

Care Of The Patient On Ventilator

The concept of the Intensive Care Unit (ICU) has been accepted as an integral part of any bigger hospital, even in the District and Taluk hospitals. The ICU, when it becomes busy, becomes the nerve centre and probably the biggest money spinning area of the hospital. In any busy ICU, at a given time, 25 to 40% of the patients will be found attached to the ventilator. Proper understanding of the ventilator mechanics, constant monitoring of these patients and frequent adjustments in the ventilator settings are the essence of the care of the patients on ventilator therapy.

The patients on the ventilator need all the supportive measures which any other patient in the ICU is getting. This includes frequent change in posture to avoid pressure sores, proper padding of pressure points to prevent nerve palsies, preventing injuries to the orofacial tissues and the eyes due to the endotracheal tubes and connectors, accurate management of fluid balance, temperature regulation, maintenance of adequate nutrition according to the disease condition, adequate sedation for the patients on ventilators and prevention of nosocomial infection, especially, the ventilator associated pneumonia. Each one of these items is a separate topic which can be discussed at length and detailed discussion about these is not undertaken in this lecture. I am going to concentrate only on the possible complications due to the airways and due to the mechanical ventilation, how to take care of the patients against these complications and how to get them out of the ventilators early. The ***ventilatory care of the patients*** will be dealt under the following headings:

Complications due to the airways

Management of the secretions in the airway

Monitoring for the lung injury due to the mechanical ventilation

Periodical assessment of the patient for weaning from the ventilator

Complications due to the airways

All patients on ventilator will have either the endotracheal tube or the tracheostomy tube in place through which they are ventilated.

Endotracheal tubes (ETT)

The complications due to the ETT depend on the route of insertion (oral or nasal), size and possible change of position of the tip of the ETT. Complications due to the nasal ETT are epistaxis and paranasal sinusitis. Epistaxis is usually due to the local tissue trauma and transient. Nasal tubes block the ostia of the paranasal sinuses and maxillary sinus is almost always involved. Air filled sinus in the X-ray helps in the diagnosis, though needle aspiration is confirmatory. Occasionally, fever in ICU patient, may be due to this sinusitis. Nasal ETT has the advantage of better tolerance and the lesser need for sedation and less frequent chance of dislodgement. Complications due to oral ETT include necrosis of the mucosa at the back of the mouth, dental trauma, fissure wounds at the angle of the mouth where the tube is fixed and obstruction to the tube due to bite by the patient.

Laryngeal damage can occur with both nasal and oral ETTs. The damage may be in the form of ulceration, granulomas, vocal cord palsies and laryngeal oedema. The injuries become evident

after 72 hrs. In the majority, they do not lead to airway obstruction and usually resolve within weeks of extubation.

The apparatus dead space due to the ETT is about 40 ml (for adult sized tubes) which is nearly half of the anatomical dead space of the adult. The work of breathing is directly proportional to the length and inversely proportional to the diameter of the ETT. Increased work of breathing leads to turbulent air flow and increased resistance. Therefore, in adults, the diameter of the endotracheal tubes should be at least 7 mm, preferably more than 8 mm.

The position of the ETT should be checked on a regular basis till extubation or tracheostomy. Occasionally, verification of the proper position of the ETT may need X-ray head and neck, using portable X-ray machine. Land marks to be remembered are that vocal cord lies at the level of C5-C6, and bifurcation of the trachea lies at T4-T5, when the X-ray is taken in the neutral position i.e., the inferior border of the mandible projects over the cervical spine at C5-C6 level. It should be remembered that the tip of the ETT move up to 2cm downwards during flexion of the neck and upwards during extension.

Migration of the ETT can occur during suctioning and turning the patient, usually into the right bronchus. This may lead to obstruction of the left bronchus and atelectasis of the left lung. In such situation, the whole tidal volume will be diverted to the normal lung and may lead to pneumothorax. To prevent such migration of the ETT, tubes should be fixed in such a way that the 21cm mark is against the edge of the teeth and the position verified periodically.

Tracheostomy:

Tracheostomy is preferred to ETT in prolonged ventilation. The advantages are patient comfort, easy and effective clearance of secretion, decreased resistance to breathing and less laryngeal injury. Patient can take oral food and if special tubes are used, can even vocalise.

