Sri Lakshmi Narayana Institute of Medical Sciences

Date: 02.05.2018

From
Dr. Nithianandam
Professor and Head,
Department of Anaesthesia
Sri Lakshmi Narayana Institute of Medical Sciences
Bharath Institute of Higher Education and Research
Puducherry

To
The Dean,
Sri Lakshmi Narayana Institute of Medical Sciences
Puducherry

Sub: Request for Permission to conduct value-added course: Mechanical Ventilation

Dear Sir,

With reference to the subject mentioned above, the department proposes to conduct a value-added course titled: Mechanical Ventilation for undergraduates from Jan -June 2019. We solicit your kind permission for the same.

Kind Regards

Dr. NITHIANANDAM. S

FOR THE USE OF DEANS OFFICE

Names of Committee members for evaluating the course:

The Dean: Dr A.SUGUMARAN

The HOD: Dr.NITHIANANDAM. S

The Expert: Dr KALASREE M

The committee has discussed about the course and is approved.

Dean Subject Expert HOD

OFFICE OF THE DEAN

Sri Lakshmi Narayana Institute of Medical Sciences

OSUDU, AGARAM VILLAGE, VILLIANUR COMMUNE, KUDAPAKKAM POST,
PUDUCHERRY - 605 502.
[Recognised by Medical Council of India, Ministry of Health letter No. U/12012/249/2005-ME (P -II) dt. 11/07/2011]
[Affliated to Bharath University, Chennai - TN]

Circular

08.06.2018

Sub: Organizing Value-added Courses: Mechanical Ventilation - reg

With reference to the above mentioned subject, it is to bring to your notice that Sri Lakshmi Narayana Institute of Medical Sciences, **Bharath Institute of Higher Education and Research** is organizing **"Mechanical Ventilation"** course in Jan-June 2019. The course content is enclosed below."

The application must reach the institution along with all the necessary documents as mentioned. The hard copy of the application should be sent to the institution by registered/ speed post only so as to reach on or before 15/06/2018. Applications received after the mentioned date shall not be entertained under any circumstances.

Dean

DEAN
SRI LAKSHMI NARAYANA INSTITUTE OF MEDICAL SCIENCES
OSUDU, AGARAM VILLAGE,
KUDAPAKKAM POST,
PUDUCHERRY - 605 502

Encl: Copy of Course content

COURSE PROPOSAL

Course Title: Mechanical Ventilation

Course Objective:

1. To enable the students to learn about mechanical ventilation- its definition, understanding its principles, indications and contraindications and monitoring a patient on mechanical ventilation.

2. To be acquainted with the newer modes of ventilation

Course Outcome:

On successful completion of the course the students will have skill in identifying patients who require mechanical ventilation and have basic knowledge about the common modes of ventilation

Course Audience: II year MBBS students

Course Coordinator: Dr.NITHIANANDAM. S

Course Faculties with Qualification and Designation:

1. Dr. M Kalasree-Associate Professor

2. Dr Chandrasekar - Assistant Professor

Course Curriculum/Topics with schedule (Min of 30 hours)

S.No	Date	Topics	Time	Hours	Faculty
1	05.01.2019	Introduction	2-4PM	2	Dr Chandrasekar
2	19.01.2019	Anatomy and physiology of respiration	2-4PM	2	Dr. Kalasree
3	02.02.2019	Respiratory failure	2-4PM	2	Dr Chandrasekar
4	09.02.2019	indications	2-4PM	2	Dr. Kalasree
5	16.02.2019	Types of mechanical ventilation	2-4PM	2	Dr Chandrasekar
6	23.02.2019	Negative pressure ventilation	2-4PM	2	Dr. Kalasree
7	02.03.2019	Positive pressure ventilation	2-4PM	2	Dr Chandrasekar
8	09.03.2019	Modes of ventilation	2-4PM	2	Dr. Kalasree
9	16.03.2019	Group discussion	2-4PM	2	Dr Chandrasekar
10	23.03.2019	Triggering mechanism	2-4PM	2	Dr. Kalasree
11	30.03.2019	Monitoring a patient with mechanical ventilation	2-4PM	2	Dr. Kalasree
12	06.04.2019	complications	2-4PM	2	Dr Chandrasekar
13	20.04.2019	Skill station and demonstration	2-4PM	2	Dr. Kalasree
14	27.04.2019	Prone ventilation	2-4PM	2	Dr Chandrasekar
15	04.05.2019	Final assessment	2-4PM	2	Dr. Kalasree

REFERENCES

1) Mason R.J, Braaddus V.C.Murray and Nadel`s :Textbook of Respiratory Medicine. 5th edn. Philadelphia:Saunders;2010

2) George R.B,Light R.W. Chestmedicine:Essentials of Pulmonary and Critical Care Medicine. 4th edn . Philadelphia:Lippincott;2000.

3) Fauci AS, Braunwald E, Kasper DL, Hauser SL, Longo DL, Jameson JL, et al., editors,Harrison’s principles of internal medicine. 17th ed. New York: McGraw Hill; 2008.

VALUE ADDED COURSE

1. Name of the program & Code

MECHANICAL VENTILATION, ANAES 08

2. Duration & Period

30 hrs: Jan 2019-June 2019

3. Information Brochure and Course Content of Value Added Courses

Enclosed as Annexure- I

4. List of students enrolled:

Enclosed as Annexure- II

5. Assessment procedures:

Multiple choice questions- *Enclosed as Annexure- III*

6. Certificate of Participation:

Enclosed as Annexure- IV

7. No. of times offered during the same year:

1 Time JAN 2019-JUNE 2019

8. Year of discontinuation: 2019

9. Summary report of each program year-wise

Value Added Course Jan 2019-June 2019					
Sl. No	**Course Code**	**Course Name**	**Resource Persons**	**Target Students**	**Strength & Year**
1	ANAES 08	MECHANICAL VENTILATION	DR. KALASREE	II MBBS	20

10. Course Feed Back

Enclosed as Annexure- V

RESOURCE PERSON

DR. KALASREE

COORDINATOR

Dr S NITHIANANDAM

Annexure

MECHANICAL VENTILATION

TOPICS:

Anatomy and physiology of respiration

Modes of ventilation

complications

Respiratory failure

indications

Types of ventilation

Modes of ventilation and triggering mechanism

PEEP and NEEP

Triggering mechanism

Monitoring a patient with mechanical ventilation

Prone ventilation

Skill station and demonstration

Group discussion

Final assessment

Introduction

Mechanical ventilation is a procedure often performed in patients in respiratory failure, which is defined broadly as the inability to meet the body's needs for oxygen delivery or carbon dioxide removal. A ventilator delivers air, usually with an elevated oxygen content, to a patient's lungs via an endotracheal tube to facilitate the exchange of oxygen and carbon dioxide.

The indications for mechanical ventilation include airway protection, treatment of hypoxemic respiratory failure (low blood oxygen), treatment of hypercapnic respiratory failure (elevated carbon dioxide in the blood), or treatment of a combined hypoxic and hypercapnic respiratory failure. On some occasions, patients are also intubated and placed on mechanical ventilation for procedures. However, intubation and initiation of mechanical ventilation require a great degree of vigilance, as committing to this therapy can affect the patient's overall course of treatment.

mechanical ventilation is managed by Respiratory Therapists (RTs), highly-trained medical professionals who specialize in the care of respiratory illnesses. In addition to managing ventilators, RTs have expertise in other forms of oxygen administration and respiratory support, provide medications for respiratory disorders, and assess patients for extubation readiness. RTs are essential for the appropriate care of mechanically ventilated patients. Conversely, mechanical ventilation traditionally has not been taught as a core component of many medical and nursing practices outside of Critical Care and Anesthesiology. As such, to collaborate effectively with RTs in the care of ventilated patients, additional education is warranted. Ventilator management can seem intimidating due to varied and confusing terminology (with many clinicians using synonyms for the same modes or settings), slight variation among brands of ventilators, unfamiliarity, or ceding management to others.

For COVID-19 patients, ventilators are often crucial, given the nature of the illness. But note that intubation and initiation of mechanical ventilation require a great degree of vigilance. The data supporting the importance of good ventilator management continues to increase, and appreciating the fundamental principles of ventilation is essential for all clinicians involved in the care of these patients.

VENTILATOR BASICS Control (target) variables are the targets that are set, based on the mode of mechanical ventilation chosen. For example, there are pressure-controlled and volume-

controlled modes of ventilation. In pressure-control, the clinician sets a designated pressure that is delivered with every breatn. In volume-control, they set a designated tidal volume instead. Conditional variables are the dependent variables in mechanical ventilation. For example, in volume controlled modes of ventilation, the tidal volume is a set parameter, while the pressure is a conditional variable, and can vary from breath to breath. Trigger - the factor that initiates inspiration. A breath can be pressure-triggered, flow-triggered, or time-triggered. Cycle – the determination of the end of inspiration, and the beginning of exhalation. For example, the mechanical ventilator can be volume, pressure, or time cycled.

