Oxygen therapy

Discovered by Joseph Priestley and Antoine Lavoisier in 1774

Oxygen cascade:

Oxygen tension:(1kpa=7.5 mmHg) Inspired air(160 mmHg). expired air(116). Alveoli(104). Arteries(100). Veins(40). cells(2)

O2 is carried in blood: Attached to hemoglobin: responsible for O2 saturation Dissolved in blood: free; responsible for O2 tension(PO2) O2 is metabolized in citric acid cycle: Aerobic 38 ATP, Anaerobic 2ATP + Lactic acid By mitochondria **Utilized oxygen** = Arterial PO2 – Venous PO2

Oxygen flux: The amount of oxygen leaving the left ventricle in a minute = C.O cc/min x Arterial O2 saturation% x Hb gm/100 x 1.34 ml of O2 attached to each 1 gm Hb = $5000 \times 97/100 \times 16/100 \times 1.34 = 1000$ cc O2/min . Only 250 cc is used by cells

Oxygen dissociation curve

Gives the relation between PO2 and Hb saturation with O2 Shift to the right : Anemia Acidosis Increase PCO2 Increase temp. Increase 2,3-diphosphoglyceric acid(DPG)

P50: the PaO2 needed to make Hb saturation 50% = 26 mmHg



PO₂(mmNg)

Indications of O2 therapy:

Cyanosis (reduced Hb>5gm) Chest injuries Shock To decompress distended bowel by nitrogen CO poisoning High metabolic rate

Hypoxia: lack of O2

Hypoxic hypoxia: high altitude, diffusion hypoxia Anemic hypoxia: decrease Hb, CO poisoning, Sickle, massive blood transfusion Stagnant hypoxia: Shock, embolism Histotoxic hypoxia: cyanide poisoning, tissue edema

Methods to overcome hypoxia:

Measures to increase oxygen availability Raise the arterial oxygen tension Raise the hemoglobin content Remove acidosis Increase the cardiac output Measures to reduce oxygen requirement Paralysis and IPPV Digitalization Prevent hyperpyrexia Induce hypothermia (in 30C O2 requirement is only 40% of normal)

O2 therapy is indicated when PO2 decreases below 60 mmHg

Delivery of oxygen:

Nasal cannula: minimal patient discomfort extent 1cm into nares 6L/min Face nask: flow 5-8L/min Oxygen tent Hyperbaric oxygen chamber.....indications

Hazards of oxygen therapy:

-Retrolental fibroplasia: specially in neonates with low birth weight. High O2 result in vasoconstriction of immature retinal vessels leading to neovascularization and scarring which leads to impaired vision.

-CO2 retention: In chronic obstructive airway diseases the patient get used to high PCO2 and low PO2. When we remove the low PO2 they get CO2 retention.

-Atelectasis: Normally nitrogen in alveoli is in equilibrium with that in the pulmonary capillary blood and its loss is unlikely, this nitrogen act as a splint to alveolar wall. When oxygen is given, it will wash the alveolar nitrogen leading to collapse.

-Pulmonary oxygen toxicity: Prolong exposure of alveoli to O2 causes pulmonary damage by formation of hyaline(fibrin) membranes with alveolar septal thickening and endothelial destruction, also there will be destruction of mucociliary clearance.

Postoperative hypoxia:

Diffusion hypoxia

Lower functional residual capacity(FRC) than closing volume as in geriatrics and smokers Decrease cardiac output which will decrease O2 flux

Hypoventilation: Recurarization: because of vasoconstriction

Opiates such as fentanyl

Obstruction: tongue, secretion, vomiting, foreign bodies Pain

Dry gas harmful effects:

Ciliary paralysis: Dry gas arrested the activity of mucociliary transport system. If combined with increased musus viscocity(thick) will lead to inspissation and encrustation of mucus leading to inflammation and atelectesis

Decrease mucus flow from tracheobronchial epithelium: In children as they have narrow and small airways so will be easily blocked

Hazards of Humidification:

Overheating leads to hyperpyrexia, it is good in burn patients only as they loss heat Overhydration: specially in small children

Infection: due to contamination of the chamber with bacteria specially G-ve ...pneumonia Increased airway resistance, specially if the solution nebulised by ultrasound not heat Bronchopneumonia-like changes