Chapter 5

Respiratory Management and Ventilators

Patients often require mechanical ventilatory support in the postoperative period.

The duration of support differs with or without reactive pulmonary vascular disease.

Patients without pulmonary vascular disease may be extubated in 4-6 hours postoperatively. Patients with pulmonary vascular disease need prolonged ventilator support and special care, and these patients usually have postoperative pulmonary hypertension crises. These candidates are as follows:

Complete AV canal, truncus arteriosus, obstructed total anomalous pulmonary venous connection (TAPVC), multiple VSD's, and patients with late pulmonary artery (PA) bands or inadequate PA bands.

Patients with pulmonary vascular disease do require supplementary monitoring during the postoperative period for adequate management of ventilator support. These patients require in brief the following management:

Pulmonary artery line is inserted in OR for monitoring and infusion of drugs (see below).

Use of paralyzing agents (vecuronium) and sedation (fentanyl) in the postoperative period.

Support with mechanical ventilation for a minimum of 24 hours.

After stabilization of respiratory and cardiovascular hemodynamic status and improvement, withdraw paralyzing agent first.

Patient should not be manipulated or disturbed unless absolutely necessary.

Avoid loud noises to prevent pulmonary hypertensive crises.

5.1 Initial Ventilator Orders

5.1.1 Ventilator Settings

Pressure vs. Volume controlled: Volume to begin with and it meets the goals of ventilation.

i) Mode:

PRVC (pressure regulated volume controlled ventilation) if available / or SIMV (synchronous intermittent mandatory ventilation) with or without pressure support (PS).

PRVC guarantees tidal volume (TV), and limits the peak airway pressure.

Decelerating wave pattern on the flow in PRVC is well tolerated.

ii) Respiratory rate (RR): 20

For small children this is lower than their usual spontaneous rate, but larger TV that is delivered increases the minute ventilation.

For large children decrease rate to 15 or less.

For small infants or neonates increase RR to 30 or higher.

iii) PEEP (positive end-expiratory pressure): 5 mm Hg.

It is slightly above the physiologic range.

PEEP should not be so high as to cause barotrauma / cardiac compromise.

iv) FiO_2 (inspired oxygen concentration): (1.0) or 100%

Allows maximal preoxygenation if any thing adverse happens.

Start to wean once everything is stable.

Deleterious effects occur from FiO_2 1.0, even for brief periods in those with:

1. Single ventricular physiological correction with outflow into PA (pulmonary artery) and AO (ascending aorta) occurs in series.

2. Systemic arterial to pulmonary artery shunts (i.e., modified Blalock-Taussig or central shunt).

The cause of adverse effects are due to pulmonary vasodilatation (↓ PVR), and ↑ pulmonary blood flow, and both can lead to systemic hypoperfusion and hypotension.

In above situations, rapid weaning of FiO_2 (preferably to room air or 0.3) should be done to maintain SaO_2 of 70-80%.

Tailor the FiO_2 to maintain SaO_2 of 80-85% in situations (i.e., bidirectional Glenn, hemi-Fontan) where free mixing of pulmonary venous and systemic venous blood occurs.

v) Tidal volume (TV): 8-10 mL/kg.

It gives good alveolar distention without significant barotrauma.

Generally use PEEP to maintain lung volume and smaller TV to avoid barotrauma.

TV of 10-12 mL/kg may also be used as the standard range.

vi) Inspiratory time (I time): 0.5 to 1 second.

It is physiological, but use longer I-time for adolescents and bigger children.

The shorter I-times allow maximal time for exhalation (i.e., in asthmatics and obstructive lung disease).

5.1.2 Ventlatory Goals

Following parameters are, especially, useful for patients with pulmonary vascular disease.

Keep PaO_2 100 mm Hg, blood fully saturated with O_2 (100%) ♣

Keep $PaCO_2$ 25-30 mm Hg.

Keep pH between 7.5 to 7.55

The patients may require frequent hand ventilation with 1.0 FiO_2.

Suction the airway frequently to prevent pooling of secretions and atelectasis.

The ♣ ideal SaO_2 is 80 to 85% with PaO_2 of 40 mm Hg in the following:

Bidirectional Glenn, hemi-Fontan, stage I Norwood correction of hypoplastic left heart syndrome (HLHS), aorto - pulmonary shunts (i.e., modified Blalock-Tausig, or a central shunt), and lesions or corrections in which free mixing of pulmonary venous and systemic venous blood occurs.

5.1.3 Precautions on Ventilator

A. Peak Pressures:

The airway peak pressures should be kept under 35 if at all possible.

May change to pressure control (PC) mode of ventilation if peak pressure goes into the 40's to 50's persistently. The latter may imply restrictive lung disease or non-compliant lungs with obstructive pattern.

One should try other manipulations before change to PC mode.

Improve bronchodilation and evaluate pause pressure.

B. Oxygenation:

Inability to wean the FiO_2 is a concern, and get the FiO_2 under 60% soon as possible. Failure to wean implies shunting due to lack of airway recruitment (PEEP too low) / or adult respiratory distress syndrome (ARDS). Increasing the mean airway pressure may be of use.

C. Ventilation:

The adequacy of ventilation is based on patient's needs, and is reflected on $PaCO_2$.

Shock and metabolic acidosis require higher rates for compensation.

Obtaining a blood gas early 15-20 minutes after on the ventilator will give proper direction.

5.2 Controls in Mechanical (Positive Pressure) Ventilation

5.2.1 Oxygenation

Oxygen uptake by the lungs, though is dependent on a number of factors, it can be manipulated by mechanical ventilation as described below:

i) Arterial oxygen tension (PaO_2):

Manipulated by altering inspired oxygen concentration (FiO_2), alveolar pressure, and ventilation.

ii) Ventilation-perfusion matching:

Intrapulmonary shunting decreases by re-opening the collapsed alveoli. (PEEP) helps to re-open alveoli, and splint the alveoli open and increases in lung compliance (see Figure 21 and 22).

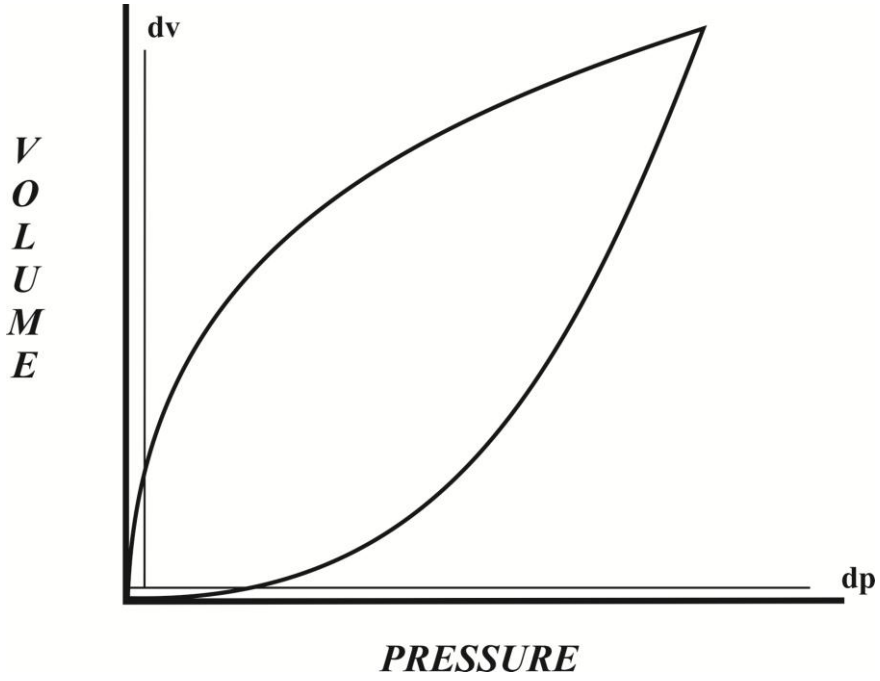

Figure 21 *Lung compliance (unit change in lung volume per unit change in pressure (dv / dp) is represented by a volume loop curve.*

