate in the brainstem and travel through the oculomotor, facial, glossopharyngeal, and vagus nerves. (Figure 12)The sacral component involves preganglionic neurons originate in the sacral spinal cord (S2-S4) and travel through the pelvic splanchnic nerves. They terminate into the terminal ganglia located near the target organs, where preganglionic fibres synapse with postganglionic neurons at the level of the target organs.. The parasympathetic nervous system basically causes the rest-and-digest response, that decreases heart rate, blood pressure, and breathing rate, constricts pupils and stimulates digestion.

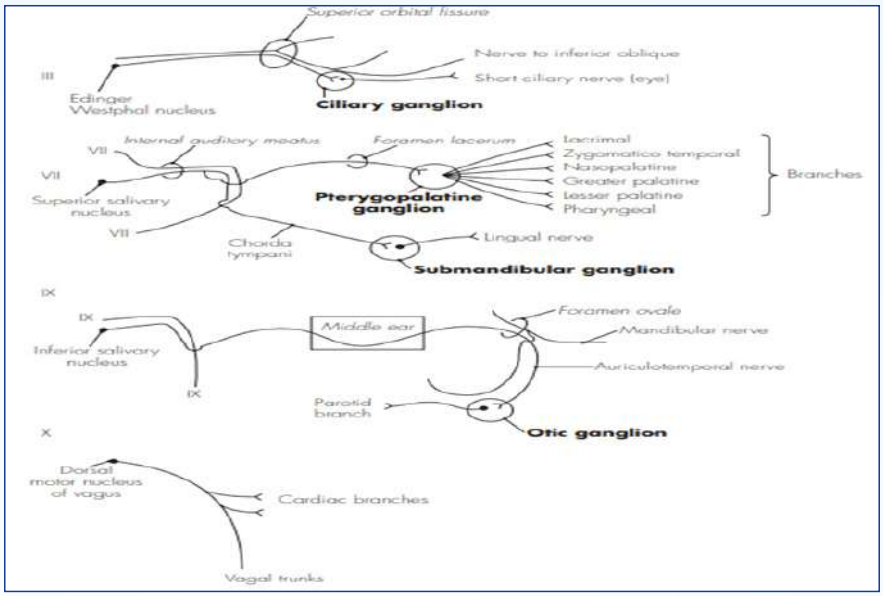

Figure 12. The parasymapathetic nervous system

Understanding the anatomical and functional aspects of the ANS is crucial for anaesthetists to manage patients' physiological responses during surgery. By understanding the effects of the sympathetic and parasympathetic nervous systems, anaesthetists can adjust medications and interventions to maintain optimal hemodynamic stability and prevent complications.

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Comments:

1. CNS can be brought first followed by airway, thoracic, cardiovascular
2. Mention about abdominal, musculoskeletal anatomy as well
3. Summary box highlighting key airway and regional anaesthesia considerations based upon clinical anatomy

Rapid review of Onco-Anaesthesia

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Introduction

Onco-Anaesthesia is a specialized field at the intersection of oncology and anaesthesia, focusing on the perioperative care of cancer patients. As cancer treatments become more complex, the role of anaesthesiologists in managing the unique challenges these patients present is increasingly critical. This review highlights key aspects of oncoanaesthesia, including patient assessment, anaesthetic techniques, pain management, and post-operative care.

Patient Assessment

Preoperative Evaluation

A thorough preoperative assessment is essential for cancer patients. This includes:

1. **Medical History:** Understanding the type and stage of cancer, previous treatments (surgery, chemotherapy, radiotherapy), and current medications.
2. **Physical Examination:** Assessing the functional status using the Eastern Cooperative Oncology Group (ECOG) performance status or Karnofsky scale.
3. **Laboratory Tests:** Routine blood tests, coagulation profiles, and specific tests based on the cancer type (e.g., renal function in patients with renal tumours).
4. **Multidisciplinary Collaboration:** Coordination with oncologists, surgeons, and other specialists to evaluate the risks and benefits of surgery.

Specific Challenges in Oncoanaesthesia

1. **Comorbidities:** Cancer patients often present with multiple comorbid conditions, such as cardiovascular disease, diabetes, and pulmonary disorders. These comorbidities can complicate anaesthetic management and require tailored strategies to mitigate risks.
2. **Malnutrition and Cachexia:** Many cancer patients experience malnutrition or cachexia, impacting their physiological status. This can lead to increased perioperative morbidity and necessitate nutritional interventions prior to surgery.
3. **Psychological Factors:** Anxiety and depression are common in cancer patients, affecting their overall health and recovery. Anaesthesiologists must be prepared to address these psychological factors and may collaborate with mental health professionals for comprehensive care.
4. **Tumour Location and Size:** The anatomic location of tumours can significantly impact anaesthetic management. For instance, thoracic or abdominal surgeries may pose greater risks for respiratory complications.
5. **Immunotherapy and Targeted Therapies:** Patients receiving these treatments may have unique reactions to anaesthesia. Understanding the side effects and mechanisms of these therapies is crucial for safe perioperative management.

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Anaesthetic Techniques

Choice of Anaesthetic Agents

The choice of anaesthetic agents can be influenced by:

1. **Drug Interactions:** Cancer patients may be on multiple medications that can interact with anaesthetics.
2. **Physiological Considerations:** Changes in pharmacokinetics and pharmacodynamics due to cancer or treatment effects, such as altered liver function or volume status.
3. **Immunocompromised State:** Many cancer patients are immunocompromised, requiring careful selection of agents to minimize risks.

Regional Anaesthesia

Regional anaesthesia (RA) can be advantageous in oncoanaesthesia for several reasons:

1. **Reduced Opioid Use:** RA techniques can lead to less postoperative pain and reduced opioid requirements.
2. **Improved Recovery:** Enhanced recovery after surgery (ERAS) protocols often incorporate RA to facilitate quicker mobilization and recovery.
3. **Specific Applications:** Techniques like neuraxial blocks or peripheral nerve blocks may be particularly beneficial for specific surgeries, such as breast or abdominal procedures.

Advanced Anaesthetic Techniques

1. **Enhanced Recovery After Surgery (ERAS) Protocols:** ERAS protocols emphasize multimodal analgesia and the minimization of opioid use. Incorporating preoperative education, optimizing fluid management, and utilizing minimally invasive surgical techniques are key components.
2. **Depth of Anaesthesia Monitoring:** Using advanced monitoring technologies to assess the depth of anaesthesia can help tailor anaesthetic delivery, potentially improving outcomes and reducing the risk of awareness during surgery.
3. **Enhanced Fluid Management:**

    - **Goal-Directed Fluid Therapy (GDFT):** This strategy focuses on optimizing fluid administration based on real-time monitoring of haemodynamics, reducing complications like fluid overload, particularly important in cancer patients who may have pre-existing organ dysfunction.
    - **Crystalloids vs. Colloids**: Understanding the appropriate use of crystalloids versus colloids can help tailor fluid therapy in cancer patients, considering their unique pathophysiology and response to surgery.

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Pain Management

Acute Pain Management

Effective pain management in cancer patients is crucial and may involve:

1. Multimodal Analgesia: Combining different analgesic modalities to achieve better pain control and minimize opioid side effects.
2. Opioid Protocols: Tailored opioid protocols considering the patient's previous exposure and tolerance levels.
3. Adjuvant Medications: Utilizing non-opioid analgesics (NSAIDs, acetaminophen) and adjuvant medications (antidepressants, anticonvulsants) for neuropathic pain.

Chronic Pain Management

Chronic pain is prevalent among cancer survivors and may require:

1. **Interventional Techniques:** Procedures like nerve blocks, intrathecal drug delivery systems, or palliative care consultations.
2. **Psychosocial Support:** Integrating psychological support and counseling as part of a holistic approach to pain management.

Postoperative Care

Monitoring and Complications

Postoperative monitoring in oncoanaesthesia should focus on:

1. **Respiratory Function:** Patients may have compromised pulmonary function due to malignancy or treatment.
2. **Cardiovascular Stability:** Careful monitoring for potential complications, including arrhythmias or thromboembolic events, especially in patients with metastatic disease.
3. **Oncological Considerations:** Early identification and management of complications related to cancer treatment, such as infection or delayed wound healing.

Discharge Planning

Effective discharge planning includes:

1. **Patient Education:** Providing clear instructions on pain management, signs of complications, and follow-up appointments.
2. **Coordination with Home Care:** Arranging for home healthcare services if needed, especially for patients with significant comorbidities or those undergoing extensive treatments.

Ethical Considerations

Informed Consent

Informed consent in oncoanaesthesia is complex and requires:

1. **Transparency:** Discussing the risks and benefits of anaesthesia in the context of the patient's cancer diagnosis and treatment plan.
2. **Cultural Sensitivity:** Recognizing the diverse backgrounds and values of patients, which may influence their treatment preferences.

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End-of-Life Considerations

For patients with terminal cancer, anaesthetic management may involve:

1. Palliative Care Principles: Focus on comfort and quality of life rather than curative approaches.
2. Communication: Ongoing discussions about goals of care and patient wishes.

Future Directions in Oncoanaesthesia

1. Research and Evidence-Based Practice: Ongoing research is vital for advancing oncoanaesthesia. Large-scale studies and clinical trials are needed to establish best practices, optimize anaesthetic protocols, and improve patient outcomes.
2. Integration of Technology: The use of technology, including artificial intelligence and machine learning, may enhance patient monitoring, predict complications, and streamline anaesthetic delivery.
3. Patient-Centred Approaches: Emphasizing patient-centred care models, where patient preferences and values are prioritized, can lead to improved satisfaction and adherence to treatment plans.
4. Interdisciplinary Collaboration: Strengthening collaboration among oncologists, surgeons, anaesthesiologists, pain management specialists, and palliative care teams is essential for delivering holistic and coordinated care.