The decision to go in for tracheostomy is usually taken at the end of 5 to 7 days. If the patient is likely to be extubated in the following week, tracheostomy is postponed. Otherwise, tracheostomy is done.

Tracheostomy can be done by open surgical method or through percutaneous methods. Percutaneous methods involve puncturing the anterior wall of the trachea, passing a guide wire and threading the tracheostomy tube over it. Original percutaneous method is the percutaneous dilatational tracheostomy. Occasionally, in emergency, cricothyroidotomy is done to save the patient. This should always be followed by regular tracheostomy later on.

Mortality due to tracheostomy is less than 1%. But in 3to10% of cases, major adverse events can occur. Bleeding and infection are the two major complications and are less common following percutaneous techniques than in open surgery. Accidental decannulation, if it occurs before the tract is formed, is an emergency situation. Attempts to pass the tracheostomy tube blindly may lead to false passage. It is preferable to intubate the patient immediately and then try to negotiate the tracheostomy tube. Late complication following tracheostomy is tracheal stenosis which can appear 6 months after the closure of tracheotomy stoma. Majority of the cases are asymptomatic and the incidence is 0 to 15%.

Management of the cuff of ETT / Tracheostomy tube:

The cuff is inflated till the air leak is not heard. The cuff pressure should not exceed 25mm of Hg since it may compress the underlying capillaries and cause mucosal ischemia and tracheal necrosis. Elongated cuff disperses the pressure over larger area and so high volume low pressure cuff should always be used. Volume of the leak can be estimated as the difference between inflation volume and the expired volume as indicated in the ventilator. Cuff leak need not always be due to rupture of the cuff. It can be due to non uniform contact between the cuff and the trachea. The leak in cuff of the ETT should not be attempted to be sealed by blindly injecting more air into the cuff. It may damage the vocal cord if ETT is malpositioned. The tube should be changed. In tracheostomy tube the leak can be obliterated by injecting more air. Then, the cuff pressure is measured and if it exceeds 35 cm of H₂O or if the leak persists, a larger tube should be used. Use of foam rubber cuff which is inflated at normal atmospheric pressure avoids damage to the tracheal mucosa. The tube with preinflated foam cuff is introduced after deflating the cuff and after positioning the tube, the cuff is allowed to inflate to the atmospheric pressure and seal the trachea (Birona Fome-Cuf tracheostomy tube).

It should be remembered that the cuff does not give guarantee against aspiration. Aspiration of mouth and gastric content into the lungs is reported in more than 50% of ventilator dependent patients and in $\frac{3}{4}$ of the patients, aspiration is clinically silent. One litre of saliva is produced daily (0.6ml per min) and each micro litre of saliva contains one billion micro organisms. This points out the importance of oral decontamination which should be included in the protocol of VAP bundle.

Management of secretions in the airway

The airway secretion protects the underlying mucosa. The amount of the secretion is increased enormously during ventilatory therapy, due to the presence of so many foreign bodies like ETT, oral airway etc. Normally respiratory secretions are in two layers, the inner hydrophilic layer to keep the respiratory mucosa moist and the outer or luminal layer which is hydrophobic which traps the dust and debris in the respiratory tract. The hydrophobic layer is constituted by mucoproteinous strands bridged by disulfide links. The luminal layer, when it traps the dust and debris, it leads to thick viscous tenacious secretions which can block small and sometimes, even the larger airways. The treatment options available to remove this excessive secretion are either saline instillation or mucolytic therapy (apart from frequent suction).

Saline instillation which was once very common has two disadvantages. Since the luminal layer is hydrophobic, instillation of saline may not liquefy the secretion. Secondly, instillation of saline through the ETT / tracheostomy tube may dislodge the organisms colonised in these tubes into the lungs. It is estimated that instillation of 5 ml of saline may dislodge 300,000 colonies of viable bacteria into the lungs.