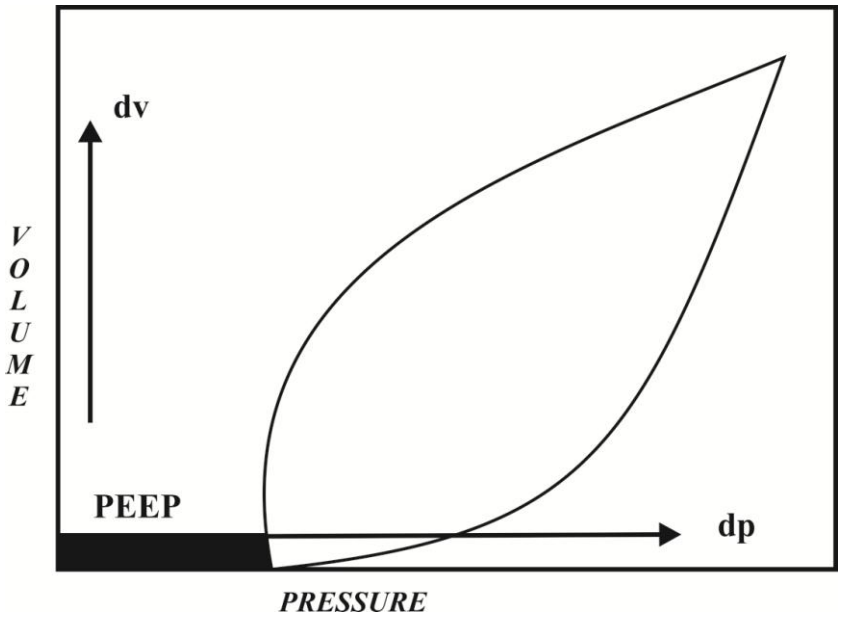

Figure 22 *Addition of PEEP shifts the volume loop curve upward and right representing increased lung compliance with increased air entry. PEEP= positive end expiratory pressure, dv- change in lung volume, dp= change in alveolar pressure.*

To improve oxygenation do the following:

Increase FiO_2, increase mean alveolar pressure, increase mean airway pressure ↑ PEEP, and ↑ I: E ratio.

5.2.2 Carbon Dioxide (CO_2) Elimination

CO_2 elimination via the lungs is largely dependent on alveolar ventilation.

$$Alveolar\ ventilation = Respiratory\ rate \times (Tidal\ volume - Dead\ space\ volume)$$

To improve CO_2 elimination: ↑ respiratory rate and ↑ tidal volume.

5.2.3 Inspiratory Time, Inspiratory Pause, and I: E ratio

Inspiratory time is the time over which the set tidal volume is delivered (as in volume control ventilator) or the pressure is maintained (as in pressure control ventilation).

Inspiratory time or I; E ratio:

These parameters are set in time-cycled modes; set tidal volume is delivered in that time. The ventilation modes can be pressure control (PC), volume control (Siemens and Drager ventilators), or pressure regulated volume control (PRVC).

Inspiratory flow is set in volume-cycled modes, (Puritan-Bennett and Bear ventilators). Inspiration ends when set TV is delivered. In pressure support mode patient determines the duration of inspiration.

Inspiratory pause time: It is only set in modes where a fixed tidal volume is set and delivered. Expiratory time is whatever time is left over before the next breath, i.e., as in volume control and volume preset SIMV modes.

I: E ratio:

(Inspiratory time + Inspiratory pause time): Expiration.

Usually set to 1:2 to mimic usual pattern of breathing.

Advantages/disadvantages of longer inspiratory times:

i) Improves oxygenation by ↑ mean airway pressure (MAP). Longer period of high MAP is existent over the entire respiratory cycle, by allowing re-distribution of gas from more compliant alveoli to less compliant alveoli.

ii) Reduces expiratory time and increased risk of gas trapping, intrinsic PEEP and barotrauma. Poorly tolerated by the patient and deeper level of sedation is needed.

iii) Decreases peak pressure by decreasing the inspiratory flow rate.

5.2.4 Inverse-ratio Ventilation

I: E ratio: 1:1 or 2 to 3:1.

Pressure-controlled inverse-ratio ventilation may be used in for children with severe respiratory distress syndrome (ARDS).

5.2.5 Trigger Sensitivity (TS)

Determines how easy it is for the patient to trigger the ventilator to deliver a breath. TS may be flow-triggered or pressure (negative pressure) triggered.

Flow triggering is generally more sensitive.

Smaller the flow or the smaller the negative pressure the more sensitive the trigger. Increasing the TS is preferable to improve patient-ventilator synchrony (stops "fighting" the ventilator). Excessively high sensitivity may result in false or auto-triggering.

5.3 Cardiovascular Effects of Mechanical Ventilation (MV)

5.3.1 Preload

Positive intrathoracic pressure reduces venous return.

Reduction in preload is exacerbated by high inspiratory pressure, prolonged inspiratory time, and PEEP.

5.3.2 Afterload (T)

Afterload is equivalent to ventricular wall tension (T) during myocardial contraction.

$$T = Ptm \times R/2H$$

Ptm = transmural myocardial pressure, R = radius, and H = wall thickness of ventricle.

Ptm = (intracavity pressure of the ventricle − pleural pressure)

Positive pressure ventilation by ↑ pleural pressure and ↓ transmural pressure decreases afterload.

5.3.3 Cardiac Output (CO)

Reduced preload will tend to ↓ cardiac output, but reduced afterload tend to ↑ cardiac output. Net effect on CO depends on left ventricle (LV) contractility.

In patients with normal LVcontractility, mechanical ventilation (MV) tends to ↓ CO.

In patients with decreased LV contractility, MV tends to ↑ cardiac output.

Failure to wean from MV may be due to failure to cope with increased preload and afterload.

5.3.4 Myocardial Oxygen Consumption

It is reduced by positive pressure ventilation with improved myocardial oxygen supply/demand balance and improved myocardial performance.