Conclusion

Oncoanaesthesia plays a pivotal role in the perioperative management of cancer patients, addressing the complexities introduced by their unique medical and psychological needs. A multidisciplinary approach, effective pain management strategies, and careful monitoring can optimize outcomes and enhance the quality of life for these patients. As cancer therapies evolve, anaesthesiologists must remain adept in managing the intricacies of oncoanaesthesia to ensure safe and effective perioperative care.

Rapid review of Trauma anaesthesia:

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Introduction:

Anaesthesiologists play a vital role throughout the trauma care continuum, from prehospital care to rehabilitation, thanks to their expertise in acute care, airway, and pain management, perioperative care, and postoperative critical care. Here's a quick review of key concepts in trauma
anaesthesia:

Preparation: Trauma patients preparation occur in two different clinical settings

Prehospital phase:

- Treated at scene by ambulance service personnel and transported to nearby hospital
- Events are coordinated with the clinicians at the receiving hospital.
- Emphasizes on airway maintenance, control of external bleeding and shock, immobilization of the patient, and immediate transport to the closest appropriate facility.

Hospital phase: A advance planning for the arrival of trauma patients is essential, that include rapid trauma patient resuscitation, preparing the airway equipment trolly, arranging warm intravenous crystalloid for infusion, protocol for additional medical assistance and transfer agreements with various hospitals.

Approach to Trauma Victims(ATLS Concept)

- **Primary Survey along with Resuscitation:** It is identification of immediate life-threatening injuries and their management in the right sequence. It encompasses ABCDEs of trauma care to be done within 10 second.

  Airway: Maintaining airway with restriction of cervical spine movement

  Breathing: Assess ventilation and oxygenation; provide supplemental oxygen as needed.

  Circulation: Maintaining circulation with Control of hemorrhage; establish IV access, and monitor vital signs.

  Disability: Perform neurological assessment (GCS). Check Pupil

  Exposure/Environment: Undress the patient for a complete examination while preventing hypothermia.
- **Adjuncts to primary survey with resuscitation:** It include continuous ECG, pulse oximetry, carbon dioxide (CO_2) monitoring, arterial blood gas (ABG) analysis, and assessment of ventilation. Also, place urinary Catheter (to monitor urine output & hematuria) and Ryles tube (decompress gastric distention & assess for evidence of blood). Other test include lactate, X-ray examination (Chest & pelvis), FAST, eFAST and DPL.
- **Secondary Survey:** It is overall examination of patient's body, from head to toe, including back examination. This includes detailed history and physical examination with reassessment of vital signs.
- **Adjuncts to secondary survey:** In this specialised diagnostic test should be performed like additional X -ray of spine / extremities, CT scans of Head / chest / Abdomen or spine, Bronchoscopy / Angiography / Trans-esophageal USG; contrast urography and angiography.

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- **Reevaluation:** Trauma patients must be reevaluated repetitively to ensure that new findings are not missed and to discover any deterioration further.
- **Definitive care:** Patients may require certain specialised care according to the injury and the facilities available at particular centre. So, if such care is not available, patients should to transfer to such specialised centre for definitive management.

Important Concepts and Terms in Trauma Management

Triage

It means sorting of patients based on the resources required for treatment and the resources that are actually available. Triage order is based on the ABC priorities (airway with cervical spine restriction, breathing, and circulation with hemorrhage control). 4 conventional triage categories are- Green Triage Tag Color: Victim with relatively minor injuries, Yellow Triage Tag Color: Victim's transport can be delayed, Red Triage Tag Color: Victim can be helped by immediate intervention and transport, Black Triage Tag Color: Expectant managment

Airway Management

- Trauma patients are at risk of "difficult airway" or worsen an existing anatomical predisposition. All trauma patients considered as full stomach and are at increased risk of aspiration. Rapid sequence intubation (RSI) or Drug assisted Intubation(DAI) should be used for management for airway in trauma. DAI is defined as any use of medications to facilitate endotracheal intubation (ETI), with or without neuromuscular blocking agents
- Manual in line of Stabilisation (MILS) technique should be used in case of cervical spine injury, when cervical collar is not applied during intubation.
- Head Tilt and chin lift technique should be used to open the airway. Jaw thrust technique should be used to open the airway, in suspected case of cervical spine injury.

Resuscitation

- Established at least 2 wide bore IV catheters (14/16 G) as soon the patients arrive in ED.
- Initiate IV fluid therapy with warm Crystalloid. A bolus of 1 litre of isotonic fluid in adults and 20 mL/kg in paediatric patients weighing less than 40 kilograms over a period of 15 to 30 minutes is administered.
- Amount of resuscitation fluid will depend upon patient response to fluid therapy, keeping in mind the fluid received prehospital setting.
- Early resuscitation with blood and blood products must be considered in patients with evidence of class III and IV haemorrhage to prevent development of coagulopathy and thrombocytopenia.
- Blood Replacement & Type of Blood: Initiation of blood transfusion depend upon the patients response. Patients who are transient responders or nonresponders require pRBCs, plasma and platelets as an early part of their resuscitation. Fully crossmatched pRBCs are preferable for this purpose. Crossmatched PRBC/ Type O blood : Blood typed FFP/ prethawed blood type AB negative plasma : platelets are preferred in 1: 1: 1 ratio in adult patients. However, in paediatric, 20 mL/kg of PRBCs, 20 mL/kg of FFP, and 10 mL/kg of platelets in children.

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Massive Transfusion: It is defined when > 10 units of pRBCs within the first 24 hours of admission or more than 4 units in 1 hour.

Shock

In trauma, shock is classified into hemorrhagic or non-hemorrhagic. Hemorrhage is the most common cause of shock in trauma patients. The non-hemorrhagic shock includes cardiogenic shock, cardiac tamponade, tension pneumothorax, neurogenic shock, and septic shock. Different class of shock is mentioned below in the table with amount of blood loss and its sign and symptoms.

TABLE 3-1 SIGNS AND SYMPTOMS OF HEMORRHAGE BY CLASS

PARAMETER	CLASS I	CLASS II (MILD)	CLASS III (MODERATE)	CLASS IV (SEVERE)
Approximate blood loss	<15%	15–30%	31–40%	>40%
Heart rate	↔	↔/↑	↑	↑/↑↑
Blood pressure	↔	↔	↔/↓	↓
Pulse pressure	↔	↓	↓	↓
Respiratory rate	↔	↔	↔/↑	↑
Urine output	↔	↔	↓	↓↓
Glasgow Coma Scale score	↔	↔	↓	↓
Base deficit*	0 to −2 mEq/L	−2 to −6 mEq/L	−6 to −10 mEq/L	−10 mEq/L or less
Need for blood products	Monitor	Possible	Yes	Massive Transfusion Protocol

* Base excess is the quantity of base (HCO_3^-, in mEq/L) that is above or below the normal range in the body. A negative number is called a base deficit and indicates metabolic acidosis.

Data from: Mutschler A, Nienaber U, Brockamp T, et al. A critical reappraisal of the ATLS classification of hypovolaemic shock: does it really reflect clinical reality? *Resuscitation* 2013;84:309–313.

Damage control Resuscitation:

It is a systematic approach for managing trauma patients with severe injuries, beginning in the emergency room and continuing through the operating room and ICU. DCR involves haemostatic resuscitation, permissive hypotension (where appropriate) and damage control surgery. DCR aims to to maintain circulating volume, control haemorrhage and correct the 'lethal triad' of coagulopathy, acidosis and hypothermia until definitive intervention is appropriate

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Damage Control Surgery

Severely injured patients often do not have the physiologic reserve to tolerate definitive repair It is a surgical intervention to restore physiology rather than correct the anatomy. It involves performing only necessary amounts of surgery to control bleeding, remove nonviable tissue, stabilize fractures and restore extremity perfusion The term 'damage–control' originates from US Navy referring to the ability of a ship to absorb damage while maintaining mission integrity

Permissive hypotension:

This is tactic of limiting fluid resuscitation and allowing for a perfusion pressure that is lower than usual, while any bleeding is under control. Permissive hypotension, also known as hypotensive resuscitation. Ideally, this approach would have begun during the pre-hospital care period. There is still debate over the precise artery pressure target. With the exception of severe traumatic brain injuries (TBI) or spinal injuries, which call for constant systolic arterial pressures more than 90mmHg, European standards recommend systolic arterial pressures of 80–100 mm Hg.

Acute Traumatic Coagulopathy (ATC): It is an endogenous coagulation dysfunction that can develop within minutes of major trauma. Contrary to previous beliefs, it is not a consumptive process rather it is influenced by tissue damage, systemic hypoperfusion, and inflammatory mediator release finally affecting the balance between vascular endothelium, platelets, and pro/anti-coagulant factors. The diagnosis of trauma induced coagulopathy is often made by observing bleeding from wounds or puncture sites and the differential diagnosis between dilutional and consumptive coagulation abnormalities is diagnosed and monitored using laboratory tests like INR, aPTT, fibrinogen, FDP and point of care tests like ROTEM and TEG

Use of Antifibrinolytics: CRASH-2 trial recommend early administration of tranexamic acid. The trial showed that administering 1 g within 3 hours of injury (1 g in a 10-minute bolus followed by 1 g infused over 8 hours) reduced hemorrhage-related mortality. However, if given after 3 hours, it increases bleeding-related mortality. The usual dose of tranexamic acid is 10 to 15 mg/kg followed by 1 to 5 mg/kg/hr. The dose of -aminocaproic acid is 100 to 150 mg/kg followed by 15 mg/kg/hr.

Anaesthesia Considerations In Trauma Patients

Anaesthetic for trauma patients can be challenging due to airway injuries, hemodynamic instability, occult injuries, and, often with limited information on co-morbidities, medications, and allergies. Key steps include:

- Obtain relevant history and conduct a thorough airway assessment and quick physical examination.
- Evaluate airway, breathing, and circulation.
- Document mental status, neurological signs, heart sounds, breath sounds, arterial pressure, pulse rate, and skin color (pallor, icterus, cyanosis).
- Determine the nature and extent of injuries in consultation with the surgeon.

