Breathe Easy: RT Student Resource for Mechanical Ventilation

BREATHE EASY: RT STUDENT RESOURCE FOR MECHANICAL VENTILATION

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Additional content was also adapted from:

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A list of more detailed changes can be found in Changes from Adapted Resource.

Cover photo of Fanshawe Respiratory Therapy students class of 2024.

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ABOUT THIS BOOK

Mechanical ventilation is a complex and dynamic field, and this Open Education Resource (OER) covers a wide range of topics, including the physiology of breathing, indications and contraindications for mechanical ventilation, types of mechanical ventilators, modes of ventilation, patient-ventilator interactions, liberation from mechanical ventilation, and management of complications associated with mechanical ventilation. With contributions from experts in the field and evidence-based information, the purpose of this OER is to serve as a supplementary tool for respiratory therapy students and respiratory therapists new to acute care practice, to enhance their comprehension of mechanical ventilation, as it is a necessary component for entry to practice into the respiratory therapy profession in Canada.

While the fundamentals of mechanical ventilation remain consistent across different age groups and patient populations, the emphasis of this resource is specifically on the ventilation of adult patients.

Topical Overview

This OER provides a foundation of mechanical ventilation concepts and basics. By the end, you will understand the basics of ventilation, settings, ventilation modes, how to initiate ventilator settings and when and why to make changes based on patient status. Topics include:

- What ventilation is and how it affects the lungs.
- Common ventilator modes and basic ventilator settings.
- How to set up a ventilator based on patient demographics and their clinical presentation.
- Blood gas interpretation and how to change the ventilator based on blood gas values.
- Specific pathophysiologies and how they impact ventilator strategies and settings.
- Common misconceptions and inappropriate ventilation strategies.

Accessibility Statement

We are actively committed to increasing the accessibility and usability of the textbooks we produce. Every attempt has been made to make this OER accessible to all learners and is compatible with assistive and adaptive technologies. We have attempted to provide closed captions, alternative text, or multiple formats for on-screen and off-line access.

The web version of this resource has been designed to meet Web Content Accessibility Guidelines 2.0, level AA. In addition, it follows all guidelines in Appendix A: Checklist for Accessibility of the *Accessibility Toolkit – 2nd Edition*.

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CHANGES FROM ADAPTED RESOURCE

This book was adapted from Basic Principles of Mechanical Ventilation by Melody Bishop, the following content was revised, changed or added to create this version.

Overall	Updated formatting and images/diagrams.
Chapter 1	1.2 Mechanics of breathing. Added info on Boyle's Law. 1.3 FRC, Intrinsic PEEP and Respiratory Pressures. Added detailed definitions on pressures in respiratory physiology. 1.4 Ventilation. New section created.
Chapter 2	Changed title from Oxygenation and Fundamentals of mechanical ventilation to Fundamentals of mechanical ventilation. 2.0 Chapter overview. All new content created. 2.1 Ventilators. The basics. All new content created. 2.2 The physiology of a mechanically delivered breath. Added details including graphics of pressures in the lungs. 2.3 A brief history of control and spontaneous modes. Moved from chapter 3 2.4 Phase variables. All new content created. 2.5 Oxygen on a ventilator. No change 2.6 Supplemental oxygen and mechanical ventilation. No change 2.7. The relationship between PEEP and FiO2. No change 2.8 Basic ventilation modes. The ventilation family tree. Moved from original Chapter 3 2.9 Ventilation modes: Alphabet Soup. Moved form original Chapter 3 and added "The 10 maxims to understanding ventilation modes." 2.10 Settings and definitions. Moved from original Chapter 3 2.11 ventilation settings table. Moved form original chapter 3
Chapter 3	Changed Chapter title from Basic ventilator modes and settings to Ventilator Settings 3.1 Mandatory settings in control mode. New content created and imported from original Chapter 5 (The relationship between pressure and volume) 3.2 Choosing Initial Settings for a Control Mode. Moved from original Chapter 5 3.3 Idea Body Weigh. Moved form original chapter5 and added details on tidal volume calculations including a video. 3.4 Volume Control. Added details and graphic diagram. 3.5 Pressure Control. Added details and graphic representation. 3.6 Ventilator alarms. Moved form chapter 5. Added details.
Chapter 4	Adapted from original Chapter 7. Changed photo and added information to " What is Non invasive ventilation" 4.2 added information, including the table Current guidelines 4.3 added information and graphic. 4.5 Added information and video 4.7 new content Other sections were not changed from the original content.
Chapter 5	5.1 Using Control modes of ventilation. Moved from original chapter 4 and added details on ventilation modes including graphic diagrams. 5.2 The Time variable. Moved from original chapter 4 and added details on Flow related to the time variable. 5.3 I: E. Moved from original Chapter 4 and added details including a video. 5.4 Putting it all together. No change. 5.5 Volume Control ventilation. Moved from original chapter 4. Added details and graphics. 5.6 Pressure Control Ventilation. Moved from original chapter 4. Added details and graphics. 5.7 Other ventilation modes. All new content created.
Chapter 6	6.1 Spontaneous modes. A review. No change 6.2 Spontaneous modes vs control modes. No change 6.3 Indications and contraindications. No change 6.4 Types of spontaneous modes. Added details, graphic and a video. 6.5 Pressure Support Ventilation settings. Added details on Back up Ventilation. 6.6 A word of caution. No change 6.7 Volume Support Ventilation. All new content created. 6.8 Volume Assured pressure support. All new content created. 6.9 Proportional assist ventilation. All new content created.
Chapter 7	7.1 originally from Chapter 2. Image of the hand and a short paragraph kept, and the rest of the section is new. Remaining chapter is all new content.
Chapter 8	8.1 to 8.5 No change 8.6 Patient ventilator interaction. New content created.

Chapter 9	9.1 How much do I change ventilation. Added details and examples. 9.2 How much do I change oxygenation. New content. Added details and examples, including videos and graphic diagrams. 9.8 New content 9.9. Ventilation in ARDS. New content created. 9.10. Putting it all together. No change
Chapter 10	10.2 SIMV. Moved from original Chapter 6. 10.3 Additional weaning strategies. New content created

CHAPTER 1 | HOW THE HUMAN BODY BREATHES - PHYSIOLOGY OF VENTILATION

Chapter Outline

1.0 CHAPTER OVERVIEW

In order to understand mechanical ventilation, you must first have a basic understanding of how the human body is designed to breathe. Oxygen is the food of the human body. Every cell requires it to create energy and carry out its various functions. On the other hand, carbon dioxide (CO_2) is the garbage of the human body that every cell creates as a byproduct of its functions. It is commonly known that the lungs are the organ that take the oxygen from the air and bring it into the body, and that they expel (exhale) CO_2 at the same time. But what is the mechanism that triggers this to happen and how exactly does this exchange occur?

The effectiveness of Mechanical Ventilation in helping with treating various lung condition depends on how it is applied. Therefore, it is essential for respiratory therapists to have a comprehensive understanding of respiratory physiology to ensure appropriate application and prompt response to any complications that may arise. This chapter aims to provide the reader with a review of respiratory physiology concepts required for understanding principles of mechanical ventilation, including intrathoracic pressure changes associated with spontaneous breathing, negative pressure breathing, and positive pressure breathing. By gaining a good understanding of these principles, respiratory therapists can optimize the use of Mechanical Ventilation and improve patient outcomes.

Application

How and when will you apply the concepts you are learning in this chapter? The mechanism of breathing is fundamental to the real-world use of ventilators. The health care professional will benefit greatly from understanding the natural process that the mechanical ventilator is intending to replicate (or imitate as closely as possible) to better aid them in using a ventilator effectively in practice.

Learning Objectives

At the end of this chapter, you will be able to:

1. Explain the body mechanics of inspiration and expiration.
2. Identify key parts of the body involved in breathing.
3. Understand how surface tension contributes to lung recoil
4. Define FRC and Intrinsic PEEP and the importance to the alveoli.
5. Describe the basic pressure changes that occur in the lungs during the respiration process.
6. Describe the timing and components of the respiratory cycle
7. Describe minute ventilation, alveolar ventilation and dead space.
8. Describe compliance, resistance and time contact and their impact on breathing

Key Terms

In this chapter, you will learn about all of the following key terms. These terms will be used throughout this book, so it is important to take the time to master them and practice your recall often.

- respiratory system
- lungs
- lobes
- alveoli, alveolus
- oxygen (O_2)
- carbon dioxide (CO_2)
- inspiration
- expiration
- chemoreceptors
- diaphragm
- negative pressure
- positive pressure
- Functional Residual Capacity (FRC)
- Positive End-Expiratory Pressure (PEEP)
- surfactant
- cm H_2O
- Palveoli
- Ppleural
- P_{ao}
- Pleural space
- Compliance
- Resistance
- Time constant

Whenever these terms are first introduced in this chapter, they are bolded. However, if you need additional information about a term than what is provided here, you can research it in The Free Dictionary: Medical Dictionary.

1.1 ANATOMY OF BREATHING

In order to fully comprehend the principles of mechanical ventilation, it is important to have a thorough understanding of the intricate mechanisms that govern human respiration. This chapter serves as a brief yet comprehensive overview of the respiratory anatomy and physiology that is crucial for comprehending the nuances of mechanical ventilation. The **respiratory system** is responsible for breathing. The **lungs** are the major organ responsible for this. We have two lungs in the human body. The left lung has two **lobes**, or sections, while the right lung has three lobes.

> Please take a moment now to learn about alveoli (singular: *alveolus*), the small sacs inside the lungs that fill with air. Check out "The Alveoli in Your Lungs" (2018) from Healthline for a helpful description of what they look like and how they work.

Thinking of the lung as a balloon is a great simple analogy but it is important to review the physiology of the respiratory system to fully explain how the lungs behave. The lungs are not simply one big balloon. It is like they are made up of millions of little balloons all stuck together—similar to bubble wrap. These little balloons are referred to as **alveoli**.

The millions of "small balloons" are still able to take in the same overall volume of air as a single balloon of the same size but by dividing the volume of air into alveoli versus one big balloon, the total combined surface area of the lungs goes up exponentially. Why does this matter? Well, each individual alveolus is surrounded by blood vessels called "capillaries". Where the alveoli and capillaries meet is where the actual exchange of gases occurs. The large increase in surface area where this exchange can occur allows for more active areas of gas exchange, and this exchange can happen at a more efficient and rapid pace. Oxygen diffuses in from the alveoli to the bloodstream, and carbon dioxide diffuses out of the blood into the alveoli to be exhaled out of the lungs.

Apply Your Learning

Can you locate the lungs, the alveoli and the capillary network in the diagram below? Don't worry if the rest of the labels look confusing to you—you'll learn a lot more about the lungs as you progress through this course!

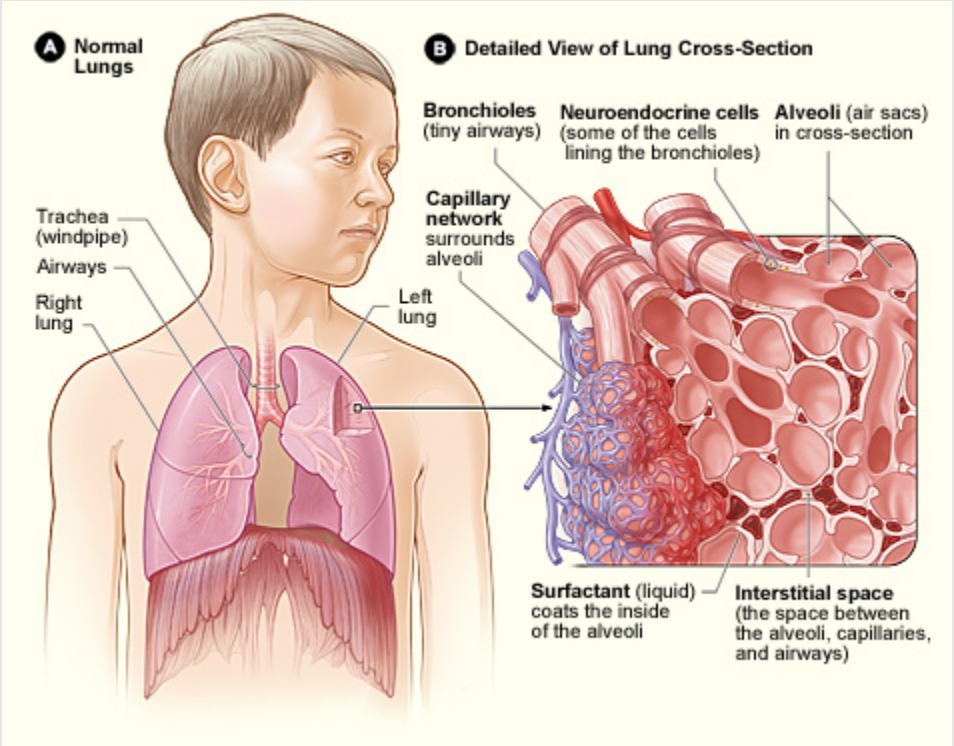


Figure 1.1.1: "Normal lungs with a detailed view of lung cross-section" by National Heart Lung and Blood Institute, CC0

1.2 MECHANICS OF BREATHING

Oxygen (O_2) is the food of the human body. Every cell in your body requires oxygen to create energy and carry out its various functions. On the other hand, **carbon dioxide (CO_2)** is the garbage of the human body that every cell creates as a byproduct of its functions. It is common knowledge that the lungs are the organ that take the oxygen from the air and bring it into the body and conversely expel (exhale) CO_2 at the same time. This process of taking in oxygen and getting rid of CO_2 occurs with every breath.

There are two parts of every breath:

1. **inspiration** (also called inhalation) and
2. **expiration** (also called exhalation)

During inspiration, your body takes in the air. Normal ambient air is made up of approximately 78% nitrogen and 21% oxygen, as well as a small amount of various other trace gases. This amount of oxygen is more than adequate for healthy lungs to take in and circulate through the body for normal daily activities.

But what is the mechanism that triggers a breath to happen, and how exactly does this exchange occur?

The human body has a specific baseline amount of oxygen that it must have in order to function. It also has a maximum level of carbon dioxide that it is comfortable with keeping in the body at any given time. At any point, if the level of O_2 drops below, or the level of CO_2 climbs higher than, the body's favoured level, the brain kicks in and asks the lungs to breathe in more oxygen or breathe out more CO_2.

Key Takeaway

The brain is the trigger that initiates a breath based on the body's "set baseline" for oxygen need or carbon dioxide (CO_2) removal.

How does this happen? The central nervous system has **chemoreceptors** that sense the levels of oxygen and carbon dioxide in the body. This sensing is done independent of each other. The central chemoreceptors measure the level of carbon dioxide, and when it gets too high or low, will trigger the brain to adjust how the body is breathing. Remember: the human body exhales CO_2 so if the central chemoreceptors measure that levels of carbon dioxide are too high in the blood, then they send a message that the body needs to breathe out more carbon dioxide. Conversely, the peripheral chemoreceptors are responsible for sensing the oxygen levels. If the blood levels of oxygen drop, this will send a message that the body needs to breathe in more oxygen. This signal will trigger a breath.

If you want to learn more about the role chemoreceptors play in respiratory drive, check out the entry on Chemoreceptors from Pathway Medicine.

Apply Your Learning

Why does your body breathe faster when you are doing cardiovascular exercise? What chemoreceptors are involved in this process?

When a message or "trigger" occurs by the chemoreceptors sensing a need for a breath, the brain asks for the breath by stimulating the **diaphragm** to contract. The diaphragm is the large muscle that lines the bottom of the lungs. In this animation, the diaphragm, located below the two lungs, is red and the lungs are blue.

One or more interactive elements has been excluded from this version of the text. You can view them online here: https://ecampusontario.pressbooks.pub/mcvresource/?p=29#video-29-1

Video: "Lungs Front Breathing Diaphragm GIF" via GfyCat [0:09]

The diaphragm is "glued" to the bottom of the lungs by the pleural space so that when the diaphragm contracts, this pulls the lungs down and open.

Object Lesson

Think of pulling open an accordion. As you pull on the end of the accordion, it gets bigger and fills with air.

Figure 1.2.1: Photo by music4life , Pixabay License

It is important to understand this concept as it relates to pressure and volume in the lungs. As the diaphragm contracts and pulls the lungs open, it results in a vacuum state in the lungs—**negative pressure**. Just like a vacuum, it sucks air into itself. That negative pressure in the lungs causes air to passively flow into the lungs as long as that negative pressure exists.

Object Lesson

Think of a slinky with one end in each hand. When one hand is placed lower than the other, the slinky falls towards it. When you move the other hand lower, the slinky falls in the other direction. In the same way, when there is lower/more negative pressure in lungs, the air rushes in until the pressure is equal or becomes higher in the lungs—then, the air exits the lungs.

Figure 1.2.2: Photo by Roger McLassus, CC BY-SA 3.0

Key Takeaway

If there is a negative pressure in a space, air will naturally flow to fill this space. Just like water flowing downstream, air always flows from higher pressure to lower pressure.

In other words, air flows into the lungs because a pressure gradient exists. During inspiration, in a normal spontaneous breath, as the diaphragm and respiratory muscles contract, thoracic volume increases, and pressure in the lungs decreases (pressure in the alveoli P_{alv}) decreases. Air moves into the lungs due to the pressure gradient between airway opening $\left(P_{ao}\right)$ and pressure in the alveoli $\left(P_{alv}\right)$. Boyle's Law explains the relationship between pressure and volume during breathing. When temperature remains constant, as the volume increases, the pressure decreases.

Watch:

"Boyles Law from Respiratory System – Anatomy & Physiology Online" By Primal Pictures 3D Anatomy & Physiology [1:56]

So how does air stop flowing into the lungs? Once the lungs sufficiently fill—based on the amount of air that the brain has decided it requires to satisfy its need—it turns "off" the diaphragm. Contraction stops and the muscle relaxes. The diaphragm is no longer pulling the lungs open. The vacuum has turned off and the negative pressure has subsided. It's important to remember that the lungs are full of air at this point. Air has been passively filling them for a while.

Object Lesson

Lungs act like balloons. When no other force is acting on them, the surface of each alveolus has a tension to it—exactly like a rubber outside of an inflated balloon. When nothing stops it, the lung naturally wants to empty, just like the elastic recoil of a balloon that isn't tied and is allowed to leak.

Figure 1.2.3: "Balloon Arch" by Ishmael Orendain, CC BY 2.0

A great example of the natural mechanics of the lungs is an archer's bow. The diaphragm actively pulls the lungs open like the hand on the back of the bow. As soon as that active force stops, the elastic recoil of the bow kicks in and resets it back to its relaxed state.

This collapsive force is accomplished by surface tension and is identical to what you notice

with balloons. The fuller the balloon is, the higher the tension wanting the balloon to shrink.

Figure 1.2.4: Photo via Pixnio, CC0

Thinking back to the flow of air related to pressure, at the beginning of exhalation when the lungs are full and the surface tension of the lungs is wanting to collapse the lungs, there is high **positive pressure** in the lungs. Think of how high the pressure gets in a balloon when it is full. Remember, air always flows from high to low pressure, so the air flows out of the lungs (exhalation).

Key Takeaway

The lungs act exactly like balloons when no outside force is acting (i.e., surface tension when full of air, or being pulled open by the diaphragm) on them or stopping them, they passively will want to deflate.