Mucolytic therapy is now being frequently used to make the tenacious secretions less viscous so that it can be easily removed. N acetyl cysteine (NAC) having a sulfidryl containing tripeptide can disrupt the sulfidryl links of the mucoproteinous luminal layer and thus liquefies the secretion. It is available as 10% and 20% solutions and can be instilled into the trachea or given as aerosol therapy. Aerosol is a little irritating and can cause bronchospasm especially in asthmatics. Direct instillation is preferred when there is obstruction. If instillation through the ETT does not

relieve the obstruction, bronchoscopy is done and the drug instilled directly onto the plug. Following relief from obstruction the instillation can be continued two or three times a day for one to two days and not more than that, since NAC is very hypertonic and can lead to bronchorrea.

Monitoring for the lung injury due to the mechanical ventilation

Alveolar rupture:

Ventilator induced lung injury ultimately leads to clinically apparent alveolar rupture which occurs in 25% of the patients receiving mechanical ventilation. Escape of gas from the alveoli produces dissection along the tissue planes and leads to interstitial emphysema, pneumomediastinum, subcutaneous emphysema, pneumoperitoneum and finally to pneumothorax. Each of these can occur alone or in combination.

Pneumothorax occurs in 5 to 15% of patients who are ventilator-dependent. The risk factors leading to pneumothorax are high inflation volume, high inflation pressure, PEEP (peak end expiratory pressure) and diffuse lung injury inherent to the underlying lung disease. In many situations of accidental pneumothorax, the absence of breath sounds may not be clinically detectable due to the presence of transmitted sounds and appearance of subcutaneous emphysema over the neck and the upper chest may be the first indication of pneumothorax. Radiological diagnosis may be difficult, especially in supine patients and the CT chest has revealed pneumothorax in situations where the X-ray chest was normal. Basilar and subpneumonic collections of air are characteristic of pneumothorax in the supine position. Redundant skin folds over the back of the chest in the supine patient can mimic pneumothorax in X-ray.

ICD (intercostal drainage) for pneumothorax is done through 4th or 5th intercostal space in the mid axillary line and the tube is advanced in the anterior and superior direction. Three chambered suction was originally employed for the ICD, the first chamber for collection of the fluid, the second chamber for effective water seal (usually 2 cm of H₂O) and the third chamber for applying the negative pressure from the wall mounted suction (to a level of 20 cm of H₂O). Application of negative pressure to the pleural cavity is potentially harmful since it may increase the air leak through the bronchopleural fistula and may keep the fistula patent.

Intrinsic PEEP:

Normally, at the end of expiration, since the alveoli have emptied their content, there is no air flow to the proximal airways and the pressures in the alveoli and the proximal airways are the same. When there is incomplete emptying of the alveoli, air is trapped in the alveoli which are at a higher pressure than the proximal airways, at the end of expiration. This is called intrinsic PEEP, also called auto PEEP, occult PEEP and in case of obstructive airway disease, dynamic hyper inflation.

Ventilator associated factors like high inflation volumes, rapid breathing, and relative decrease in the expiratory time may lead to the development of auto PEEP. Similarly, disease related factors like asthma and COPD may have increased incidence of auto PEEP. Auto PEEP is universal in all patients with asthma and COPD undergoing volume cycled ventilation and also in patients with ARDS who are ventilator dependent (though to a lesser level of <3 cm of H₂O).

Intrinsic PEEP suppresses the myocardial function same as the extrinsic PEEP and can be life threatening. This is sometimes attributed as the occult cause for cardiac arrest and failed CPR in ICU patients. Intrinsic PEEP may lead to barotrauma and pneumothorax. The work of breathing is increased by the intrinsic PEEP due to many factors – pressure needed to trigger the ventilator is increased; hyperinflation places the lungs on the flatter portion of the pressure-volume curve thus increasing the pressure needed to inhale the tidal volume; hyperinflation flattens the diaphragm and increases the muscle tension required to cause effective contraction. Intrinsic PEEP increases both the peak and plateau pressure in the proximal airways. This increase in the plateau pressure may be misinterpreted as a decrease compliance of the lungs and chest wall. PEEP pressure should be subtracted from the plateau pressure in the calculation of the compliance. Similarly intrinsic PEEP may spuriously increase the cardiac filling pressures (central venous pressure, CVP and the pulmonary artery occlusion pressure, PAOP) due to the transmission of PEEP into the lumen of the intrathoracic vessels and hence PEEP should be subtracted from the CVP or PAOP in the calculation of filling pressures.