PHYSIOLOGY TERMS

Airway resistance refers to the resistive forces encountered during the mechanical respiratory cycle. The normal airway resistance is ≤ 5 cm H2O. Lung compliance refers to the elasticity of the lungs, or the ease with which they stretch and expand to accommodate a change in volume or pressure. Lungs with a low 2 compliance, or high elastic recoil, tend to have difficulty with the inhalation process, and are colloquially referred to as “stiff” lungs. An example of poor compliance would be a patient with a restrictive lung disease, such as pulmonary fibrosis. In contrast, highly compliant lungs, or lungs with a low elastic recoil, tend to have more difficulty the exhalation process, as seen in obstructive lung diseases. Atelectasis is a complete or partial collapse of the entire lung or area (lobe) of the lung. It occurs when the tiny air sacs (alveoli) within the lung become deflated or possibly filled with alveolar fluid. Derecruitment is the loss of gas exchange surface area due to atelectasis. Derecruitment is one of the most common causes of gradual hypoxemia in intubated patients and can be minimized by increasing PEEP. Recruitment is the restoration of gas exchange surface area by applying pressure to reopen collapsed or atelectatic areas of the lung. Predicted Body Weight is the weight that should be used in determining ventilator settings, (never use actual body weight). Lung volumes are determined largely by sex and height, and therefore, these two factors are used to determine predicted body weight. The formula for men is: PBW (kg) = 50 + 2.3 (height (in) – 60) The formula for women is: PBW (kg) = 45.5 + 2.3 (height (in) – 60)

PHASES of MECHANICAL BREATHING Initiation phase is the start of the mechanical breath, whether triggered by the patient or the machine. With a patient initiated breath, you will notice a slight negative deflection (negative pressure, or sucking). Inspiratory phase is the portion of mechanical breathing during which there is a flow of air into the patient’s lungs to achieve a maximal pressure, the peak airway pressure (PIP or Ppeak), and a tidal volume (TV or VT). Plateau phase does not routinely occur in mechanically ventilated breaths, but may be checked as an important diagnostic maneuver to assess the plateau pressure (Pplat). With cessation of air flow, the plateau pressure and the tidal volume (TV or VT) are briefly held constant. Exhalation is a passive process in mechanical breathing. The start of the exhalation process can be either volume cycled (when a maximum tidal volume is achieved), time cycled (after a set number of seconds), or flow cycled (after achieving a certain flow rate).

Peak Inspiratory Pressure (PIP or Ppeak) is the maximum pressure in the airways at the end of the inspiratory phase. This valve is often displayed on the ventilator screen. Since this value is generated during a time of airflow, the PIP is determined by both airway resistance and compliance. By convention, all pressures in mechanical ventilation are reported in "cm H2O." It is best to target a PIP < 35 cm H2O. Plateau Pressure (Pplat) is the pressure that remains in the alveoli during the plateau phase, during which there is a cessation of air flow, or with a breath-hold. To calculate this value, the clinician can push the "inspiratory hold" button on the ventilator. The plateau pressure is effectively the pressure at the alveoli with each mechanical breath, and reflects the compliance in the airways. To prevent lung injury, the Pplat should be maintained at < 30 cm H2O.

Positive End Expiratory Pressure (PEEP) is the positive pressure that remains at the end of exhalation. This additional applied positive pressure helps prevent atelectasis by preventing the end-expiratory alveolar collapse. PEEP is usually set at 5 cm H2O or greater, as part of the initial ventilator settings. PEEP set by the clinician is also known as extrinsic PEEP, or ePEEP, to distinguish it from the pressure than can arise with air trapping. By convention, if not otherwise specified, "PEEP" refers to ePEEP. Intrinsic PEEP (iPEEP), or auto-PEEP, is the pressure that remains in the lungs due to incomplete exhalation, as can occur in patients with obstructive lung diseases. This value can be measured by holding the "expiratory pause" or "expiratory hold" button on the mechanical ventilator. Driving pressure (ΔP) is the term that describes the pressure change that occurs during inspiration, and is equal to the difference between the plateau pressure and PEEP (Pplat – PEEP). For example, a patient with a Pplat of 30 cm H2O and a PEEP of 10 cm H2O would have a driving pressure of 20 cm H2O. In other words, 20 cm H2O would be the pressure exerted to expand the lungs. Inspiratory time (iTime) is the time allotted to deliver the set tidal volume (in volume control settings) or set pressure (in pressure control settings).

Expiratory Time (eTime) is the time allotted to fully exhale the delivered mechanical breath. I:E ratio, or the inspiratory to expiratory ratio, is usually expressed as 1:2, 1:3, etc. The I:E ratio can be set directly, or indirectly on the ventilator by changing the inspiratory time, the inspiratory flow rate, or the respiratory rate. By convention, decreasing the ratio means increasing the expiratory time. For example, 1:3 is a decrease from 1:2, just like 1/3 is less than 1/2. 4 Peak inspiratory flow is the rate at which the breath is delivered, expressed in L/min. A common rate is 60 L/min. Increasing and decreasing the inspiratory flow is a means of indirectly affecting the I:E ratio. Fraction of inspired oxygen (FiO2) is a measure of the oxygen delivered by the ventilator during inspiration, expressed at a percentage. Room air contains 21% oxygen. A mechanical ventilator can deliver varying amounts of oxygen, up to 100%.

VENTILATOR MODES Assist Control (AC) is a commonly used mode of ventilation, and one of the safest modes of ventilation in the Emergency Department. Patients receive the same breath, with the same parameters as set by the clinician, with every breath. They may take

additional breaths, or over-breathe, but every breath will deliver the same set parameters. Assist control can be volume-targeted (volume control, AC/VC) where the clinician sets a desired volume, or pressure-targeted (pressure control, AC/PC) where the clinician selects a desired pressure.

Synchronized Intermittent Mandatory Ventilation (SIMV) is a type of intermittent mandatory ventilation, or IMV. The set parameters are similar to those in AC, and the settings can be volume controlled (SIMV-VC) or pressure controlled (SIMV-PC). Similar to AC, each mandatory breath in SIMV will deliver the identical set parameters. However, with additional spontaneous breaths, the patient will only receive pressure support or CPAP. For example, in SIMV-VC we can set a TV, and as long as the patient is not breathing spontaneously, each delivered mechanical breath will achieve this tidal volume. However, spontaneous breaths in this mode of ventilation will have more variable tidal volumes, based on patient and airway factors. Pressure Regulated

Volume Control (PRVC) is a type of assist-control thatcombinesthbestattributes of volume control and pressure control. The clinician selects a desired tidal volume, and the ventilator gives that tidal volume with each breath, at the lowest possible pressure. If the pressure gets too high and reaches a predefined maximum level, the ventilator will stop the air flow and cycle into the exhalation phase to prevent excessive airway pressure and resulting lung injury. In this mode of ventilation, thepressure target

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Anatomy and physiology of respiration

Introduction to mechanical ventilation for junior ICU trainees and nurses.

This page is written with the assumption that the reader has a basic understanding of respiratory physiology and respiratory failure

The problem

Getting oxygen in

Oxygen uptake via the lungs is dependent on a number of factors. Some can be manipulated to a large extent by mechanical ventilation:

- PAO2, which in turn can be manipulated by altering:

    - inspired oxygen concentration (FIO2)
    - alveolar pressure
    - ventilation
- ventilation-perfusion matching - by re-opening collapsed alveoli, thereby reducing intra-pulmonary shunting
    - positive end-expiratory pressure (PEEP) helps re-open alveoli and splint open alveoli

Getting carbon dioxide out

- Carbon dioxide elimination via the lungs is largely dependent on alveolar ventilation.
- Alveolar ventilation = Respiratory rate x (tidal volume - dead space)

Main controls

To improve oxygenation:

- increase FIO2
- increase mean alveolar pressure
    - increase mean airway pressure
        - increase PEEP
        - increase I:E ratio
- re-open alveoli with PEEP

To improve CO2 elimination:

- increase respiratory rate
- increase tidal volume

Other controls

Inspiratory time, inspiratory pause and I:E ratio

- inspiratory time is the time over which the tidal volume is delivered or the pressure is maintained (depending on the mode)
    - in time-cycled modes either inspiratory time or I;E ratio are set (flow is adjusted to ensure that the set tidal volume is delivered in that time). These modes include:
        - pressure control
        - volume control (Siemens and Drager ventilators)
        - pressure regulated volume control
    - in volume-cycled modes the flow is set and inspiration ends when the set tidal volume has been delivered. These modes include:
        - volume control (Puritan-Bennett and Bear ventilators)
    - in pressure support mode the patient determines the duration of inspiration
- inspiratory pause time is only set in modes where a fixed tidal volume is set and delivered (volume control and volume preset SIMV modes)

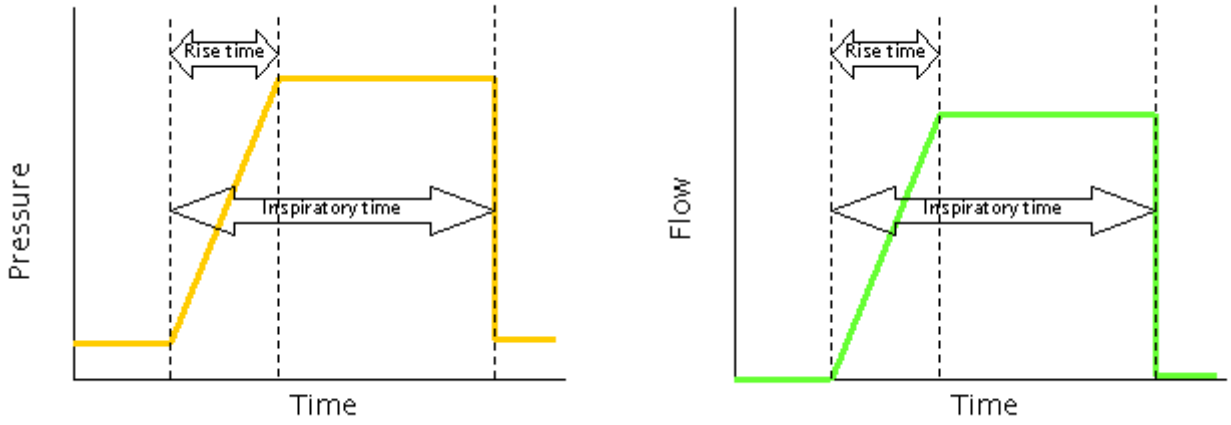
- expiratory time is whatever time is left over before the next breath
- I:E ratio
    - =(inspiratory time + inspiratory pause time):expiration
    - usually set to 1:2 to mimic usual pattern of breathing
- in general longer inspiratory times:
    - improve oxygenation by:
        - increasing the mean airway pressure (longer period of high pressure increases mean airway pressure over the entire respiratory cycle)
        - allowing re-distribution of gas from more compliant alveoli to less compliant alveoli
    - increase risk of gas trapping, intrinsic PEEP and barotrauma by reducing expiratory time
    - are less well tolerated by the patient, necessitating a deeper level of sedation
    - decrease peak pressure by decreasing inspiratory flow