During expiration, thoracic volume (and lung volume) decreases, resulting in an increase in alveolar pressure (P_{alv}). Because (P_{alv}) is now higher than P_{ao}, flow changes direction, and air flows out of the lungs. Eventually, as air flows out of the lungs, at the end of expiration, P_{ao} and (P_{alv}) become equal, air movement stops.

To summarize, the cascade of events during a breath in and out, is all about the flow of air from HIGH to LOW pressures. It starts in inspiration, with the lungs having a negative (lower) pressure from the diaphragm contracting. Air flows in until the diaphragm stops contracting, which stops that negative pressure. Inspiration stops. At this point, the lungs are full of air and are experiencing a higher pressure than the outside world. Exhalation starts as air flows from high to low pressure once again. Thinking of balloons and Slinkys, the likely conclusion is that this passive flow of air out of the lungs (exhalation) would continue until the pressures have equalized. This is true of balloons but does not occur in the human body because of one very important principle: **intrinsic PEEP**. We'll learn all about this on the next page.

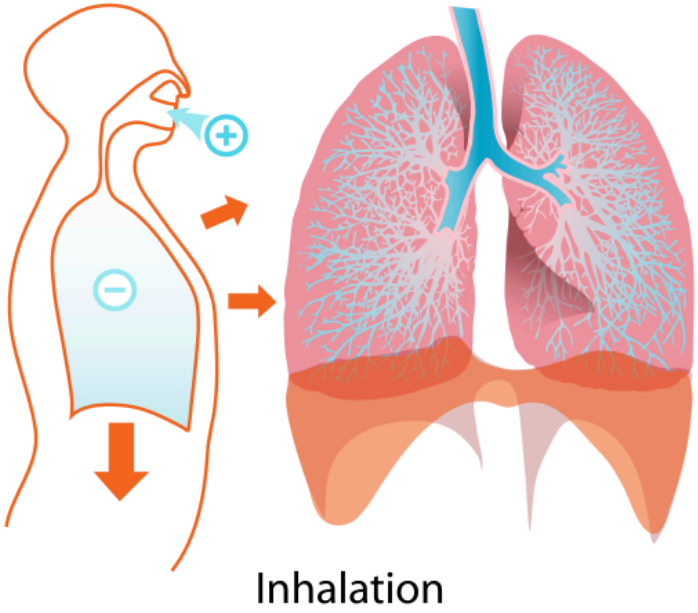


Figure 1.2.5: "Inhalation" by LadyofHats, CCO

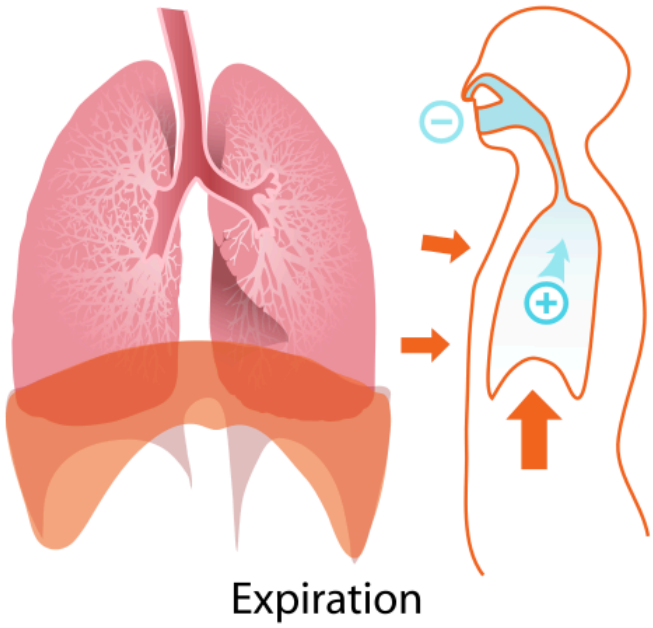


Figure 1.2.6: "Expiration" by LadyofHats, CCO

1.3 FRC, INTRINSIC PEEP AND RESPIRATORY PRESSURES

Going back to the balloon analogy, consider the process of blowing up a balloon. At what point is the balloon the hardest to blow up?

- When it is completely empty? or
- When there is a little air in it already?

An empty balloon requires very powerful and sustained breath to start inflating; but if there is a little air in the balloon already, you can blow it up with a gentler breath and you wouldn't have to push so hard.

The lungs are no different. The initial little bit of air in the lungs requires a higher amount of pressure in order to open up the lungs a little, while a lung with some air can inflate much more easily. This is where the human body is so smart. The body stops the lungs from fully emptying—it keeps a small amount of air in the lungs at all times. This is referred to as the **functional residual capacity (FRC)** of the lungs. A term for the air left in the body at the end of exhalation is the intrinsic (physiologic) **Positive End-Expiratory Pressure (PEEP).** PEEP is very important, as it protects the lungs from the high blowing pressures that would normally be needed to inflate them from empty—decreasing the risk of trauma. We will revisit this concept in subsequent chapters.

Key Takeaway

The lungs never fully empty. Some air remains in the alveoli at all times. This is known as FRC or intrinsic PEEP. Positive End-Expiratory Pressure (PEEP) refers to the pressure left in the lungs at the end of exhalation—also known as the residual air left in the balloon to stop it from fully collapsing.

Object Lesson

Think for a moment about a balloon that you have reused for three different parties. The first time you blew it up, the rubber seemed sturdy, but over time, it became more fragile and compromised. The fourth time you tried to blow it up,

the balloon popped even though it was not yet full. What happened here? The high pressure damaged the balloon over time. In the same way, your lungs can be damaged by consistent exposure to high pressure.

The main physiologic mechanism to achieve FRC is **surfactant,** a naturally produced lubrication that coats all the alveoli of the lungs. Surfactant stabilizes the surface of the alveoli so they do not collapse and helps facilitate the reserve of the FRC we explained earlier.

Object Lesson

You can think about surfactant as the body's own "door stop." It keeps the door slightly ajar so the latch doesn't close. In technical terms, surfactant decreases the surface tension on the alveoli. Surface tension is the force—just like the elastic of the balloon that wants to deflate the alveoli fully during expiration.

Photo by Conrad Bakker, CC BY-NC-ND 2.0

Pressures in Respiratory Physiology

When dealing with breathing, it is important to have a good understanding of the pressure changes in the lungs during inspiration, expiration and at rest. Because pressures in the lungs are fairly low, they are usually measured in cmH_2O and referenced to atmospheric pressure (P_{atm}), which is given the value zero. Any pressure above atmospheric pressure is considered positive pressure, and any pressure below atmospheric pressure is considered negative.

As you may have already guessed, cm H_2O literally refers to the weight of a centimeter (cm) of water (H_2O) on a given spot. The atmospheric pressure is $0\ cmH_2O$ and any additional pressure would be above that.

Object Lesson

In this representation, the zero point represents the reference pressure. Pressure values above zero indicate positive pressure, while pressure values below zero indicate negative pressure.

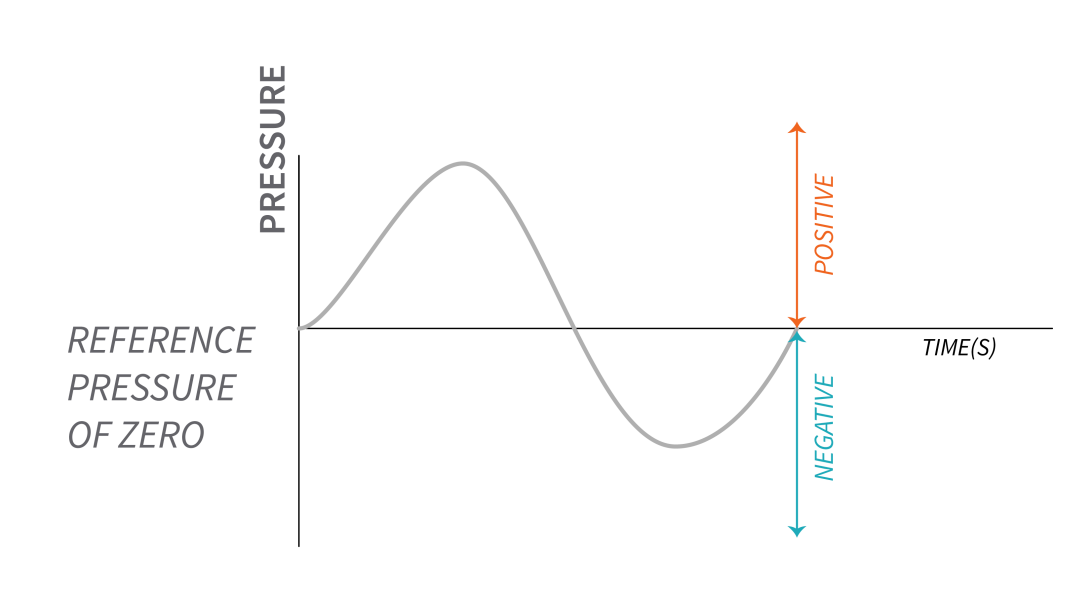


Figure 1.3.1: "Reference Pressure" by Freddy Vale, CC BY-NC-SA 4.0

Pressure unit equivalents for 1 atmosphere (atm)

760 mmHg

760 Torr

$1034\ cmH_2O$

Definition of Pressures and Pressure Gradients

In respiratory physiology, and inadvertently in mechanical ventilation, we will be referring to four main pressures and four important gradients. It is important to understand the impact of these pressures and gradients on respiration, so that we can apply physiology principles to mechanical ventilation, in order to safely ventilate patients.

Pressure at the airway opening, is sometimes called pressure at the mouth (P_m) or airway pressure (P_{aw}), or proximal pressure, when

mechanical ventilation is applied. This pressure is equal to atmospheric pressure. Similarly, the **pressure at the body surface** (P_{bs}) is equal to atmospheric pressure. Other pressures that are instrumental in the pressure gradients of breathing are the $P_{alveoli}$ (pressure in the alveoli) and the $P_{pleural}$ (pressure in the pleural space). **Pleural pressure** (P_{pl} or intrapleural pressure) is the pressure measured in the space between visceral and parietal pleurae. **Alveolar pressure** (P_{alv}) is determined by the volume of air in the alveoli and the pressure being exerted around the alveoli—by the pleural pressure or the diaphragm. this pressure is sometime referred to as intrapulmonary pressure. This pressure depends on the elastic recoil of the lungs, specifically how compliant the lungs are. A stiff lung will require more pressure to inflate.

$33 \text{ ft } H_2O$
14.7 psi
29.9 in. Hg

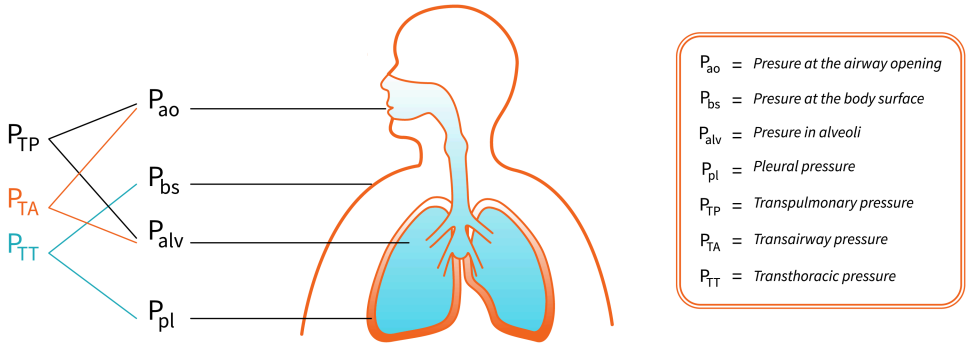


Figure 1.3.2: "Human Respiratory System" by Freddy Vale, CC BY-NC-SA 4.0

Four main pressure gradients determine different phases of the respiratory cycle, as follows:

Transairway pressure (P_{TA}) is the pressure gradient between airway opening pressure and alveolar pressure. $P_{TA} = P_{ao} - P_{alv}$. This pressure is reflective of airflow conditions, as it is the pressure required to generate flow through the airways. The gradient is always between airway opening and alveoli.

Transthoracic pressure (P_{TT}) describes the pressure gradient required to expand and contract lungs and the chest wall in the same time. $P_{TT} = P_{pl} - P_{bs}$. P_{TT} is equal to the outward recoil force of the thorax when there is no airflow. The stronger this outward recoil force, the greater the P_{TT}.

Transpulmonary pressure (P_{TP}) is the pressure gradient required to maintain alveolar inflation. $P_{TP} = P_{alv} - P_{pl}$. In mechanical ventilation this gradient is often referred to as distending pressure.

Transrespiratory pressure (P_{TR}) is the pressure gradient across the entire respiratory system, including airway opening, lungs, and chest wall. The components of this gradient are the respective gradients for airway, lungs and chest wall: the airway is represented by the transairway pressure (P_{TA}), lungs are represented by the transpulmonary pressure (P_{TP}) and chest wall is represented by transthoracic pressure (P_{TT}).

Now, let's revisit inspiration and expiration from a pressure differential viewpoint. Remember, the lungs are glued to the diaphragm and the surrounding thorax by the **pleural space**. Think of this space as a large suction that is all around the lung. Just like the word *suction* hints at, the pleural space is a negative pressure at rest. Before inspiration occurs, the pleural space negative pressure (-5 cmH_2O) keeps a small amount of air in the lungs (equal to $+5 \text{ cmH}_2O$) that cancels out the negative pressure in the

lungs. This equalization stops the flow of air in or out and puts the lungs in a "rest state" until the diaphragm contracts and causes a negative pressure.

Key Takeaway

Since the pleural space has a negative pressure, to equal zero, air must be left in the lungs!

As the diaphragm contracts, the volume of the thoracic space increases and pressure in the pleural space decreases (it becomes more negative in relation to atmospheric pressure). The pressure in the pleural space drops from about $-5\ \mathrm{cmH_2O}$ to about $-10\ \mathrm{cmH_2O}$ at end inspiration. The negative pressure in the pleural space is transmitted to the alveoli, so alveolar pressure (P_{alv}) becomes negative. Pressure at the airway opening is still atmospheric $(P_{ao} = 0)$ while P_{alv} is negative. Air flows from a point of high pressure to a point of low pressure. The gradient is always between P_{ao} (pressure at the airway opening or atmospheric pressure) and P_{alv}. This negative pressure gradient created from the contraction of the diaphragm, allows air to flow from airway opening into the lungs. Transpulmonary pressure (P_{TP}) gradient widens, allowing lung inflation. Transairway pressure gradient (P_{TA}) is about $-5\ \mathrm{cmH_2O}\ (0 - [-5])$. As volume of air builds up in the alveoli, alveolar pressure return to zero. Airflow stops at end inspiration, because there is no longer a pressure gradient present $(P_{alv} = P_{ao})$.

During expiration, respiratory muscles relaxation and elastic recoil of the lung tissue, results in a decrease in thoracic space and lung volume. Pleural pressure (P_{pl}) returns to about $-5\ \mathrm{cmH_2O}$. As soon as the diaphragm relaxes, and stops the suction, the volume of air and therefore pressure in the chest is now higher than the pressure at the mouth. This state reverses that pressure gradient and air flows out of the lungs—in other words, exhalation occurs—until the pressures equilibrate.

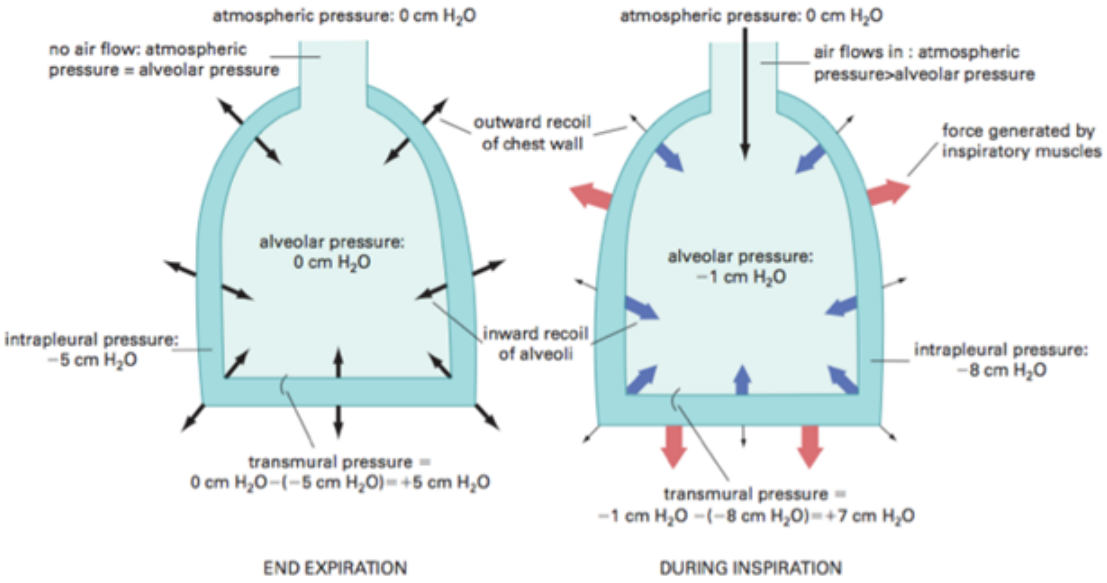


Figure 1.3.3: "Lung Pressures – Inspiration and Expiration"

Let's review the process of inspiration and expiration again, this time using graphics. Becoming familiar with visual depictions of

respiratory patterns is highly recommended, as graphics serve as valuable aids and tools in mechanical ventilation when it comes to recognizing issues and enhancing patient prognosis.