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Anaesthesia Techniques

Most commonly **general anaesthesia** preferred in major trauma cases. **Regional Anaesthesia** considered in specific situations (e.g., lower limb injuries), but be cautious of potential hemodynamic instability, should be avoid in cases of significant hypovolemia or neurologic compromise. Tailor anaesthetic plan based on specific injuries (e.g., thoracic vs. limb injuries) can be considered.

Pharmacological Considerations:

Pre-medication is Often omitted due to urgency, but consider analgesics. For induction, agents that minimize hemodynamic impact impact (e.g., etomidate, ketamine) are used, with doses are carefully titrated and lowered : The higher the shock index, the greater the recommended dosing reduction. For maintenance of anaesthesia a low concentration volatile anaesthetic, total intravenous anaesthesia, opioid, and neuromuscular blocking drugs are used. In Inhalational agents, those having a low blood-gas partition coefficient (desflurane or sevoflurane) are recommended for quick titration.The chosen volatile agent may be raised to ≥0.5 minimum alveolar concentration (MAC) if systolic blood pressure improves to ≥90 mmHg. Nitrous oxide (N O) gas is generally avoided due to increased risk of worsening tension pneumothorax / pneumocephalus, increasing ICP and worsening pulmonary hypertension. For sedation and analgesia consider use of opioids and adjuncts like ketamine or lidocaine or giving various peripheral nerve block. Neuromuscular Blockers should be use with caution as there is risk for prolonged neuromuscular blockade in trauma patients.

Monitoring: Standard monitoring technique as per ASA is applied in trauma patients. Consider arterial lines and central venous catheters for patients with significant injuries or unstable hemodynamic; for fluid resuscitation and medication administration. Point of care testing like ROTEM, TEG, ABG, RBS should be used.

Intraoperative complications: Intraoperatively trauma patients can have persistent hypotension, hypothermia (due to shock & fluid resuscitation), coagulation disorder, electrolyte and acid-base abnormality and results in death due to uncontrol bleeding, air embolism and brain herniation.

Peripheral nerve Block: Different peripheral nerve block, nerve plexus, and myofascial plane block can be used in trauma patients for surgery as well as pain control.

Head Injury:

- The Glasgow Coma Scale (GCS) score is used as an objective clinical measure of the severity of brain injury.
- A GCS score of 8 or less define stage of coma or severe brain injury. Patients with a brain injury who have a GCS score of 9 to 12 are categorized as having "moderate injury," and individuals with a GCS score of 13 to 15 are designated as having "mild injury."
- In assessing the GCS score, if there is right/left or upper/lower asymmetry, the best motor response is noted to calculate the score.
- Traumatic Brain Injury: It's crucial to maintain adequate ventilation, oxygenation, and cerebral blood flow (CBF) to prevent further brain damage. Goals for concurrent traumatic brain injury management include:

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- SBP > 100 mmHg
- SpO2 > 90%
- pCO2 35 to 45 mmHg

TABLE 6-2 GLASGOW COMA SCALE (GCS)

ORIGINAL SCALE	REVISED SCALE	SCORE
Eye Opening (E)	Eye Opening (E)	
Spontaneous	Spontaneous	4
To speech	To sound	3
To pain	To pressure	2
None	None	1
	Non-testable	NT
Verbal Response (V)	Verbal Response (V)	
Oriented	Oriented	5
Confused conversation	Confused	4
Inappropriate words	Words	3
Incomprehensible sounds	Sounds	2
None	None	1
	Non-testable	NT
Best Motor Response (M)	Best Motor Response (M)	
Obeys commands	Obeys commands	6
Localizes pain	Localizing	5
Flexion withdrawal to pain	Normal flexion	4
Abnormal flexion (decorticate)	Abnormal flexion	3
Extension (decerebrate)	Extension	2
None (flaccid)	None	1
	Non-testable	NT

GCS Score = (E[4] + V[5] + M[6]) = Best possible score (5, worst possible score 3)
*If an area cannot be assessed, no numerical score is given for that region, and it is considered "non-testable." Source: www.glasgowcomascale.org

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Comments:

1. Pain management can be a sub section for trauma cases
2. Post trauma anaesthesia care can highlighted with bit of focus of post-traumatic stress disorder

Rapid review of pain

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Pain is a multidimensional personal experience that influences not only physical well-being but also psychological, social and spiritual aspects of life. The prevalence of chronic pain in India ranges from 19 – 37% and increases steeply beyond the age of 65. 1-3 Higher number of females are affected as compared to males. Chronic pain negatively impacts the quality of life and is responsible for loss of work hours.

Understanding Pain: Definitions and Classifications

The International Association for the Study of Pain (IASP) defines pain as an unpleasant sensory and emotional experience associated with, or resembling that associated with, actual or potential tissue damage. It is classified as acute or chronic pain. Acute pain serves as a protective mechanism that alerts individuals to potential pathology like fractures, sprains, infections etc. It may be associated with symptoms and signs of sympathetic overactivity such as tachycardia, hypertension and sweating. If pain persists past the normal healing time, usually more than 3 months, it is classified as chronic pain. It is associated with mood disturbances and can sometimes lead to anxiety and depression, which can further complicate management. The transition from acute to chronic pain can be influenced by various factors, including genetics, psychological factors, environmental influences, and the nature of the initial injury or disease.

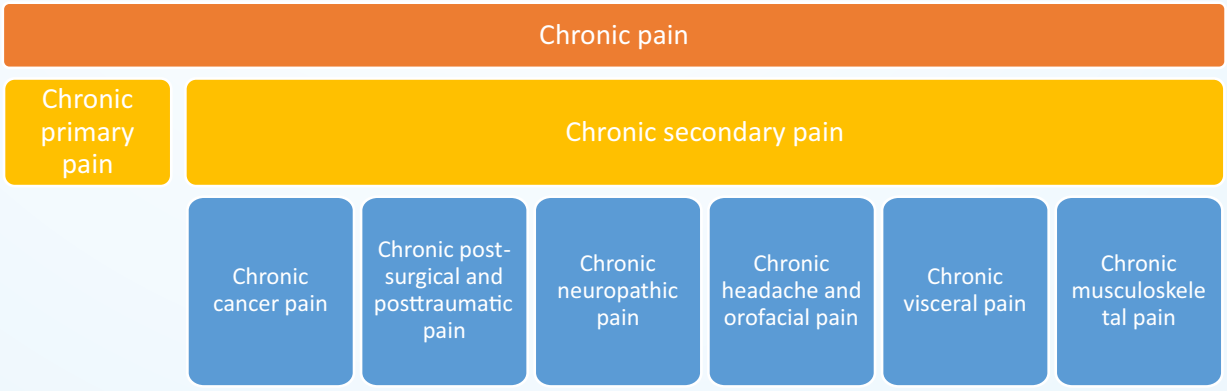
On the basis of pathology, pain can be classified into Nociceptive and neuropathic pain. Nociceptive pain arises from the activation of nociceptors, whereas Neuropathic pain results from damage or dysfunction in the nervous system.

Nociceptive pain can be categorized as:

- Somatic Pain: Arising from skin, muscles, and joints, it is described as sharp and well-localized.
- Visceral Pain: Pain originating from internal organs (e.g., pancreatitis, appendicitis) is often described as deep, aching, or cramping.

Neuropathic pain is commonly described as a burning, shooting or electric shock-like sensation. It is categorized as:

- Peripheral neuropathic pain: Postherpetic neuralgia, peripheral nerve injury and diabetic neuropathy
- Central neuropathic pain: Post stroke pain, spinal cord injury, etc.



ICD-11 Classification of chronic pain

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According to ICD-11 classification 4, chronic pain is classified into

- Chronic primary pain - Chronic pain in 1 or more anatomic regions associated with significant emotional distress or significant functional disability that cannot be better explained by another chronic pain condition. It includes back pain that is neither identified as musculoskeletal or neuropathic in origin, chronic widespread pain, fibromyalgia and irritable bowel syndrome. This denotes pain as a disease entity and not just a symptom of an underlying disease. 5
- Chronic secondary pain. The latter is further classified into

  i. Chronic cancer pain – Pain may be present as a result of musculoskeletal, visceral or neural invasion, or associated with the treatment ie. surgery, radiotherapy and chemotherapy.

  ii. Chronic postsurgical and posttraumatic pain

  iii. Chronic neuropathic pain - It is caused by a lesion or disease of the somatosensory nervous system. It is characterized by hyperalgesia (heightened pain disproportionate to the painful stimulus) or allodynia (painful response to a nonpainful stimulus). It can present after a stroke, diabetic neuropathy, nerve injury or compression.

  iv. Chronic headache and orofacial pain – Headache and orofacial pain are said to be chronic if they span over at least 50% of the days during a minimum of 3 months. Most common chronic orofacial pains include temporomandibular disorders.

  v. Chronic visceral pain – Pain originating from the visceral organs usually manifests as referred pain to the somatic dermatomes that share innervation with the affected viscera. The pain may arise due to persistent inflammation, obstruction or compression, ischemia or a combination of these mechanisms.

  vi. Chronic musculoskeletal pain – It arises, as the name suggests, from the bone, joint, muscles or related soft tissues.

The Neurobiology of Pain

Pain perception begins with the activation of nociceptors, which are specialized sensory receptors that respond to potentially damaging stimuli such as mechanical injury, extreme temperatures or chemical irritants. These receptors transmit signals through peripheral nerves to the spinal cord. The spinothalamic tract is the primary pathway, transmitting signals to the thalamus and then to various areas of the cortex responsible for pain perception, including the somatosensory cortex, which processes the sensory aspect of pain. Certain projections reach the limbic system, which is involved in the emotional response to pain.