Trigger sensitivity

- this determines how easy it is for the patient to trigger the ventilator to deliver a breath
- in general increased sensitivity is preferable in order to improve patient-ventilator synchrony (ie to stop the patient "fighting" the ventilator) but excessively high sensitivity may result in false or auto-triggering (ie ventilator detects what it "thinks" is an attempt by the patient to breath although the patient is apnoeic)
- triggering may be flow-triggered or pressure triggered. Flow triggering is generally more sensitive.
- the smaller the flow or the smaller the negative pressure the more sensitive the trigger

Rise time

- determines speed of rise of flow (volume control mode) or pressure (pressure control and pressure regulated volume control modes)
- very short rise times may be more uncomfortable for the patient
- long rise times may result in a lower tidal volume being delivered (pressure control mode) or higher pressure being required (volume control and pressure regulated volume control modes)



Modes of ventilation

In general a ventilator can be set to deliver:

- a certain volume of gas in a set period of time
    - the pressure generated in the lung will then be dependent on the resistance and compliance of the respiratory system
    - known as volume control mode

- a certain level of pressure for a set period of time
    - the tidal volume delivered will then be dependent on the resistance and compliance of the respiratory system
    - pressure control and pressure regulated volume control modes
- in assist-control modes (volume control, pressure control, pressure regulated volume control) the ventilator guarantees that the patient will receive the set minimum number of breaths, although he/she is able to demand (trigger) more
- in pressure support modes the patient only receives breaths when he/she triggers the ventilator

Complications

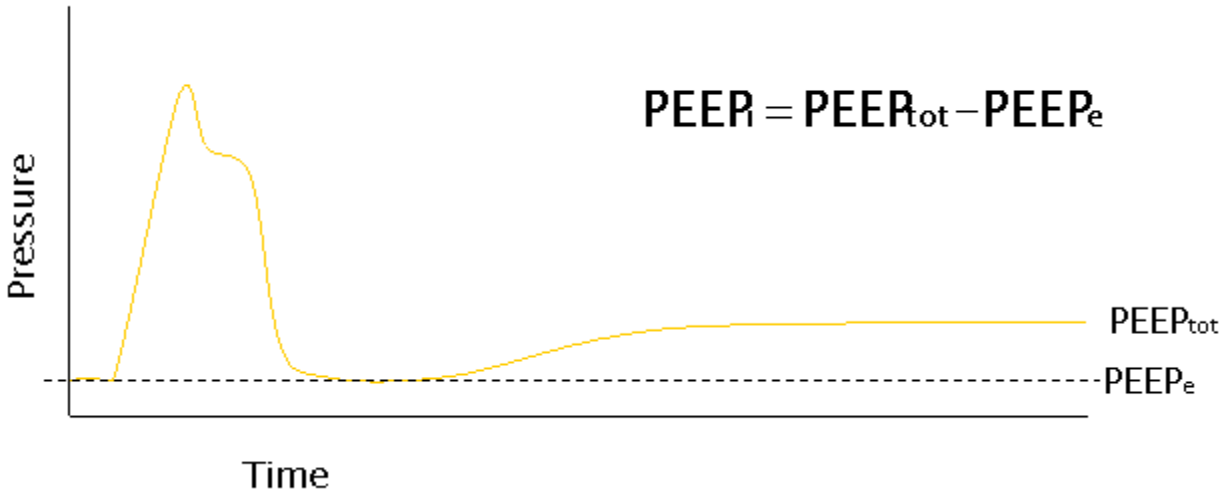
Respiratory

- nosocomial pneumonia
- barotrauma
    - not only due to high pressures also due to high volumes and shear injury (due to repetitive collapse and re-expansion of alveoli and due to tension at the interface between open and collapsed alveoli
    - causes:
        - pneumothorax
        - pneumomediastinum
        - pneumopericardium
        - surgical emphysema
        - acute lung injury
- gas trapping
    - occurs if there is insufficient time for alveoli to empty before the next breath
    - more likely to occur:
        - in patients with asthma or COPD
        - when inspiratory time is long (and therefore expiratory time short)

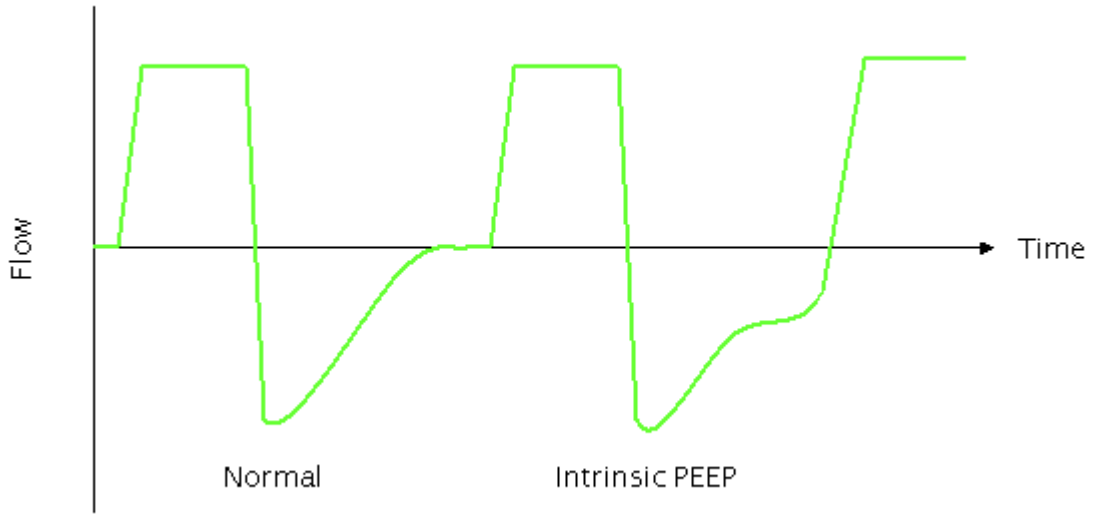
        - when respiratory rate is high (absolute expiratory time is short)
    - results in progressive hyperinflation of alveoli and progressive rise in end-expiratory pressure (known as intrinsic PEEP)
    - may result in:
        - barotrauma
        - cardiovascular compromise due to high intrathoracic pressure. In an extreme case can lead to cardiac arrest with pulseless electrical activity.

Measuring intrinsic PEEP

- quantitative measurement of intrinsic PEEP can be obtained in an apnoeic patient by using the expiratory pause hold control on the ventilator. This allows equilibration of pressures between the alveoli an the ventilator allowing the total PEEP to be measured. The value for total PEEP can be read from the airway pressure dial or the PEEP display
- Intrinsic PEEP=Total PEEP-Set PEEP



- Examination of the flow-time curve from the ventilator gives an indication that there is intrinsic PEEP but does not give an indication of the magnitude. The patient does not need to be apnoeic.



Cardiovascular effects

Preload

- positive intrathoracic pressure reduces venous return
- exacerbated by
  - high inspiratory pressure
  - prolonged inspiratory time
  - PEEP

Afterload

= ventricular wall tension (T) during contraction

$$T = \frac{P_{tm} \times R}{2H}$$

wherePtm=transmural pressure, R=radius and H=wall thickness

Ptm=intracavity pressure-pleural pressure

By increasing pleural pressure positive pressure ventilation decreases transmural pressure and hence afterload

Cardiac output

- reduced preload will tend to decrease cardiac output
- reduced afterload will tend to increase cardiac output

- net effect depends on LV contractility. In patients with normal contractility positive pressure ventilation tends to decrease cardiac output while in patients with decreased contractility it tends to increase cardiac output
- effect on cardiac function also important to remember when weaning patients. Failure to wean may be due to failure to cope with increased preload and afterload

Myocardial oxygen consumption

- reduced by positive pressure ventilation

Respiratory failure

We breathe oxygen from the air into our lungs, and we breathe out carbon dioxide, which is formed in our body as a waste gas. Breathing is essential to life itself. Oxygen must pass from our lungs into our blood for our tissues and organs to work properly.

Respiratory failure is a serious condition that develops when the lungs can't get enough oxygen into the blood. Buildup of carbon dioxide can also damage the tissues and organs and further impair oxygenation of blood and, as a result, slow oxygen delivery to the tissues.

During a physical exam your doctor may do the following:

- **Check for a bluish color** on your lips, fingers, or toes.
- **Listen to your heart** with a stethoscope to check for a fast or irregular heartbeat.

- **Listen to your lungs** with a stethoscope for rapid breathing or any unusual sounds when you breathe. He or she will also see if your chest moves unevenly while you breathe.
- **Measure your blood oxygen level** with a clip on a finger, called pulse oximetry.
- **Measure your blood pressure** to check if it is too high or low.
- **Measure your temperature** to check for a fever and ask if you have recently had a fever.

Diagnostic tests and procedures

To diagnose respiratory failure, your doctor may order some of the following tests and procedures.

- **Arterial blood gas tests** to measure levels of oxygen, carbon dioxide, pH, and bicarbonate. A sample of your blood will be taken from your arteries. These tests help determine whether you have respiratory failure and what type it is.
- **Blood tests** to help find the cause of your respiratory failure. Blood tests can also help your doctor see how well your other organs are working.
- **Bacterial cultures** using samples of your blood, urine, or phlegm (a slimy substance that you cough out) to check for a bacterial infection.
- **Bronchoscopy** to check for blockages, tumors, or other possible causes of respiratory failure.
- **Chest X-ray** to identify any lung or heart conditions that may be causing respiratory failure.
- **Chest computed tomography (CT) scan** to image the lungs and look for inflammation or damage.
- **Electrocardiogram (EKG or ECG)** to check your heart rhythm and how well your heart is working.
- **Echocardiography** to check how well your heart is working.
- **Lung ultrasound** to check for lung conditions such as pleural effusion.
- **Lung biopsy** to collect samples of your lung tissue.
- **Pulmonary function tests** to measure how well your lungs are working

Any condition or injury that affects breathing can cause respiratory failure. The condition or injury may affect your airways or lungs. Or it may affect the muscles, nerves, and bones that help you breathe.

