CARE OF PATIENT ON VENTILATOR ;

WHAT A NURSE SHOULD KNOW.

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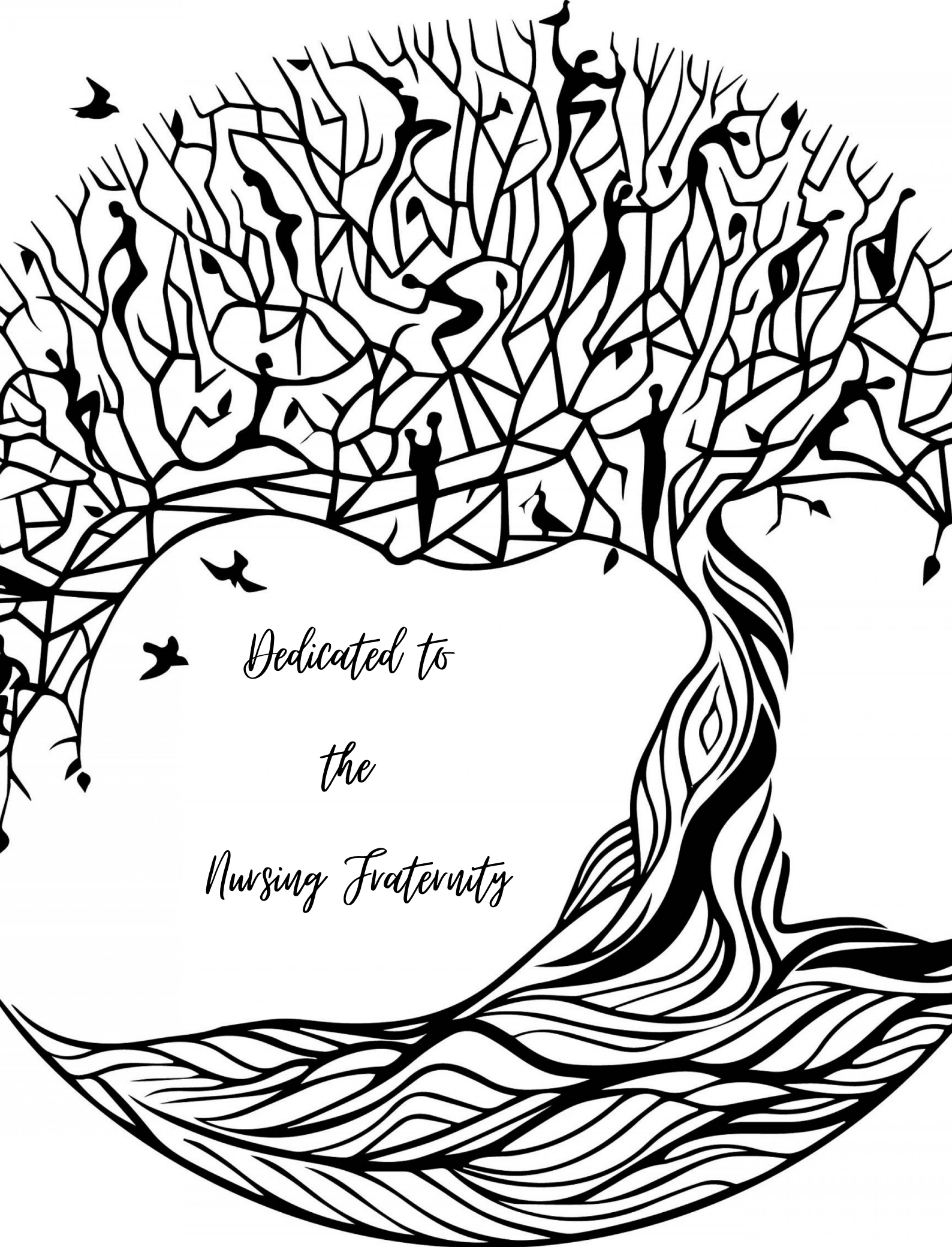
Dedicated to
the
Nursing Fraternity

PREFACE

Close involvement of the society is the destiny of Science. And Science is evolving with time constantly. Mechanical Ventilators has been bringing benefits to mankind since the first half of nineteenth century. And of late, the devastating Covid pandemic has brought in fore again the need of successful ventilatory management of critically ill patients.

This manual of 'Care of a Patient on Ventilator' for Nursing fraternity has been collated &written to tell the story of this field to the current and future generations. Every chapter abounds with enthusiasm and knowledge ofMr. Stanley Jones, Gopi Krishnan, Simi &Selvi; an amazing bunch of highly capable Nursing Officers and my respected and dear friends.They convey theintellectual fascination of intricacy of Ventilator management, its profundity, its breadth, and its importance as technology, and tell the story in an intriguing and visually appealing way. I believe this book will become a cornerstone of Nursing education.

Each chapter in this manual has been written with the demand of imparting practical knowledge in mind and to handhold towards furthering the skills of the nursing care givers.

The journey started with the history of the invention of the machine and basics of normal breathing and Ventilatory breaths. It slowly unfolded into different types of ventilatory modes and when and how to choose which mode and how to monitor and troubleshoot different alarms and potential problems which may arise during the course of mechanical Ventilation. I must specially mention about the chapter on different clinical scenarios which has been added very thoughtfully to specifically prepare the nursing community what to expect in those different day to day life threatening scenarios in Critical care ICU/ ITU and respond quickly and unflinchingly.

We are proud of our profession. And the nursing fraternity is the main pillar of the medical system. Southern railway HQ Hospital has remained in the forefront of Railway Medical Services towards dispensing advanced health care since 1970s with first CABG surgery of the country being performed in this very hospital. That rich tradition of clinical excellence is continuing over the generations. The department of Nursing at Perambur Railway Hospital have taken up the mantle now with this novel approach of writing this book on mechanical ventilation.I am not sure whether this is the first such book being published by Indian Railway Nursing fraternity, but even if it is not so, it should be one of the very first handbooks written by them. Kudos to the Perambur hospital Nursing fraternity for this academic inclination in furthering their knowledge and achieving such phenomenal feat. Wishing them all success and more such wonderful and impressive advancements in future and make life better for the patients undergoing therapy at Railway Hospitals.

Dr. Soumitra Sinha Roy

NOTE FROM AUTHORS

At the outset we thank God almighty for the grace, blessings and guidance throughout the successful completion of the book."

We are living in this age of COVID pandemic and the necessity to change gave birth of this book "Care of patient on ventilator; what a nurse should know". When the first and second waves of COVID were hitting us with its maximum brutality there was no other way but to equip ourselves with what we had and what we could afford in terms of manpower, equipment and knowledge. Time and luck were against us as we lost countless lives even though we tried our level best. Once the second wave came to its conclusion we were determined not to lose one more life because of our lack of knowledge or skill in taking care of patient on ventilator. We hope this book shall be of great help for those who share the same view of upgrading themselves even though there is no call.

As new information is available there is always a requirement of change. We have incorporated maximum information in this book even though in the view of possible human errors or advancement in the science' we advice readers discretion and wisdom when using and applying information given in the book.

We thank each and everyone who helped us creating this book.

Guided By

Dr.SOUMITRA SINHA ROY
M.D.,DTCD (Gold Medalist), MRCP (UK),
FCCP (USA),MRCP (UK) (Resp.Med.)
Fellow (Pulmonology),
Fellow (Interventional Pulmonology)
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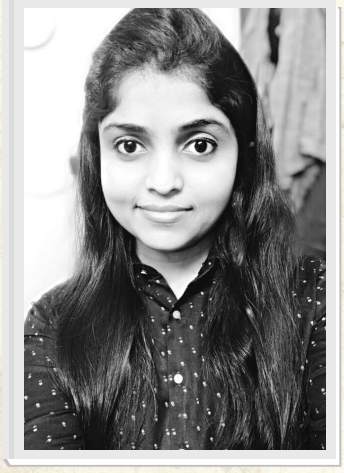

DR S.SHILMA
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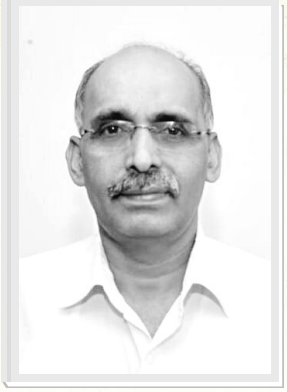
Message



I am very happy to know that the nursing department of Railway hospital is bringing out a manual on 'Care of a patient on ventilator' for nurses.

The Covid pandemic has been a trying time for all, especially for the health care personnel, who at times were overwhelmed with the number of seriously ill patients who required respiratory support. I understand that Patients with Covid can deteriorate very rapidly and this requires nursing personnel to act immediately to provide ventilator support to the patients. This book which is an excellent instructional guide to nurses as to how to handle these patients, will be useful not only to those who are new to ventilator technology, but, will also be a refresher guide to those experienced in managing patients on ventilators.

I appreciate the time and efforts put in by the nursing department to bring out such a detailed book in spite of their busy schedules.

I congratulate Mr.Stanley Jones and his team for their excellent effort.

John Thomas
General Manager
Southern Railway

27.09.2021

Message

I am very happy to hear that the nursing department of Railway hospital, Perambur is bringing out a manual on 'Care of a patient on ventilator' for nurses.

The ongoing Covid pandemic made us realize the importance of having adequately trained medical and para medical staff to handle patients who require respiratory support. The decision of the nursing department of Railway hospital, Perambur to come out with this book on care of patients requiring ventilator support is commendable and timely.

I had the privilege of going through the book. It is a ready reckoner for the nursing staff when they take care of patients on the ventilator, explaining the basics of the terminology used with easy to understand illustrations, the indications and types of ventilators available, detailed explanation of the various parts of the ventilator, diagrammatic representation of the procedure to ventilate, potential problems which may arise and how to trouble shoot. The authors have taken a lot of effort to share their knowledge so that patients on ventilators at Railway Hospital are under the care of competent care givers ensuring the safety of our patients.

I am sure this book will benefit not only nursing staff but even doctors not previously trained in ventilator use.

I would like to place on record my appreciation for the efforts taken by the nursing department of Railway hospital to put this book together and hope that they will update it at regular intervals with the feedback received on this book and with advances in ventilator technology. I hope that this effort will also encourage others to bring out similar books on other aspects of patient care so that the patients of the Railway family are the ultimate beneficiaries.

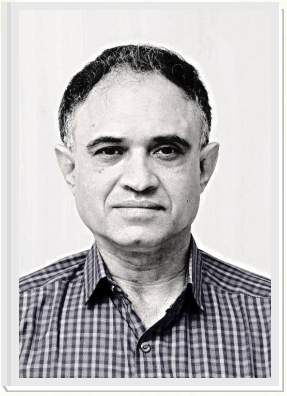
Bina John

Dr.Bina John

27.09.2021

President SRWHQO

Message



This manual on "Care of a patient on Ventilator — what a nurse should know" is a highly commendable effort by the team of outstanding Nurses from Railway Hospital, Perambur, who were part of team Medical SR that saw us through two difficult waves of the COVID Pandemic.

The manual is very appropriately timed and would be of tremendous use for training of Nurses in this very critical area of patient care particularly during the COVID Pandemic.

I congratulate Mr. Stanley Jones and his team for this outstanding piece of work.

B.G.Mallya
Additional General Manager
Southern Railway

27.09.2021

Message

A purposeful life is well lived in the service of our fellow beings, more so in the lives of medical professionals as it involves safeguarding the lives of others. The ongoing COVID-19 pandemic has imparted great pressure on critical care practices as increasing number of patients are in need of critical care all over the world. Identification of high-risk patients for clinical deterioration has been essential to ensure access to intensive treatment of severe conditions in a timely manner with emergency actions that demand decisiveness on the appropriatecourse of action Progress of COVID-19 in patients often lead respiratory distress requiring intubation and ventilation. Going by the adage that necessity is the mother of invention, I am glad to note that four of our nursing staff have found the time and energy to offer efficient and simpler ways of handling a ventilator. This book was written by our dedicated ICU staffs Shri.D.Stanly Jones, Shri.G.Gopi Krishnan, Smt.P.D.Simi and Smt.S.Selvi, who have relentlessly worked during the COVID-19 pandemic and saved many lives. This book covers a plethora of topics including, basic physiology of the lungs, ventilation and its accessories, maintenance of the ventilator, management of patients on ventilation, prevention of complications and weaning patients off ventilation. During this pandemic, a need for more ICU trained nurses arose; hence, such a book is written in simple language would help the paramedical staff handle patients on ventilation. This gains significance during the difficult times of the pandemic and also short supply of the machines. The thoughtful act of the staff is to be appreciated.

Dr.V.Nirmala Devi
P.C.M.D
Southern Railway

27.09.2021

Message

I take immense pleasure in writing a foreword for this book. This book has been scripted in a easily comprehensible and lucid language which would aid nursing staff in grasping cardinal concepts which are a prerequisite for catering to patients on ventilator. It is to be noted that knowledge in providing respiratory care by means of invasive and non-invasive ventilation is of paramount importance in management of COVID patients. The book gives good practical knowledge in managing patients which would help and guide the readers in providing quality intensive care.

I congratulate the sincere efforts taken by the team of authors Mr. Gopikrishnan G, Mrs. Simi P.D, Mrs. Selvi .S and Mr. D. Stanly Jones in coming up with this book which would benefit the paramedical staff in an unparalleled way.

Dr.C.V.N. MURTHY
Medical Director
Southern Railways Head Quarters Hospital
Perambur

27.09.2021

Message

It gives me immense pleasure to know about the skill and knowledge of my nursing fraternity in care of critically ill patients, by giving an extra mile to prepare a manual to educate the caregivers by their team work; able to brought this book on,

"CARE OF PATIENT ON VENTILATOR: WHAT A NURSE SHOULD KNOW".

This book has a great depth of knowledge, and it will be useful for not only the nurses but also other group of professional involved in patient care including doctors. My best wishes for the team who brought this manual and some more to come in future.

Dr.K.Muruganandam, MD(GM)., DNB(GM).
Additional Chief Health Director
HOD,Dept.of Medicine
Southern Railways Head Quarters Hospital
Perambur

27.09.2021

Message

I have gone through the manual 'Care of patient on ventilator; what a nurse should know', authored and compiled by COVID warriors team headed by Br. Stanley Jones. First, I would like to appreciate him for his persistent effort of compiling the manual in spite of this hard and hectic pandemic time. The overriding goal has been the mobilisation of information related to the science and skills of ventilator care. The format of the book is excellent and has particular emphasis on the common procedures and queries that a nursing staff encounters while caring a patient on ventilator set up. It affords the paramedics to assimilate right from the fundamentals in easy way. This manual will be an enormous help to those who are working in tertiary care setup especially in an intensive care unit where the bread and butter turns out to be ventilator care. I wish the manual a great success.

V Ganesh

Ms. Ganesh Kumari
Assistant Nursing Officer
Southern Railways Head Quarters Hospital
Perambur

29.09.2021

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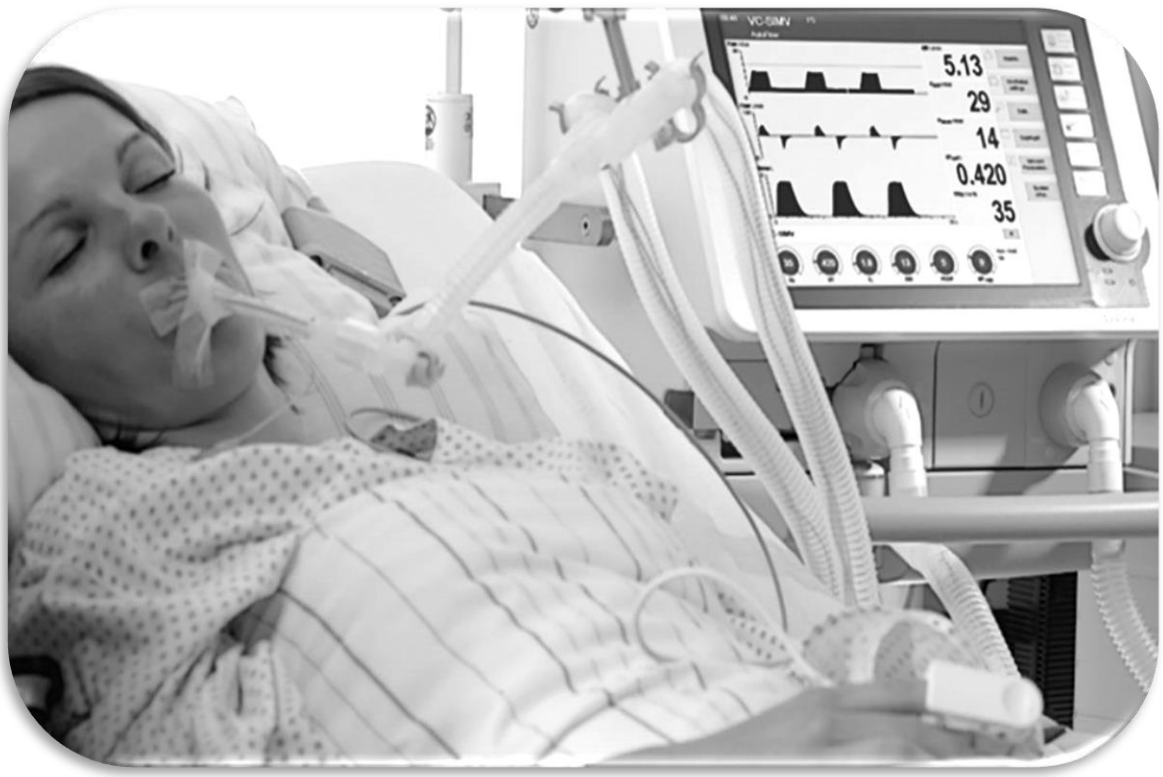
WHAT A NURSE SHOULD KNOW.



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Hi I am SATERN MAN*. My sole purpose is helping the nurses of Southern Railways to improve and update themselves in patient care. Every nurse's fear when it comes to patient care is intubation and mechanical ventilation. This book is meant to help the nurses in understanding the concept of mechanical ventilation, care of patient in ventilator & trouble shooting the ventilator. Author is trying here to compile information from various sources and explain each and every concept of ventilator in a simple and palatable way so that nurses can understand them easily.

NB: The name SATERN is adopted from the name of workshop series "Systematic Approach To Empower Registered Nurses of Southern Railway"

CHAPTER 1

MECHANICAL VENTILATION: BASIC REVIEW

VERY BASIC RESPIRATORY PHYSIOLOGY

What do the lungs do?

Yes, the simple answer is gas exchange:

Oxygenation?!!!

Oxygenation is the process of oxygen diffusing passively from the alveolus to the pulmonary capillary, where it binds to hemoglobin in red blood cells or dissolves into the plasma. Insufficient oxygenation is termed hypoxemia. This is to be differentiated from hypoxia, which is abnormally low oxygen content in a tissue or organ.

Ventilation

The mechanical process of exchange of air between the lungs and the atmosphere so that oxygen can be exchanged for carbon dioxide in the alveoli (the tiny air sacs in the lungs).

Always remember: Normal breathing is a negative pressure phenomenon; mechanical ventilation is POSITIVE pressure ventilation.

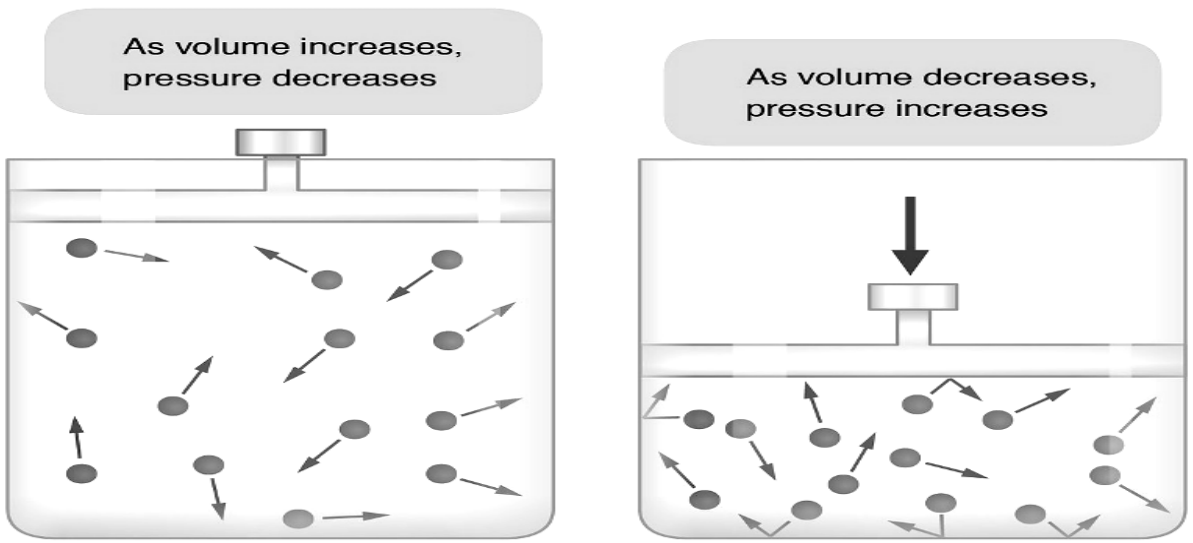
Mechanics of Breathing

The processes of inspiration (breathing in) and expiration (breathing out) are vital for providing oxygen to tissues and removing carbon dioxide from the body. Inspiration occurs via active contraction of muscles – such as the diaphragm and chest wall muscles causing negative intra thoracic pressure and negative intra Pleural pressure which drives in the air – whereas expiration tends to be passive, unless it is forced.

The Lungs and Breathing

The space between the outer surface of the lungs and inner thoracic wall is known as the pleural space. This is usually filled with pleural fluid, forming a seal which holds the lungs against the thoracic wall by the force of surface tension. This seal ensures that when the thoracic cavity expands or reduces, the lungs undergo expansion or reduction in size accordingly.

During breathing, the contraction and relaxation of muscles acts to change the volume of the thoracic cavity. As the thoracic cavity and lungs move together, this changes the volume of the lungs, in turn changing the pressure inside the lungs.



Boyle's law states that the volume of gas is inversely proportional to pressure (when temperature is constant). Therefore:

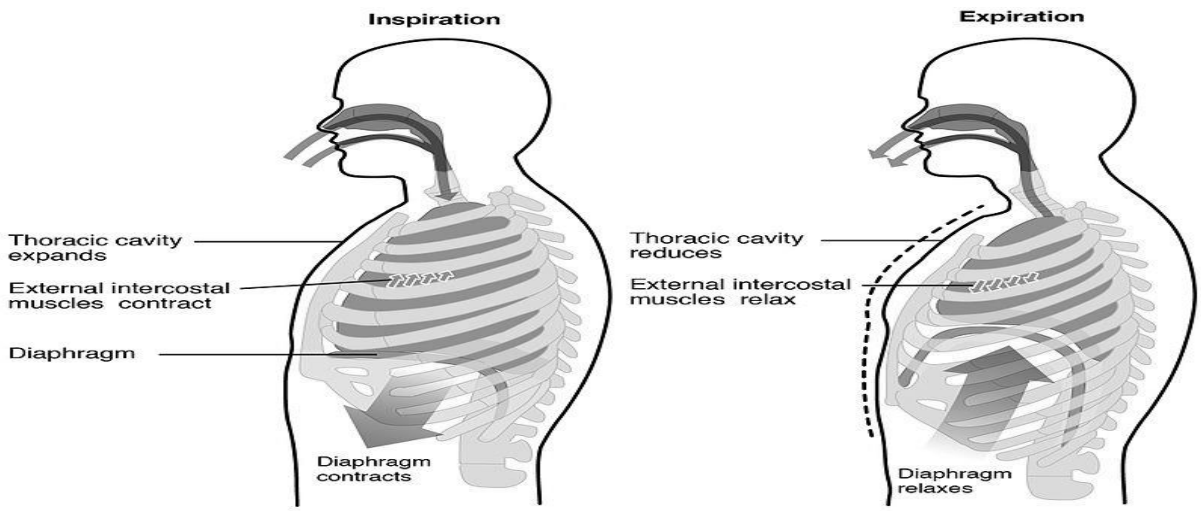
- When the volume of the thoracic cavity increases – the volume of the lungs increases and the pressure within the lungs decreases.
- When the volume of the thoracic cavity decreases – the volume of the lungs decreases and the pressure within the lungs increases.

Process of Inspiration

Inspiration is the phase of ventilation in which air enters the lungs. It is initiated by contraction of the inspiratory muscles:

- Diaphragm – flattens, extending the superior/inferior dimension of the thoracic cavity.
- External intercostal muscles – elevates the ribs and sternum, extending the anterior/posterior dimension of the thoracic cavity.

The action of the inspiratory muscles results in an increase in the volume of the thoracic cavity. As the lungs are held against the inner thoracic wall by the pleural seal, they also undergo an increase in volume.



As per Boyle's law, an increase in lung volume results in a decrease in the pressure within the lungs. The pressure of the environment external to the lungs is now greater than the environment within the lungs, meaning air moves into the lungs down the pressure gradient.

Process of Passive Expiration

Expiration is the phase of ventilation in which air is expelled from the lungs. It is initiated by relaxation of the inspiratory muscles:

- Diaphragm – relaxes to return to its resting position, reducing the superior/inferior dimension of the thoracic cavity.
- External intercostal muscles – relax to depress the ribs and sternum, reducing the anterior/posterior dimension of the thoracic cavity.

The relaxation of the inspiratory muscles results in a decrease in the volume of the thoracic cavity. The elastic recoil of the previously expanded lung tissue allows them to return to their original size.

As per Boyle's law, a decrease in lung volume results in an increase in the pressure within the lungs. The pressure inside the lungs is now greater than in the external environment, meaning air moves out of the lungs down the pressure gradient.

Forced Breathing

Forced breathing is an active mode of breathing which utilises additional muscles to rapidly expand and contract the thoracic cavity volume. It most commonly occurs during exercise.

Active Inspiration

Active inspiration involves the contraction of the accessory muscles of breathing (in addition to those of quiet inspiration, the diaphragm and external intercostals).

All of these muscles act to increase the volume of the thoracic cavity:

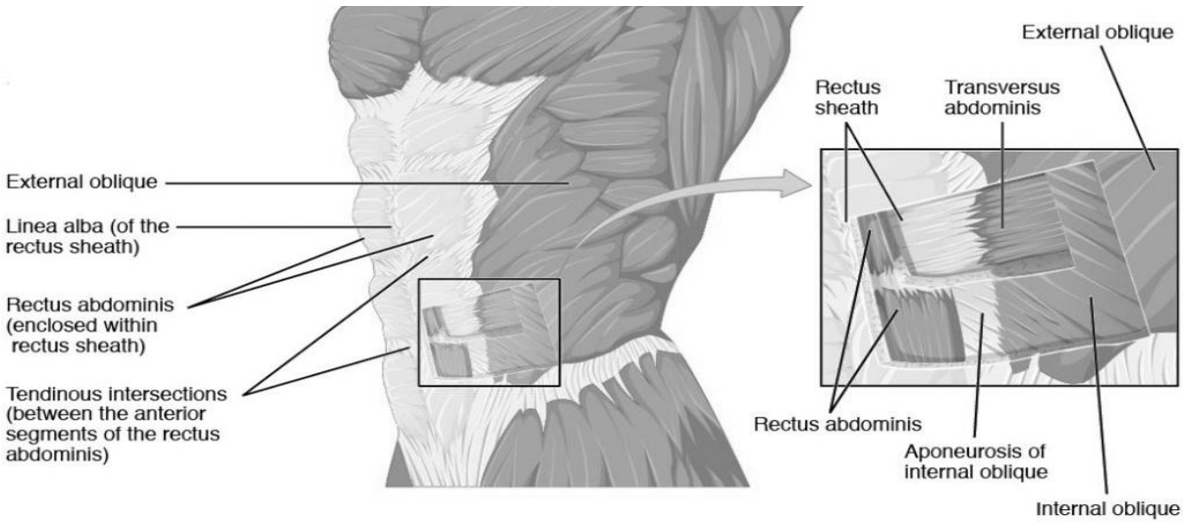
- **Scalenes** – elevates the upper ribs.
- **Sternocleidomastoid** – elevates the sternum.
- **Pectoralis major and minor** – pulls ribs outwards.
- **Serratus anterior** – elevates the ribs (when the scapulae are fixed).
- **Latissimus dorsi** – elevates the lower ribs.

Active Expiration

Active expiration utilises the contraction of several thoracic and abdominal muscles. These muscles act to decrease the volume of the thoracic cavity:

- **Anterolateral abdominal wall** – increases the intra-abdominal pressure, pushing the diaphragm further upwards into the thoracic cavity.
- **Internal intercostal** – depresses the ribs.
- **Innermost intercostal** – depresses the ribs.

The muscles of the antero lateral wall are utilised in forced expiration



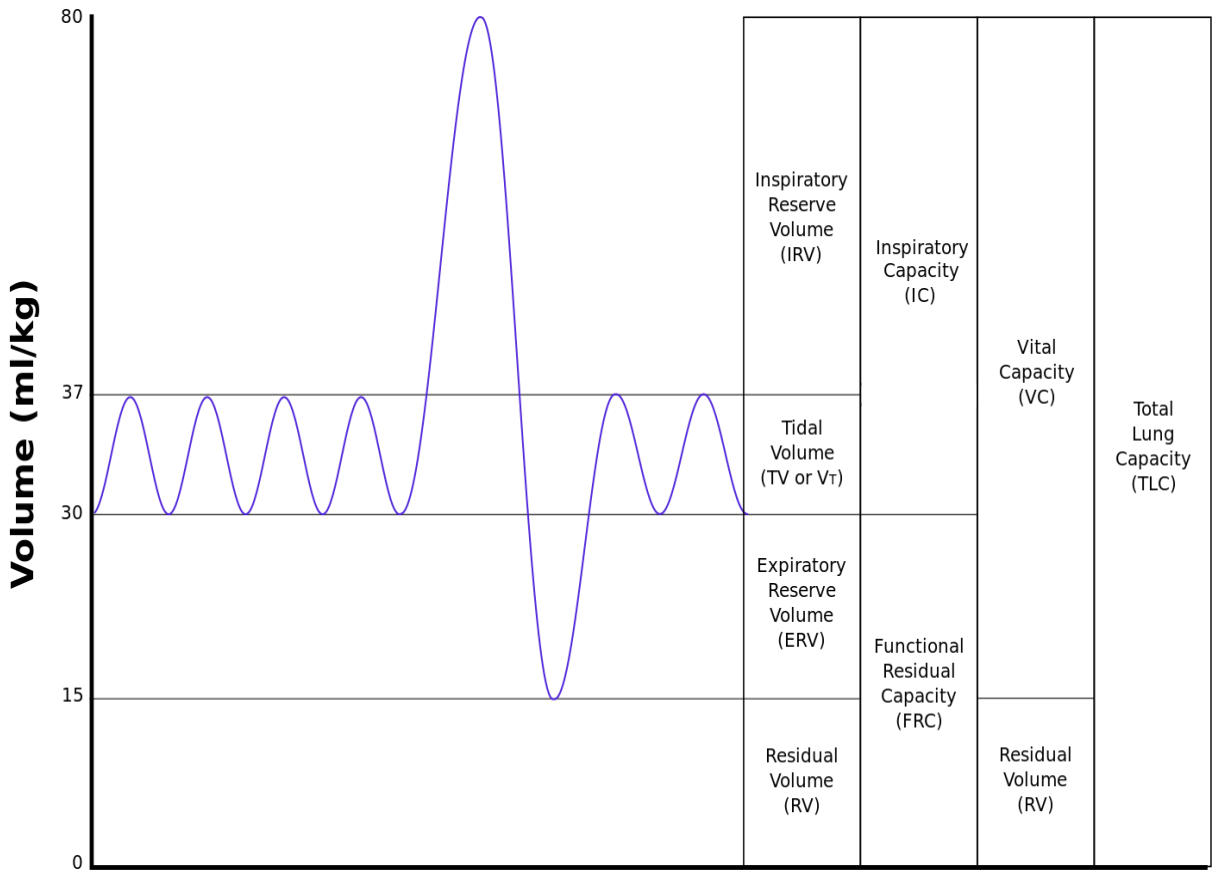
Some basic physiologic concepts to review

Air flow

The flow of air through the complete system i.e.: Machine, tubing, the Airways & lungs.

Volume

Lung volumes are also known as respiratory volumes. It refers to the volume of gas in the lungs at a given time during the respiratory cycle. Lung capacities are derived from a summation of different lung volumes. The average total lung capacity of an adult human male is about 6 litres of air.



Lung volumes and capacities

Volume	Value (litres)	
	In men	In women
Inspiratory reserve volume (IRV)	3.3	1.9
Tidal volume (TV)	0.5	0.5
Expiratory reserve volume (ERV)	1.1	0.7
Residual volume (RV)	1.2	1.1

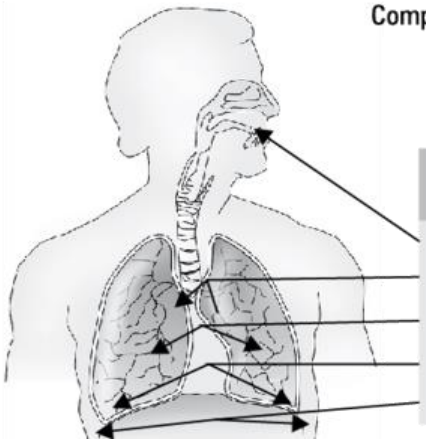
Capacities	Average value (litres)		Derivation
	In men	In women	
Vital capacity	4.8	3.1	IRV + TV + ERV
Inspiratory capacity	3.8	2.4	IRV + TV
Functional residual capacity	2.4	1.8	ERV + RV
Total lung capacity	6.0	4.2	IRV + TV + ERV + RV

Pressures

Peak Pressure (P peak): This is the total of pressure generated by the ventilator to overcome airway (ET tube and bronchus) resistance and alveolar resistance to attain peak inspiratory flow and to deliver desired tidal volume

Plateau pressure: is the pressure that is applied by the mechanical ventilator to the small airways and alveoli. The plateau pressure is measured at end-inspiration with an

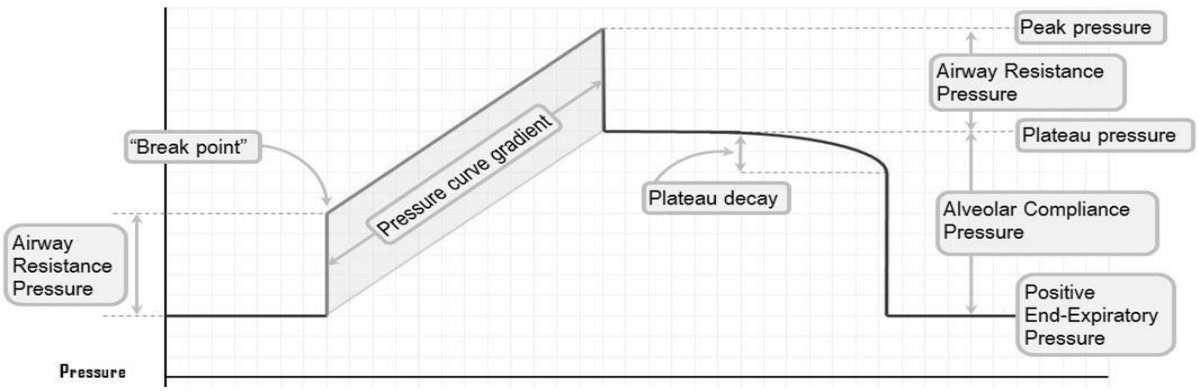
inspiratory hold maneuver on the mechanical ventilator that is **0.5 to 1 second**. Pplat less than 30 cm H_2O is recommended to avoid barotrauma.



Components of total airway resistance

Part of respiratory system	Percentage of total resistance (%)	Value of resistance ($cmH_2O/L/s$)
Oral cavity/pharynx	19	0,5
Central bronchi	19	0,5
Peripheral bronchi	8	0,2
Lung parenchyma	8	0,2
Thoracic wall	46	1,2

PEEP (Positive End Expiratory Pressure): At the end of mechanical or spontaneous exhalation, PEEP maintains the patient's airway pressure above the atmospheric level by exerting pressure that opposes passive emptying of the lung. **In simple language it is the pressure which helps the alveoli to keep open.** Low levels of PEEP (3 to 5 cmH_2O) are routinely used in patients on mechanical ventilation. This practice is important to: (I) keep lungs open at the end of expiration, thus promoting alveolar stabilization ; (II) prevent opening and closing of distal small airways and alveolar units ; and (III) increase lymphatic flow through the thoracic duct, which may facilitate drainage of lung edema . However, higher levels of PEEP may cause regional over distension and impairment of cardiac performance . The pros and cons of PEEP depend on the degree of lung injury.



Driving pressure

In patients without spontaneous breathing efforts (i.e., sedated and/or paralyzed on controlled mechanical ventilation), the driving pressure of the respiratory system is defined as the difference between plateau pressure and positive end-expiratory pressure (P_{plat}-PEEP), and can also be expressed as the ratio of tidal volume to respiratory system compliance (Vt/C_{rs}).

Since driving pressure is a way of representing the tidal volume adjusted for the respiratory system compliance, lower driving pressure may be associated with lower mortality due to a resultant reduction in cyclic lung inflation during mechanical ventilation. The tagret value of driving pressure is set to be 13-15 cm of H_2O.

Compliance

It is the change in volume occurs in an elastic structure due to a change in pressure. For example when you inflate a balloon by blowing air into it, it becomes bigger due to the change in volume by the pressure you delivered; likewise it is the change in the lungs by volume when pressure is exerted by the machine (ventilator).

Compliance is the ability of a hollow organ (vessel) to distend and increase volume with increasing transmural pressure or the tendency of a hollow organ to resist recoil toward its original dimensions on application of a distending or compressing force.

Elastance

It is the change occurs in pressure by delivering volume. If we take the previous example; when blowing in 1 liter of air to the balloon it inflates. But what will happen if the stiffness of the balloon increases?

Elastance is the pressure required to inflate the lungs. One half of this pressure is spent to inflate the lungs, and the other half is used to

inflate the chest wall in normal lungs. Normally, the elastance of the lungs and chest wall is similar. In the early stages of ARDS(Acute Respiratory Distress Syndrome), fluid from the smallest blood vessels in the lungs starts to leak into the alveoli- the tiny air sacs where oxygen exchange takes place. The lungs become smaller and stiffer and it becomes hard to breath.

Resistance

Resistance describes the opposition to a gas flow entering the respiratory system during inspiration, which is caused by frictional forces. Resistance is calculated as the ratio between the pressure driving a given flow and the resulting flow rate (V).

In a spontaneously breathing adult, normal airway resistance is estimated at **2 to 3 cm H2O/L/sec**. In the ventilated patient, resistance can be measured by dividing the [peak pressure minus the plateau pressure] by the flow rate in liters per second. In mechanically ventilated patients with a normal lung and an artificial airway, inspiratory resistance is 10–15 cmH2O/L/sec

Remember in ventilator;

Flow is volume divided by time.

Volume is flow multiplied by time.

Pressure is flow multiplied by resistance.

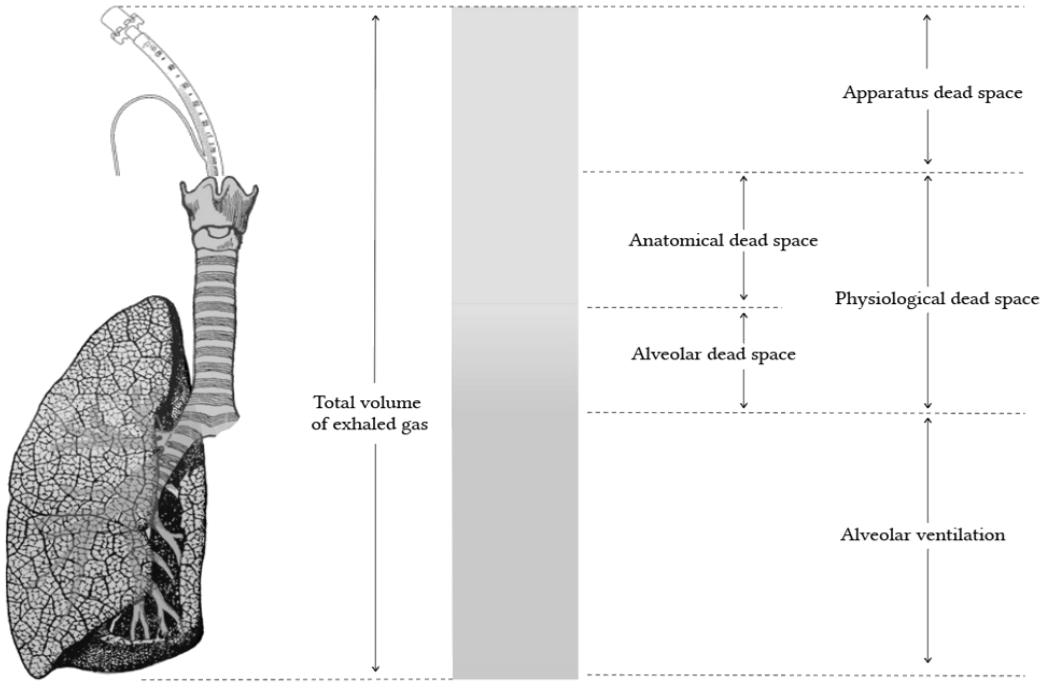
Resistance is the change in pressure divided by flow.

Compliance is volume divided by change in pressure.

Dead Space: the portion of each breath that does not participate in gas exchange.

Anatomic dead space: the volume of the conducting airways.

Physiologic dead space: also includes the contribution of alveoli that are well-ventilated but poorly perfused.



Why intubate and mechanically ventilate your patient?

1. Airway protection: If someone is intubated for "airway protection," as opposed to hypoxia or hypercapnia, the fact that the patient is not awake enough to maintain an open airway to promote gas exchange

2. Hypoxic respiratory failure

- Right-to-left shunt(A shunt is an abnormal communication between the right and left sides of the heart or between the systemic and pulmonary vessels, allowing blood to flow directly from one circulatory system to the other. A right-to-left shunt allows deoxygenated systemic venous blood to bypass the lungs and return to the body).

- Ventilation Perfusion (V/Q) mismatch.(It is a condition in which one or more areas of the lung receive oxygen but no blood flow, or they receive blood flow but no oxygen).

- pO2 < 50 on 100% NRB
- Decreased ambient FiO2
- Diffusion block
- Ventilation Perfusion (V/Q) mismatch.(It is a condition in which one or more areas of the lung receive oxygen but no blood flow, or they receive blood flow but no oxygen).

3. Hypercarbic respiratory failure pH < 7.30. pCO2 > 50. (Acute)

- Increased pCO2
- Acute hypercapnic respiratory failure develops **over minutes** to hours; therefore, pH is less than 7.3.
- Chronic respiratory failure develops over several days or longer, allowing time for renal compensation and an increase in bicarbonate concentration. Therefore, the pH usually is only slightly decreased.

A Change in pCO2 of 10 mm Hg

= a change in pH of 0.08

CHAPTER 2

THE MACHINE

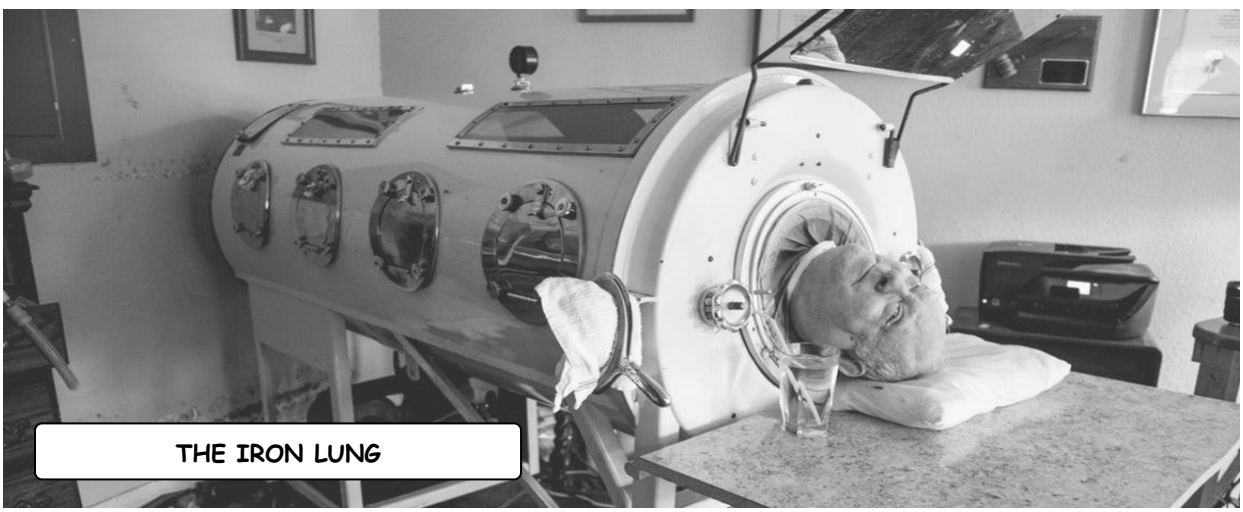
A ventilator is a machine that provides mechanical ventilation by moving breathable air into and out of the lungs, to deliver breaths to a patient who is physically unable to breathe, or breathing insufficiently.