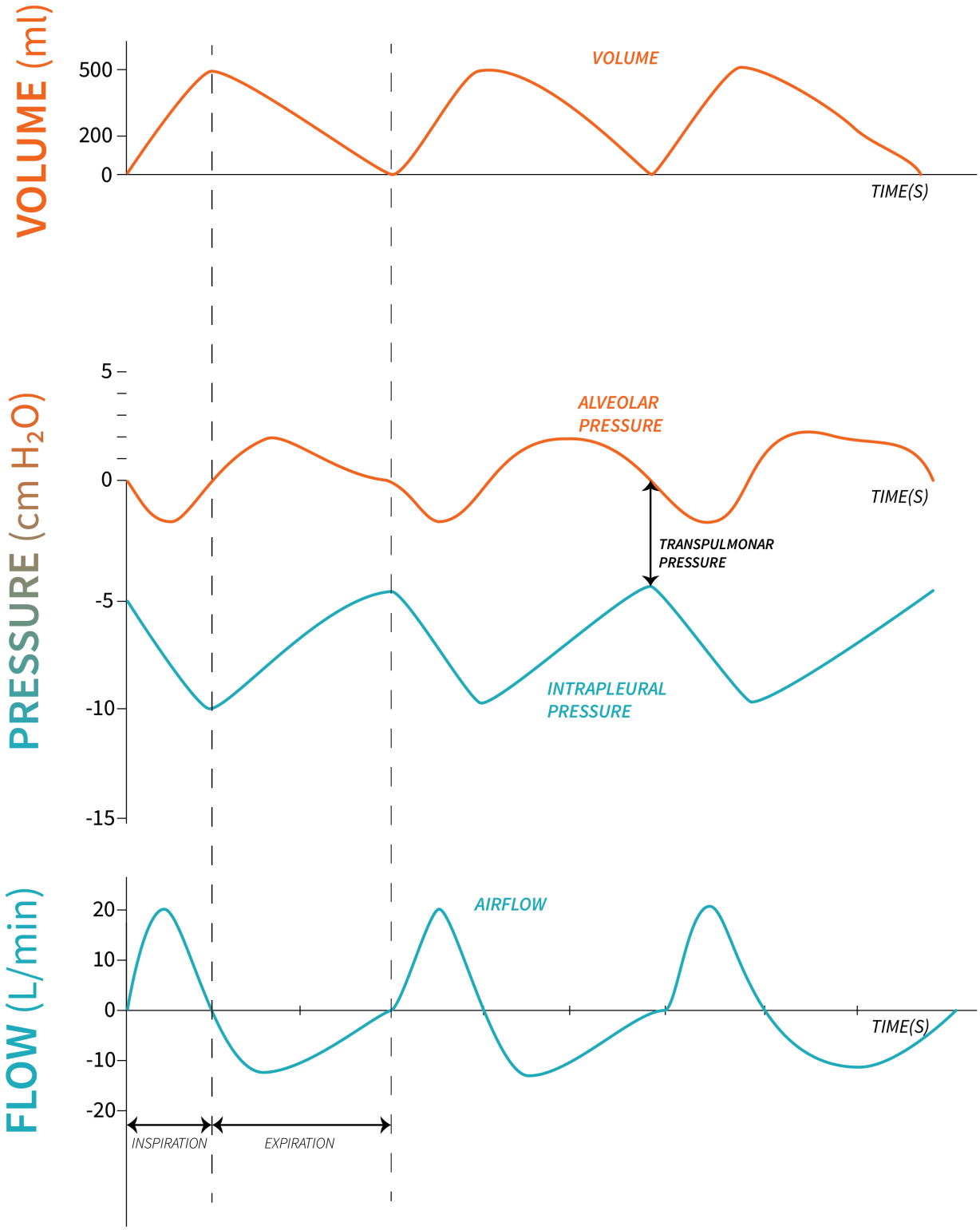

VOLUME (ml)
500
200
0
VOLUME
TIME(S)
PRESSURE (cm H2O)
5
0
-5
-10
-15
ALVEOLAR PRESSURE
TIME(S)
TRANSPULMONAR PRESSURE
INTRAPLEURAL PRESSURE
FLOW (L/min)
20
10
0
-10
-20
AIRFLOW
TIME(S)
INSPIRATION
EXPIRATION

Figure 1.3.4: "Inspiration and Expiration" by Freddy Vale, CC BY-NC-SA 4.0

1.4 VENTILATION

The Respiratory Cycle

When we talk about ventilation, we refer to the cyclic movement of air between atmosphere and alveoli. With every inspiration, we move a volume of air, called tidal volume (V_T), from ambient into the alveoli, and with every expiration we remove that tidal volume of air from the alveoli back to the ambient. The time required for this process is called total cycle time (**TCT**). In healthy adults, the average tidal volume at rest is approximately $500\ \mathrm{mL}$, although this can vary depending on factors such as age, sex, height, weight, and physical activity level. During exercise or other physical exertion, tidal volume can increase to facilitate increased oxygen uptake and carbon dioxide elimination.

Key Takeaway

Tidal volume refers to the volume of air that is inhaled or exhaled during a single breath in normal breathing. It is the amount of air that moves in and out of the lungs with each respiratory cycle, and is typically measured in milliliters (mL) or liters (L).

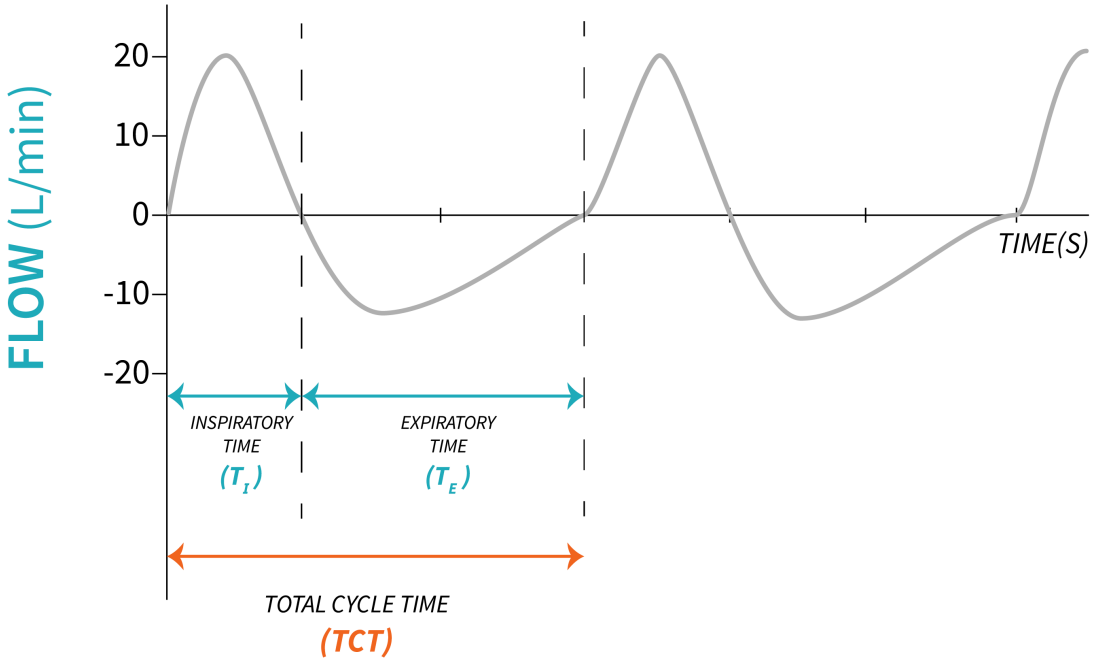


Figure 1.4.1: "Flowtime Graph with Respiratory Cycle" by Freddy Vale, CC BY-NC-SA 4.0

Under normal physiological circumstances, lungs move tidal volume 12-18 times per minute. This is normal respiratory rate (RR).

The timing of one respiratory cycle can be divided into inspiratory time (T_I), time required for inspiration, and expiration time (T_E), time required for expiration. In mechanical ventilation, the relationship between inspiration and expiration is often expressed as a ratio $\mathrm{I{:}E}$ and it can be determined easily when inspiratory and expiratory times are known.

Let's calculate the $\mathrm{I{:}E}$ ratio for an adult individual breathing with an inspiration of 2 seconds and an expiration of 4 seconds.

$$TCT = T_I + T_E$$

$$TCT = 2\ \mathrm{s} + 4\ \mathrm{s} = 6\ \mathrm{s}$$

$$\mathrm{I{:}E} = 2 : 4$$

$$\mathrm{I{:}E} = 1 : 2$$

In mechanical ventilation, in the $\mathrm{I{:}E}$ ratio, the numerator is always expressed as 1 (divide the numerator by itself). In this example, we divide the numerator, 2, by 2. To keep the ratio constant, we divide the denominator by 2 as well. In other words, we simplified the ratio.

Key Takeaway

Total cycle time is a function of respiratory rate per minute $(1\ \text{min} = 60\ \text{s})$:

$$\frac{60\ \text{s}}{RR} = TCT$$

Example:

For a rate of 15 breaths/min, TCT is 4 s

$$\frac{60\ \text{s}}{15} = 4\ \text{s}$$

Minute Ventilation

The product of respiratory rate and tidal volume, expressed in litres per minute, is the total volume of air moving in and out of the lungs. This total volume is called minute ventilation (V_E), and it is an important element that we will be using often as we learn more about mechanical ventilation.

Object Lesson

Minute Ventilation: $V_E \left(\frac{\text{L}}{\text{min}}\right)$

$$V_E = RR \times V_T$$

For an adult individual, breathing at a respiratory rate of 15 breaths per minute, with a tidal volume of $500\ \text{mL}$:

$$V_E = 15\frac{\text{breaths}}{\text{min}} \times 500\ \text{mL}$$

$$V_E = 7500\frac{\text{mL}}{\text{min}}$$

Expressed in $\frac{\text{L}}{\text{min}}$: $7500\frac{\text{mL}}{\text{min}} = 7.5\frac{\text{L}}{\text{min}}$ $\qquad (1\ \text{L} = 1000\ \text{mL})$

V_E for this individual is $7.5 \frac{\text{L}}{\text{min}}$

Alveolar ventilation

Alveolar ventilation describes the total volume of air moving in and out of the lungs, including the volume of dead space. In the context of mechanical ventilation, dead space refers to the portion of the tidal volume that does not participate in gas exchange. This occurs when air is inhaled into the *anatomical dead space* (V_{Danat}) of the respiratory system, which includes the trachea, bronchi, and bronchioles, but does not reach the alveoli where gas exchange occurs. Anatomical dead space is usually 1 mL/lb. Ideal body weight is (2.2 mL/Kg).

Alveolar dead space (V_{Dalv}) refers to areas of the lung where gas exchange is impaired due to ventilation-perfusion mismatch or decreased blood flow, resulting in air being inhaled and exhaled without exchanging oxygen and carbon dioxide. These areas of the lung are ventilated, but not perfused or poorly perfused with mixed venous blood, resulting in poor gas exchange.

Imagine oxygen and carbon dioxide molecules as people waiting at a bus stop. Just as waiting at a bus stop without a bus prevents people from reaching their desired destination, impaired alveolar ventilation limits the proper exchange of oxygen and carbon dioxide in the lungs in the absence of or reduced blood flow. This is a situation where airflow occurs, but the gas exchange process within the alveoli doesn't happen as expected.

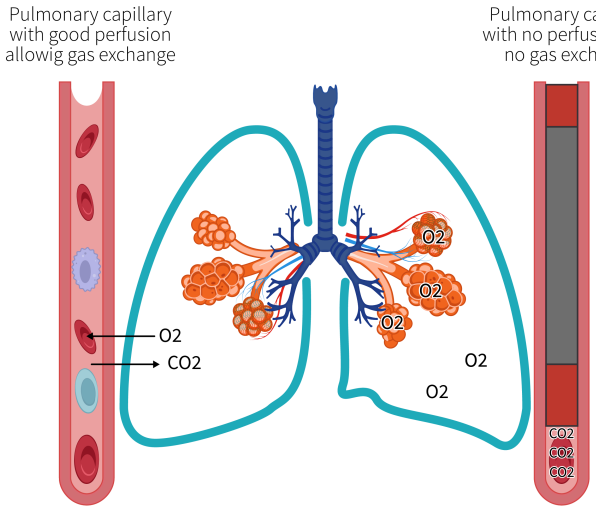


Figure 1.4.2: "Dead Space Ventilation Diagram" by Yvonne Drasovean, CC BY-NC-SA 4.0

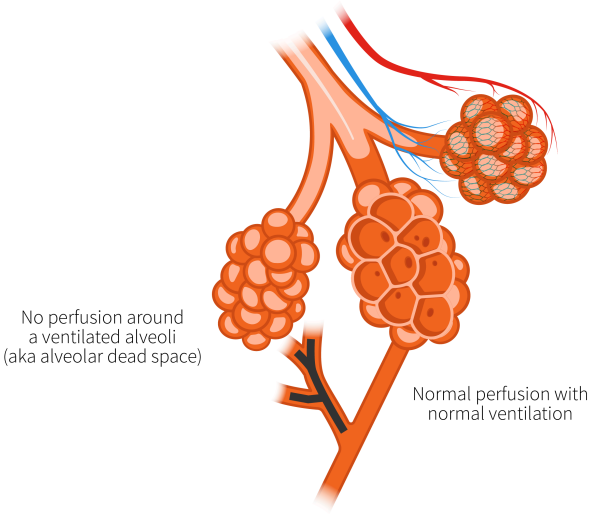


Figure 1.4.3: "Alveolar Dead Space" by Yvonne Drasovean, CC BY-NC-SA 4.0

The sum of anatomical dead space and alveolar dead space determines the *physiological dead space* $(V_{D\ phys})$:

$$V_{Dphys} = V_{Danat} + V_{Dalv}$$

Physiological dead space can be used to determine the efficiency of ventilation.

$$V_A = RR(V_T - V_{Dphys})$$

In clinical practice, using data obtained from bedside monitoring, physiological dead space is often expressed as a ratio to tidal volume ($\frac{V_D}{V_T}$).

$\frac{V_D}{V_T} = \frac{(PaCO_2 - PECO_2)}{PaCO_2}$ Where $PaCO_2$ is the partial pressure of O_2 in arterial blood, $PECO_2$ is the partial pressure of CO_2 in exhaled air. Normal values for $\frac{V_D}{V_T}$: 0.20 - 0.40.

Lung Compliance

A simple definition describes compliance as the change in volume per change in pressure, and it is measured in $\frac{\text{mL}}{\text{cmH}_2\text{O}}$.

$$C = \frac{\Delta V}{\Delta P}$$

Compliance is the reciprocal of elastance ($C = \frac{1}{E}$) and is the result of the elastic forces and surface tension that oppose lung inflation. The surface of each alveolus experiences tension in the absence of any other forces. This surface tension arises from cohesive forces between liquid molecules at the air-liquid interface within the alveoli. Imagine each alveolus as a miniature sphere that expands and contracts independently in all directions, pressing against adjacent alveoli. In order to expand the millions of alveoli in the lungs, a specific amount of inflation pressure is necessary. This is the elastic recoil pressure, also called distending pressure, and it becomes greater as the alveolar radius decreases, as explained by LaPlace's Law. To prevent the collapse of alveoli, which require a significant amount of pressure to reopen, pulmonary surfactant is released to decrease the surface tension within the alveoli as the radius decreases. In conditions where pulmonary surfactant is reduced or absent, such as certain diseases, high ventilating pressures may be necessary, which can potentially cause harm to the lungs. Because the lungs consist of elastic tissue, they tend to empty on their own when unobstructed, much like an untied balloon that gradually deflates due to the principles of Hooke's law of elasticity.

One or more interactive elements has been excluded from this version of the text. You can view them online here: https://ecampusontario.pressbooks.pub/mcvresource/?p=307#oembed-1

Video: "Balloon Deflating 01" By 101GreenLight TV [0:15]

LaPlace's Law

LaPlace's Law: $P = \frac{2ST}{R}$

Where:

$ST = \text{Surface tension } (\frac{\text{dyne}}{\text{cm}^3})$

$P = \text{pressure acting against the surface tension} (\frac{\text{dyne}}{\text{cm}^2})$

$R = \text{radius of the sphere}$

Object Lesson

A balloon that is easy to inflate, is considered **compliant** (with reduced elasticity). This balloon can be described as more compliant, less elastic.

One or more interactive elements has been excluded from this version of the text. You can view them online here: https://ecampusontario.pressbooks.pub/mcvresource/?p=307#oembed-2

Video: "Balloon Inflate Animation" By Clint DiClementi [0:08]

In clinical practice, you may come across various pathological conditions that affect the compliance of the lungs. Pulmonary fibrosis, for instance, can cause a decrease in compliance, resulting in minimal volume changes for any given change in pressure. Conversely, emphysema can cause an increase in compliance due to a loss of tissue elasticity. Generally, the respiratory system has a total compliance of approximately $100 \frac{\text{mL}}{\text{cmH}_2\text{O}}$, with a range of $50 \text{ - } 170 \frac{\text{mL}}{\text{cmH}_2\text{O}}$. This total compliance is comprised of two main components: lung compliance and chest wall compliance, both of which play a crucial role in the elastic behaviour of the respiratory system.

Key Takeaway

Compliance varies with changing conditions such as patient position, respiratory effort and disease process. Any change in lung or chest wall conditions, or both, will affect the total compliance of the respiratory system and the pressure required to inflate the lungs.

A graphic representation of change in volume over change in pressure describes the compliance curve. As described above, you will notice that the position of the curve varies with conditions of the respiratory system. The compliance curve of a patient with emphysema is shifted to the left, compared to a normal compliance curve, as less pressure is required to inflate the lung. The compliance curve of a patient with lung fibrosis will be shifted to the right compared to the normal compliance curve, as more pressure is required to inflate the lung. Monitoring compliance is a critical parameter in mechanical ventilation, as it provides valuable information that can guide adjustments to the ventilator settings and improve patient outcomes.

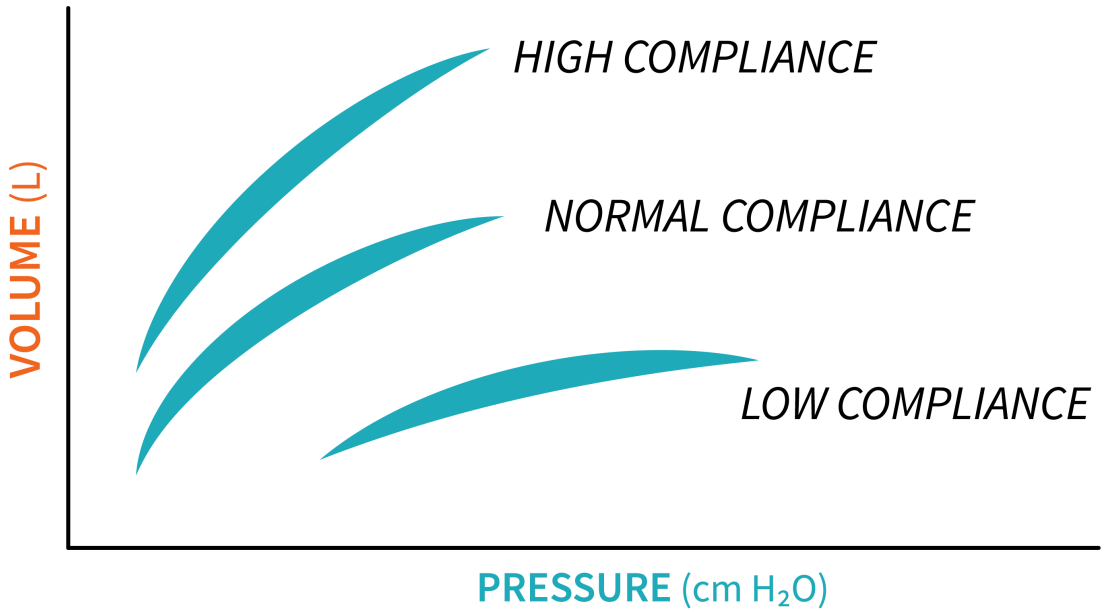


Figure 1.4.4: "Compliance Curve" by Freddy Vale, CC BY-NC-SA 4.0

Check Your Knowledge

Why does an inflated lung want to recoil inward?

Answer

An inflated lung wants to recoil inward due to surface tension and elasticity.

One or more interactive elements has been excluded from this version of the text. You can view them online here: https://ecampusontario.pressbooks.pub/mcvresource/?p=307#oembed-3

Video: "Lungs Breathing – 3D Medical Animation" By AnimatedBiomedical [0:16]

Airway resistance

As air enters the lungs through the conducting airways, it experiences frictional forces, resulting in a resistance to flow known as airway resistance. This resistance can be quantified as the amount of pressure needed to overcome the frictional forces that oppose the inflation of the lungs. The pressure required to overcome this resistance is what we described earlier as transairway pressure (P_{TA}). Air movement caused by lung and chest wall movement during breathing also encounters resistance as a result of the viscous resistance of lung tissue and surrounding tissues and organs. This viscous tissue resistance generally remains constant under normal circumstances and it represents about 20% of total resistance. Viscous resistance may increase in certain pathological conditions such as ascites and obesity. Airway resistance represents about 80% of total resistance. Under normal circumstances airway resistance is about $0.5 \text{ to } 2.5 \text{ cmH}_2\text{O}$.