When you can't breathe well, your lungs can’t easily move oxygen into your blood or remove carbon dioxide. This causes a low oxygen or high carbon dioxide level in your blood. Learn more about how your lungs normally exchange oxygen and carbon dioxide

Respiratory failure can be caused by:

- **Conditions that make it difficult to breathe in and get air into your lungs.** Examples include weakness following a stroke, collapsed airways, and food getting stuck in and blocking your windpipe.
- **Conditions that make it difficult for you to breathe out.** Asthma causes your airways to narrow, while COPD can cause mucus to buildup and narrow your airways, which can make it hard for you to breathe out.

- **Lung collapse.** When no air is able to enter your lungs, one or both lobes may collapse and cause a condition called atelectasis. This collapsing of the lung can happen in certain situations, such as when the lungs become extremely weak, mucus blocks one of the large airways, a rib is broken or fractured, or severe pain in the lung makes it difficult to take a deep breath. Chest trauma or lung injury can also cause air to leak from the lung, filling the space around the lung within the chest. This air could cause the lung to collapse, called a pneumothorax.
- **Fluid in your lungs.** This makes it harder for oxygen to pass from the air sacs into your blood and for carbon dioxide in your blood to pass into the air sacs to be breathed out. Pneumonia, acute respiratory distress syndrome (ARDS), drowning, and other lung diseases can cause this fluid buildup. It can also be caused by the inability of the heart to pump enough blood to the lungs (called heart failure). Severe head injury or trauma can also cause sudden fluid buildup in the lungs.
- **A problem with your breathing muscles.** Such problems can occur after a spinal cord injury or when you have a nerve and muscle condition such as muscular dystrophy. It can also happen when your diaphragm and other breathing muscles do not get enough oxygen-rich blood, when the heart is not pumping well enough (cardiogenic shock), or when you get a severe infection called sepsis.
- **Conditions that affect the brain's control over breathing.** In opioid overdose, for example, the brain may not detect high levels of carbon dioxide in the blood. Normally, the brain would signal to you to deepen your breathing so that you breathe out the carbon dioxide. Instead carbon dioxide builds up in the body, while oxygen levels fall, leading to respiratory failure.

Indications for mechanical ventilation

- **Nerve and muscle disorders** such as amyotrophic lateral sclerosis, Guillain-Barre syndrome, and myasthenia gravis. A stroke can also affect the area of the brain that controls breathing.
- **Lung and airways diseases,** such as asthma, cystic fibrosis, COPD, and interstitial lung diseases. Fluid buildup in the lungs or pulmonary embolism (a blood clot in your lungs) can also lead to respiratory failure.
- **Infections** in your brain or spinal cord (such as meningitisexternal link), lungs (such as pneumonia), or airways (such as bronchiolitis). Watch this video to learn how infection with SARS-CoV-2, the virus responsible for COVID-19, can affect the lungsexternal link.
- **Airway blockages,** such as when food or another object gets stuck in your airways or your epiglottis swells. Your epiglottis is the flap at the back of your throat that prevents food or drink from getting into your airways when you swallow. During obstructive sleep apnea, your upper airway becomes blocked repeatedly during sleep, reducing or completely stopping airflow.
- **Chest or back injuries** that damage your ribs or lungs.
- **Severe scoliosis,** a condition in which the spine is curved from side-to-side.
- **Severe allergies to food or medicine** can cause your throat to swell up.

Phases of mechanical ventilation and Types of mechanical ventilation

Bag mask ventilation. You may wear a mask attached to a bag to get more air into your lungs. This is often done while you are waiting for a complex procedure to treat the cause of your respiratory failure.

Noninvasive positive pressure ventilation (NPPV). This treatment uses mild air pressure to keep your airways open while you sleep. You wear a mask or another device that fits over your nose or your nose and mouth. A tube connects the mask to a machine that blows air into the tube. Continuous positive airway pressure (CPAP) is one type of NPPV. For more information, visit the CPAP Health Topic. Although it focuses on CPAP treatment for sleep apnea, it explains how CPAP works.

A mechanical ventilator, if the oxygen level in your blood doesn't increase, or if you're still having trouble breathing. A ventilator is a machine that supports breathing. It blows air—or air with increased amounts of oxygen—into your airways and then your lungs. Using a ventilator, especially for a long time, can damage your lungs and airways and cause infections such as pneumonia.

A tracheostomy to deliver oxy

Modes of ventilation and Triggering

Although mechanical ventilation can be a complex and seemingly elusive topic, expectations are that physicians and healthcare professionals who deal with critically ill patients have a basic familiarity with the management of a patient on a ventilator. Additionally, providers must also understand how applying mechanical ventilation affects patient physiology and response to disease states. The focus of this article will be on the management of the intubated patient in the

first few hours of care on mechanical ventilation and will review the basics of mechanical ventilation.

The primary indications for mechanical ventilation are:

1. Airway protection in a patient who is obtunded or has a dynamic airway, e.g., from trauma or oropharyngeal infection
2. Hypercapnic respiratory failure due to a decrease in minute ventilation
3. Hypoxemic respiratory failure due to a failure of oxygenation
4. Cardiovascular distress whereby mechanical ventilation can offload the energy requirements of breathing
5. Expectant course, e.g., anticipated patient decline or impending transfer

Function

Mechanical ventilation works by applying a positive pressure breath and is dependent on the compliance and resistance of the airway system, which is affected by how much pressure must be generated by the ventilator to provide a given tidal volume (TV). The TV is the volume of air entering the lung during inhalation.[1] Compliance and resistance are dynamic and can be affected by the disease state(s) that led to the intubation. Understanding the changes in compliance and resistance will allow you to pick the proper ventilator strategies.

There are four stages of mechanical ventilation.

] There is the trigger phase,

the inspiratory phase,

the cycling phase

, and the expiratory phase

. The trigger phase is the initiation of an inhalation which is triggered by an effort from the patient or by set parameters by the mechanical ventilator. The inhalation of air into the patient defines the inspiratory phase. The cycling phase is the brief moment when inhalation has ceased but before exhalation has begun. The expiratory phase is the passive exhalation of air from the patient.

Once the decision is made to place a patient on mechanical ventilation, the clinician may be presented with multiple different options on how to set up the ventilator. There are many kinds of modes of ventilation, such as assist-control (AC), synchronized intermittent mechanical ventilation (SIMV), and pressure support ventilation (PSV). [2] The ventilator can then be set to provide a given volume or pressure. Several leading voices in emergency medicine and critical care recommend utilizing volume assist control (VAC) as it is simple to use, safe, and available on all ventilators. [3] Further, it provides complete ventilator support which offsets fatigue in patients who are critically ill.

After choosing the mode, the rest of the parameters have to be set on the ventilator. These parameters are the respiratory rate (RR), inspiratory flow rate (IFR), the fraction of inspired oxygen (FI02), and positive end-expiratory pressure (PEEP). Respiratory rate is generally adjusted to move towards normocapnia, or to offset severe acidosis. The inspiratory flow rate is how fast the inspiration is provided, generally expressed in liters/minute. [2] FI02 is the fraction of inspired air and should be set to the lowest level to achieve an SP02 of 92-96%, as hyperoxemia has been shown to increase mortality in critically ill patients. [4] PEEP is used to increase the functional residual capacity, and to stent open collapsable alveoli thus reducing atelectatic-trauma. [1] Finally, all patients on mechanical ventilation should have the head of the bed elevated to at least 30 degrees and have continuous waveform end-tidal carbon dioxide (CO2) (ETCO2) monitoring. According to a 2016 Cochrane review on ventilator-associated pneumonia (VAP), "a semi-recumbent (30º to 60º) position reduced clinically suspected VAP by 25.7% when compared to a 0° to 10° supine position", however, they acknowledge that the data is severely limited. [5]

Clinical Significance

Three clinical strategies may be chosen to assist in ventilator management.

Lung Protective Strategy:

This strategy should be used for any patient with the potential to develop acute lung injury (ALI) or whose disease state risks progression to acute respiratory distress syndrome (ARDS). This low tidal volume (LTV) strategy developed after the landmark ARDSnet trials, specifically the ARMA study, which showed that low tidal volume ventilation in patients with ARDS improved mortality. [7] This method is used to avoid barotrauma, volume trauma, and atelectatic trauma. Pneumonia, severe aspiration, pancreatitis, and sepsis are examples of patients with the acute potential to develop ALI and should be managed with the LTV strategy.

Tidal volume (TV) should be initially set at 6 ml/kg based upon ideal body weight.[8][7][9][10] As patients develop ALI and progress into ARDS, their lungs become progressively recruited and develop shunts, which leads to decreased functional lung volume. [3] A low tidal volume strategy offsets the decreased functional lung volume. Tidal volume should not be adjusted based on minute ventilation goals. Respiratory rate is adjusted based upon minute ventilation goals and the acid-base status of the patient. An initial rate of 16 breaths/minute is appropriate for most patients to achieve normocapnia. [11] A blood gas should be sent approximately 30 minutes after initiation of mechanical ventilation and RR adjusted based upon the acid-base status and PaCO2 of the patient. Normocapnia is a PaCO2 of 40 mmHg. If the PaCO2 is significantly greater than 40, then the RR should be increased. If the PaCO2 is significantly lower than 40, then the RR should be decreased. It is important to remember that the ETCO2 is not a reliable indicator of PaCO2 as the ETCO2 can be affected by the physiological shunt, dead space and decreased cardiac output. [3] Inspiratory flow rate should be set at 60L/minute. If the patient appears to be trying to inhale more during the initiation of inspiration, then it can be increased. [3]

Immediately after intubation, an attempt should be made to reduce the FI02 to 40% to avoid hyperoxemia. [4] From there, adjustments of the FI02 and PEEP are simultaneously controlled in the lung-protective strategy. Difficulty in oxygenation in ALI is due to de-recruited alveoli and

physiological shunt. To counteract this, you should increase the FIO2 and PEEP together. The oxygenation goal of 88%-95% should follow the ARDSnet protocol. [9]