Ventilators are computerized microprocessor-controlled machines but patients can also be ventilated with a simple, hand-operated bag valve mask. Ventilators are chiefly used in intensive-care medicine, home care, and emergency medicine (as standalone units) and in anesthesiology (as a component of an anesthesia machine).

Ventilators are sometimes called "respirators", a term commonly used for them in the 1950s (particularly the "Bird respirator"). However, contemporary hospital and medical terminology uses the word "respirator" to refer instead to a face-mask that protects wearers against hazardous airborne substances.

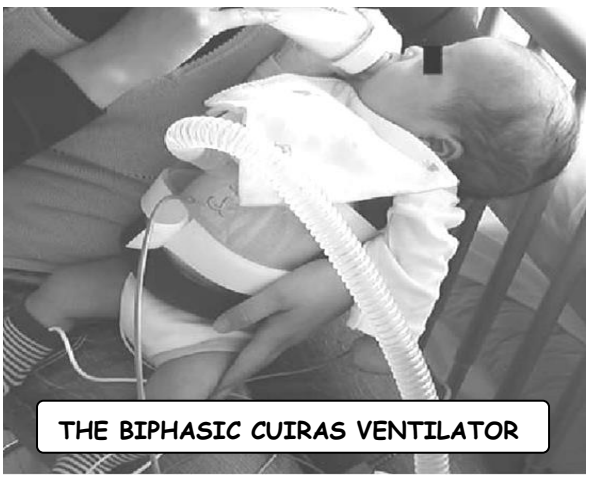
A BIT OF HISTORY…

The history of mechanical ventilation begins with various versions of what was eventually called the iron lung, a form of noninvasive negative-pressure ventilator widely used during the polio epidemics of the twentieth century after the introduction of the "Drinker respirator" in 1928, improvements introduced by John Haven Emerson in 1931, and

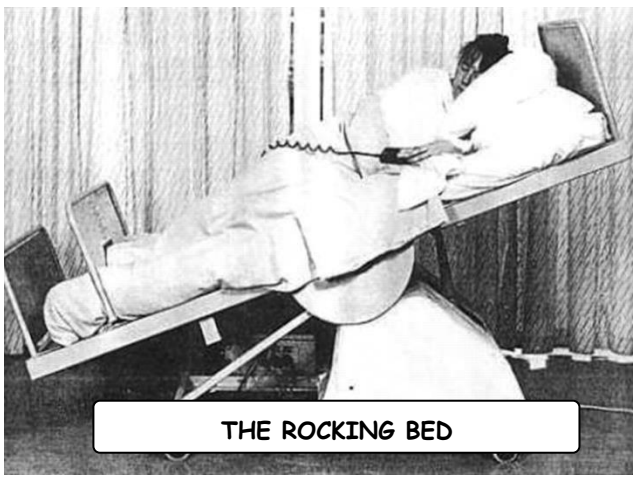


THE IRON LUNG

the Both respirator in 1937. Other forms of noninvasive ventilators, also used widely for polio patients, include Biphasic Cuirass Ventilation, the rocking bed, and rather primitive positive pressure machines.

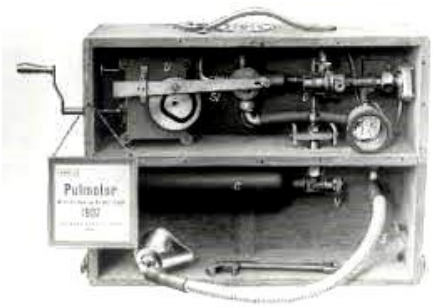


THE BIPHASIC CUIRAS VENTILATOR



THE ROCKING BED

The 1955 release of Forrest Bird's "Bird Universal Medical Respirator" in the United States changed the way mechanical ventilation was performed, with the small green box becoming a familiar piece of medical equipment. The unit was sold as the Bird Mark 7 Respirator and informally called the "Bird". It was a pneumatic device and therefore required no electrical power source to operate.

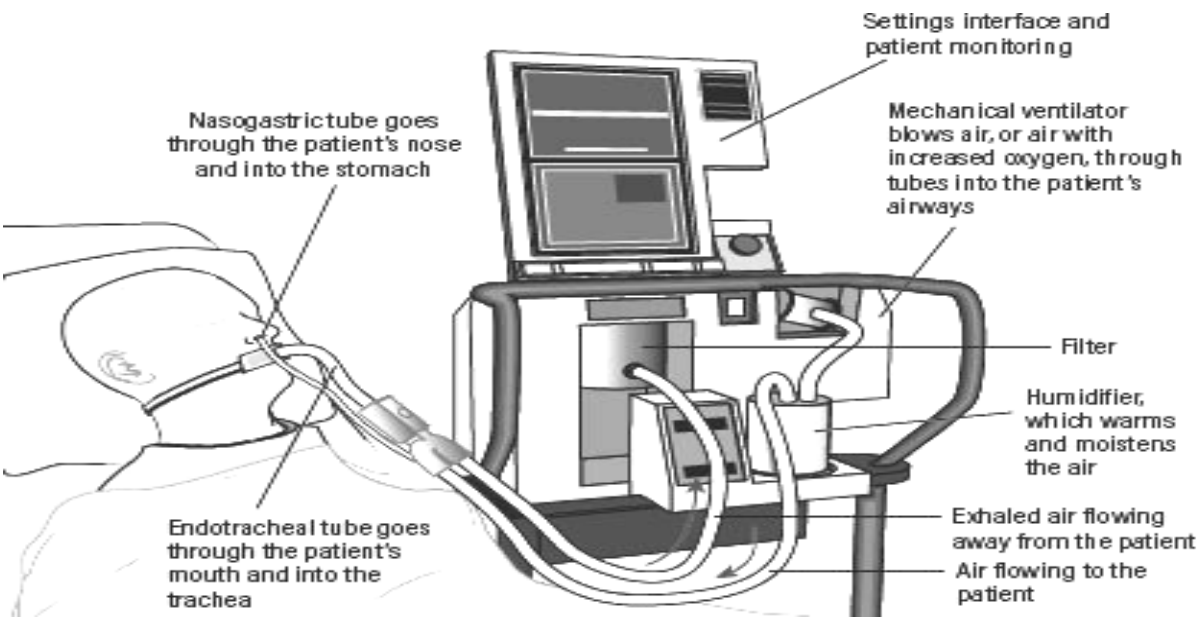


The Pulmotor, an early device for **POSITIVE PRESSURE VENTILATION**, was introduced in 1907 by German businessman and inventor Johann Heinrich Dräger and his son Bernhard. The Pulmotor was a transportable device that disbursed oxygen through ace mask until a set pressure was reached in the lungs, at which point it switched to exhalation. Another early 20th century device called a "rhythmic inflation apparatus" pumped air into a sealed box around a patient's head.

FOR THE EXTRA READERS

An open-source ventilator is a disaster-situation ventilator made using a freely-licensed design, and ideally, freely-available components and parts. Designs, components, and parts may be anywhere from completely reverse-engineered to completely new creations, components may be adaptations of various inexpensive existing products, and special hard-to-find and/or expensive parts may be 3D printed instead of sourced.On April 23, 2020, NASA reported building, in 37 days, a successful COVID-19 ventilator, named VITAL ("Ventilator Intervention Technology Accessible Locally"). On April 30, NASA reported receiving fast-track approval for emergency use by the United States Food and Drug Administration for the new ventilator.

PARTS OF MECHANICAL VENTILATOR



While you are taking care of a mechanically ventilated patient; the machine, tubing and patient together must be considered a single system. The following illustrations are based on a specific device (Carefusion-vela) only but it will help you to understand the concept.

Panels

Front panel

A. Effort indicator

B. Mode indicator

C. Screen indicator

D. Alarm status indicator

E. Power indicator

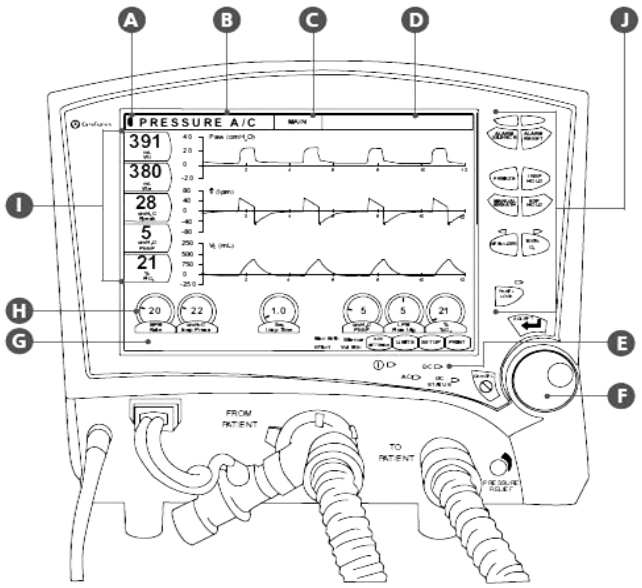
F. Data dial

G. Message window

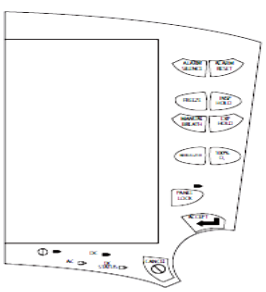
H. Primary controls

I. Monitored parameters

J. Membrane buttons



Membrane button functions



Alarm Silence: Pressing this button disables the audible portion of an alarm for 60 seconds (± 1 second) or until the ALARM SILENCE button is pressed again. This button is not functional for a VENT INOP alarm.

Alarm Reset: Cancels the visual indicator for alarms that are no longer active.

Freeze: The FREEZE button freezes the current screen and suspends real-time update of data until pressed again. When the screen is frozen, you can scroll through displayed waveforms, trends or loops using the Data Dial to move the cursor on screen.

Inspiratory Hold: When the INSP HOLD button is pressed and held, once the preset volume of a volume breath has been delivered, the patient is not allowed to exhale for a maximum of 6 seconds.

Expiratory Hold: When the EXP HOLD button is pressed and held, at the start of the next breath interval, the ventilator does not allow the patient to inspire or exhale for a maximum of 6 seconds.

Manual Breath: Pressing this button during the expiration phase of a breath delivers a single mandatory breath at current ventilator settings. No breath is delivered if the button is pressed during inspiration.

Synchronized Nebulizer: When an in-line nebulizer is attached and the NEBULIZER button is pressed, the ventilator supplies nebulized gas to the patient at 6 L/min. When nebulization is active, the nebulizer flow is synchronized with the inspiratory phase of each breath and can be adjusted in increments of 1 minute for a maximum of 60 minutes. You may end the nebulization period early by pushing the NEBULIZER button again.

Caution: Using the nebulizer may impact patient volumes. During volume control breaths, approximately 50 mL is added to the Tidal Volume for every 0.5 seconds of inspiratory time. If this added volume is undesirable for your patient, adjust the set Tidal Volume appropriately. This added volume also slightly increases the Peak Pressure. Properly set High-Pressure alarms help protect the patient from injury

100% O2: When this button is pressed, the ventilator increases the oxygen concentration delivered to the patient to 100% for 3 minutes. If the 100%O2 button is pressed again within the three-minute period, the maneuver is cancelled and the ventilator returns to the prior settings for FiO2.

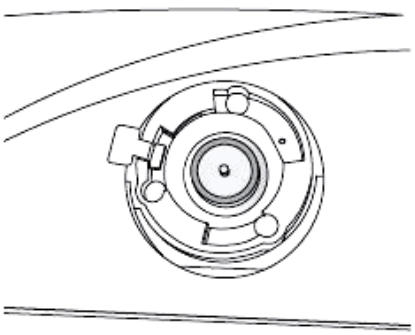
Panel Lock: The PANEL LOCK button disables all front panel controls except MANUAL BREATH, 100 %O2, ALARM RESET, ALARM SILENCE, and the PANEL LOCK button.

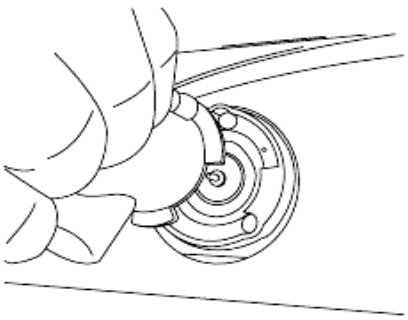
Accept: Accepts data entered into a field on the touch-screen.

Cancel: Cancels data entered into a field on the touch-screen. The ventilator continues to ventilate at current settings.

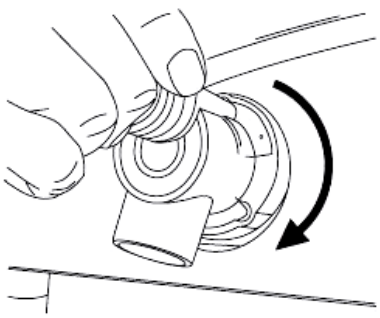
Circuit assembly

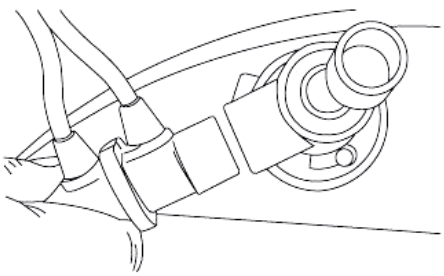
Carefully seat the rim of the diaphragm on the exhalation valve, and gently press around the rim to ensure it is seated evenly as shown. Line up the fins of the exhalation valve body with the openings in the exhalation valve housing.



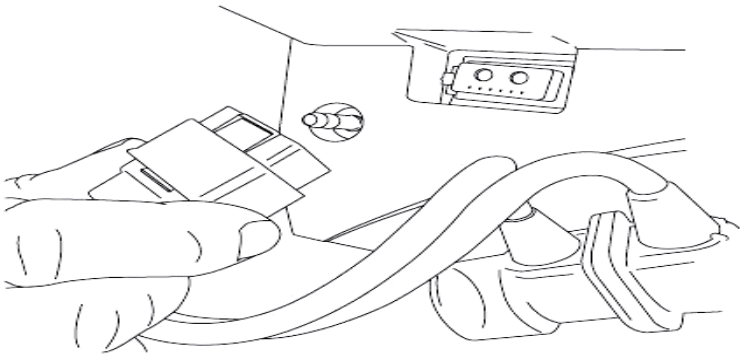


Rotate clockwise until it clicks into place. Attach the flow sensor with the flow tubes in an upright position.

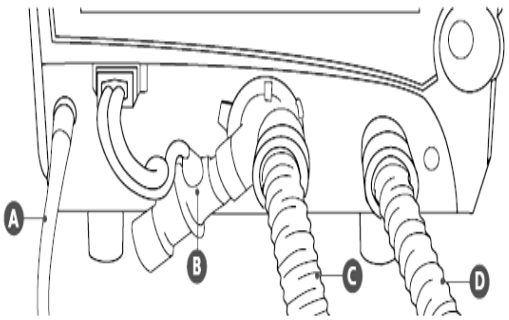




To connect the smart connector for the variable orifice flow sensor, pull back the locking sleeve. Push the locking sleeve into the receptacle, and slide it forward to secure. To remove, repeat these steps in reverse order.



Complete patient circuit assembly.



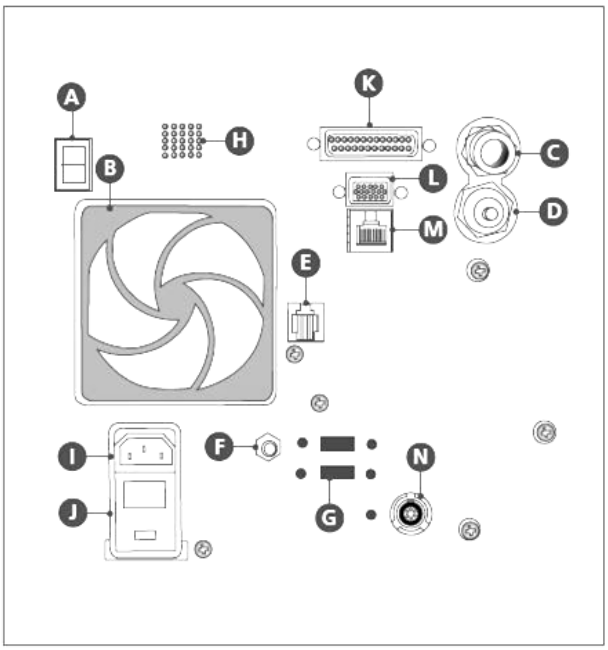
A. Nebulizer output

B. Expiratory flow sensor

C. Expiratory limb of patient-circuit.

D. Inspiratory limb of patient-circuit.

Rear panel



A. Power switch

B. Fan and fan filter

C. High-pressure oxygen fitting

D. Low-pressure oxygen fitting

E. Nurse call system connection

F. Ground terminal

G. Future options

H. Alarm speaker

I. Power cord connector

J. Fuses

K. Parallel printer port

L. Video output port

M. MIB port

N. CO2 power box connector

The primary controls

The primary breath controls are operator set controls, which directly affect the way a breath is delivered to your patient. They are displayed along the bottom of the touch-screen. Only the controls that are active in the currently selected mode of ventilation are displayed.

Displayed control	Description	Range
bpm Rate	Breath rate in breaths per minute	2 to 80 bpm
mL Vt	Tidal volume in milliliters	50 to 2,000 mL
cmH2O Insp Pres	Inspiratory pressure in centimeters of water pressure	1 to 100 cmH2O
L/min Peak Flow	Peak inspiratory flow in liters per minute	10 to 140 L/min
sec Insp Time	Inspiratory time in seconds	0.30 to 10 sec
sec Insp Pause	Sets an inspiratory pause, which is in effect for each volume breath delivered	Off, 0.1 to 2.0 sec
cmH2O PSV	Pressure Support in centimeters of water pressure	Off, 1 to 60 cmH2O

cmH2O PEEP	Positive end-expiratory pressure in centimeters of water pressure	0 to 35 cmH2O
L/min Flow Trig	Sets inspiratory flow-trigger point in liters per minute	1 to 10 L/min
fiO_2 %O2	Controls the percentage of oxygen in the delivered gas	21% to 100%

Ventilation types

Mechanical ventilation is termed "invasive" if it involves any instrument inside the trachea through the mouth, such as an endotracheal tube, or the skin, such as a tracheostomy tube. Face or nasal masks are used for non-invasive ventilation in appropriately selected conscious patients.

Non invasive ventilation (NIV)

Non-invasive ventilation (NIV) is the delivery of oxygen (ventilation support) via a face mask and therefore eliminating the need of an endotracheal airway. NIV achieves comparative physiological benefits to conventional mechanical ventilation by reducing the work of breathing and improving gas exchange.

Research suggests that noninvasive ventilation after early extubation looks helpful in reducing the total days spent on invasive mechanical ventilation. The intervention is recognized as an effective treatment for respiratory failure in chronic obstructive pulmonary disease, cardiogenic pulmonary oedema and other respiratory conditions without complications such as respiratory muscle weakness, upper airway trauma, ventilator-associated pneumonia, and sinusitis.

The non invasive positive pressure ventilation (NIPPV)

NIPPV describes the delivery of oxygen at either constant or variable pressures via a face mask, such as Bi-level Positive Airway Pressure (BiPAP) and Constant Positive Airway Pressure (CPAP)

CPAP

CPAP is the most basic level of support and provides constant fixed positive pressure throughout inspiration and expiration, causing the airways to remain open and reduce the work of breathing. This results in a higher degree of inspired oxygen than other oxygen masks. When indicated for home use it is usually via a low flow generator and is commonly used for patients requiring nocturnal CPAP for sleep apnoea.

High flow systems used in a hospital environment are designed to ensure that airflow rates delivered are greater than those generated by the distressed patient. As well as having an effect on respiratory function it can also assist cardiac function where patients have a low cardiac output with pre-existing low blood pressure. It is also commonly used for severe obstructive sleep apnoea and also for type 1 respiratory failure, for example, acute pulmonary oedema (by recruiting collapsed alveoli).

Indications

When a patient remains hypoxic despite medical intervention, Atelectasis - Complete or partial collapse of a lung or lobe, Rib fractures - to splint the rib cage open; to stabilize the fracture and prevent damage to the lung, Type I respiratory failure, Congestive Heart Failure, Cardiogenic pulmonary oedema, Obstructive sleep apnoea, Pneumonia: as an interim measure before invasive ventilation or as a ceiling of treatment.

Nasal CPAP is more commonly used with infants.

BiPAP

NIV is often described as BiPAP, however, BiPAP is actually the trade name. As the name suggests provides differing airway pressure depending on inspiration and expiration. The inspiratory positive airways pressure (iPAP) is higher than the expiratory positive airways pressure (ePAP). Therefore, ventilation is provided mainly by iPAP, whereas ePAP recruits underventilated or collapsed alveoli for gas exchange and allows for the removal of the exhaled gas. In the acute setting, NIV is used in type 2 respiratory failure (for example in a COPD exacerbation), with respiratory acidosis (pH < 7.35).

Indications

Type II respiratory failure, Acidotic exacerbation of chronic obstructive pulmonary disease (COPD), Increased work of breath causing ventilatory failure, for example, hypercapnia (increased CO2 in arterial blood gas), fatigue or neuromuscular disorder, Weaning from tracheal intubation

Contraindications of NIV

Coma, Undrained pneumothorax, Frank haemoptysis, Vomiting blood (haematemesis), Facial fractures, Cardiovascular system instability, Cardiac Arrest, Respiratory Failure, Raised ICP, Recent upper GI surgery, Active Tuberculosis, Lung abscess, No additional contraindications in the pediatric population.

Precautions :Emphysema - check chest x-ray for bullae, Patient compliance, Skin integrity and Airway obstruction.

Patients Unlikely to Do Well On NIV

Agitation, encephalopathic, uncooperative, Severe illness including extreme acidosis (pH <7.2),.Presence of excessive secretions or pneumonia, Multiple organ failure,

Haemodynamic instability, Inability to maintain a lip seal, Inability to protect the airway, Overt respiratory failure requiring immediate intubation.

Setting up the Equipment

Do not set up NIV unless you are familiar with the equipment, circuits, masks, etc.and are confident as to how safely to establish the patient on NIV and appropriately respond to blood gas results.

Introduce the treatment to the patient slowly.

Patients need to keep their mouth closed if using a nasal mask.

Some patients are less suited to NIV; however, each situation should be individually assessed.

NIV should generally be used in ICU or critical care environments.

Parts of the Machine

Bi-level positive airway pressure (BiPAP) generator

Anti-bacterial filter

Smoothbore tubing

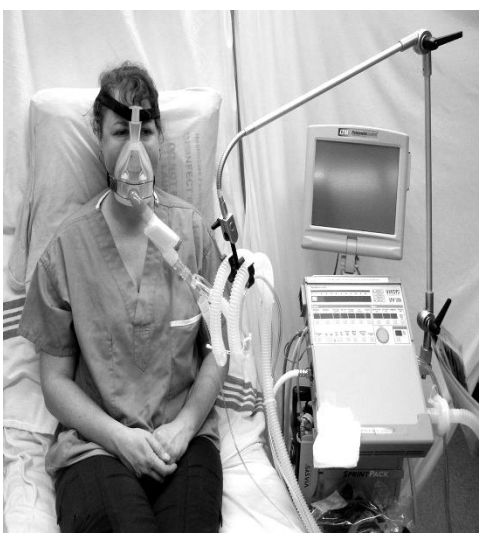
Exhalation port

Face mask, spacer and headgear

Oxygen tubing

Heated humidifier and tubing (if required)

Oximeter with an integral recorder



Instructions on Using the Ventilator

Introduce the patient slowly to the equipment and all its parts.

Ensure the mask fits comfortably and that the patient can experience the mask on their face without the ventilator.

Allow the patient the opportunity to feel the operation of the machine through the mask on their hands or cheek before applying it over their nose or mouth.

Allow the patient the opportunity to practice breathing with the ventilator, either by holding the mask in place or allowing them to hold it in place.

Adjust the settings initially for comfort and establish whether the patient can relax comfortably in a sleeping position.

Provide opportunities for the patient to feedback any discomfort.

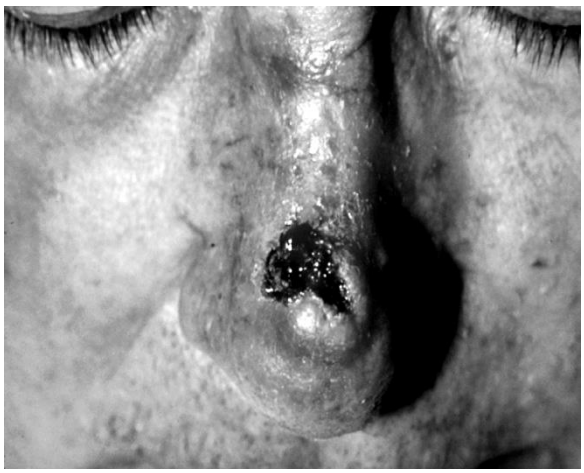
Assess and adjust the performance of the ventilator during an afternoon nap to optimise gas exchange and comfort.

Progress to an overnight study, continuing to monitor and optimise gas exchange and sleep quality.

Complications

Facial Pressure Ulcers Pressure ulcers associated with the use of NIV is a growing clinical problem due to the increased popularity of the intervention. The development of pressure ulcers is associated with poor clinical outcomes, increased complications, and length of hospital stay that compound with the consequences of acute illness. Medical devices such as NIV masks have unique risk factors including - the existence of a microclimate individual to the device, the method in which the device is secured, that devices may obscure the skin, and that the areas at risk are not routinely checked.. The

alternating airflow from bi-level positive pressure means that a seal is important to avoid ventilator asynchrony. Therefore strap tension is increased, with the risk of pressure damage a secondary consideration. It is important to consider that the patient may not be able to respond to an uncomfortable mask fit or excessive load delivered to vulnerable areas of skin due to sedation, medication, or neurological disease or injury. Furthermore, the patient may be too weak to reposition the device. Prophylactic interventions should also be considered.



Eye Irritation

It is important to ensure the mask is fitted correctly if it is not it can cause oxygen to leak upwards to the eyes, causing eye irritation and conjunctivitis.

Retention of Secretions

The use of a full face mask may interfere with the ability to cough and the effective clearance of secretions. As well as this, the positive pressure created may compromise the patient's ability to generate sufficient expiratory flow rates affecting the mobilization of secretions and also a resistance to cough leading to the retention of secretions.

The non invasive positive pressure ventilation in our setting includes NPPV SIMV, NPPV AC and NPPV CPAP/PSV.

NPPV Assist Control (A/C): Is delivered as a Pressure Control breath. Any patient trigger will receive a Pressure Control breath, and the breathing pattern is normally time-

cycled. NPPV AC is used with patients that require maximum support noninvasively. Every breath although patient-triggered is a controlled breath.

NPPV SIMV: Is a Pressure Control SIMV mode. The SIMV timed synchronized mandatory breaths are Pressure Control breaths, and the spontaneous breaths are either CPAP type breaths or, at the user's discretion, Pressure Support (PSV) Breaths. NPPV SIMV is used with patients that require mandatory support in addition to the patient's spontaneous rate.

NPPV CPAP/PSV: Consists of CPAP breathing at the user preset baseline pressure with the option of using Pressure Support (PSV) as an adjunctive adjustable pressure (decreases work of breathing) over and above the set CPAP level. NPPV CPAP/PSV is used for spontaneous breathing patients not requiring mandatory breaths from the ventilator.

Invasive ventilation

Invasive ventilation is positive pressure delivered to the patient's lungs via an endotracheal tube or a tracheostomy tube.

Breath types

We can categorize the breaths into the following types

- Spontaneous
- Supported
- Assisted
- Controlled/ Mandatory

All breaths are defined by three variables:

- Trigger (initiates the breath)
- Limit (terminates the breath)
- Cycle (how often is the breath delivered)

Types of breath and control	Trigger	Limit	Cycle
Spontaneous	Patient	Patient	Patient
Supported	Patient	Machine	Patient
Assisted	Patient	Machine	Machine
Controlled	Machine	Machine	Machine

Mandatory breath

Mandatory breaths can be triggered by the machine, the patient or the operator.

There are three mandatory breath types delivered by the VELA ventilator:

• Volume Breaths

• Pressure Breaths

• Pressure Regulated Volume Controlled (PRVC)

Volume Breaths: A Volume Breath is available in the Assist Control and SIMV mode. The ventilator delivers the set tidal volume with a constant flow during an inspiration time with a set inspiratory pause, according a set frequency.

If the compliance and/or the resistance of the respiratory system of the patient changes, the Peak Pressure can vary from breath to breath.

In **Volume A/C**, all breaths are mandatory and controlled by the ventilator per operator settings. The patient may initiate all breaths; however, when there is no patient effort, breaths are delivered at the set breath rate.

In **Volume SIMV**, both mandatory and demand breath types can be delivered. The operator sets a (SIMV) rate. When this rate is, for example, six breaths per minute, the ventilator will guarantee these six breaths (synchronized with patient effort). In between

these six breaths, the patient can breathe spontaneously. The spontaneous breaths can be supported with Pressure Support (PSV).

Pressure Breaths: A Pressure Breath is available in the Assist Control and SIMV mode. The ventilator delivers the set inspiratory pressure with variable flow until this pressure level is achieved. Flow is then regulated to maintain this pressure level for the duration of the set inspiratory time. The monitored Peak Pressure is equal to the sum of the **Inspiratory Pressure** + the **PEEP** settings. Pressure Breaths guarantee an inspiratory pressure; however, volumes may vary from breath to breath depending on the compliance of the lungs.

In **Pressure A/C**, all breaths are mandatory and controlled by the ventilator per operator settings. The patient may initiate all breaths; however, when there is no patient effort, breaths are delivered at the set breath rate.

In **SIMV PSV**, both mandatory and demand breath types can be delivered. The operator sets a (SIMV) rate. When this rate is, for example, six breaths per minute, the ventilator will guarantee these six breaths (synchronized with patient effort). In between these six breaths, the patient can breathe spontaneously. The spontaneous breaths can be supported with Pressure Support (PSV).

Pressure Regulated Volume Control (PRVC)

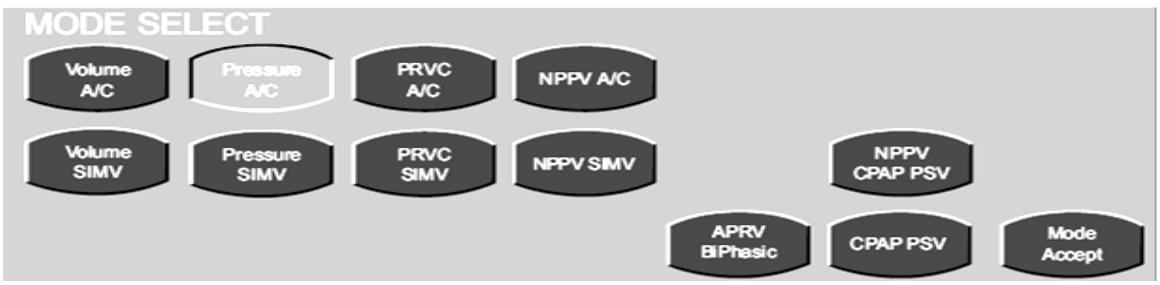
In Pressure Regulated Volume Control (PRVC) breaths, the pressure level is modulated up or down to achieve a preset tidal volume. When PRVC is selected, a volume controlled breath to the set tidal volume is delivered to the patient (test breath). Depending on the measured compliance, the ventilator sets the best possible inspiratory pressure to deliver that target volume during the first pressure controlled breath. The next breath and all subsequent breaths are delivered as pressure controlled breaths. Inspiratory pressure is adjusted automatically by the ventilator to maintain the target volume based on the dynamic compliance of the previous breath.

The patient triggered breaths are controlled by pressure and flow or time-cycled. They can be either Pressure Supported (PSV) or Spontaneous. All Patient triggered breaths are accompanied by the yellow patient demand indicator.

PSV (Pressure Support Ventilation): Pressure Supported (PSV) breaths are active when CPAP/PSV, SIMV or APRV/Biphasic is selected.

The patient-triggered breath will be supported with the set amount of PSV (+ PEEP).

Spontaneous: A Spontaneous breath is a demand breath where the pressure level during inspiration is preset at the PEEP level.



Different modes on the ventilator

Mode	Description
Volume A/C	Volume breath with Assist ventilation (default)
Pressure A/C	Pressure breath with Assist ventilation
Volume SIMV	Volume breath with Synchronized Intermittent Mandatory Ventilation (SIMV)
Pressure SIMV	Pressure Breath with SIMV
CPAP/PSV	Continuous Positive Airway Pressure (Demand Breath) with Pressure Support Ventilation (PSV)

APRV/Biphasic	Spontaneous demand breath at two alternating baseline pressure levels or controlled ventilation cycled by time
PRVC A/C	Pressure Regulated Volume Controlled breath with Assist Ventilation
PRVC SIMV	Pressure Regulated Volume Controlled breath with SIMV and an adjustable level of Pressure Support for spontaneous breaths.
NPPV A/C	Non-invasive Positive Pressure with Assist Ventilation
NPPV/SIMV	Non-invasive Positive Pressure with SIMV
NPPV/CPAP PSV	Non-invasive Positive Pressure and Continuous Positive Airway Pressure (Demand Breath) with Pressure Support Ventilation (PSV)

Apnea backup ventilation

The Apnea Mode choices appear when the APRV/Biphasic, CPAP/PSV or NPPV/CPAP PSV mode is selected. Apnea Backup is active in all SIMV and CPAP modes. In SIMV, the Apnea Backup breaths are delivered at the current ventilator breath settings (Volume or Pressure). Apnea Backup defaults to a breath rate of 12 unless a higher rate is set. The ventilator ceases Apnea Backup and resumes ventilation at the current settings once the patient initiates two breaths in a row or the Alarm Reset button is pushed.

CHAPTER 3

THE ACCESSORIES

There are certain tools which may come in handy when you take care of a patient in ventilator. Can you say a simple but most useful form of a ventilator? YES!!! AMBU™ (**Artificial Manual Breathing Unit)** it is. The **Bag Valve Mask (BVM)** ventilator was there and will be for a long time to save the patient in emergency. Let's discuss some of the aids the nurse might need in taking care of a ventilator patient.

Essential items needed for intubation

BVM (Bag Valve Mask Ventilator), Gloves, Lubricant jelly, Laryngoscope, Bougie and stylet, Oxygen source and Bain circuit, Suction tubing and apparatus, Endo tracheal tube, Oro pharyngeal airways, Syringes, Medications, Fixators, Scissors, Ventilator circuit, Filters, Et cuff manometer, Stethoscope, Crash cart and defibrillator.

Oxygen central out let

Oxygen cylinder with flow meter

BVM masks

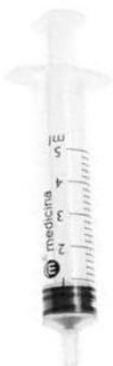
Bag Valve Mask ventilator

Bain circuit

Suction catheter

Yankauer catheter

Enteral syringe



IV syringe

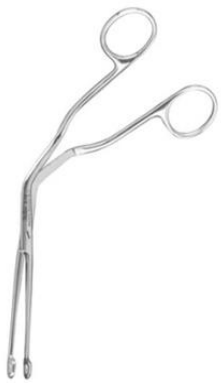
Lubricant jelly

Endotracheall tube

Laryngoscope with blades

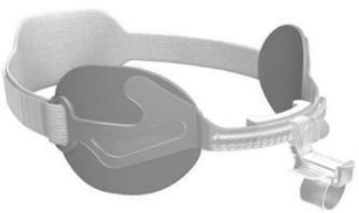
Bougie

Guedels airway



Magils forceps

Scissors



ET Tube holder

Fixator tapes

ET Cuff Manometer

Stethoscope

The Bag Valve Mask Apparatus.

Bag-Valve-Mask (BVM) apparatus are also known as manual resuscitators and as self-inflating resuscitation systems. Examples include **Laerdal™, Ambu™, Hsiner™, Mayo™, and Air Viva™**

Uses

- administration of high flow O2
- provision of PEEP (positive end-expiratory pressure)
- provision of controlled ventilation
- provision of augmentation of spontaneous ventilation

The BVM is a self-inflating resuscitation device. It is a bag made of plastic materials that re-expand after being manually collapsed. Various sizes e.g. Laerdal 240 mL, 500 mL, 1600 mL bag sizes for infants, children and adults

The parts of BVM are

- oxygen inlet nipple
- air intake valve
- oxygen reservoir with two one way valves
    - reservoir is at least the volume of the bag
    - oxygen flow rate equal to, or higher than, the minute volume of the patient allows 100% oxygen to be delivered
    - inlet valve allows room air to enter if fresh gas flow is inadequate and an outlet valve allow oxygen to flow out if pressure is excessive
- non-rebreathing valve that directs fresh flow of oxygen to the patient and prevents exhaled gas re-entering the bag
- standard 15 mm adapter for attaching to masks or tubes
- able to attach PEEP valve to exhalation port (either "built in" or detachable)

Have you ever used the pop off valve in BVM. You can hold down pop off valve (releases at about 60 cmH2O) to give increased pressure in the circuit

- Masks come in a range of sizes and designs
    - opaque or clear plastic
    - firm or air inflated cushion
    - mouldings vary but are designed to minimise dead space and fit
    - some have specific names (e.g. Rendall Baker Mask for paediatrics).

FOR THE EXTRA READERS

In BVM ventilation, a self-inflating bag (resuscitator bag) is attached to a nonrebreathing valve and then to a face mask that conforms to the soft tissues of the face. The opposite end of the bag is attached to an oxygen source (100% oxygen) and usually a reservoir bag. The mask is manually held tightly against the face, and squeezing the bag ventilates the patient through the nose and mouth. Unless contraindicated, airway adjuncts such as nasopharyngeal and/or oropharyngeal airways are used during BVM ventilation to assist in creating a patent airway. Positive end expiratory pressure (PEEP) valves should be used if further assistance is needed for oxygenation without contraindications to its use.

Successful BVM ventilation requires technical competence and depends on 4 things:

- A patent airway
- An adequate mask seal
- Proper ventilation technique
- PEEP valve as needed to improve oxygenation

Establishing a patent airway for BVM ventilation requires

- Keeping the oropharynx clear of physical obstructions (eg, secretions, vomitus, foreign bodies)
- Proper patient positioning and manual maneuvers to relieve tongue and soft tissue obstruction of the upper airway
- Airway adjuncts such as a nasopharyngeal or oropharyngeal airway to facilitate effective air exchange (see also Airway Establishment and Control)

Remember!!!
Rapid provision of successful ventilation and oxygenation is the goal.

Indications for BVM Ventilation

- Emergency ventilation for apnea, respiratory failure, or impending respiratory arrest
- Pre-ventilation and/or oxygenation or interim ventilation and/or oxygenation during efforts to achieve and maintain definitive artificial airways (eg, endotracheal intubation)

Contraindications to BVM Ventilation

Absolute contraindications:

- BVM ventilation is absolutely contraindicated in the presence of **complete upper-airway obstruction**, **Congenital diaphragmatic hernia** etc.
- A legal contraindication (do-not-resuscitate order or specific advance directive).

Relative contraindications:

- It is relatively contraindicated after paralysis and induction (because of the increased risk of aspiration)

Complications of BVM Ventilation

If bag-valve-mask ventilation is used for a prolonged period of time or if improperly performed, air may be introduced into the stomach. If this occurs and gastric distention is noted, a nasogastric tube should be inserted to evacuate the accumulated air in the stomach.

Equipment for BVM Ventilation

- Gloves, mask, gown, and eye protection (ie, universal precautions)
- Oropharygeal airways, nasopharygeal airways, lubricating ointment
- Bag-valve apparatus
- PEEP valve
- Variably sized ventilation face masks
- Oxygen source (100% oxygen, 15 L/minute)
- Nasogastric tube

- Suctioning apparatus and catheter; Magill forceps (if needed to remove easily accessible foreign bodies and patient has no gag reflex) to clear the pharynx as needed
- Pulse oximeter
- Capnography equipment if available.

How do you check if BVM is working properly??

There are three quick checks to make sure an BVM is working properly:

1. Squeeze to make sure the BVM self inflates, which indicates the valves are working correctly.
2. Block the patient connection (where the device would enter the patient's mouth) and attempt to squeeze the bag. The pop-off valve should open.
3. Remove the reservoir and block the patient intake valve. The bag should not inflate.

Additional Considerations for BVM Ventilation

- Two-person bag-valve-mask (BVM) ventilation is used whenever possible. Bag-valve-mask ventilation can be done with one person or two, but two-person BVM ventilation is easier and more effective because a tight seal must be achieved and this usually requires two hands on the mask.
- Unless contraindicated, a pharyngeal airway adjunct is used when performing BVM ventilation. An oropharyngeal airway is used unless the patient has an intact gag reflex; in such cases, a nasopharyngeal airway (nasal trumpet) is used. Bilateral nasopharyngeal airways and an oropharyngeal airway are used if necessary for ventilation.
- Among the many factors that can make achieving an air-tight seal difficult are facial deformity (traumatic or natural), a thick beard, obesity, poor dentition,

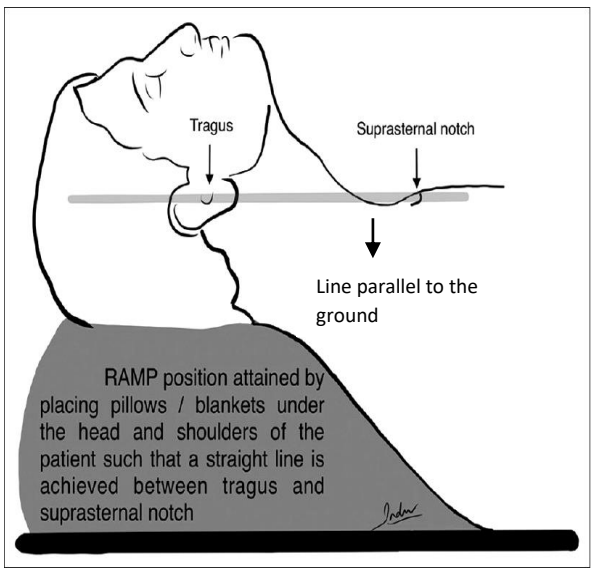
trismus, and cervical pathology. In such situations, BVM is attempted, but if it is unsuccessful, a supraglottic airway is placed (unless contraindicated).

- A positive end expiratory pressure (PEEP) valve may be used during BVM to improve oxygenation. PEEP can increase alveolar recruitment and thus oxygenation if oxygenation is compromised even with 100% oxygen due to atelectasis. PEEP has also been shown to prevent lung injury. However, PEEP should be used cautiously in patients who are hypotensive or pre-load dependent because it reduces venous return.

Positioning for BVM Ventilation

The sniffing position—only in the absence of cervical spine injury

Position the patient supine on the stretcher.



Align the upper airway for optimal air passage by placing the patient into a proper sniffing position. Proper sniffing position aligns the external auditory canal with the sternal notch. To achieve the sniffing position, folded towels or other materials may need to be placed under the head, neck, or shoulders, so that the neck is flexed on the body and the head is extended on the neck. In obese patients, many folded towels or a commercial ramp device may be needed to sufficiently elevate the shoulders and neck. In children, padding is usually needed behind the shoulders to accommodate the enlarged occiput.

Head and neck positioning to open the airway: Sniffing position

If there is concern for cervical spine injury:

- Position the patient supine or at a slight incline on the stretcher.
- Position yourself at the head of the stretcher.

- Avoid moving the neck and, if possible, use only the jaw-thrust maneuver or chin lift without head tilt to manually facilitate opening of the upper airway.

Aligning the external auditory canal with the sternal notch may help open the upper airway to maximize air exchange and establishes the best position to view the airway if endotracheal intubation becomes necessary.

The degree of head elevation that best aligns the ear and sternal notch varies (eg, none in children with large occiputs, a large degree in obese patients).