5.4 Evaluation of Gas Exchange

5.4.1 Arterial Blood Gas (ABG)

Table 5.1 *The normal arterial blood gas values.*

	PaO_2 (mm Hg)	**$PaCO_2$ (mm Hg)**	**$'HCO_3$ (mEq/L)**	**pH**
Infants < 2 years	90	34	20	7.40
Children < 18 years	96	37	22	7.39
Adult	90-110	35-45	22-26	7.35-7.45

PaO_2 = Arterial oxygen tension, $PaCO_2$ = arterial carbon dioxide tension.

5.4.2 Analysis of Acid-Base Disturbance

It is based on Henderson-Hasselbach Equation:

i) 10 mm Hg rise or fall in $PaCO_2$ accompanies in 0.08 fall or rise in pH respectively.

ii) 0.15 rise or fall in pH accompanies 10 mEq/L fall or rise in HCO_3^- respectively.

iii) Determine the primary disturbance by first noting pH; $PaCO_2$ and then HCO_3^-.

Analyze the acid base distrubance by a secondary compensatory change (see Table 5.2).

Table 5.2 *Analysis of the acid base disorder.*

	pH	**$PaCO_2$ (mm Hg)**	**HCO_3 (mEq/L)**
Acute respiratory acidosis (pure)	↓ (7.26)	↑ (55)	(24)↔
Acute resp acidosis (compensated)	↓ (7.26)	↑ (55)	27, ↑ by 1 mEq for each 10 mm Hg rise of $PaCO_2$
Chronic resp acidosis (compensated)	↓ (7.3)	↑ (55)	31, ↑ by 4 mEq for each 10 mm Hg rise of $PaCO_2$
Acute resp alkalosis (pure)	↑(7.48)	↓ (25)	(24)↔
Acute resp alkalosis (compensated)	↑ (7.48)	↓ (25)	21, ↓ by 1-3 mEq for each 10 mm Hg fall of $PaCO_2$
Chronic resp alkalosis (compensated)	↑ (7.44)	↓ (25)	19, ↓ by 3-5 mEq for each 10 mm Hg fall of $PaCO_2$
Acute metabolic acidosis (compensated)	↓(7.2)	↓(30)	16, ↓ $PaCO_2$ by 1-1.5 × fall in HCO_3
Acute metabolic alkalosis (compensated)	↑ (7.5)	↑(44)	36, ↑ $PaCO_2$ by 0.25-1 × rise in HCO_3

5.4.3 Venous Blood Gas (VBG)

Peripheral venous sample:

It is affected by local environmental, circulatory, and metabolic conditions.

May have good correlation for pH, but not for PaO_2.

Central Venous sample:

It may correlate well with acid base status.

Useful for determining shunt fraction and oxygen consumption by estimation of PaO_2 and SvO_2.

5.4.4 Capillary Blood Gas (CBG)

It has good correlation for arterial pH, but correlation is only moderate value for $PaCO_2$.

It correlates poorly for $PaCO_2$.

5.4.5 Pulse Oximetry

Oxygen saturation estimation is done non-invasively through light absorption by pulse oximetry, It differentiates characteristics of oxygenated and deoxygenated hemoglobin.

Oxygen saturation empirically derived from algorithm based on oxyhemoglobin dissociation curve. It doesn't estimate O_2 delivery to tissues.

For accuracy patients heart rate should match oximeter pulse reading.

The readings are unreliable in hypothermia, hypovolemia, and shock due to improper detection of patient's pulse.

Spurious reading (high or low) occurs in methemoglobinemia, patient motion, under xenon or surgical lamps, and electrosurgical interference.

Spurious decrease by intravenous dyes, e.g., methelene blue or nail polish.

Spurious increase by carboxyhemoglobin (smoke inhalation) and cyanide poisoning.

Best to monitor oxygenation in acutely ill, during sleep, during feeding, during periods of exercise, brdycardias, or periods of apnea.

Marginal decrease in O_2 saturation may imply poor oxygen carrying capacity (i.e., anemia, acute or chronic blood loss).

5.4.6 Capnography

Infrared spectroscopy measures the CO_2 concentration of expired gas or the end-tidal CO_2. The end-tidal CO_2 ($ETCO_2$) approximates $PaCO_2$.

Capnography monitors ventilation in mechanically ventilated.

It is useful for detection of ET tube leaks which results in ($\downarrow ETCO_2$)

Pulmonary vascular obstruction and V/Q mismatches $\downarrow ETCO_2$.

5.4.7 Oxy-hemoglobin Dissociation Curve

Most of O_2 in blood is bound to Hb (one molecule of Hb binds to 4 molecules of O_2). The affinity of O_2 to Hb is dependent on O_2 tension and O_2 satuaration curve is sigmoid (see Figure 23). The P50 (i.e., 50% of hemoglobin is satuarted with O_2) value therefore varies.

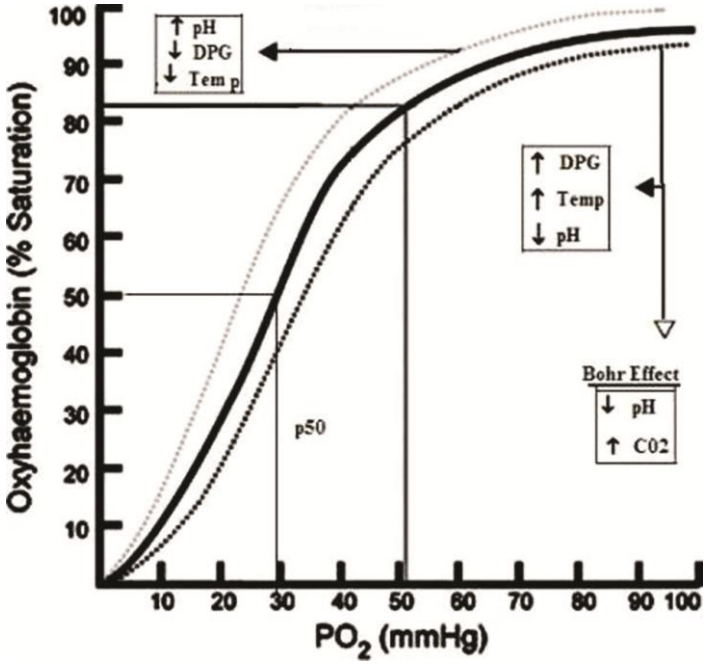

Figure 23 *Oxy-hemoglobin dissociation curve. Note the curve is sigmoid and oxygen saturation of hemoglobin molecule plateaus beyond PO_2 > 70 mm Hg. Solid line represent a normal curve. Normal P50 (O_2 saturation of Hb is 50%) value is PO_2 of 30 mm Hg. Shift of curve rightwards (broken line) ↑ the P50 value (PO_2 > 30 mm Hg) and oxygen is readily released from Hb, a situation that persists in tissues. Shift of curve to left (broken line) ↓ the P50 value (PO_2 < 30 mm Hg) with increased binding of O_2 to Hb molecule. Note ↑ DPG (diposhoglycerate), ↑ temperature, ↓ Ph, and increased CO_2 (Bohr effect) shifts the curve to right with more release of O_2 from hemoglobin. ↓ Temperature, ↓ DPG, and alkalosis increase the binding of O_2 to Hb with less O_2 release.*

Shift of curve to left: (P50 value ↓ i.e., O_2 tension, in which 50% of hemoglobin is saturated with O_2 is lower (PO_2 40 mm Hg or less). More O_2 is bound to hemoglobin (as in lungs to facilitate O_2 binding).