- Transduction: The conversion of noxious stimuli into electrical signals or action potentials by nociceptors. This process is facilitated by the release of inflammatory mediators, such as prostaglandins, bradykinin and substance P, which sensitize nociceptors and enhance their response to stimuli.
- Transmission: The conduction of pain signals along peripheral nerves to the spinal cord. Two main types of nociceptive fibers are A-delta fibers (fast, myelinated, transmit sharp, localized pain) and C fibers (Slow, Unmyelinated, transmit dull, aching, or throbbing pain)

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- Modulation: The alteration of pain signals within the spinal cord by excitatory and inhibitory interneurons influenced by descending pathways from the brain. Structures such as the periaqueductal gray and the rostral ventromedial medulla inhibit or facilitate pain transmission at the spinal cord level. This modulation involves endogenous opioid systems, serotonin and norepinephrine pathways, impacting the overall experience of pain.
- Perception: The brain's interpretation of pain signals, influenced by emotional and cognitive factors.

  With chronicity of pain, alterations in the pain pathways may lead to central sensitization due to increased excitability of neurons in the central nervous system, resulting in heightened sensitivity to pain stimuli and the perception of pain even in the absence of noxious stimuli. 6

Pain Assessment

Accurate pain assessment is critical for effective management and requires a thorough understanding of the patient's experience. It includes the intensity, quality, location, duration, and impact of pain on daily activities. Several tools are available for such assessment. These can be unidimensional and multidimensional.

- Visual Analog Scale (VAS): A 100 mm line is drawn where patients mark a cross on the given line that represents their pain intensity. The examiner then manually measures the distance from the "no pain" end to this cross.

  No pain _____ Pain as bad as could be

  The downside is that it is more time-consuming and susceptible to measurement errors. A validated alternative is the use of mechanical VAS where subjects position a slider on a linear pain-scale.7 The examiner can directly read the pain intensity on a millimetre-scale on the other side of the slider. VAS can also be recorded and assessed electronically on touch screen computers and pen-based electronic diaries.
- Numerical Rating Scale (NRS): Patients rate their pain on a scale from 0 (no pain) to 10 (worst pain imaginable). Therefore, there are only 11 possible answers in a 0–10 point NRS. It is feasible to administer it verbally and hence useful for telephonic interviews. It also has good patient compliance.
- McGill Pain Questionnaire (MPQ): A multidimensional tool that assesses pain quality and intensity through descriptive words. It consists of three major measures — pain-rating index, word descriptors and pain intensity. The pain-rating index consists of numerical grading of words describing sensory, affective and evaluative aspects of pain. The MPQ is the most extensively reviewed tool to measure pain affection.
- Brief Pain Inventory (BPI): Multidimensional scale that assesses pain severity and the impact of pain on daily functions.

Management of Chronic pain

Routine blood investigations and imaging are not recommended for every patient presenting with chronic pain. However, specific tests may be ordered on an individualized basis, including baseline hematological and biochemical studies when certain medications with potential systemic side effects are prescribed. While treating chronic pain, it is essential to address both the pain itself and any comorbid conditions, especially psychiatric disorders, thereby targeting "total pain".

Rapid review of pain

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Pharmacological management

Pharmacological management of pain often begins with non-opioid analgesics. Among these, Non-Steroidal Anti-Inflammatory Drugs (NSAIDs) are effective and most commonly used for managing acute and chronic nociceptive pain. Some of the frequently used non-selective NSAIDs are ibuprofen, aceclofenac and naproxen.

Drug	Average analgesic Dose	Maximal daily dose (mg)	
Ibuprofen	200 – 400 mg 4 - 6 hourly	2400	
Diclofenac	50mg 8 hourly	150	More selective for COX2 than COX1
Naproxen Mefenamic acid	500 mg first dose, 250 mg subsequently 6 – 8 hourly	1500	
Ketorolac	20 mg initially, 10 mg subsequently	40 (oral) 120 (iv/im)	Limit dose to 5 days, as it can precipitate renal failure in dehydrated patients
Celecoxib	200 – 400 mg 12 – 24 hourly	400	
Etoricoxib	60 – 90 mg	120	

Before initiating NSAIDs patient's renal status, hepatic and cardiovascular status need to be assessed as NSAIDs are associated with higher risk of peptic ulcer disease. Diclofenac is associated with the highest reported increase in adverse gastrointestinal and cardiovascular events. Less severe and common adverse effects include headache, nausea, diarrhoea and dizziness. Among COX 2 selective NSAIDs, etoricoxib is commonly prescribed. Topical NSAIDs like diclofenac sodium 1.5% topical solution, diclofenac hydroxyethyl pyrrolidine 1.3% patch and diclofenac sodium gel 1% are also available and useful for treating pain due to soft-tissue injuries and osteoarthritis. Acetaminophen is used for mild to moderate pain. It is generally well-tolerated but can lead to liver toxicity in excessive doses. Recommended maximum dose is 3g/day.

For patients with acute postoperative pain, cancer pain, and severe chronic pain unresponsive to other treatments, opioids are administered. Commonly used opioids are tramadol, tapentadol, morphine and buprenorphine.

For cancer pain, opioids are given on following principles:

- By the mouth - Simple, effective, convenient, can be taken at home
- By the clock- For continuous pain relief, round the clock administration
- By the individual - Less frequent or reduced dose in renal or hepatic dysfunction.
- By the ladder – WHO Analgesic ladder

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Drug	Average analgesic Dose	Maximal daily dose (mg)	
Tramadol	50mg 8th or 6th hourly	400 mg	
Morphine	5-10 mg 4th hourly Breakthrough pain – same as the 4th hourly dose		
Buprenorphine	0.2- 0.6mg 6 -8 hourly		

Common side effects of opioids include constipation, sedation and respiratory depression. Opioids are known for causing dependence and addiction of prolonged use.

Additionally, medications such as antiepileptics (gabapentin or pregabalin) and antidepressants (tricyclics like amitriptyline and serotonin-norepinephrine reuptake inhibitors like duloxetine), muscle relaxants, N-methyl-d-aspartate (NMDA) receptor antagonists and alpha 2 adrenergic agonists are also possible pharmacological therapies for managing chronic pain.

Drug	Average analgesic Dose	Maximal daily dose (mg)	
Gabapentin	100 – 400 mg, 8 hourly	2400	
Pregabalin	75mg HS, then BD as required	600	Pregabalin is as effective as gabapentin with fewer side effects
Amitriptyline Nortriptyline	10 – 25 mg HS, titrated to 75 mg/ day	150	
Duloxetine	30 – 60 mg/ day		
Carbamazepine	Titrated to 100 - 400mg TDS		

Non-pharmacological management

In conjunction with pharmacological interventions, non-pharmacological strategies play a vital role in managing pain, particularly in musculoskeletal conditions. Techniques may include exercise therapy to improve strength, flexibility and function; manual therapy techniques like massage and manipulation and modalities like heat, cold, ultrasound and electrical stimulation. Cognitive behavioural therapy is an evidence-based psychological intervention that addresses maladaptive thought patterns and behaviours and develop coping strategies for managing chronic pain. Complementary therapies like acupuncture, biofeedback, mindfulness and meditation, art and music therapy, hypnotherapy, reassurance and diversional therapy aim at reducing stress and promoting relaxation. These have been used with variable response to treatment.

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Interventional pain management

When conservative management fails, interventional pain management techniques may be considered. Some of these in brief are as follows:

- Nerve blocks: Injection of local anesthetics, steroids, etc., near specific nerves. Common types include:
  - Peripheral Nerve Blocks: Target specific nerves (e.g. median nerve block for carpal tunnel syndrome, peripheral nerve injury).
  - Sympathetic blocks: For sympathetically maintained pain for example, sphenopalatine block for headache, stellate or lumbar sympathetic block for complex regional pain syndrome, phantom limb pain, etc.
  - Epidural Steroid Injections: Used for radicular pain, delivering medication to the epidural space to reduce inflammation.
- Musculoskeletal interventions: For management of intra-articular pathologies, myofascial pain syndrome, tendinitis, etc.
- Radiofrequency Ablation: Radiofrequency ablation uses thermal lesioning or neuromodulation of specific nerves involved in pain transmission. It has various applications in chronic pain management, some of which are joint pain (facet, shoulder, hip etc.), or neuropathic pain syndromes (persistent post-surgery pain, lumbar radicular pain, trigeminal neuralgia, etc.)
- Spinal Cord Stimulation: It is a neuromodulation technique that involves implanting a device that delivers electrical impulses to the spinal cord, interrupting pain signals. This technique is often used for chronic pain conditions unresponsive to other treatments.

Pain management in special populations

A focus on broader context of pain including genetic, developmental, environmental and individual factors is essential while managing pain in paediatric population. Acetaminophen is widely used for management of acute pain. There are little studies on chronic daily administration of acetaminophen and NSAIDs and daily use can produce rebound headaches.

Opioid tolerance and opioid-induced hyperalgesia are likely to proceed more rapidly in children. They have been shown to have detrimental effects on endocrine function as well. Therefore, if needed, it is recommended to use opioids with caution. Use of tramadol is contraindicated in patients less than 12 years of age because of potential respiratory complications.

While taking care of elderly, common barriers encountered are

- Physiologic impairments (reduced peripheral intraepidermal nerve fiber density, small fiber neuropathy, decreased speed of processing nociceptive stimuli)
- Sensory impairments (Visual and hearing)
- Cognitive impairment

Acetaminophen and NSAIDs can be considered for short-term treatment and should be used with caution for long-term treatment for older patients with chronic pain. Co-administration of gastroprotective agents (Proton pump inhibitors or misoprostol) is recommended.

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According to the 2023 American Geriatrics Society Beers Criteria update8, opioids should be avoided except for pain management in the setting of severe acute pain because they may cause ataxia, impaired psychomotor function, syncope and falls. Antidepressants and antiepileptics should be used with caution as they may exacerbate or cause SIADH or hyponatremia. Sodium levels should be monitored closely while starting or changing dosages.

