Complications of Mechanical Ventilation*

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With increasing utilization of mechanical ventilation during the past decade or so, complications related to its use have also increased. Ventilators are primarily indicated when acceptable safe levels of oxygenation and ventilation cannot be maintained by other means.

The goal of assisted mechanical ventilation should be to improve arterial oxygen, CO$_2$ tensions, and pH to acceptable safe levels compatible with life or normal functioning.

The following are the complications which may occur during assisted mechanical ventilation:

1) Reduced venous return and cardiac output.
2) Complications related to endotracheal tubes and tracheostomies.
3) Atelectasis.
4) Infection.
5) Oxygen toxicity.
6) Acid-base, fluid, and electrolyte disturbances.
7) Gastrointestinal problems.
8) Technical and mechanical problems.
9) Emotional stress.
10) Miscellaneous: cardiac arrhythmias, pulmonary emboli, pneumothorax, and so forth.

Reduced venous return and cardiac output are commonly observed in patients with significant volume depletion. It is important that blood pressure and urine output should be checked at regular intervals. A central venous pressure catheter may need to be inserted for proper fluid replacements. The two other common causes of decreased venous return are prolonged severe hypoxemia and impaired sympathetic nervous system activity.

Normal people can compensate for the decreased venous return by a reflex increase in venous tone and pressure provided: 1) mean airway pressure does not go above approximately 15 mm Hg, 2) effective blood volume is adequate, 3) sympathetic nervous system function is unimpaired. If any one or a combination of any of the above factors is not met, mechanical ventilation results in decreased cardiac output and lowering of arterial blood pressure.

Proper adjustments in ventilators to reduce the mean intrathoracic pressure may need to be performed to improve decreased venous return. In this respect, volume cycled respirators are probably advantageous over pressure cycled respirators.

Negative pressure during exhalation may occasionally be used to improve venous return to the heart. However, this is somewhat contraindicated in patients with chronic obstructive pulmonary disease. It may cause closure of airways leading to clinical or subclinical atelectasis.

The following complications are related to endotracheal intubation and tracheostomies:

1) Tube in right main bronchus which causes massive atelectasis of the left lung. If not recognized this usually causes rapid deterioration of pulmonary function.
2) Dislodgement, kinking of the tube, obstruction by secretions, laryngeal edema and damage, herniation of cuff over end of the tube.
3) Complications related to tracheostomy: bleeding, infection, necrosis, tracheal stenosis, erosion of vessels, obstruction, and tracheoesophageal fistula.

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The incidence of atelectasis is increased especially in patients requiring prolonged assisted mechanical ventilation. The administration of high concentrations of \( O_2 \) in inspired air causes further increase in the incidence of atelectasis.

Adequate humidification of the inspired gas, chest physiotherapy, proper suction, and hydration are essential in prevention and treatment of atelectasis. Bronchoscopy should be performed if atelectasis does not improve with conservative measures.

Atelectasis may be first manifested by decreasing compliance and increased alveolar-arterial \( O_2 \) pressure gradient.

To prevent atelectasis, a proper breathing pattern to obtain optimum ventilation should be maintained. Intermittent sighing is also an important therapeutic measure to prevent atelectasis. Inflation hold and continuous position pressure ventilation are additional steps that may be used in prevention and treatment of this problem.

As to \( O_2 \) toxicity, two factors are primarily responsible for \( O_2 \) toxicity in the lungs: 1) high \( O_2 \) tension in inspired air, 2) prolonged exposure to high inspired \( O_2 \) tension. Patients may be at risk of developing \( O_2 \) toxicity if they are given inspired air containing 50% or greater \( O_2 \) concentration for prolonged time. There seems to be an individual susceptibility. As a general rule, just enough \( O_2 \) concentration in the inspired air should be given to maintain a safe and adequate level of oxygen tension in the arterial blood for a given patient.

It should be noted that most pressure cycled ventilators administer higher \( O_2 \) concentrations than one may think. On the air dilute setting, these ventilators usually deliver somewhere between 60 to 95% \( O_2 \) in inspired air.

This is prevented by running the ventilator with compressed air and bleeding 1 to 3 L of \( O_2 \) into the system. By measuring inspired \( O_2 \) concentration, proper adjustments should be made to administer the desired \( O_2 \) concentrations in the inspired air.