Step-by-Step Description of Procedure

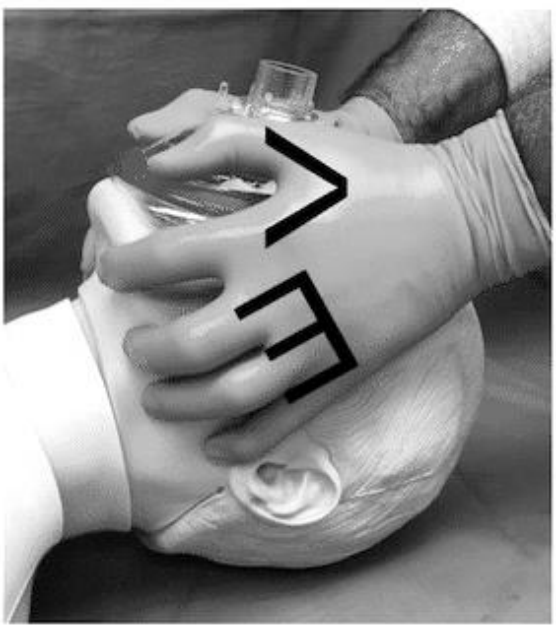
- Insert an oropharyngeal airway (unless the patient has a gag reflex) or one to two nasopharyngeal airways prior to bag-valve-mask (BVM) ventilation.
- Select a mask that fits over the mouth and nose but spares the eyes.
- Do two-person BVM ventilation if possible. (NOTE: The accompanying video presents the one-person technique first.)

Two-person mask technique

- In the two-person technique, the more experienced operator handles the mask, because maintaining a proper mask seal is the most difficult task. The second operator squeezes the bag.
- Stand at the head of the stretcher and have the second operator stand to the side.
- Using both hands, hold the mask between your thumbs and index fingers placed on either side of the connector stem.
- Making sure not to place your hands or the mask on the patient's eyes, first place the nasal portion of the mask over the nose high enough to cover the bridge

without air leaks. Next, lower the mask over the chin and allow it to seal along the two malar eminences. Cover the bridge of the nose, the two malar eminences, and the patient's lower lip by the mask to achieve a proper seal. Stretching the internal portion of the mask before placing it over the nose and mouth can help create a tighter seal.

- Traditional hand placement is the **"C-E" grip**, placing the middle, ring, and little fingers (the "E") under the mandible and pulling the mandible upward, while the thumbs and index fingers create a "C" and then press down against the mask. There is V-E grip also if you are using two hands.

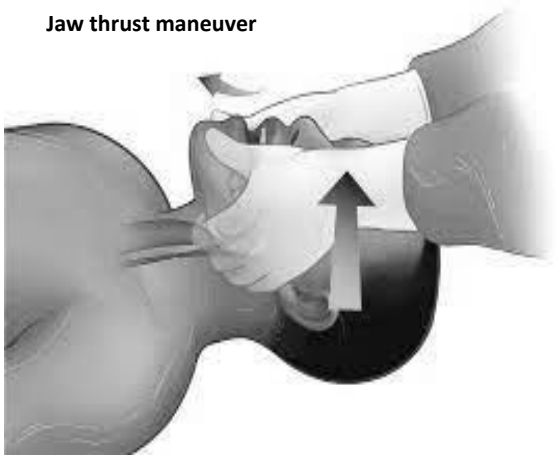


- An alternative, often preferred, method (1, 2) can be used in which the thenar eminences (muscles at the base of the thumb) hold the mask to the face. Place the thenar eminences (the base of the thumbs in the palm) along each lateral edge of the mask. Then lower the mask onto the face and place the other 4 fingers under the mandible. Press the mask to the face with the thenar eminences while pulling the mandible upward with the fingers. Head tilt may be applied concurrently. This technique is easier to perform, allows the use of stronger hand muscles to maintain

a proper seal, minimizing fatigue, and enables 4 fingers rather than 3 to lift the mandible (accomplishing chin lift and **jaw thrust**).

- If using the traditional hand placement, provide a head tilt–chin lift maneuver by pulling up on the mask and patient's face with your middle, ring, and little fingers while holding the mask onto the patient's face, to further open the airway. If your hands are large enough, place your little fingers behind the mandibular rami to do a jaw-thrust maneuver . This re-positioning helps to direct air into the trachea rather than into the esophagus and prevents gastric distention.

Jaw thrust maneuver



- Be sure to pull up only on the bony parts of the mandible, because pressure to the soft tissues of the neck or under the chin may obstruct the airway.
- Once a proper seal is achieved, have the second operator attach the bag to the mask and begin ventilation.

One-person mask technique

- Using one hand, hold the mask, with your thumb and index finger wrapped around the connector stem of the mask. Most operators use their non-dominant hand to grasp the mask, but either hand can be used as long as a good mask seal can be maintained.
- Making sure not to place your hand or the mask on the patient's eyes, first place the nasal portion of the mask over the nose, and then lower the body over the patient's mouth. The bridge of the nose, the two malar eminences, and the mandibular alveolar ridge must be covered by the mask in order to achieve a proper seal.

- Now extend your middle, ring, and little fingers underneath the patient's mandible, and pull it upward into the mask. This maneuver is similar to that of the head tilt–chin lift technique and further opens the airway.
- While maintaining this upward traction on the mandible, press the mask downward onto the face to attain a tight mask seal. If your hand is large enough, place your little finger behind the mandibular ramus to do a jaw-thrust maneuver to further open the airway.
- Be sure to pull up only on the bony parts of the mandible, because pressure to the soft tissues of the neck or under the chin may obstruct the airway.
- Once a proper seal is achieved, use your other hand to begin ventilation.

Bag ventilation and oxygenation

- For each breath, steadily and smoothly squeeze the bag, to deliver a tidal volume of 6 to 7 mL/kg (or about 500 mL for an average size adult) over 1 second, and then release the bag to allow it to reinflate. If using a 1000-mL volume bag, squeeze only halfway to obtain the correct tidal volume.
- In cardiac arrest cases, do not exceed 8 to 10 breaths per minute (ie, one complete breath every 6 to 7.5 seconds).
- Observe for proper chest rise during ventilations; in practice, you can use a tidal volume just large enough to cause the chest to rise.
- Monitor the patient, checking breath sounds and, if possible, end-tidal carbon dioxide and pulse oximeter. (Pulse oximetry may not be useful during cardiac arrest due to poor peripheral perfusion.) Assess if adequate ventilation is continuous and sustainable or is requiring too much physical effort. If available, use waveform capnography, an excellent indicator of mask seal and proper ventilation.
- If oxygenation is inadequate despite proper form and use of 100% oxygen, attach a positive end expiratory pressure (PEEP) valve to recruit more alveoli for gas exchange. Set the PEEP valve initially at 5 and increase as needed to improve

oxygen saturation. However, avoid PEEP in hypotensive patients,(PEEP is permitted if essential with vasopressors and fluid support).

- If ventilation or oxygenation is still not adequate, prepare for other airway maneuvers such as a supraglottic airway or endotracheal intubation.

Aftercare for Bag-Valve-Mask Ventilation

- Continue bag-valve-mask (BVM) ventilation until either a definitive artificial airway (eg, endotracheal tube) is achieved or spontaneous ventilation is adequate (eg, following naloxone administration for an opioid overdose).
- If a patient becomes more conscious or a gag reflex returns while doing BVM ventilation with an oropharyngeal airway in place, remove the oropharyngeal airway and provide continued treatment as appropriate. A nasopharyngeal airway may be better tolerated.
- If endotracheal intubation is necessary, ventilate using maximum FiO2 through a non-rebreather mask for 3 to 5 minutes before inserting the tube if feasible; if this is not feasible because intubation must proceed immediately, pre-oxygenate the patient by giving 5 to 8 vital capacity breaths using a PEEP valve.

Warnings and Common Errors for BVM Ventilation

- Do not place your hands or the mask on the patient's eyes. Doing so may damage the eyes or cause a vagal reaction.

Tips and Tricks for BVM Ventilation

Neither excessive force nor rapid insufflation should be used to ventilate; doing so increases gastric distention, compromising ventilation. **A nasogastric tube** is inserted to help decompress the stomach when possible.

How do you clean a BVM after use?

If a BVM is of the reusable type, it must be disassembled and thoroughly cleaned, first of any "gross soiling" through meticulous hand-rinsing, washing, and re-rinsing. Some BVMs can be sterilized in an autoclave at 134° C or 272° F.

Otherwise, it must be sterilized through the use of the recommended disinfectant solution. Glutaraldehyde 2% is an appropriately high-level disinfectant; the bag must be immersed in the solution for at least 20 minutes.

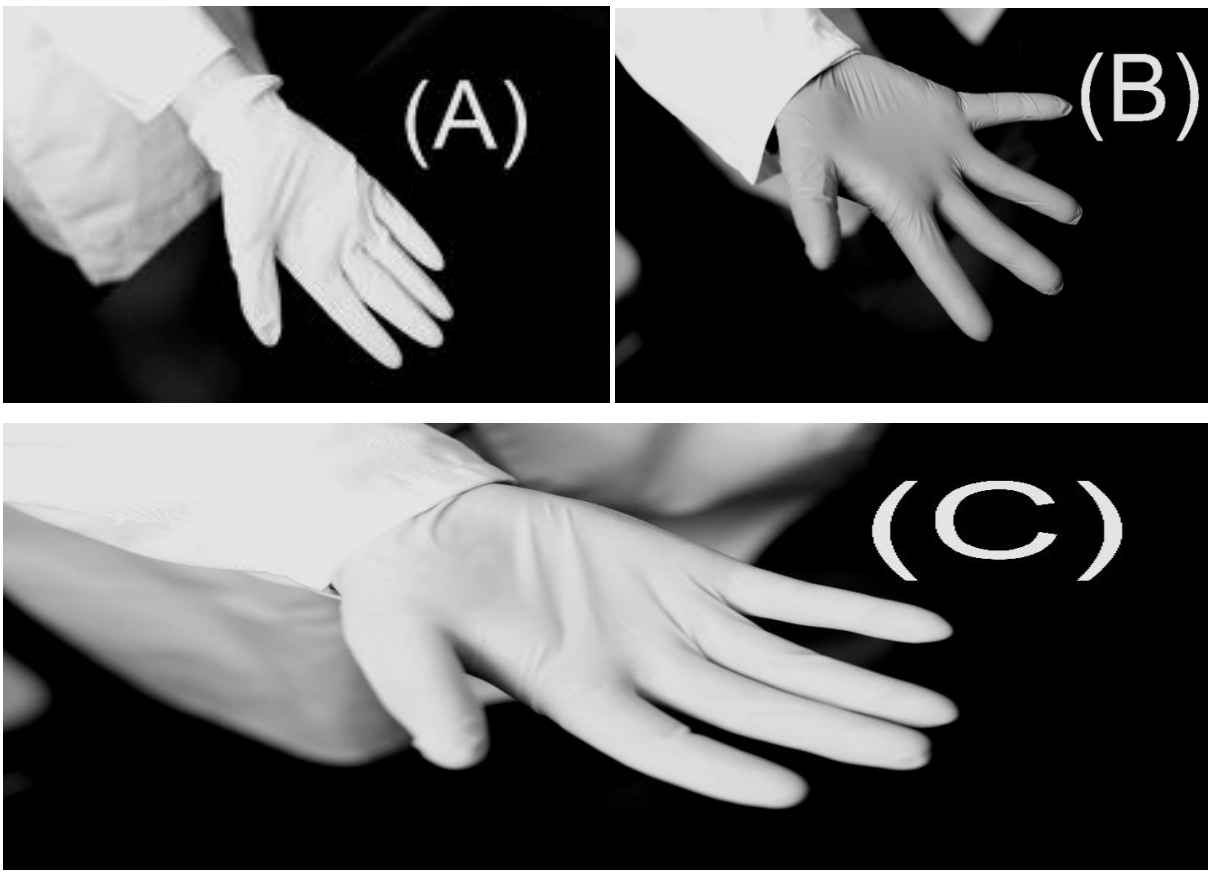
Because of the complicated structure of the BVM, it is recommended that disposable models be used in cases where proper cleaning and sterilization protocols and equipment are not in place.

The Gloves

Did you know that it's essential to wear the right sized protective equipment in your medical setting? Exam gloves are worn daily by many medical professionals and it's very important these exam gloves fit.

Hand protection is key during routine patient exams, procedures and handling sick patients. It's important for medical personnel to know the right size exam gloves to use at work. First, you must decide which type of exam glove to use. This can include natural rubber latex, vinyl, nitrile, latex free gloves, etc. Once you have determined the type of exam glove, proper sizing is crucial.

If you have exam gloves that are too big, they can easily be pulled off or liquids can make its way inside the glove. This can also be distracting to the wearer. If you have exam gloves that are too tight, it can cause fatigue and decrease dexterity. The images to the right indicate a pair of exam gloves that are (A) too loose, (B) too tight and (C) a good fit.



To make sure you're wearing a pair of the right sized exam gloves, you'll want to measure your hands. You want to measure around the knuckles of your dominant hand (above the V of your thumb). If you have a measurement of 8 inches, you would wear a size 8 glove or perhaps an 8 $\frac{1}{2}$ to be safe.

Lubricating Jelly

Why to lubricate endo tracheal tube before insertion?

Is it simply for easy insertion? You may already know ; Applying gel to an ETT before insertion helps it slide more easily into the trachea and prevents tissue damage, but it serves another important function: The gel enhances the seal produced by the cuff, especially high-volume, low-pressure cuffs.

Remember work around the cuff with gentle pressure using your thumb and index finger and of course wear sterile gloves

Occasionally the endotracheal tube may become "caught up" along the epiglottis. Because it is difficult to predict when this may happen, pre-lubricate the endotracheal tube cuff and tip with a thin layer of water-soluble lubricant, such as K-Y jelly or lignocaine jelly. This lubricant can also minimize the degree of surface trauma to the trachea and tracheal rings as the tube passes the vocal cords.

FOR THE EXTRA READERS

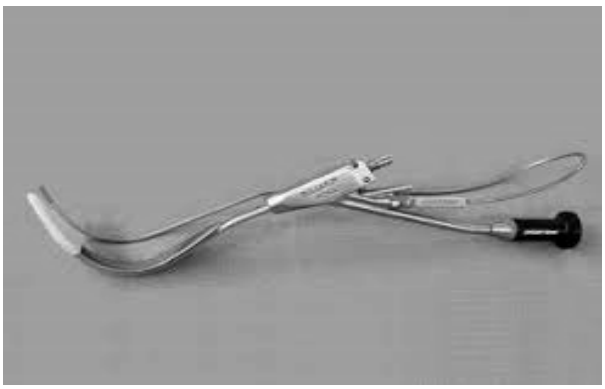
Lubrication of the ETT cuff with water soluble jelly may delay the increase in cuff pressure during general anesthesia with N_2O. However, the clinical significance of this effect may be limited.

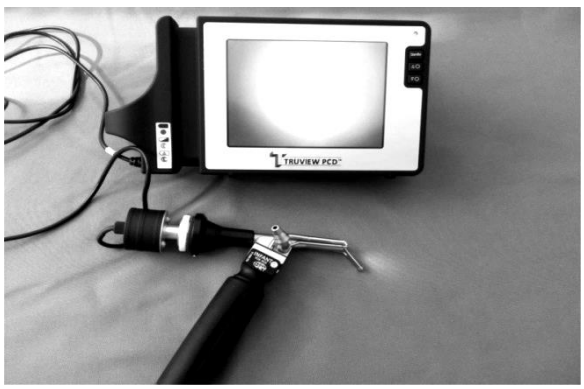
Laryngoscope

Visualization of the larynx by direct or indirect means is referred to as laryngoscopy and is the principal aim during airway management for passage of a tracheal tube.

Laryngoscopy is a term describing visualization or examination of the larynx by distraction of the upper airway structures, typically for the purpose of tracheal intubation and airway management in modern anesthesia

and critical care practice as well as in many trauma scenarios. For nearly a century, direct laryngoscopy has been the standard technique for tracheal intubation. In this approach, a rigid laryngoscope is used to expose the laryngeal inlet under direct vision or line of sight to facilitate placement of a tracheal tube beyond the vocal cords. Alternatively, indirect techniques for tracheal intubation have been developed that do not require direct vocal cord visualization. These newer approaches include the design and use of malleable or rigid optical stylets, rigid indirect laryngoscopes such as the Bullard and TruView EVO2, fiberoptic technology, and video laryngoscopes, in which video camera systems provide a focused view of the laryngeal inlet.



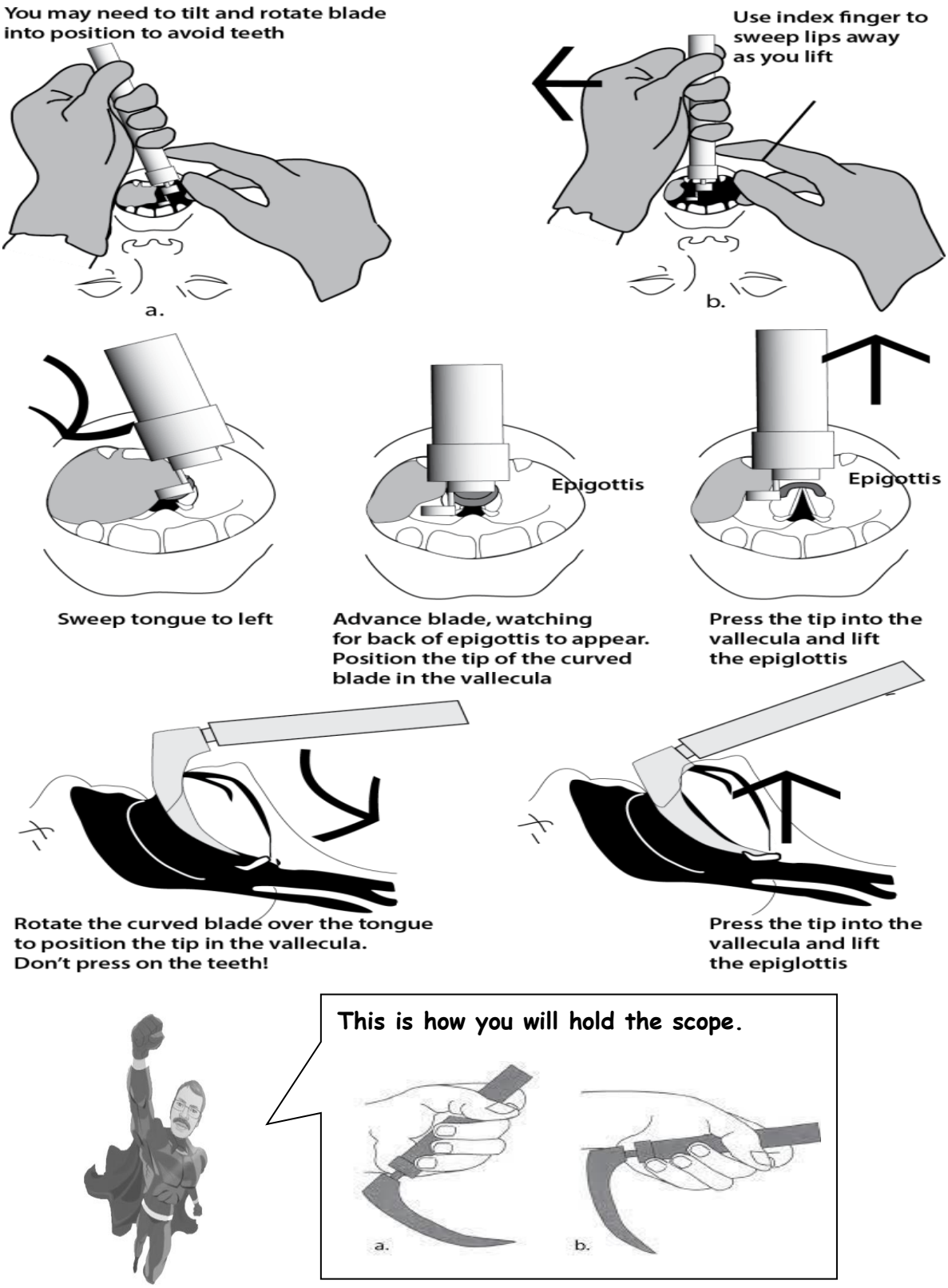


The most commonly used scopes and blades are either Mac or Miller type.

How to select the correct blade size?

The blade length excluding the base is measured by placing the proximal blade at the upper incisor teeth with the blade tip extending to the angle of the mandible. If the blade tip is within 1 cm proximal or distal to the angle of the mandible, it is an appropriate blade length for intubation. Once you inserted the blade there should be no movement at the wrist joint Lift the scope towards the ceiling.

How to visualize the larynx using laryngoscope?



Standard Laryngoscopes — Recommended Cleaning, Sterilization, and Instructions for Use.

Operating Instructions

1. Engage the blade by aligning the slot of the blade on to the hook pin of the handle and apply a sufficient force as shown in Figure 1.**Use your thumb to attach and detach blades.**
2. Apply force upward to bring the blade in operating position as in Figure 2.
3. To bring the blade to standby position, apply force downward as in Figure 2.

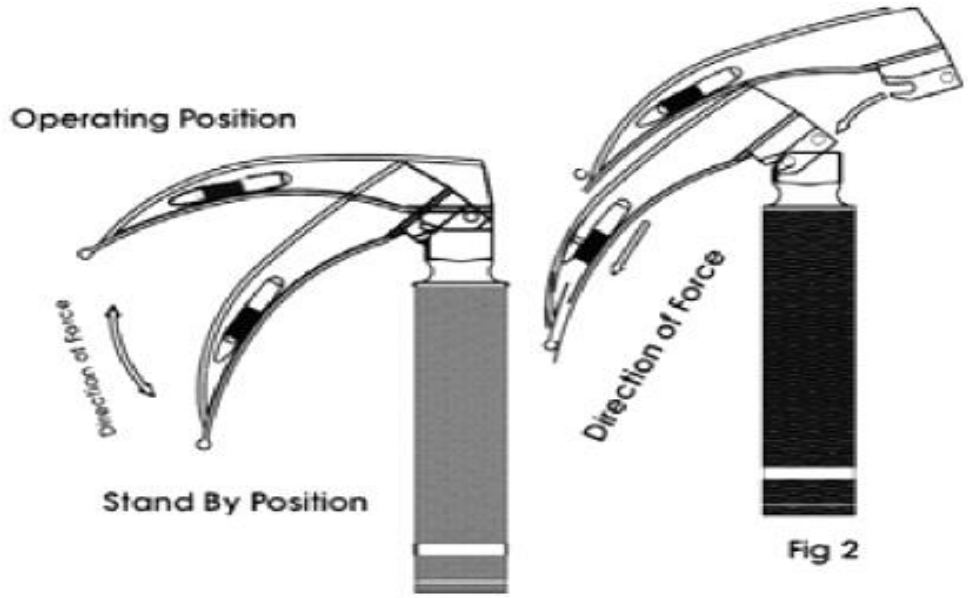


Fig 2

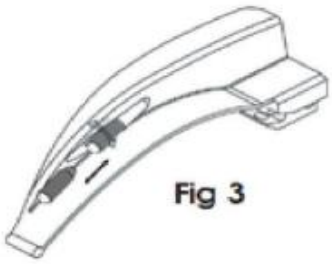


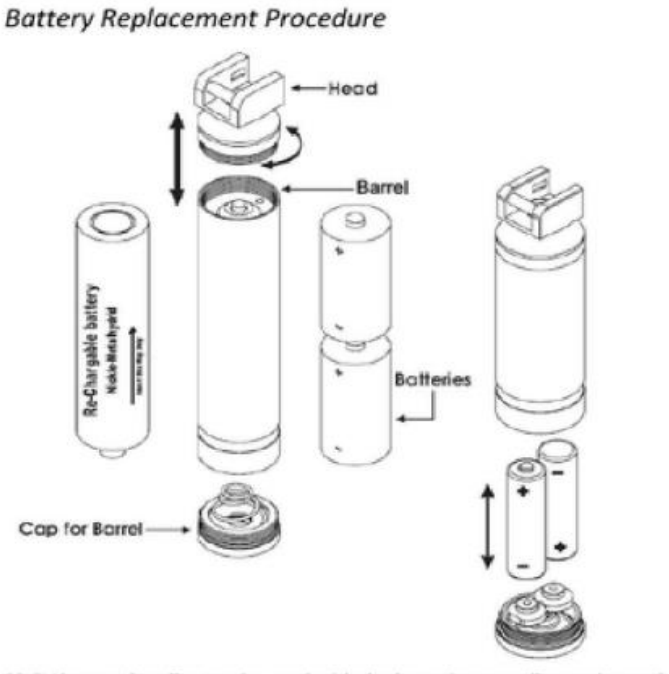
Fig 3

Bulb Replacement Procedure

1. Unscrew the bulb counter-clock wise direction until free as shown in Figure.
2. Replace the bulb and verify that the bulb is sufficiently tight before use.

Battery replacement procedure

The cells should be changed according to the manufacturer's instructions. Make sure you are inserting the cells in correct polarity and remember to tight the caps.



Make sure everything is tight and working before using and don't forget to ready the suction too.

Cleaning Procedure

Attention: Remove batteries before cleaning, high level disinfection or sterilization of the laryngoscope system.

Blades and Handles - Cleaning

1. Immediately after use, the laryngoscope system should be rinsed under cool running tap water until all visible soil is removed. Ensure that all hard to reach areas are flushed with the running tap water.

2. Immerse the sealed laryngoscope system in a enzymatic cleaner solution, prepared in accordance to the manufacturer's recommendations or for a minimum of two (2) minutes.

3. Remove device from enzymatic cleaner solution and rinse with lukewarm running tap water for a minimum of one (1) minute to remove all residues and visible soils,

4. Immerse the device in enzymatic detergent. Remove bottom cap and brush items thoroughly using a soft bristle brush, ensuring all visible soils are removed.

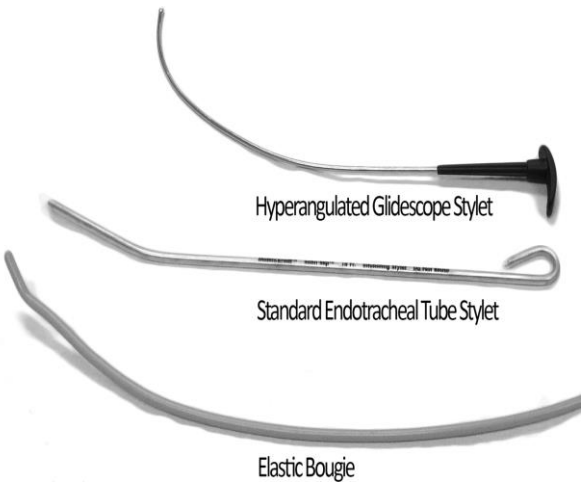
5. Rinse under running reverse osmosis/de ionized (RO/DI) water to remove detergent residuals.

6. Dry with a lint free cloth or filtered pressurized air.

7. The bulb may be cleaned with a cotton ball dampened in alcohol.

Bougie and Stylet

The tracheal tube introducer, known as the Bougie, is typically used **to aid tracheal intubation in poor laryngoscopic views** or after intubation attempts fail. The effect of routine Bougie use on first-attempt intubation success is unclear.



The stylet is a rigid but malleable introducer which fits inside the endotracheal tube and **allows for manipulation of the tube shape**; to facilitate passage of the tube through the laryngeal inlet.

There are different studies done to determine whether the Bougie is better than the stylet or vice versa, but there is no such evidence to show one is better than other.

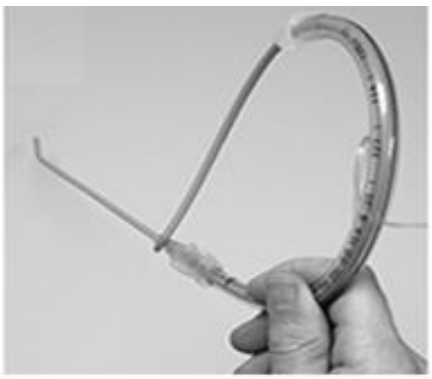
FOR THE EXTRA READERS

Bougie-assisted Endotracheal Intubation

The bougie is typically held by the intubator 20- 30 cm proximal to the coude tip. It should be inserted via the side of the mouth, rather then down the center, so that rotation of the bougie provides better control of the tip in the vertical plane ,it is typically inserted directly into the trachea and then used as a guide over which the

endotracheal tube can be inserted.It can be preloaded with an endotracheal tube or an assistant can pass the endotracheal tube over the free end of bougie while the intubator maintains visualisation of the bougie/ cords and ensures the placement of the bougie remains secure. The assistant should continuously walk their hands down the bougie as the endotracheal tube is advanced over it. The tracheal tube should be introduced through the cords, over the bougie, using a 90° anti-clockwise rotation to prevent its beveled point from getting caught in the arytenoids.

when used to confirm endotracheal placement the bougie is passed down the endotracheal tube and there should be 'hold up' at 30-40cm depth, if this does not occur the bougie is likely to be in the esophagus. The bougie may be passed into one of the main bronchi by twisting the angled tip in the preferred direction, this may be useful to facilitate endobronchial intubation (e.g. in massive hemoptysis where other equipment is unavailable)


The 'Kiwi grip' can be used by a solo operator – the bougie is curled upon itself and preloaded with an endotracheal tube, as can the 'pistol grip' (when the bougie is held together with the laryngoscope in one hand

Make sure that the cleanliness of bougie

Check any break in the wand or stylet

Use correct length of bougie stylet

After use .

clean wand and stylet separately in running water

clean with spirit

soak in disinfectant

Oxygen source and Bain Circuit

You need to make sure adequate supply of oxygen is there at the time of intubation. Even if you have central oxygen outlet in the scene keep an oxygen cylinder handy.

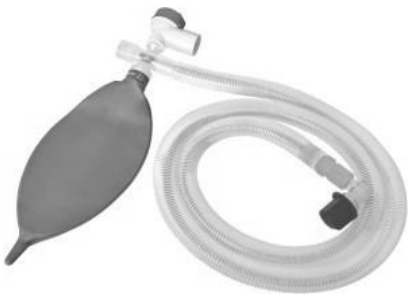
Connect an oxygen regulator to your central oxygen outlet and check adequate oxygen levels are available. You need to keep a back up oxygen tank or cylinder incase central outlet failure happens. You must check the oxygen pressure in the cylinder by connecting the pressure gauge, flow meter and opening the valve. Or using the cylinder wrench, turn the oxygen cylinder valve at least one half turn counter-clockwise. Check the pressure gauge to ensure that there is ample oxygen in the tank.

Bain circuit

The Pethick Test for the Bain Circuit

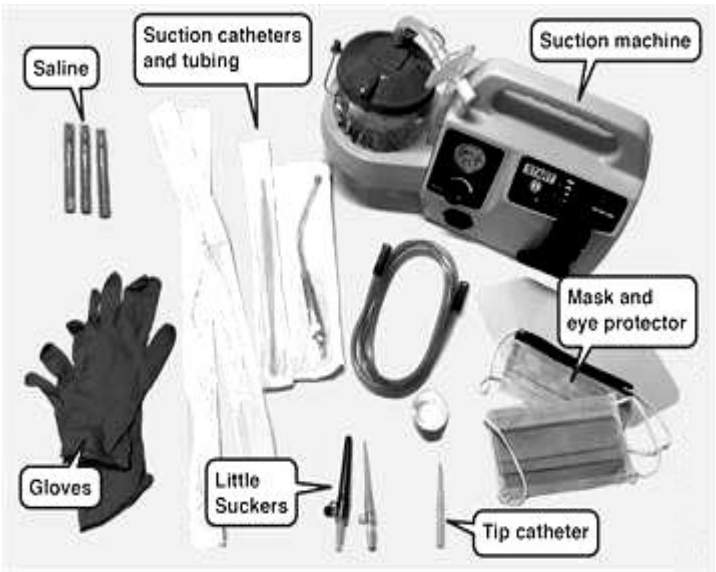
1. Occlude the patient's end of the circuit (at the elbow).
2. Close the APL valve.
3. Fill the circuit, using the oxygen flush valve.
4. Release the occlusion at the elbow and flush. A Venturi effect flattens the reservoir bag if the inner tube is patent.

Bain circuits use a high fresh gas flow to the patient's airway and to wash out the exhaled carbon dioxide. - An inner tube delivers fresh gases and an outer corrugated tube serves as the outlet for exhaled gases. A Bain circuit ensures that there is no re-breathing of the exhaled gases.



Suction tubing and apparatus.

Endotracheal suctioning (ETS) is a necessary practice carried out in intensive care units. In involves the removal of pulmonary secretions from a patient with an artificial airway in place. All intensive care nurses should be aware when performing this intervention of the potential hazards a patient is exposed to, and should endeavor to prevent or minimize these.



The current dilemma facing nurses is the overwhelming view that ETS should be performed only when indicated as necessary by assessment, to minimize the exposure of the patient to the hazards of ETS, but also recognition that ETS is a necessary procedure to maintain a patent airway and clear secretions. As we; nurses are accountable for all aspects of this practice, we should be able to make an informed choice about the frequency with which ETS is performed.

For the extra readers

Suction only as needed: The literature does not agree on an appropriate frequency of suctioning, but one 2009 study in Intensive and Critical Care Nursing suggested it should be performed only when necessary due to its risks and adverse effects, including that patients often describe it as "painful and uncomfortable, and may result in a choking sensation." Despite the discomfort, it is often a necessary procedure that can improve breathing.

Increase oxygen in different amounts for adults and children: While pre-oxygenation at 100% is the typical practice with adults before, during and after intubation, that same

level of oxygen can cause hyperoxia-over oxygenation-in preterm infants according to a 2010 article in Respiratory Therapist. However, some oxygen is necessary in infants to prevent bradycardia-and apnea so, many NICUs recommend oxygen at 10 to 20 percent before intubation.

Use low vacuum pressure unless otherwise necessary: A number of studies recommend using the lowest vacuum pressure to reduce risks of hypoxia and atelectasis, and tracheal mucosa injury. However, the pressure has to be strong enough to remove the secretion. So researchers suggest nurses and respiratory therapists don't rely on the manometer dial of the suctioning equipment alone. "It depends on the suction catheter-ET-tube ration, the duration of the procedure and the volume and viscosity of the secretions." Ultimately, they recommend the lowest possible suction pressure—usually between 80 and 120 mmHg, unless the secretion is not responding.

Choose the right suction catheter size: If a suction catheter is too large for the ET or there is too much vacuum pressure, atelectasis can occur. Ideally, the general recommendation is to use a suction catheter "that has an external diameter less than 50% of the size of the [endotracheal tube (ETT)] inner diameter," according to Respiratory Therapist. Or, put another way, a suction catheter that "occludes less than one-half the internal diameter of the ETT lumen," and to always use the smallest suction catheter possible that will still allow for effective aspiration.

Weigh the pros and cons of open or closed suctioning systems: While the literature is inconclusive as to whether open trachea or closed tracheal suctioning systems cause greater infections or trauma to tissues, many health care professionals have come to prefer closed suctioning systems (CSS or CTSS) for their convenience and speed. According to a study in the Egyptian Journal of Chest Diseases and Tuberculosis, CTSS had the following advantages over OTSS: "improved oxygenation; decreased clinical signs of hypoxemia; maintenance of positive end-expiratory pressure; limited environmental, personnel and patient contamination; and smaller loss of lung volume."Maintaining PEEP is

very important and with perceived reduced infections CSS in intubated patients is becoming standard of care in most health care facilities.

Use continuous suction: Both continuous and intermittent suctioning can cause some damage to the trachea, however, when using the CTSS system, researchers recommend using continuous suctioning, and otherwise there is a risk of alveolar collapse.

Use shallow suction depth when possible: While studies in neonates found no major differences in heart rate and oxygen saturation between deep or shallow suctioning, deeper suctioning can cause mucosal injury, bleeding, and even vagal stimulation and bradycardia. However, sometimes deep suctioning is needed, particularly when there are larger amounts of mucus in the lower airways. In lieu of more conclusive studies, the general recommendation is to "minimize the use of deep suctioning."

There are two types of ET tube suctioning Deep and Shallow; each has its pros and cons. For deep ETS, the catheter is inserted until it is beyond the tip of the ETT, or until it touches the carina. Deep suctioning is usually needed when there are large amounts of secretions in the lower airways. The drawback with deep ETS is that there is some degree of mucosal injury and the potential for bleeding, as well as the possibility of vagal stimulation and bradycardia. Pulling the suction tube 0.5 to 1 c.m may help in preventing the complications. Withdraw suction tube applying suction in a rotary fashion.

With shallow ETS, the catheter is inserted only to the tip of the ETT, thereby avoiding injury to the airway. Premeasured suctioning requires that the approximate depth to the tip of the ETT be estimated by using a suction catheter that has graduated centimeter markings The centimeter marking of the ETT at the lip is then noted before the suction catheter is inserted to the same distance that exists from the lip to the tip of the ETT. There is also no cough stimulated with shallow ETS, which means that the maneuver will only clear secretions from within the lumen of the ETT.

Total duration of suction

A longer duration of suction can increase the negative pressure within the lungs and reduce lung volumes. A longer duration of suction also increases the risk of hypoxia and its

associated complications. Recommended a suction duration of **10 to 15 seconds for adults** and **5 seconds or less for pediatric patients and neonates**, is a good to minimize complications. It is also beneficial if we allow **20-30 Seconds** of rest for the patient between each attempts of suctioning.

Indications for suctioning

Visible or audible secretions – rattling or bubbling sounds, audible with or without a stethoscope Decreased oxygen saturation levels

Bradycardia / tachycardia

Increased pCO2

Deteriorating blood gas values

Changes in respiratory rate and pattern with increase respiratory distress

Change of colour (cyanosis, pallor, mottled)

Suspected endotracheal tube obstruction

Ventilator alarms i.e. Increased proximal airway pressure / decreased tidal volume

Decreased breath sounds / absent chest movement

Increased airway pressure when ventilated (decreased tidal volumes)

Decreased chest excursion / asymmetry

Patient agitation

Precautions with Endotracheal Suctioning

Raised ICP

Pulmonary Hypertension

Pulmonary Oedema

Pulmonary Haemorrhage

These conditions may be exacerbated by suctioning and extra precautions taken

.

Potential complications of endotracheal suctioning

Respiratory

Hypoxia

Bronchospasm

Tracheobronchial mucosal trauma resulting in potential pulmonary hemorrhage

Contamination of airway leading to nosocomial infection

Unplanned Extubation

Atelectasis (loss of ciliary function / glottis closure)

Right upper lobe collapse (excessive suction pressures)

Pneumothorax

Cardiovascular

Vagal response bradycardia

Haemodynamic instability

Pulmonary vasoconstriction

Neurological

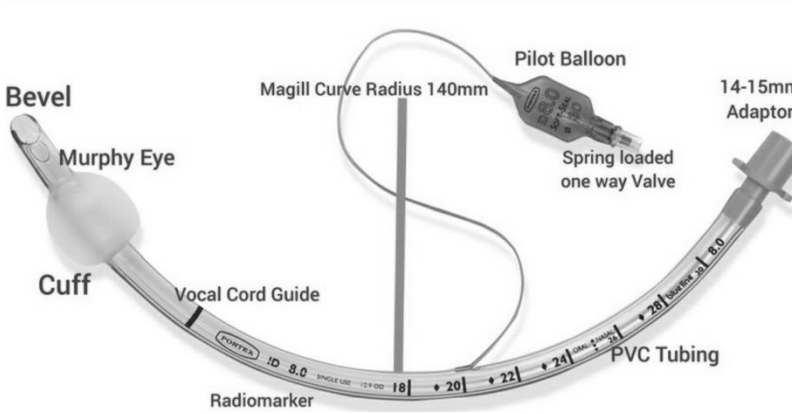
Changes in cerebral blood flow velocity / Raised intracranial pressure

Decreased oxygen availability in cerebral blood flow increases risk of IVH and

Hypoxic-ischemic encephalopathy.

Endotracheal tube

An endotracheal tube is a flexible plastic tube that is placed through the mouth into the trachea to help a patient breathe. The endotracheal tube is then connected to a ventilator, which delivers oxygen to the lungs. An endotracheal tube is placed



when a patient is unable to breathe on their own, when it is necessary to sedate and "rest" someone who is very ill, or to protect the airway. The tube maintains the airway so that air can pass into and out of the lungs.

How do you calculate paediatric ET tube length?

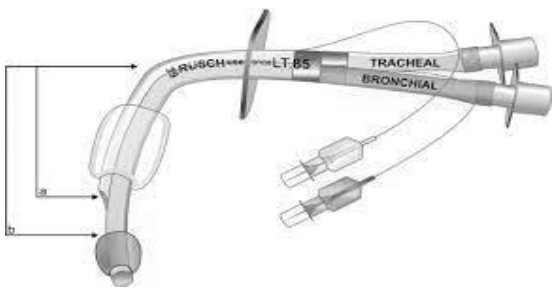
The endotracheal tube (ETT) size formula, **(age/4) + 3.5**, with a cuffed tube makes more sense anatomically. Classic teaching is that we should use the formula **(16+age)/4** or **(age/4) + 4** to calculate the uncuffed paediatric ETT size. There is one more equation (PALS) **(age/2+12)** to find out the depth of insertion of ETT

TYPES

Oral or nasal,

Cuffed or uncuffed, preformed tube,

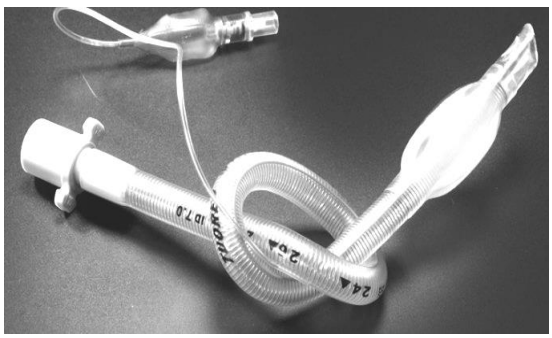
Reinforced tubes, and double-lumen endobronchial tube.



Complications and Risks

Bleeding

Oesophageal placement of the tube: One of the most serious complications is improper placement of the endotracheal tube into the oesophagus. If this goes unnoticed, the lack of oxygen to the body could result in brain damage, cardiac arrest, or death.



Temporary hoarseness when the tube is removed Injury to the mouth, teeth or dental structures, tongue, thyroid gland, voice box (larynx), vocal cords, windpipe (trachea), or oesophagus.

Infection

Pneumothorax (collapse of a lung):

Aspiration of contents of the mouth or stomach during placement which can, in turn, result in aspiration pneumonia

Persistent need for ventilator support .

Atelectasis: Inadequate ventilation (a respiratory rate that is too low) can result in collapse of the smallest of airways, the alveoli resulting in atelectasis (partial or complete collapse of a lung).

Long term complications

Tracheal stenosis, or narrowing of the trachea:

Tracheomalacia

Spinal cord injuries

Tracheoesophageal fistula (an abnormal passageway between the trachea and esophagus)

Vocal cord paralysis: A rare complication that can cause permanent hoarseness

Choose correct size of ETT

Check for leakage or break in the cuff and ETT

Lubricate ETT before intubation

After intubation secure properly

Check cuff pressure with manometer

Oro pharyngeal Airway.

Oro pharyngeal airway (also known as an oral airway, OPA or Guedel pattern airway) is a medical device called an airway adjunct used to maintain or open a patient's airway. It does this by preventing the tongue from covering the epiglottis, which could prevent the person from breathing.

In intubated patients Guedels can be used to prevent tube biting and to access the oropharynx during suctioning. This maneuver may, however, be hazardous in children between 5 and 10 years of age with loose deciduous teeth or to adult patients having loose teeth. **And remember Use the airway only in unconscious patients and or minimally responsive. Otherwise stimulation of gag can cause aspiration .It may be wise to avoid oropharyngeal airway completely in an otherwise calm patient who is not vigorously biting tube to prevent injury to lips, mouth and teeth**

How to insert an oropharyngeal airway

As necessary, clear the oropharynx of obstructing secretions, vomitus, or foreign material. Determine the appropriate size of the oropharyngeal airway. Hold the airway beside the patient's cheek with the flange at the corner of the mouth. The tip of an appropriately sized airway should just reach the angle of the mandibular ramus. Next, begin inserting the airway into the mouth with the tip pointed to the roof of the mouth (ie, concave up).To avoid cutting the lips, be careful not to pinch the lips between the teeth and the airway as you insert the airway. Rotate the airway 180 degrees as you advance it into the posterior oropharynx. This technique prevents the airway from pushing the tongue backwards during insertion and further obstructing the airway. When fully

inserted, the flange of the device should rest at the patient's lips. Alternatively, use a tongue blade to depress the tongue as you insert the airway with the tip pointed to the floor of the mouth (ie, concave down). Use of the tongue blade prevents the airway from pushing the tongue backward during insertion.