It occurs in alkalosis, hypothermia, and hypocarbia.

Shift of curve to right: (P50 value ↑ ie: O_2 tension in which 50% of hemoglobin is satuarted with O_2 is higher (PO_2 55-60 mm Hg). Less O_2 is bound to hemoglobin to facilitate delivery to tissues.

It occurs in acidosis, hyperthermia, hypercarbia, and due to ↑ 2, 3 diphospho-glycerate levels.

Bohr effect (effect of CO_2 and H^+ ion on oxyhemoglobin (O_2Hb) dissociation curve):↑ CO_2 and ↑ hydrogen ions in blood shift O_2Hb curve to right and ↓O_2 binding to Hb.

↓ CO_2 and ↓ H^+ ions in blood shifts O_2Hb curve to left and ↑ O_2 binding to Hb

Haldane effect effect of O_2Hb dissociation curve on CO_2 transport is shown in figure 24.

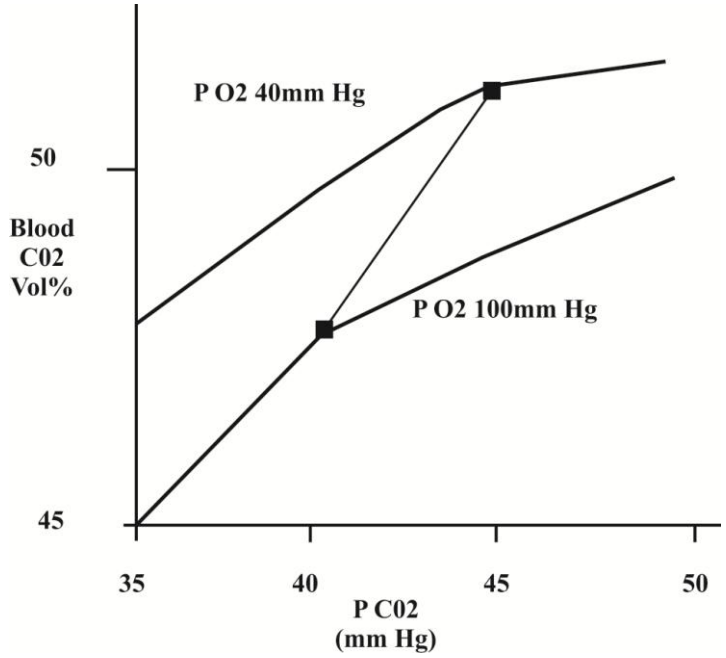

Figure 24 *Section of blood carbon dioxide dissociation curve and Haldane effect. Increased binding of oxygen to hemoglobin (oxyhemoglobin) dissociates* $\boldsymbol{C0_2}$ *from blood, a situation that persists in lungs. Decreased oxyhemoglobin saturation of blood (as present in tissues) facilitates binding of* $\boldsymbol{C0_2}$ *to Hemoglobin. The curve is almost linear. Note at blood* $\boldsymbol{P0_2}$ *of 100 mm Hg, the blood* $\boldsymbol{P0_2}$ *is 40 mm Hg and* $\boldsymbol{C0_2}$ *content of blood is 48 volumes% (as in lungs). Note at blood* $\boldsymbol{P0_2}$ *of 40 mm Hg, the blood* $\boldsymbol{P0_2}$ *is 45 mm Hg and* $\boldsymbol{C0_2}$ *content of blood is 52 volumes % as exists in tissues.*

This effect is more pronounced than Bohr effect (effect of $C0_2$) on O_2 transport.

↑ binding of O_2 to Hb (shift of O_2Hb curve to left), and ↓ binding of CO_2 to Hb releases CO_2 from hemoglobin (as in normally ventilated lungs).

↓ binding of O_2 to Hb (shift of O_2Hb curve to right), ↑ binding of CO_2 to Hb to take up CO_2 released from metabolic pool (as in tissues).

Under normal conditions, the net total transport of $C0_2$ is 4 mL/dL of blood carried from tissues to lungs. It occurs in three forms. i) 7% in dissolved form (0.3 mL), ii) 70% as plasma bicarbonate (2.8 mL), (carbonic acid is formed in red cells by an enzyme carbonic anhydrase, and HCO_3 transfer from red cell to plasma is facilitated by chloride shift to red cells; inhibition of carbonic anhydrase in red cells interfere with CO_2, transport and tissue PCO_2 increases to 85 to 90 mm Hg), iii) 22 to 25% in combination with Hb molecule and plsama proteins (0.8 mL to 1 mL).

It is ideal to maintain adequate tissue perfusion and flow, with adequate alveolar ventilation to promote optimal O_2 transport and delivery CO_2 transport and elimination.

5.5 Management of Acute Respiratory Decompensation and Evaluation of Hypoxia / Hypercarbia

5.5.1 Hypoxia

Blood PaO_2 of < 70 mm Hg on supplemental O_2 and SaO_2 of < 95% is considered as hypoxia if it occurs in all biventricular corrections with separated systemic and pulmonary circulations.

A. Evaluate for several following causes of hypoxia:

1) Hypoxemic hypoxia: It is characterized by a low arterial O_2 tension.

It occurs due to V/Q mismatch (pulmonary embolism, atelectasis, pulmonary edema), intracardiac shunts, and hypoventilation.

2) Circulatory hypoxia: It occurs due to decreased capillary flow or perfusion.

It is commonly due to crtical decrease in systemic cardiac output, decreased pulmonary blood flow (through shunts, or imbalance in systemic/pulmonary circulation in single ventricle physiology), and sepsis.

3) Anemic hypoxia: Occurs due to deficient O_2 carrying capacity of hemoglobin.

It is observed in anemias (low hemoglobin content of blood), carbon monoxide poisoning with elevated carboxyhemoglobin levels. ↑ carboxyhemoglobin levels in blood do not affect blood PO_2 and it may be normal in presence of tissue hypoxia, and methemoglobinemia (nitroglycerin (NTG) infusion toxicity).

Nitroglycerin infusion toxicity (methemoglobinemia):

(Nitroglycerin converts ferrous iron to ferric with ↓ binding of Hb molecule to O_2.

(SaO_2 and PaO_2 spuriously ↑ or normal. The blood turns into choclate brown color on exposure to atmospheric O_2.

4) Hisotoxic hypoxia: It is due to decreased O_2 utilization at a cellular level.

It occurs in cyanide poisoning (cyanide is a byproduct of nitroprusside). It is clinicaaly related to nitroprusside infusion toxicity. Liver enzme rhodnase converts cyanide to thiocynate and is excreted by kidney.

The toxic levels of thoicynate is 5-10 mg/dL and should be treated with thiosulphate infusion (see Chapter 16, section).

B. Management of hypoxia:

Act immediately and perform following maneuvers:

1) Disconnect the ventilator.