When we view the airways as a series of tubes, including the trachea, bronchi, and smaller airways, airway resistance can be defined as the constant factor that relates pressure to the flow of air.

$$R = \frac{\Delta P}{\Delta \dot{V}}$$

Key Takeaway

Relate airway resistance back to definitions of pressures. To determine airway resistance, transairway pressure (P_{TA}) will be considered. To determine respiratory system resistance, transrespiratory pressure will be considered.

It is worth noting that the pressure needed to produce airflow during lung inflation accounts for the entirety of airway resistance, even though resistance may fluctuate depending on the specific level of the bronchial tree being examined. Although the right and left main bronchi vary in size, the same driving pressure is transmitted through both of them based on the combined resistance, even tough different flow through these airways created different resistance. Large airways are considered connected in parallel, with the total resistance of elements being less than the resistance of each elements considered individually. Smaller airways are connected in series with large airways as they progress though the bronchial tree. Resistance through the smaller airways will require different pressures for the same flow. The total resistance through smaller airways connected in series is higher than the resistance of an individual element.

Air flow through the conducting airways encounters different patterns: laminar and turbulent flow, and often a combination of these two. Laminar flow requires less pressure to move flow than turbulent flow. Poiseuille's Law describes the factors affecting resistance during laminar flow.

Poiseuille's Law

Poiseuille's Law is written as: $\dot{V} = \frac{\Pi \Delta P R}{8nl}$

Where:

$\dot{V} =$ flow rate
$\Delta P =$ driving pressure
$R =$ radius of tube
$\Pi =$ mathematical constant
$n =$ viscosity of the fluid
$l =$ length of the conducting tube

This video offers a summary of the impact of compliance and resistance on breathing.

One or more interactive elements has been excluded from this version of the text. You can view them online here: https://ecampusontario.pressbooks.pub/mcvresource/?p=307#oembed-4

Video: "Lungs and Chest wall Compliance | Breathing Mechanics | Respiratory Physiology" By Byte Size Med [6:20]

Time Constants

A time constant represents the time required for the lungs to fill and empty by 63%. Filling of the lungs depends on the driving

pressure required based on compliance and resistance. Therefore, a time constant is calculated as the product of compliance and resistance.

This concept is important because it can help us understand differences in distribution of ventilation throughout the lungs, especially in the presence of disease process. Different alveoli, even if adjacent, may have different time constants (different compliance and/or resistance), leading to potentially different driving pressures and volumes. Under normal physiological conditions, a time constant is 0.1 to 0.2 sec.

When inspiratory flow is constant, alveoli with decreased compliance (stiff lung) will have a shorter time constant, will fill quickly, and possibly with smaller volume. These alveoli will require more pressure to deliver normal volume. An example of this scenario is a patient with pulmonary fibrosis.

Alveoli with high resistance will have a long time constant and will fill more slowly. An example of this scenario is a patient with emphysema. If flow stops long enough at the end of inspiration, air will move from the alveoli with decreased compliance into alveoli with high resistance. This movement of air between alveoli is described as The Pendelluft effect.

$$TC = C \times R$$

$1\ TC$	63% filling or emptying
$2\ TC$	86% filling or emptying
$3\ TC$	95% filling or emptying
$4\ TC$	98% filling or emptying
$5\ TC$	lung unit fills or empties completely (99.3%)

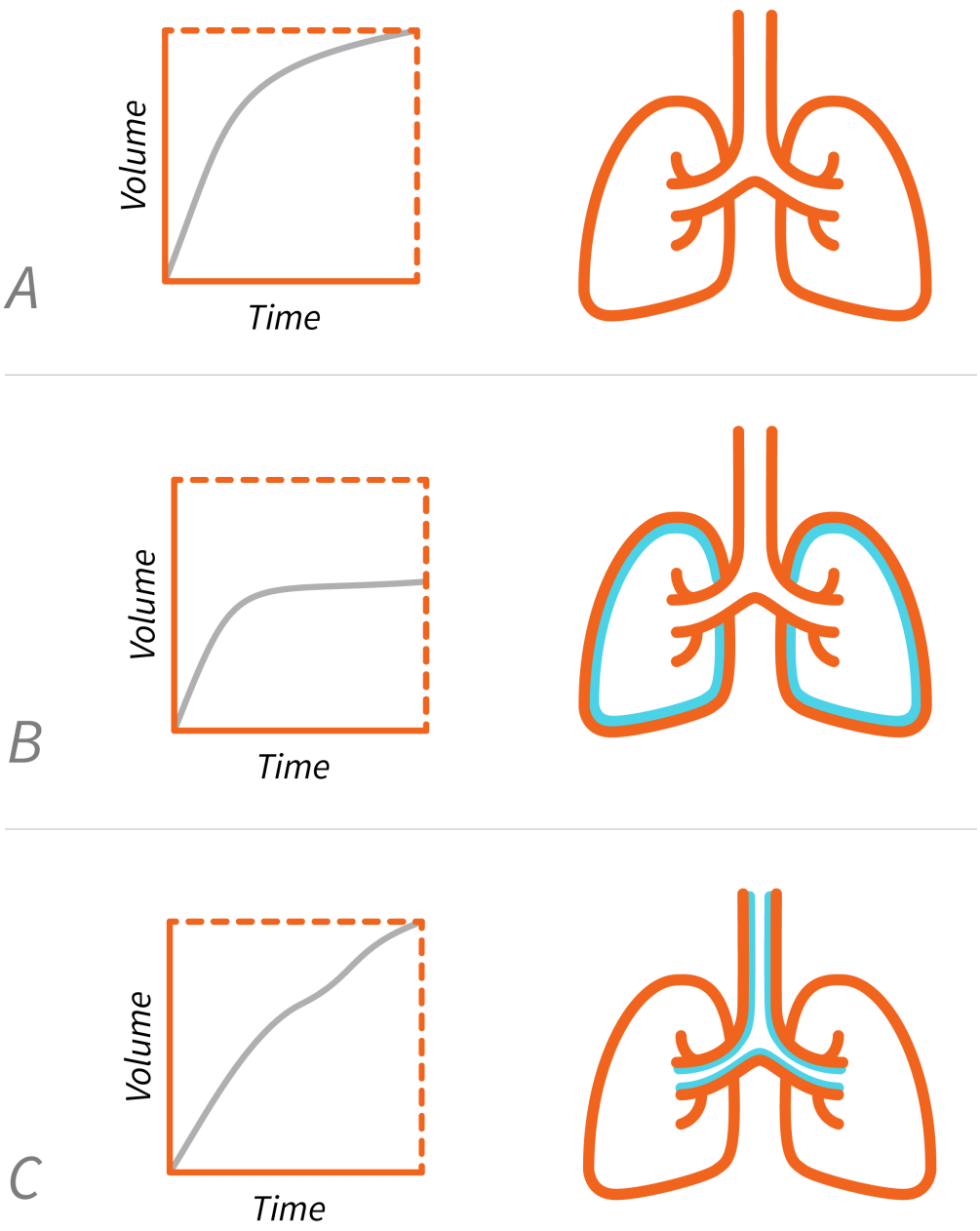


Figure 1.4.5: "Time Constant Diagram" by Freddy Vale, CC BY-NC-SA 4.0

1.5 CONCLUSION

How natural and beautiful is the flow of inhaling and exhaling—like waves upon the shore. It is a deceptively simple process because it is so fundamental to life, but when we look deeper, we learn that each step is so important to the overall flow of air.

The action of breathing seems as simple as the waves upon the shore, but a closer look reveals hidden complexities. Photo by Tim Shields, CC BY-NC 2.0

But what happens when you change the smallest little aspect of the process? For instance, what if you sedated the brain and stopped the contraction of the diaphragm? Suddenly, the passive flow of air no longer has a trigger that causes the negative pressure in the lungs—therefore no air flowing into the lungs. Or, what if the positive pressure applied overcomes the negative pleural pressure that facilitates the air being left in the alveoli? The alveoli would attempt to equilibrate this pressure differential and tend to collapse fully. Finally, instead of air flowing easily into the lungs via a pressure gradient, what if the air was forced into the lungs with a forceful push? If you think of letting a bowling ball gently roll down the alley versus hurling it down the lane as hard as you can, in the first case, the ball most likely won't knock down any bowling pins, but a forceful throw has enough force to send them all flying. The same trauma can be seen in your lungs.

Mechanical ventilation occurs by forcefully pushing air into the lungs. It occurs with a closed system by means of an endotracheal tube, and it forces the alveoli to inflate based on the settings that the ventilator is programmed to deliver. As you can already tell from just this simple description, mechanical ventilation is much different from the natural process of the body.

Like the majority of medical treatments and interventions, there is a downside to mechanical ventilation and this is it: if not done very carefully, the forced air of mechanical ventilation will cause trauma to all those delicate alveoli in the lungs. When we understand that this trauma can occur if we are not careful, it allows us to approach ventilation with the respect needed, to provide safe care for critically ill patients. Eliminating or interrupting spontaneous respiration introduces a large number of problems, as well as the concern of causing damage if ventilation is not done safely. These concerns can be easily mitigated by setting the ventilator appropriately based on the patient and situation you are dealing with. One size does not fit all when it comes to mechanical ventilation. Now that we understand the natural state of breathing, we should also understand how important it is to match the natural breathing process as much as possible with our ventilation settings.

Review

The cascade of breath, in and out, is all about the flow of air from high to low pressures. It starts in inspiration, with the lungs having a negative (lower) pressure from the diaphragm contracting. Air flows in until the diaphragm stops contracting, which stops that negative pressure. Inspiration stops. At this point, the lungs are full of air and are experiencing a higher pressure than the outside world. Exhalation starts as air flows from high to low pressure once again. This continues until the pressure in the lungs sufficiently drops—yet the lungs always maintain FRC, thereby decreasing the pressure needed to open the lungs again.

For a helpful video-based overview of the respiratory process, please watch the Crash Course video on the Respiratory System:

One or more interactive elements has been excluded from this version of the text. You can view them online here: https://ecampusontario.pressbooks.pub/mcvresource/?p=36#oembed-1

Video: "Respiratory System, Part 1: Crash Course Anatomy & Physiology #31" By CrashCourse [9:21] *Transcript Available*

Additional Resources

If you would like more information about the mechanics of breathing, please try these resources:

- *Mechanics of Ventilation*, UBC Critical Care Medicine
- "The Process of Breathing," *Anatomy and Physiology*
- The Inspiratory and Expiratory Process, Deranged Physiology
- Static, Dynamic and Specific Compliance, Deranged Physiology
- Time Constants, Deranged Physiology

1.6 SELF-CHECK

Chapter 1 Quiz

See how well you recall the concepts covered in this chapter by completing the following 5-question quiz.

Don't forget to check your score!

An interactive H5P element has been excluded from this version of the text. You can view it online here: https://ecampusontario.pressbooks.pub/mcvresource/?p=39#h5p-1

CHAPTER 2 | FUNDAMENTALS OF MECHANICAL VENTILATION

Chapter Outline

2.0 CHAPTER OVERVIEW

In Chapter 1, we talked about spontaneous breathing as an elegant and effortless process. Comparing spontaneous breathing to mechanical ventilation is like comparing a gentle stream to powerful rapids. During mechanical ventilation, the diaphragm is being bypassed, or in many cases (i.e., sedation, paralysis) knocked out completely. Air is pushed in by generating a high pressure outside the lungs. Mechanical ventilation pushes air into the lungs with a driving force that is generated inside the ventilator and delivered into the lungs through the ventilator circuit and endotracheal tube. We are now dealing with a positive pressure being applied to the lungs instead of the negative pressure that is usually generated with spontaneous breathing via the diaphragm. This driving force (or positive pressure) can be very traumatic to the fragile alveoli in the lungs—imagine wanting to water your garden but instead of a sprinkler, you use a power washer—however, you will begin to learn how to minimize this trauma for the patient while ensuring effective oxygenation.

In this chapter, we will learn why and when mechanical ventilation is used, and the physiology of a mechanically delivered breath. We will explore supplemental oxygen and its impact on the body. Finally, we will revisit PEEP and FiO_2, but this time within the context of mechanical ventilation.

Mechanical ventilation has evolved so much since its infancy, and it is a constantly changing world. In particular, medical researchers have extensively studied the effects of positive pressure ventilation and how traumatic it is to the lungs. Interrupting the physiological process of breathing is never a good thing, and scientific advances are constantly trying to improve medical treatments to mimic the natural breathing process and rhythm. At the end of the day, as close to "natural" as you can achieve is better for the body and safer for the lungs. As such, there is a constant drive to update and improve modes of ventilation and ways to deliver breaths to mimic the natural physiologic process more closely.

As technology improves and ventilators become more advanced, more and more changes and ventilation modes and strategies are continually introduced. This chapter briefly describes causes of respiratory failure in the context of mechanical ventilation requirements, but it is not meant to replace a detailed pathophysiology textbook or resource. Pairing this information with a general understanding of lung mechanics provides a competent understanding of methods to decrease damage to the lungs and how to use ventilator settings to facilitate lung protective strategies. These concepts can be applied to most ventilation modes and strategies. Lung protective strategies will be explored further in later chapters.

Application

It is essential that any respiratory therapy student who is working with ventilators, understand mechanical ventilation and be familiar with all of the ventilator settings in order to learn more about achieving goals for patient care.

Learning Objectives

At the end of this chapter, you will be able to:

1. Understand basic functions of a ventilator
2. Describe the physiology of a positive pressure breath
3. Differentiate control and spontaneous breaths
4. Differentiate between control and spontaneous ventilation modes
5. Identify settings on a ventilator and which modes they apply to

Key Terms

In this chapter, you will learn about all of the following key terms. These terms will be used throughout this book, so it is important to take the time to master them and practice your recall often.

- control mode
- spontaneous mode
- asynchrony
- trigger
- volume control
- Respiratory Rate (RR)
- Tidal Volume (V_T)
- Pressure Control (PC) or Inspiratory Pressure (P_{insp})
- Pressure Support (PS)
- Inspiratory Time (I_T)
- Flow $(\dot{V})$
- Plateau pressure P_{pl}
- Peak Inspiratory Pressure (PIP)
- Positive End Expiratory Pressure (PEEP)
- Fraction of inspired oxygen (FiO_2)
- Compressible volume
- Pneumatic circuit

Whenever these terms are first introduced in this chapter, they are bolded. However, if you need additional information about a term than what is provided here, you can research it in The Free Dictionary: Medical Dictionary.

2.1 VENTILATORS: THE BASICS

Types of Ventilators

A mechanical ventilator can be described as a device that aids or takes over a patient's breathing. Essentially, a ventilator can be thought of as a computerized machine capable of generating airflow. There are three different types of devices that can perform this function: negative pressure ventilators, positive pressure ventilators and high frequency ventilators.

Negative Pressure Ventilators

Negative pressure ventilation mimics physiological breathing. The most known example of negative pressure ventilator is the Iron Lung (Figure 2.1.1).

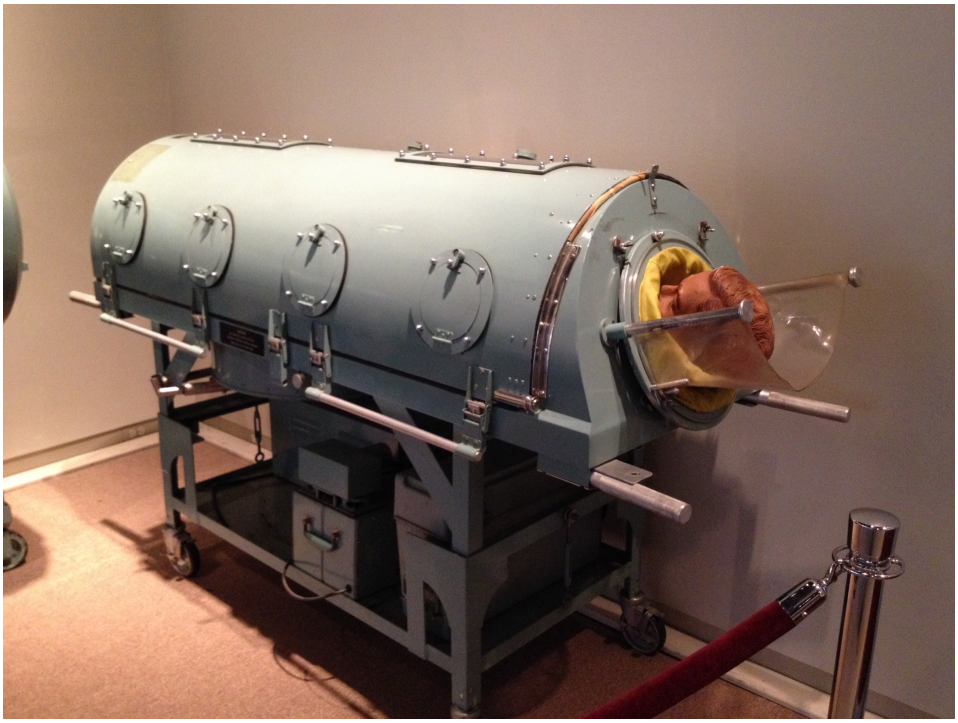


Figure 2.1.1: "Iron Lung" by Arcticseahorse, CC0

The patient's entire body, including the thorax, is enclosed in an airtight container, with sub atmospheric pressure $(\mathrm{P} < 0\ \mathrm{cmH_2O})$. The patient's head is exposed to atmospheric pressure $(\mathrm{P} = 0\ \mathrm{cmH_2O})$. A negative pressure ventilator, operates by creating negative pressure or vacuum around the patient's chest or body. Instead of pushing air into the lungs like a positive pressure ventilator, a negative pressure ventilator gently pulls or draws air out of the lungs. The negative pressure generated around the patient's thorax, is transmitted to the pleural space and consecutively to the alveoli. This creates a pressure differential, causing the chest to expand and allowing air to flow into the lungs naturally. Negative pressure ventilation closely resembles physiological breathing. Expiration occurs passively due to the normal elastic recoil of the lungs and chest wall, when the negative pressure around the thorax is removed.

Object Lesson

During negative pressure ventilation, inspiration occurs when negative pressure is applied to the chest, transmitted to pleural space and alveoli, creating a pressure gradient that allows airflow into the lungs (Remember, air flows down a gradient from high pressure to low pressure).

Another type of negative pressure ventilation is the chest cuirass (Figure 2.1.2).