Ventilator circuit

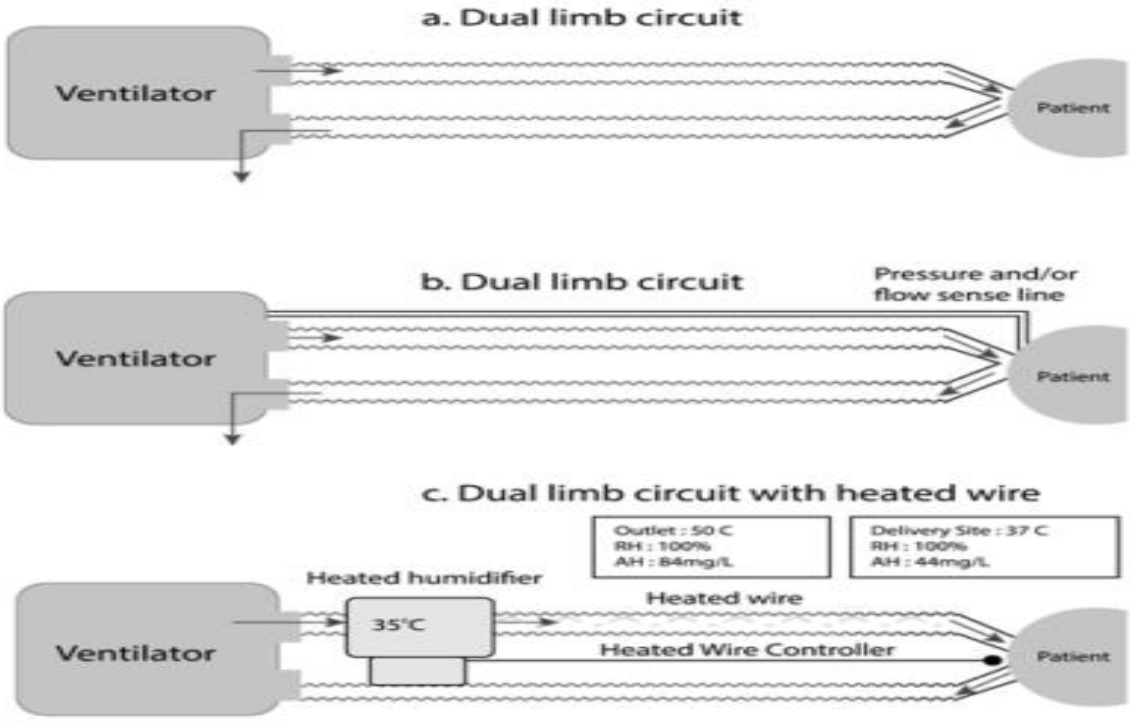
The ventilator circuit refers to the tubing that connects the ventilator to the patient, as well as any devices that might be connected to the circuit. The circuits are primarily divided into dual limb and single limb circuits.

There are multiple configurations of dual and single limb circuits (outlined below).

Note on humidification & circuit configuration: some dual and single limb circuits may contain a heated wire in the inspiratory limb to optimize heat & humidification delivery to the patient and to prevent excess condensation from accumulating when using an active heated humidification system. If an active heated humidification system is used in the absence of a heated wire inspiratory limb, a water trap is often needed. Some water traps may allow for emptying without circuit disconnect (an important consideration with COVID19).

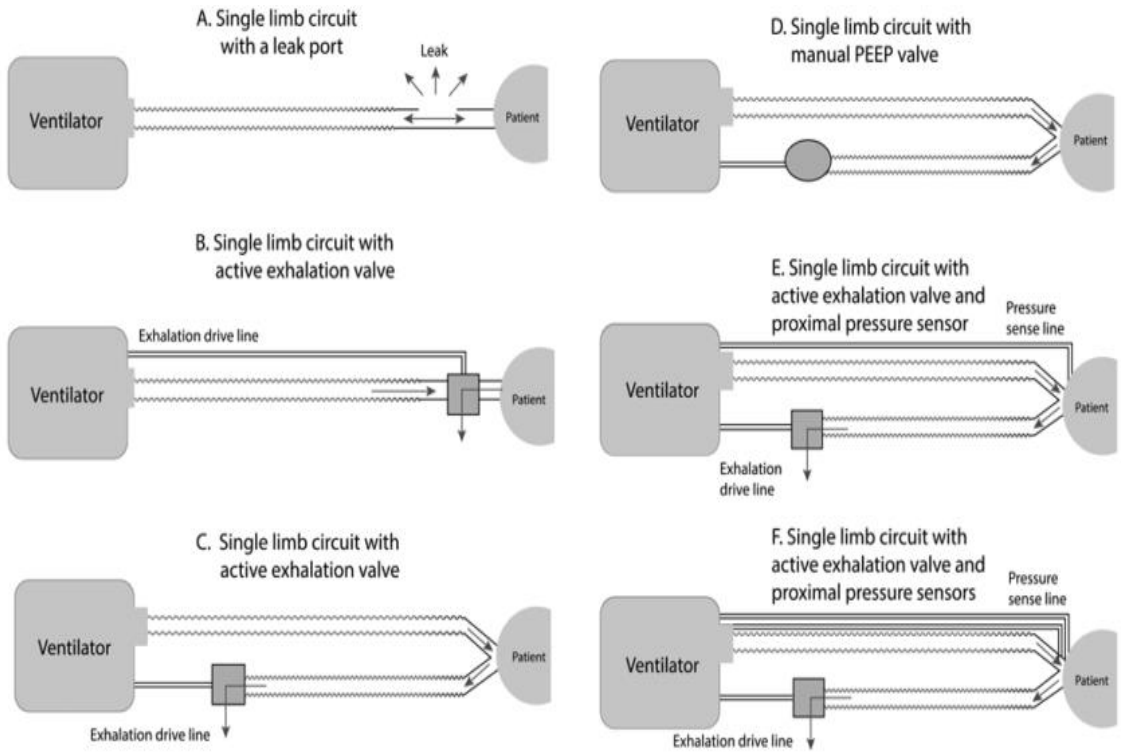
FOR THE EXTRA READERS

Dual limb circuit (Figure a, b and c) – used by most traditional critical care ventilators. Flow/pressure and PEEP are commonly measured/controlled in the machine, and thus no additional circuit transducer tubing is needed



(a). Some circuits do use proximal flow/pressure sensors **(b)**. These may include a heating element in the inspiratory limb and port for temperature monitoring **(c)**. Standard single limb with built in leak (figure A) – mostly for non invasive devices

- Standard single limb circuit with active exhalation valve and internal PEEP – (figure B and C)
- Standard single limb circuit with active exhalation valve and manual PEEP – (figure D)
- Standard single limb circuit with active exhalation valve, internal PEEP and proximal pressure sensor – (figure E) – this is one of the most common single limb circuit setups
- Standard single limb circuit with active exhalation valve, internal PEEP and two proximal pressure/flow sensors – (figure F) – this is usually a proprietary circuit type that is commonly encountered and allows measurement of exhaled tidal volume



Filters

Ventilator filters play a key role in protecting the safety of patients on mechanical ventilation and reducing the risk of cross contamination. Filtration can help to prevent the inhalation of harmful pathogens. There are different kind of filters used with circuit mainly Heat and Moisture Exchangers (HME) and viral filters.

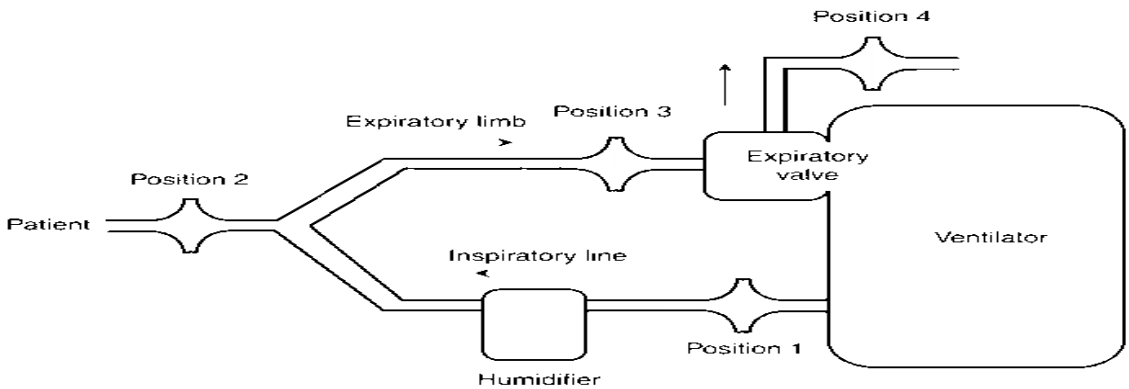
Heat and Moisture Exchanger Filter (HMEF) is usually incorporated with a microbiological filter that provides passive humidification. HME Filter provides adequate humidification of inspired gas reduces the airway resistance, and lowers the incidence of infections and maintains the respiratory loads. Heat and Moisture exchanger filter is designed to enhance the protection against airborne microbes like bacteria virus etc. It is commonly employed in ICU for ventilation purposes to ensure proper humidification of inspired air.

Bacterial–viral air filters are medical devices used in respiratory ventilators or breathing circuits to protect patients, equipment, and/or the environment from viruses and bacteria.

They may be either electrostatic or mechanical, based on their working principle: electrostatic filters use an induced electrostatic charge to capture particles, while mechanical filters use a pleated porous membrane. Mechanical filters can reach higher filtration efficiency than electrostatic filters, but they impose higher airflow resistance. Air filters are classified upon their efficiency: efficiency particulate air (EPA), high-efficiency particulate air (HEPA), and ultra-low penetration air filters retain a minimum of 99.95%, 99.97%, and 99.999% of 0.3 μm particles, respectively. Heat moisture and exchangers (HMEs) retain heat and humidity from exhaled air and return them to the patient during the following inspiration. HMEs also provide a filtration function that can be either electrostatic or mechanical, and they can be classified as either EPA or HEPA based on their filtration efficiency. HMEs are passive humidifiers and, as such, they should be placed at the inlet of the airway interface. HMEs must not be used with humidified gasses because the humidity retained by the hygroscopic membrane may increase airflow resistance.

Connecting a bacterial–viral filter to the breathing circuit modifies its mechanical characteristics: (1) it increases the compliance of the circuit; (2) it increases dead space if placed at the airway interface; (3) it adds a resistance that causes a pressure drop between the inlet and the outlet of the filter

FOR THE EXTRA READERS



The filters can be connected to various positions of a ventilator and circuit.

Position 1

The bacterial–viral filter on the inspiratory limb has two functions: (1) protecting the equipment from the rare event of contamination with exhaled air, (2) protecting the patient in case he/she breathes room air through the safety valve that some ventilators open in case of sudden failure. The inspiratory filter does not prevent environmental contamination. The mechanical characteristics of inspiratory filters remain stable over time because the gas flowing through them is clean and dry. The pressure drop across the filter may affect the inspiratory flow and pressure waveforms.

Position 2

A filter between the breathing circuit and the airway interface protects the patient, equipment, and environment from airborne contamination. HMEs should be placed only in position 2. This configuration increases dead space, thus affecting gas exchange. In adults, a filter between the Y-piece and the airway interface increases minute ventilation or arterial partial pressure of carbon dioxide. Position 2 should be avoided in neonates because filter dead space (e.g., 8–10 mL for the smallest filters) is very high compared with the patient tidal volume. The ventilator cannot detect the effects of a filter between the Y-piece and the airway interface. Therefore, monitoring ventilation waveforms over time is recommended to identify the possible consequences of increased filter resistance.

Positions 3 and 4

Expiratory filters prevent bacteria or virus transmission to the environment. They do not add dead space, but they increase circuit compliance and resistance, similarly to inspiratory filters. While the work required to overcome the resistance of the inspiratory line is provided by the ventilator, increasing the expiratory line resistance prolongs the time to exhale. This issue is particularly critical in patients at risk of developing intrinsic

positive end-expiratory pressure (PEEP; e.g., patients with a small endotracheal tube or with obstructive diseases). The humidity of the gas flowing through the expiratory filter may significantly increase its resistance and nearly occlude it. The highest risk occurs using an HME filter in position 3 or 4 with humidified gasses and non-heated expiratory lines. HME filters must not be used with humidified gasses and must be connected only in position 2. If the expiratory limb of the breathing circuit is not heated, a water trap on the expiratory line may reduce the risk of rainout inside the filter. While the filter is in place, check that the airway pressure reaches the pre-set PEEP level and set the "high PEEP" alarm, if present, at a value only slightly higher than the desired PEEP.

The expiratory filter is a single-patient device; it should also be changed if PEEP tends to increase compared with the pre-set value, in the presence of rainout, after nebulization, and, in any case, every 12–24 h. Caution should be used during filter replacement because **opening the breathing circuit is an aerosol-generating procedure**. Always remember the adult and pediatric filters are different in weight and size.

Cuff pressure manometer

It is recommended to maintain endotracheal tube cuff pressure within a range of 20 to 30 cm H2O to prevent complications of cutting of mucosal capillary blood circulation and thus prevents necrosis and tracheal stenosis later. Cuff pressure gauges have been

established as a standard device in many clinical institutions. More and more societies of anesthesiology and intensive care throughout the world are endorsing the systematic control of cuff pressure in the recommendation of their guidelines. Ventilator-associated pneumonia (VAP) is the leading nosocomial cause of mortality in the Intensive Care Unit and has significant impact on hospital costs and length of stay. A leading cause of VAP is micro-aspiration of potentially infectious secretions through micro channels formed from in folding of redundant cuff material after inflation. Cuff pressure management can contribute to reduce tracheal ischemia and subsequent complications. Availability of the device and precision of the measurement adds to the success of treatments.

How to use cuff pressure manometer

Cuff inflation should be done slowly to avoid over inflation and can be monitored by measuring cuff pressure. Simply palpating the pilot balloon is not an appropriate way to assess cuff inflation and may cause harm. Instead, cuff pressure should be measured using cuff manometer, and should be performed regularly and whenever air is added to or taken out of the cuff. A cuff manometer is designed to measure cuff pressure, and can be used to inflate the cuff or remove air from the cuff of the ET tube. **The gauge shows pressure in centimeters of water.** There is also an inflator bulb and air vent button that can be used to adjust pressure by adding or releasing air. **The inflator bulb** can be used to add air to the cuff if the pressure is too low. **The red air vent button located on the back** of the manometer can be depressed to release air if the pressure is too high. **To measure the cuff pressure, attach the manometer to the pilot balloon** of the ET tube and **note the needle** on the manometer. That will reflect the measured pressure. **Recommended cuff pressure is highlighted in green** on the face of the gauge. Continue squeezing the inflator bulb, or releasing air as needed to ensure the cuff is inflated properly.

Keep a stethoscope (For auscultation of lung field), ET tube Fixators and Scissors at the ready. The patient may collapse at any time of intubation and mechanical ventilation. So it is necessary to keep all the emergency drugs and crash cart including defibrillator nearby.

FOR THE EXTRA READERS

Medications used for intubation

Pre-- treatment – agents should be given 3 minutes prior to intubation (can be given in any order)			
Drug	**Dose**	**Indication**	**Other notes**
Fentanyl	2---3 mcg/kg	Elevated ICP, cardiovascular disease (ischemic coronary disease, aneurismal disease, great vessel rupture or dissection, intracranial hemorrhage)	Fentanyl helps decrease catecholamine discharge secondary to intubation, thus decreasing the risks associated from BP increases in pts with CV disease, aortic dissections, etc. Be careful if the patient is already hypotensive
Lidocaine	100 mg	Head injury, traumatic brain injury, unknown mechanism of injury, elevated ICP	Lidocaine will help protect the patient from increases in intracranial pressure caused by intubation

Summary of Induction Agents						
Agent	**Usual Emergency Induction Dose**	**Onset (sec)**	**Duration of Action (min)**	**Indications**	**Adverse Effects**	**Comment**
Midazolam	0.2-0.3 mg/kg IV	60-90	15-30	Not routinely recommended for RSI. May use for post-intubation management	Respiratory depression Apnea Paradoxical agitation	Not recommended for RSI. Patient response may be extremely variable
Thiopental	3 mg/kg IV	<30	5-10	Patients with elevated ICP or status epilepticus who are hemodynamic-ally stable	Histamine release Myocardial depression Venodilation Hypotension	Not routinely used Avoid intra-arterial injection (may cause gangrene) Pregnancy category C
Propofol	1.5 mg/kg IV	15-45	5-10	Hemodynamically stable patients with reactive airway disease or in status epilepticus	Hypotension Myocardial depression Reduced cerebral perfusion pressure Pain on injection	Ultra-short acting Negative CV effects limits use for induction in RSI
Ketamine	1.5 mg/kg IV	45-60	10-20	Good option for patients with reactive airway disease or who are hypovolemic, hemorrhaging, or in shock	Increased: BP HR Intraocular pressure	Not recommended in hypertensive or normotensive patients. Use caution in patients with cardiovascular disease

Etomidate	0.3 mg/kg IV	10-15	4-10	Used in almost all patients for emergency RSI. May consider alternative agent if patient is septic or in status epilepticus	-Adrenal insufficiency -Pain on injection -Myoclonic activity	Communicate to subsequent providers that patient received etomidate if patient septic

Paralytic Summary – Depolarizing

Agent	Usual Emergency Induction Dose	Onset (sec)	Duration (min)	Indications	Adverse Effects	Comments
Succinylcholine	1.5 mg/kg IV Increase to 2 mg/kg IV in myasthenia gravis 4 mg/kg IM (only in life threatening situations)	45	6-10	Essentially all patients except those with: Malignant hyperthermia Hyperkalemia ->5d after burn, crush, denervation, severe infection	Hyperkalemia Muscle fasciculations Elevated IOP	Bradycardia may occur after repeated doses, have atropine ready in the event it occurs

Paralytic Summary – Nondepolarizing						
Agent	**Usual Emergency Induction Dose**	**Onset (sec)**	**Duration (min)**	**Indications**	**Adverse Effects**	**Comments**
Rocuronium Can also be given for pre treatment purpose as a defasciculating agent.	0.6 mg/kg	60-75	40-60	RSI when succinylcholine contraindicated	No, clinically significant ADEs	Ensure contingency plan in place in the event of failed airway.
Vecuronium	0.01 mg/kg followed 3 minutes later with 0.15 mg/kg	120-180	45-65	Not recommended for RSI unless a nondepolarizing agent is indicated and rocuronium is not available	No clinically significant ADEs	Ensure contingency plan in place in the event of failed airway

THE RAPID SEQUENCE INTUBATION

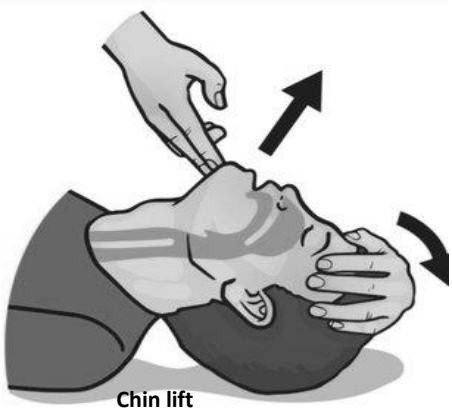
Rapid sequence intubation involves sequential steps that lead to successful endotracheal intubation. These steps allow for adequate assessment of the choice, dose, timing, and sequence of administration of sedatives, analgesics, and paralytics while ensuring that all equipment is ready and the patient's clinical status is optimized. Rapid

sequence intubation using NMBAs is the standard of care and is associated with a reduction in complications compared to using sedatives alone.

The following steps make up rapid sequence intubation; **preparation, pre-oxygenation, pretreatment, paralysis and induction, positioning, placement and confirmation, and following these, post-intubation management**. These are commonly called the "7Ps" of rapid sequence intubation and are described below.

Preparation includes assessing the degree of difficulty of a patient's airway and establishing adequate intravenous access and continuous monitoring (telemetry, blood pressure, and pulse oximetry). As mentioned prior, ETTs of multiple sizes should be available and all tested for cuff leaks. Laryngoscopes, both curved and straight and in multiple sizes, should be available. All laryngoscopes should be checked for a functioning light source. Bedside suction devices should be easily accessible. Of note, even if airway assessment does not reveal any obvious evidence of difficulty, a backup plan should be readily available. Adequate nursing staff and respiratory therapists must be present to assist with intubation, monitoring, administering drugs, and preparing the ventilator.

Pre-oxygenation involves providing the highest possible oxygen concentration at high flows for 3 to 5 minutes. This allows the patient to tolerate longer periods of apnea without causing hypoxia during rapid sequence intubation. The upper airway patency needs to be maintained with **chin lift** or jaw thrust maneuvers that facilitate oxygen entry into the airways. For patients in whom achieving high oxygen saturation is not possible, pre-oxygenation can be performed with non-invasive positive pressure ventilation masks or



Chin lift

positive end-expiratory pressure (PEEP) valves that can be added to the bag-valve-mask.

The effectiveness of pre oxygenation can be assessed by the ETO_2 value of 90% or an SpO_2 value of more than 95%.

Pre-treatment is an additional step involving administering medications that may optimize the clinical setting where intubation is being done. For example, intravenous fluids, anxiolytics with benzodiazepines, or opioid medications may be used before administrating sedatives and NMBAs. Typically a short-acting opioid such as intravenous **fentanyl** and or **Midazolam** a fast acting benzodiazepine is administered for pre-treatment. In patients with reactive airway disease, a short-acting beta-agonist (albuterol) may be administered during this step to minimize airway resistance. Rarely, in patients with shock, pretreatment with alpha-adrenergic inotropic agents may circumvent further reduction in mean arterial pressure following intubation due to loss of sympathetic tone from the use of specific induction agents.

Paralysis with induction involves the simultaneous administration of the medications for sedation and paralysis that have been decided earlier in the preparation phase based on clinical status, allergies, and potential contraindications. During rapid sequence intubation, the dose of these drugs should be pre-calculated and administered intravenously as a bolus and never titrated. The onset and duration of action should all be taken into consideration. **Etomidate** should be given in a dose of 0.15 mg/kg to 0.3 mg/kg intravenously, depending on the stability of the patient. **Ketamine** is to be given in a dose of 2 mg/kg intravenously. **Propofol** is given in doses of 0.5 mg/kg to 2 mg/kg intravenously, depending on hemodynamic stability. Immediately after the induction agent, the paralytic agent of choice is administered intravenously. **Succinylcholine** is given in a 1.5 mg/kg dose.

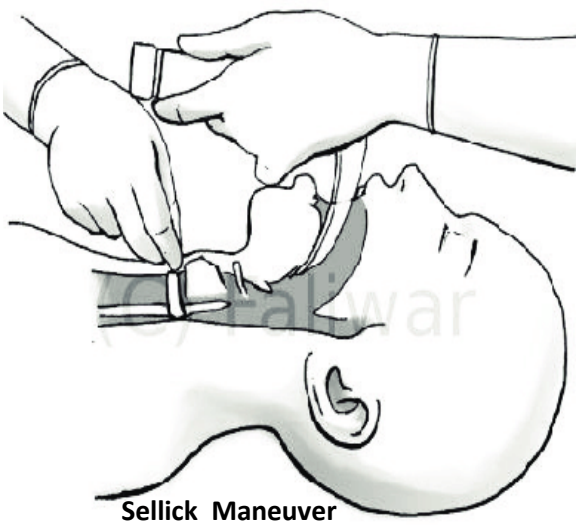
Vecuronium is used as an adjunct for continued paralysis in patients for upto a maximum of 24 hours post intubation.(Injection :Load: 0.08-0.1 mg/kg IV push over 60 sec OR 0.04-0.06 mg/kg IV push if following succinylcholine, PLUS maintenance: 0.01-0.015 mg/kg IV push 20-45min after loading as needed. Continuous Infusion ; Load: 0.001 mg/kg/min IV starting 20 min post bolus recovery maintenance: 0.0008-0.0012 mg/kg/min).

Fentanyl is a synthetic opioid that binds to opiate receptors resulting in decreased pain. Endotracheal intubation results in sympathetic nervous stimulation that can cause significant increases in blood pressure. Fentanyl can be given prior to induction agents reduce increases in blood pressure in patients with increased cranial pressure or in other clinical settings where a rapid rise in blood pressure is undesirable. Its rapid onset and metabolism relative to other opiates make it a common choice for premedication. Loading dosage can be 25-100 mcg over 1-2 minutes and as an infusion the dosage is 1-2 mcg/kg/hr.

Midazolam is a fast acting benzodiazepine that provides anxiolysis and some amnesia, facilitating endotracheal intubation. Like fentanyl, it can be given before induction agents to facilitate endotracheal intubation. Load: 10-50 mcg/kg (dose range 0.5-4 mg) slow IV injection or infusion over several minutes; repeat q5-15min PRN. Maintenance: Initial, 20-100 mcg/kg/hr infusion; titrate up or down 25-50% PRN

Atropine occasionally is used as a premedication. Its anticholinergic effects reduce ACH-mediated bradycardia that can accompany endotracheal intubation.

The protection and positioning phase is vital, as the patient is now paralyzed, and the airway must be protected from aspiration. Minimal bag-mask ventilation should be used to keep oxygen saturations adequate (**Modified RSI**); this will be possible only if pre-oxygenation was adequate. One can perform the **Sellick maneuver, in other words, apply pressure over cricoid cartilage to occlude the esophagus if necessary**. **Cricoid pressure is indicated in patients identified as having an elevated risk of aspiration**. Common indications for implementing cricoid pressure include patients who have recently eaten, gastroparesis, gravidity, nausea, recently vomited, hiatal hernia, or known

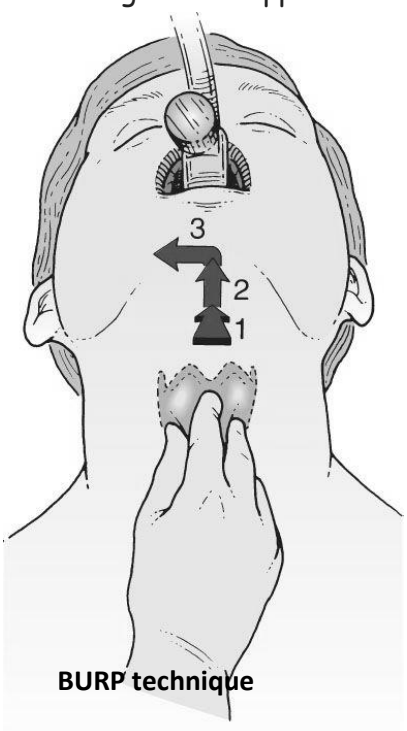


Sellick Maneuver

incompetent esophageal sphincter(s), increased intraabdominal pressure, inebriation, impaired neurological, and/or upper respiratory reflexes. Cricoid pressure is commonly used in emergency settings as multiple attempts at intubation, emergent situations

The practitioner who is performing the maneuver will often stand on the right side of the patient. The practitioner will use anatomical landmarks, including the thyroid cartilage and cricothyroid membrane, to identify the cricoid ring. Once identified, the practitioner may use the dominant hand or whichever hand is most able to apply appropriate pressure without impeding the team member's function performing the intubation. The thumb will remain on the patient's right side of the cricoid ring, with the second digit placed on the contralateral side of the cricoid ring. The practitioner may place the third digit on the same side as the second digit if this is found to be more comfortable.

While the patient is awake, a 10 N or 1 kg pressure is appropriate until induction is complete. Once the patient is unconscious, approximately 30 N or 3 kg will be applied in a posterior direction to the cricoid ring. **The constant pressure should be maintained until the endotracheal tube is appropriately placed with the cuff inflated to protect the airway**. When applying cricoid pressure in pediatric patients, the pressure should be decreased, considering the increased deformability of soft structures. A pressure of 20 N is effective when the patient is in a head-up position. A double-handed maneuver may be implemented when there is a concern for cervical spine instability. In such situations, the practitioner will use his or her free hand to support the posterior cervical spine while providing cricoid pressure. **Cricoid pressure should not be confused** with a technique commonly referred to as **"BURP"** (backward, upward,



BURP technique

rightward, pressure on the thyroid cartilage), used to **aid in the visualization of the glottis during intubation.**

Placement should occur once adequate sedation and paralysis have been obtained. Direct laryngoscopy should be performed, and once glottis is visualized definitively, an appropriately sized endotracheal tube with stylet should be placed through the vocal cords under direct visualization. After that, the endotracheal tube cuff is inflated with cuff pressure manometer (20-30 cm H_2O) and the stylet removed. Placement should be confirmed by end-tidal carbon dioxide detection, quantitative or colorimetric methods. Auscultation over both lung fields and the epigastric region should also be performed to ensure equal breath sounds on both sides in the chest and absent in the epigastric region. A chest radiograph should be performed to determine the depth of airway intubation. The endotracheal tube tip should be located more than 2 cm but less than 5 cm from the carina on chest radiography.

Post-intubation management involves securing the endotracheal tube, connecting the endotracheal tube to a mechanical ventilator, and evaluating and managing potential post-intubation complications. Appropriate sedation agents should be initiated, as discussed earlier. Most induction agents have short half-lives.

FOR THE EXTRA READERS

Commonly used sedatives for patients on ventilator

Benzodiazepines act through the Gamma-aminobutyric acid (GABA) receptor. This is a neuroinhibitory receptor that causes neurons to be less excitable when benzodiazepines bind to it. These drugs have anxiolytic, sedative, and hypnotic effects at increasing doses . The two most commonly used drugs for ICU sedation in this class are **midazolam** and **lorazepam**. Both of these drugs are lipophilic, although midazolam is more so in plasma. This allows it to quickly cross the blood–brain barrier, resulting in a more rapid onset of

action (≤1 min) than lorazepam. Lipophilicity also causes both midazolam and lorazepam to accumulate in adipose tissues, where they are not readily metabolized.

A potential adverse effect of benzodiazepines is respiratory depression. These drugs shift the CO_2 response curve to the right. Unlike the opiates, benzodiazepines tend to reduce both respiratory rate and tidal volume, so the "slow and deep" breathing pattern of opiates is less commonly seen with these drugs. These drugs have antiepileptic properties that make them useful for seizures, and they are valuable for use in alcohol and chronic benzodiazepine withdrawal. Rarely, there may also be a paradoxical reaction to the drug, resulting in agitation. This unusual response is seen more frequently in elderly patients.

Although benzodiazepines have traditionally been used as first-line agents, randomized controlled trials comparing them with newer agents such as propofol or dexmedetomidine clearly show that benzodiazepines lead to worse outcomes, including delirium, oversedation, delayed extubation, and longer time to discharge.

Propofol is another commonly used ICU sedative. The mechanism of action is not well understood, but evidence supports the theory that it acts through modulation of neurotransmitter release, including GABA, and has direct effects on the brain. This GABAergic agent is a lipophilic drug that quickly crosses the blood–brain barrier, with an onset of action on the order of seconds to minutes. There is also an extremely rapid redistribution of propofol to peripheral tissues, again on the order of minutes, coupled with a large volume of distribution. These pharmacokinetic properties make propofol ideal for early recovery of consciousness after discontinuation of continuous infusions, even when administered for prolonged periods. Hypotension is a common occurrence with propofol as a result of decreases in venous and arterial tone and decreased cardiac output, although this is usually of little hemodynamic consequence in volume-resuscitated patients . Propofol is formulated in a lipid emulsion, and thus triglycerides should be monitored every 3–7 days while the patient receives continuous infusion, and the 1.1 kcal/ml must be accounted for when formulating a nutrition plan .

The propofol infusion syndrome is an adverse reaction characterized by bradycardia and cardiac failure potentially resulting in asystole in the setting of metabolic acidosis, rhabdomyolysis, and hyperkalemia. This condition was originally described in children and led to warnings against propofol in pediatric intensive care .

Fospropofol, a prodrug of propofol, is emerging as a potential alternative agent for sedation in the ICU. It is metabolized in vivo to the active drug propofol, but the parent drug is water-soluble, with a much smaller volume of distribution than propofol . Contamination of the drug, which is a problem in the lipid formulation of propofol, is less of a concern with the water-soluble fospropofol. The onset of action is slightly longer than that of propofol because it must first be metabolized to the active form, but it is still on the order of minutes. It is safe to use in moderate renal insufficiency but has not been studied yet in liver failure.

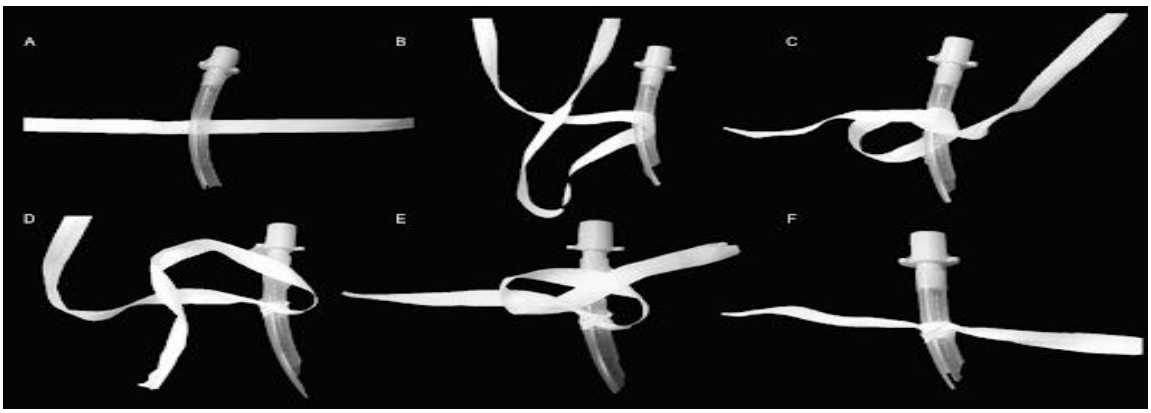
Dexmedetomidine is an a_2 agonist that acts centrally to inhibit norepinephrine release. It has both sedative and analgesic effects, making it a potentially ideal drug for ICU sedation. It does not have the respiratory depressant effects that are present with most other sedative drugs .It allows for a more awake, interactive patient and is associated with less delirium than benzodiazepines. The primary significant side effects of dexmedetomidine infusion are bradycardia and hypotension, which may be mitigated by avoiding a loading dose and initiating a slow infusion rate. In addition, a withdrawal syndrome characterized by agitation, tachycardia, and hypotension can result on discontinuation of a long-term infusion.

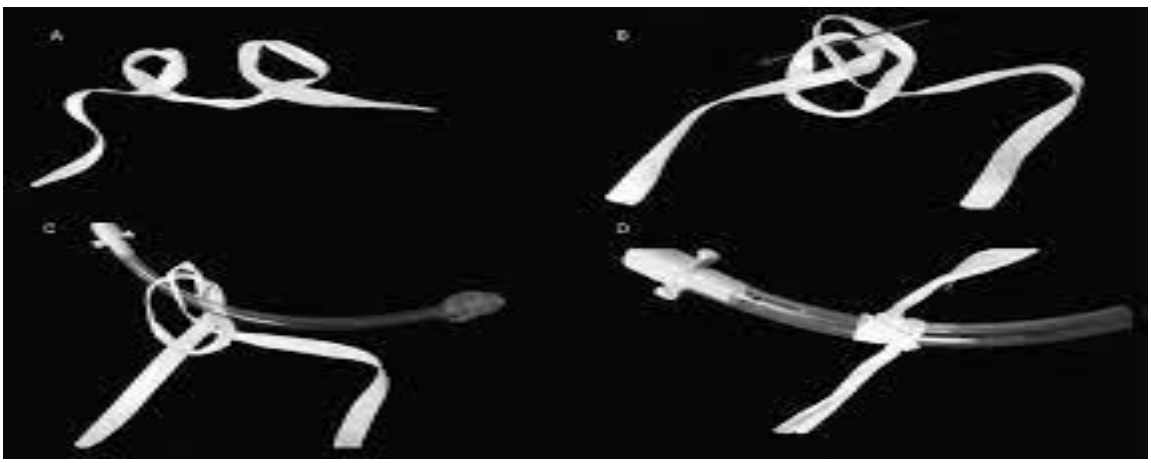
Methods of fixing ET tubes

The accidental dislocation and self extubation of endo tracheal tube is undoubtedly one of the most gruesome challenges faced by an intensive care nurse. Fixing the endotracheal tube correctly will be the simple most solution for this trouble. We can use different methods and items to fix the endo tracheal tube in place. The use

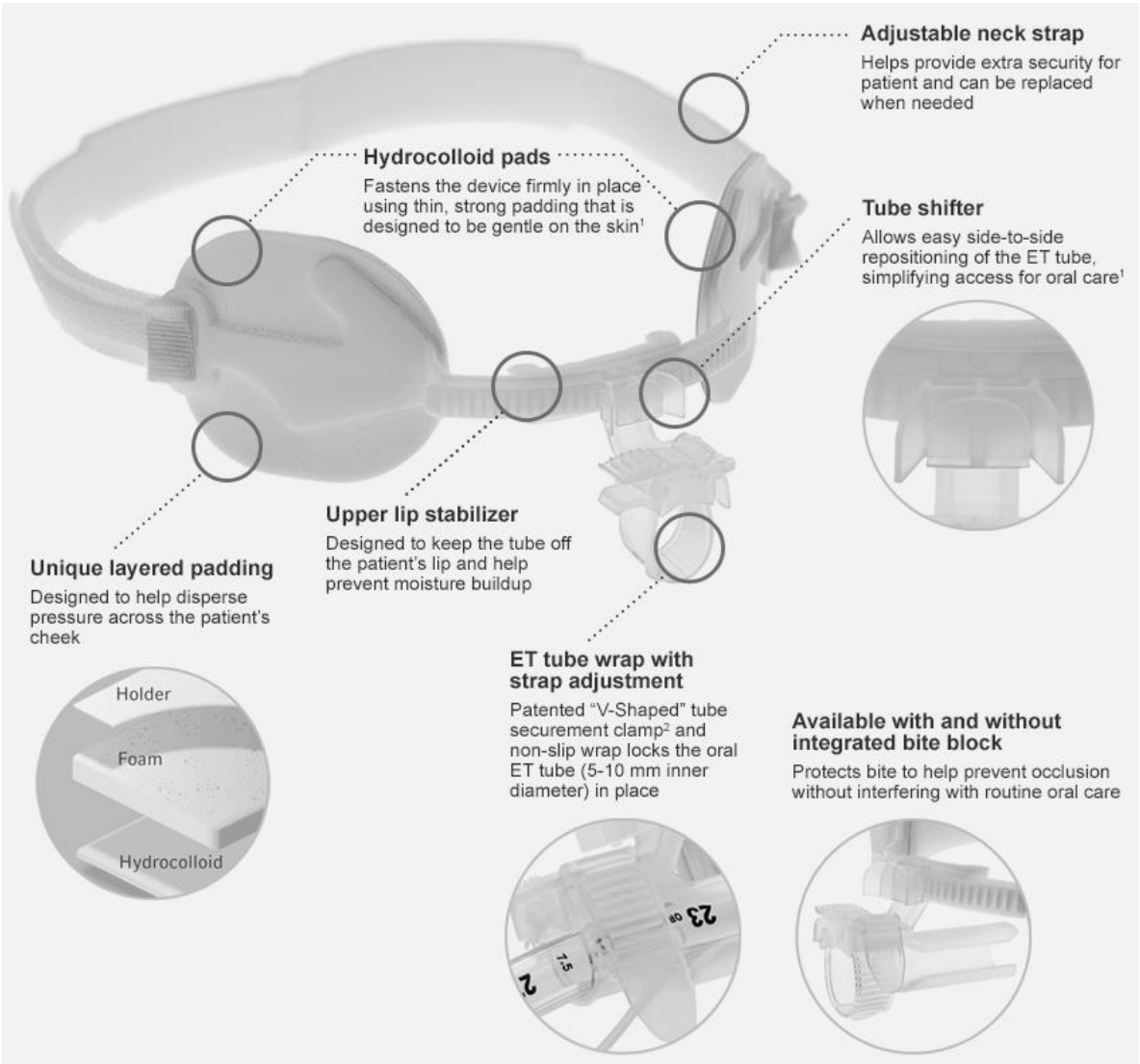
of bandage rolls, adhesive tapes and dedicated ET holders will be of help.