Table 1.ARDSnet PEEP/FIO2 Protocol [9]

Upon connecting a patient to mechanical ventilation, it is essential to frequently reassess its effects on the patient, especially the alveoli. This assessment is by examining the plateau pressure and driving pressure. The plateau pressure is the pressure applied to small airways and alveoli. The plateau pressure should be under 30, to prevent volume trauma, which is an injury to the lung secondary to overdistension of the alveoli. To obtain the plateau pressure, one must perform an inspiratory pause. Most ventilators have a button to calculate it. The driving pressure is the ratio of the tidal volume to the compliance of the lung, providing an approximation of the "functional" amount of lung that hasn't been de-recruited or shunted. [12] The driving pressure can be calculated simply by subtracting the amount of PEEP from the plateau pressure. [12] The driving pressure should remain below 14. If plateau and driving pressures start to exceed these limits, then decrease TV to 4ml/kg. Respiratory rate can be increased to compensate for the decrease in minute ventilation, though permissive hypercapnia might be necessary. Permissive hypercapnia is a "ventilation strategy to allow for an unphysiologically high partial pressure of carbon dioxide (PCO) to permit lung-protective ventilation with low tidal volumes." [13] Recruitment maneuvers have been found to increase mortality in moderate to severe ARDS and should not be routinely used. [14]

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Airway Pressure Release Ventilation:

APRV is a form of continuous positive airway pressure (CPAP) that is characterized by a timed pressure release while allowing for spontaneous breathing. [16](See Figure 1) While previously considered a rescue strategy, APRV has recently gained acceptance as a primary ventilatory mode. Its indications for Acute Lung Injury (ALI)/Acute Respiratory distress syndrome (ARDS), multifocal pneumonia, and severe atelectasis make it a very attractive ventilatory option.

APRV functions by providing a continuous pressure to keep the lungs open with a timed-release to a lower set pressure.[17][18] The continuous pressure phase of APRV transmits pressure to the chest wall which allows for the recruitment of both proximal and distal alveoli. The prolonged continuous pressure phase with the short release phase avoids the continuous cycles of recruitment-derecruitment that occur in pressure/volume control vent settings.[19] This helps to avoid atelectrauma, barotrauma and resulting ventilator induced lung injury. [19](See Figure 2) The timed release allows for a passive exhalation and improved clearance of CO2. Since APRV relies upon spontaneous ventilation it requires less sedation compared to conventional modalities thus mitigating adverse events due to sedation. Spontaneous breathing has the benefit of increasing end-expiratory lung volume, decreasing atelectasis, and improves ventilation to dependent lung regions. [19] Spontaneous breathing, further, improves the hemodynamic profile by decreasing intrathoracic pressure, thus

.

Enhancing Healthcare Team Outcomes

The management for a patient on mechanical ventilation requires an interprofessional team involving physicians, nurses, and respiratory therapists. Good communication among the team is paramount. Respiratory therapists provide a crucial role in managing the ventilated patient, and their expertise should be utilized extensively. [22] Finally, only one dedicated professional should be in charge of the ventilator and vent changes should not be made without communication with others in charge of the patient. [Level III]

Types oPhase variables: triggering, limits, cycling and PEEP

Phase variables in mechanical ventilation are parameters which control the phases of a mechanical breath. Triggering controls the initiation of inspiration, cycling controls the initiation of expiration, and limits are set to maintain control over the three main parameters while inspiration is taking place. PEEP is also viewed as a phase variable, for lack of a better classification, and is the variable which reigns over the otherwise very boring expiratory phase. CICM have shown some interest in these matters over the years, and the

In short:

- The trigger variable determines how and when the ventilator ends exhalation and commenced inhalation.
  - The trigger setting can be time, flow, pressure or volume
  - This variable determines whether a mode of ventilation is “mandatory” (machine triggered) or “spontaneous” (patient-triggered)
- The limit variable restrict the maximum value which the parameters can achieve during inspiration.
  - Reaching the limit variable during inspiration does not abort the inspiratory phase
  - This is distinct from the alarm variables, which are activated whenever their values are breached, and which abort inspiratory flow or open the expiratory valve
  - Several limit variables can be selected simultaneously.
- The cycling variable is measured during the inspiratory phase; it is the mechanism used to end inspiration and commence expiration
  - Time and flow are the most common settings for this varible, though volume-cycled and pressure-cycled ventilation is also possible
  - Only one cycling variable can be set at any given time
- The PEEP variable is the pressure setting which determines the pressure maintained by the circuit bias flow during expiration.

Trigger variable

The trigger variable determines when a breath is delivered. This variable distinguishes “mandatory” from “spontaneous” modes of ventilation; where “mandatory” refers to the fact that the ventilator decides when you take a breath, usually according to a timer. In the dark ages of critical care, this was the only sort of ventilation available to the slightly comatose patient.

Limit, or “target” variables

The limit variable is the unimaginative name given to the limits of the mechanical breath. One usually has only minimal control over these, as they are integral parts of the selected mode of ventilation. In short, a limit variable is the maximum value a variable can attain during inspiration; and this is something distinct from the alarm settings, which can also be viewed as “limits”. Alarm settings tend to terminate the inspiration by opening the expiratory valve, whereas the main characteristic of the limit variable is that the inspiratory phase continues even after the limit has been reached.

Specifically, the term “limit variable” refers to the inspiratory phase: during inspiration, the ventilator won’t let that parameter (flow, volume, pressure, etc) get beyond its limit value. That is not to say that those parameters are completely ignored during the other phases: there are still limits in place but they fall into the territory of safety parameters. For example, let us take this pressure control mode of ventilation as depicted below.

The PEEP is set as 10 cm H_2O, and the pressure control variable is 20 cm H_2O. Thus, the pressure limit during inspiration is 30 cm H_2O. The pressure will get no higher than this during inspiration. In addition to this limit, there is also as safety limit which is set as one of the ventilator alarms, which is (conventionally) set as 40 cm H_2O. This is not unique to the inspiratory phase- breaching this limit during any phase of ventilation will abort the delivery of flow to prevent injury. This is clearly a confusing distinction: both values can be referred to as “limits”. Because of this, the ISO has moved away from referring to this phase variable as “limit” – they would prefer us to call it a “target” variable, whereas the term “limit” should be restricted to the abovementioned safety alarms.
Limit variables can include flow, volume and pressure. Time is obviously not a limit variable because it would defy logic. All limit variables can be active simultaneously, i.e. one can have a mechanical breath which is pressure-limited flow-limited and volume-limited.

Cycling variable

The cycling variable determines how and when the ventilator transitions from inspiration to expiration. The ventilator measures this variable during the inspiration phase. The cycling variable is distinct from the limit variable, in that it ends the inspiratory phase when it is reached. When the set parameter for this variable is achieved, the ventilator opens the expiratory valve, and expiration may begin. Typical methods of ventilator breath cycling include:

- Time-cycled ventilation (mandatory modes)
- Flow-cycled ventilation (spontaneous modes, eg. pressure support)
- Volume-cycled ventilation
- Pressure-cycled ventilation

Unlike the limit variable (of which several can be active simultaneously), there can only be one cycling variable. In general, time and flow are the most popular settings. Volume and pressure cycled ventilation is something of a historicalfootnote.

PEEP – the expiratory phase variable

The expiratory phase in modern ventilators is generally a fairly passive and unexciting time, where the only thing happening is a slow bias gas flow seeping out of the circuit via the expiratory solenoid valve. This phase generally only has one variable applied to it, which is PEEP. In the absence of any pressure, one should probably refer to it as ZEEP (zero end-expiratory pressure). Historically, during the Dark Age of critical care many physicians held heretical beliefs regarding the need to actively assist patients with expiration, and s

NEEP (negative end-expiratory pressure)

was used as a means of promoting expiratory airflow. A representative example from that era is this article by Hill et al (1965), instructing people on the correct use of a -4 cm H2O NEEP for post-operative cardiac surgery patients via the Engstrom respirator. Around the 1960s-1970s people finally realised that this practice was insane, and ventilator manufacturers stopped incorporating sub-ambient pressure into their equipment.

Control variables, or “target” variables

The control variables are the independent limit variables in a mode of ventilation. In essence, the control variable is the constant to which all other variables are enslaved. There are only two possible control variables: pressure and flow. These are discussed in another chapter because they seemed like something fundamentally important and therefore deserving of a separate page.

Monitoring a patient under mechanical ventilation

- **espiratory rate**—To detect apnea, set or measured respiratory rate can be measured by airflow sampling, capnography, inductive plethysmography, oscillometry frequency-based changes, ECG, or ventilation acoustics.
- **Physical examination**—Vigilant physical assessment of chest excursion is a necessity for accurate monitoring of mechanical ventilation. Asymmetric chest motion or unilateral breath sounds may indicate pneumothorax, endobronchial intubation, or atelectasis. Paradoxical chest motion can signify flail chest or respiratory muscle dysfunction. Poor synchrony of a patient's breathing pattern with the ventilator's drive may indicate that the ventilator settings are inappropriate or that the patient's depth of anesthesia is too light. Tympanic percussion or tracheal deviation could help diagnose a pneumothorax. Audible endotracheal leaks around the airway cuff indicate insufficient air or a potential cuff rupture.
- **Movement of reservoir bag**—Free and unencumbered movement of reservoir bag during spontaneous ventilation assures a patent airway or early detection of circuit obstruction.
- **Breath sounds**—Continuous auscultation with a precordial or esophageal stethoscope is extremely valuable in detecting disconnects, leaks, airway obstruction by secretions or bronchospasm, and apnea.

MONITORING GAS EXCHANGE

Adequacy of mechanical ventilation can be determined by the ability of the patient to maintain ventilation and oxygenation. Pulse oximetry and capnography are two standard American Society of Anesthesiologists (ASA) monitors that are utilized for this purpose. Furthermore, arterial blood gas analysis provides significant insight into ventilatory status. A low Pao_2 on an arterial blood gas (ABG) indicates hypoxemia—a dysfunction of the ability to oxygenate arterial

blood. A number of ventilator factors can directly affect the Pao_2: chiefly, the Fio_2, positive end-expiratory pressure (PEEP) level, and the patient's lung function. It is important to interpret the Pao_2 as a function of these dependent variables, as a "normal" Pao_2 does not necessarily indicate ideal physiologic pulmonary function.