Pain during pregnancy and lactation pose unique challenges for the physician. Sub optimal treatment of pain may lead to anxiety, depression, insomnia, poor quality of life and may affect maternal and fetal bonding. Challenges faced are fear of using drugs in pregnancy and physiological changes affecting pharmacokinetics and pharmacodynamics of the drugs. Drugs considered safe during pregnancy and lactation are acetaminophen. If pain is controlled on acetaminophen, use of mild opioid or opioid-acetaminophen combination is considered. Close fetal monitoring during the second trimester if high doses of NSAIDs ae required. Periodic fetal ultrasound, including fetal echocardiography should be used to monitor amniotic fluid volume and patency of ductus arteriosus. NSAIDs should be discontinued after 34 – 36 weeks of gestation to reduce risks of peripartum bleeding, neonatal hemorrhage and persistent fetal circulation.9

Ibuprofen and Naproxen are considered safe during lactation. Opioids like Oxycodone, Pentazocine, Meperidine and Codeine are not recommended for lactating mothers as clinically therapeutic levels are achieved in infants, producing adverse effects.

Epidural steroid injection can be given in the 2-3rd trimester of pregnancy if needed. Risk to the fetus following a single dose of an epidural corticosteroid appears to be low. Trigger point injections, fascial plane blocks and peripheral nerve blocks with local anaesthetic and steroid can be performed without any adverse effects.

Emerging therapies and future research areas

Utilizing virtual reality for distraction, neural reprocessing and mindfulness training are being used to treat acute and post-surgical pain, and has shown promising results in managing chronic pain conditions, especially low back pain.10 Some VR technologies have demonstrated a comparable analgesic effect to opioid medications. Wearable medical technology are being used to monitor complex physiological signals that can be used as a measure of pain. Artificial intelligence has become another revolutionary innovation in the treatment of chronic pain. It can predict or influence prospective outcomes like pain in candidates for neuromodulation.

Numerous novel medications such as sodium channel blockers, peptides, and cannabinoids have been added to the armamentarium of pain physician. Psychedelic medications are being studied to treat cancer pain, phantom limb pain, fibromyalgia, neuropathic pain and cluster headaches. The proposed mechanism involves serotonin 2A (5-HT2A) receptor agonism resulting in “reset” of regions of functional connectivity (FC) in the brain that are crucial in many central neuropathic conditions.

In conclusion, a thorough comprehension of pain mechanisms and meticulous clinical assessment are imperative for accurate diagnosis. Successful pain management necessitates a comprehensive approach integrating pharmacological, interventional, and non-pharmacological strategies, while also acknowledging the psychological, emotional, and social dimensions of pain.

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Rapid review of cardiac anaesthesia

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Heart at the centre of a series circulation

Anatomically speaking, heart is an organ with two atria and two ventricles where one chamber exists on each side in a manner that features the pulmonary and systemic circulation in series. The systemic venous return here reaches the right atrium and ventricle; to be coupled to the pulmonary circulation for the requisite oxygenation and the pulmonary venous blood reaches the left atrium and ventricle to provide for the systemic perfusion. The heart being the centre of both the circulations efficiently needs to pump and receive blood, depending on the interplay between the cardiac and the extracardiac factors.

The Cardiac-tropies:

Tropy (Greek for change) refers to the effect of different external stimuli, which produce various alterations in the cardiac activity, a concept introduced by TW Engelman. There are 5 tropy's which have been described in literature namely:

1. Inotropy which describes the change in contractility.
2. Chronotropy entails the change in rate.
3. Dromotropy refers to change in conduction.
4. Bathmotropy deals with change in excitability.
5. Lusitropy pertains to the cardiac relaxation.

The Oxygen Supply-Demand dynamics:

Adequate myocardial performance is intricately linked to the oxygen supply-demand matching which is a dynamic entity which pivots to both the sides of the scale depending on the factors that aim to prevent ischemia by aligning oxygen supply and decreasing demand. Normal oxygen supply of myocardium is 80-100ml/100mg amounting to 4-5% of the cardiac output (CO) with a high extraction ratio of 70-80% compared to 25-30% as in other tissues. Factors determining myocardial oxygen supply are oxygen content of blood, coronary vessel patency, presence of stenosis or collaterals, coronary vascular resistance, metabolic coupling, autonomic nervous system, hormonal factors and last but not the least, the coronary perfusion pressure (CPP).

CPP = Aortic diastolic pressure – Left Ventricular End Diastolic Pressure (LVEDP)

Factors determining myocardial oxygen demand are constituted primarily by the heart rate, contractility and, the wall stress. Left ventricular (LV) myocardial perfusion occurs in diastole due to coronary anatomy and intracardiac factors, whereas the right ventricle (RV) is perfused throughout the cardiac cycle. Any factor that decreases the diastolic time such tachycardia will cause decrease in the coronary perfusion.

Cardiac Monitoring, in nutshell:

Basic cardiac monitoring includes heart rate, rhythm, Electrocardiograph (ECG), arterial pressure (systolic, diastolic, and mean) and waveform, central venous pressure (CVP) and waveform. Heart rate and rhythm can be monitored by a basic five electrode ECG system which will give all limb leads along with a ventricular lead which is placed at V5 position for assessing the LV. 90% of the ischemic episodes can be diagnosed by simultaneous monitoring of leads II and V. As for arrhythmias, lead II can be particularly helpful. Speaking of ischemia, it is new ST

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elevation present at the J point in at least two anatomically contiguous leads > 0.1 mV in all the leads other than the leads V2-V3 and for the leads V2-V3, the following described cut points are: ≥ 0.2 mV in men ≥ 40 years, > 0.25 mV in men < 40 years and ≥ 0.15 mV in women.

In hemodynamic terms, the rate pressure product (RPP) is used to assess cardiac exercise workload and predict the corresponding risk, as calculated by multiplying the heart rate and systolic blood pressure. The quotient of mean arterial pressure (MAP) and heart rate, the Buffington ratio predicts blood flow to the collateral dependant myocardium with a value < 1 forecasting the risk of ischemia.

CO can be measurement by thermodilution method with Pulmonary artery catheter (PAC); the "gold standard", involving the injection of 5-10 ml cold injectate to the right atrium with temperature measured against time in the pulmonary artery giving the CO by Stewart Hamilton equation. Higher the CO, faster the blood flow with shorter, smaller, and steeper thermodilution curve. Continuous thermodilution by placement of thermal filament in PAC generates real-time CO values. The non-invasive CO monitoring subset include: uncalibrated pulse contour analysis with FloTrac, LiDCO rapid, etc and calibrated pulse contour analysis by transpulmonary thermodilution with PiCCO Plus. Other modalities are Doppler ultrasound, bioreactance and bioimpedance. Of these, the common modality in practice is the uncalibrated pulse contour analysis which measures the systolic, diastolic, and mean systemic pressures along with the CO, stroke volume variation and the pulse pressure variation which helps in assessing an estimate of the preload, and the afterload, in addition.

PAC and the anaesthesiologist:

PAC opens the window to wide range of variables including direct measured parameters such as CVP, pulmonary artery pressure, Pulmonary capillary wedge pressure (PCWP), blood temperature, mixed venous oxygen saturation, and other derived parameters such as systemic vascular resistance (SVR), pulmonary vascular resistance (PVR), cardiac index (CI), stoke volume index (SVI), right ventricular stroke work index, left ventricular stroke work index. Use of PAC being a debate, is still a good tool in experienced hands aiding in real-time assessment of volume status, LV and RV function. The ischemia prone subset also benefits by early detection of V-waves secondary to ischemia. All in all, when PA catheter is used, it is one modality by which can give a real-time measurement of PCWP/LVEDP. Elucidating on LVEDP, it is ideally measured when the tip is wedged in West zone III of the lung as pulmonary venous pressure exceeds alveolar pressure here, making the part of the pulmonary circulation to be in physiological continuity with LV at the end-diastole. Any factor which disrupts this physiological continuity can cause underestimation or overestimation of LVEDP. Overestimation (PCWP>LVEDP) can occur in positive-pressure ventilation (PPV0 with high levels of positive end-expiratory pressure, increased intrathoracic pressure, non–West lung zone III PAC placement, Chronic obstructive pulmonary disease, left atrial myxoma, increased pulmonary vascular resistance and mitral valve disease. Underestimation (PCWP<LVEDP) occurs in cases of non-compliant LV and aortic regurgitation.

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Systolic dysfunction: Ejection Fraction and beyond

Systolic dysfunction refers to the inability of myofibrils to shorten against load, resulting in a failure to generate adequate pressures and CO. With the most frequently employed parameter to quantify it being LV Ejection Fraction (LVEF). LVEF represents the ejection phase dimensionless volumetric ratio of SV to End Diastolic Volume (normal range 55% - 65%). Alternate Indices include Pressure-Volume Analysis (End-systolic pressure-volume relation and stroke work-end-diastolic volume relation), Isovolumic Contraction dP/dtmax, and ejection phase parameters [Fractional area change and wall thickening]. In patients with lets' say mitral regurgitant lesions, nonetheless, LVEF may overestimate the ventricular function, as anyways when there is decreased effective SV and a fraction of the blood from LV is ejection into the more compliant left atrium, amounting to the regurgitant fraction which eats into the EF. To overcome this, newer modalities such as strain rate by tissue Doppler and speckle tracking which measures the inherent myocardial function. 3D volume assessment techniques have also been developed to exactly measure the end systolic and end diastolic volume and EF.