Techniques of ET fixation using bandage rolls or ties





Using Et tube holders

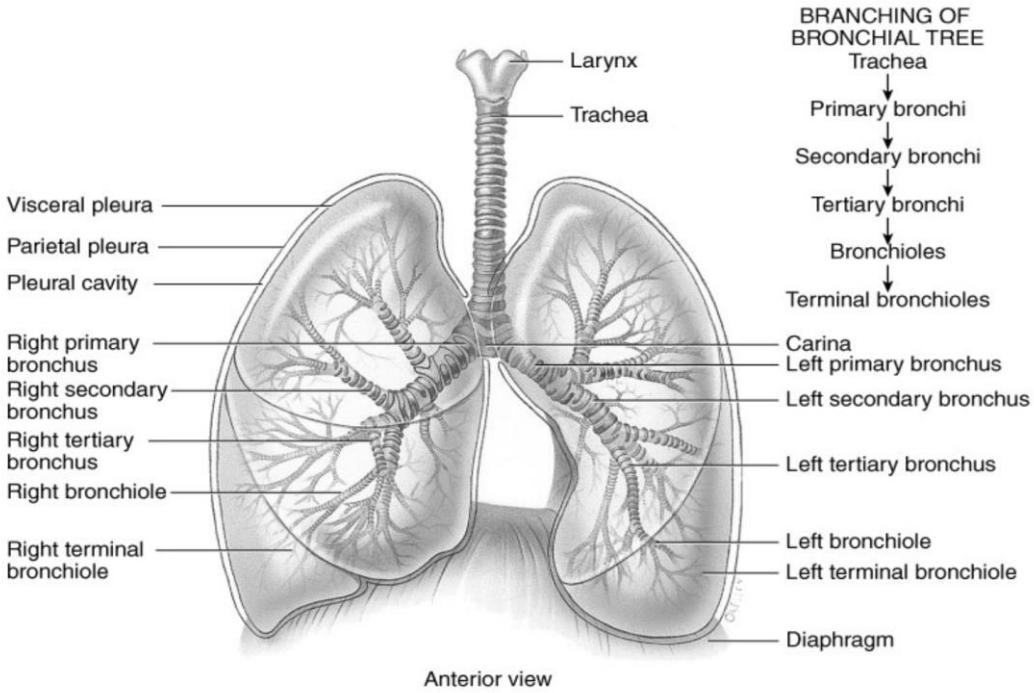


CHAPTER 4

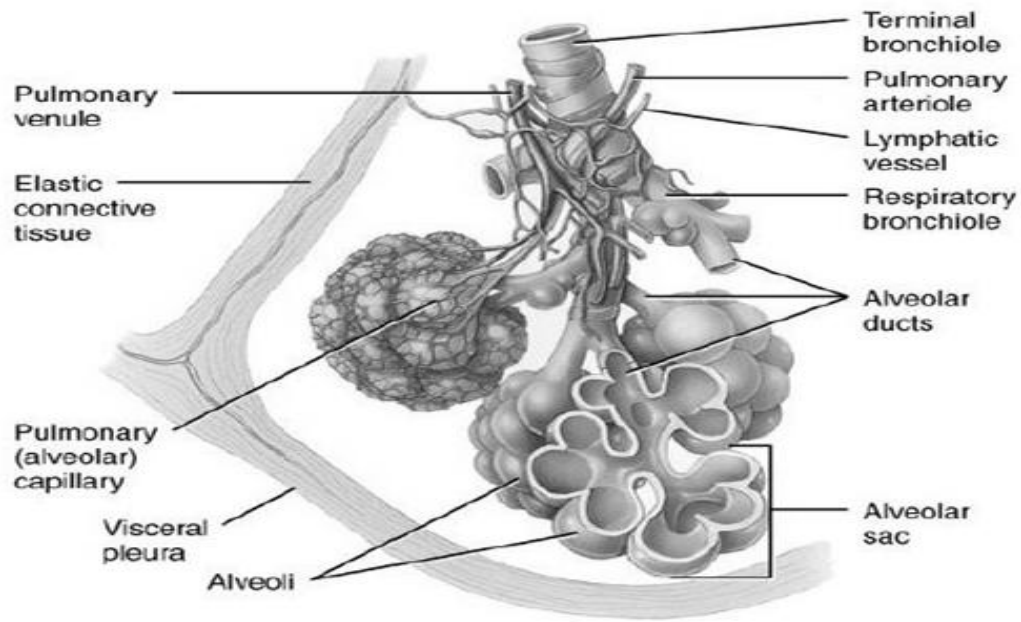
MECHANICS OF VENTILATION

.The Mechanics of ventilation

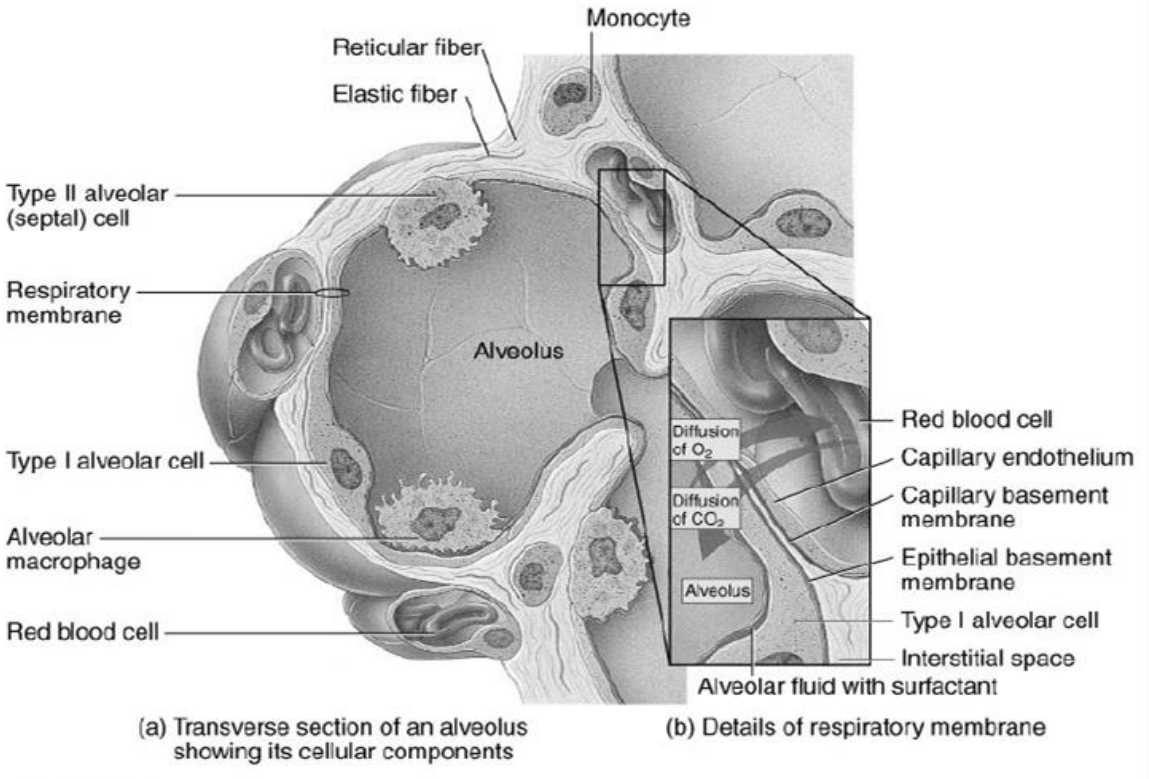
The functional anatomy of lungs



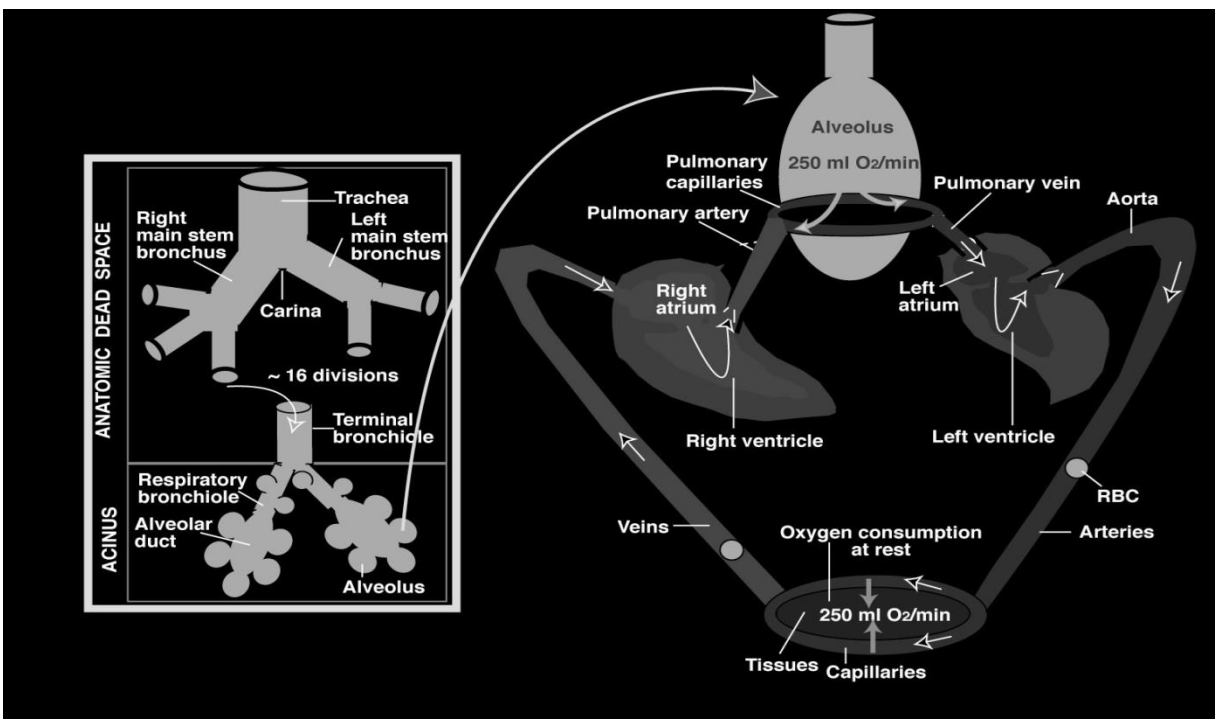
Anterior view



(a) Diagram of a portion of a lobule of the lung



Anatomic Dead Space – area within the respiratory tract in contact with surfaces where gas exchange cannot take place



Structural Changes in Restrictive and Obstructive Lung Disease

Restrictive Lung Diseases – problem with lungs or chest wall, stiff or chest wall deformed making it difficult to inflate the lungs, airways are normal

Interstitial Fibrosis

Chronic inflammation in the space located at the respiratory membrane results in fibrosis, and later scar tissue, eg: Silica exposure, Asbestos exposure

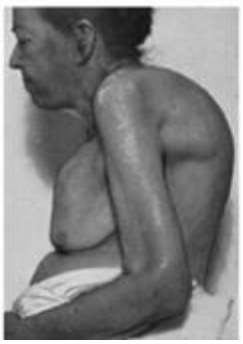
Chest Wall Deformities

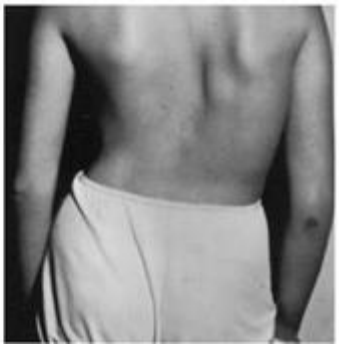
Abnormal curvature of the spine

Kyphosis

Scoliosis

Obesity





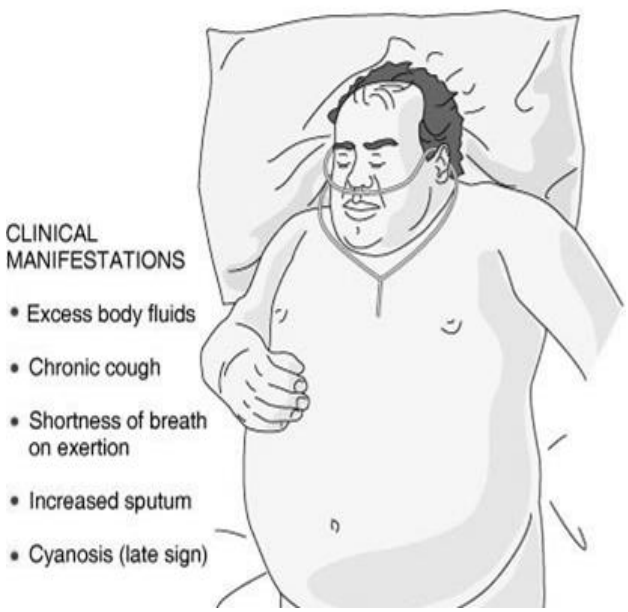
Obstructive Lung Diseases – airways are blocked or resisted, limiting air flow through these passages.

Chronic Bronchitis

Upon chronic insult, goblet cells proliferate at the expense of fewer ciliated cells

Sub mucosal glands hypertrophy. Secretions accumulate and form blockages. With stagnant secretions bacteria multiply, causing inflammation.

Coughing, Expectorating large amounts of mucous, May present as a "blue-bloater" (People with **chronic bronchitis** are sometimes called "blue bloaters" because of their bluish-colored skin and lips. Blue bloaters often take deeper breaths but can't take in the right amount of oxygen).



Asthma

Bronchial smooth muscle cells constrict, edema forms in bronchioles; episodes of wheezing and shortness of breath.

Can be of two types

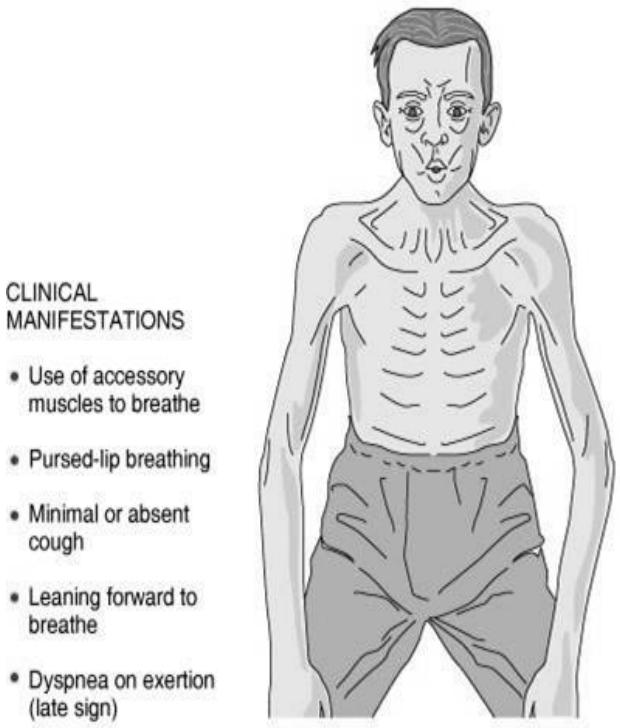
Allergic (extrinsic) Pollen, Molds Animal dander

Nonallergic (intrinsic) Infection, Emotional upset

Treated with bronchodilators and anti-inflammatory drugs.

Emphysema

Result of alveoli losing their elasticity, lungs enlarge but cannot deflate – alveoli merge with a loss in surface area; Extra air trapped in alveoli and large air pockets called bullae; Cause of elastic breakdown not really known. Rare cases where individual inherits a deficiency of alpha1-antitrypsin, result cannot breakdown elastase (released by macrophages in lung) and elastic fibers are destroyed.



To really diagnose should use a biopsy to see if the removed lung tissue floats

Diagnosis typically clinical for irreversible changes seen in pulmonary function tests

Patient may present as a "pink-puffer" (People with emphysema are sometimes called "pink puffers" because they have difficulty catching their breath and their faces redden while gasping for air).

Chronic Obstructive Pulmonary Disease

A combination of emphysema and chronic bronchitis

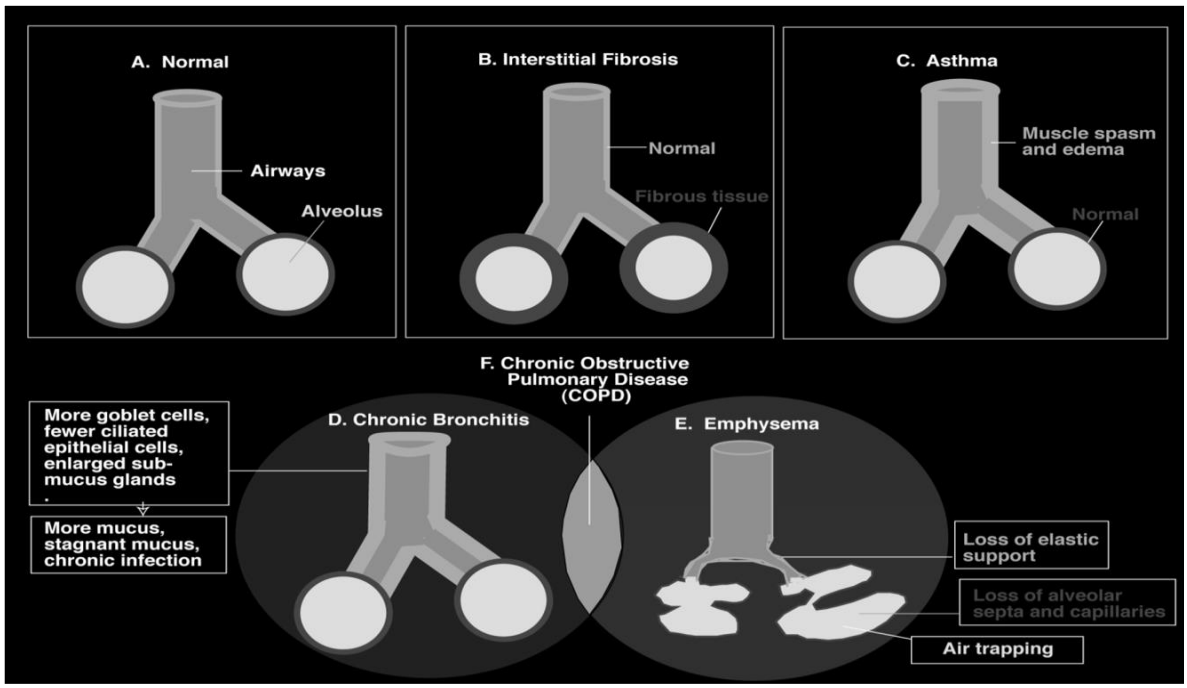
Typically associated with cigarette smoking

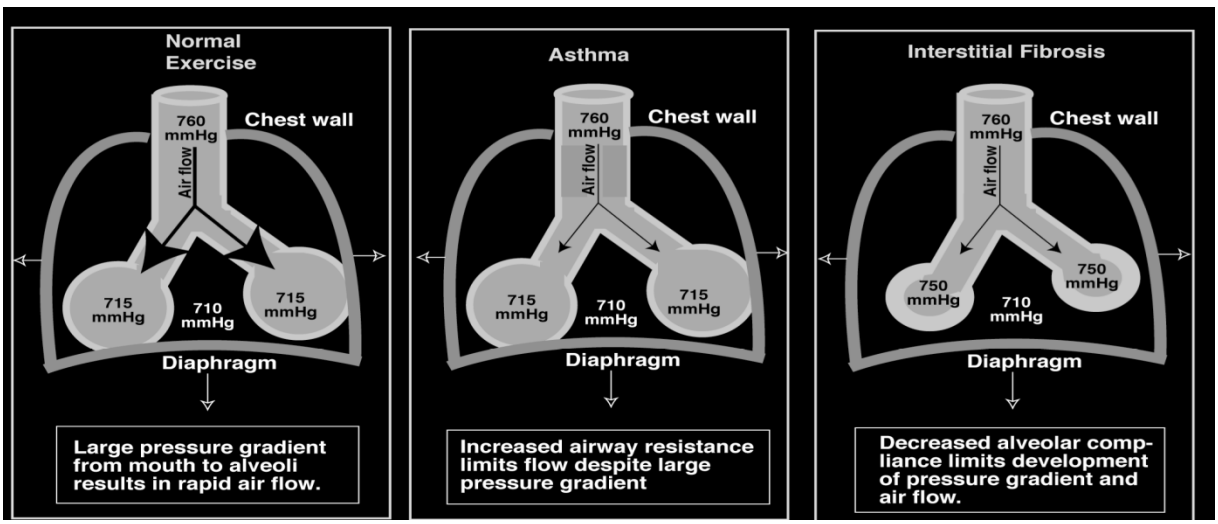
Damage cannot be reversed

Acute inflammation can be treated by Antibiotics, Bronchodilators, Nebulizer to liquefy secretions, **Deep suctioning to remove secretions.**

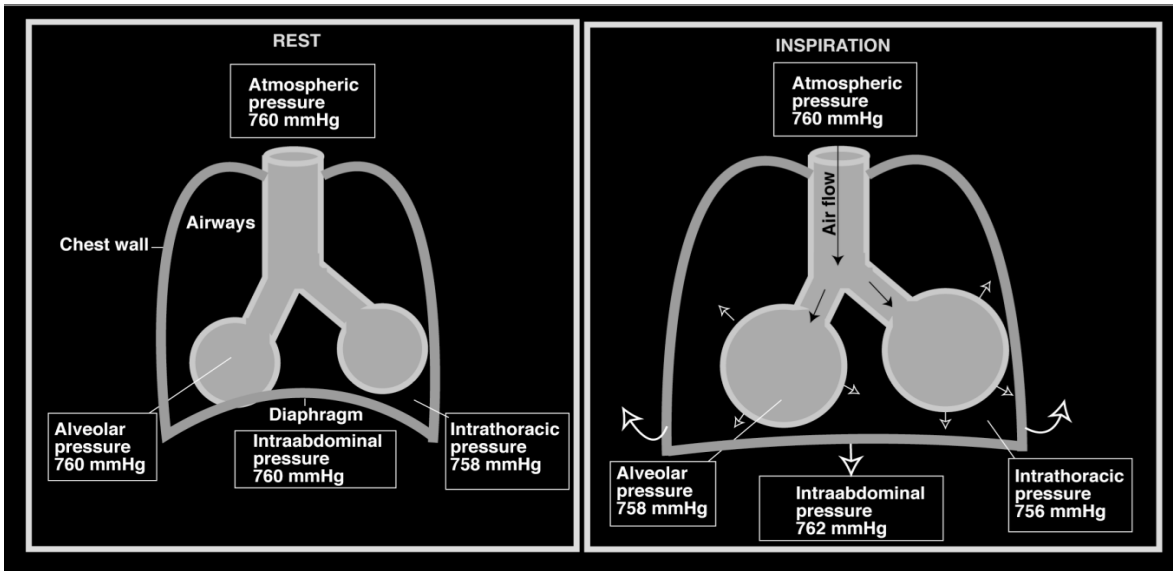
FOR THE EXTRA READERS

The ventilation apparatus and pressures

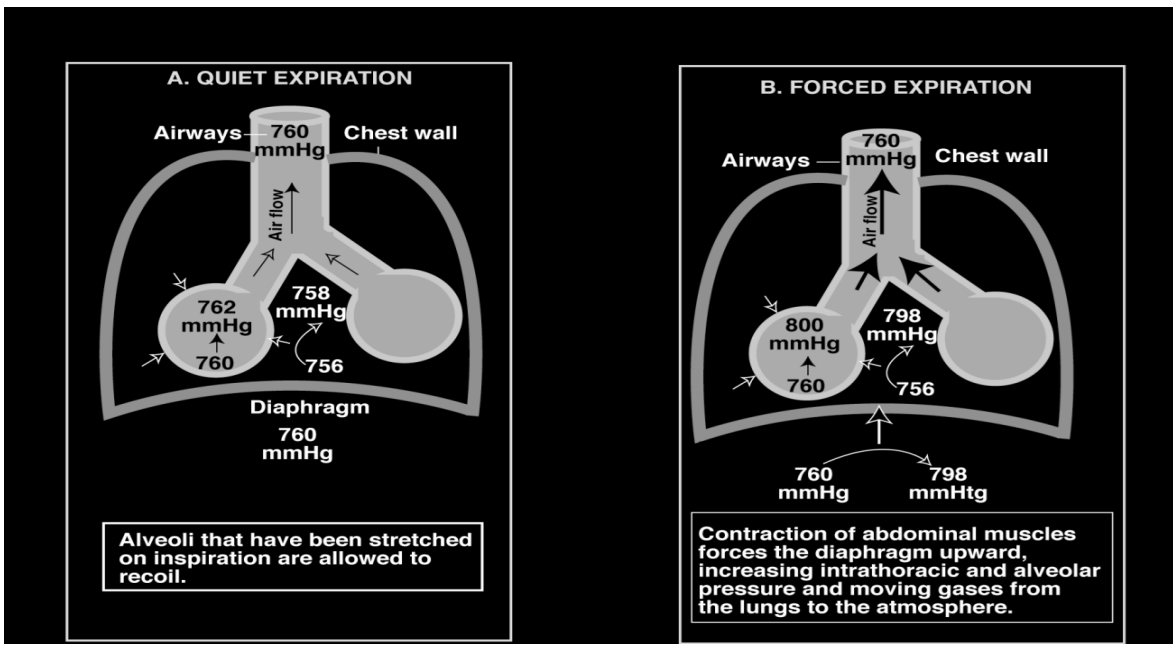




Abnormal inspiration



Expiration is passive



CHAPTER 5

MODES OF VENTILATOR AND TROUBLE SHOOTING SIMPLIFIED.

Positive Pressure ventilators

Based on the use of the invasive artificial airway to deliver ventilation, it is 2 types.

Non-Invasive and invasive Ventilators.

Non Invasive ventilators.

This is mainly used in home-based set up to assist breathing. Examples are CPAP and BiPAP machines.

Invasive Ventilators.

They are 3 Types

1. Pressure cycled.
2. Volume cycled.
3. Time Cycled.
4. Flow cycled.

Here we are discussing Pressure and Volume cycled ventilators because time cycled ventilators are not used widely or used mainly in infants only.

Indications for Positive Pressure Ventilators

Based on arterial blood gas report and clinical symptoms.

To decrease the work of breathing

To Reverse life-threatening hypoxia

To Support Acute Ventilatory Failure

Indications for Positive Pressure Ventilators

1. **based on arterial blood gas report and clinical symptoms.**

a. PaO2 <50 mm of Hg with FiO2 > 60%.

b. PaO2 >50 mm of Hg with p.H < 7.25.

c. Respiratory rate > 35/min.

2. **To decrease the work of breathing.**

Leading factors are.

a. Airway obstruction.

b .Reduced Respiratory compliance.

Lung compliance is the elasticity of the lungs to expand, it will reduce due to pathological conditions like A.R.D.S.

c. High CO2 Production.

2. **To Reverse life-threatening hypoxia.**

Leading factors are.

a. V/Q mismatch: There is a mismatch in ventilation and perfusion ratio, normally the value is one.

b. Shunt.

c. Gas exchange limitation.

3. **To Support Acute Ventilatory Failure.**

Leading factors are.

a. Respiratory Center failure.

b. Mechanical Disruption.

c. Neuromuscular disorders.

d. Reduced Alveolar Ventilation.

e. Pulmonary vascular disruption.

some conditions also lead to respiratory failure, such conditions are thoracic and abdominal surgery, drug overdose, inhalation injury, C.O.P.D, Multiple trauma, Shock, Multi-organ failure, etc.

The physiological effect of Positive pressure ventilators

1.Decreased Cardiac Output.

2.Increased incidence of barotrauma.

3. Decreased urine output. It is due to decreased cardiac output. So adequate intravenous fluid therapy to generate normal stroke volume is essential.

Basic modes of Positive Pressure ventilator

1. **Controlled Mechanical Ventilation or C.M.V.**

It may be pressure controlled or volume-controlled ventilation.

In pressure-controlled ventilation, a preset inspiratory pressure used to deliver required tidal volume. It may range from 5 to 35 cm of H2O to get a target Tidal volume of 6 to 8 ml / Kg of Bodyweight. For example, a patient with 70 kg required a tidal volume of 420 to 560 ml.

The important point is that there is no patient's triggering factor required to start ventilation. The ventilator will deliver a set rate of breath per minute irrespective of the patient's effort of breathing if present.

Example of Pressure Control Ventilation

2. **Assist/control (Triggered) mechanical ventilation**

The ventilator will respond to the patient triggering of breath with either a pre-set tidal volume or a pre-set level of the pressure support level.

The pressure Trigger may set from -1 to -10 cm of H2O. Initially, it may set to generate minimum negative pressure that is -1, gradually it can increase with the prognosis of the patient's condition.

The volume trigger may set from 1 to 7 liter/minute. The patient should generate a minimum amount of tidal volume to initiate a ventilator breath.

In addition, a pre-set back up the rate of breaths will occur. If the patient does not trigger at the required rate. So it is safe to use when compared to Controlled mandatory ventilation mode.

In this ventilation, the ventilator will assist all patient's effort of breathing, if the patient is not taking the breath, the ventilator will deliver the breath.

Pressure Support Ventilation or PSV mode

A pre-set level of inspiratory pressure support is delivered when the patient triggers a breath. The tidal volume of each breath depends upon the lung compliance and respiratory rate and can be adjusted to the level of pressure support to maintain normal range.

Synchronized Intermittent Mandatory Ventilation or SIMV

It is the support mode of the volume-cycled ventilator. The ventilator will deliver a pre-set frequency of breaths but allows spontaneous breaths to be taken in between. Ventilator breaths are synchronized with these spontaneous breaths.

The main difference from assist control ventilation is, it will not assist all breath initiated by the patient but synchronize with the patient's breath at a regular flow rate or deliver the breath if the patient's breath is inadequate in number.

These are basic modes of ventilators, but nowadays the variety of ventilator modes are available according to patient's respiratory needs. For example, a combination of pressure cycled and volume cycled mode and inverse ratio ventilation etc. The primary reason is to improve oxygenation and remove excess CO2.

How to choose a ventilator mode

- Controlled Mechanical Ventilation or C.M.V is used to provide full ventilator support (when the patient is apnoeic).
- Synchronized Intermittent Mandatory ventilation or SIMV is used when the patient is able to initiate some breaths but still requires ventilator assistance at a constant level; to maintain CO2 removal and oxygenation.

Mechanical Ventilator settings

The ventilator settings are used to achieve the required tidal volume. and minute volume along with the normal range of P.a.O2; and PaCO2.

Respiratory rate (breaths/min, f)

- It is usually set at 10 to 15 breaths/min. but may be altered to manipulate the minute volume, P.O2, and P.CO2.

Tidal Volume

It is set at 6 to 8ml / kg of body weight. But recent researches recommend s a tidal volume of 6ml/Kg or even less.

Tidal volume can be altered if there is difficulty in optimizing PO2 and PCO2.

Minute Volume

Target range from 2.5 to 12 Liters/min.

It is the product of tidal volume and Respiratory rate.

Pressure support (cm of H2O)

It ranges from 5 to 35 cm of H2O. It is adjusted according to the target tidal volume required.

Inspiratory: Expiratory Ratio

The normal is 1:2, but may vary from 2:1 to 1:4 in order to increase the time for inspiration in severe airflow limitation or to increase expiratory time in severe COPD.

Trigger or Sensitivity

Volume-based trigger ranging from 1 to 7 Liters/minute.

Pressure based trigger ranging from -1 to -10 cm of H2O.

Initial weaning stage; a minimum trigger needed to set to initiate the ventilator-assisted breath.

Positive end-expiratory pressure or PEEP (cm of H2O)

Usually set between 5 to 10 cm of H2O.The aim of PEEP is to reduce the alveolar collapse during each exhalation and to increase the area of gas exchange with minimum FiO2.

How to connect the mechanical ventilator to a patient

Following things to be ready before connecting with the ventilator.

1. Power supply/battery backup.
2. Ventilator.
3. O2 and air supply.
4. Humidifier.
5. Ventilator Circuit.
6. Catheter Mount
7. E.T. Tube or Tracheostomy tube.
8. AMBU bag- for giving manual breath, if needed.

Before connecting to the ventilator, a self-test run is mandatory to assure the working status of the ventilator. Also, select the required mode and settings for the patient's condition. First make sure uninterrupted power supply, oxygen, and air supply to the ventilator.

Connect the ventilator circuit with the ventilator, humidifier to be attached to the inspiratory limb or tube. Catheters mount to be connected to the Y end tip of the ventilator circuit. Then it connects to the endotracheal or tracheostomy tube. Ensure that the cuff of the endotracheal tube is inflated.

Mechanical Ventilator Troubleshooting

1). High Airway pressure.

Causes.

E.T. Tube obstruction.

Pneumothorax.

Severe Bronchospasm.

A buildup of secretion.

Patient coughing.

Increased peak airway pressure resulting from increased tidal volume or inspiratory time is too short.

Displacement of the tube. Either upward or downward.

Nursing Interventions

If severely compromised; remove from ventilators and manually ventilate.

Perform suction to clear secretion.

If the cause is complete obstruction of E.T, then re-intubate.

Auscultation of lungs for wheezing; reduction in air entry and altered breath sound.

If there is a pneumothorax; immediate insertion of a chest drain or it will cause cardiovascular compromise.

Check blood gas to assess ventilation status.

If necessary administer sedatives as per order.

Review ventilator settings.

2. Low airway pressure

Manifested by sounds of leakage, decreased minute volume, low airway pressure.

Causes

Disconnection.

The major leak from the ventilator.

Burst cuff from E.T. Tube.

A leak from the circuit.

Broncho-Pleural fistula with a massive air leak from the chest drain.

Nursing Interventions

Check the patient's attachment to the ventilator.

Check tubing's connections for leakage.

Check the cuff pressure.

Check inspiratory tidal volume; to assess ventilator delivering adequate volume.

Check levels set for the alarm.

If still persists manually ventilate.

3.Low minute volume

Manifested by.

Low Minute volume alarm.

Audible cuff leak.

Desaturation alarm.

Causes

Disconnection from the ventilator.

Ventilator tube leakage.

Broncho-pleural fistula with a chest drain in situ.

Nursing Intervention

Unless the cause of low minute volume is immediately traced out; manually ventilate the patient.

Check ventilator tubings for leakage.

Review ventilator settings.

Check cuff leakage, auscultate the trachea if necessary.

Monitor air leakage through the chest drain.

4.High minute volume

Manifested by.

High M.V alarm.

Causes.

Patient making the respiratory effort.

Possible ventilator malfunction.

Nursing intervention

Check the cause of tachypnoea such as possible hypoxia or hypercapnia.

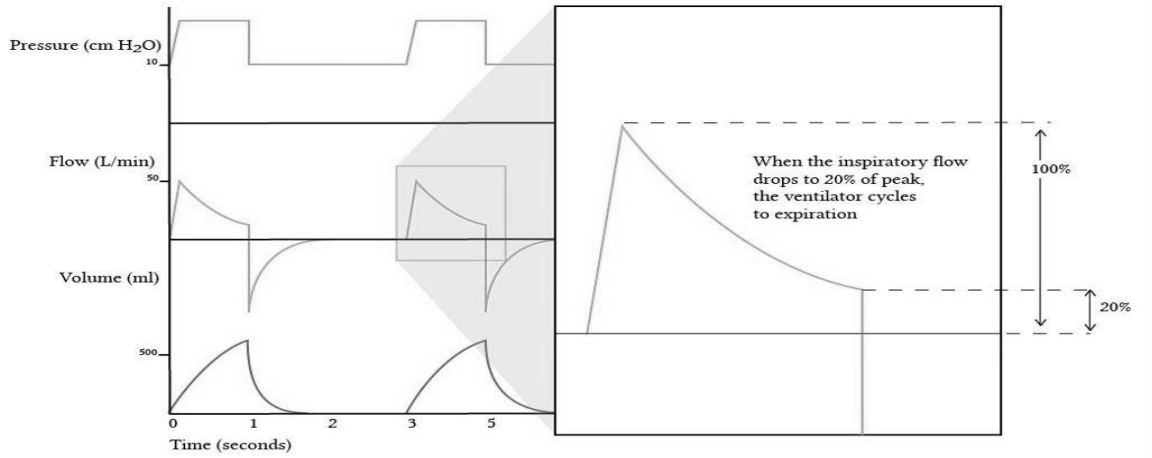
Review Ventilator settings.

So that is all about the basics of mechanical ventilation.

FOR THE EXTRA READERS

Flow cycled ventilation

With flow-cycled ventilation, the ventilator cycles into the expiratory phase once the flow has decreased to a predetermined value during inspiration.



The flow cycling variable can be a fixed flow value in L/min or a percentage fraction of the peak flow rate achieved during inspiration. In some models it is probably something that can be pre-set at an absolute value, eg 5L/min. Some of the ventilators expresses the flow cycling variable as a percentage of peak inspiratory flow and allows a range of settings (between 1% and 70%.).

There are some implications for the flow cycled ventilation. The patients with restrictive lung disease will have poor respiratory compliance and their flow rate will drop rather quickly; as the result their tidal volumes will be lower. In contrast, the patient with emphysematous lung disease may have a very high lung compliance and will get larger tidal volumes with the same settings on the ventilator. Exposure to pressure will be different as well: the higher inspiratory pressure will be more sustained and the waveform will be more "square" with a lower flow cycling setting.

Flow-cycled ventilation has many advantages:

- It is more comfortable for the patient by preventing frustrated expiratory efforts; if the patient needs to terminate a breath and exhale the inspiratory flow ceases and the ventilator cycles to expiration rapidly. With conventional settings,

the inspiratory time is rarely uncomfortably prolonged. This is the main advantage of this method.

- It is limited by changes in lung compliance and airway resistance, which could theoretically prevent inadvertent ventilator-induced lung injury (i.e. with poorly compliant lungs, the ventilator will cycle to expiration rather than continue to apply distending pressure).

There are also some disadvantages:

- Tidal volumes may be poor in patients with poor lung compliance, resulting in inadequate minute volume
- Patient comfort depends on intelligent settings; inappropriately low and inappropriately high settings could result in uncomfortably deep and prolonged inspiration or "double triggering" due to insufficient inspiratory time and tidal volume.

Some commonly found technical alarms in ventilator

XDCR fault

Screen shows the error message "XDCR Error" and an alarm is sounding when the device is powered on. This error means the transducer, a power adapting device, is not working properly.

How to trouble shoot

Connection Problem: Check if all of the tubings and pins connecting to the transducer are securely connected. If any tubes is loose or disconnected, reconnect those pins and tubes.

Incorrect Voltage: Check if the voltage being applied to the transducer is the appropriate voltage level for that transducer.

Faulty Transducer: If all other problems are fixed and the message still appears, the transducer itself may be faulty. In that case replace the transducer with a new one.

Vent INOP

Vent INOP alarm comes when the machine's electronics or valves cease functioning or the ventilator find itself unsafe for functioning. If unattended this can be life threatening to

patient as the ventilator cannot meet the desired breathing requirements of the patients. Disconnect the patient from the ventilator and connect to a new one. Switch off the ventilator. The ventilator ceases operating, the audible alarm sounds continuously and the Vent INOP LED is lit. Press the Silence/Reset button to silence the audible alarm. Inform to your biomedical engineer or the service personnel.

O_2 inlet low

This alarm comes generally when the oxygen inlet pressure drops from a minimum required for the ventilator to maintain the set FiO2. Check the patient for distress and the SpO2. Check the Oxygen inlet pressure sense by the ventilator.

Reconnect and the check the high pressure Oxygen tubing for leak. If the problem persists connect the ventilator to a different oxygen source or AMBU the patient using portable O2 cylinders till the problem is resolved. If the central outlet pressure decreases and there is a loss of oxygen pressure contact the concerned person immediately to re establish the service.

CHAPTER 6

CARE OF PATIENTS ON VENTILATOR & PREVENTING COMPLICATIONS.

There are certain points you must remember

Review communications

Check ventilator settings and modes

Suction appropriately

Assess pain and sedation needs

Prevent hemodynamic instability

Prevent infection

Manage the airway

Meet the patient's nutritional needs

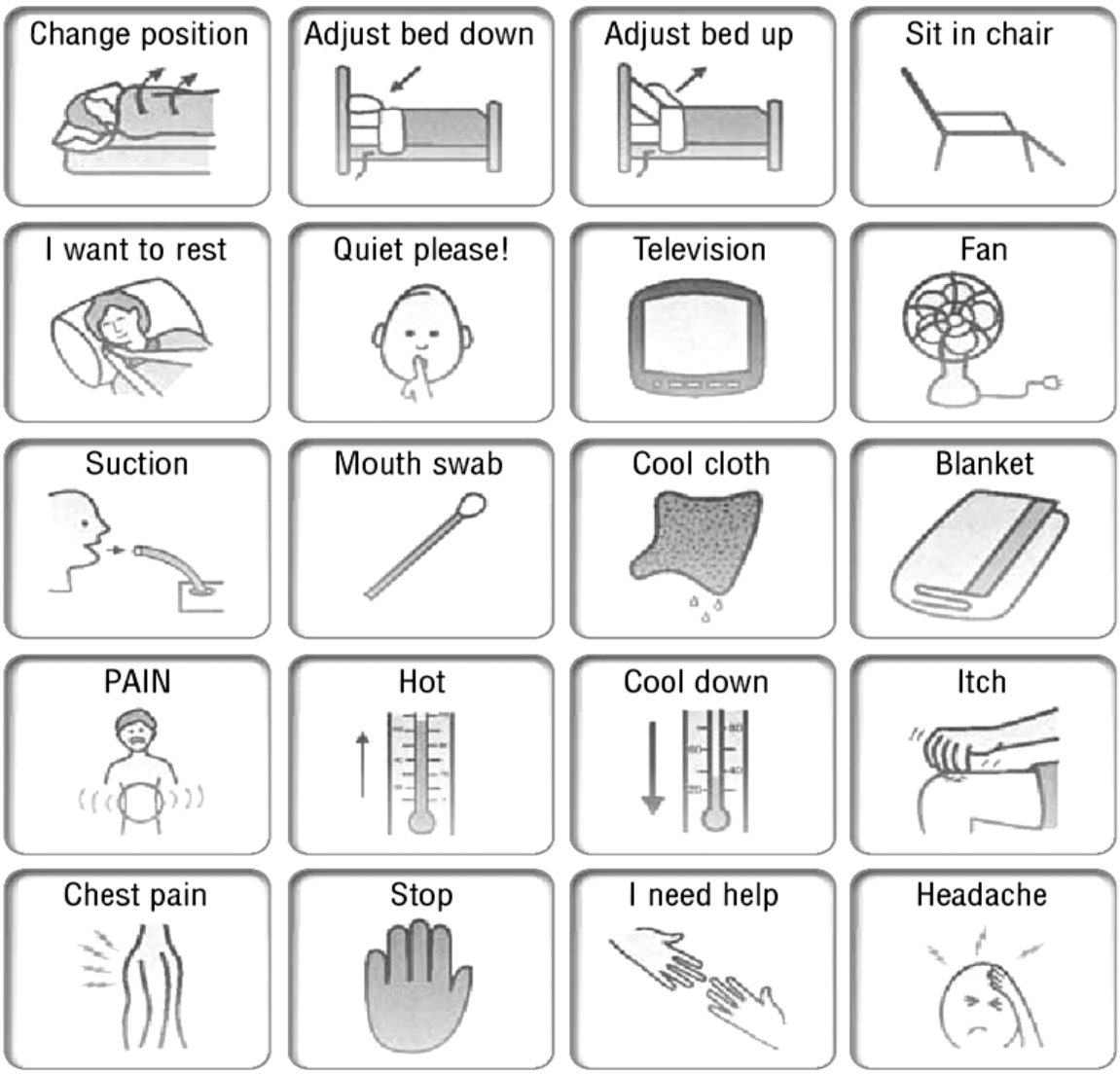
Wean the patient from the ventilator appropriately

Educate the patient and family

Care essential 1: Review communications

Communication among care providers promotes optimal outcomes. For mechanically ventilated patients, care providers may include primary care physicians, pulmonary specialists, hospitalists, respiratory therapists, and nurses.

To make sure you're aware of other team members' communications about the patient, find out the goals of therapy for your patient when obtaining report. Why is the patient on a ventilator? To improve oxygenation? Boost ventilation? Permit sedation? Reverse respiratory muscle fatigue? Why is the patient on your unit? Because the patient has an underlying condition that complicates weaning from the ventilator? What is the do-not-resuscitate status?



Communicating with the patient is essential, too. Provide writing tools or a communication board so the patient can express her needs. Ask simple yes/no questions to which the patient can nod or shake her head.

Care essential 2: Check ventilator settings and modes

When you enter the patient's room, take vital signs, check oxygen saturation, listen to breath sounds, and note changes from previous findings. Also assess the patient's pain and anxiety levels.

Read the patient's order and obtain information about the ventilator. Compare current ventilator settings with the settings prescribed in the order. Familiarize yourself with

ventilator alarms and the actions to take when an alarm sounds. Locate suction equipment and review its use. Look for a bag-valve mask, which should be available for every patient with an artificial airway; be sure you know how to hyperventilate and hyper oxygenate the patient.

Ventilator settings and modes

Generally, ventilators display ordered settings and patient parameters. Check the following settings:

respiratory rate, the number of breaths provided by the ventilator each minute. Manually count the patient's respiratory rate, because the patient may be taking her own breaths at a rate above the ventilator setting.

fraction of inspired oxygen (FiO2), expressed as a percentage (room air is 21%).

tidal volume (TV or VT), the volume of air inhaled with each breath, expressed in milliliters

peak inspiratory pressure (PIP), the pressure needed to provide each breath. Target PIP is below 30 cm H2O. High PIP may indicate a kinked tube, a need for suctioning, bronchospasm, or a lung problem, such as pulmonary edema or pneumothorax.

To find out which ventilation mode or method your patient is receiving, check the ventilator itself or the respiratory flow sheet. The mode depends on patient variables, including the indication for mechanical ventilation.

Modes include those that provide specific amounts of TV during inspiration, such as assist-control (A/C) and synchronized intermittent mandatory ventilation (SIMV); and those that provide a preset level of pressure during inspiration, such as pressure support ventilation (PSV) and airway pressure release ventilation. PSV allows spontaneously breathing patients to take their own amount of TV at their own rate. A/C and continuous mandatory ventilation provide a set TV at a set respiratory rate. SIMV delivers a set

volume at a set rate, but lets patients initiate their own breaths in synchrony with the ventilator.

Some patients may receive adjuvant therapy, such as positive end-expiratory pressure (PEEP). With PEEP, a small amount of continuous pressure (generally from +5 to +10 cm H2O) is added to the airway to increase therapeutic effectiveness. In many cases, PEEP is added to reduce oxygen requirements.

Finally, determine if a capnography monitor is recording the patient's partial pressure of exhaled carbon dioxide (pCO2). Capnography, which reflects ventilation, can detect adverse respiratory events, such as tracheal-tube malpositioning, hypoventilation, and ventilator circuit problems. The capnography waveform should be square; generally, the value should be in the normal pCO2 range of 35 to 45 mm Hg. To better understand your patient's ventilation status, check for trends in waveforms and values rather than focusing solely on single events.

Care essential 3: Suction appropriately

Patients receiving positive-pressure mechanical ventilation have a tracheostomy, endotracheal, or nasotracheal tube. Most initially have an

endotracheal tube; if they stay on the ventilator for many days or weeks, a tracheotomy may be done. Tracheotomy decisions depend on patient specifics.

Although specific airway management guidelines exist, always check your facility's policy and procedure manual. General suctioning recommendations include the following:

- Suction only as needed-not according to a schedule.
- A thorough chest Physio therapy after a mucolytic nebulization followed by suctioning may give the best outcome
- Hyper oxygenate the patient before and after suctioning to help prevent oxygen desaturation.
- Don't instill normal saline solution into the endotracheal tube in an attempt to promote secretion removal.
- Limit suctioning pressure to the lowest level needed to remove secretions.

- Suction for the shortest duration possible.

  If your patient has an endotracheal tube, check for tube slippage into the right mainstem bronchus, as well as inadvertent extubation. Other complications of tracheostomy tubes include tube dislodgment, bleeding, and infection. To identify these complications, assess the tube insertion site, breath sounds, vital signs, and PIP trends. For help in assessing and managing tube complications, consult the respiratory therapist.

  If your patient has a tracheostomy, perform routine cleaning and care according to facility policies and procedures.

Care essential 4: Assess pain and sedation needs

Even though your patient can't verbally express her needs, you'll need to assess her pain level using a reliable scale. Keep in mind that a patient's acknowledgment of pain means pain is present and must be treated. Two scales that help you evaluate your patient's sedation level are the Richmond Agitation Sedation Scale and the Ramsay Sedation Scale.

RASS (Richmond Agitation Sedation Scale)		
4	Combative	Overtly combative, violent, immediate danger to staff
3	Very agitated	Pulls or removes tubes or catheters; Aggressive
2	Agitated	Frequent non purposeful movement, fights ventilator
0	Alert and calm	
-1	Drowsy	Sustained awakening to voice (≥ 10sec)
-2	Light sedation	Briefly awakens with eye contact to voice(<10 sec)
-3	Moderate sedation	Movement or eye opening to voice but no eye contact
-4	Deep sedation	No response to voice but movement or eye opening to physical stimulation
-5	Cannot be aroused	No response to voice or physical stimulation

RAMSAY SEDATION SCALE	
Score	**Level of sedation**
1	Patient is anxious and agitated or restless, or both
2	Patient is co-operative ,oriented and tranquil
3	Patient response to commands only
4	Patient exhibits brisk response to light tactile stimuli or loud auditory stimulus
5	Patient exhibits sluggish response to light tactile stimuli or loud auditory stimulus
6	Patient exhibits no response

Should you restrain an agitated ventilator patient to prevent extubation? Research shows self-extubation can occur despite physical restraints. It's best to treat agitation and anxiety with medication and nonpharmacologic methods, such as communication, touch, presence of family members, music, guided imagery, and distraction.

Care essential 5: Prevent infection

Ventilator-associated pneumonia (VAP) is a major complication of mechanical ventilation. Much research has focused on how best to prevent VAP. The Institute for Healthcare Improvement includes the following components in its best-practices VAP prevention "bundle":

- Keep the head of the bed elevated 30 to 45 degrees at all times, if patient condition allows. Healthcare providers tend to overestimate bed elevation, so gauge it by looking at the bed frame rather than by simply estimating.
- Every day, provide sedation and paralytic "vacations" and assess readiness to extubate, indicated by vital signs and arterial blood gas values within normal ranges as well as the patient taking breaths on her own.
- Provide peptic ulcer disease prophylaxis, as with a histamine-2 blocker such as famotidine.
- Provide deep vein thrombosis prophylaxis, as with an intermittent compression device.
- Perform oral care with chlorhexidine daily.