Clinically it is easy to detect the presence of intrinsic PEEP, but difficult to measure it. Various methods are used to quantify the auto PEEP. Listening to the flow of air at the end of expiration may give a clue. Expiratory flow tracings are also useful in the assessment. The most accurate method to assess the auto PEEP is by measuring intra oesophageal (pleural) end expiratory pressure. But the most popular method of assessing the auto PEEP is by end expiratory occlusion method. The expiratory tubing is occluded at end expiration (to be timed accurately and to be done just before the starting of the next inspiration). In normal persons, since there is no air flow at the end of expiration, the airway pressure remains the same. But in the presence of intrinsic PEEP, due to the continuing flow of gas even at the end of expiration, the proximal airway pressure increases during occlusion. Another method is to add an extrinsic PEEP which will neutralise the intrinsic PEEP to prevent flow of air during end expiration. Failure of the extrinsic PEEP to increase the peak inspiratory pressure is evidence of intrinsic PEEP.

Management of intrinsic PEEP is by avoiding excessive inflation pressure, facilitating alveolar emptying by optimising the expiratory duration and in rare cases by applying extrinsic pressure just enough to neutralise the intrinsic pressure (extrinsic PEEP should decrease the plateau pressure).

Periodical assessment of the patient for weaning from ventilator

In 20 to 25% patients who are on ventilator, it may be difficult to wean them from the ventilator.

Readiness Criteria:

The following indicators are to be evaluated before planning weaning from ventilator: a) oxygenation should be adequate with minimal oxygen support b) patient should be haemodynamically stable with minimum vasopressor support c) patient should be awake and aware of the surroundings, when not sedated and d) the co-morbid conditions, especially sepsis and electrolyte derangement, should be under control. Two bedside measurements can be undertaken to assess the patient for weaning from ventilator.

Rapid-shallow breathing index (RSBI) – the ratio of respiratory rate to tidal volume (RR/Tv) is the RSBI. This is normally 40 to 50 per litre. Patients with the ratio >100/litre do not tolerate weaning. Serial measurements are more useful than single measurement.

Maximum inspiratory pressure (P_Imax) – This needs the full cooperation of the patient. After exhaling to near residual volume, the patient should inhale as forcibly as possible against a closed valve. Normal persons can generate a negative pressure of 90 to 120 cm of H₂O, more in males than in females. The threshold for deciding weaning is 20 cm of H₂O. This has more of a negative predictive value than positive.

Spontaneous breathing trial (SBT):

This can be done either when the patient is attached to the ventilator or when he is on 'T' piece oxygen (called 'T' piece trial).

The advantage of doing the test when the patient is connected to the ventilator is that we can monitor the respiratory rate and the tidal volume and notice the rapid shallow breathing. The disadvantage is the increased work of breathing due to the ventilatory tubings and the necessity of generating extra pressure to open the actuator valve. To obviate this disadvantage, the patient should always be on the pressure support ventilation when SBT is undertaken when the patient is connected to the ventilator.

'T' piece trial is done with the patient receiving gas at a high flow, more than the patient's inspiratory flow rate. This high flow rate creates a 'suction' effect and helps in the removal of CO₂ and also prevents dilution by the atmospheric air. We cannot monitor the respiratory rate and tidal volume on the screen. 'T' piece trial is preferred in patients breathing rapidly or who have high minute ventilation.

SBT is done for 30 to 120 minutes initially. It is better to administer 10% extra oxygen during SBT. Patient should be able to maintain SPO₂ (oxygen saturation) above 90% and the ET_{CO}₂ (end tidal carbon dioxide) should be normal or constant throughout the trial. Patients who were on ventilator for short period will require short period of SBT (30 to 120 minutes) and those who were on ventilator for long period may require long periods of SBT (even up to 8 hrs).

When the patient is breathing rapidly during SBT, it should be differentiated whether it is due to anxiety or ventilatory failure. If the minute ventilation is high and the ET_{CO}₂ is low, it is likely to be due to anxiety and sedatives can be given judiciously in such situations.