2) Use hand ventilation:

While giving appropriate Fi O_2 for the patient (i.e., 1.0 for biventricular corrections), perform the following tasks:

i) Perform thorough physical examination and ensure adequate hemodynamic status and perfusion.

ii) Clear the airway secretions (from upper and lower airways), assure airway patency (i.e., ET tube) and correct position of ET tube, bilateral chest movement, and bilateral ventilation of lungs.

iii) Get ABG, mixed venous blood gas, and chest roentgenogram.

A. If manual ventilation is adequately accomplished:

Check the hemodynamic status of the patient, and if the patient is:

1) Hemodynamically stable:

Address or exclude ventilator malfunction, reconnect the ventilator, and evaluate the patient by the results of the tests performed as discussed below.

i) Roentgenogram: Position of ET tube, pulmonary infiltrates, pulmonary edema, atelectasis, and pleural fluid collections in chest cavity.

ii) Intrapulmonary shunting: Normal < 5%. Greater degrees suggest respiratory causes, which improve with additional oxygen due to ↑ recruitment of alveolar-capillary gas exchange units.

Intracardiac shunts (fixed) show no improvement in gas exchange with above measures.

iii) Oxygen transport: The oxygen content of blood and systemic cardiac output are assessed along with estimate of pulmonary / systemic blood flow ratio (see Chapter 17).

Blood flow is also evaluated by echocardiogram and clinical indices of perfusion.

iv) Oxygen extraction ratio: Normal 25-35, against O_2 content of blood.

Increased ratios suggest increased metabolic demand as a cause of hypoxia.

v) Oxygenation index: Normal < 35

Increased index suggests progressive respiratory failure.

2. Hemodynamically unstable patient:

Exclude acute and large accumulations of pleural air and/or fluid/blood, acutely diminshed pulmonary blood flow (PBF) or pulmonary embolus (PE) in larger children and adolescents. Also exclude sepsis, hypovolemia, dimished cardiac output and/or cardiac compression.

Stabilize hemodynamic status with judicious administration of fluids, and institute appropriate measures based on clinical, radiological, and echocardiographic diagnosis.

Manipulate the ventilator settings depending on the cause and optimize O_2 transport. Consider need for ECMO (see Chapter 11).

B. Manual ventilation is difficult:

Immediately evaluate and address the following causes:

1) Obstructed ET tube:

Perform endotracheal suction, differentiate from kinked tube and blocked tube due to mucous plugs, and blood clot. Change the ET tube if required.

2) No-obstruction of ET tube:

Consider mucous or thrombus obstructing distal to tube or tube malposition with distal obstruction, re-assess bilateral breath sounds, and perform deep suction of the tube with normal saline lavage.

If pulmonary edema with stiff lungs, bronchospasm, aspiration, or cardiac decompensation are suspected, perform appropriate measures depending on cause. Give bronchodilators and modify ventilator settings as needed. Consider ECMO support.

5.5.2 Hypercarbia

Hypercarbia is defined as $PaCO_2$ of > 45 mm Hg on supportive ventilation. Evaluate the patient for the following causes of hypercarbia:

A. Inadequate CO_2 elimination from lungs:

1) Ventilator circuit problems:

Malfunction such as disconnection, inadequate TV because of poor circuit capacitance, and air leaks (suggested by ↓ end-tidal CO_2 and ↑ $PaCO_2$).

Address the above and reconnect the ventilator.

2) Inadequate tidal breaths:

These are secondary to mucous plugs, atelectasis, bronchospasm, pleural space occupying fluid / air, and increased physiologic dead space with inadequate compensation.

Treat above and improve alveolar recruitment by increasing TV and PEEP.

3) Increased dead space ventilation (↑ physiologic dead space):

It occurs due to ↓ pulmonary blood flow, pulmonary vessel obstruction as in pulmonary embolism (PE) in older chidren, and decreased alveolar perfusion secondary to ↓ cardiac output.

Address measures to improve pulmoanry blood flow and cardiovascular dynamics.

B. Excess CO_2 production:

It occurs in shivering, rewarming, fever, hypermatabolic state, sepsis, and increased respiratory quotient (RQ). The normal RQ = 0.8

$$\text{RQ} = \frac{CO_2 \ production \quad in \ mL/minute}{O_2 \ consumption \quad in \ mL/minute}$$

Increased RQ (i.e., 0.1) occurs in excessive carbohydrate caloric intake (as in patients on total parenteral nutrition (TPN).

Address above causes of ↑ CO_2 production, limit carbohydrates, and provide lipids as source of energy.

Conrol shivering my muscle paralysis.

5.6 Principles of Use of Conventional Ventilators and Settings

5.6.1 Ventilator Cycle

The ventilators are broadly divided into volume cycled or pressure cycled depending on whether preset volume or pressure is generated by the ventilator to deliver a breath.

(I) Volume Cycled Ventilation

Controls:

a) Respiratory rate (RR). b) Positive end expiratory pressure (PEEP).

c) Inspired oxygen concentration (FiO_2). d) Inspiratory time (I time).

e) Tidal volume (TV).

The pressure limits are set to avoid elevated inspiratory pressures.

It provides constant ventilation under varying lung compliance.

Part of TV is lost to the compliance of the breathing circuit (3-4 cm/H_2O).

Unable to compensate for volume loss due to air leaks.

Advantages / Disadvantages:

Delivers known tidal volume, but risk of baro-trauma exists.

Uses:

Used in the most ventilated patients, patients in OR (including neonates), and in the postoperative period.

Control of pH and $PaCO_2$: Control minute ventilation and set both the respiratory rate and the tidal volume.

Control of $PaCO_2$: Adjust the FiO_2 and PEEP, may also adjust PIP (positive inspiratory pressure) by adjusting the tidal volume. Larger TV yields bigger Pmax, but it is rarely done in practice.

(II) Pressure Cycled Ventilation

Controls:

a) Rate. b) PEEP. c) FiO_2. d) Inspiratory time. e) Peak inspiratory pressure (PIP).

Inspiration is terminated when airway pressure reaches preset peak value.

TV is dependent on airway pressure and compliance of lungs and chest wall.

Time cycled-pressure limited ventilation is identical to pressure cycle ventilation with addition of inspiratory pause (IP). IP maintains airway pressure at preset maximum for a predetermined time. IP causes airway-alveolar pressure equilibration under varying resistances of ventilatory units and delivers constant TV, and is a common mode in infants and children.

Advantages / Disadvantages:

Ideal with constant lung/chest wall compliance, and ET tube size (small) mandate air leaks as in infants and children.

No guarantee of TV, but it is since pressure limited, ↓ risk of barotrauma.

Incomplete exhalation limits TV, and compensatory ↑ RR (breath stacking) may result in abnormal increase in mean airway pressure.

Uses:

Patients where pressure is a concern, as in ARDS and in neonates.

Control pH and $PaCO_2$ by controlling minute ventilation as below:

Set the RR, but the TV is managed indirectly.

TV is directly proportional to alveolar distension pressure (DP).