Other measures that decrease VAP risk include extubating the patient as quickly as possible, performing range-of-motion exercises and patient turning and positioning to prevent the effects of muscle disuse, having the patient sit up when possible to improve gas exchange, and providing appropriate nutrition to prevent a catabolic state. Assess the patient's tolerance when the patient performs an activity by checking vital signs, oxygenation status, and pain and agitation levels.

Keeping bacteria out of oral secretions also reduces VAP risk. Use an endotracheal tube with a suction lumen above the endotracheal cuff to allow continuous suctioning of tracheal secretions that accumulate in the subglottic area. Don't routinely change the ventilator circuit or tubing. Brush the patient's teeth at least twice a day and provide oral moisturizers every 2 to 4 hours.(If the patient is receiving anticoagulation therapy brushing can be substituted by chlorhexidine mouth washes)

Care essential 6: Prevent hemodynamic instability

Monitor the patient's blood pressure every 2 to 4 hours, especially after ventilator settings are changed or adjusted. Mechanical ventilation causes thoracic-cavity pressure to rise on inspiration, which puts pressure on blood vessels and may reduce blood flow to the heart; as a result, blood pressure may drop. To maintain hemodynamic stability, you may need to increase I.V. fluids or administer a drug such as dopamine or norepinephrine, if ordered.

High levels of inspiratory pressure with PSV and PEEP increase the risk of barotrauma and pneumothorax. To detect these complications, assess breath sounds and oxygenation status often. To help prevent these conditions, use the lowest pressure level for ventilator-delivered breaths and adjust the level as tolerated.

Care essential 7: Manage the airway

The cuff on the endotracheal or tracheostomy tube provides airway occlusion. Proper cuff inflation ensures the patient receives the proper ventilator parameters, such as TV and oxygenation. Following hospital policy, inflate the cuff and measure for proper inflation

pressure using the minimal leak technique or minimal occlusive volume.(Minimal occlusive volume (MOV) is one of the four methods used to monitor cuff inflation. This method involves the addition of sufficient air to abolish an air leak on inspiration determined by auscultation over the trachea). These techniques help prevent tracheal irritation and damage caused by high cuff pressure; always practice them with an experienced supervision. Never add air to the cuff without using proper technique.

When performing mouth care, suction oral secretions and brush the patient's teeth, gums, and tongue at least twice a day using

a soft pediatric or adult toothbrush. Use a tonsil suction device if your patient needs more frequent suctioning.

With assistance from an experienced colleague, change the tracheostomy tube or tracheostomy ties and endotracheal tube-securing devices if they become soiled or loose. Incorrect technique could cause accidental extubation.

Care essential 8: Meet the patient's nutritional needs

For optimal outcomes, ventilator patients must be well nourished and should begin taking nutrition early. But like any patient who can't swallow normally, they need an alternative nutrition route. Preferably, they should have feeding tubes with liquid nutrition provided through the gut. If this isn't possible, the healthcare team will consider parenteral nutrition.

Patients with tracheostomy tubes may be able to swallow food. Follow the physician's orders and consult speech and respiratory therapists.

Care essential 9: Wean the patient from the ventilator appropriately

As your patient's indications for mechanical ventilation resolve and he/she's able to take more breaths on his/ her own, the healthcare team will consider removing him/her from the ventilator. Weaning methods may vary by facility and provider preference. Although protocols may be used to guide ventilator withdrawal, the best methods involve teamwork, consistent evaluation of patient parameters, and adjustment based on these changes.

Some patients may need weeks of gradually reduced ventilator assistance before they can be extubated; others can't be weaned at all. Factors that affect ease of weaning include underlying disease processes, such as chronic obstructive pulmonary disease or peripheral vascular disease; medications used to treat anxiety and pain; and nutritional status.

Care essential 10: Educate the patient and family

Seeing a loved one attached to a mechanical ventilator is frightening. To ease distress in the patient and family, teach them why mechanical ventilation is needed and emphasize the positive outcomes it can provide. Each time you enter the patient's room, explain what you're doing. Reinforce the need and reason for multiple assessments and procedures, such as laboratory tests and X-rays. Communicate desired outcomes and progression toward outcomes so the patient and family can actively participate in the plan of care.

Caring for a patient on mechanical ventilation requires teamwork, knowledge of care goals, and interventions based on best practices, patient needs, and response to therapy. Mechanical ventilation has become a common treatment, and nurses must be knowledgeable and confident when caring for ventilator patients.

FOR THE EXTRA READERS

Patient-Ventilator Associated Complications

Airway dislodgment or disconnection

Circuit leaks

Tracheal and Oral Injury

Inadequate humidity

Excessive rain out in ventilator tubing

Airway obstructions

Pulmonary Complications

Ventilator-Induced Lung Injury (VILI)

Volutrauma

Barotrauma

Biotrauma

Atelectrauma

Oxygen Toxicity

Ventilator-Associated Pneumonia (VAP)

VAP is one of the most serious complications of invasive ventilation. Endotracheal intubation is the major risk factor for ventilator-associated pneumonia. Endotracheal intubation breaches airway defenses, impairs cough and mucociliary clearance, and facilitates micro aspiration of bacteria-laden secretions that pool above the inflated endotracheal tube cuff. In addition, bacteria form a biofilm on and within the endotracheal tube that protects them from antibiotics and host defenses. The highest risk of VAP occurs during the first 10 days after intubation. Ventilator-associated pneumonia occurs in 9 to 27% of mechanically ventilated patients. The most important pathogens causing VAP are Pseudomonas aeruginosa, methicillin-sensitive Staphylococcus aureus, and methicillin-resistant S.aureus (MRSA). Pneumonia in critically ill, mechanically ventilated patients more typically causes fever and increased respiratory rate or heart rate or changes in respiratory parameters, such as an increase in purulent secretions or worsening hypoxemia can be suggestive of VAP. There are specific protocols formulated to prevent VAP.

Ventilation Care Bundle

- Head of bed elevated at least 30 degrees/upright positioning
- Daily sedation breaks with assessment to wean/extubate
- Stress ulcer prophylaxis
- Deep vein thrombosis (DVT) prophylaxis
- Thorough regular teeth brushing with subglottic suctioning

Interventions for VAP	Checkers
Direct elements that decrease infection	
Hand hygiene and Aspiration prophylaxis	Nurse
Head of bed elevation 30-45^0	Nurse
Adequate ET cuff pressure	Respiratory therapist/ trained nurse
Oral cavity secretion clearance before changing position	Nurse
Oral care with chlorhexidine every 8 hour	Nurse
Elements that decrease contamination to respiratory tract devices	
High level sterilization and correct storage of devices	Nurse
Moisten the devices with sterile water	Respiratory therapist/ trained nurse
Indirect elements that decreases infection.	
Daily sedation vacation and assessment of readiness of extubation	Doctor
Peptic ulcer prophylactic drugs	Doctor
Deep vein thrombosis prevention	Doctors/Nurses
Indication for intubation	Doctor

Wash your hands. It's the most important key. Do you know what the five moments of hand hygiene? Clubbing this practice with Ventilator care bundle you can save lots of lives.

There are five moments of hand hygiene

Moment 1 - before touching a patient.

Moment 2 - before a procedure.

Moment 3 - after a procedure or body fluid exposure risk.

Moment 4 - after touching a patient.

Moment 5 - after touching a patient's surroundings.

Cardiovascular and Renal Complications

Reduced venous return, cardiac output and hypotension

Decline in urine output

Gastrointestinal and Nutritional Complications

Gastritis and ulcer formation

Malnutrition

Neuromuscular and Psychological Complications

ICU acquired weakness, DVT and pressure ulcers Sleep deprivation, sedation, delirium, depression

The Ventilator induced Lung Injury (VILI)

The delivery of positive pressure to the airways during mechanical ventilation can potentially cause the lungs to be damaged in varying circumstances;

Volutrauma : Alveolar over-distension

Barotrauma : High to excessive alveolar pressures

Biotrauma :Inflammatory mediators

Atelectrauma : Repeated recruitment and collapse

Oxygen Toxicity : High FiO2

VILI may occur in previously healthy lungs or aggravate pre-existing conditions such as ALI, ARDS.Approximately 25% of patients who are Mechanical Ventilation develop VILI.

Volutrauma and Barotrauma

Damage to the lung occurs as a result of repeated stretching (over- distention) of alveolar structures and excessive transpulmonary pressures to the point of rupture.

As a result, air leaks into the plural space resulting in conditions including pneumothorax, pneumomediastinum and subcutaneous emphysema.

Volutrauma = transpulmonary pressures >30 to 35 cm H2O.

Barotrauma = increasing transpulmonary pressures >50cm H20.

Biotrauma

Widespread alveolar damage may lead to an increase in inflammatory cytokines in the lungs, resulting in VILI.Systemic cytokine release with bacterial translocation are involved in the systemic inflammatory response (SIRS), potentially leading to multiple organ dysfunction (MODS), increasing mortality.

Atelectrauma

Recurrent alveolar re-opening and collapsing of the under-recruited alveoli during ventilation causes injury affecting surfactant functioning, collapse of the dependant portions of the lung and regional hypoxia .

Oxygen toxicity

Oxygen concentrations nearing 100% are known to cause oxidant injuries in the airways. Increased reactive oxygen species (ROS) lead to inflammation, secondary
tissue injury, depletion of cellular antioxidant defences and cell death

Lung Protective Ventilation Strategies

Volutrauma and Barotrauma are the most common causes of ventilated-associated lung injuries (VALI), resulting from tidal volume settings that generate high or excessively high pressures.

Lung Protective Ventilation Strategies

Aim to Prevent Injury	**Variable**	**ARDS net Protocol Protective Ventilation Strategies**
Recruit alveoli without volutrauma	Tidal Volume	≤ 6ml/kg of predicted body weight
Recruit alveoli without barotrauma	Plateau Pressure	≤ 30 cm H20

Biotrauma	Set Vт and target Plateau Pressures	As above, use of neuromuscular blockers may help
Volutraum & barotrauma	Ventilation Rate Ratio of breath Duration (Insp/Exp)	6-35bpm, adjusted to achieve an arterial pH 7.3 to 7.45 1:1 to 1:3
Atelectrauma, shearing Injury	Positive-End Expiratory Pressure (PEEP)	≥ 8 cm H_2O , PaO_2 55-80 mm Hg, open lung ventilation strategies
Oxygen Toxicity	Oxygenation goal	PaO_2 55-80 mm Hg, SpO_2 88-95%, Higher PEEP, recruitment strategies

P-SILI

Patient self-inflicted lung injury (P-SILI) is a controversial, emerging concept, whereby intense inspiratory efforts by spontaneously breathing patients – whether assisted or unassisted – may exacerbate lung injury. Concerns about P-SILI must be balanced against the harms from prolonged controlled ventilation, over-sedation, and paralysis, when allowing patients to breathe spontaneously.

P-SILI may occur in spontaneously breathing patients receiving:

Assisted ventilation via an endotracheal tube

Non-invasive ventilation (NIV)

No ventilatory support

P-SILI may result from intense inspiratory effort, resulting in:

Swings in transpulmonary pressure (i.e. lung stress) causing the inflation of big volumes in an aerated compartment markedly reduced by the disease-induced aeration loss.

Abnormal increases in transvascular pressure, favouring negative-pressure pulmonary edema

An intra-tidal shift of gas between different lung zones, generated by different transmission of muscular force (i.e. pendelluft, Pendelluft phenomenon is defined as **the displacement of gas from a more recruited nondependent (ND) lung region to a less recruited dependent (D) lung region**. This phenomenon may cause lung injury)

Diaphragm injury

Increased lung inflammation

P-SILI may be more likely if:

Vigorous spontaneous breathing efforts

Severe lung disease (e.g. ARDS patients with a PaO2/FiO2 ratio below 200 mmHg may be an "at risk' group)

How to Calculate Predicted Body Weight

Tidal volume (V_T) is the volume of the patient's breath, measured in mls. Measuring the patient's height is a must to calculate a patient's predicted body weight (PBW).

Demi-span

This is measured with the arm outstretched straight, palm forwards, from the base of the middle/ring finger to the sternal notch using a non-stretch tape measure

Females: Height in cm = (1.35 x demi-span cm) + 60.1

Males: Height in cm = (1.4 x demi-span cm) + 57.8

Other formulations for calculation of ideal body weight (IBW)

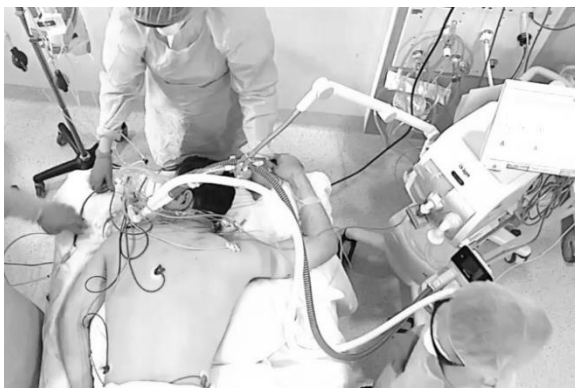
Male: 50.0Kg+2.3 Kg for every inch over 5 feet.

Female: 45.5 Kg+2.3Kg for every inch over 5 feet.

For obese patients

Adjusted body weight= IBW+0.4 (Actual body weight-IBW).

Importance of prone ventilation



Prone ventilation refers to the delivery of mechanical ventilation with the patient lying in the prone position. **Prone ventilation is NOT considered a mode of mechanical ventilation**. Volume-controlled and pressure-controlled modes of ventilation are the typical modes of ventilation that are delivered in the prone position. Other modes of ventilation including high frequency ventilation and other methods of improving gas exchange (eg, extracorporeal membrane oxygenation [ECMO]) are not generally administered in the prone position but may be used in conjunction with prone positioning.

The prone position alters the mechanics and physiology of gas exchange to result consistently in improved oxygenation. Prone positioning improves gas exchange by reducing the ventral-dorsal trans pulmonary pressure difference, reducing dorsal lung compression, and improving lung perfusion.

Studies have consistently shown that in most patients with ARDS (up to 70 percent), prone ventilation increases PaO_2 allowing a reduction in the FiO_2 . Among patients whose oxygenation improves during prone ventilation, some continue to have improved oxygenation for hours after they return to the supine position and many improve each time prone ventilation is repeated.

Indications

Severe ARDS with refractory hypoxemia. Other cases include but not researched extensively are

Patients with diffuse pulmonary edema and dependent alveolar collapse

Patients with elevated intra-abdominal pressure appear more likely to increase their PaO_2 during prone ventilation than patients with normal intraabdominal pressure.

Contra indications

Absolute contraindications to prone ventilation include spinal instability, patients at risk of spinal instability (eg, rheumatoid arthritis), unstable fractures (especially facial and pelvic), anterior burns, chest tubes, and open wounds, shock, pregnancy, recent tracheal surgery, and raised intracranial pressure

Procedure of prone ventilation

Timing of initiation - After a 12 to 24 hour stabilization period of supine ventilation, maintain a low threshold for initiating prone ventilation early (up to 36 hours). Some studies are showing 2-8 hours a day or continuous prone up to 18 hours will be beneficial.

Modes and settings on ventilator- Volume-controlled and pressure-controlled modes of ventilation are the typical modes of ventilation that are delivered in the prone position. A low tidal volume and optimization of PEEP is also recommended.

Positioning-there is no standard procedure to position the patient. The staff who performs this procedure must be ready to tackle the possible respiratory and hemodynamic incompatibility. Some uses the log roll technique to turn the patient from supine to prone. Considering the security of the artificial airway is vital.

Monitoring-There is no additional monitoring needed for the patient even though the nurse must be more cautious for hemodynamic and respiratory compromise.

Nutrition- Enteral nutrition by means of a Ryle's tube is advocated in these cases, even thought the risk of emesis must be noticed.

Sedation- Most of the patients in prone position needs increased sedation and sometimes neuromuscular blockade.

Skin integrity- The nurse should be aware of a possible issue with skin integrity as the patient is lying on his abdomen for a longer period of time. Changing the arm and head position two hourly and use of pressure relievers on pressure points may help to reduce the risk.

CHAPTER 7

INTRODUCTION TO VENTILATOR WAVEFORMS

Ventilator waveforms are graphical descriptions of how a breath is delivered to a patient. These include three scalars (flow versus time, volume versus time, and pressure versus time) and two loops (pressure-volume and flow-volume).

Thorough understanding of both scalars and loops, and their characteristic appearances, is essential to being able to evaluate a patient's respiratory mechanics and interaction with the ventilator.

Basic things to understand

Before reviewing the graphics associated with mechanical ventilation, it is important to understand the concepts of how ventilators can deliver breaths. A mechanical breath is classified based on three main variables-**how the breath starts, how the breath is delivered by the machine, and how the breath is stopped**. A breath can be started by either the patient (referred to as a supported or assisted breath) or by the machine (referred to as a controlled breath). This variable is also referred to as **the trigger**. After the breath is started, **the gas is delivered to the patient in a set pattern that is sustained throughout the course of inspiration**. This is also referred to as **the target variable**. The two main **target variables** are either a **specific inspiratory flow rate or a pressure goal**. The delivery of the **breath is stopped when a certain amount of time has elapsed, a goal amount of volume has been inspired, or the ventilator senses a**

decrease in the flow taken in by the patient. This is the **cycle variable**. Any combination of the above variables can define the type of breath delivered by the machine.

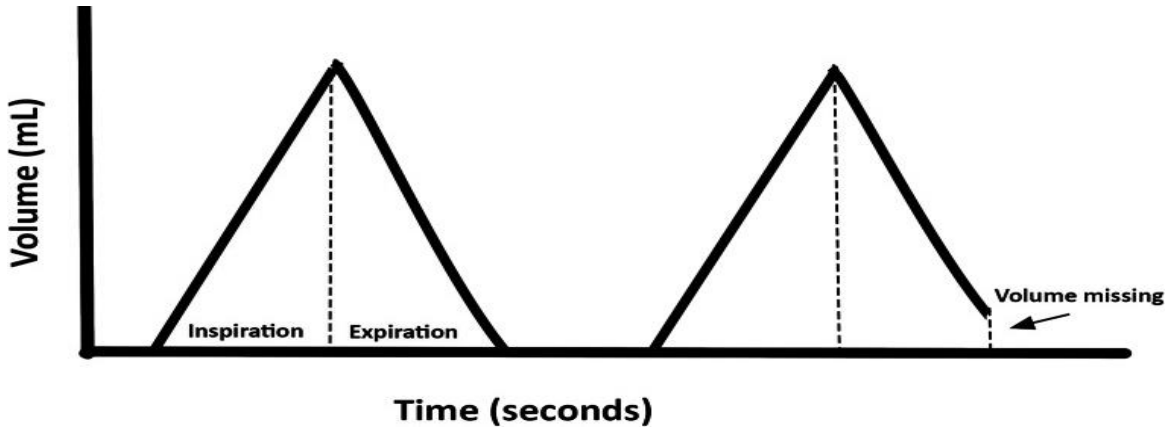
The Scalars or scalar waveforms

Now that we have reviewed the basic ways breaths are delivered from a ventilator, it is important to understand how those breaths are represented in graphical form. The first of these graphics are termed scalars. Scalars on conventional mechanical ventilators are representations of specific respiratory parameters over time. The three scalars commonly utilized are volume, pressure, or flow plotted on the vertical y-axis against time plotted on the horizontal x-axis. Pressure and flow are measured values, while the volume of each breath is a calculated value. Each scalar represents the entire breath from the beginning of inspiration to the end of expiration. Most ventilators have these three scalars displayed on the main screen.

For the extra readers

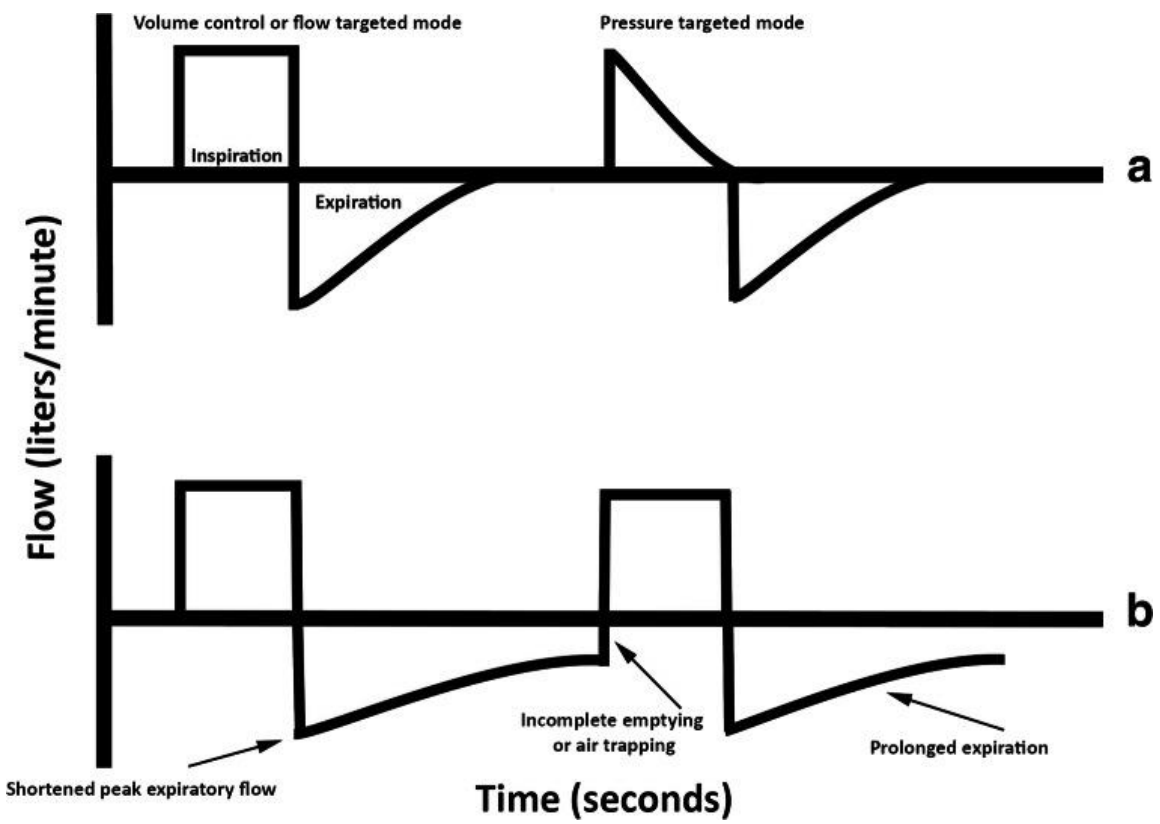
Volume versus Time Scalar

The volume versus time scalar is the graphical representation of the amount of gas delivered into the lungs by the ventilator over time. It is calculated from the measurement of flow. The **upslope** is the **inspiratory volume** and the **downslope** is the **expiratory volume** .Inspiratory and expiratory volumes should be similar, and differences can indicate air leaks in the system or intrinsic positive end-expiratory pressure (i.e. auto-PEEP or air trapping). This is shown when the part of the curve representing expiration decreases as expected but plateaus and never reaches the baseline of zero volume before the next breath. In addition to showing potential air leaks or trapping, the volume versus time scalar can be used to evaluate the volume of a patient's spontaneous breath and the effect adjustment of the ventilator settings may have on tidal volume.



Flow versus Time Scalar

Gas flow in between the patient and the ventilator is represented by the flow versus time scalar. **Inspiratory flow is a positive value** on the graph, whereas **expiratory flow is a negative value**. The **area under the curve represents the volume moved during the phases of breathing**. The shape of the inspiratory limb of the curve depends on the mode of ventilation. In **pressure-targeted modes**, the peak inspiratory pressure (PIP) and inspiratory time are set and flow is variable. At the beginning of the breath, flow is delivered at a high rate but then tapers off over the



course of inspiration, resulting in a **decelerating shape of the curve**. **Pressure-supported modes** may also have a flow pattern that is **decelerating or sinusoidal in shape**. In **volume control or flow-targeted modes**, the tidal volume, inspiratory time, and inspiratory flow are set resulting in a **constant flow or square shape to the flow scalar**. In **volume control modes, flow is constant until the goal tidal volume is achieved, resulting in a constant rise in the pressure and a higher PIP, but a lower mean airway pressure**. If the goal is to minimize mean airway pressure we may choose this mode of ventilation for this reason. In **pressure control modes, since a specific PIP is delivered for a period of time, the overall PIP is lower but the mean airway pressure is higher**. This may be beneficial in situations with increased airway resistance

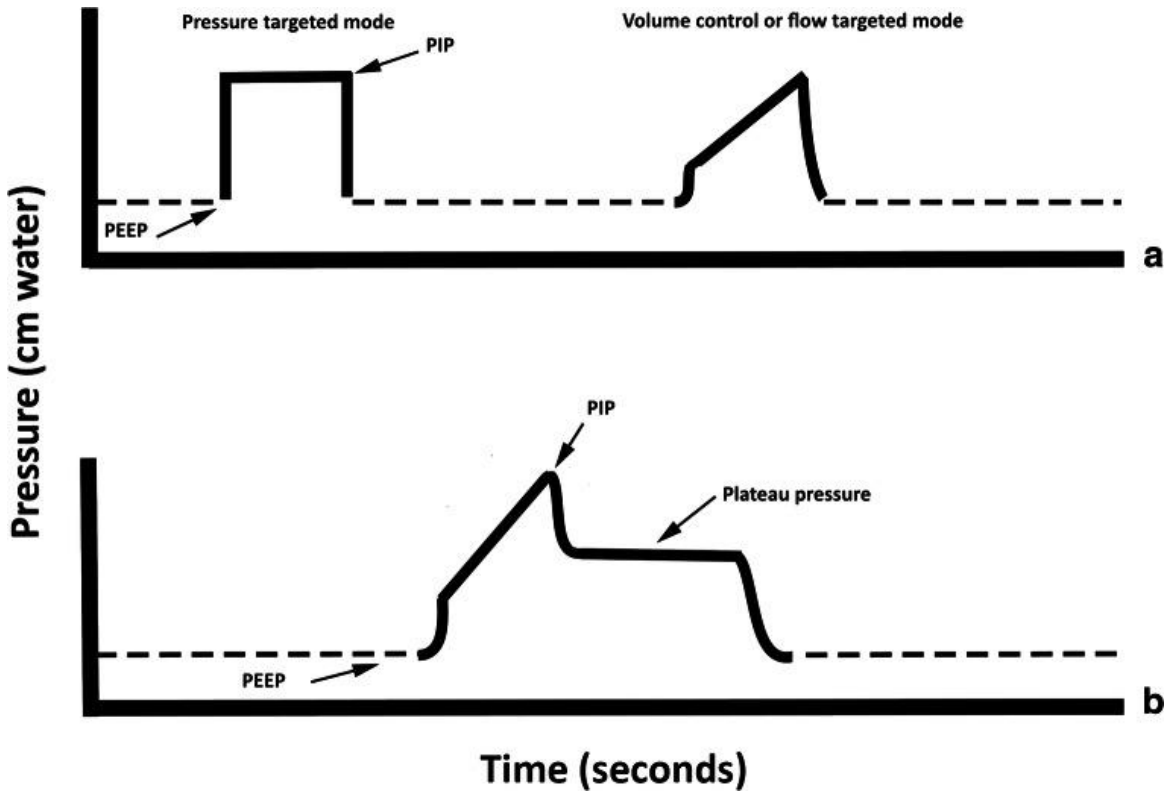
In addition to helping identify the mode of ventilation, the flow versus time scalar can provide useful **information about a patient's exhalation**. **The shape of the expiratory limb** of the curve is **affected by the resistance to air flow and the compliance of the lung**. In instances of **higher airway resistance from some obstructive process**, the flow scalar will show a **decreased peak expiratory flow and a prolonged time for the expiratory curve to return to a baseline of zero flow.** All of this represents that the resistance to airflow is limiting how fast and how much volume the lung empties. Conversely, when compliance is decreased, the peak expiratory flow rate increases.

Air trapping or auto-PEEP (also referred to as intrinsic PEEP) can also be identified on the flow scalar. Under normal conditions, the expiratory limb of the curve returns to a baseline of zero flow prior to the next breath being initiated. However, if expiration of air is still ongoing when inspiration starts, then the lungs are not emptying completely and air trapping occurs. This is shown in the scalar when **the expiratory limb does not return to baseline before the new breath starts**.

Pressure versus Time Scalar

The pressure scalar provides good information about airway compliance. It **represents the pressure in the airway as a function of time**. This can be a fixed or variable amount

depending on the mode of ventilation . During **pressure control** ventilation, the **pressure delivered is constant and the pressure scalar is square-shaped**. In **volume control ventilation**, the pressure scalar is **ascending due to the rise in pressure with a constant flow pattern** . In the typical graphic displayed on the ventilator, **the baseline pressure indicates the PEEP** and the **maximum pressure at the end of the curve indicates the PIP**.



In addition to the PIP and the PEEP, the pressure versus time scalar can show a few other pressure measurements when specific ventilator maneuvers are done. The first is the **plateau pressure (P_{plat})**. This is the **pressure in the airway** under static conditions, or **when there is no air flow**. PIP is mathematically calculated by **adding** the **pressure created by airway resistance**, the **pressure related to the lung's compliance**, and **the total PEEP**.

When there is no flow in the system, the pressure due to airway resistance drops to zero, and the resultant value is **the P_{plat}**. This value is thought to represent the **pressures at the alveolar level**, and is **obtained by doing an inspiratory hold** on the ventilator. It is **best measured in a volume control mode** as compared to a pressure control mode. Volume-targeted modes use constant flow and therefore have a larger pressure differential between PIP and P_{plat} when an inspiratory hold maneuver is completed and flow abates. In contrast, during pressure-targeted ventilation, the descending flow pattern means flow is nearing zero during inspiration, allowing for less of a pressure drop when inspiration is held. Regardless of mode the P_{plat} is measured in, **a large difference between the PIP and the P_{plat} indicates high resistance in the airway as can exemplified in severe bronchospasm**. Use of a P_{plat} target less than 30cm of H_2O in adults with acute respiratory distress syndrome (ARDS), along with a low tidal volume strategy, has been shown to be beneficial for improved outcomes and less barotrauma.

An additional pressure measurement that can be obtained from the pressure versus time scalar is the intrinsic PEEP. This is obtained by completing an expiratory hold maneuver on the ventilator.

Loops

The next ventilator graphics that are important to understand are the loops. These graphics are one of the two variables, either pressure or flow, plotted against the volume during a breath. Each loop consists of an inspiratory and expiratory curve and allows for evaluation of respiratory mechanics. There are two loops-the pressure-volume curve and the flow-volume curve.

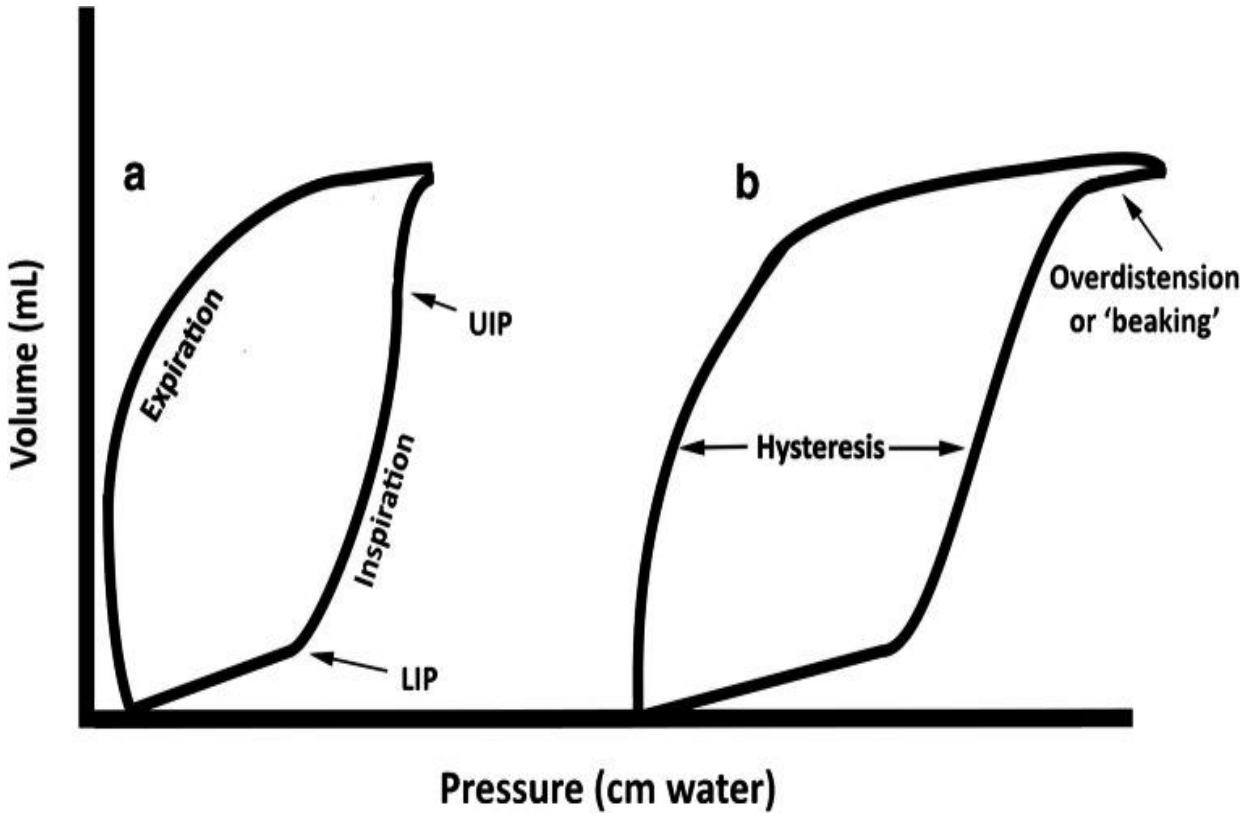
Pressure-Volume Curve

The pressure-volume loop shows pressure (in cm water) plotted along the x-axis and volume (in mL) along the y-axis . The curve **starts in the lower left corner near the origin of the graph**, with this point **representing functional residual capacity**. If **PEEP is present**, then the **curve begins at that level of pressure** along the x-axis. As **inspiration** progresses, the **curve rises in a counterclockwise direction until it ends in the upper right hand corner when either the goal volume or pressure is reached.** The inspiratory limb of the curve takes on a **sigmoidal shape**, with an **initial flat part** indicating **movement of air into collapsed airways with low compliance**, the **middle steep part** indicating **lung recruitment**, and the **flattening of the curve** again representing the **end of inspiration**.

Rapid changes to the slope of the limb, called inflection points, signify instances where compliance changes suddenly.

A **lower inflection point (LIP)** correlates to the **opening of collapsed alveolar units and a sharp rise in volume**. The **steep par**t of the curve after the LIP occurs when **compliance is high and increased volume into the airways leads to a minimal increase in pressure**. An **upper inflection point (UIP)** occurs at the **end of inspiration when accumulation of more pressure leads to minimal increase in volume, compliance is low again**, and the curve may take on a **beaking appearance representing over distension** . Inflection points are **more easily appreciated in volume control modes than in pressure-targeted modes** .

Once **exhalation begins**, the curve continues in a **counterclockwise** direction, but starts its descent back to the origin. **The shape** of the graph in a **ventilated patient under ideal conditions resembles a football** and the slope of the whole loop correlates to lung compliance . When the **slope of the curve is flatter**, this represents **decreased compliance**. Conversely, a **steeper slope** to the curve indicates **increased compliance.**



The use of inflection points to prescribe ventilator settings that allow for a lung protective strategy has been described. It has been suggested that setting the **PEEP just above the LIP in ARDS patients may lead to less barotraumas and improved survival**. Another recommendation is to aim for ventilating patients between the LIP and UIP on the curve. However, these recommendations remain controversial as there are limitations to using the pressure-volume curve in this way.

Another important feature of the pressure-volume curve is called **hysteresis** . In the respiratory system, **hysteresis is failure of the lung tissue to act the same with inspiration and expiration**. It takes more energy to inflate the lungs than to deflate them, representing that the lung volume at any given pressure is different depending on the phase of ventilation. This is shown in the pressure-volume curve as the inflation and deflation limbs taking on different shapes, with hysteresis representing the area between the two limbs. Hysteresis in the lungs is related to alveolar air-liquid surface forces and the opening and closing of alveoli. Changes in resistance to air flow will affect the

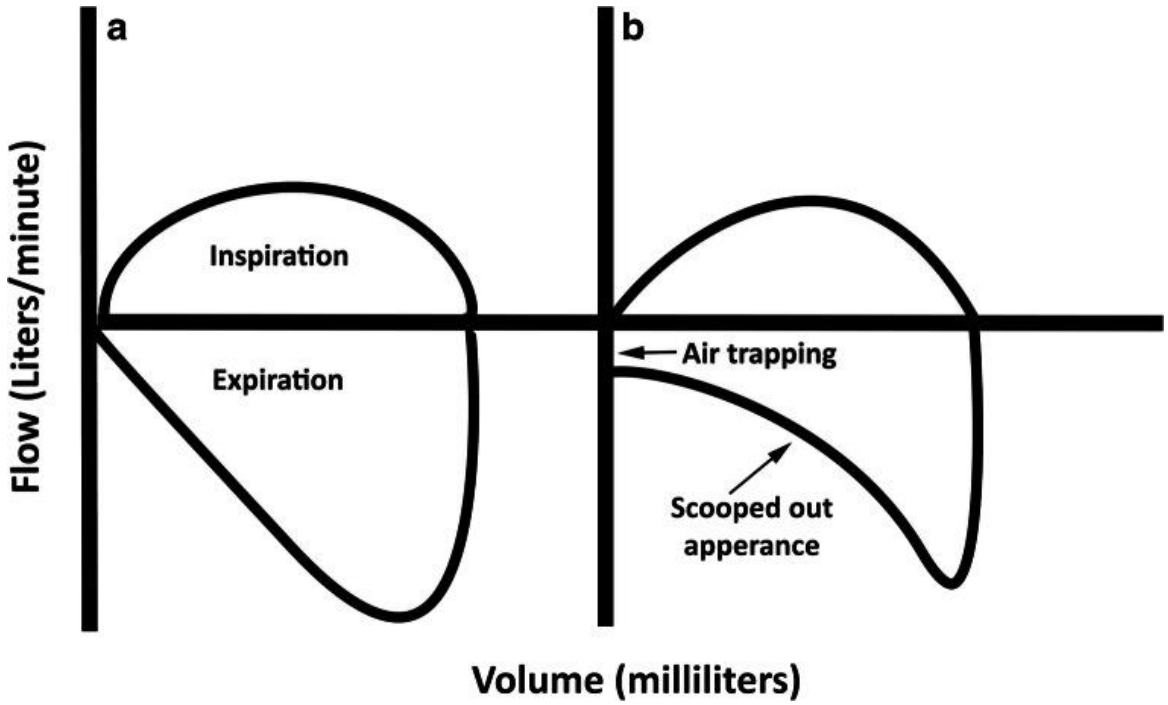
hysteresis, with the **curve appearing wider with increasing resistance**. It may be possible to identify changes to either inspiratory or expiratory resistance, therefore identifying potential etiologies of ventilator changes, by comparing the shape of successive pressure-volume curves.

Flow-Volume Curve

The **flow-volume** loop describes **how air flows in and out of the lungs during a breath**. In this graph, the volume (in milliliters) is on the x-axis and the flow rate (in liters/min) is on the y-axis. Conventionally, during pulmonary function testing, inspiration is below the x-axis and expiration is above. However, this is often reversed on ventilators with the inspiratory limb of the curve on top and the expiratory limb on the bottom. This is dependent of ventilator software.

Further discussion of this curve will assume that inspiration is above the x-axis and expiration is below. With the first breath, the inspiratory limb starts at the intercept of the x- and y-axes, where both flow and volume equal zero, and travels in a clockwise fashion. As the volume rises, and the curve travels along the both axes further, flow also increases. Volume continues to rise as flow starts to decrease, with the curve continuing on the x-axis but changing directions on the y-axis. Once the flow reaches zero, when the curve crosses the x-axis, inspiration is over and expiration begins. The curve continues moving in a clockwise fashion, this time with volume decreasing, as flow increases and then decreases similarly to before. The curve is complete when both volume and flow reach zero, signifying the end of expiration, completion of the breath, and complete emptying of volume inspired.

The **shape of the inspiratory portion** of the curve is often dependent on the mode of ventilation. For example, in **volume-controlled or flow-targeted modes**, since flow remains the same throughout inspiration, this limb takes **on a square shape**. In **pressure-controlled modes**, flow is **represented as descending** as volume increases during inspiration.



Several important pieces of information about air flow in and out of the lungs can be obtained from evaluation of the flow-volume loop, particularly the expiratory limb. First, the loop provides for **measurement** of a **peak expiratory flow rate**. A **lower peak expiratory flow rate indicates potential obstruction**, such as can be seen with **broncho constriction**. Also seen in this situation is an **expiratory limb** with **more concavity or a "scooped out" appearance**, representing **lower flows at a given volume**, as would be expected with **an obstructive process**. The expiratory limb may also show air trapping when it does not return to zero along the y-axis, or return to zero flow, before another breath is started. In addition to information about airway obstruction, **air leak may be identified** when the volumes in the **inspiratory and expiratory sides of the curve are different**. This may be seen when flow drops to zero suddenly, but volume does not.

Ventilator wave forms and asynchrony

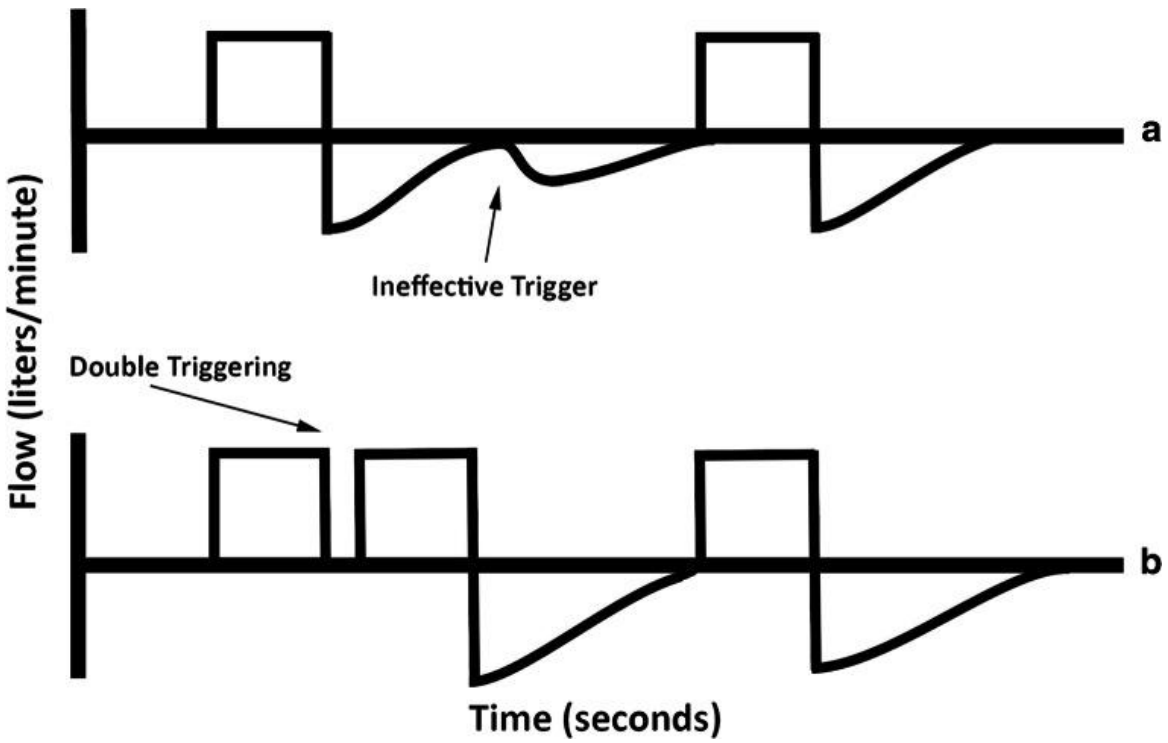
When a patient is interacting poorly with the ventilator, this is called asynchrony. Patient-ventilator asynchrony (PVA) can be associated with negative side effects including increased sedation needs, increased work of breathing, ventilation-perfusion mismatch, increased dynamic hyperinflation, and slower weaning. It can also be associated with worse outcomes including increased length of mechanical ventilation, increased length of stay, and increased mortality. Pediatric patients experience PVA frequently.