Abdominal paradox, i.e., the abdomen moving inwards during inspiration, during SBT indicates that the patient should be reconnected to the ventilator.

SBT should be done daily until patient is successfully weaned from the ventilator. Failure of spontaneous breathing may be due to low cardiac output, overfeeding and respiratory muscle weakness.

Transition from positive pressure ventilation to spontaneous breathing can result in decrease in the cardiac output (CO) due to an increase in the left ventricular after load. This may lead to pulmonary congestion, reduced lung compliance and hence difficulty in spontaneous

breathing. The diaphragm, like the heart, heavily depends on the cardiac output for its blood supply and reduced CO can decrease the strength of the diaphragmatic contraction. Increased oxygen extraction ($SaO_2 - SvO_2$) and increase in the arterial-end tidal PCO_2 gradient, when done before the SBT and when the patient experiences breathing difficulty during SBT indicates reduced CO. In patients with coronary artery disease, failure to tolerate spontaneous breathing may be due to the onset of acute myocardial ischemia which is often silent. 12 lead ECG should always be done when such situation is suspected. Continuous Positive Airway Pressure (CPAP) offsets the increased after load caused by the negative intrathoracic pressure during spontaneous ventilation and therefore non invasive ventilation (NIV) has been effectively used during cardiogenic pulmonary oedema and this can be employed during weaning and after extubation.

When the daily intake of calories exceed the daily requirement (the resting energy expenditure) the excess CO_2 produced should be eliminated only through the lungs and this can add to the difficulty in sustaining spontaneous breathing in ventilator dependent patients. The caloric intake and need should be matched daily in consultation with the dietician.

It is a wrong conception that disuse of diaphragm during prolonged mechanical ventilation leads to atrophy of the diaphragmatic muscle. Diaphragm being an involuntary muscle is always stimulated by the respiratory centre even when the patient is on ventilator, more so during respiratory insufficiency. Only critical illness polyneuropathy and myopathy can lead to diaphragmatic weakness.

Tracheostomy closure / Extubation:

Extubation or closure of tracheostomy can be planned if the patient is able to protect his airways and there is no evidence of laryngeal oedema.

Patient should have a good gag and cough reflex (tested by placing a piece of paper 2cm from the tracheostomy tube and asking him to cough). The respiratory secretions should be brought down to minimum so that the patient will be able to cough out in the post extubation period.

Laryngeal oedema is seen in 40% of prolonged intubation and in 5% of the patients it can lead to respiratory obstruction in the post extubation period. Cuff-leak test can be done to identify the laryngeal oedema before extubation. If there is a decrease in the expired volume after deflation of the cuff, laryngeal oedema is unlikely. In patients in whom larger and snugly fitting ETTs had been used or when the cuff is adherent to the respiratory mucosa, this test may not be useful. If the patient is with tracheostomy and on fenestrated tracheostomy tube, cuff leak test can be done in that patient also (laryngeal obstruction is possible even after tracheostomy, due to the previous intubation or ischemic injury). Steroid has no proven value to prevent laryngeal oedema, especially after prolonged intubation.

Even though the cross sectional area of glottis in adult person is about $66mm^3$ and the cross sectional area of an 8 sized ETT is only $50mm^3$, patients frequently develop increased work of breathing after extubation. This is unexplainable.

If the patient develops stridor in the post extubation period, in case of children, epinephrine can be used as an aerosol. It is not effective in adults. Helium-oxygen (heliox) can be used in the post extubation period and in mild laryngeal oedema.

To summarise, the ventilatory care of the patient includes, apart from the general care which has been mentioned in the introduction, looking for, preventing and managing the complications due to prolonged intubation / tracheostomy, increased secretions due to the introduction of foreign bodies into the airway, complications due to the positive pressure ventilation (normal respiration is negative pressure ventilation), and prevention and management of auto PEEP. The different modes of ventilation which may be required for managing different disease situations and their advantages and disadvantages were not discussed here. The ultimate aim of the intensivist is to wean the patient from the ventilator as early as possible. As soon as the need for the active respiratory support has subsided, daily and regular tests are to be done to assess the fitness of the patient to be weaned from the ventilator.