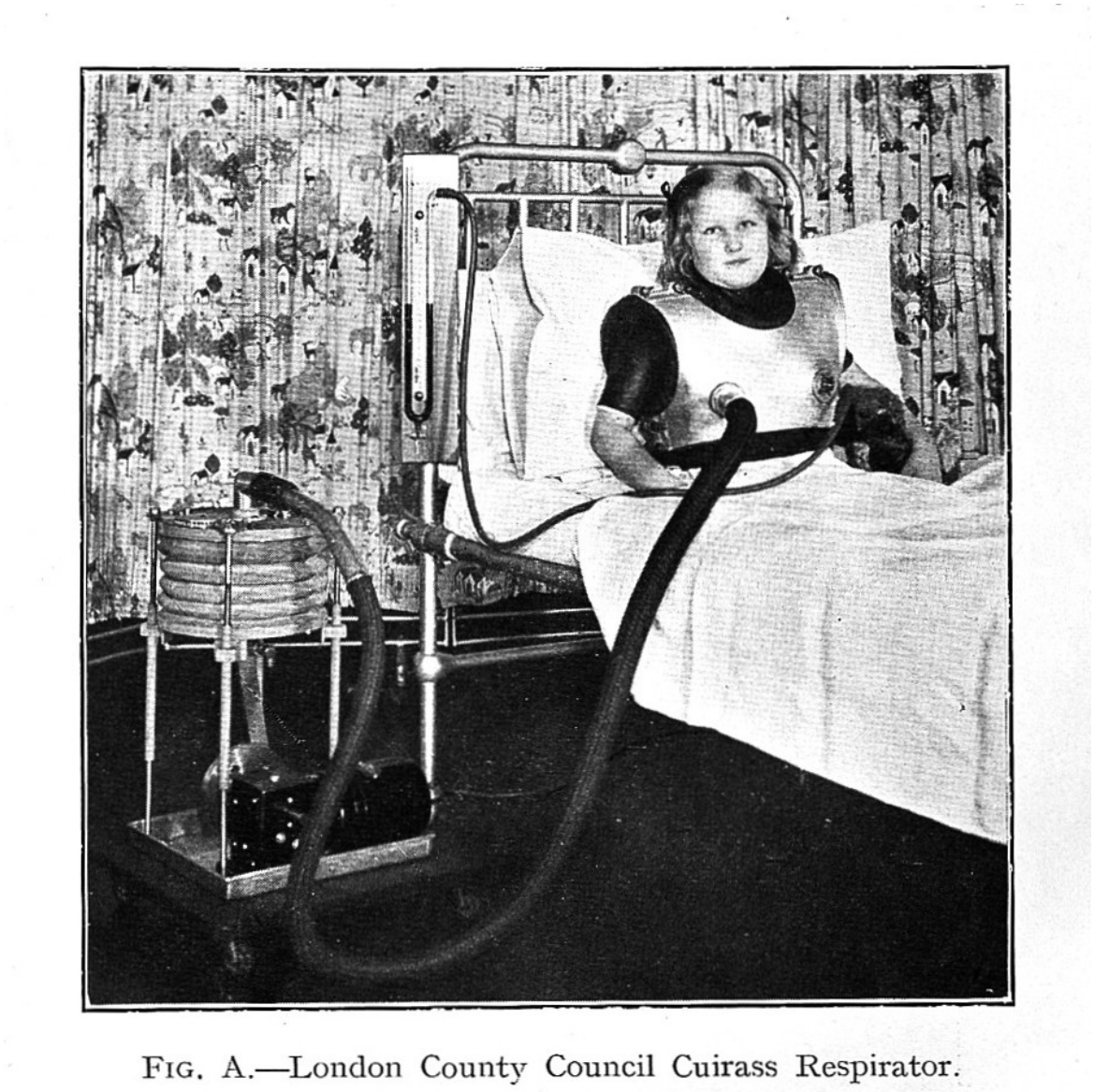


FIG. A.—London County Council Cuirass Respirator.

Figure 2.1.2: Photo via Wellcome Images, CC BY 4.0

Negative pressure ventilation is less commonly used compared to positive pressure ventilation and is often employed in specialized cases or certain conditions where positive pressure ventilation may not be suitable.

Positive Pressure Ventilators

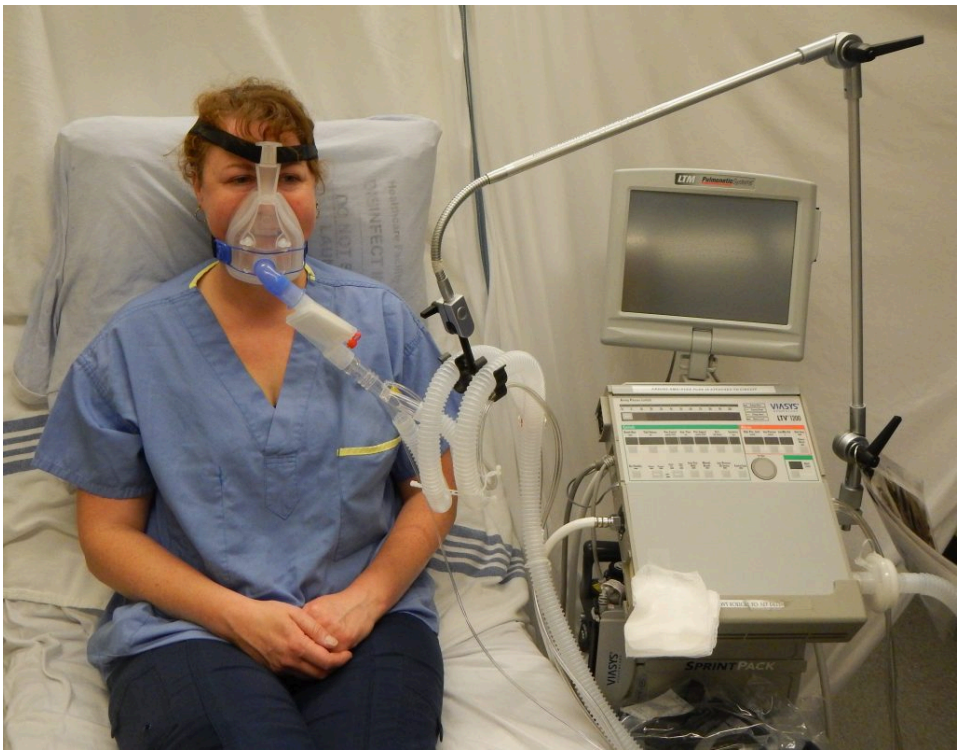


Figure 2.1.3: "BiPAP using a Ventilator" by James Heilman, MD, CC BY-SA 4.0

Positive pressure ventilation requires the use of a device that is capable of generating airflow and creating pressure that is greater than atmospheric pressure. In simple terms, this is a machine that will push air into the patient's lungs by creating a pressure gradient between airway opening and alveoli. To perform this function safely, the following elements are required: power source – power conversion and transmission system- control system and output system, with alarms for every one of these systems. The main functions of the ventilator can be described as four phases: initiation of inspiration, limitation of inspiration (stop lung inflation), allow exhalation and determine baseline.

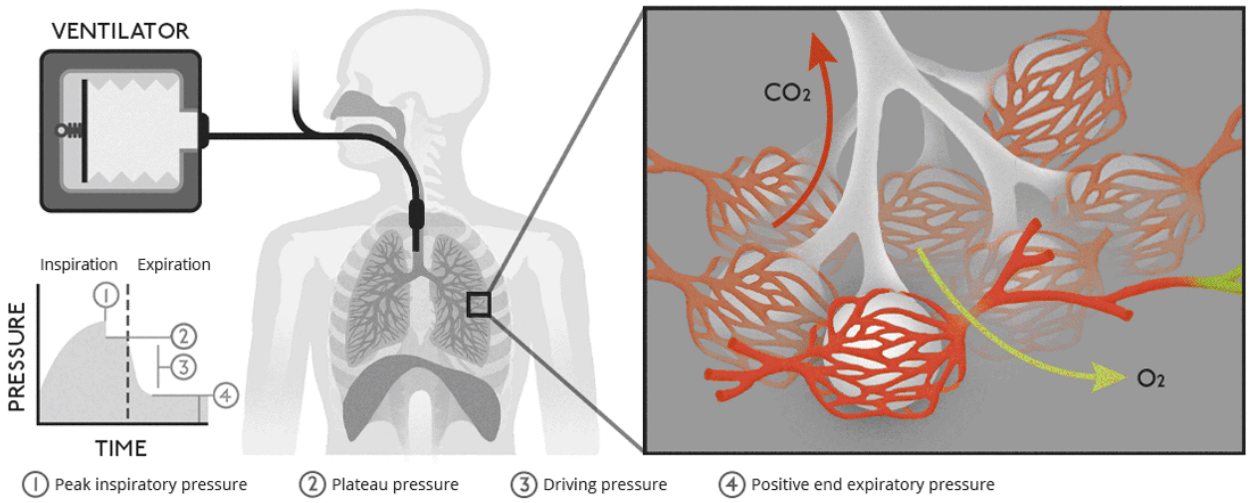


Figure 2.1.4: Animation by Eleanor Lutz, CC0

1. Power Source:

A reliable power source is necessary for adequate function. Ventilators can be electrically powered, pneumatically powered or both.

Electrically Powered Ventilators

Electrically powered ventilators are designed to operate on electricity from the main power supply of the facility with a backup battery option for uninterrupted use during power outages or when transporting the patient.

Pneumatically Powered Ventilators

Pneumatically powered ventilators require a source of pressurized gas, usually oxygen or air, at a pressure of 50 psi. This gas source can be an integrated compressor or a connection to an external oxygen and/or air supply.

A special pneumatically powered ventilator is a **fluidic ventilator.** It operates based on fluid dynamics principles rather than traditional mechanical mechanisms. This type of ventilator is used in specific clinical situations where conventional mechanical ventilators may not be suitable or available.

Most ICU ventilators we currently use require both electrical and pneumatical power source, and are microprocessor controlled. Two 50 psi provide power to generate gas flow, while electricity powers a microprocessor and various valves are powered electrically that control the pattern and direction of flow.

Object Lesson

Fluidic ventilators are often used in emergency situations or field hospitals due to their portability.

2. Power conversion and transmission:

This part of the ventilator is made up by the drive mechanism and the output control mechanism, and it generates and delivers pressurized gas. The drive mechanism achieves this through a variety of methods, including direct application of high pressure gas, though a pressure reducing valve, through mechanical fans or turbines, compressed gas sources, or a combination of both. The flow of gas generated, is then directed towards the patient via the output control valve. Examples of output control valves: diaphragm, electromagnetic plunger,

proportional solenoid valves. Detailed description of various types of drive mechanisms and output control valves are available in respiratory therapy equipment textbooks.

3. Control system:

This component of the ventilator plays a crucial role in precisely regulating the pressure, volume, and flow directed towards the patient. The control system not only measures these parameters, but also governs the functionality of the exhalation valve. Essentially, it acts as the decision-making system of the ventilator, using a combination of pneumatic and electronic devices. While older references may include mechanical components in the control system, they now remain relics of the past, primarily found within the pages of historical accounts. Nowadays, ventilators rely on microprocessor control using technology for advanced and precise ventilation management. Ventilator control systems can use open-loop (also called unintelligent) or closed-loop (intelligent) feedback systems.

Open loop system refers to a ventilator that is only able to deliver a set amount of gas to the patient as programmed by the respiratory therapist without the ability to adjust or sense the actual outcome (unable to recognize potential leaks or other changes). Imagine a barista pouring coffee into a mug based on open-loop feedback type instructions. The barista will pour the entire carafe of coffee into the mug without considering whether it exceeds the mug's capacity. As a result, the excess coffee spills over the edges being wasted and ending up on the kitchen counter (Figure 2.1.5).

Figure 2.1.5: "Coffee poured in White Cup" by Cameron Rainey, CC0

A **closed loop system** refers to a ventilator that can deliver a predetermined volume or amount of gas to the patient's lungs, measure the exhaled volume by the patient, compare the delivered and exhaled volumes, and then make adjustments to the ventilation parameters based on this comparison. The goal is to adapt the delivery of gas to optimize patient outcomes and maintain appropriate ventilation. The control system of the ventilator monitors and adjusts ventilation parameters as programmed by respiratory therapists on the user interface (control panel). Examples of these parameters include tidal volume, respiratory rate, inspiratory-to-expiratory ratio, and positive end-expiratory pressure.

The **pneumatic circuit** is made up by a series of tubes that direct gas flow to the patient. There are two components of the pneumatic circuit: internal and external. The internal circuit directs gas flow from the power source to the external circuit. A simplified schematic of a ventilator circuit is illustrated below. For details on specifics of ventilator circuits and components, please refer to manufacturer information packages.

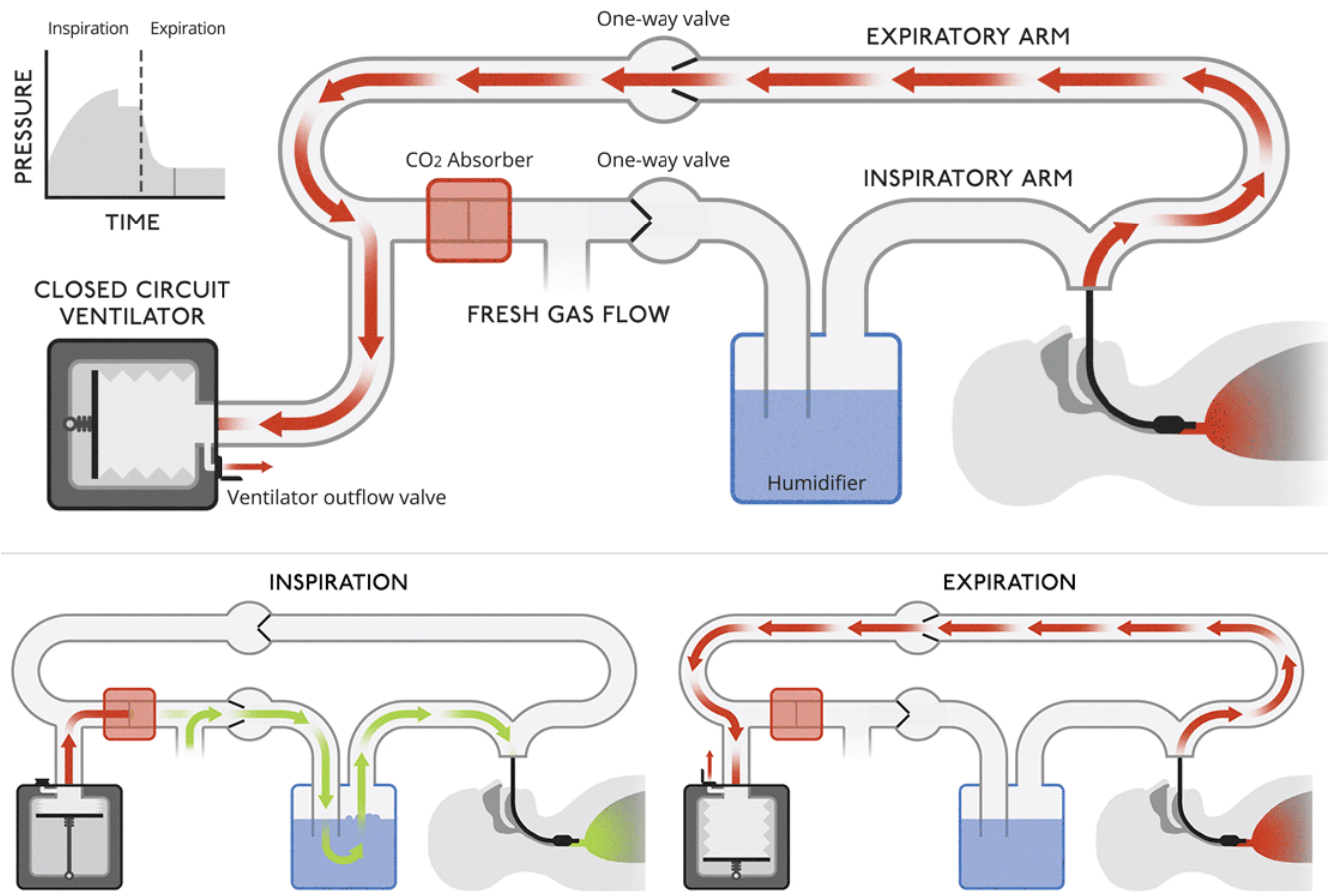


Figure 2.1.6: Animation by Eleanor Lutz CC0

The external circuit consists of tubing that carries the gas flow from the ventilator to the patient and may include additional components like filters, humidifiers, and heat exchangers to optimize patient comfort. The circuit tubing is generally corrugated plastic (22 mm inside diameter for adults), which has universal connectors ($22\ \text{mm}$ outside diameter, $15\ \text{mm}$ inside diameter) that connects the ventilator to the endotracheal tube (ETT), tracheostomy tube, or noninvasive interface.

Plastic circuits used for conventional ventilation will have a certain compliance and resistance, that must be taken into consideration.

Circuit Compliance

When the ventilator generates pressure in the circuit, the plastic material will allow for expansion, due to its compliance (this is often seen as movement, as the circuit tends to straighten during inspiration). This tubing compliance represents the volume of gas compressed in the circuit for every cmH_2O of pressure generated by the ventilator. Because of this, some of the volume delivered will be lost (or trapped) in the corrugated circuit during inspiration. During expiration, this volume of gas leaves the circuit through the exhalation valve.

The volume lost in the circuit due to tubing compliance is referred to as **compressible volume.** Most ventilators have the ability to determine and make adjustments, so that the compressible volume does not significantly affect patient ventilation.

Circuit Resistance

Plastic tubing used with most ventilators is usually wide enough in diameter to minimize resistance to flow. The resistance to flow through a typical ventilator circuit generates a pressure of less than $0.5\ cmH_2O$ at $30\ L/min$ flow rate. Ventilators have the ability to determine and account for circuit resistance. It is important to note that any addition to the length of the circuit, must be taken into consideration by the respiratory therapist. For example, adding length to the circuit for transport purpose may increase resistance. Another example is adding inspiratory or expiratory filters that may become saturated when high humidity is used, increasing resistance in the circuit (Figure 2.1.7).

Figure 2.1.7: "Puritan Bennett 840 Expiratory Filter" by Julia Rios, CC BY-NC-SA 3.0

The Exhalation Valve

The purpose of the exhalation valve is to close the circuit during inspiration so that air flow is directed towards the patient's lungs, and open during expiration, to allow air flow to leave the patient's lungs. This valve allows for unrestricted flow from the patient, or it can increase resistance to flow, in order to maintain an elevated baseline with positive pressure at the end of exhalation (PEEP). Exhalation valves are often flow or threshold resistors, spring loaded valves, diaphragm valves, magnetic and electromagnetic valves. Details on these devices can be found on respiratory therapy equipment textbooks.

Floating Exhalation Valve

The traditional mechanism of operation of exhalation valve allows it to open when exhalation starts, or if pressure in the circuit exceeds the upper pressure limit set by the respiratory therapist. A floating exhalation valve, sometimes called active exhalation valve, is designed to provide more precise control over the exhalation process and improve ventilation efficiency. The microprocessor in the

ventilator actively regulates this valve to release of excess expired air when pressure builds up in the circuit. It responds to changes in pressure within the patient's lungs and adjusts the valve opening accordingly. This allows for better synchronization between the patient's breathing pattern and the ventilator's support. The main advantage of a floating exhalation valve is its ability to optimize ventilation by adjusting the resistance to exhalation based on the patient's needs.

2.2 THE PHYSIOLOGY OF A MECHANICALLY DELIVERED BREATH

Once someone is intubated and attached to a ventilator, the lungs are now a "sealed system." This means that whatever air is pushed out of the ventilator has nowhere else to go except into the lungs (inhalation), and air that leaves the lungs has nowhere to go except for back to the ventilator (exhalation).

Object Lesson

As you learned in Chapter 1, lungs act like balloons. Now, imagine a balloon sealed to the end of a straw. When you blow air through the straw, it must go into the balloon. The balloon will inflate when the pressure you blow is hard enough to overcome the strength of the rubber. If you fill the balloon up and stop blowing, air will come rushing back into your mouth as the balloon relaxes.

In the case of mechanical ventilation, inspiration occurs by air being pushed from the ventilator into the lungs by creating positive pressure at the mouth. Inspiration continues until the breath has been fully given—either what is set by the ventilator (mandatory/control breaths) or based on the patient demand (spontaneous breaths).