FOR THE EXTRA READERS

Asynchronies Related to Breath Initiation The first type of asynchrony is associated with the initiation, or trigger, of a breath. This occurs when the ventilator fails to respond to a patient's effort to take a breath. There are several types of **trigger asynchronies including ineffective triggering leading to delayed or missed breaths, double triggering, and auto-triggering**. When a **patient initiates** a breath, but **the machine fails to recognize** this attempt appropriately and **either a breath is delayed or not given** at all, this is referred to as **ineffective triggering**. This can be seen on the flow

versus time waveform as a deflection from the baseline expiratory flow with either a minimal decrease in airway pressure or no change at all, and no breath delivered . The easiest way to **troubleshoot** this issue is to look **at how sensitive the trigger is set on**

the machine and adjust it to make it easier for the patient to initiate a breath. Another potential **etiology of ineffective triggering** is the presence of **intrinsic PEEP (or auto-PEEP)**, a pressure that must be offset by the patient's effort before the ventilator will recognize the patient is triggering a breath. The higher the intrinsic PEEP, the more pressure that will need to be overcome. **Interventions designed to decrease intrinsic PEEP (increasing time of expiration or decreasing resistance to airflow with bronchodilators) or increase external PEEP** may help decrease the amount of ineffective triggering.

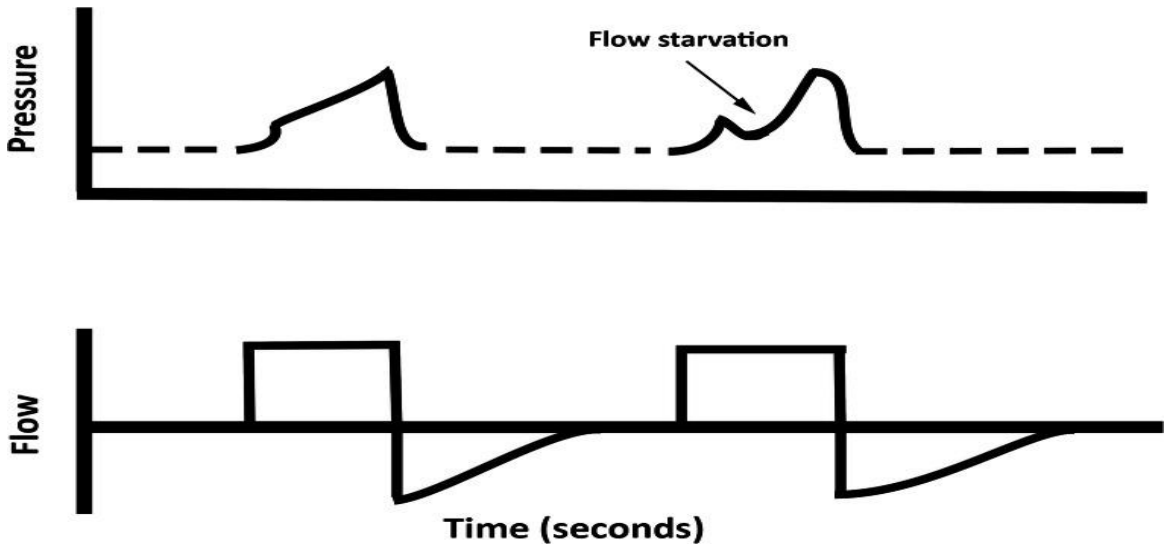


Another type of asynchrony associated with the initiation of a breath is **double triggering. This is when a patient wants to take a breath with a longer inspiratory time than the ventilator settings**, which may result in a second breath being triggered immediately after the first. This can be seen on all three scalars as one breath being followed immediately by a second without any time for exhalation. This can usually **be improved by matching the patient's inspiratory demands** better (e.g., increasing the inspiratory time, increasing the tidal volume).

Reverse triggering may **look similar to double triggering** on ventilator graphics. This **happens when the ventilator breath leads to contraction of the diaphragm that may be interpreted by the machine as the patient initiating a breath**. It is often **seen in patients under heavy sedation**. This may be shown as more volume or flow on the scalars at the end of a ventilator delivered breath, or may look like another breath triggered before the first breath's cycle is complete.

The last type of triggering asynchrony is called **auto-triggering** (also called **auto-cycling**). This occurs when **multiple breaths are delivered by the ventilator that was not initiated by the patient**. Some etiologies of auto-triggering include air leaks in the system, inappropriately set trigger sensitivity, condensation in the ventilator tubing, or detection of cardiac movement.

Asynchronies Related to Flow



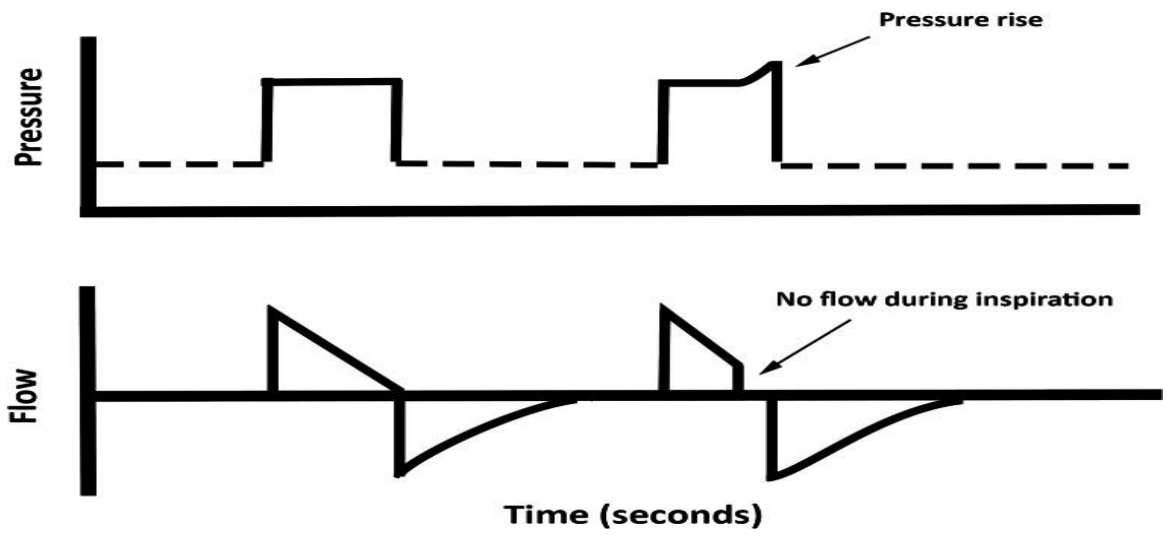
When a **patient is not receiving as much flow as they would like from the ventilator with each breath**, this is one type of asynchrony termed **flow starvation**. It can be identified by a dip in the pressure versus time scalar without associated changes in the flow or volume scalars. This represents that the **patient is generating more negative pressure to be able to pull more volume**, but **is not getting enough to meet their needs**. It is often due to inadequately set flow on the ventilator. It is more often seen in

modes where flow is constant as in volume control modes. Increase of the set flow or changing to a non-constant flow pattern may fix this problem.

Asynchronies Related to Cycling

These asynchronies are related to the inspiratory time either being too short or too long compared to the patient's desired inspiratory time. **When the ventilator stops a breath before the patient's inspiratory effort has finished**, this is called **premature cycling**. It can be seen on the flow versus time scalar as an additional upward deflection after inspiration is completed by the ventilator. It may also result in double triggering and an added breath as described above . Changes to the delivered flow or inspiratory time can help fix premature cycling.

Conversely, when **the ventilator's set inspiratory time is too long compared to the patient's inspiratory time**; this is referred to as **delayed cycling**. It results in a patient trying to exhale while inspiration is still occurring, and being unable to do so, there is a rise in pressure at the end of the pressure versus time scalar and a period of zero flow during inspiration. It can also be fixed by adjusting the flow rate or inspiratory time, depending on the mode of ventilation.



Modern ventilators provide multiple graphics that help guide the clinician in evaluation and management of respiratory failure. In addition to describing the basics of how a breath is

delivered to a patient, these graphics also give valuable information regarding how well a patient is interacting with the ventilator.

Pressure support-induced apneas

During sleep, the apneic threshold for PaCO2 increases, and lowering PaCO2 level below this threshold caused by excessive ventilation rapidly lead to a specific form of poor patient ventilator interaction, characterized by central apneas that negatively influence sleep in the absence of backup ventilation. High levels of pressure support ventilation (PSV) may cause sleep disruption from periodic breathing. If PSV delivers a higher-than-needed alveolar minute ventilation, hyperventilation and hypocapnia will occur that will be followed by apnea causing continuous sleep fragmentation . Avoiding excessive levels of assistance is very important with PSV, especially during sleep. The use of modes with backup ventilation such as assist-control mode or synchronized intermittent mandatory ventilation may also prevent periodic breathing but they may have other downside effects if they put the respiratory muscles excessively at rest. Apneas during PSV may be worsened in patients with heart failure

CHAPTER 8

TAKING THE PATIENT OFF FROM VENTILATOR SUPPORT.

Once a patient is stable and in a position to move towards spontaneous breathing it is important that steps are taken to wean the patient of the dependency of supported breathing. Weaning is the gradual withdrawal of a patient from assisted breathing on a life-support system or other form of therapy. Weaning a patient from a ventilator occurs when the condition of the patient improves and a decision is made to remove them from the ventilator through a trial of spontaneous breathing through the endotracheal tube and eventually extubation (removal of the tube).

Weaning is gradual reduction of ventilation. A new systematic review suggests that noninvasive ventilation after early extubation helps in reducing the total days spent on invasive mechanical ventilation; also the patients spending less time on invasive ventilation had lower rates of ventilator-associated-pneumonia . In some cases this process is rapid and uneventful; however, for some patients the process may be prolonged for days or weeks. Weaning is a term that is used in two separate ways. Firstly, it implies the termination of mechanical ventilation and secondly the removal of any artificial airway.

Assessment of readiness for extubation

Basic pre-conditions

- Resolution of the condition which had required the intubation and ventilation
- Patient-directed mode of ventilation (eg. PSV)
- Haemodynamic stability (the patient is unlikely to need massive fluid resuscitation in the near future, and their cardiac function is satisfactory to endure the increased demand from hard-working respiratory muscles)
- Adequate muscle strength

Airway protection assessment

- Good cough reflex on tracheal suctioning
- Good gag reflex on oropharyngeal suctioning
- Adequate neurological performance (obeying commands, or at whatever cognitive baseline previously permitted spontaneous breathing)

Gas exchange criteria

- Adequate oxygenation: SpO_2 over 90% on FiO2 under 40%
- Normal acid base status (pH >7.25), i.e. no significant respiratory acidosis

Lung mechanics criteria

- Adequate oxygenation: FiO2 40%
- PEEP less than 8 cmH_2O
- Satisfactory tidal volume: V_T > 5ml/kg
- Satisfactory vital capacity: VC > 10ml/kg
- Satisfactory MIP: less than 20-25 cmH_2O (i.e pressure trigger)
- Satisfactory RSBI (Rapid shallow breathing index): **Tidal volume in liters/Respiratory rate**; less than 105 is an indicator for successful attempt of extubation.

The level of consciousness is satisfactory and the patient is cooperative

There needs to be some minimum of patient cooperation, or at least the promise of adequate airway patency at extubation. You are not going to extubate somebody whose GCS would mandate intubation if they were found on the sidewalk.

The patient can raise their head off the pillow, and their arms off the bed.

This is a crude rapid test for muscle weakness. If the patient is able to do this, they probably don't have critical illness neuromyopathy.

There is an adequate cough.

If the tracheal suctioning results in a vigorous cough, one can expect secretions dribbling into the carina should do the same. One can rely on such a patient to clear their own sputum, instead of letting it fester in their airway.

There is an adequate gag.

If the oropharyngeal suctioning results in a vigorous gag, one can expect oral secretions will also be detected, and the patient will protect their lower airway from their upper airway secretions.

The patient can generate a good tidal volume with zero pressure support.

We are talking 10ml per kg of ideal body weight, at least. It means they probably have enough available lung tissue to support good ventilation post-extubation

The patient can overcome a -20 cmH_2O pressure trigger

This is the pressure trigger of a patient-triggered mode of ventilation. If they are able to generate such a negative pressure, one can make some assumption about the strength of their respiratory muscles.

The procedure of extubation

- Explanation of the procedure to the patient, assuring them it is only for a trial period
- The ventilator support is gradually reduced (e.g. reducing pressure during pressure support)
- The patient is placed into a better postural position (e.g. sitting upright or half-sitting)
- The airway is suctioned
- The patient is disconnected from the ventilator and given oxygen or mechanical assistance (CPAP)
- The patient is encouraged to breathe spontaneously
- The patient is monitored for signs of labored breathing, anxiety or an increase in $PaCO_2$
- Extubation should occur as soon as possible because breathing through an endotracheal tube increases the work of breathing
- Encourage the patient to cough after being extubated.

Patients may be extubated when they are alert, show a stable breathing pattern and control their airway. It is better to extubate patients after 2 hours of fasting/stopping feed to avoid chances of aspiration. Difficulties in weaning patients from a ventilator can occur due to:

- Inspiratory muscle atrophy
- Fatigue
- Paralysis of the diaphragm
- A fear of suffocation.

Role of physiotherapy

The respiratory care involves optimization of ventilation, airway clearance, prevention of pulmonary complications, and hastening weaning from mechanical ventilation.

Techniques used by physiotherapy to help improve patient breathing and wean patients off ventilators may include:

- Suctioning
- Postural drainage
- Central lavage (Pediatrics)
- Percussion
- Vibrations

FOR THE EXTRA READERS

Extubation of patient is not as simple as pulling out the endotracheal tube after deflating it. You should expect some complications and the need for re intubation.

Mechanical causes of difficult extubation

Possible causes of inability to remove the tracheal tube are failure to deflate the cuff caused by a damaged pilot tube, trauma to the larynx, cuff herniation, adhesion to the tracheal wall and surgical fixation of the tube to adjacent structures. Sequelae can vary from aspiration to fatal haemorrhage if undue force is applied. The problem is usually solved by puncturing the cuff transtracheally or using a needle inserted into the stump of

the pilot tube; rotation and traction of the tube; using a fibreoptic scope for diagnosis; and surgical removal of tethering sutures.

Cardiovascular response

Tracheal extubation is associated with a 10–30% increase in arterial pressure and heart rate lasting 5–15 min. Patients with coronary artery disease experience a 40–50% decrease in ejection fraction.

Respiratory complications

The incidence of coughing and sore throat, hypoxemia, difficulty swallowing, difficulty to speak can be expected. Active and passive heavy smokers, patients suffering from chronic obstructive pulmonary disease and children with mild to moderate upper respiratory tract infections have a high incidence of bronchospasm at extubation.

Airway obstruction

A differential diagnosis of post-extubation upper airway obstruction (UAO) includes laryngospasm, laryngeal oedema, haemorrhage, trauma and vocal cord paralysis/dysfunction.

Post-obstructive pulmonary oedema

The incidence of post-obstructive pulmonary oedema is ~1:1000 anaesthetics; most patients are children or young fit adults. The common pattern is an episode of airway obstruction at emergence followed by rapid onset of respiratory distress, haemoptysis, and bilateral radiological changes consistent with pulmonary oedema.

Tracheomalacia

Softening or erosion of the tracheal rings leading to tracheal collapse and UAO may be primary or secondary to a prolonged insult by a retrosternal thyroid or other tumours,

enlarged thymus, vascular malformations, and prolonged intubation. Failed extubation, complicated by inspiratory stridor or expiratory wheezing, may be the first signs of the condition. Techniques for extubation include deep extubation to avoid coughing and maintenance of continuous positive airway pressure (CPAP) to maintain airway patency.

Pulmonary aspiration

One-third of cases of pulmonary aspiration occur after extubation.

Post extubation stridor

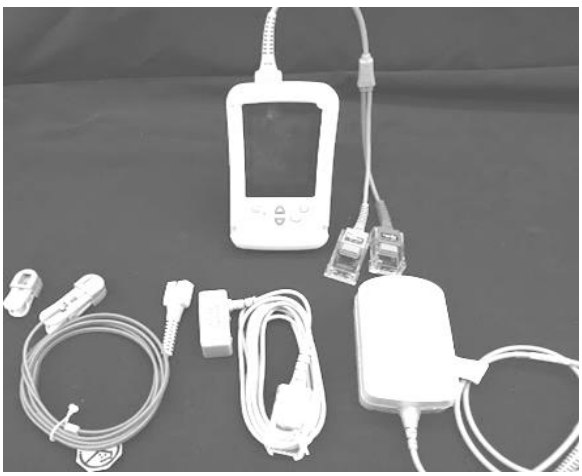
The incidence of laryngotracheal oedema leading to upper airway narrowing can cause post extubation stridor.

CHAPTER 9

A PEEK INTO ABG, $ETCO_2$ & CERTAIN DRUGS OF ICU.

The Arterial Blood Gas analysis and $ETCO_2$ monitoring are the accurate tools to find out the effectiveness of ventilation in patients. In this chapter we are discussing the drug dilution and dosage of various drugs which we commonly use in patients on ventilator

END TIDAL CAPNOGRAPHY



End-tidal carbon dioxide ($ETCO_2$) is the level of carbon dioxide that is released at the end of an exhaled breath. $ETCO_2$ levels reflect the adequacy with which carbon dioxide (CO_2) is carried in the blood back to the lungs and exhaled. Available evidence has established that $ETCO_2$ measurement can provide an indication of cardiac output and pulmonary blood flow. Non-invasive methods for $ETCO_2$ measurement include capnometry and capnography. Capnometry provides a numerical value for $ETCO_2$. In contrast, **capnography delivers a more comprehensive measurement that is displayed in both graphical (waveform) and numerical form. For this reason, capnography is currently the most**

widely recommended method for monitoring ETCO$_2$. Capnography devices are configured as either sidestream or mainstream. In a **sidestream configuration**, the CO_2 sensor is located in the monitoring device, which is at a distance from the patient. The exhaled CO_2 is diverted from the airway into the device via a sampling tube of six to eight feet in length, which is attached to the breathing circuit fitted to the patient. In the case of a mainstream configuration, the CO_2 sensor and a sampling cell are integrated into a small device that connects directly at the airway, between the breathing circuit and endotracheal tube (ETT). **Sidestream devices can monitor both intubated and non-intubated patients**, while **mainstream devices are most often limited to intubated patients**.

By using capnography, a patient's ventilation status is monitored in real time. Health care providers are able to identify potential breathing complications (such as airway obstruction, hyperventilation, hypoventilation, or apnoea) and respond accordingly with a change in clinical management (for example, providing supplemental oxygen or reassessing the patient). Capnography may also capture an otherwise self-resolving incident of respiratory depression, which might also lead to unnecessary interventions.

The capnogram

An end-tidal capnography waveform is a simple graphic measurement of how much CO2 a person is exhaling. The normal end-tidal capnography wave form is basically a rounded rectangle. When a person is breathing out CO2, the graph goes up and when breathing in the graph goes down.

Phase 1 is inhalation. This is the baseline. Since no CO2 is going out when a patient is breathing in, the baseline is usually zero.

Phase 2 is the beginning of exhalation. CO2 begins to travel from the alveoli through the anatomical dead space of the airway causing a rapid rise in the graph as the CO2.

Phase 2 measures the exhaled CO2 from the alveoli mixed with the gas that was in the dead space. This part of the graph goes up as the more concentrated CO2 gases from lower in the lungs rise up past the sensor.

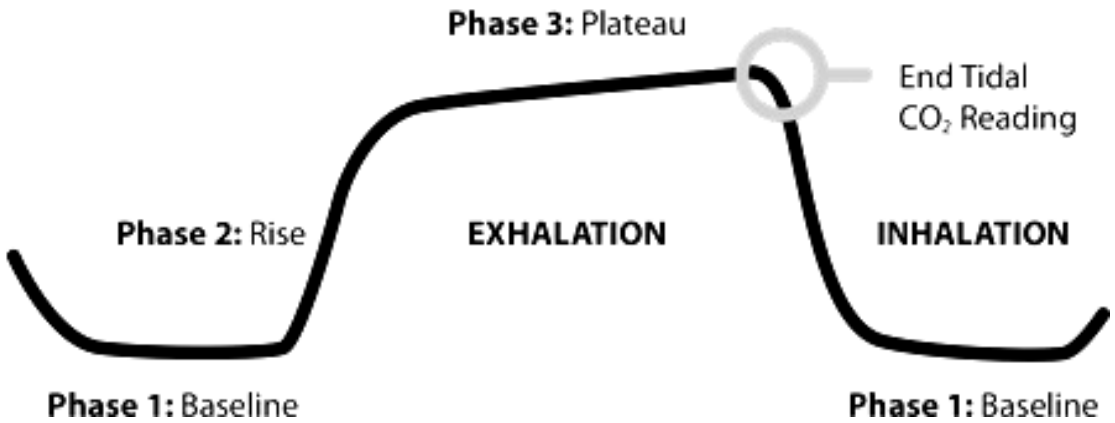
Phase 3 is when the sensor is receiving the CO2-rich gas that was in the alveoli. Because this is a fairly stable amount, the graph levels off into a plateau. The measurement at the end of the tide of respiration, the peak measurement at the very end of phase 3, is the EtCO2 reading.

After the end of phase 3, the patient inhales again, bringing clear air past the sensor, dropping the graph back down to zero to start over again at phase 1.

Although it can be intimidating to try and memorize what each phase (and the angles between them) represents, you can think of it as follows: The left side shows how quickly and easily air is moving out of the lungs; the right side shows how quickly and easily air is going in; the top shows how easily the alveoli are emptying.If all we wanted to read from capnography was ventilation, this would be enough, but to indirectly measure a patient's perfusion and metabolic status we must understand how CO2 gets to the lungs to be exhaled.

Normal wave form

Figure 1: Normal end-tidal capnography waveform

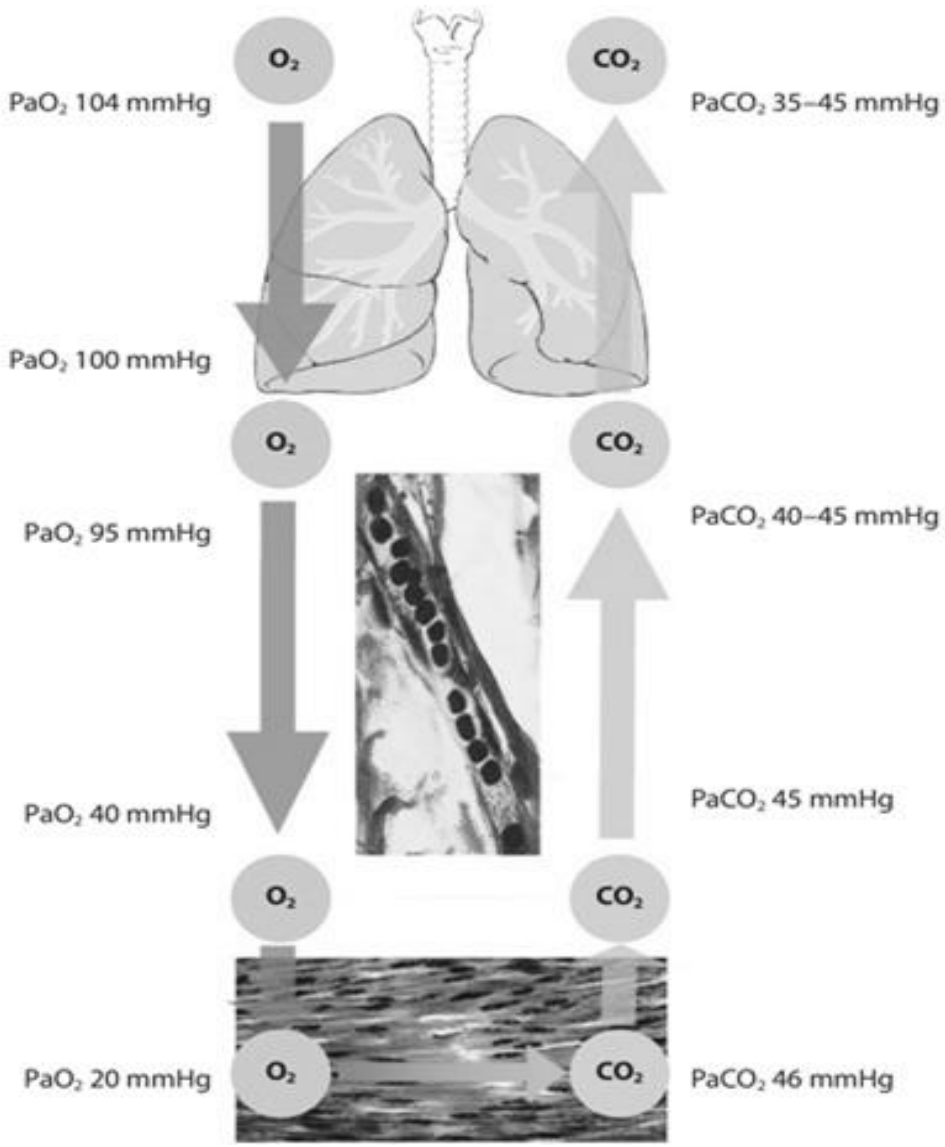


Patients with below conditions can have abnormal end-tidal capnography readings:

Abnormally high body temperature, Shivering, Sepsis, Endocrine disease, Bradypnoea, Brady cardia, pulmonary embolism

Knowing the pressures.

Many factors affect how oxygen gets into the body and CO_2 gets out; however, the biggest influence is the partial pressures of these gases.



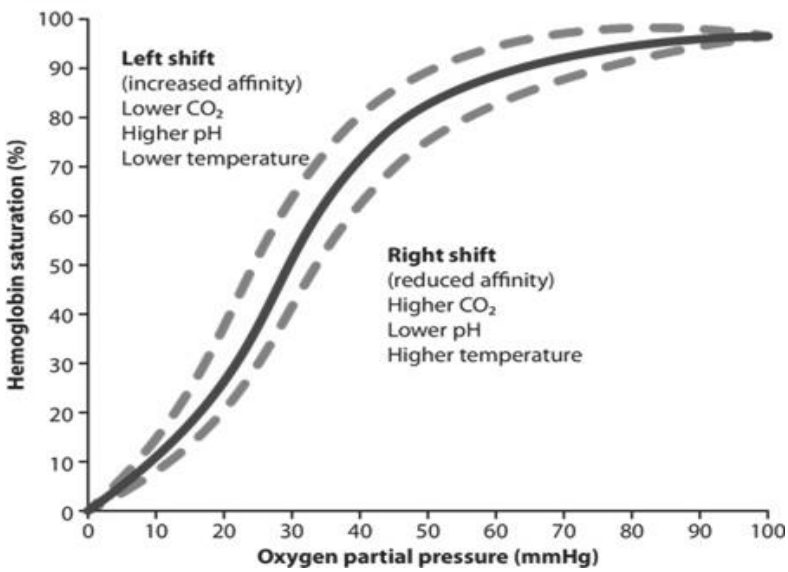
Although hemoglobin, myoglobin and other body chemicals play a part in transporting gasses, it can be helpful to begin by just picturing the partial pressures pushing the gasses from one part of the body to the next.

The normal partial pressure of oxygen in ambient air is approximately 104 mmHg. It gets humidified and absorbed by the body as it's inhaled, bringing the partial pressure down to 100 mmHg by the time the oxygen reaches the alveoli. The partial pressure of oxygen in the alveoli is known as PaO_2.

Oxygen is then pushed from the partial pressure of 100 mmHg in the alveoli to the lower partial pressure of 95 mmHg in the capillaries surrounding the alveoli. Oxygen gets carried through the circulatory system, getting absorbed along the way.

By the time the oxygen gets to the end of its journey, it has a partial pressure of approximately 40 mmHg, still high enough to allow it to move into muscles and organs that have a lower partial pressure of approximately 20 mmHg.

If the organs are functioning normally, the oxygen is metabolized, producing the CO_2 that we're ultimately going to measure. Although the journey back involves CO_2 moving primarily through the body's buffer system as bicarbonate (HCO3-) its movement is still largely governed by partial pressures.



The partial pressure of carbon dioxide (PCO_2) as it leaves the organs is approximately 46 mmHg, just high enough to push it into the capillaries which have a partial pressure of only 45 mmHg.[4] CO_2 travels through venous circulation largely untouched.

In the end it moves from 45 mmHg at the capillaries surrounding the alveoli into the alveoli themselves. From the alveoli to exhalation the CO_2 is approximately 35-45 mmHg. At this level it will get exhaled and measured by the EtCO_2 sensor, letting us know that the patient's metabolism, perfusion and ventilation are all working properly taking up oxygen, converting it to CO_2 and releasing it at a normal rate (or not).

If you were to know one more thing about oxygen and CO_2 transport, it's that high CO_2 reduces the affinity of hemoglobin for oxygen. Referred to as the Bohr effect, during normal body function this is a good thing, (the high CO_2 in muscles and organs help hemoglobin release needed oxygen). However, prolonged periods of high CO_2 and associated acidosis make it hard for hemoglobin to pickup and transport oxygen. This can be seen as a shift of the oxyhemoglobin dissociation curve to the right.

Conversely, if the patient has low CO_2, perhaps because of hyperventilation, it will cause an increased affinity for oxygen, allowing hemoglobin to pick oxygen up more easily. However, if the low CO_2 is prolonged, the hemoglobin may not release the oxygen into the organs. This is referred to as the Haldane effect and is seen as a shift of the oxyhemoglobin dissociation curve to the left. In this case you may have a "normal" pulse oximetry reading even though organs aren't getting the oxygen because hemoglobin is saturated with oxygen, but this oxygen remains "locked" to the hemoglobin. In this way your EtCO_2 reading can help you better interpret the validity and meaning of other vital signs like pulse oximetry, blood pressure and more.

PQRST IN ETCO_2 Monitoring ???

Now that we've peeked behind the curtain as to how CO_2 is produced in metabolism and transported via perfusion, let's use the PQRST (proper, quantity, rate, shape and trending) method to different types of emergency calls.

We read PQRST in order, asking, "What is proper?" Consider what your desired goal is for this patient. "What is the quantity?" "Is that because of the rate?" If so, attempt to correct the rate. "Is this affecting the shape?" If so, correct the condition causing the irregular shape. "Is there a trend?" Make sure the trend is stable where you want it, or improving. If not, consider changing your current treatment strategy.
Below are several examples.

Advanced Airway/Intubation

P: Ventilation. Confirm placement of the advanced airway device.

Q: Goal is 35-45 mmHg.

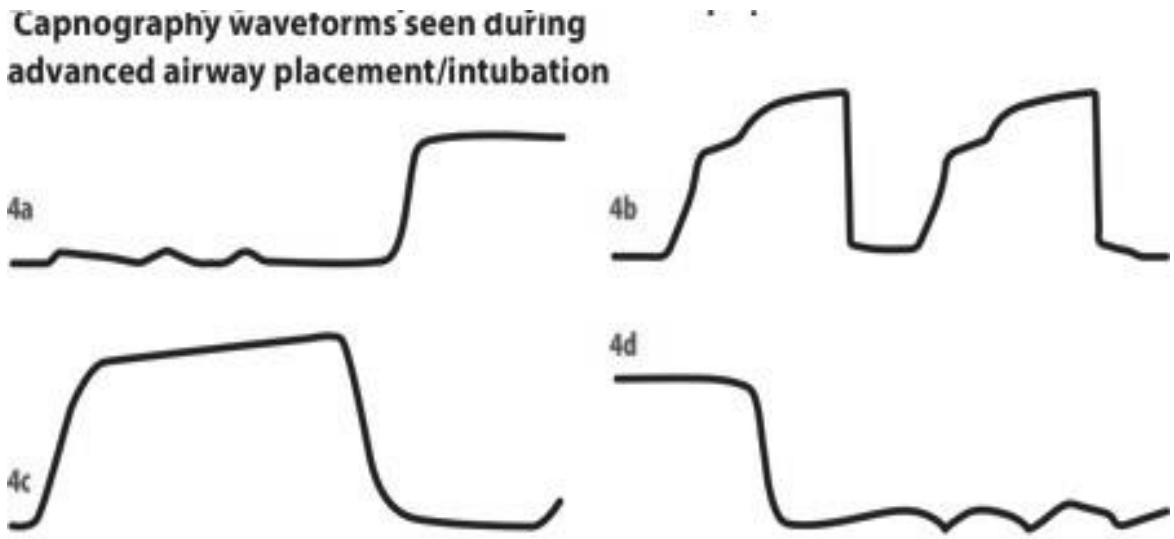
R: 10-12 bpm, ventilated.

S: Near flat-line of apnea to normal rounded rectangle EtCO_2 waveform. If the top of the shape is irregular (e.g., like two different EtCO_2 waves mashed together) it may indicate a problem with tube placement. This shape can indicate a leaking cuff, supraglottic placement, or an endotracheal tube in the right mainstem bronchus. This shape is produced when one lung-often the right lung-ventilates first, followed by CO_2 escaping from the left lung. If the waveform takes on a near-normal shape then the placement of the advanced airway was successful.

T: Consistent Q, R and S with each breath. Watch for a sudden drop indicating displacement of the airway device and/or cardiac arrest.

Capnography waveforms seen during

Advanced airway placement/intubation



4a:Near flat line ofapnea to normal rounded rectangle; 4b:Irregular top indicating problem with airway placement;4c: Near normal shows successful intubation; 4d: Sudden drop indicating displacement of airway or cardiac arrest

Wave forms in different scenarios

Capnography waveforms illustrating problematic ventilation

A: Faster ventilation/Rapid exhalation. B: Slower ventilation/longer exhalation with Co_2 building between breaths

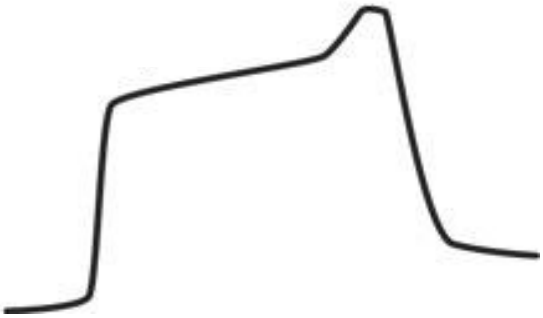
Capnography waveforms indicating ROSC after cardiac arrest

Capnography waveforms trending down in shock

Capnography waveforms illustrating emphysema or leaking alveoli in pneumothorax.

Capnography waveforms indicating hypoxia due to asthma

Capnography waveforms indicating poor lung compliance also seen in obese and pregnant patients



Capnography waveforms indicating hypoxia due to mechanical ventilation

ARTERIAL BLOOD GAS ANALYSIS

AN ARTERIAL BLOOD GAS (ABG) analysis can tell you about the patient's oxygenation (via PaO2 and SaO2), acid-base balance, pulmonary function (through the PaCO2), and metabolic status.

Endotracheal (ET) intubation and mechanical ventilation may be prescribed for patients who can't maintain adequate oxygenation or ventilation or who need airway protection. The goal of mechanical ventilation is to improve oxygenation and ventilation and to rest fatigued respiratory muscles.

Mechanical ventilation is supportive therapy because it doesn't treat the causes of the illness and associated complications. However, ventilator support buys time for other

therapeutic interventions to work and lets the body reestablish homeostasis. When using this lifesaving intervention, clinicians should take steps to avoid or minimize ventilator-induced lung injury (VILI), Patients should be weaned from ventilatory support if their condition permits.

A critically ill patient's clinical status can change rapidly and dramatically, and the need for ventilator support in terms of oxygenation or minute ventilation can vary at different stages of the illness. ABG analysis is an indispensable diagnostic tool for monitoring the patient's condition and evaluating the response to interventions. By reviewing the patient's ABGs and clinical status, clinicians can adjust ventilator settings to improve oxygenation, ventilation, and acid-base balance, or wean the patient from ventilator support.

Normal values for ABGs vary slightly among labs, but in general are:

- PaO2, 80 to 100 mm Hg
- SaO2, 95% to 100%
- pH, 7.35 to 7.45
- PaCO2, 35 to 45 mm Hg
- HCO3-, 22 to 26 mEq/L.

Serum lactate is often obtained along with ABGs; results are normally less than 2 mmol/L in critically ill patients.4-9

For mechanically ventilated patients, the key means of improving oxygenation are to increase the FiO2 or increase positive end-expiratory pressure (PEEP). Remember that a patient's minute ventilation equals respiratory rate times tidal volume (VT). Therefore, any intervention that alters respiratory rate or VT can help manage hypercapnia or hypocapnia and rectify acid-base imbalance.

- In volume control mode ventilation, increasing the VT, respiratory rate, or both will reduce PaCO2 and improve ventilation.

- In pressure control mode ventilation, interventions to improve ventilation include increasing the inspiratory pressure, respiratory rate, or both; prolonging inspiratory time; and decreasing airway resistance by administering bronchodilators, suctioning airway secretions, or using a larger diameter ET tube.
- In pressure support mode ventilation, interventions to improve ventilation include increasing the pressure support level and decreasing airway resistance by administering bronchodilators, suctioning airway secretions, or using a larger diameter ET tube.

Steps to interpreting ABGs

Follow this five-step approach to interpreting your patient's ABGs.

1. Is the patient hypoxemic? Look at the PaO2 and SaO2.

2. What's the acid-base balance? Check the pH.

3. How is the patient's pulmonary status? Look at the PaCO2.

4. What's the patient's metabolic status? Review the HCO3-.

5. Do you detect any abnormalities or compensation? What's the primary cause of the acid-base imbalance, and which derangement is the result of secondary (compensatory) change? Matching PaCO2 and HCO3- parameters with the pH can help you determine the primary cause and secondary change. Examine the serum lactate, hemoglobin, glucose, and electrolyte results.

Case 1: Meeting changing needs

A male patient, age 52, was admitted to the ICU via the Casualty due to respiratory distress and hypotension secondary to neutropenic sepsis. The patient required fluid resuscitation and I.V. positive inotropes. His history included diarrhea and fever for the last 3 weeks, diffuse large B-cell lymphoma treated with chemotherapy, hepatitis C virus, alcohol abuse, and cirrhosis.

Follow the five-step approach to analyze his admission ABGs:

- PaO2 of 81.3 mm Hg (while on supplemental oxygen at 6 L/minute via simple face mask) indicates his oxygenation was adequate.
- pH of 7.14 indicates acidosis.
- PaCO2 of 41.8 mm Hg indicates his minute ventilation is adequate for his metabolic status.
- HCO3- of 13.8 mmol/L reflects a metabolic alteration toward acidosis.
- serum lactate level of 5.8 mmol/L indicates hyperlactatemia.

The patient's metabolic alteration moved his pH toward acidosis, but he had no respiratory derangement. Specifically, his PaCO2 and PaO2 values showed that his respiratory system could maintain adequate ventilation and oxygenation with supplemental oxygen of 6 L/minute. **This ABG profile shows uncompensated metabolic acidosis.**

The patient's metabolic acidosis was most likely caused by diarrhea and aggravated significantly by sepsis and septic shock. With an acute episode of septic shock, the patient's admission ABGs didn't demonstrate respiratory compensation for metabolic acidosis, although it usually occurs fairly quickly.

Lactic acidosis is characterized by hyperlactatemia associated with metabolic acidosis. The patient's lactate and pH values confirmed the diagnosis of lactic acidosis. Hypotension decreases tissue perfusion and impairs oxygen delivery, causing tissue hypoxia.

Tissue hypoxia leads to anaerobic metabolism and increases lactate production. Hepatic dysfunction reduces lactate clearance. This patient's lactic acidosis was most likely caused by septic shock and the preexisting cirrhosis.

In addition to antibiotics, the patient received continued I.V. fluid resuscitation and positive inotropes to maintain his mean arterial pressure (MAP) above 70 mm Hg. Because his oxygenation and ventilation were adequate, mechanical ventilatory support wasn't initiated at this stage, but he continued to receive supplemental oxygen.

Three-and-a-half hours after the previous ABG results, a new ABG analysis showed: pH, 7.29; PaCO2, 35.3 mm Hg; PaO2, 99.7 mm Hg; HCO3-, 17 mEq/L; and lactate, 5.77 mmol/L. The patient had less profound uncompensated metabolic acidosis with a marginal decrease in lactate level, but had developed signs of increased work of breathing: restlessness, shortness of breath, accessory muscle use, and diaphoresis. Because he was at increased risk for respiratory muscle fatigue, he was put on noninvasive ventilation with continuous positive airway pressure (CPAP) of 10 cm H2O.

After the patient had been on CPAP for 4 hours, the ABG analysis showed: pH, 7.34; PaCO2, 32.4 mm Hg; PaO2, 95.9 mm Hg; HCO3-, 19 mEq/L; and lactate, 6.7 mmol/L. The drop in the PaCO2 level to below the normal range suggests that the patient was hyperventilating to blow off more carbon dioxide and raise pH. **In other words, his respiratory system was compensating for the metabolic acidosis**. Now, the diagnosis was **partially compensated metabolic acidosis**. However, his lactate level was still quite high.

Hyperlactatemia may indicate inadequate tissue perfusion, but the patient's MAP had been maintained above 70 mm Hg and his urine output was more than 0.5 mL/kg/hour, indicating adequate tissue perfusion. The slow reduction of his hyperlactatemia was most likely due to impaired liver function.

The patient was weaned off CPAP and humidified oxygen at 6 L/minute was administered via simple face mask. On the next day, his ABGs were pH, 7.41; PaCO2, 34.2 mm Hg; PaO2, 90.7 mm Hg; HCO3-, 20 mEq/L; and lactate, 5.41 mmol/L. His metabolic derangement had been corrected significantly by establishing and maintaining adequate tissue perfusion. Moreover, acid-base balance had been restored by his respiratory compensation. At this stage, the diagnosis was **fully compensated metabolic acidosis.**

On the fourth day postadmission, the patient's temperature increased to 103.5[degrees] F (39.7[degrees] C) and his oxygenation dropped. An ABG analysis revealed: pH, 7.34; PaCO2, 52.6 mm Hg; PaO2, 60.7 mm Hg (indicating hypoxemia); HCO3-, 27.6 mEq/L; and

lactate, 2.44 mmol/L. Fever increases oxygen consumption and carbon dioxide production. The patient's respiratory system was unable to maintain adequate oxygenation and ventilation to meet his metabolic demand, as indicated by his hypoxemia and hypercapnia. His carbon dioxide retention led to respiratory acidosis. On the other hand, his metabolic process was attempting to elevate the pH, which suggested metabolic compensation for his respiratory acidosis.

The above ABG profile is consistent with **partially compensated respiratory acidosis**. A marked reduction in lactate levels is the result of decreased lactate production and increased lactate clearance due to improved tissue perfusion since admission.

Bilevel positive airway pressure (BiPAP) refers to setting inspiratory positive airway pressure (IPAP) and expiratory positive airway pressure (EPAP) separately. The gap between IPAP and EPAP creates a pressure support. Compared with CPAP, BiPAP is more effective in eliminating carbon dioxide because of the pressure support generated by the gap between IPAP and EPAP.15,16 BiPAP (IPAP, 15 cm H2O; EPAP, 7 cm H2O) with an FiO2 of 0.40 was started to treat the patient's hypoxemia and hypercapnia as well as to rest respiratory muscles.

With the above settings, a pressure support of 8 cm H2O was generated (15 cm H2O - 7 cm H2O = 8 cm H2O). Also, antibiotics were changed based on the latest culture and sensitivity results.

After half an hour, the patient's ABG analysis showed: pH, 7.39; PaCO2, 42.1 mm Hg; PaO2, 90.1 mm Hg; HCO3-, 25.4 mEq/L; and lactate, 2.40 mmol/L, which suggested his hypoxemia, carbon dioxide retention, and respiratory acidosis had all been corrected.

The next day, his ABG analysis while on BiPAP was pH, 7.27; PaCO2, 58.9 mm Hg; PaO2, 89.5 mm Hg; HCO3-, 26.8 mEq/L; and lactate, 1.78 mmol/L.

Partially compensated respiratory acidosis recurred, which could have been the result of inadequate spontaneous breathing and drowsiness. Because of the patient's increasing risk of BiPAP intolerance, he was **endotracheally intubated and ventilated with pressure support mode** ventilation with the following settings: FiO2, 0.40; PEEP, 10 cm H2O; and pressure support, 16 cm H2O.

Thirteen hours later, the patient's ABGs were pH, 7.51; PaCO2, 34.1 mm Hg; PaO2, 99.2 mm Hg; HCO3-, 26.8 mEq/L; and lactate, 2.39 mmol/L.

Pressure support ventilation augments spontaneous tidal volume and blows off more carbon dioxide. This patient's most recent ABG values showed that the ventilator support had **turned his respiratory acidosis to alkalosis**. Because both respiratory and metabolic alterations moved pH toward alkalosis, he developed mixed respiratory and metabolic alkalosis.

Two days later, the patient's ventilator settings had been weaned to FiO2, 0.30; PEEP, 10 cm H2O; and pressure support, 12 cm H2O. ABG values were pH, 7.59; PaCO2, 28.4 mm Hg; PaO2, 156.3 mm Hg; HCO3-, 26.6 mEq/L; and lactate, 1.60 mmol/L. His mixed respiratory and metabolic alkalosis was worsening. The decrease of PaCO2 levels from 34.1 to 28.4 mm Hg suggested he'd been **overventilated**.