Diastolic Dysfunction:

Cardiac diastole comprises of the following phases: isovolumic relaxation, rapid filling, diastasis, and the atrial Kick. Herein, Diastolic dysfunction (DD) refers to impaired relaxation or compliance of the ventricles, resulting in high filling pressures, which may precede systolic dysfunction, with as high as 28% prevalence in patients > 60y of age. While anesthetizing patients with DD, a range of perioperative factors (tachycardia, volume shifts, atrial fibrillation (AF) with loss of normal sinus rhythm, anemia, hypoxia, electrolyte disturbances, sympathetic stimulation, hypertensive crisis, shivering, etc.) can lead to the precipitation of diastolic heart failure (DHF) in such patients where elderly, obese, hypertensive, diabetic, cancer, anemic patients, and those with history of DHF can be peculiarly predisposed. While no definite recommendation exists on the preference to general/regional anesthesia, epidural does score over spinal, owing to slower hemodynamic changes. Invasive pressures be closely monitored in patients with DD undergoing major surgeries, and caution be executed with the volume status-swings. Good anesthetic-analgesic practices be practiced, avoiding hypertensive crisis, tachycardia, arrhythmias, preserving normal sinus rhythm. Maintaining systolic blood pressure (SBP) within 10–20%, the pulse pressure (PP) should be less than the diastolic BP (DBP). PP indeed increases ventricular wall stress and impairs early diastolic filling. "Rule of 70s" is a useful managing guide here: age > 70, diastolic BP > 70, HR = 70 per minute, and PP < 70. The use of nitroglycerine + titrated phenylephrine combination in difficult cases preserves vascular distensibility, prevents preload reduction, maintaining coronary perfusion at the same time, and stroke volume with minimal cardiac work. It be emphasized here that pure sympathomimetics should be avoided in settings of DD, however inodilators be preferred. Postop hypoxia-hypercarbia, AF, shivering increase propensity to DHF and no knee-jerk large fluid boluses be administered where continuous positive pressure can be particularly helpful in hypoxic setting.

Right heart and PAH:

In general, right heart differs from that of the left in structure and the operating pressures. The RV is different in structure (has an inflow or sinus part and outflow or conus part), coronary perfusion

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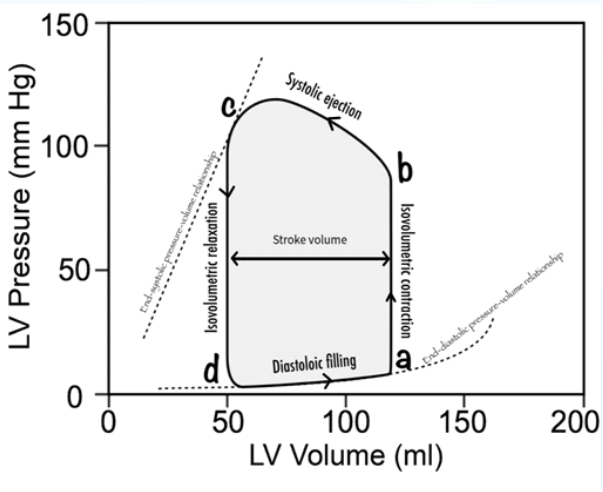
(perfused in both systole and diastole) and function (peristaltic systole which begins earlier and lasts longer). In addition, there are other parameters such as ventricular interdependence, effect of ventilation and pulmonary artery pressure which come in to play in the functioning of this dynamic yet "forgotten chamber". Ventricular interdependence is the mechanical interaction of both ventricles owing to the interventricular septum and pericardial constraint. Pulmonary artery pressure is coupled closely to the RV and issue arises when there is pulmonary hypertension which causes the RV to work against increased afterload. RV hence remodels causing RV hypertrophy (Laplace law). Some patients can have a functional pulmonary regurgitation (PR) and/or tricuspid regurgitation (TR), further causing volume overload catalysing a downward spiral deteriorating RV function.

LV PV Loops:

Pressure Volume (PV) loop represents the changes in LV pressure associated with the changes in chamber volume which occur during a cardiac cycle, with the following information:

1. Pressures:
   - SBP the highest point on the curve
   - DBP the point of opening of aortic valve
   - End-systolic blood pressure being the point where the aortic valve closes
2. Volumes:
   - End-diastolic volume the point where the mitral valve closes and end-systolic volume the point where the aortic valve closes.
   - SV is the difference between the end-diastolic and end-systolic volumes.
   - EF is the ratio of SV to the end-diastolic volume
3. Pressure-volume relationships:
   - Isovolumetric contraction and the relaxation
   - End-diastolic pressure-volume relationship characterizes the ventricular elastance
   - End-systolic pressure-volume relationship relates to the contractility
   - Effective arterial elastance line connects the point of end-diastolic volume to that of the end-systolic pressure and volume, it provides an estimate of the afterload.
4. Areas: Total mechanical energy forms the Stroke work ie. the cumulative area of the P-V loop



a = mitral valve closure
b = aortic valve opening
c = aortic valve closure
d = mitral valve opening

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Cardiopulmonary interactions: the neighbourhood

PPV transmits its' effects to both the left and the right heart but has predominant direct effects on the right heart and derived effects on the left heart. PPV causes increase in the intra thoracic pressure, which is transmitted to the central veins and right atrium causing decrease in the RV preload and to the pulmonary arteries elevating the PVR in-turn the RV afterload. The net effect translates into a decrease in the RV SV. PPV causes fall in the LV preload by causing fall in the pulmonary venous pressure. Also prompting to fall in LV end systolic transmural pressure as well as increased pressure gradient between intrathoracic aorta and extra-thoracic vessels, amounting to decrease in CO in a healthy heart but helping a failing heart by decreasing the preload and afterload. These beneficial effects can be tapped by providing PPV in patients with failing LV whilst considering its effects on RV in the era of HFNC and CPAP warranting judicious use. PPV additionally removes the transudated fluid from the alveoli, helping in resolving pulmonary edema.

POCUS, heart of the matter:

Speaking of RV function, preload, afterload, contractility, cardiopulmonary interaction, ventricular interdependence and pericardial constraint; the practising anaesthetist must be aware of the latest member in the armamentarium, POCUS (Point Of Care Ultra-Sonography). It involves a swift bedside assessment of the heart for preload and volume responsiveness (by assessing the ventricular dimensions, caval size and the collapsibility, RV collapsibility, hepatic venous Doppler and renal venous doppler), pericardial effusion/tamponade, RV dysfunction (RV contractility, EF, hepatic and portal venous Doppler) and LV dysfunction (LV contractility, EF, LV outflow velocity time integral, and SV). At the same time, lung can be assessed with ultrasound to look for pneumothorax, pulmonary oedema, consolidation, lung collapse and pleural effusion; abdomen for free fluid and aortic examination for aortic pathology.

Desirable cardiac grid:

Desirable cardiac grid for efficient functioning of heart is to have combination of an adequate preload, good contractility with normal heart rate and optimal afterload, at which adequate CO is generated with optimal oxygen supply demand balance. To understand this better we can go through the optimal cardiac grid in common valvular diseases and cardiac tamponade.

In mitral and aortic stenosis which are fixed output states, the CO depends on an optimal preload with low heart rates (50-70 beats/min) to allow time for adequate LV filling in case of MS and adequate ejection along with good coronary perfusion time in AS (perfusion in diastole). Here, a volume overload can indeed lead to pulmonary edema as the SV is low and fixed. In addition to maintaining contractility any decrease in the SVR as to maintain the systemic perfusion. Maintenance of sinus rhythm can be cardinal in decreased LV compliance as atrial contraction can contribute up to 40% of LV filling.

In mitral and aortic regurgitation, which operate at increased LV volumes, on the other hand, an augmented preload helps in adequate forward SV. Normal or high heart rates with careful afterload reduction and good contractility is necessary to reduce the amount of regurgitant volume and LVEDP (increase in CPP). In any of the above setting if there is elevated pulmonary pressures (due to left atrial pressures) be solicitous in avoiding hypercapnia, hypoxia or light anaesthesia plane that leads to pulmonary vasoconstrictive responses.

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Of note, cardiac tamponade warrants a high heart rate, adequate preload promoting RV filling, optimized contractility preserving CO in a milieu of fixed reduced SV with avoidance of decrease in SVR to maintain perfusion pressure.

Pregnancy with MS

Pregnancy causes increase in blood volume due to increase in red cell volume as well as plasma volume with latter predominating to contribute to a relative dilutional anaemia. CO increases throughout pregnancy with a sharp rise at around the end of the 1st trimester and escalates through 2nd trimester to as high as 45% above normal in singleton pregnancy and 15% more than singleton pregnancy in twin pregnancy. Systemic arterial pressures decrease in pregnancy with majority of the decline around 8-10 weeks with diastolic pressure drop more than systolic and mean pressures. Heart rate in pregnancy increases by about 10-20 beats/minute which is 20-25% above baseline non pregnant values with no significant increase in left and right ventricular contractility causing the CO to remain normal or slightly elevated. Albeit all these CVP and LVEDP remain unchanged in pregnancy. A glance at these changes will give one, a flash that all the cardiovascular changes of pregnancy are the opposite to desirable cardiac grid of mitral stenosis thus crippling the heart with worsening of the parturient by 1-2 New York Heart Association (NYHA) grades.

Heart failure, not always low EF:

As discussed above, patients with DD are predisposed to DHF, with a special emphasis on the predilection to major adverse cardiovascular event, pulmonary edema, and myocardial ischemia-infarction despite the preservation of EF, ie. >50%. It be however reiterated that HF with preserved ejection fraction (HFpEF) is a wider clinical entity with multiple phenotypes with DHF being an important member of the HFpEF syndrome.

Suggested reading:

- Miller's Anesthesia,10th Edition
- Kaplan's Cardiac Anesthesia: 8th edition
- Braunwald's Heart Disease: A Textbook of Cardiovascular Medicine, 12th Edition
- Thompson A, Fleischmann KE, Smilowitz NR, de Las Fuentes L, Mukherjee D, Aggarwal NR, et al. 2024 AHA/ACC/ACS/ASNC/HRS/SCA/SCCT/SCMR/SVM guideline for perioperative cardiovascular management for noncardiac surgery: a report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. Journal of the American College of Cardiology. 2024 Nov 5;84(19):1869-969.
- Otto CM, Nishimura RA, Bonow RO, Carabello BA, Erwin III JP, Gentile F, et al. 2020 ACC/AHA guideline for the management of patients with valvular heart disease: executive summary: a report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. Journal of the American College of Cardiology. 2021 Feb 2;77(4):450-500.