Remember from Chapter 1, air always flows from higher to lower pressure.

In this scenario, inspiration is triggered by the application of positive pressure at the mouth through the ventilator, rather than by the contraction of the diaphragm and the creation of negative pressure, as observed during a spontaneous breath. This positive pressure delivered by the ventilator initiates the inflow of air into the lungs. At the beginning of inspiration pressure is applied at the mouth making P_m positive, compared to P_{alv}, which is zero at this point (or less than P_m). This results in a pressure gradient, and air flows into the alveoli following a positive transairway pressure gradient. $\mathrm{PTA} = P_m - P_{alv}$.

As the lungs inflate, P_{alv} becomes progressively more positive (P_{alv} Increases). Under normal circumstances—in healthy lungs—at the end of inspiration, when tidal volume is delivered, these pressures equilibrate $(P_{alv} = P_m)$. The positive pressure in the lungs is now transmitted to the pleural space resulting in P_{pl} becoming more positive. This effect can cause physiological

changes and even complication, deepening on lung compliance and resistance and the state of the cardiovascular system. These complications will be addressed in a later chapter. Positive pressure breathing attempts to inflate the lungs by increasing transpulmonary pressure gradient.

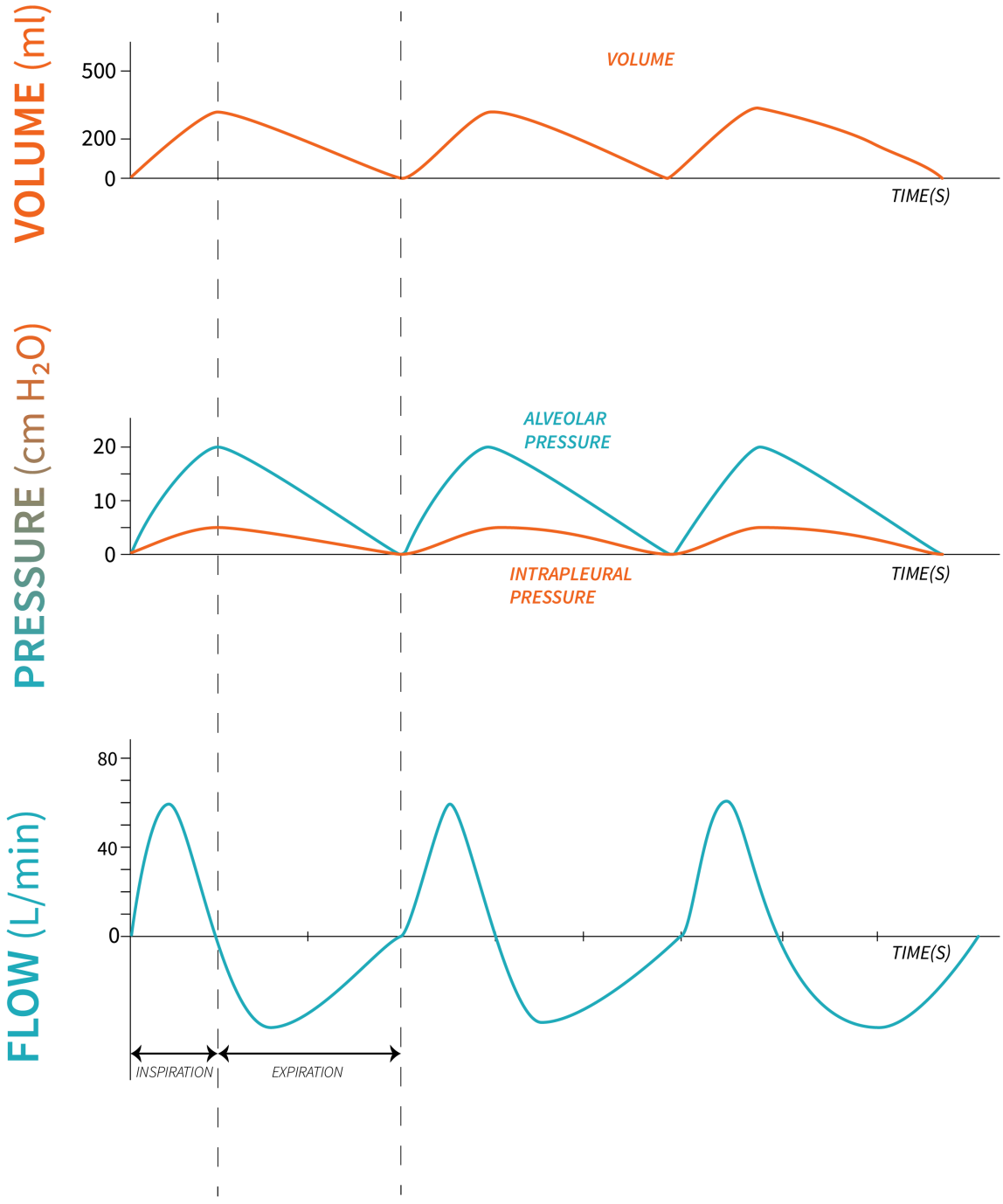


Figure 2.2.1: "Positive Pressure Ventilation" by Freddy Vale, CC BY-NC-SA 4.0

When full, the inflated lungs have a higher pressure than the outside atmosphere once the driving pressure stops from the ventilator. At the end of inspiration, the ventilator stops delivering positive pressure. Exhalation remains a passive process and it begins when a valve opens (exhalation valve), and the ventilator allows the air to flow through a filter out to atmosphere—the full alveoli will be higher pressure than the atmosphere, similar to the concept of spontaneous breathing where, during exhalation, the pressure in the lungs is higher than the pressure at the mouth (P_m). Air flows from high to low pressure from the lungs back to the ventilator.

Apply Your Learning

Refer to the following diagram of a patient who requires a mechanical ventilator (Figure 2.2.2). Can you understand why the mechanical ventilator is a sealed system? It is also a good idea to start becoming familiar with the parts of the ventilator. You'll learn a lot more about them in future chapters.

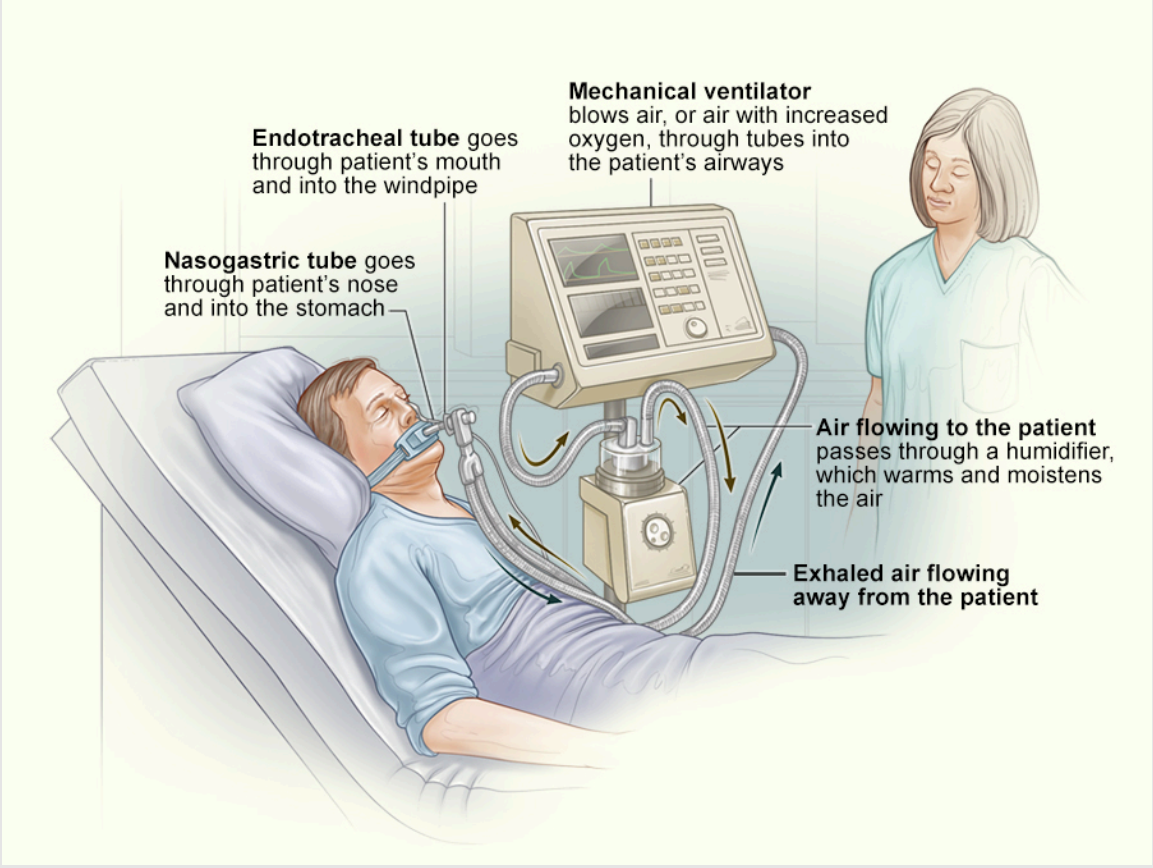


Figure 2.2.2: Ventilator Support by the National Heart, Lung, and Blood Institute, CC0

Lung mechanics and Definition of Pressures in Positive Pressure Ventilation

During Positive Pressure Ventilation (PPV), pressures in the lungs are continuously monitored and displayed on the ventilator screen (PPV). Let's examine a graphic representation of pressures in the lungs at various points in the respiratory cycle, during positive pressure ventilation (Figure 2.2.3).

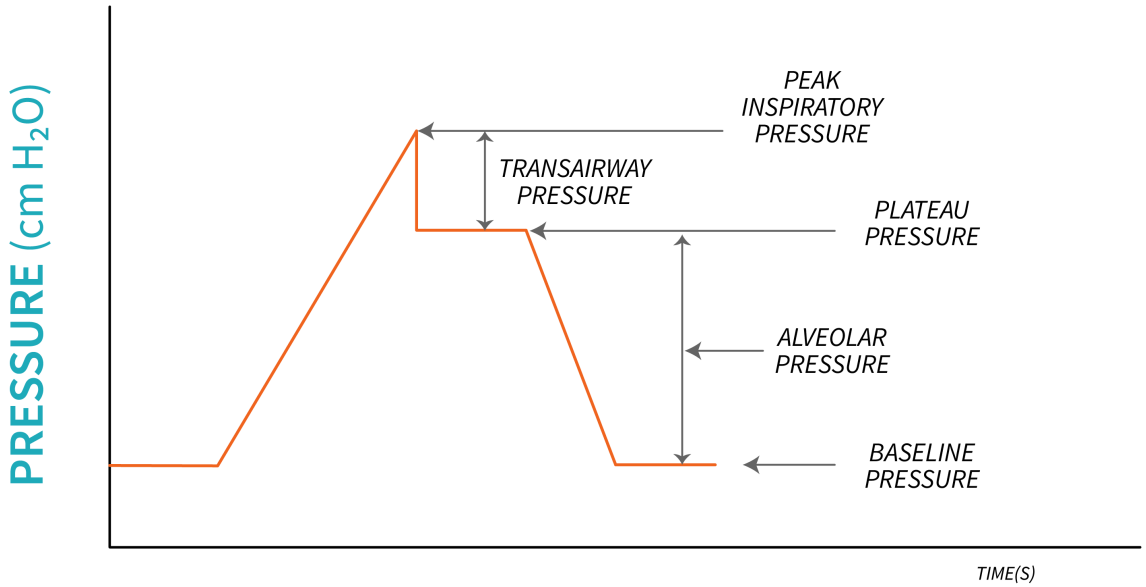


Figure 2.2.3: "Pressures in the Lungs during Positive Pressure Ventilation" by Freddy Vale, CC BY-NC-SA 4.0

Baseline Pressure

Similar to spontaneous breathing, pressures are referenced to a baseline pressure during PPV. This pressure is exerted by the volume of air left in the lungs at the end of an expiration. We learned in Chapter 1 that this is the Functional Residual Capacity (FRC), or when positive pressure is applied, Positive End-Expiratory Pressure (PEEP). As the name suggests, during PPV, we often select a baseline pressure that is higher than zero (atmospheric pressure). When PEEP is set on a ventilator, the volume of air remaining in the lungs at the end of an exhalation is increased. PEEP added through the ventilator increases FRC and is called **Extrinsic PEEP**. A potential complication of PPV is air trapped in the lungs, most often when a patient does not have enough time to exhale completely is **Intrinsic PEEP or Auto-PEEP**.

Peak Inspiratory Pressure

As inspiration begins, and air flows into the lungs, pressure increases progressively, reaching its highest point at the end of inspiration. This is called Peak Inspiratory Pressure (PIP). The pressure required to inflate the lung is the distending pressure, or alveolar pressure.

Plateau Pressure

This pressure is a measure of the compliance of the lungs and it depends on the passive recoil of the lungs, and it is used frequently as part of assessment of ventilated patients. It is measured under static conditions, when flow stops, at the end of an inspiration, with the ventilator performing an **inspiratory hold** before expiration begins. This Inspiratory hold lasts between 0.5 to 1.5 seconds, while the ventilator prevents the exhalation valve from opening to allow exhalation. At the end of an inspiration, during a breath hold, flow stops as the gradient between pressure at the airway opening (P_{ao}) is equal to the pressure in the alveoli (P_{alv}). $P_{ao} = P_{alv}$. During this time, the pressure in the lungs is determined by the the elastic recoil of the lungs and relaxation of respiratory muscles, and it stabilizing to a plateau because there is no air movement. It is measured by the ventilator as plateau pressure (P_{plat}). Note that, if the patient is actively breathing during this inspiratory hold, a plateau will most likely not be obtained and may lead to inadequate measurements.

From the pressure-time graphics above, you will notice that the peak inspiratory pressure is the sum of the pressure required to overcome the resistance to flow through the airways (transairway pressure, P_{TA}), the pressure required to inflate alveoli, and the pressure already present in the lung—baseline pressure.

Key Takeaway

You may notice the terms Plateau Pressure and Alveolar pressure being used interchangeably, however, they refer to different concepts. It is important to understand how these pressures are related. Plateau pressure encompasses the effect of elastic recoil on the volume of air in the alveoli and includes the elastic recoil of the ventilator circuit as well. It emphasizes the concept of the patient's lungs and the ventilator functioning as a closed system. On the other hand, alveolar pressure specifically refers to the intrapulmonary pressure focusing on the pressure within the lungs themselves.

Using the equation of motion, we can describe the dynamics of breathing and examine the differences between spontaneous breaths, negative breaths, and positive breaths, as well as the factors that impact the movement airflow into and out of the lungs.

$$P_{mus} + P_{vent} = P_E + P_R$$

Going back to the idea that the patient connected to the ventilator is a sealed system, the equation of motion help describe the interactions between ventilator and lungs. This equation is basically a mathematical representation of the relationship between pressure, volume, and flow in the lungs, where muscle pressure together with ventilator pressure, equal elastic recoil pressure and resistance to flow pressure. This will help us better understand the dynamics of breathing during positive pressure ventilation under various pathophysiological conditions. The left side of the equation, $\left(P_{mus} + P_{vent}\right)$, describes the pressure required to inflate the lungs, with a dynamic component, the transairway pressure, required to overcome resistance to flow in the conducting airways, and a static component, alveolar pressure, which will maintain lung inflation. This is simply the Peak Inspiratory Pressure (PIP) described above.

For a non-ventilated patient, this pressure, (P_{mus}), is provided by the contraction of respiratory muscles. For a ventilated patient, the ventilator will provide the remaining power needed for a breath (P_{vent}) depending on their ability to generate partial pressure by contraction of respiratory muscles.

Key Takeaway

For a patient unable to generate a respiratory effort (due to sedation, paralysis, head injury), the ventilator will provide all the power necessary to generate a breath, P_{vent}.

The right side of the equation describes the factors that oppose lung inflation.

Resistive pressure P_R, the dynamic element opposing lung inflation describes the pressure required to overcome the resistance to flow through the conducting airways, which we learned in Chapter 1. This is also called transairway pressure, P_{TA}. The relationship between transairway pressure, resistance and flowrate can be written as:

$$P_{TA} = R \times \dot{V}$$

Elastic recoil pressure, P_E, the static component that opposes lung inflation, is the pressure required to inflate the lungs with a certain volume based on elastance (a measure of lung stiffness). This is also called alveolar distending pressure (P_{alv}) and it illustrates how changes in lung volume can affect the elastic recoil pressure generated by the lungs. We learned in Chapter 1 that compliance (C) is the reciprocal of elastance (E). $E = \frac{1}{C}$ and the pressure required to inflate the lung is alveolar pressure P_{alv}. Therefore, we can write the relationship between the pressure exerted by the elastic recoil of the lungs, the elastance and the volume of air present in the lungs as follows:

$$P_{alv} = \frac{V}{C}$$

So, to inflate the lungs, the pressure generated by respiratory muscles and/or ventilator must be equal to the sum of the pressures required to overcome compliance and resistance of the lungs. The equation of motion can be rewritten:

$$P_{mus} + P_{vent} = P_{alv} + P_{TA}$$

2.3 A BRIEF HISTORY OF CONTROL AND SPONTANEOUS MODES

Based on the type of breath delivered, basic modes of ventilation can be mostly categorized as follows:

- Control mode
- Spontaneous modes

Control modes exist to replace the whole process of breathing. When on a control mode, the ventilator completely controls all phases of a breath including:

1. **Initiation:** Breathing is no longer controlled by the chemoreceptors and diaphragm. The ventilator will push breaths into the lungs at the interval set by the clinician.
2. **Inhalation:** The amount of air pushed into the lungs is determined by what is set. The lungs will inflate because it is a sealed system.
3. **Termination of inhalation:** Once the set amount of air is pushed in, the ventilator will stop pushing air into the lungs.
4. **Exhalation:** The lungs will passively deflate as air moves from high pressure to low pressure.

The control mode is utilized for patients who are not able to breathe on their own or who are not breathing or compensating enough to support the body's needs. Every single step in the breathing process is determined by what is set by the health care professional. Virtually nothing to do with the respiratory process is decided by the patient.

Key Takeaway

Ventilation modes can be divided into two main types: (1) control or assist control and (2) spontaneous.

Spontaneous modes are exactly what the name suggests. They allow the patient to be much more involved in the breathing "decision-making process." The patient can control much more of the pattern and size (or volume) of their breaths, and they can cycle through the phases of a breath based on what they want to do. Spontaneous modes are used in situations where patients have an intact drive to breathe—meaning they are still initiating breaths regularly and the physiological "trigger" in the brain is still functioning to initiate a breath (remember the chemoreceptors?). In spontaneous modes, the patient does have more control over their breathing pattern, but some aspects of the breath are still dependent on what is set on the ventilator and not fully patient-driven.

Object Lesson

A good analogy to understand the difference between control and spontaneous modes is driving to a destination. A control mode is like getting a ride to a destination in a taxi. You are the passenger. The taxi driver determines the speed the car is going as well as the route they take when driving.

Photo via PickPik, CC0

On the other hand, a spontaneous mode is similar to driving an automatic car. The person driving has control over the route they are taking and the speed, but the engine is doing some of the "decisions" of driving—like what gear you are in and shifting gears for you. This is similar to a spontaneous mode. Though respiration is mostly patient-driven and in the patient's control, there are still some small parameters that are set on the ventilator to assist or supplement the process.