Hypocapnia and respiratory alkalosis can be caused by pain, agitation, severe anemia, hypoxia, brainstem injury, or excessive mechanical ventilation. With this patient, the absence of pain, agitation, anemia, and other conditions suggest the most likely cause for his hyperventilation would be over ventilation caused by pressure support. Consequently, both pressure support and PEEP were reduced to 5 cm H2O. In less than 2 hours, a repeat ABG showed: pH, 7.49; PaCO2, 37.2 mm Hg; PaO2, 96.9 mm Hg; HCO3-, 28.2 mEq/L; and

lactate, 1.53 mmol/L. Lowering the pressure support corrected his hypocapnia and eliminated respiratory alkalosis. Now, he had only uncompensated metabolic alkalosis.

Three days later, the patient was extubated and placed on an air-entrainment **(Venturi) mask** with an FiO2 of 0.30. His ABGs were now pH, 7.46; PaCO2, 42.4 mm Hg; PaO2, 114.8 mm Hg; HCO3-, 29.8 mEq/L; and lactate, 1.10 mol/L, reflecting **minor uncompensated metabolic alkalosis.** Because hepatic dysfunction reduces the production of HCO3- and proteins (buffers), the patient's minor metabolic alkalosis most likely resulted from his cirrhosis.

Case 2: Dual pathology and permissive hypercapnia

An 84-year-old male patient with renal dysfunction developed acute respiratory distress syndrome (ARDS). He was receiving an I.V. furosemide infusion. Because of severe hypoxemia and profound hypercapnia, he was intubated and ventilated with high levels of FiO2, PEEP, and pressure support for a prolonged period.

When the patient was ventilated with pressure support mode ventilation at an FiO2 of 0.45, PEEP of 17.5 cm H2O, and pressure support of 12 cm H2O, his ABGs were:

- PaO2 of 85 mm Hg, indicating no hypoxemia with ventilatory support.
- pH of 7.39 (within normal limits).
- PaCO2 of 65 mm Hg, indicating his minute ventilation was inadequate and causing hypercapnia and respiratory acidosis.
- HCO3- of 38 mEq/L, reflecting a metabolic alteration toward alkalosis, most likely caused by the furosemide infusion or compensatory changes for hypercapnia and respiratory acidosis.
- lactate of 1.21 mmol/L, indicating adequate tissue perfusion.

ARDS resulted in respiratory acidosis. On the other hand, the furosemide infusion and metabolic compensation for respiratory acidosis led to metabolic alkalosis. The above mixed acid-base disorders produce a normal pH.

Some patients may need a high level of ventilatory support to achieve and maintain optimal ABG values. This places them at risk for developing VILI from large tidal volumes or positive pressure, or oxygen toxicity from high FiO2 values, and may delay weaning. **In patients with refractory hypoxemia or profound hypercapnia, mild hypoxemia or permissive hypercapnia are acceptable for a short time because this lung-protective ventilation strategy can minimize VILI**.

The mechanical ventilation protocol summary developed by the National Institutes of Health; National Heart, Lung, and Blood Institute; and ARDS Clinical Network recommend maintaining PaO2 between 55 and 80 mm Hg or SpO2 between 88% and 95%, and pH between 7.30 and 7.45 in patients with ARDS.

To restore this patient's acid-base balance and provide adequate oxygenation, clinicians allowed permissive hypercapnia, making no change in ventilator settings despite his PaCO2 of 65 mm Hg. Eventually, the patient recovered, was extubated, and was discharged home.

Case 3: Dehydration

A 79-year-old woman was admitted to the ICU after a right hemicolectomy with the following ventilator settings: FiO2, 0.40; PEEP, 10 cm H2O; and pressure support, 10 cm H2O. Her ABGs were:

- PaO2 of 85.2 mm Hg, indicating no hypoxemia with ventilatory support.

- pH of 7.27, indicating acidosis.
- PaCO2 of 41.5 mm Hg, indicating pulmonary ventilation was adequate for her metabolic status.
- HCO3- of 18.6 mEq/L, reflecting a metabolic disturbance toward acidosis.
- lactate of 1.57 mmol/L, a normal level suggesting that her tissue perfusion was adequate.

This is **uncompensated metabolic acidosis.** The patient subsequently developed sepsis and renal failure. Because of renal failure and severe metabolic acidosis, continuous veno-venous hemodiafiltration (CVVHDF) was started with fluid removal at 150 mL/hour.

Five days later, her ABGs were: pH, 7.49; PaCO2, 41.2 mm Hg; PaO2, 92.9 mm Hg; HCO3-, 31.3 mEq/L; and lactate, 2.38 mmol/L. Ventilator settings were FiO2, 0.30; PEEP, 7.5 cm H2O; and pressure support, 5 cm H2O. Her metabolic acidosis had been corrected by CVVHDF(Continuous veno-venous hemodiafiltration**)**, but she developed uncompensated metabolic alkalosis. Her lactate elevation was most likely due to sepsis. Because her serum creatinine level had returned to normal and her urine output was adequate, CVVHDF therapy was terminated.

After pressure support was increased from 5 to 12 cm H2O, her systolic BP dropped from 140 mm Hg to less than 110 mm Hg. Her central venous pressure (CVP) was 5 mm Hg. (During the first 6 hours of resuscitation, the goals of sepsis-induced hypoperfusion include a CVP of 8 to 12 mm Hg). In addition, her urine output decreased significantly. Her ABGs were pH, 7.51; PaCO2, 36.3 mm Hg; PaO2, 106.2 mm Hg; HCO3-, 28.3 mEq/L; and lactate, 1.30 mmol/L. This shows worsening metabolic alkalosis because increasing pressure support blew off more carbon dioxide and elevated pH.

PEEP affects the whole respiratory cycle (inspiration and expiration). Pressure support, however, is delivered only during the inspiratory phase of spontaneous breaths. Therefore, compared with pressure support, PEEP has a more profound effect in decreasing cardiac output and lowering BP. With mechanically ventilated patients, BP often drops after PEEP is increased if the patient has inadequate intravascular volume.1,3 But a decrease in BP seldom occurs after elevating pressure support, unless the patient is profoundly dehydrated.

This patient's marked reduction in BP and urine output as well as low CVP pointed to the possibility of profound dehydration, which made her very sensitive to increasing pressure support. Dehydration also can cause metabolic alkalosis. Consequently, pressure support was lowered back to 5 cm H2O. Her systolic BP immediately increased to between 140 and 160 mm Hg. She also received a 250 mL I.V. bolus of 0.9% sodium chloride solution twice, and the I.V. maintenance fluid infusion rate was increased from 60 to 100 mL/hour. Consequently, her CVP rose to 7 mm Hg.

Next, 500 mL of 4% albumin was infused over 4 hours. Her urine output increased to 17 to 35 mL/hour. After the albumin infusion was complete, the patient's ABGs were: pH, 7.46; PaCO2, 36.1 mm Hg; PaO2, 94.3 mm Hg; HCO3-, 25.1 mEq/L; and lactate, 1.17 mmol/L.

Treating dehydration and lowering pressure support nearly resolved this patient's metabolic alkalosis in less than 6 hours.

Because a critically ill patient's condition can change rapidly and dramatically, dynamic reviews of ABGs are essential. By understanding mechanical ventilation and how to use ABG results, ventilation strategies can be changed as needed to address changes in the patient's condition.

Calculation of anion gap

Calculation of the anion gap (AG) is one of the way to assess acidosis .The equation of anion gap is as follows

AG= [Na+]-([Cl-] + [HCO3-])=12 ± 2

A normal anion gap is approximately 12 meq/L.

In patients with hypo albuminemia, the normal anion gap is lower than 12 meq/L; the "normal" anion gap in patients with hypoalbuminemia is about 2.5 meq/L lower for each 1 gm/dL decrease in the plasma albumin concentration (for example, a patient with a plasma albumin of 2.0 gm/dL would be approximately 7 meq/L.)

If the anion gap is elevated, consider calculating the osmolal gap in compatible clinical situations.

Elevation in AG is not explained by an obvious case (DKA, lactic acidosis, renal failure

Toxic ingestion is suspected

OSM gap = measured OSM – (2[Na+] - glucose/18 – BUN/2.8

The OSM gap should be < 10

If an increased anion gap is present, assess the relationship between the increase in the anion gap and the decrease in [HCO3-].

Assess the ratio of the change in the anion gap (ΔAG) to the change in [HCO3-] (Δ[HCO3-]): ΔAG/Δ[HCO3-]

This ratio should be between 1.0 and 2.0 if an uncomplicated anion gap metabolic acidosis is present.

If this ratio falls outside of this range, then another metabolic disorder is present:

- If ΔAG/Δ[HCO3-] < 1.0, then a concurrent non-anion gap metabolic acidosis is likely to be present.
- If ΔAG/Δ[HCO3-] > 2.0, then a concurrent metabolic alkalosis is likely to be present.

Correcting lactic acidosis with bicarbonate infusion is not recommended. Lactic acidosis causes a decrease in serum bicarbonate concentration that is similar in magnitude to the increase in the lactate concentration. Lactate is a metabolizable organic anion that, when oxidized, will generate bicarbonate. Thus, if the stimulus to lactic acid production is eliminated by successful treatment of the underlying disease (eg, restoration of perfusion in a patient with shock), oxidative processes will metabolize the accumulated lactate and regenerate bicarbonate. This will correct the metabolic acidosis and reduce the anion gap.

FOR THE EXTRA READERS

Knowing the medicines well surely help you to get an edge over your day to day practice. Here are some commonly used ICU drugs its dilution and calculation. **GOOD LUCK!**

INFUSION FORMULA

3mg in 50 ml ————→1ml/hour = 1mcg/min 3mg/kg in 50 ml ————→ 1ml/hour = 1mcg/kg/min

1/2 mg/kg in 50ml ————→ 1ml/hour = 10mcg/kg/hour

Percentage Concentration

X% of drug equivalence with X GRAM of drug in 100ml diluent

ALL DRUGS ARE TO BE DILUTED WITH NORMAL SALINE EXCEPT NORADRENALINE AND AMIODARONE (WITH D5%)

ACTRAPID INSULIN INFUSION

Preparation: 40 IU/ ml Use 50 mls syringe

Take 40 IU of insulin (1 ml)

Dilute with 39 ml of Normal Saline = 50 ml dilution

Rate: 1ml/ hour = 1 IU/ hour

Commence Fixed Rate IV Insulin Infusion

- **0.05** to **0.1 U/kg/hr** based on estimate weight
- **Aim to drop Blood Glucose Level (BGL)/ Glucometer (GM) 2-5 mmol/L per HOUR**
- INCREASE if BGL doesn't drop by a minimum of 2 mmol/hr
- INCREASE if serum ketone doesn't decrease by minimum of 0.5mmol/hr (if available)
- REDUCE dose if BGL dropping too rapidly > 5 mmol/hr

ADRENALINE INFUSION

Preparation: 1 ampoule 1ml (1:1000 = 1mg / ml)

Dilute 3.0 mg (3mls) of Adrenaline with 47 mls of Normal Saline in 50 mls syringe.

Rate: **1ml/hr = 1mcg/min** (Document rate on Syringe Pump & in Patient's Notes)

Dose: 1 – 10 mcg/min (starting infusion rate 0.1 mcg/kg/min)

Titrate accordingly to desired BP - (Infusion range 0.1-2.0mcg/kg/min ie for 50kg patient start with 5ml/h)

Calculations:

- Dilute 3mg adrenaline in 50mls NS (Preferable to use single strength in ED)

- 50mls —> 3mg —> 3000mcg
- 1ml —> 3000/50 = 60mcg
- 1ml/hr —> 60mcg/hr —> 60mcg/60min —> 1mcg/min
- 1ml/hr = 1mcg/min

Use single strength in ICU, especially if infusion is through a peripheral line. Make sure BP cuff is not on the Arm of the peripheral line. Regularly inspect the site of insertion of the peripheral line.

ADRENALINE INFUSION (Paediatrics)

In paediatrics patient:

Dilution: 0.15mg/kg in 50mls D5%

Dose : 0.05 -0.5 mcg/kg/min (1-10ml/hr) Rate : 1-10ml/hr Document dilution on Syringe Pump & in Patient's Notes

E.g. 10 kg patient

10 x 0.15mg =1.5mg (Add 1.5mg adrenaline in D5% to make 50mls) 1 ml/hr = 0.05 mcg/kg/min

0.05 -0.5 mcg/kg/min (1-10 ml/hr)

In smaller patients, dose can be concentrated to 0.3 mg/kg in 50ml D5% Run at 0.5-5 ml/hr (0.05-0.5 mcg/kg/min)

AMIODARONE INFUSION

Preparation: 150mg / 3mls

Loading Dose:

5mg/kg bodyweight infused over 20-120 mins Dilute 2 ampoules of amiodarone (300mg / 6mls). In 100 mls of Dextrose 5% **(Incompatible with Normal Saline).**Use microchamber or infusion pump

(Dilute 150 mg in 50 ml D5%, Run @100ml/Hour (Do in 2 syringes)

Run 300mg over 1 hour

Maintenance Dose:

Dilute 6 ampoules of Amiodarone (900 mg/18mls) In 500mls of Dextrose 5%

Run over remaining 23 hours —> 22mls/hour

Max maintenance dose 1.2gm in 24 hours

Alternative dose:

Preparation from ICU Sungai Buloh

600mg in 50ml D5% (12mg/ml)

Run at **12.5ml/h (150mg) for 2 hours** then **3ml/h (36mg/ml)**.

AMINOPHYLINE INFUSION

Preparation: 250mg / 10 mls

Loading dose: 5mg/kg in 100mls NS over 1/2 Hour

Eg: 250-500 mg in 100mls Normal Saline (or 5mg/kg) Run over 20 - 30 minutes

(Do not give bolus to patients already on oral theophylline)

Maintenance Dose:

Dose:

Non-smoker: 0.5mg/kg/hr

(i.e: BW= 50kg; run @ 5ml/Hr)

Smoker: 0.8mg/kg/hr

Elderly: 0.3mg/kg/hr

Dilute 250 mg in 50 mls syringe with Normal saline 1 ml = 250 / 50 = 5mg

1 ml /hr = 5mg /hr

Eg : Body weight 70 kg for young non smoker.

0.5 mg/kg/hour = 0.5 x 70 = 35mg /hour

35mg/hour = 35/5 = 7mls /hour (Usual dose at 6-7mls/hr).

CALCIUM GLUCONATE 10%

For hypocalcemia

Inject 10-30ml of a 10% Calcium gluconate solution IV over 10 minutes (slow bolus).

Cocktail regime (for hyperkalaemia)

I/V slow bolus 10mls of 10% Calcium Gluconate

The FIRST medication to be given immediately

Given over 2-5 minutes

Cardiac (ECG) monitoring

Effect should be evident within a few minutes and last 30-60 minutes

Can be repeated once or twice if necessary – titrate against ECG changes

Avoid use in hyperkaleamia secondary to digoxin toxicity

I/V bolus 50mls of Dextrose 50% (Glucose not required if HYPERGLYCEMIC/DKA)

I/V bolus insulin 10 IU

IV Sodium Bicarbonate (IV NaHCO3) – Consult with Emergency Physician. Given only if patient in SEVERE METABOLIC ACIDOSIS.

Oral calcium polystyrene sulphonate – orally every 6 hours. Effect takes 1-2 hours

Consider Renal replacement therapy (dialysis).

DIGOXIN

Preparation: 0.5mg in 10ml N/S Give slow bolus over 10-20 mins

Atrial fibrillation

Loading dose for the management of atrial fibrillation was 0.25 mg IV every 2 hours up to 1.5 mg. The recommended daily maintenance dose was 0.125 to 0.375 mg IV or orally

AF/Heart failure for rapid digitalization

Rapid digitalization may be achieved with a loading dose of 8 to 12 mcg/kg IV. Administer half of the total recommended loading dose as a first dose, then give one-fourth of the total dose every 6 to 8 hours for 2 doses

Example- for pt 50 kg and take 10mcg/kg will be 500mcg ie 0.5mg. Given half as first dose ie 0.25mg then one-fourth ie 0.125mg every 6 hours.

Special precaution in hypokalaemia patient as will lead to digitalis toxicity.

DOBUTAMINE

Preparation: 1 ampoule contains 250mg in 5 mls

Syringe Pump. No bolus doses. Preferable to use single strength

Method 1

(Dilution dose is Fixed, but rate is adjusted according to weight)

- **2**50mg Dobutamine in 50ml NS
- 50mls —> 250mg
- 1ml —> 5mg —> 5000mcg
- 1ml/hr —> 5000mcg/hr —> 5000mcg/60min —> 83.3 mcg/min
- 1ml/hr —> 83.3 mcg/min

In a 70kg person 1ml/hr —> 83.3 mcg/min —> 83.3 mcg/70kg/min 1ml/hr = 1.2 mcg/kg/min (Document Rate on Syringe Pump & in Patient's Notes)

Method 2

(Dilution dose is adjusted according to patient's weight. The rate is fixed) Eg 70kg patient

3 x BW = 3 x 70 = 210mg

- 210 mg in 50mls NS
- 50mls —> 210mg
- 1ml —> 210/50 = 4.2mg
- 1 ml/hr —> 4.2mg/hr —> 4.2mg/60min —> 4200mcg/60min —> 70mcg/min

—>1mcg/kg/min

Rate: 1ml/hr = 1mcg/kg/min (Document Rate on Syringe Pump & in Patient's Notes).

DOPAMINE

Preparation: 1 vial contains 200mg in 5mls

Use Syringe Pump - No bolus doses. **Preferable to use single strength.**

Method 1

(Dilution dose is Fixed, but Rate is adjusted according to Weight)

- 200mg Dopamine in 50ml NS
- 50mls —> 200mg
- 1ml —> 4mg —> 4000mcg
- 1ml/hr —> 4000mcg/hr —> 4000mcg/60min —> 66.6mcg/min
- 1ml/hr —> 66.6mcg/min

In a 70kg person 1ml/hr —> 66.6mcg/min —> 66.6mcg/70kg/min —> **1mcg/kg/min**

(Document Rate on Syringe Pump & in Patient's Notes)

Method 2

(Dilution dose is adjusted to patient's weight. The Infusion rate is fixed)

E.g. Patients weight 70kg

3 x BW = 3 x 70 = 210mg

- 210 mg in 50mls NS
- 50mls —> 210mg
- 1ml —> 210/50 = 4.2mg
- 1 ml/hr —> 4.2mg/hr —> 4.2mg/60min —> 4200mcg/60min —> 70mcg/min

—>1mcg/kg/min

Rate: 1ml/hr = 1mcg/kg/min (Document rate on Syringe Pump & in Patient's Notes)

FENTANYL INFUSION

Preparation: 100mcg / 2mls

Dilute 200mcg (2 ampoules) of Fentanyl with NS to become 20 mls **OR (600 mcg to become 60 mls.)**

1 ml —> 200/20 OR 600/60 = 10 mcg

Dose Infusion:

Dose range 0.5-1.5 mcg/kg/h

If 60kg pt may start with 3ml/h (30mcg/H)

Titrate to desired effect and BP.

FUROSEMIDE INFUSION

Preparation: 20mg/2ml ampoule

Bolus Dose: 20 - 40mg (max 160mg) undiluted IV slow bolus 5-10mins

Infusion Dose:

200mg (10 amps undiluted) = 20 mls in 20mls syringe 1 ml/hr = 10mg/hr

Run at **10-40 mg/h (1- 4 ml/h)**

Adult: maximum rate 4mg/min (exceeding this rate increase risk of ototoxicity).

Children: maximum rate of 0.5mg/kg/min.

GTN (GLYCERYL TRINITRATE) INFUSION

Preparation: 50mg/10mls Syringe Pump: 50 mls

Method 1:

LOWEST CONCENTRATION DOSE: 100mcg/ml

Dilute 5mg (1ml) of IV GTN with 50mls of Normal Saline.

Rate: 3mls/hour = 5 mcg/min (Document Rate on Syringe Pump & in Patient's Notes)

5mg in 50 mls

50 ml —> 5mg —> 5000mcg

1 ml —> 5000 / 50 —> 100 mcg

1 ml /hr —> 100 mcg /hr —> 100 mcg / 60 min —> 1.66 mcg / min

3 ml /hr = 5 mcg /min

Starting dose of infusion **5-20 mcg/min (3-12ml/h)** Can increase up to **200mcg/min (120ml/h)** Monitor BP every 5 minutes

Max concentration: 400mcg/ml. Higher concentration will be adsorbed to tubing

Starting dose 5mcg/min (3ml/hr), increase every 5mcg/min every 3-5 mins to 20mcg/min (12ml/h). If no response at 20mcg/min, may increase by 10-20mcg/min every 3-5 mins

Method 2:

HIGHEST CONCENTRATION: 400mcg/ml

20 mg IV GTN (4ml) dilute with NS to become 50 ml

Rate : 1ml/hr = 10mcg/min (Document Rate on Syringe Pump & in Patient's Notes)

20mg in 50 mls NS

50 mls —> 20mg —> 20000mcg 1 ml —> 20000 / 50 —> 400mcg

1 ml /hr —> 400 mcg / hr —> 400mcg / 60min —> 6.67 mcg/min

1 ml /hr = 6.67 mcg/min

1.5 ml/hr = 10 mcg/min

Starting dose of infusion **5-20 mcg/min (0.75-3ml/h)**

Can increase up to **200mcg/min (30ml/h)**

Monitor BP every 5 minutes

Max concentration: 400mcg/ml. Higher concentration will be adsorbed to tubing

Starting dose 5mcg/min (0.75ml/hr), increase every 5mcg/min every 3-5 mins to 20mcg/min (3ml/h). If no response at 20mcg/min, may increase by 10-20mcg/min every 3-5 mins

HEPARIN INFUSION (For DVT / Pulmonary Embolus)

UFH is no longer the standard treatment in DVT & PE. Use LMWH

Bolus dose : 80 IU/KG Infusion Dose : 18 IU/kg/hr

Preparation: 5000 IU /ml (BLUE label)

Syringe out 2 mls (10,000 IU)

Dilute with 10 ml of Normal Saline in 20 mls syringe

1 ml = 1000 IU

Dilution after IV Heparin given at **1 ml/h**

HUMAN ALBUMIN 4% (FLUID RESUSCITATION ,HYPOALBUMINEMIA)

Usual available preparation: Human Albumin 20%

100 cc Human Albumin 20% dilute with 400 cc Normal Saline 0.9% Infusion rate: 10-20 ml/kg over 1 hour

HYPERTONIC SALINE

Hypertonic saline may be more effective than mannitol in lowering intracranial pressure but no difference was found in short- term mortality.

Dose: 3-5 ml/kg Nacl 3% over 30 to 60 minutes

An adult usually 250 ml of Nacl 3% bolus preferably through CVL (due to high osmolarity & tonicity) Aim Na level 145-155 mmol/l.

KETAMINE

Preparation: 200mg in 20ml vial

Induction dose for INTUBATION: 1-2mg/kg

Analgesia: 0.2-0.3 mg/kg bolus or 0.3mg/kg in 100ml Ns over 15min less feeling unreality.

Procedural Sedation Analgesia (PSA): 0.5-1.0 mg/kg and repeat 1/3 of the dose for 5-10 minutes

Or preferred: Ketamine and Propofol (Ketafol- 2 different syringes): 0.3mg/kg of Ketamine (analgesic dose) and propofol (sedative dose) titrate 40mg + 20mg + 20mg

Alternative for PSA

Ketamine and Propofol (Ketafol- in 1 syringe)

Ketofol (1:1 mixture of 5ml ketamine 10 mg/mL and 5ml propofol 10 mg/mL-) given aliquot **3 ml** till effect The median dose of medication administered was ketamine at 0.75 mg/kg and propofol at 0.75 mg/kg.

LABETALOL

Preparation: 25mg/5ml

Bolus:

Non diluted:

Give 1ml= **5mg** bolus titrate up every 5 minutes up to 25mg (1 ampule). **Maximum dose 200mg**

Aim SBP not to lower than 160mmhg.

Do not give bolus 1 ampule (25mg) stat

Continuous infusion:

Transfer **100mg (4 amp) into 20ml** syringe

(May be given undiluted) 1ml= 5mg run **0.5-2mg/min (**6ml/h to 24 ml/H)

Dilute to final concentration of 1mg/ml, eg: 200mg (8 ampules) in 200ml NS/D5%

Hypertensive crisis: **0.5-2mg/min** (run @ 30ml/Hr-120ml/Hr). Max total dose 300mg

NOT according to body weight

Hypertension in pregnancy: 20mg/hr, doubled every 30 mins. Max dose 160mg/hr. For hypertension bleed aim MAP reduction 25% from initial highest BP within 3-12 H.

- Incompatible with sodium bicarbonate
- Patient should receive drug in supine or left lateral position. Avoid raising patient into upright position within 3 hours which may cause excessive postural hypotension.
- Avoid severely elevated blood pressure to drop rapidly which may cause catastrophic reaction eg. cerebral infarction.

LIGNOCAINE INFUSION

Second option for stable Ventricular Tachycardia after Amiodarone

Preparation:

Lignocaine Injection 2% for IV (plastic ampule) not IM (glass ampule) 100mg in 5mls (20mg in 1 ml)

Dilute 400 mg in 50 ml D5% (8mg/ml)

Dose: (Cardio protocol)

0.125 ml/kg (1mg/kg) (Max 3mg/kg) over 3 mins (for 50 kg will be **6.25 ml bolus for 3 min**) followed by 0.5ml/kg (4 mg/min) x 30 mins

for 4mg/min= 240mg/h—> **30ml/h for 30 mins**

0.25ml/kg (2 mg/min) x 2 hours

for 2mg/min= 120mg/h—> **15ml/h for 2 hours then**

0.125ml/kg (1 mg/min)= 60mg/h—> **7.5ml/h as maintenance**.

MAGNESIUM SULPHATE

Preparation: 2.47 gm / 5mls (1 amp)

For Pre-eclampsia / Eclampsia

Loading dose:

4gm (8ml) in 20ml N/S run @ 80ml/Hr (over 15 min)

Maintenance dose:

1gm (2ml) in 50ml N/S run @ 50ml/Hr (1gm/Hr). Continue maintenance @ 1gm/Hr till delivery

Check patellar reflex before & during infusion, RR > 16, Urine Output > 25mls/hr

For Severe Asthma

Dosage:

Dilute 2 gm (4 mls) in 20cc NS

Give using syringe pump over 20 minutes (run 60ml/hr)

For Tetanus (Management of autonomic dysfunction)

Dosage:

Magnesium sulfate, 40 milligrams/kg IV loading bolus, then

2 grams/h (1.5 grams/h if ≤45 kg) continuous infusion (Dilute 4 gm (8 mls) in 20cc NS run 10ml/h) to maintain blood level of 2.0–4.0 mmol/L

Used in torsades de pointes with long QT interval

Dosage:

2 grams IV over 2 min, followed by infusion of 1–2 grams/h

MANNITOL INFUSION (Adult)

Preparation: 20 %

Dose: 1g / kg

To run over 30- 60 minutes

Use microchamber Drip or infusion pump

Calculations:

Body weight 50 kg

Mannitol 20 % meaning 20g in 100ml

Total volume of Mannitol needed (1 gm/kg) = 50g

= (100ml/20g) X 50g

=250 ml of Mannitol 20 % to run in 30 minutes

or **Rule of 2** ie 200ml of 20% mannitol run over 20 minutes.

MIDAZOLAM-MORPHINE SEDATION (Adult)

Preparation:

Morphine 30 mg (3 mls) + Midazolam 30 mg (6mls) Dilute with Normal Saline to **30 mls** in a 50mls syringe

Rate:

1mls/hr = 1 mg/hr of Midazolam and Morphine

Dose:

Infusion starting dose: 3ml/hr (3 mg/hr)

To titrate to desired effect

Bolus dose may be required for faster effect (3-5mls), followed by infusion dose

MIDAZOLAM-FENTANYL SEDATION

Sedation of choice post intubation especially for hyperactive airway such as status asthmaticus (advantages of less tachycardia and no increase in salivation)

Preparation:

300mcg (3 ampoules) of Fentanyl + Midazolam 30 mg

Dilute with NS to become 30mls in 50mls syringe

Rate:

1mls/hr = 1 mg/hr of Midazolam AND 10 mcg/hr of Fentanyl

Dose:

Infusion starting dose: 3ml/hr To titrate to desired effect

Bolus dose may be required for faster effect (3-5 mls), followed by infusion dose

N-ACETYLCYSTEINE (NAC) INFUSION

NAC DOSE FOR ACUTE LIVER FAILURE IN PCM POISONING

Preparation:

2 gm / 10 mls (1ml = 200mg)

Dosage:

- 150mg/kg (Maximum 15g) in 200 mls of Dextrose 5% over 15 – 60 mins
- Then 50mg/kg (Maximum 5g) in 500 mls of D5% over 4 hour
- Then 100 mg/kg (Maximum 10g) in 1 litre of D5% over 16 hours

e.g. : Body weight = 70 kg

150 mg/kg = 150 x 70 = 10500 mg = 10.5 gm

1 ampule = 2 gm

10.5 gm = 10.5/2 = Approximate 5 ampules

5 ampules of N-Acetylcysteine in 200 mls of Dextrose 5% over 15 min

NALOXONE for Opiod Overdose

Preparation: 0.4 mg / ml

Dose:

I/V 0.4 mg – 2.0 mg

Repeated dose with interval of 2 – 3 mins Max: 6 mg

Naloxone infusion:

0.4 mg/kg in 30ml at 1ml/H (0.01mg/kg/h)

NORADRENALINE INFUSION

Preparation: 4mg/4mls (1 amp)

Dilute 4.0 mg (4mls) of Noradrenaline with 46 mls of D5 in 50 mls syringe.

Rate: 1ml/hr = 1mcg/min

Dose: 2 – 20 mcg/min. Titrate accordingly to desired response (0.05-0.5mcg/kg/min)

Calculations:

- Dilute 4mg Noradrenaline in 50mls D5 **(Preferably use single strength)**
- 50mls —> 4mg —> 4000mcg
- 1ml —> 4000/50 = 80mcg
- 1ml/hr —> 80mcg/hr —> 80mcg/60min —> 1.3mcg/min
- 1ml/hr = 1.3 mcg/min (Max infusion rate 25ml/h for a 65kg patient)

OMEPRAZOLE/ PANTOPRAZOLE

Preparation: 40mg per vial

Dose for severe Upper Gastrointestinal Tract (UGIT) Bleeding: 80mg bolus then 8mg/ hour for 72 hours

IV Bolus:

Omeprazole: 80mg (2 vial) + 20ml solvent and give over 5 min (max rate 4ml/min)

Pantoprazole: 80mg (2 vial) + 20ml NS and give over 2-15 min

IV Infusion:

40mg (1 vial) + NS = 50ml Run 8mg/hour (10ml/ hour)

PHENYTOIN INFUSION (ADULT)

Preparation: 250 mg / 5mls

Loading dose:

15-20 mg/kg (Maximum dose 1.5 gm)

Dilute in 100 mls of Normal Saline (final concentration should not exceeding 10mg/ml) Use microchamber or Infusion pump

Run over 1 hour (Rate not exceeding 50 mg/minute)

Maintenance dose:

IV or Oral: 100 mg every 6 to 8 hours

Preparation: 10mmol in 10ml with 1mEq/ml bicarbonate.

SODIUM BICARBONATE (NaHCO3) 8.4%

Indication for severe metabolic acidosis

Dosage depends on Base Excess/ HCO3 level and body water of patient. It is recommended to give bicarbonate through a central venous access.

Calculation:

(24 - pt's HCO3) or BE X BW X 0.6 X 1/3= Z ml

And only give 1/2 from Z or BE X BW/10 (Frank Shan)

IV Slow Bolus for Emergency case:

Give undiluted solution. Rate not exceeding 10mEq/minute (equivalent to 10ml/minute) For infants, dilute 1mEq/ml solution 1:1 with water for injection.

IV Infusion:

Dilute with D5 to a maximum concentration of 0.05mEq/ml, run over 2 hours.

Maximum rate is 1mEq/kg/hr

Indication for Alkaline Diuresis

(Salycylate Poisoning or Rhabdomyolysis)

75 ml of IV Sodium Bicarbonate 8.4% + 425ml of Dextrose 5% (becomes NaHCO3 1.26%) and run accordingly to 1.5 to 2 times patient maintenance.

Urine PH aim to achieve more than 6.5

TRANEXAMIC ACID

Bolus: 1gm over 10 mins

Infusion: 1gm in 500ml N/S over 8Hrs

Line should not mix with blood transfusion

VITAMIN K

For Bleeding in over warfarinization (not for pt with any heart valve replacement)

Major bleed (Life/Limb threatening)

IV Vitamin K 5 mg

Dilute dose in minimum of 50ml NS (run over 30 mins (@ 50ml/Hr) (rate: 0.16mg/min)

Non- major bleed

IV Vitamin K 1-3 mg

Dilute dose in minimum of 50ml NS

run over 30 mins (@ 50 ml/Hr) (rate:0.03-0.1mg/min)

Maximum infusion rate: 1mg/min

IV route should be used in major bleeding patient. IM route should be avoided due to risk of hematoma formation.

MAJOR ELECTROLYTE CORRECTION REGIMEN

Hyponatremia

Rapid correction should not exceed more than 12 mmol/ 24 H or 0.5 mmol/H to avoid Osmotic Demyelinating Syndrome (ODS); previously Central Pontine Myelinosis (CPM)

Calculation.

First, calculate the expected change in serum sodium from the infusion of a liter of saline solution.

Second, determine the portion of the liter required to raise the sodium the desired amount

Third determine duration for correction to avoid ODS

Examples.

Correction for 50 y/o male with weight 70kg having seizure secondary to Na 108mmol/l, using 3% Normal saline (consist of Na 513mmol/Liter) -if using NaCl 0.9% consist of 154mmol/L will lead to overload.

1- **Total sodium deficit**= Total Body Water (TBW) X (desired Na - current Na)

[0.6 X BW] X (130 - current Na)

[0.6 X 70] X (130- 108) = 924 mmol/l

2- **NACL 3%**—> 513mmol per Liter

924 mmol =(1000ml/513) X 924

= 1800 ml of NACL 3%

3- **Duration**

0.5 mmol/H—> 22/0.5= 44 hours

Thus infusion rate of NACL 3% will be 41ml/H.

Hypernatremia

Calculation of total body water (TBW) deficit.

TBW deficit (L) = TBW X [(measured Na/ Normal Na) - 1]

Example

Patient BW 60kg with Na 170mmol/l

Calculation of TBW deficit = (60kg X 0.6) X [(170/150) -1]

= 4.8 liter

To avoid cerebral oedema correction should slower within 48H to 72 H Thus to give Dextrose 5% 4800ml over 48H = 100ml/h

or if pt intubated with Ryle's tube may give water through the RT in divided dose ie 2.4L/day ie 600ml of water every 6 hly.

Hypokalaemia

Do an ECG- if have changes ie u waves, need a fast correction or patient is symptomatic such as in Periodic Paralysis

Rule of 2 ie 2 gram of KCL in 200ml of Ns run for 2 hours or

Dilute 2g in 100ml NS

Maximum 0.4mmol/kg/hour (20-30mmol/hour) 1 g KCL = 13.4 mmol K

if patient is 50kg X50= 20mmol—> 1.65 g KCL over 1 hour

infusion Maintenance: 2-4 mmol/kg/day

If patient 50kg—> for 2 mmol/kg/day will be 100mmol—> 7.5g of KCL per day.

If patient in 2 liters of maintenance drip ie 4 pints of NS thus may put 2 g KCL in each pints.

Peripheral infusion: Maximum concentration is 100mEq/100ml. Maximum rate is 10mEq/h.

Central infusion: Maximum concentration is 40mEq/100ml. Maximum rate is 40mEq/h.

Hyeprkalemia

I/V slow bolus 10mls of 10% Calcium Gluconate

The FIRST medication to be given immediately

Given over 2-5 minutes

Cardiac (ECG) monitoring

Effect should be evident within a few minutes and last 30-60 minutes

Can be repeated once or twice if necessary – titrate against ECG changes

Avoid use in hyperkaleamia secondary to digoxin toxicity

I/V bolus 50mls of Dextrose 50% (Glucose not required if HYPERGLYCEMIC/DKA)

I/V bolus insulin 10 IU

IV Sodium Bicarbonate (IV NaHCO3) – Consult with Emergency Physician. Given only if patient in SEVERE METABOLIC ACIDOSIS.

Oral calcium polystyrene sulphonate – orally every 6 hours. Effect takes 1-2 hours

Consider Renal replacement therapy (dialysis).

The dilution and administration of Dexmedtomidine,Succinyl choline,Atracurium etc are mentioned in previous chapters.

Dexmedtomidine 600mg diluted to become 60ml in NS as an infusion through syringe pump.

Atracurium 150 mg diluted to become 50 ml in NS as an infusion through syringe pump.

CHAPTER 10

LET'S BRAIN STORM.

Hi everyone, I think you may have now understood the basics of ventilator and care of patients on ventilator. Here I am giving you some scenarios which will help you refresh and register what you have learned so far.

ALLTHE VERY BEST;LET US SAVE SOME LIVES.

SCENARIO – 1

Mr.Balakrishnan 41 years old male obese with DM,HTN, hyperlipidemia, covid suspect admitted to icu with complaints of laboured breathing, restlessness, profuse sweating and anxious.

a) Why do you think patient have laboured breathing?

b) What you will primarily assess?

c) What will be your intervention?

After connecting patient on CPAP , patient become more agitated.

What may be the reason?

The patient's current BP is 165/80mmHg.

What does it indicate?

Should you go with antihypertensives?

After 2 hours ABG done , shows pH – 7.11, PCO2- 92 mmHg, PO_2- 70mmHg, HCO3- 24mmol/L. Patient seen apparently normal.

What you will prepare for; if patient condition improves ?

What you will prepare for; if patient condition worsens ?

SCENARIO -2

Mr. Narayanan 72 year old male brought to casualty with the state of unconsciousness, profuse sweating, with expiratory stridor. Patient was intubated and put on pressure cycled ventilation. After an hour the nurse noted varying tidal volume on ventilator showing ETV less than ITV.

What will be your assessment?

Still same problrem persists then what will be the probable cause?

What will be the findings or changes seen on ventilator?

What will be your further assessment for the probable cause mentioned above?

SCENARIO- 3

Mr. Chakrapani 58 years old male known case of ARDS with CKD – V on MHD (2/7) on 2nd day of ventilator - volume A/C, RR- 12/ mt, VT- 400ml, FiO2 – 40%, PEEP – 8cmH2O, plateau pressure is 18cmH2O.

ABG done. The values are pH- 7.2, PCO2 – 60mmHg, PO2- 72 mmHg, HCO3- 16mmol/L, K+- 5.8, Lactate- 2.1mmol/L, BE- -10mmol/L. SPO2 – 92%. Vital signs are stable.

What is your interpretation on ABG?

What will be the primary management?

Any changes in the ventilator setting to be made?

If acidosis in not corrected , what will be the further management?

SCENARIO - 4

Mr. Brijesh 36 year old male known case of RHD, AF on OAC, admitted with progressive drowsiness, pallor, cold peripheries, tachycardia. h/o poor drug compliance.

What will be the initial assessment and management?

VBG done and it shows pH – 7.30, PCO2- 40mmHg, PO2- 60mmHg, HCO3- 20, Hb 6.2. Patient's vital signs checked and the values are BP – 90/60mmHg, PR- 160/mt, RR- 32/mt, SPO2 – 92%.

What will be the plan of care?

What will be the expected lab abnormalities for this patient?

Chest X ray shows bilateral pleural effusion. ECG showing AF with heart rate 160/mt. State the plan of action?

Any electrolyte imbalance expected with ECG changes?

SCENARIO- 5

Mrs. Devi admitted with fever, cough and tachypnoea since 2 days. On examination patient is drowsy but is arousable . Vital signs are temperature – 101 f, PR – 104/mt, RR-42/mt, BP- 111/74mmHg SPO2- 85%. She is known case of asthma,

COPD, DM. Patient is connected to oxygen mask with O2 5L/mt but patient is still drowsy.

What may be the cause of drowsiness?

What is your recommended oxygen delivery device for COPD patients?

After sometime patient developed gasping for breath. Steep fall in saturation to 60%. Immediately intubated and connected to ventilator . The ventilator settings are volume A/C, TV- 480ml, RR- 16/mt, PSV- 12/mt, PEEP- 10cm, FiO2 – 100%. After 1 hour ABG done and the values are pH – 7.45, PCO2- 38mmHg, PO2- 55mmHg, HCO3- 25mmol/L, Lactate – 2.25mmol/L.

What are the precautions to avoid VILI in COPD patients?

What is the role of the bronchodialators in improving hypoxia in COPD patient?

SCENARIO- 6

Mr. Palani 45 years old male patient came in ED with head injury. On examination patient is unconcious , deep, shallow breathing. The vital are temparature –101f, PR- 84/mt, RR- 14/mt, BP- 100/70mmHg, SPO2- 80%. Patient intubated with7.5mm ET tube and connected to ventilator. The ventilator settings are volume A/C, TV- 460ml, PEEP- 8cm, PSV- 10cm, FiO2- 80%, RR- 20/mt. CT head done and patient is diagnosed as SAH. Patient is unable to wean from ventilator.

What will be the next line of management?

On 2nd day of tracheostomy patient developed acute breathlessness and fall in saturation.

What will be the probable cause?

Precautions to avoid dryness or secretion?

How do you clean and suction a tracheostomy tube?

SCENARIO –7

Mr. Shankar 64years old came in medical ICU with dyspnea, sweating and irritability. On examination temperature –98f, RR- 42/mt, PR- 132/mt, BP- 200/100mmHg, SPO2- 85%. Patient is known case of CKD-V on MHD 2/7, DM, HTN.

What will be the initial diagnosis?

Which is the preferred mode of ventilation for acute pulmonary edema?

After half an hour patient saturation improved with BIPAP and a shot of diuretics. Still BP is uncontrolled.

What will be the further management?

ABG taken and the values are pH- 7.29, PCO2- 40mmHg, PO2- 60mmHg, HCO3- 16mmol/L, lactate- 4mmol/L, BE- -10.

What is the interpretation of ABG and management?

After 2 hours patient is slowly developed abdominal distention.

What is the reason for abdominal distension and how will you manage?

What are the other expected complications of BIPAP?

Scenario 8

Mr. Kandasamy 63-year-old man presented with acute respiratory failure. He was intubated and given mechanical ventilation in the ED. The initial settings for the mechanical ventilator were Vt, 600 mL; respiratory rate, 10 breaths per minute; and PEEP, 5 cm H_2O, FiO2 – 100%. The patient was transferred to the ICU. He was deeply sedated and paralyzed, and no spontaneous breathing effort was noted. ABG taken and the values are pH- 7.25, PCO2 – 72mmHg, PO2- 75mmHg, HCO3-

25mmol/L. After seeing ABG, decreased the Vt to 500 mL and increased the respiratory rate to 20 /mt, PEEP increased to 10cmH2O.

What changes would you expect to occur as a result of the intervention?

During mechanical ventilation, what primarily influences oxygenation?

What Vt range is appropriate for an adult male with an ideal body weight of 66 kg?

Which mode of ventilation provides full support for all breaths (timed and patient triggered)?

How does PPV affect cardiac output?

What is the benefit in selecting a dual-control adaptive pressure ventilation mode?

By which mechanism can hypercapnia increase intracranial pressure?

SCENARIO – 9

Mrs. Vasantha 72years old admitted with difficulty in breathing, fast rapid breathing, fever, cough and cramps. On examination patient looks confused. Vital signs are temp-103f, PR- 110/mt, RR- 40/mt, BP- 122/60mmHg, SPO2- 90%. Patient is connected to oxygen mask with O2 5L/mt. ABG done and the values are pH- 7.45, PCO2 –20mmHg, PO2- 60mmHg, HCO3- 22mmol/L, Ca2+ - 0.5mmol/L. she is known case of CVA with left hemiparesis.

1) What is the cause of respiratory alkalosis?
2) What is the first line of management for alkalosis in this patient?
3) Find out the reason for cramps in this patient?
4) What are the other methods to prevent CO2 wash out?

SCENARIO – 10

Mr. Chandran 48 years old covid positive patient on 2nd day of ventilation, with 7.5mm ET tube fixed at 22cm lip level, on vol SIMV, RR- 14/mt, TV – 360ml, PSV- 10 cm, PEEP- 8cm, FiO2- 60%. Patient is coughing and restless. The nurse noted high PIP alarm in red on ventilator.

1) What could be the reason for high PIP alarm?
2) What will be the intervention?
3) How will you differentiate the alarm is because of airway resistance or lung compliance?
4) Will pneumothorax cause high PIP alarm?
5) If the alarm is because of airway resistance, what will be the possible causes?

WELL DONE!!! WE HAVE LEARNED A LOT BUT REMEMBER-

We are what we repeatedly do. Excellence, then, is not an act but a habit.

ALL THE BEST AND GOD BLESS!!