Rapid review of cardiac anaesthesia

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- Kannan M, Vijayanand G. Mitral stenosis and pregnancy: Current concepts in anaesthetic practice. Indian Journal of Anaesthesia. 2010 Sep 1;54(5):439-44.

Comments:

1. Please mention anaesthetic considerations peri-operatively
2. Commonly used monitoring and diagnostic tools
3. Peri-operative cardiac risk assessment
4. Post-operative common complications and pain management in cardiac cases

Table viva-voce-Anesthetic drugs

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Induction agents

PROPOFOL- (2–6 di-iso-propylphenol)
1% propofol, 2.25% glycerol (isotonicity), 10% soybean oil (lipid base), 1.2% egg phosphatide (emulsifier), preservative - sodium metabusulphite (pH 4.5-6.5) or NaOH (pH 7-8.5). It can be diluted with D5%

OTHER PREPARATIONS

- Ampofol - low lipid emulsion
- Aquavan - water soluble, contains pro-drug fospropofol, prevents lipid associated side effects
- Non-lipid formulations with cyclodextrin carrier

MECHANISM OF ACTION

- It binds to β subunit of GABA A receptor (chloride channel) to prevent dissociation of GABA from the receptor. Chloride influx occurs leading to hyperpolarization and inhibition of post-synaptic neurons.
- It also inhibits ACh release in hippocampus and NMDA receptor

PHARMACOKINETICS

- Onset of action: one brain-arm circulation time; end point - loss of verbal response
- Time to peak effect: 90-100 sec
- Duration of action: 10 min
- Context sensitive half time for infusion of propofol for 8 hrs is < 40 min
- Hepatic metabolism - conjugation. Extrahepatic metabolism - kidney (30%) and lungs.
- Excretion: metabolites and < 0.3% unchanged drug via kidney. 2% of drug is eliminated in feaces

PHARMACODYNAMICS
CNS

- Rapid smooth induction with clear-headed recovery
- Cerebroprotective: reduces ICP, CMRO2 and cerebral perfusion pressure
- Produces burst suppression on EEG: Anti-convulsant
- Lowers postoperative nausea and vomiting
- Increases dopamine in nucleus accumbens - results in drug abuse

RESPIRATORY SYSTEM

- Produces apnea (30–60 sec) after induction in 25–30% patients
- Decreases tidal volume and frequency of breathing
- Bronchodilation
- Laryngeal reflexes abolished

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CVS

- Decreases systolic, diastolic and mean blood pressures
- Inhibits baroreceptor reflex to hypotension
- Decreases PVR: due to inhibition of sympathetic nervous tone
- Reduction in Cardiac output, stroke volume index and cardiac index
- Heart rate: response depends on hypotension and baroreceptor suppression

HEPATIC - prolonged infusion causes hepatocellular injury

RENAL - infusion can increase phenols in urine; green colour urine. However, renal function isn't altered.

USES

- Induction of anesthesia: 1.0-2.5 mg/kg IV
- Maintenance of anesthesia: 50-150 µg/kg/min IV infusion
- Intravenous sedation: 25-75 µg/kg/min IV infusion. Prompt recovery after stopping the infusion due to short context sensitive half time
- TIVA
- PONV: 10-20 mg given IV
- Antipruritic: 10 mg IV
- Anticonvulsant: 1 mg/kg IV reduces seizure duration in patients undergoing ECT
- Laryngospasm: 0.25-0.8 mg/kg IV to deepen the plane of anaesthesia

ADVERSE EFFECTS

- Pain on injection
- Propofol infusion syndrome, if used >4mg/kg/hr for > 48 hrs
- Pancreatitis
- Thrombophlebitis
- Hypertriglyceridemia
- Immunosuppression

THIOPENTONE SODIUM – Barbituric acid derivative.
Pale yellow sodium salt mixed with 6% by weight anhydrous sodium carbonate in 0.5 or 1 gm vial. Diluted with sterile water, D5%, or normal saline to produce a 2.5% solution of pH 10.8; stable for 1 week if refrigerated.
It cannot be reconstituted with acidic solutions such as Ringer lactate, as it results in precipitation.

MECHANISM OF ACTION

- It binds to the GABA A receptor potentiating and mimicking its activity by increasing chloride conductance and causing hyperpolarization of the cell membrane. Thus, increasing the threshold of excitability of the postsynaptic neurone.
- It also inhibits the synaptic transmission of excitatory neurotransmitters, such as glutamate and acetylcholine

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- PHARMACOKINETICS
- Onset: one brain-arm circulation, End point: loss of eyelash reflex
- Peak effect : 60s
- Duration of action: 5 to 10 min
- High lipid solubility, low degree of ionization allows crossing the blood–brain barrier rapidly, producing a fast onset of action.
- Action is terminated by rapid redistribution.
- Hepatic metabolism - inactive, water-soluble metabolites are excreted in the urine.

PHARMACODYNAMICS

CNS

- Hypnotic, sedative, anticonvulsant activity, anti-analgesic
- Decreases CMRO2, slows the EEG.
- Decreases ICP through cerebral vascular vasoconstriction.

RESPIRATORY SYSTEM

- Double apnea (20% cases).

CVS

- Peripheral vasodilation causes mild BP drop
- Decrease in cardiac output because of (1) direct negative inotropic action, (2) decreased ventricular filling, and (3) transiently decreased sympathetic outflow from the CNS.
- Increased heart rate due baroreceptor reflex.
- Cardiac index and MAP, is unchanged or reduced.

OTHERS

- Sustained drug administration increases liver microsomal protein, resulting in accelerated metabolism of drugs - oral anticoagulants, phenytoin, and endogenous substances - corticosteroids, bile salts, and vitamin K.
- Accelerated heme production, which exacerbates acute intermittent porphyria in susceptible patients.

USES

- Induction agent- Dose 3-4 mg/kg
- Anti-convulsant

ADVERSE EFFECTS

- Garlic or onion taste
- Allergic reactions
- Local tissue irritation
- Tissue necrosis
- Accidental arterial injection. Treatment (1) dilution of the drug by injecting saline into the artery, (2) heparinization to prevent thrombosis, and (3) brachial plexus block

Table viva-voce-Anesthetic drugs

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- CONTRAINDICATIONS
- Respiratory obstruction, or status asthamaticus
- Severe cardiovascular instability or shock
- Porphyria

ETOMIDATE – Imidazole derivative
0.2% solution either in 35% propylene glycol or in a lipid emulsion. Latter is associated with less incidence of pain on injection, thrombophlebitis, and histamine release.

Novel Etomidate Derivatives
- Methoxycarbonyl etomidate (MOC etomidate) - doesn't inhibit adrenal steroid synthesis.
- Methoxycarbonyl-carboetomidate (MOC-carboetomidate) - longer duration of action and it doesn't cause adrenal suppression.
- Cyclopropyl-methoxycarbonyl metomidate (CPMM)
- Dimethylmethoxycarbonyl metomidate (DMMM)

PHARMACOKINETICS
- Onset of action: one brain arm circulation
- Duration of action: 5 to 10 min
- Redistribution terminates its action
- Hepatic metabolism: ester hydrolysis.
- 2% of the drug is excreted unchanged, rest eliminated as metabolites by the kidney (85%) and bile (13%).

PHARMACODYNAMICS
CNS
- Facilitates the GABA A receptor resulting in hypnosis.
- Decreases ICP.
- Associated with grand mal seizures and produces increased EEG activity in epileptogenic foci. Thus, useful for intraoperative mapping of seizure foci before surgical ablation.

RESPIRATORY SYSTEM- minimal effect.

CVS- maintains hemodynamic stability due to lack of effect on the sympathetic nervous system and baroreceptors.

ENDOCRINE EFFECT
- Dose dependent inhibition of the enzyme 11 β-hydroxylase, which results in decreased synthesis of cortisol. Thus, continuous infusion is avoided.

USES
Induction agent in cardiac patients undergoing noncardiac surgery, neurosurgical procedures - giant aneurysm clippings, haemodynamic instability
Dose: 0.2 - 0.6 mg/kg (usual dose - 0.3mg/kg)

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ADVERSE EFFECTS

1. PONV
2. Hiccups
3. Myoclonic movement
4. Pain on injection
5. Independent risk factor in the development of an emergence delirium.

KETAMINE HYDROCHLORIDE - Phencyclidine derivative

Racemic mixture of R(-) and S (+) ketamine; latter being 3-4 times more potent analgesic with a faster clearance, recovery and fewer psychomimetic side effects.

Available as partially water soluble 1%, 5% and 10% concentrations for IV/ IM use

MECHANISM OF ACTION

- Inhibition of NMDA receptor- mediated glutamatergic input to the GABA-ergic system which depresses the neuronal cortex (especially association areas) and thalamus, and stimulates parts of the limbic system (hippocampus).
- Dissociative anesthesia resembling cataleptic state.
- At the spinal cord level, it has potent anti-nociceptive effects on NMDAR and inhibits ACh release

PHARMACOKINETICS

- Onset of action: 30-60 sec
- Duration of action: 10-15 minutes.
- Only 12% bound to proteins
- Low mol wt, pKa - 7.5 (near physiologic pH) and relatively high lipid solubility, enables it to cross the blood–brain barrier rapidly.
- Short duration of action due to redistribution
- Metabolized in liver by N-demethylation to form active metabolite- nor ketamine, which is further metabolised and excreted in the urine.

PHARMACODYNAMICS

CNS

- Unconsciousness
- Analgesia. It inhibits nociceptive central hypersensitization. It also attenuates acute tolerance after opiate administration
- Increases cerebral metabolism, CBF, and ICP
- Epileptiform activity on EEG, however, not seen in the cortex. Thus, unlikely to precipitate seizure
- Pupils dilate and nystagmus occurs
- Lacrimation and salivation
- Increased skeletal muscle tone
- Emergence reactions - vivid dreaming, extracorporeal experiences, and illusions.