Begin your exploration of the history of modes with a refresher on the emergence of ventilation and some key innovations that we have already touched on in Chapter 2. Watch "How do Ventilators Work?" by TED-Ed, and then we will focus on ventilator modes:

One or more interactive elements has been excluded from this version of the text. You can view them online here: https://ecampusontario.pressbooks.pub/mcvresource/?p=66#oembed-1

Video: "How do ventilators work? – Alex Gendler" By TED-Ed [5:41] *Transcript Available*

The first version of the modern ventilation modes was a mode known as Continuous Mandatory Ventilation (CMV). It was created to artificially breathe for the patient with positive pressure, but only within the parameters set on the ventilator. The ventilator would not sense what the patient was doing or "asking for". Meaning, if the patient started to trigger breaths and wanted to breathe at a faster respiratory rate than what was set, the ventilator would not deliver it. It was completely blind to the patient.

The problems with this approach are obvious. Imagine being hooked up to a machine and waking up and not being able to breathe when you want to—you try to breathe, but there is no air at all. Not only that, but air is pushed into you when you don't want it! The CMV mode caused a lot of patient discomfort because the ventilator and the patient were not in sync. This problem is termed **asynchrony**. To reduce asynchrony, patients had to be highly sedated—and, in a lot of cases, paralyzed. Increased levels of sedation and paralysis made assessing patient's drive to breathe on their own much more difficult—which posed a challenge when deciding when to discontinue ventilation.

As technology and ventilators advanced, the problems with CMV were quickly identified and ventilators were improved to become much more sensitive to patient efforts. Microprocessors are now able to sense small changes in airflow that represent a patient-triggered breath. Since an effort can now be sensed, the ventilator can deliver a breath on a set "minimum" rate. Or, when the patient causes flow differential that the computer recognizes as an "ask," the ventilator can go above that minimum rate and deliver a breath. To differentiate this mode from the old CMV, this new mode is often termed "ASSIST/CONTROL" or A/C Ventilation since it has the ability to give breaths as set and additionally when asked for.

Introduced in 1983, the Puritan Bennett 7200 was hailed as one of the first vents to use an advanced compressed air system combined with microprocessor technology, resulting in a ventilator that provided excellent gas delivery and patient monitoring capability for its day (Squire, 2013). As time went on, the PB 7200 was replaced by smaller, more efficient technology. Puritan Bennett 7200 by Robert M Kacmarek. Used under fair dealing. All Rights Reserved.

Old control modes (CMV) that do not allow patient triggered breaths are completely archaic and do not exist in modern ventilation. A/C has completely replaced CMV as the only type of control mode. Even modes that still remain labelled as CMV on specific ventilators are not the historical version—they also allow patients to breathe above the set rate.

In the past, as patients improved, the set rate would be turned down to allow the patients to initiate all of their breathing. This did not solve all the asynchrony issues. Small variations or pauses in the physiologic breathing pattern of the patient would result in control breaths being delivered, which would stimulate the patient to breathe while the control breath was being delivered—this is referred to as double-triggering. Other asynchronies also resulted from patients wanting to breathe at different flowrates or lengths of time. These issues identified a need for a mode where the patient could fully control their own breathing pattern and length of breath, which brought spontaneous modes to the market.

Spontaneous modes were created for patients that were able to consistently trigger their own breaths with an effective drive to breathe. Spontaneous modes improved asynchrony by allowing patients to control their own air flowrates and cycling of the ventilator through the different phases of breathing. These significant changes represented some of the most significant and historic

changes in modern mechanical ventilation, moving towards a focus on mimicking physiologic breathing and normal physiologic breathing patterns.

In contrast to control modes, spontaneous modes widely remain very similar to their original versions. Though there have been some new modes that offer some additional changes to spontaneous ventilation, they are not widely used and will not be covered in the scope of this book.

> For a much more detailed historical overview and discussion of possible future innovations for ventilators, read "The Mechanical Ventilator: Past, Present, and Future" by Robert M. Kacmarek (2011).

2.4 PHASE VARIABLES: CONTROL VS. SPONTANEOUSLY INITIATED BREATHS

Control breaths are the breaths delivered according to the set respiratory rate on the ventilator. When we program a certain respiratory rate into the ventilator, this determines the total cycle time (Total Cycle Time, TCT). For example, if the respiratory rate is set at 15 bpm, that means that there are 15 breaths that will be delivered in one minute—one every 4 seconds. Even if the patient does not have a drive to breathe, the air will still be delivered every 4 seconds. However, even in control modes, patients can choose to initiate breaths *above* the set respiratory rate. This means that, if the patient has their normal physiologic drive to breathe, they could initiate additional breaths as well. But if they do not, the ventilator will always deliver the set respiratory rate *at a minimum*.

Before we further discuss controlled and spontaneous breaths, let's review the functions of the ventilator, and describe in detail how the ventilator enables a controlled versus a spontaneous breath.

Photo by Official U.S. Navy Page, CC BY 2.0

Phase variables are parameters used by clinicians to describe and design a breath that will be delivered to the patient.

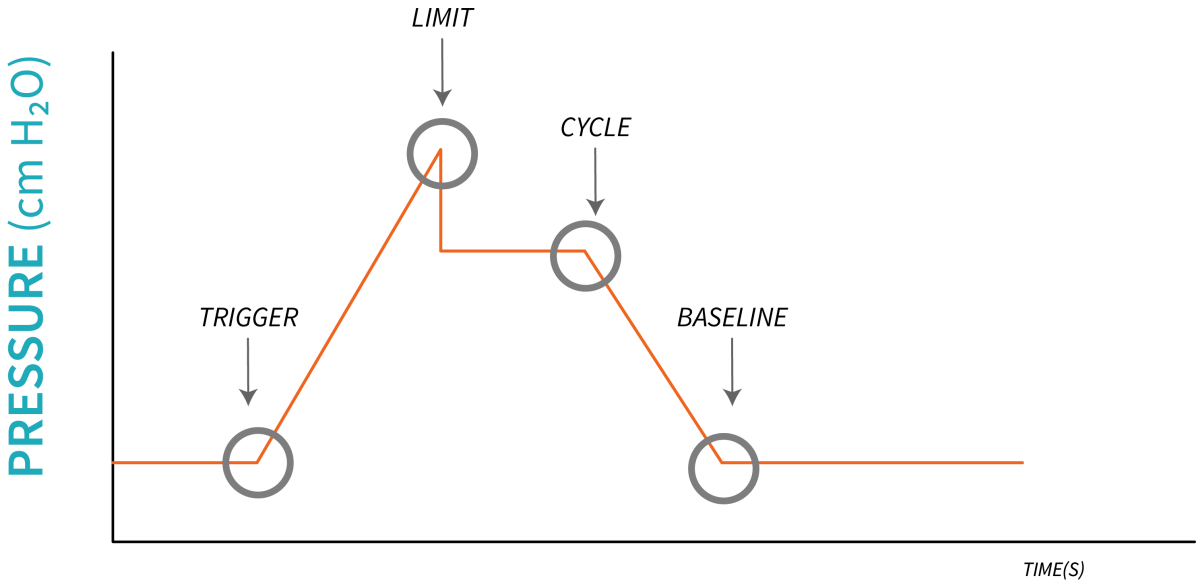


Figure 2.3.1: "Phase Variables" by Freddy Vale, CC BY-NC-SA 4.0

The ventilator employs distinct parameters known as variables to determine the initiation of each phase in the breathing cycle. The **Trigger variable** is utilized to initiate inspiration, while the **Limit Variable** ensures that lung distention remains within specified limits. Exhalation begins when the ventilator permits it based on the **Cycle variable**, and the **Baseline Variable** determines the level at which a new inspiration will begin.

Now, let's explore the functions of the ventilator in relation to these phase variables.

Table 2.4.1: Functions of the Ventilator and Phase Variables

Function	Phase Variable
Initiate Inspiratory phase	Trigger
Stop lung distention	Limit
Allow exhalation	Cycle
Determine baseline	Baseline

So how does a ventilator "sense" a patient?

The Trigger Variable

We have already established that positive pressure ventilation pushes air into the lungs. Microprocessors are constantly evaluating the flow of air that is leaving the ventilator and being delivered to the patient through the ventilator circuit. Recalling the process of normal physiologic breathing, think about the action of the diaphragm contracting and dropping down in the thorax. We have talked about how this movement creates a negative pressure and air will flow into the lungs. Remember that the patient is intubated and connected to the ventilator through the patient circuit. If a patient has the ability to initiate (trigger) a breath by contracting their

diaphragm, his negative pressure will pull air from the circuit. The microprocessors of the ventilator are calibrated to the normal flow of air and pressure in the circuit, and even a small change with air moving into the chest is sensed by the ventilator. This is called a **trigger** because the ventilator senses this change and it "triggers" the delivery of the set breath.

Ventilators can be set by the respiratory therapist to sense changes through either pressure or flow. Both triggers operate in a similar way:

- A pressure trigger is activated when the contracting diaphragm decreases the overall pressure of air in the circuit.
- A flow trigger senses air being pulled from the circuit from the drop in pressure that the diaphragm creates.

Pressure or flow triggered breaths are referred to as ***patient triggered (assisted breaths).*** If the patient does not make any efforts, is unable to trigger the ventilator, breaths will be ***time triggered (controlled breaths)*** based on the respiratory rate set by the respiratory therapist.

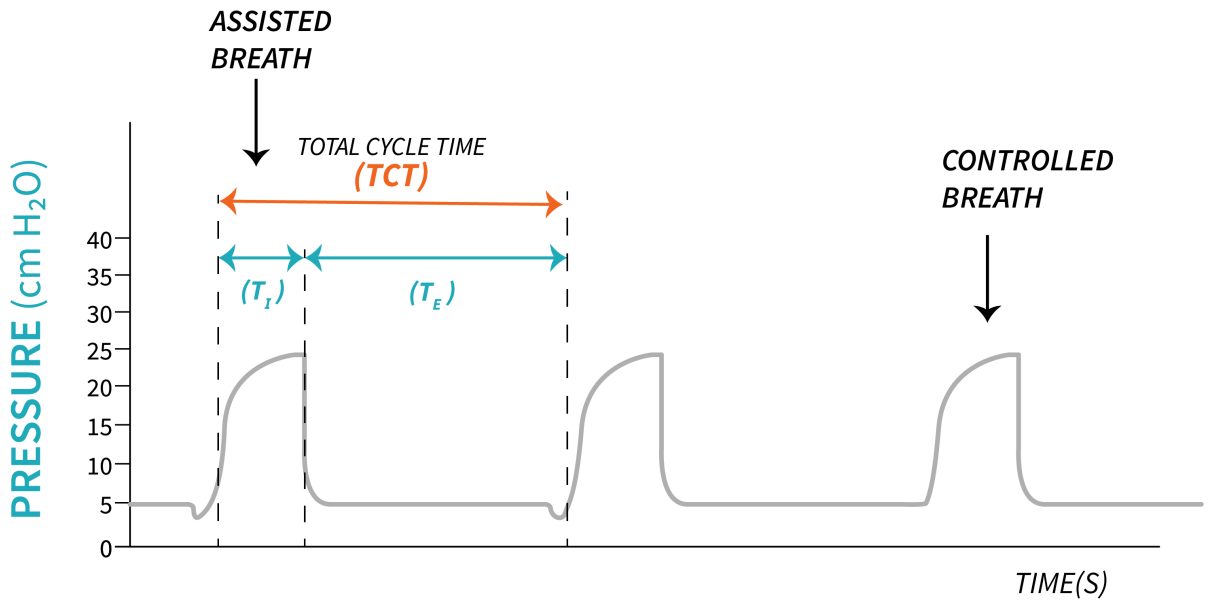


Figure 2.3.2: "Trigger Diagram" by Freddy Vale, CC BY-NC-SA 4.0

Key Takeaway

Flow triggering is more commonly used for mechanically ventilated patients as the more comfortable trigger mechanism.

It takes more effort and flow from the patient to trigger even a $-1\ cmH_2O$ change in pressure in order for the ventilator to sense a breath request than to create a change in flow in the circuit.

Flow triggers are more commonly used on most ventilators in current practice. When the flow hits the threshold of the set flow trigger, a breath is delivered. The amount of flow that the patient needs to pull is negligible before the ventilator senses the patient initiating a breath, and then the ventilator will deliver the set breath based on the mode and settings selected. A normal flow trigger is $2 - 3\ Lpm$. When you think that ventilators respond in fractions of a second, even a minute change (pull of air toward the patient) triggers the breath.

As respiratory therapist, we will be able to see the changes in pressure and flow described above at the beginning of a breath by looking on the ventilator screen at the waveforms that show the delivery of the air. Reading waveforms will be discussed in detail in a later chapter, but when it comes to patients triggering breaths in control modes, you need to remember that it will cause a small negative pull in flows and pressures before the initiation of the breath on spontaneously triggered breaths. Control breaths would not have that small negative scoop before the waveform.

An easy way to see if your patient is triggering any breaths above the mandatory respiratory rate is to compare the total respiratory rate the patient is breathing against what you have set in the settings. If you set $15\ bpm$ and you notice the patient is breathing $18\ bpm$ in total, it is obvious that the patient is triggering breaths above the set respiratory rate.

Additionally, there are ventilation modes that grant patients greater control, enabling them to initiate breaths, determine the volume and duration of each breath. These modes allow ***spontaneous breaths***, requiring minimal assistance from the ventilators.

Adjusting the trigger variable may require some trial and error, and it is closely linked to patient assessment. The ventilator needs to be responsive to the patient's effort in triggering a breath while ensuring that it doesn't add to their respiratory workload. If the sensitivity of the ventilator is set too high, it may inadvertently detect minor leaks in the circuit and trigger breaths automatically when they are not needed (auto-triggering).

Trigger Sensitivity

When patient triggering is available on a ventilator, it is crucial to establish an optimal level of trigger sensitivity. The ventilator should be set to detect the patient's respiratory effort effectively, striking the right balance between sensitivity and avoiding auto-triggering.

The Limit Variable

Another function of the ventilator is to limit the size of a breath. This does not correlate with the end of the inspiration, but rather, how big a breath can get during inspiration. The limit variable (sometimes called the target variable) represents the maximum value that a parameter can attain. This parameter can be pressure, volume, or flow.

A ***volume limited breath*** is delivered by the ventilator by measuring the flow of air delivered during a specific time period. This volume will be the maximum amount of gas that will be delivered to the patient during an inspiration. Note that multiple limit

variables can be set in the same time for one breath. When volume is the limit, you may notice a squaring of the volume waveform, which indicates that the maximum volume has been attained.

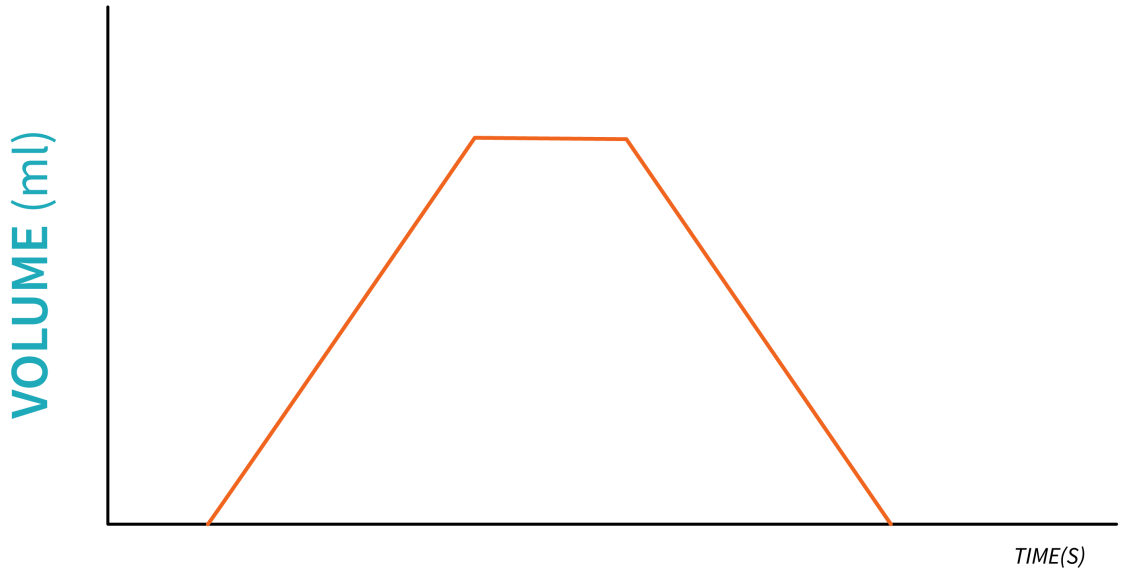


Figure 2.3.3: "Volume Limited Breath" by Freddy Vale, CC BY-NC-SA 4.0

In a ***pressure limited breath,*** the ventilator will allow pressure to rise during inspiration up to a certain value set by the respiratory therapist. The attainment of this value does not correlate with the end of the inspiration, but rather the size of the breaths and how much air will fill the lungs to reach that pressure limit. After the limit is reached, any excess pressure entering the patient circuit, will be released though a pressure release valve.

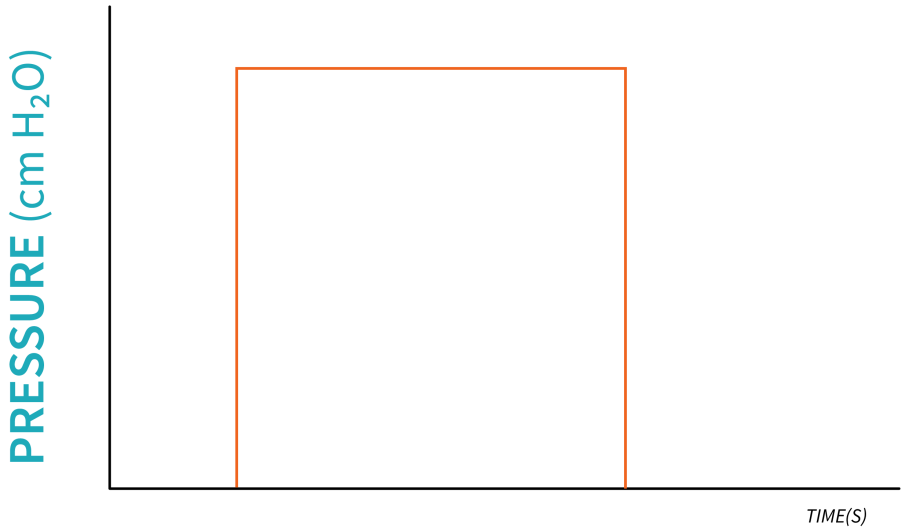
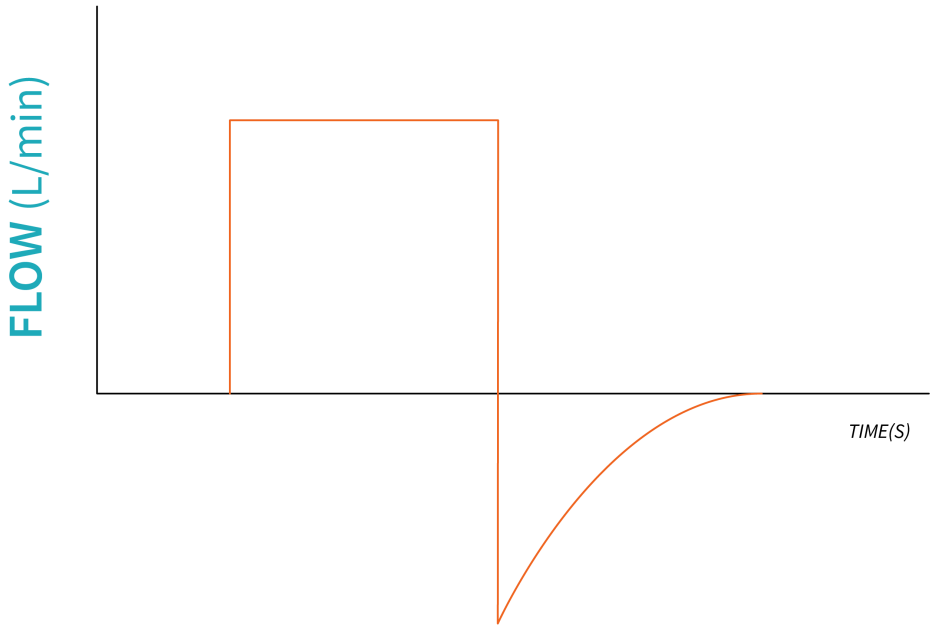


Figure 2.3.4: "Pressure Limited Breath" by Freddy Vale, CC BY-NC-SA 4.0

A ***breath is flow limited*** when the gas flow into the ventilator circuit reaches the predetermined set level before the end of the inspiration. Flow limiting often leads to patient-ventilator asynchrony, when the set flow does not reach the patient's inspiratory flow demand. Setting the flow limit requires practice with patient assessment and knowledge of physiological demands.