Alveolar distension pressure (DP) = peak inspiratory pressure – positive end expiratory pressures (PIP - PEEP).

TV varies over a range of DPs, and higher the pressures the less TV for a given DP.

Control of PaO_2: Adjust the FiO_2, PEEP, and control the PIP (rarely done clinically).

5.6.2 Ventilator Modes

Ventilator modes are divided into either control modes or support modes

Control modes are also commonly known as assist control mode.

Unless specified, all the following modes described below are volume-cycled ventilators. Newer ventilators and Servo 300 operate in either pressure or volume cycle.

(I) Control Modes

i) AC (assist control) or VC (volume control):

At preset rate and tidal volume, ventilator delivers a full mechanical breath.

Breath is initiated by the patient or at the set interval. Patient breathing spontaneously above the set rate will generally receive a full set breath.

Used commonly for patients with weak respiratory effort.

Allows synchrony with the patient for maximal support.

Contraindications: commonly none.

Advantages / Disadvantages:

Comfortable mode, but hyperventilation occurs if not closely monitored.

May not wean the patient from the ventilator in this mode.

Ventilators: LP-10 (a portable ventilator), Servo 900, and infant Star

ii) PC (Pressure Control):

The breath is controlled by the Pmax or swing pressure but not by the set tidal volume. It works basically like IMV.

Used for neonates and patients with high airway pressures (such as ARDS) to avoid barotrauma.

Contraindications: None. Not tolerated by an awake patient.

Advantages / Disadvantages:

Since pressure limited, it decreases the risk of barotrauma.

Tidal volume is not guaranteed.

Ventilators: All.

iii) Pressure Regulated Volume Control (PRVC):

Volume control / assist control mode.

Adjusts the gas flow rate to deliver set TV at or below the set maximum pressure. Used in patients with high airway pressures, and can also be used in any patient. May be better mode of ventilation in awake patients than SIMV.

Contraindications: None.

Advantages / Disadvantages:

Delivers guaranteed tidal volume and minimizes barotrauma.

PRVC ventilation mode has no specific disadvatage.

Ventilators: Servo 300

iv) Intermittent mandatory ventilation (IMV):

Operates on pressure or volume modes.

Set breath is delivered at a fixed interval. There is no patient interaction.

Used commonly in neonates on the Sechrist.

May be used as a weaning mode.

Contraindications: None. Not tolerated in older patients.

Advantages / Disadvantages:

Delivers regular guaranteed breath.

Does not allow patient to breath with the ventilator.

Does not work with the patient.

Ventilators: Sechrist and others.

(II) Control and Support Modes

These modes of ventilation have set rates.

Spontaneous patients breaths are not controlled.

Can be used in weaning.

i) SIMV (synchronous IMV):

Delivers breath within an interval based on the set rate.

Ventilator waits part of the interval for spontaneous breath from the patient.

Spontaneous breath (SB) is used as a trigger to deliver a full breath.

Delivers a breath automatically at the end of the period if SB is not sensed.

Any other patient breaths during the cycle are not supplemented.

Used commonly in many settings.

Can be used as a weaning mode.

Contraindications: None.

Advantages / Disadvantages:

Allows working with the patient and is well tolerated.

Any other breaths during the cycle are not supplemented.

Ventilators: All except Sechrist.

ii) SIMV / PS:

Operates on Combination of the two modes (SIMV + IMV).

Patient's extra breaths in the cycle are supplemented with pressure support (PS).

Used in most situations, including weaning.

Contraindications: None.

Advantages / Disadvantages:

Allows synchrony with the patient. Overcomes resistance in the endotracheal tube and allow easier spontaneous breathing.

This mode of ventilation has no particular disadvantage.

PS does not complement if patient is not spontaneously breathing.

Ventilators: All except Sechrist.

(III) Support Modes

i) PS (pressure support):

Supports each spontaneous breath with a supplemental air flow.

Achieves a preset pressure to get the air in lungs.

Used in spontaneously breathing patient to overcome the airway resistance of the ET tube.

This mode can be very helpful for weaning.

Advantages / Disadvantages:

Overcomes resistance of ET tube and conducting airways.

Makes spontaneous breathing easier.

High flow rate on the Servo 900C is uncomfortable for some small patients.

Contraindications:

In patient not spontaneously breathing, i.e., patients on muscle relaxants.

Ventilators: All types except Sechrist.

ii) Volume Support:

To maintain a minimum set goal of minute ventilation.

Variable level of pressure support is delivered on each breath.

Minute ventilation is a goal; so the patient's respiratory rate may fall below the 'set' rate. It can also be used as a weaning mode.

Stronger the patient's own respiratory effort, less support is needed from the ventilator, therefore, the level of pressure delivered will get smaller.

Advantages / Disadvantages:

Decreased number of attempts is needed to wean from a ventilator compared to traditional weaning. One should need experience to understand and wean chronically ventilated patients.

Contraindications:

In patients that are not spontaneously breathing, as it has no back-up rate.

Ventilator: Servo 300 only.

iii) CPAP (continuous positive airway pressure):

It is an equivalent to PEEP + PS, i.e., positive pressure is maintained through out the respiration. Inspiratory pressure is also maintained at the CPAP level.

Supports on inspiration but offers resistance on exhalation.

Improves functional residual capacity, ↑ gas exchange, and ↓ work of breathing.

Used for upper airway / soft tissue obstruction or in patients with a tendency for airway collapse.

Used as a final mode prior to extubation.

Contraindications:

In a patient without spontaneous respiratory effort.

Obstructive pulmonary disease (like asthma, COPD)

Advantages / Disadvantages:

Simple, easy to use, but provides no supportive ventilation.

Ventilators: All. It can also be done without a ventilator.

5.7 High Frequency Jet Ventilation (HFJV)

HFJV may be effective in adult and children with severe adult respiratory distress syndrome (ARDS) failing to be managed by conventional ventilation.

5.7.1 Indications

1. Increased PIP (> 70-80 cm H_2O in adults) or mean airway pressures on standard ventilators.

2. Hypoxemic respiratory failure in preterm and ARDS in infants and children.

3. Bronchopleural fistula 4. Upper airway instrumentation.

♣ Overall there is not enough evidence to conclude that HFJV reduces mortality or longterm morbidity in patients with acute lung injury or acute respiratory distress syndrome as compared to conventional ventilation.

5.7.2 Procedure

Give TV of 3-4 mL/kg or < dead space volume (Vd) at rates of 50-300/minute by high frequency jet (HFJV), positive pressure (HFPPV) or high frequency oscillation (HFOV)

Give HFJV with 14 gauze needle injector and sliding venture tube for a large TV. The 14 gauze needle injector and injector – lumen ET tube provide a smaller TV.

Jet ventilator settings:

Frequency: 100-150%, duty cycle (inspiratory time) 20-30%, drive pressure of 5-50 Psig. Adjust TV by altering % inspiratory time (duty cycle) and drive pressure.

Note:

All high-frequency ventilatory devices do not yield the same clinical results.

In infants HFJV improves hypoxemic respiratory failure unresponsive to HFOV.