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RESPIRATORY SYSTEM

- Minimal effect on the central respiratory drive
- Bronchial smooth muscle relaxant; useful in treating status asthmaticus

CVS

- Dominant indirect effect due to activation of the sympathetic system increases in HR and MAP
- Direct cardio depressive effect, seen if trauma or sepsis has depleted catecholamine stores

USES

- Pre-emptive or preventive analgesia: 0.15-0.25 mg/kg IV
- Sedation and analgesia: 0.2-0.8 mg/kg IV over 2-3 min; 2-4 mg/kg IM. Suitable for sedation of pediatric patients undergoing NORA and burns dressing.
- Induction of anesthetic: 0.5–2 mg/kg IV; 4-6 mg/kg IM (Especially in congenital heart disease with right-to-left shunts, unstable cardiovascular or trauma patients)
- Maintenance of general anesthesia: 0.5-1 mg/kg with N20 50%
- Adjunct in Epidural/caudal: preservative-free S (+) ketamine (0.5 to 1 mg/kg) is effective. (Ketamine's preservative - chlorobutanol is neurotoxic)
- Anti-depressant: 0.5 mg/kg, given as a 40-minute infusion. It results in a dramatic mood change within a day, often lasting for 3 to 12 days. A maintenance dose every 2 to 4 days lengthen this effect.
- Treatment of acute and chronic pain - cancer pain, CRPS, phantom and ischemic limb pain etc

CONTRAINDICATIONS

- In spontaneously breathing patients with brain trauma, it can increase ICP and cause apnea.

however, it retains response of CBF to carbon dioxide, and is neuroprotective in mechanically ventilated brain trauma patients.

- In patients with an open eye injury or other ophthalmologic disorder, it can cause increase in intraocular pressure.
- It can't be used in IHD patients as it has propensity to increase HR, BP and myocardial oxygen consumption. Also, unsuitable for patients with severe right heart dysfunction, as it increases PVR.

Opioids

REMIFENTANIL-Synthetic opioid (4-anilido-piperidine compound)
Pure μ-opioid receptor agoinst, Potency similar to fentanyl, Rapid onset and offset
Ultra short duration of action mandates infusion to ensure sustained opioid effect.
Pharmakokinetics-Peak effect: 1-3 minute
Context sensitive half life: 4 minutes after 4 hr infusion
Metabolism by non specific plasma and tissue esterase, thus, not affected by renal or hepatic failure. Major metabolite - remifentanil acid (inactive) undergoes renal excretion.

Allows rapid emergence from anesthesia without postoperative respiratory depression
Suppress automatic, hemodynamic, and somatic responses to noxious stimulation
Exposure to high doses of remifentanil may paradoxically reduce the pain threshold after its discontinuation, resulting in postoperative hyperalgesia.
Uses:
1. Induction and maintenance of anaesthesia-Loading dose: 1-2 g/kg, Maintenance infusion rate: 0.1-1.0 g/kg/min
2. Component of TIVA (0.25-0.5 g/kg/min)
3. Post operative analgesia (0.05-0.15 g/kg/min)
4. Conscious sedation
CONTRAINDICATION: not used in neuraxial adjuvant as the preparation has glycine

Muscle relaxants

SUCCINYLCHOLINE (=Suxamethonium)- two molecules of acetylcholine linked together.
Clear aqueous solution - 50 mg/ml
Routes - IM, IV
MECHANISM OF ACTION
Depolarizing block (= phase I block) produces prolonged depolarisation of end plate, which leads to desensitisation of eAChR and subsequent relaxation. It is preceded by muscle fasciculation resulting from initial muscle activation.Phase II block (seen on neuromuscular monitoring) occur on repetitive dosing. The neuromuscular membrane becomes depolarised but is desensitised to acetylcholine.
USES
1. Facilitate tracheal intubation during RSI. Dose: 1-1.5 mg/kg IV in adults; 2.5 mg/kg IM
1. Treatment of laryngospasm. Dose: 0.1-0.5 mg/kg IV; 3-4 mg/kg IM
PHARMACOKINETICS & PHARMACODYNAMICS
Peak effect: 60 seconds.
Duration of action: 9 to 13 minutes
The short duration of action is due to its rapid hydrolysis by butyrylcholinesterase (plasma cholinesterase)
SIDE EFFECT

- CVS- sinus bradycardia, junctional rhythm, and even sinus arrest may occur. SCh at autonomic nervous system ganglia may produce ganglionic stimulation and associated increases in heart rate and systemic blood pressure. Ventricular dysrhythmias have been attributed to autonomic stimuli associated with laryngoscopy and tracheal intubation.
- Hyperkalemia - 0.5 mEq/dL increase in healthy individuals. Severe hyperkalemia seen in conditions associated with upregulation of extra junctional acetylcholine receptors (burn, severe metabolic acidosis, closed head injury, hemiplegia or paraplegia, muscular dystrophies, Guillain-Barré syndrome). SCh is to be avoided post 24 hrs of acute burns upto 2 years.
- Myoglobinuria - damage to skeletal muscles, especially to pediatric patients, in patients with malignant hyperthermia or occult muscular dystrophy.

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- Increased Intraocular Pressure - peaks at 2-4 min and returns to normal by 6 min.
- Increased Intragastric Pressure
- Increased Intracranial Pressure
- Myalgia
- Masseter Spasm
- Trigger agent for malignant hyperthermia

ADRENALINE-Available in 1ml ampoule containing Adrenaline bitartrate salt
Routes - IV/IM/SC/IO/ET
RECEPTORS -beta1>beta2> alpha1
ACTIONS
Alpha1 receptors on heart increases SBP, DBP, MAP
Beta2 receptors on lungs cause bronchodilation, inhibits histamine release
USES

- CPR - asystole, PEA, ventricular tachycardia, ventricular fibrillation
  1 mg IV/IO (adult); 0.01mg/kg IV (children) every 3-5 min
  Endotracheal dose: 2-2.5 times of IV dose
- Anaphylaxis
  50 mcg IV (adult); 1mcg/kg IV (children); 300-500 mcg (IM)
- Septic shock - though it is not a first line of drug for hypotension. Vasoconstrictor action to increase cardiac output
  0.05-2 mcg/kg/min IV infusion
- Bradyarrhythmia (AHA) - 2-10 mcg/min IV infusion titrated to response in an adult patient
- Treatment of severe croup, severe asthma, bronchospasm

Racemic epinephrine (2.25%) nebulisation - 0.05 ml/kg to a max of 0.5 ml diluted in NS to 5 ml.L-adrenaline - 0.5ml/kg of 1:1000 solution (max 5ml)
300 mcg S/C, can be repeated after 20 mins up to 3 doses
ADVERSE EFFECTS

1. Cardiac arrhythmia
2. Local ischemic necrosis

Suggested Reading:

- Gropper MA. Miller's anesthesia. 10th ed. Vol. 1. Philadelphia, PA: Elsevier; 2024.
- Cullen BF, M. Christine Stock, Ortega R, Sharar SR, Holt NF, Connor CW, et al. Barash, Cullen, and Stoelting's Clinical Anesthesia. Lippincott Williams & Wilkins; 2023.
- Flood P, Rathmell JP, Urman RD. Stoelting's Pharmacology and Physiology in Anesthetic Practice. 6th ed. Philadelphia: Wolters Kluwer Health; 2021

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Comments:
The article has been written very elaborately so please reduce the total word count to 1500-1600 only. You can replace the text by table wherever feasible to cut short on the word count. Thank you

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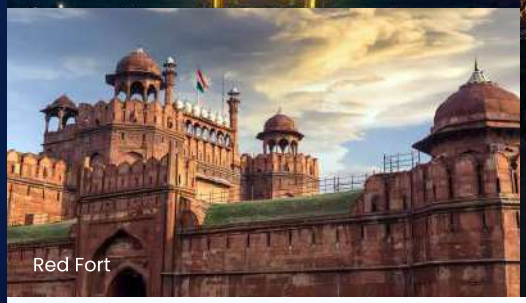
Eternal Vigilance


Akshardham Temple


Lotus Temple


Red Fort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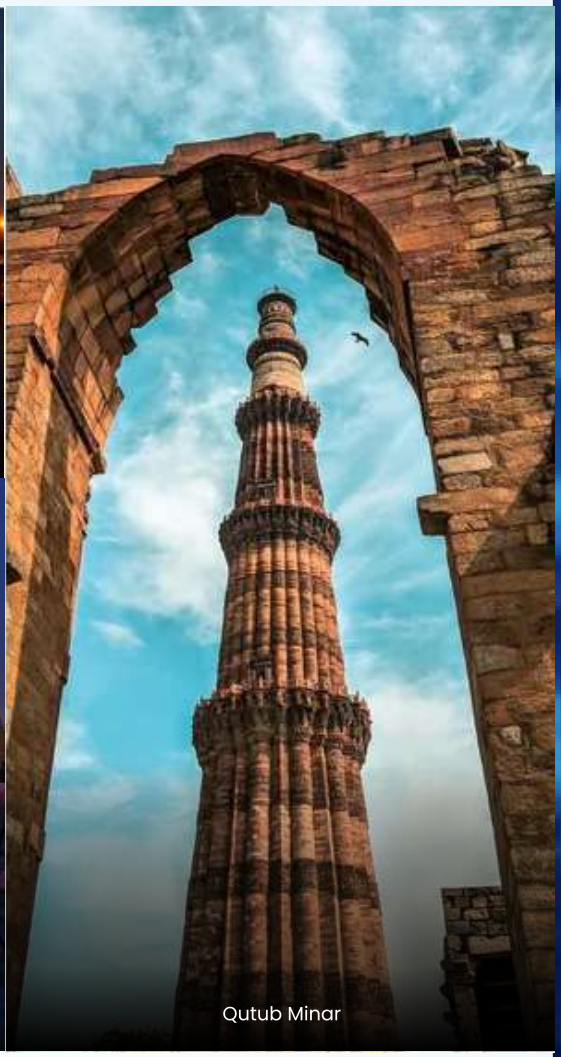
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