MONITORING VENTILATORY DRIVE AND BREATHING PATTERN

Partial ventilator support can also be utilized through pressure support and synchronized ...

Caring for the Mechanically Ventilated Patient Mechanical ventilation is utilized in intensive care and long-term care settings to assist patients who require additional respiratory support. This handy reference guide provides critical patient care essentials, tips for trouble-shooting ventilator alarms, and potential complications. Care Essentials for Patients on Mechanical Ventilation

• Maintain a patent airway. Per policy, note endotracheal (ET) tube position (centimeters) and confirm that it is secure.

• Assess oxygen saturation, bilateral breath sounds for adequate air movement, and respiratory rate per policy

. • Check vital signs per policy, particularly blood pressure after a ventilator setting is changed. Mechanical ventilation increases intrathoracic pressure, which couldaffect blood pressure and cardiac output.

• Assess patient's pain, anxiety and sedation needs and medicate as ordered.

• Complete bedside check: ensure suction equipment, bag-valve mask and artificial airway are functional and present at bedside. Verify ventilator settings with the prescribed orders

. • Suction patient only as needed, per facility policy; hyperoxygenate the patient before and after suctioning and do not instill normal saline in the ET tube; suction for the shortest time possible and use the lowest pressure required to remove secretions. Monitor for upper airway trauma as evidenced by new blood in secretions.

• Monitor arterial blood gas (ABG) after adjustments are made to ventilator settings and during weaning to ensure adequate oxygenation and acid-base balance.

• To minimize the risk for ventilator-associated pneumonia (VAP), implement best practices such as strict handwashing; aseptic technique with suctioning; elevating head of bed 30-45 degrees (unless contraindicated); providing sedation vacations and assessing patient's readiness to extubate; providing peptic ulcer disease prophylaxis; providing deep vein thrombosis prophylaxis; and performing oral care with chlorhexidine, per your facility policy.

VENTILATOR ALARMS Alarm Potential Causes Interventions High peak inspiratory pressure (PIP)

• Blockage of ET tube (secretions, food, kinked tubing, patient biting on ET tube)

• Coughin

• Bronchospasm

• Lower airway obstruction

• Pulmonary edema

• Pneumothorax

• Assess lung sounds.

• Suction airway for secretions

Ventilator/patient dyssynchrony • Assess breath sounds for increased consolidation, wheezing, and bronchospasm; treat as ordered. Low pressure alarm

• Air leak in ventilator circuit or in the ET tube cuff • Locate leak in ventilatorsystem.

• Check pilot balloon as an indicator of ET tube cuff failure

. • Replace tubing as needed, per policy. Low minute ventilation (VE)

• Low air exchange due to shallow breathing or too few respirations • Check for disconnection or leak in the system.

• Assess patient for decreased respiratory effort. Low O2 saturation (SpO2)

COMPLICATIONS RELATED TO MECHANICAL VENTILATION Patient Complication Potential Causes Interventions Cardiovascular issues • Decrease in venous return to the heart due to positive pressure applied to the lungs. • Assess for adequate volume status by checking heart rate, blood pressure, central venous pressure and urine output. • Assess patient for increasing autopeep, which can increase risk for cardiac tamponade. Barotrauma/pneumothorax • Positive pressure applied to lungs. • Elevated mean airway pressures may rupture alveoli. • Notify healthcare provider. • Prepare patient for possible chest tube insertion. • Avoid high pressure settings for patients with chronic obstructive pulmonary disease (COPD), acute respiratory distress syndrome (ARDS), or history of pneumothorax. Infection • Breaks in ventilator circuit. • Decreased mobility. • Impaired cough reflex. • Use aseptic technique. • Provide frequent mouth care. • Support proper nutritional statuS

1. Care of a patient under mechanical ventilation

Fluids. You may be given fluids to improve blood flow throughout your body. Fluids are usually given through an intravenous (IV) line inserted in one of your blood vessels.

Nutritional support. You may need a feeding tube to make sure you get enough of the right nutrients while you are on a ventilator.

Physical therapy. This can help maintain muscle strength and prevent sores from forming. Movement may also help shorten the time you are on a ventilator and improve recovery after you leave the hospital.

Positioning your body. For severe respiratory failure, your doctor may recommend that you spend most of the time lying facedown, which helps oxygen get to more of your lungs.

Pulmonary rehabilitation. This program of education and exercise teaches you breathing techniques that can improve your oxygen levels.

Blood-thinning medicine. If you are very sick or got sick very quickly, this medicine can prevent blood clots from forming. If you cannot use a blood thinner for some reason, your doctor may order special stockings or devices to increase the pressure on your legs.

Prone ventilation

it took almost 40 years from the early recommendation by BRYAN [1] to use prone positioning in patients with acute respiratory distress syndrome (ARDS) to the demonstration of its beneficial effect on patient survival Meanwhile, research has explored the mechanisms by which prone positioning could improve oxygenation and reduce ventilator-induced lung injury (VILI). Furthermore, several randomised controlled trials (RCTs) have been performed to test the effect of prone positioning on patient outcome. Of interest over these 40 years was the continuous interaction between pathophysiological advances and the search for clinical evidence of efficiency, and the continuous refinement in trial design. The aims of this article are to summarise the rationale for prone positioning, the level of evidence supporting its use, its limitations and its place in the current management of ARDS.“Acute Respiratory Distress Syndrome”Edited by S. John Wort and Stefano NavaNumber 1 in the Series

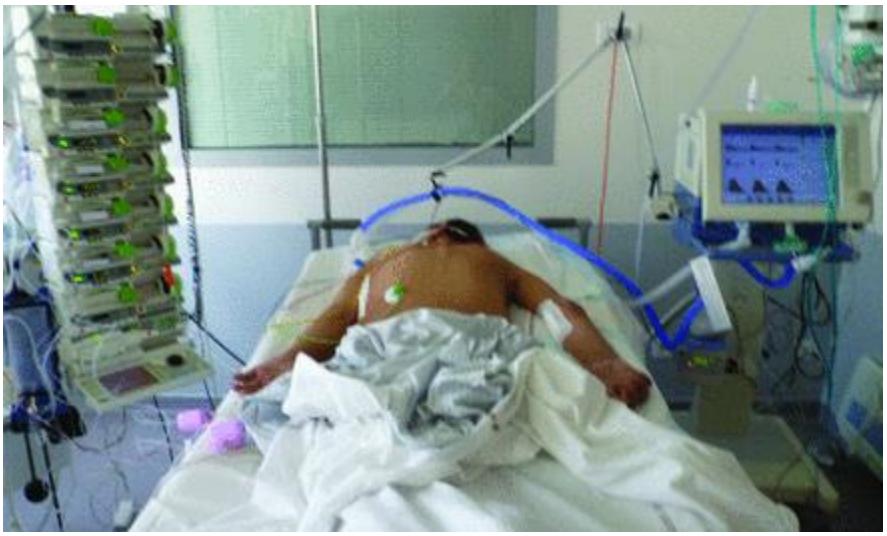


Figure 1.
A patient with acute respiratory distress syndrome receiving mechanical ventilation in the intensive care unit while in the prone position.

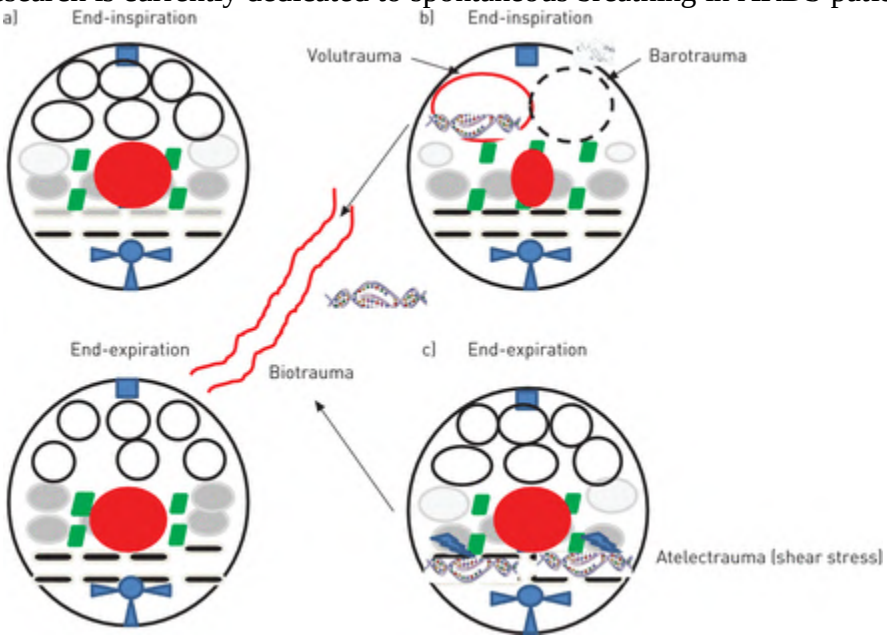
Rationale

Oxygenation

From an historical point of view, the first rationale for using prone positioning in ARDS patients is improvement in oxygenation. We now know that this goal is less important than other rationale that will be discussed later. A large number of reports have shown that the improvement in oxygenation in ARDS was frequent, observed in ~75% of the cases, and sometimes dramatic, *i.e.* during a prone session. The oxygenation response is commonly defined as an increase in the arterial oxygen tension (P_{aO2}) by 20% or an increase in the P_{aO2}/inspiratory oxygen fraction (F_{IO2}) ratio by ≥20 mmHg from the supine position. Therefore, from the onset prone positioning has mainly been thought of and used as a rescue therapy to relieve life-threatening hypoxaemia. For instance, in 1997 MURE *et al.* [3] reported on 13 ARDS patients, mostly secondary lung injury (indirect lung injury stemming from nonrespiratory severe sepsis/septic shock), who received low positive end-expiratory pressure (PEEP) and high F_{IO2}. 11 of the patients had a P_{aO2}/F_{IO2} ratio of <100 mmHg in the supine position and in four patients this ratio was <50 mmHg. In six patients, the P_{aO2}/F_{IO2} ratio increased by a factor of at least four in the prone position. From their results, the authors concluded that prone positioning should be used as a first-line therapy before the use of nitric oxide and extracorporeal membrane oxygenation (ECMO). Moving forward into the RCT era, the effects on oxygenation were then assessed for prone positioning using a group, not just as a single session. Therefore, the effects on oxygenation in the prone position group were compared to the supine position group. Meta-analyses showed a significantly greater risk, ~30–40%, for better oxygenation in the prone position group [4, 5].