Infection, especially with gram negative organisms, may be a dangerous problem. Nebulizers, humidifiers, and tubes with warm, humid inspired gases may be a source of a good culture media for many gram negative organisms.

The respiratory tubing should be changed at least once daily and sterilized before it is used again. No respirator should be used for another patient unless proper sterilization techniques have been utilized.

Acid-base, fluid, and electrolyte disturbances: one of the common iatrogenic complications of assisted mechanical ventilation is post-hypercapnic metabolic alkalosis. This is observed in patients with chronic respiratory acidosis. As a result of rapid lowering of \( P_{CO_2} \), \( pH \) may become alkalotic, since \( HCO_3^- \) cannot be lowered significantly under these circumstances.

Severe alkalosis can cause confusion, convulsions, coma, and even death. To prevent this, arterial \( pH \) should be monitored, and increased \( P_{CO_2} \), should be reduced gradually in chronic hypercapnia.

Marked hyperventilation with severe respiratory alkalosis alone can cause tetani with symptoms and signs related to it. And again, arterial gas tensions and \( pH \) need to be monitored to prevent this problem.

Some patients on prolonged assisted mechanical ventilation may develop fluid retention and even pulmonary congestion and edema, primarily interstitial type.

Another complication of mechanical ventilation is the development of inappropriate ADH-like electrolyte abnormality in some patients. This appears to be related to stimulation of ADH through thoracic volume chemoreceptors by positive pressure breathing. Therefore, patients should be weighed and electrolytes should be checked repeatedly.

Gastrointestinal complications consist of acute gastrointestinal bleeding, acute gastric dilation, ileus, and chronic aspiration during assisted ventilation. The physician should always consider these problems, and if they occur, proper steps must be taken.

Technical and mechanical problems arise quite often. A physician or inhalation therapist should be prepared to cope with a respirator in case it becomes disconnected from the patient or malfunctions or in case the tube becomes obstructed. These complications can be disastrous for the patient within a few minutes. The ventilatory and \( O_2 \) requirements of these patients change quite often, so necessary adjustments must be made accordingly.

Emotional problems quite often arise from the inability to talk and communicate properly with people. Sincere understanding of the patient’s problem and taking time to explain to the patient...
what is being done is extremely important in stabilizing the patient’s emotional status. In general, patients receiving intensive respiratory care have a tendency to develop many cardiopulmonary problems such as pulmonary emboli, cardiac arrhythmias, digitalis intoxication, and so forth. Other complications of assisted ventilation are pneumothorax and mediastinal emphysema, which may cause further deterioration of the pulmonary function; these must always be considered whenever pulmonary function of the patient becomes worse.

Lastly, weaning the patient from ventilation may be a problem. The following guidelines are somewhat useful in deciding when to start weaning a patient:

1) Tidal volume should approach 5 to 6 ml/kg and vital capacity exceeds 10 ml/kg.
2) Alveolar-arterial O₂ pressure gradient should be less than 300 mm Hg on 100% breathing.
3) The ratio between dead space and tidal volume (VD/VT) should be less than 50%.
4) Inspiratory negative pressure should be more than 20 cm H₂O negative pressure.

Weaning is in general complete when vital capacity reaches 20 ml/kg. It should be noted that many patients may require additional O₂ for some days or for weeks after weaning.

Proper humidification must be provided when the patient is off the ventilator. Frequent short periods of spontaneous ventilation, that is, five to ten minutes per hour, should be attempted. As the patient’s tolerance increases, time off the respirator is frequently increased. Vital signs, general appearance of the patient, and arterial gas studies are examined during these periods. The patient should not be allowed to sleep without assisted ventilation until he has tolerated three to four hour periods of spontaneous breathing during the day.

During the weaning period, emotional support is extremely important. A patient who can communicate can get better emotional support. We insert a plastic tracheostomy tube which can be plugged to permit the patient to talk during the period of weaning. This has been very useful during this critical period.

In conclusion, it must be recognized that properly trained physicians, nurses, and inhalation therapists are necessary for the care of acutely and critically ill patients with major pulmonary problems, to prevent and treat complications. Team work is essential for the proper management of those patients who require assisted mechanical ventilation.

BIBLIOGRAPHY