Lung volume recruitment strategy during HFOV ↑ gas exchange without compromise in diffusion of oxygen (DO_2). There will be incremental increases in MAP (mean airway pressure) to achieve a PaO_2 of > 60 torr, with an FiO_2 of < 0.6. This results in ↑ gas exchange and improvement in PaO_2/FIO_2 ratio, i.e., reductions in the oxygenation index ($p < \cdot 01$) and FiO_2.

5.7.3 Advantages / Disadvantages

↓ MAP (mean airway pressure) and ↓ pulmonary barotrauma.

↓ intracranial pressure and ↓ interference in cardiac output.

Inadequate humidification with inspissation of secretions in the ET tube.

Decreased expiratory volume and tracheal mucosal injury.

5.8 Weanining from Conventional Mechanical Ventilation (MV)

5.8.1 Prerequisites for Weaning from MV

The patient should exhibit the following characteristics before attempted weaning from MV:

i) Stabilized postoperative cardiovascular function and adequate cardic output and perfusion.

ii) Cessation of bleeding (without need for re exploration) and adequate organ function.

iii) Intact neurological function.

Procedure of weaning:

First stop or wean infusion of paralytic agents and decrease the sedation.

Patients are usually weaned on SIMV/IMV rate with progressive ↓ in IMV rate.

May add pressure support (PS) to patient's respiratory effort, and then wean from IMV rate.

5.8.2 Criteria for Cessation of MV and Extubation

If the following criteria are met, the patient will be successfully weaned from mechanical ventilation.

A. Ventilatory mechanics:

PIP (peak inspiratory pressure) usually < 14-16 cm H_2O

PEEP (positive end-expiratory pressure) < 2-3 cm H_2O (infants) < 5 cm H_2O in children and adolescents

PS < 5 cm H_2O in children, < 8 cm H_2O in adolescents.

IMV rate < 4 in infants and children: children may be weaned to CPAP mode beofre extubation.

Maximum negative inspiratory force > 20-25 cm.

TV (tidal volume) > 6 mL/kg

VC (vital capacity) > 10-15 mL/kg (adults and adolescents).

Mechanics of patient's spontaneous breathing:

The patient has sufficient respiratory effort with ability to clear secretions and has bilateral breath sounds. Patient's respiratory rate < 25 to 35 (depends on age) and has maximum negative inspiratory force > 20-25 cm.

B. Oxygenation:

PaO_2 > 70 on FiO_2 of < 40% or P (A-a) O_2 of < 350 mm Hg on FiO_2 of 100%

Ventilation:

$PaCO_2$ of < 40 mm Hg

5.8.3 Contraindications for Early Extubation

Low cardiac output state.

Pulmonary hypertension and reactive pulmonary vascular disease.

Major central nervous system derangement.

Excessive bleeding requiring re-exploration.

Pulmonary edema (may use diuretics and higher PEEP few hours before extubation trial) and paralyzed diaphragm (detected by ultrasound).

5.8.4 Preparation for Extubation

Decrease FiO_2 gradually to 0.4

Decrease PIP to 20 cm H_2O

Decrease PEEP to 3 cm H_2O

Ventilator rate set to 5-8 breaths/minute.

Some patients may need CPAP (continuous positive airway pressure) trial before extubation.

A. Observe prior to extubation:

1) Signs of respiratory distress.

2) Patient respiratory excursions.

3) Arterial blood gases:

Following blood gas results are ideal for successful extubation:

SaO_2 98-100%, PCO_2 35-40 mm Hg, normal pH

Ideal blood gas in special situations for a successful extubation is:

SaO_2 75-80%, $PaCO_2$ 35-40 mm Hg, and normal pH.

These situations are in aorta-pulmonary shunts, hemi-Fontan, stage I Norwood correction, and single ventricle physiology with outflow tract (ascending aorta and pulmonary artery) in series.

B) Steps Prior to Extubation:

1) Suction the airway and hand ventilate with oxygen (appropriate FiO_2 for the patient), and extubate during inspiratory effort.

2) Give humidified oxygen via tent or mask. FiO_2 is usually 0.1 higher than FiO_2 on ventilator. Watch for strider / bronchospasm, and treat with an appropriate inhalation therapy.

3) Always have ET insertion set available at the bedside.

C. Airway edema and extubation:

May use dexamethasone prior to extubation if airway edema is suspected, or for an extubation after a prolonged intubation and ventilatory support (see Chapter 16-H).

5.8.5 Problems in Weaning from Mechanical Ventilation

a) Ventilator impedance:

Spontaneous breathing is difficult through ventilator circuit resistance.

PEEP (3-5 cm H_2O) and PS (5 cm H_2O) would overcome the resistance of the circuit of ventilators.

b) ET tube:

Resistance of a tube (size may be small to allow spontaneous breaths) or an obstructed tube.

c) Fluid and Electrolytes:

Fluid overload (pulmonary congestion/edema) and electrolyte imbalance (i.e., hypophosphatemia).

d) Acid-Base status:

Acid base status (need pH 7.3 to 7.34 for good respiratory drive) is important for successful extubation.

e) Airway problems:

Bronchospasm, excessive secretions, or over sedation impairs weaning.

f) Nutrition:

High carbohydrate (> 35%) caloric intake ↑ ventilator demands, and malnutrition with poor respiratory muscle strength impairs weaning.

g) Over exertion / fatigue:

Respiratory muscle fatigue (RR > 35 to 50/min depending on age) on a spontaneous breathing. Use CPAP to support patient breaths without mechanical ventilation (MV).

h) Cardiovacular function:

Failure to wean may be due to increased preload and after load, and reevaluate cardiac function.

5.8.6 Parameters Predictive of Successful Extubation

The following parameters are usually suggestive that the patient can be extubted without any significant postoperative respiratory morbidity.

Normal $PaCO_2$ and PEEP < 2-3 cm H_2O (infants); < 5 cm H_2O (children).

PIP < 14-16 cm H_2O.

IMV rate < 2-4 (infants). Children may be weaned to CPAP.

FiO_2 < 40% to maintain PaO_2 70 mm Hg, (in biventricular correction).

Maximum negative inspiratory pressure > 20-25 cm H_2O.

♣ Accurate prediction of extubation is difficult. Patient's ability to breathe spontaneously is multifactorial. Clinical (i.e., age, duration of mechanical ventilation, ventilation mode) and physiological characteristics (RR, TV, lung compliance, maximal inspiratory force, and airway resistance) influence extubation success in infants and children.

5.8.7 Other Weaning Indices for Successful Extubation

1. RR (Respiratory rate): breaths/min < 45

2. VT (Tidal volume): mL/kg > 5.5

3. Rapid shallow breathing index (RSBI): $\frac{\text{Breaths per minute}}{\text{mL per kg}} < 8$

4. Compliance, resistance, oxygenation and pressure (CROP) index: mL/kg/breaths/min.

Patients who are successfully extubated would show the following:

Lung compliance (Cdyn: 0.59 ± 0.91 versus 0.39 ± 0.14 mL/kg/cm H_2O).

Higher PaO_2/FiO_2 ratio (382.4 ± 181.2 versus 279.8 ± 93.9).