Ventilator-induced lung injury

The second rationale to use prone positioning is in the prevention of VILI [9]. Preventing VILI has been established as the primary goal of mechanical ventilation after the ARDS network demonstrated that lower V_T improved survival compared to higher V_T in ARDS patients [10]. This trial was the ultimate demonstration, after decades of research [11, 12], that lung overdistension was detrimental not only to lung healing but also to patient outcome. Lung overdistension is the first component of VILI and, thus, preventing lung overdistension is the main concern of intensivists when setting mechanical ventilation. As it has become clear that lung overdistension pertains to volutrauma [13], not barotrauma (fig. 2), the relevant measurement should not be airway pressure but transpulmonary pressure determination. Transpulmonary pressure is the difference between alveolar pressure (airway pressure at zero airflow when the alveoli and conducting airways are communicating) and pleural pressure. Transpulmonary pressure can even be high if airway pressure is low. The typical example is pressure support ventilation when mechanical breaths are synchronised with a patient's inspiratory effort, a combination that may promote very high V_T, causing a risk for volutrauma [14]. Active research is currently dedicated to spontaneous breathing in ARDS patients.



a) The distribution of normally aerated (white circles), poorly aerated (grey circles), non-aerated (black rectangle) and consolidated (green rectangle) lung areas during acute respiratory distress syndrome while in the supine position during end-inspiration and end-expiration. b) Barotrauma (alveolar rupture with air leaks) and volutrauma (overdistension in the normally aerated lung areas). c) Atelectrauma, *i.e.* shear stress in the poorly aerated lung areas close to the consolidated non-recruitable lung areas. Biotrauma (biochemical and biological response) results from volutrauma and/or atelectrauma with activation of pro-inflammatory mediators within the lungs and distant end organs. The red circle represents the heart.

The second component of VILI is called atelectrauma (fig. 2), which results from the repeated opening and closing of the small airways [15]. When V_T is reduced, atelectrauma may be less important than overdistension as three large RCTs failed to demonstrate a beneficial effect on

patient outcome of higher PEEP compared to lower PEEP [16–18]. However, individual meta-analysis found a statistically significant lower mortality rate in the higher PEEP group in the subgroup of patients with a P_{aO2}/F_{IO2} ratio ≤200 mmHg [19].

A clear and direct demonstration that prone positioning can prevent VILI was reported by BROCCARD *et al.* [20] who ventilated normal dogs with 77 $mL \cdot kg^{-1}$ measured body weight V_T to reach a transpulmonary plateau pressure of 35 cmH_2O. During 6 h of such mechanical ventilation in the supine position the lungs were macroscopically and microscopically severely injured. When the dogs received the same ventilator settings for 6 h in the prone position the lung injury was markedly reduced. Furthermore, the histological lung injury due to high V_T was more homogeneously distributed throughout the lungs in the prone position.

Further findings pertaining to VILI prevention using prone positioning have increased over time in the literature. Prone positioning makes the following more homogeneously distributed in the anterior-to-posterior direction throughout the lungs: lung densities (**fig. 3**) [7, 21], as previously discussed; intrapulmonary shunt [22]; lung ventilation [23]; and transpulmonary pressure [24]. By favouring such a homogenisation, prone positioning prepares the lung to receive the strain imposed by mechanical ventilation [25], and hence makes the distribution of the resulting stress more homogeneous across the lung. Stress/strain homogenisation is associated with less risk for VILI. Furthermore, the global lung stress and strain is reduced in the prone position. MENTZELOPOULOS *et al.* [26] found that transpulmonary pressure, *i.e.* lung stress, and V_T to end-expiratory lung volume ratio, *i.e.* lung strain, were lower in the prone position than in the supine position. GALIATSOU *et al.* [27] performed a lung CT scan in ARDS patients in the supine position and then in the prone position, same patient restricted to different interventions. The authors found that the prone position was associated with significant alveolar recruitment and less hyperinflation compared to the supine position, and that these effects were more important in lobar than diffuse ARDS anatomical pattern. These findings were confirmed and even expanded on by CORNEJO *et al.* [28], who assessed the lung recruitability in the supine position. The prone position promoted lung recruitment and reduced overdistension in patients in both categories of low and high recruitability at either low or high PEEP in the prone position. However, tidal recruitment and derecruitment, *i.e.* cyclic opening and closing of the small airways (atelectrauma), and tidal hyperinflation were significantly reduced in the prone position in only the subgroup of ARDS patients who had high recruitability in the supine position and who were receiving higher PEEP in the prone position.

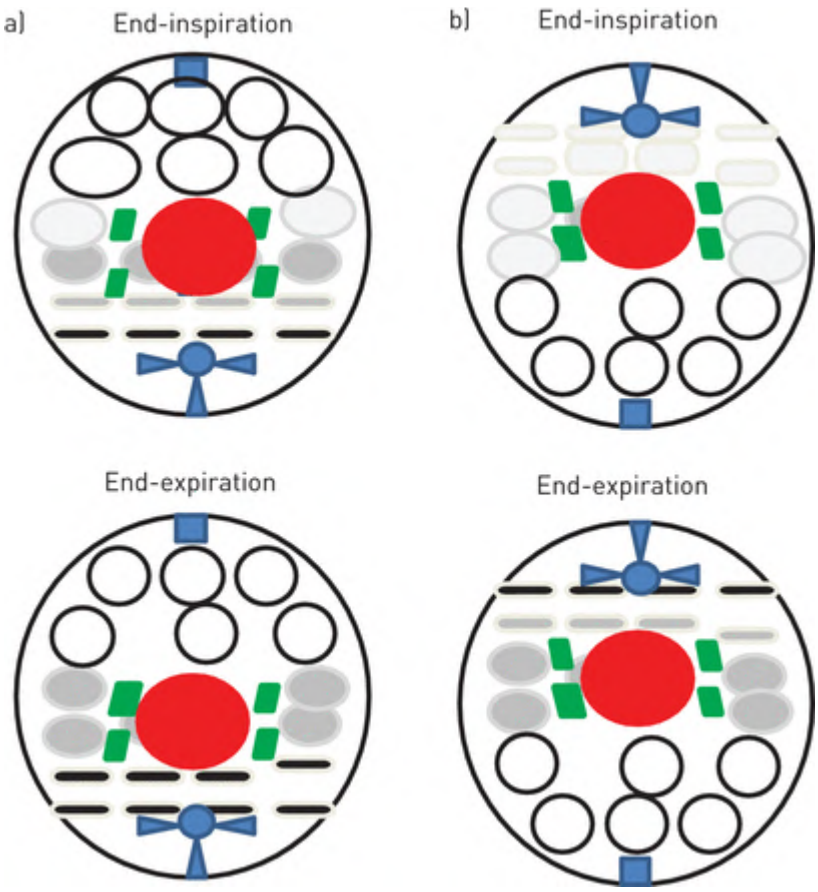


Figure 3.
Homogenisation of the distribution of lung aeration as a result of moving from a) the supine to b) the prone position during acute respiratory distress syndrome. The red circle represents the heart. White circles: normally aerated lung areas; grey circles: poorly aerated lung areas; black rectangle: non-aerated lung areas; green rectangle: consolidated lung areas.

VILI is subtended by biochemical and biological events implicated in the regulation of lung inflammation, a process termed biotrauma (**fig. 2**). PAPAZIAN *et al.* [29] found that the prone position was associated with lower lung concentration of interleukin (IL)-8, IL-6 and IL-1β compared to the supine position in ARDS patients ventilated with 6 $mL \cdot kg^{-1}$ predicted body weight V_T and higher PEEP. In rodents subjected to injurious mechanical ventilation (V_T 18 $mL \cdot kg^{-1}$ and PEEP 0 cmH_2O), prone ventilation has been shown to maintain the expression of mitogen-activated protein kinase (MAPK)-phosphatase 1, a pivotal regulator in VILI, while the supine position was associated with a significant downregulation [30]. Mice deficient in MAPK-phosphatase 1 were more susceptible to VILI [30].
So, there is large body of evidence supporting the fact that prone position ventilation has relevant beneficial physiological effects in ARDS. These benefits were progressively discovered together with major advances in mechanical ventilation. Whether these physiological benefits can translate into improvement in patient outcome will be discussed in the next section.

Limitations

Our trial has some limitations as the two groups were not completely balanced (by chance) at the time of randomisation. In particular, the Sequential Organ Function Assessment score was lower in the prone group. This was observed even though in order to be included the patients had to have a mean arterial blood pressure ≥65 mmHg. Even though this criterion was met by every patient in both groups, the rate of patients receiving vasopressors was greater in the supine group. However, in the multivariate analysis the effect of the prone position was still highly significant.

From the onset it was pointed out that the procedure of prone positioning exposes serious complications, in particular those related to airways, such as endotracheal tube displacement (main stem intubation or non-scheduled extubation), endotracheal tube obstruction or kinking, and vascular access kinking or removal. In our trial [32], as in others [31, 33], the rate of complications was significantly greater in the prone group compared to the supine group [4]. However, the mortality was not higher in the prone group. The same was true in the PSII RCT, in which the use of rotoprone was implicated in the observed higher rate of complications in the prone group. Therefore, the caregivers were very reluctant to expand the use of prone positioning in their intensive care unit given the lack of a clear significant benefit. The benefit/risk ratio was judged to not be in favour of extensive use of prone positioning. In the PROSEVA trial, for the first time, the rate of serious complications was similar between the two groups. This finding is probably the result of the expertise and skills of the centres involved in the trial that performed the procedure safely. Following the PROSEVA trial the benefit/risk ratio has greatly improved due to the significant increase in survival.
However, there is another side-effect of prone positioning that may cause some concern to the patients, their family and the caregivers, namely pressure ulcers. Previous trials and meta-analyses, also reported a greater incidence of pressure ulcers with the use of prone positioning. Furthermore, the facial location of pressure ulcers may have a psychological impact on the patients and their family. In the PROSEVA trial, as an ancillary study, the location and stage of pressure ulcers at time of inclusion, 7 days after inclusion and at intensive care unit discharge was prospectively reported in both groups.

Place of prone positioning in the management of ARDS patients

Three interventions have proven beneficial in ARDS: lower V_T [10], neuromuscular blocking agents (ACURASYS (ARDS etCurarisationSystématique) trial) [38], and prone positioning [2]. Lower V_T is the common strategy that must be applied to any ARDS patient regardless of the level of hypoxaemia [39]. Two recent meta-analyses have been published, which included PROSEVA in addition to the previous RCTs. Both found that prone positioning improves survival irrespective of the level of hypoxaemia provided a lower V_T is set [40, 41].
Neuromuscular blocking agents and prone positioning were investigated in ARDS patients with a P_{aO2}/F_{IO2} ratio <150 mmHg at PEEP ≥5 cmH_2O. An F_{IO2} of at least 0.60 and a 12–24-h stabilisation period were added to the inclusion criteria. Clearly, these two interventions are tightly linked and should be used together as first-line therapy in patients exhibiting the criteria mentioned above. The recent Berlin proposal split ARDS patients into mild, moderate and severe categories at 300, 200 and 100 mmHg P_{aO2}/F_{IO2} ratio thresholds, respectively. It is unclear

whether mortality does regularly increase from the mild to the severe ARDS category in the Berlin definition [42].

In conclusion, there is now a large body of evidence supporting the fact that prone positioning improves mortality in patients with severe ARDS. Accordingly, prone positioning should be used as a first-line therapy in this setting.

VALUE ADDED COURSE

MECHANICAL VENTILATION

Annexure II

STUDENT ENROLLMENT LIST (JAN-JUNE 2019)

S.No.	University no	Name of the student	Year / CRRI	Signature
1.	U17MB352	PRIYANKA KUMARI	II nd	
2.	U17MB353	PRIYANKA SINGH	II nd	
3.	U17MB354	RAAGAVI .S	II nd	
4.	U17MB355	RAHUL RAI	II nd	
5.	U17MB356	RICHI SWARN	II nd	
6.	U17MB357	RINI DAS	II nd	
7.	U17MB358	RISHABH SUMAN	II nd	
8.	U17MB359	RISHIKA	II nd	
9.	U17MB360	RISHIRAAJ KAR	II nd	
10.	U17MB361	RIYA M.A	II nd	
11.	U17MB362	ROFIQUL ISLAM	II nd	
12.	U17MB363	ROHAN DAS	II nd	
13.	U17MB364	SAKSHI SHARMA	II nd	
14.	U17MB365	SAMYUKTHA	II nd	
15.	U17MB366	SANORITA	II nd	
16.	U17MB367	SANTOSHKUMAR NK	II nd	
17.	U17MB368	SAPTARSHI CHATTOPADHYAY	II nd	
18.	U17MB369	SATHIYA JAINAUB T.S.	II nd	
19.	U17MB370	SHABAN OS	II nd	
20.	U17MB352	PRIYANKA KUMARI	II nd	

RESOURCE PERSON

DR. KALASREE

COORDINATOR

Dr S NITHIANANDAM

ANNEXURE III

MCQ: MECHANICAL VENTILATION

1. An artificial airway , like an endotracheal tube is used in the following type of ventilation
    a. Positive pressure
    b. Negative pressure
    c. Both A&B
    d. None of the above
2. Iron lung ventilators used after the polio epidemics in the first half of 20^{th} century belong to
    a. Positive pressure ventilation
    b. Negative pressure ventilation
    c. CPAP
    d. None of the above
3. One of the following is a risk of keeping high PEEP
    a. Hypotension
    b. Hypertension
    c. Hyperthermia
    d. Hypothermia
4. What are the ventilators parameters adjusted to maintain the optimum minute ventilation
    a. FiO2 & PEEP
    b. Tidal volume
    c. Respiratory rate
    d. Both B&C

5. Minute ventilation is equal to

a. FiO2 * PEEP

b. FiO2 \ PEEP

c. Tidal volume* Respiratory rate

d. Tidal volume\ Respiratory rate

6. One of the following modes of ventilation code out the patient efforts to breathe

a. Assist control mode

b. Pressure control mode

c. SIMV

d. CMV

7. CPAP & BIPAP modes are usually used

a. As a weaning protocol

b. To delay inhibition

c. In conjunction with bronchodilators & steroids

d. All the above

8. Pressure is applied to abdomen and thorax to draw air into lungs through upper airway in following type of ventilation

a. Positive pressure

b. Negative pressure

c. Both A&B

d. None of the above

9. Respiratory alkalosis is possible in

a.CMV

b. SIMV

c. Assist control mode

d. Pressure control mode

10. Ventilator parameters adjusted to maintain optimum oxygenation

a. FiO2

b. PEEP

c. Tidal volume

d. Both A &B

Priyanka singh.

7/10

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Shaban os.

8/10

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Annexure V
Student Feedback Form

Course Name: **MECHANICAL VENTILATION**
Subject Code: **ANAES 08**

Name of Student: _____ Roll No.: _____

We are constantly looking to improve our classes and deliver the best training to you. Your evaluations, comments and suggestions will help us to improve our performance

Sl. NO	Particulars	1	2	3	4	5
1	Objective of the course is clear					
2	Course contents met with your expectations					
3	Lecturer sequence was well planned					
4	Lectures were clear and easy to understand					
5	Teaching aids were effective					
6	Instructors encourage interaction and were helpful					
7	The level of the course					
8	Overall rating of the course	1	2	3	4	5

*** Rating: 5 – Outstanding; 4 - Excellent; 3 – Good; 2– Satisfactory; 1 - Not-Satisfactory**

Suggestions if any:

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Annexure V
Student Feedback Form

Course Name: **MECHANICAL VENTILATION**
Subject Code: **ANAES 08**

Name of Student: Raagavi - S Roll No.: U17MB354

We are constantly looking to improve our classes and deliver the best training to you.

Your evaluations, comments and suggestions will help us to improve our performance

Sl. NO	Particulars	1	2	3	4	5
1	Objective of the course is clear			✓		
2	Course contents met with your expectations				✓	
3	Lecturer sequence was well planned					✓
4	Lectures were clear and easy to understand				✓	
5	Teaching aids were effective					✓
6	Instructors encourage interaction and were helpful				✓	
7	The level of the course					✓
8	Overall rating of the course	1	2	3	4	5

*** Rating: 5 – Outstanding; 4 - Excellent; 3 – Good; 2– Satisfactory; 1 - Not-Satisfactory**

Suggestions if any:

Riya M.A

Annexure V
Student Feedback Form

Course Name: **MECHANICAL VENTILATION**
Subject Code: **ANAES 08**

Name of Student: Riya M.A Roll No.:
VMMB361

We are constantly looking to improve our classes and deliver the best training to you.

Your evaluations, comments and suggestions will help us to improve our performance

Sl. NO	Particulars	1	2	3	4	5
1	Objective of the course is clear			✓		
2	Course contents met with your expectations				✓	
3	Lecturer sequence was well planned					✓
4	Lectures were clear and easy to understand				✓	
5	Teaching aids were effective				✓	
6	Instructors encourage interaction and were helpful					✓
7	The level of the course					✓
8	Overall rating of the course	1	2	3	4	5

*** Rating: 5 – Outstanding; 4 - Excellent; 3 – Good; 2– Satisfactory; 1 - Not-Satisfactory**

Suggestions if any:

Satisfactory

Date: 05.12.2018

From
Dr M Kalasree
Head Of Department Incharge
Department of Anaesthesia
Sri Lakshmi Narayana Institute of Medical Sciences
Puducherry

To
The Dean,
Sri Lakshmi Narayana Institute of Medical Sciences
Puducherry

Sub: Completion of value-added course: MECHANICAL VENTILATION

Dear Sir,

With reference to the subject mentioned above, the department has conducted the value-added course titled: Mechanical Ventilation in Jan- June 2019 for 20 students. We solicit your kind action to send certificates for all the participants, whose name list is attached with this letter. Also, I am attaching the photographs captured during the conduct of the course.

Kind Regards,

Dr. M Kalasree

Encl: Certificates

Photographs

Sri Lakshmi Narayana Institute of Medical Sciences

Affiliated to Bharath Institute of Higher Education & Research

(Deemed to be University under section 3 of the UGC Act 1956)

CERTIFICATE OF MERIT

This is to certify that _SANORITA_____ has actively participated in the Value Added Course on Mechanical Ventilation held during January - June 2019 Organized by Sri Lakshmi Narayana Institute of Medical Sciences, Pondicherry- 605 502, India.

Dr.KALASREE M
RESOURCE PERSON

Dr. KALASREE M
COORDINATOR

Sri Lakshmi Narayana Institute of Medical Sciences

Affiliated to Bharath Institute of Higher Education & Research

(Deemed to be University under section 3 of the UGC Act 1956)

CERTIFICATE OF MERIT

This is to certify that __RIYA M A_____ has actively participated in the Value Added Course on Mechanical Ventilation held during January - June 2019 Organized by Sri Lakshmi Narayana Institute of Medical Sciences, Pondicherry- 605 502, India.

Dr.KALASREE M
RESOURCE PERSON

Dr.KALASREE M
COORDINATOR


MECHANICAL
VENTILATION