Lower $PaCO_2$ (41.3 ± 6.7 versus 47.3 ± 8.5 mm Hg).

Spontaneous breathing parameters in successfully extubated would commonly show the following parameters:

Lower respiratory rates (RR) 36.6 ± 17.9 versus 52.8 ± 23 breaths/min.

Larger tidalvolumes (VT) 7.3 ± 2.6 versus 44.9 ± 1.8 mL/kg.

Negative inspiratory force [NIF]: 41.8 ± 15.4 versus35.1 ± 12.5 cm H_2O.

5.9 Chest Physiotherapy (CP), Re-intubation, and Oxygen Delivery

CP is often done when taking the child off the ventilator (extubation).

Active chest physiotherapy (tapping or vibrating on the chest) may not be helpful for all infants. With use of CP only a few children need to go back on the ventilator, and there is no evidence of harm receiving a short course of chest physiotherapy following extubation.

Humidification systems and the drug surfactant reduced the need for CP.

Expiratory flow increase (EFI) is a method of common chest physiotherapy technique used in Europe. EFI is performed three times daily until, at least, 24 hours after extubation. There is low incidence of post extubation atelectasis in infants with use of EFI technique. EFI does not appear to increase the incidence of brain lesions in many studies.

5.9.1 Inhalation Therapy

It is often used in addition to CP after extubation to improve patient breathing and clearance of airways during the postoperative period. Both nebulizers and metered-dose inhalers (MDIs) can be adapted for use in ventilator circuits. MDIs deliver beta-adrenergic and anticholinergic bronchodilators. MDIs offer several advantages over nebulizers for routine bronchodilator therapy (i.e, combination of an MDI with an elbow adapter).

Nebulizers are used to deliver bronchodilators, antibiotics, and surfactants.

Beta-adrenergic and anticholinergic bronchodilators are effective in ventilated patients. Inhaled corticosteroids advocated in infants with bronchopulmonary dysplasia. The sympathomimetic drugs used in nebulizers are isoproterenol hydrochloride, isoetharine mesylate, metaproterenol sulfate, fenoterol, and albuterol.

♥ A combination of fenoterol and ipratropium bromide is more effective than a single drug for nebulization.

5.9.2 Emergency Re-intubation

Use straight (Miller) laryngoscope blade for children < 10 years.

Use curved (Mac Intosh) laryngoscope blade for older children.

The measurement of the depth of insertion of endotracheal tube (ET) at teeth or lips = 3 × ET tube size.

Procedure: Have the patient lying supine on firm surface, head slightly extended, and in midline. Open mouth with right thumb and index finger. Insert laryngoscope blade holding with left hand and slide it into the right side of the mouth pushing the tongue to the left. Then advance the blade to epiglottis--

Straight blade tip lifts the epiglottis. The scope is lifted straight up to visualize cords.

Curved blade tip is positioned between the vallecula and epiglottis, and the scope is lifted up to visualize cords. Advance the ET tube from the right corner of the mouth a few centimeters below the level of cords. Check for correct position by auscultation and chest x-ray.

Procedures prior to re-intubation:

1). Give 100% oxygen via bag and mask.

2). Administer medications:

Vagolytic (optional): To prevent bradycardia and reduce secretions.

Atropine 0.01-0.02 mg/kg IV, minimum 0.1 mg, maximum 1 mg.

Lidocaine (optional): To blunt cardiovascular response.

Dose: 1-2 mg/kg IV.

3). Cricoid pressure (Sellick maneuver): To prevent regurgitation.

It is done by an assistant.

4). Sedation: Midazolam 0.05 mg-0.1 mg/kg IV / or fentanyl 1-5 mcg/kg IV.

5). Paralytic: Vecuronium 0.1 mg/kg IV / or Pancuronium 0.04-0.1 mg/kg IV.

5.9.3 Selection of Endotracheal Tubes

ET tube size determination: Use the formula below to determine the size.

$$\text{Internal diameter of ET tube} = \frac{(Age*+16)}{4}$$

* Age in years, note this formula doesn't apply for infants < 1 year.

The internal diameter of ET tube that is appropriate for an infant is also determined as shown in table 5.3. Use uncuffed tubes for children and infants less than 8 years.

Table 5.3 *Endo tracheal tube sizes by age in children and adults.(inner diameter in mm).*

Premature	**2.5–3**	**New born**	**3.0-3.5**	**Infant < 1 year**	**3.5-4**
1 year	4.0-4.5	3 years	4.5-5	6 years	5.0-5.5
10 years	6.0-6. 5	Adolescent	7.0-7.5	Adult	7.5-8.0

5.9.4 Oxygen Delivery Contraptions

(I) Nasal Cannulae

At a flow rate of 1/8 to 4 liters/minute (low flow), provides FiO_2 of 22 to 40%. The FiO_2 delivery is influenced by age, inspiratory flow, minute ventilation, and entrained ambient gas. High flows cause dry mouth, headaches, and gastric distension.

(II) Oxygen Masks

Masks should be clear so regurgitation can be detected.

Increase the dead space.

Cause O_2 retention if O_2 flow isn't adequate.

Simple Mask:

Delivers FiO_2 of 35%-55% at a flow rates of 6-10 liters/minute.

FiO_2 delivery is influenced by O_2 flow relative to TV and inspiratory flow.

Increased inspiratory ambient air flow through vents decreases FiO_2.

Partial-rebreathing Mask:

Delivers FiO_2 of 60-95% and is influenced by tidal volume and inspiratory flow.

O_2 Flow rate should be 6 liters/minute to prevent CO_2 retention.

Expired gas (CO_2) mixes with O_2 in reservoir bag (valve less).

Expired gas also escapes through mask ports.

Insufficient O_2 flow / a leak will collapse the reservoir.

Non-rebreathing Mask:

Delivers FiO_2 100%, requires tight seal and a high gas flow.

Reservoir bag has one-way valve preventing expired gas to enter bag.

Valve opens on negative inspiratory pressure, allowing airflow from bag.

Expired gas escapes through exhalation ports.

Safety valve prevents ambient air to enter bag if O_2 source is disconnected.

Venturi Mask:

Delivers specific O_2 concentrations between 24%-50%.

O_2 flows at a set flow rate through jets (of various sizes) and ambient air flows through entrained ports. Development of back pressure in a jet results in less ambient air entrained and unpredictable increase in FiO_2.

Tracheotomy Mask:

Delivers controlled O_2 flow and humidity.

FiO_2 is unpredictable and should be analyzed on an individual patient.

May be used with venturi circuit with a predictable FiO_2.

(III) Oxygen Hoods

Achieve high O_2 concentration.

Used in patients who do not tolerate masks and canulae.

Require high O_2 flow (10 liters/minute) to prevent CO_2 accumulation.

Develops O_2 gradients in a hood.

(IV) Oxygen Tent

Delivers FiO_2 up to 50% and can deliver humidified air.

Require high O_2 flow to prevent CO_2 accumulation.

Develops O_2 gradients in tent, O_2 leaks, claustrophobic, and hazardous with surrounding sparks.