"Flow-Limited Breath" by Freddy Vale, CC BY-NC-SA 4.0

An important breath limiting parameter that is worth mentioning here is the **Maximum Safety Pressure.** This limit variable is

different than pressure limit. All ventilators will have this feature for the purpose of protecting the patient's lungs from damage caused by excessive pressure. Most ventilators will end inspiration and initiate expiration when this Maximum Safety Pressure is reached.

Example: Maximum Safety Pressure

Let's assume the Maximum Safety Pressure is set to $40\ cmH_2O$. When volume is set as the limit variable, and the pressure required to deliver the set volume reached $40\ cmH_2O$, the ventilator will end inspiration, allow the exhalation valve to open and release the air.

The Cycle Variable

The Cycle variable is used to determine the opening of the exhalation valve and end inspiration. Only one parameter is used at a given time, and it can be one of the four: volume, pressure, flow, and time.

During a ***volume cycled*** breath, inspiration stops, and we say that the ventilator cycles into exhalation once the target volume is delivered. This may be a problem when lung mechanics change. This is when the limit variable and maximum safety pressure play a crucial role. If the ventilator is programmed to deliver the target volume regardless of alterations in lung mechanics, excessive pressure may be necessary to achieve that volume. Without an appropriate pressure limit, the pressure delivered to the lungs can escalate to unsafe levels. This inherent drawback of volume cycling, related to safety, is one example of why this cycling mechanism is not frequently used.

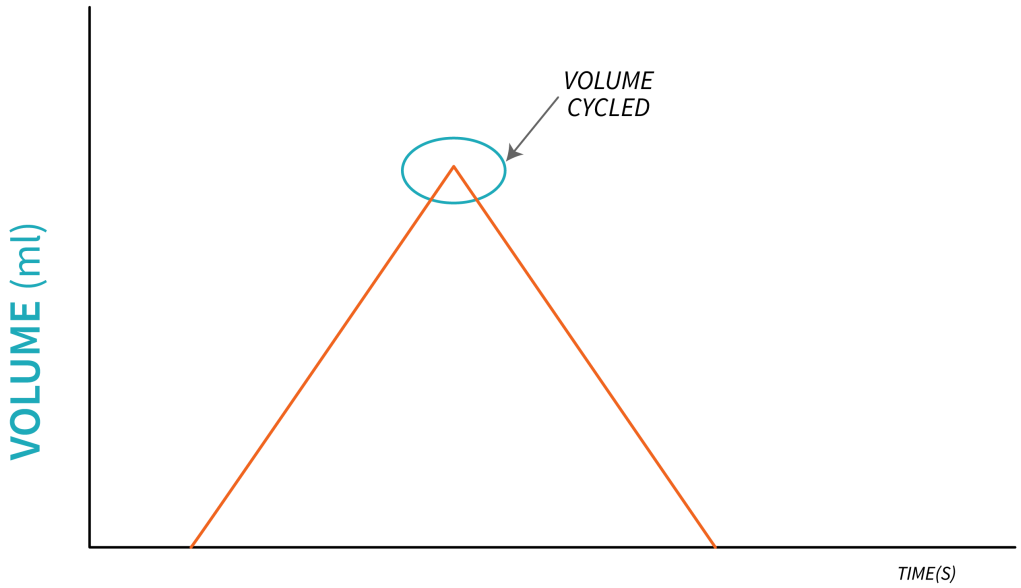


Figure 2.3.5: "Volume-Cycled Breath Diagram" by Freddy Vale CC-BY-NC-SA

During a ***Pressure cycled breath***, inspiration stops when a certain preset pressure is achieved. This signals the ventilator to open the exhalation valve and allow air to flow out of the lungs. The tidal volume delivered to the lungs depends on the preset pressure, duration of inspiration, flow delivered and patient's lung mechanics. This cycling mechanism is sometimes seen during volume limited breaths when the pressure in the lungs exceeds the maximum safety pressure. In this case, the ventilator will end inspiration in order to protect the lung from high pressure damage, and tidal volume is not delivered.

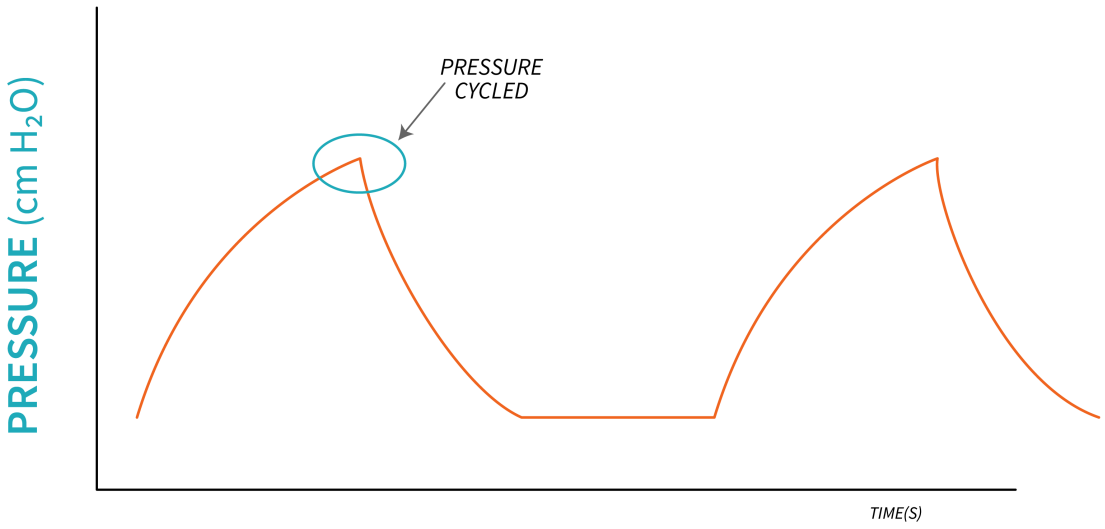


Figure 2.3.6: "Pressure-Cycled Breath Diagram" by Freddy Vale, CC BY-NC-SA 4.0

During a ***Flow cycled breath***, the ventilator opens the exhalation valve and initiates exhalation when inspiratory flow drops to a certain level, preset by the respiratory therapist. As inspiration starts, flow increases exponentially to reach a certain inspiratory pressure. As the lungs fill with air, flowrate decreases. When it reaches a certain level (usually a percentage of the peak inspiratory flowrate), exhalation starts.

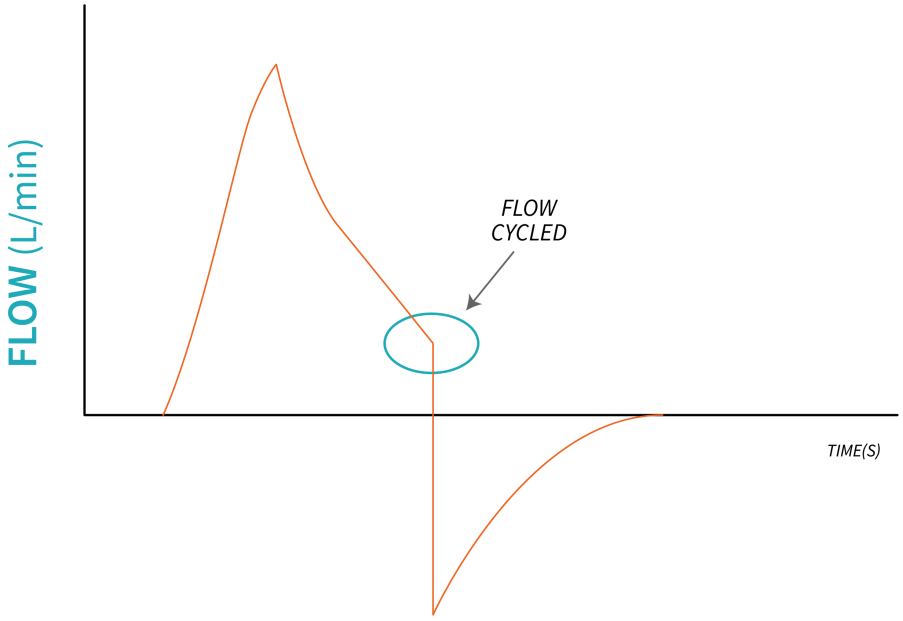


Figure 2.3.7: "Flow-Cycled Breath Diagram" by Freddy Vale CC BY-NC-SA 4.0

Example

Peak Inspiratory flow: $80\ \text{Lpm}$.

Ventilator cycle signal is set at 25% of the peak inspiratory flow. This means that the ventilator initiates expiration when inspiratory flow drops to 25% of $80\ \text{Lpm}$ (at $20\ \text{Lpm}$).

In a ***time cycled breath***, inspiration ceases upon reaching a specific time interval, which is typically observed in controlled, mandatory breaths where the Inspiratory time is predetermined. The ventilator permits exhalation once the inspiratory time has elapsed, and this cycling mechanism remains unaffected by alterations in lung mechanics.

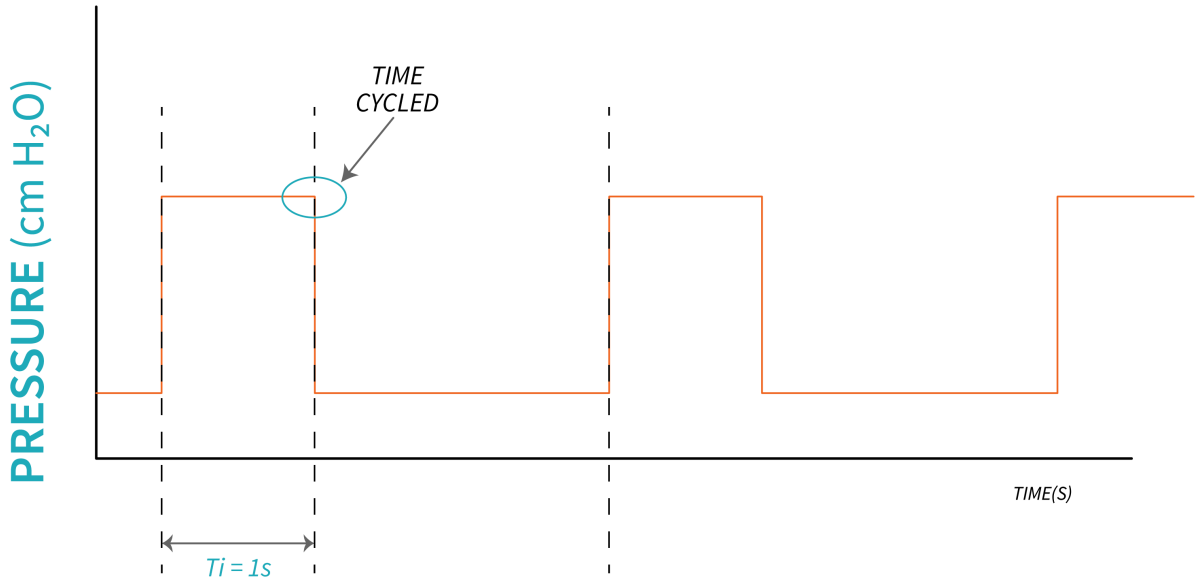


Figure 2.3.8: "Time-Cycled Breath Diagram" by Freddy Vale, CC BY-NC-SA 4.0

The Baseline Variable

This variable describes the expiratory phase and it is usually based on the pressure exerted by the volume left in the lungs during expiration. As previously discussed, this is represented by positive end expiratory pressure, PEEP. All breaths will start from PEEP level and end at the same level. For healthy lungs, a PEEP of $5 - 8\ cmH_2O$ is usually selected as baseline. For low compliant lung (such as in ARDS), a higher PEEP will be used, and in some cases a baseline of zero is used.

It is important to monitor the return of airflow to its baseline during each breath because inadequate time for expiration can lead to air trapping, resulting in intrinsic PEEP (auto PEEP).

2.5 OXYGEN ON A VENTILATOR: SETTING THE FIO2

Oxygen is *always* set on a mechanical ventilator—regardless of the mode or type of breath. Since the air delivered by a mechanical ventilator compromises all of the air the patient will be breathing in, the composition or percentage of oxygen must be set on the ventilator. On the ventilator, oxygen is set by the FiO_2. You would adjust the FiO_2 between 0.21 and 1.0 to ensure your patient is getting enough oxygen.

Key Takeaway

FiO_2 is always set on a mechanical ventilator, no matter the mode the patient is in.

The principles of assessing oxygen need that we have previously learned in our respiratory therapy training can be applied when adjusting settings on the ventilator. If the SpO_2 is less than 92%, increase the FiO_2 the ventilator is supplying to help increase the oxygen available to the lungs, and by extension you will increase the amount of oxygen in the body. Increasing the FiO_2 will increase the partial pressure of oxygen in the alveoli of the lungs, which in turn increases the amount diffusing across the alveolar-capillary membrane to attach to hemoglobin and also increases the amount of oxygen dissolving into the bloodstream. Remember, air moves from high pressure to low pressure. So, if the partial pressure of oxygen is higher in the alveoli then the blood, a higher pressure difference will increase the movement of oxygen from the high end of the gradient to the low end—moving the oxygen from the alveoli across the alveolar-capillary membrane into the bloodstream. Watch the following video from 5:22 to 5:44 for a demonstration:

One or more interactive elements has been excluded from this version of the text. You can view them online here: https://ecampusontario.pressbooks.pub/mcvresource/?p=51#oembed-1

Video: "Oxygen transport presentation" By Jennifer Pinto [8:20] *Transcript Available*

Oxygen Free Radicals

But don't start thinking that's all there is to it! You cannot simply set the oxygen and walk away. First, it is important to note that oxygen is classified as a drug and, like any other medication, it has its own negative side effects. Medical research has concluded that exposure to high amounts of oxygen for extended periods of time can cause damage to the lungs. A high level of oxygen in the body creates a byproduct called **oxygen free radicals** that cause scarring for the delicate alveoli-capillary membrane and harden it, making

it more difficult for oxygen to get through. More details on these complications will be covered in a later chapter. The best way of minimizing exposure to oxygen free radicals is ensuring that patients get just enough oxygen to support their needs, but not so much that they have extra oxygen circulating in the body.

For further learning on oxygen free radicals, see "Free Radicals and Reactive Oxygen."

It's easy to misunderstand this concept of oxygen causing damage. One of the most pervasive and misunderstood ideas is the "danger" of oxygen delivery leading many medical professionals to avoid giving oxygen. Remember that oxygen is the food for every cell of the body. Without this food, cells will die! Hypoxia can damage the brain in approximately four minutes. Damage caused by exposure to too much oxygen is a much slower process. It is essential to understand that you must give more oxygen if levels are low and *do not withhold oxygen* if your patient is showing signs of lower SpO_2. Just avoid delivering higher oxygen than you need to for extended periods of time.

Key Takeaway

When choosing between treating hypoxia (low oxygen) and hyperoxia (too-high oxygen), it is always better in the short term to treat life-threatening hypoxia. Brain trauma and hypoxic injury can happen in a matter of minutes, versus the release of oxygen free radicals that has a slower onset of hours or days.

Object Lesson

It's like the children's story of "Goldilocks and the Three Bears". With oxygen, we really don't want "too little," which leads to hypoxia, causing tissue damage. We also don't want "too much," especially for too long, which leads to hyperoxia, causing release of oxygen free radicals and damage to cellular membranes. We really want it to be "just right," with an SpO_2 greater than 92% but less than 100%. Oxygen levels delivered can be **titrated** up or down, which means that it will be increased or decreased slowly in a step-wise fashion, to ensure SpO_2 is $92 - 99\%$ for healthy individuals.

Photo via NYPL's Public Domain Archive, Public Domain

SpO_2 only goes as high as 100% once all the hemoglobin is bound to oxygen. Any additional oxygen that is in the lungs is not captured by hemoglobin. This means that a person with an SpO_2 of 100% on FiO_2 0.60 could potentially still have an SpO_2 of 100% on FiO_2 0.50. There could be an excess of oxygen present. This excess of oxygen would drive the PaO_2 much higher than the normal targeted range of $80 - 100$ mmHg since the high concentration of oxygen in the alveoli would drive more oxygen to diffuse into the plasma.

You can test for excess oxygen by taking an arterial blood gas (ABG sample) and test the PaO_2. As stated, the normal is $80 - 100$ mmHg. A person with an SpO_2 of 100% could have an PaO_2 of 100 mmHg or upwards of 400 mmHg. PaO_2 that are higher than 100 mmHg are indicative of over oxygenation, or hyperoxia. High levels greater than 100 mmHg increase the risk of oxygen free radicals and lung damage.

Since there is no way to tell from an SpO_2 of 100% whether the person is getting just enough oxygen or too much, an easy way to avoid this situation entirely is to target an SpO_2 of $92 - 99\%$ and not let the SpO_2 sit at 100% unless the oxygen is down to minimum (i.e., FiO_2 0.21).

Remember that oxygen content (CaO_2) equation? You may recall that PaO_2 was part of the dissolved oxygen portion. As long as the individual has adequate hemoglobin that is functioning appropriately, high PaO_2s do not contribute a significant amount of oxygen compared to PaO_2 of 100.

Let's compare two examples of the content of oxygen in the blood. Both examples have the same amount of hemoglobin saturated within a safe range. Patient X is getting "just enough" oxygen to saturate their hemoglobin to 97% (a safe level), but not excess oxygen with the partial pressure of oxygen at 100 mmHg. Comparatively, Patient Y is getting too much oxygen. Their hemoglobin is fully saturated with the partial pressure of oxygen in their blood reading above the normal levels. Compare the oxygen content of these two patients below.

Table 2.5.1: Content of Oxygen in the Blood – Examples

Patient X: Receiving FiO_2 0.40	Patient Y: Receiving FiO_2 1.00
Hgb 85, SpO_2 97%, PaO_2 100 $\Rightarrow$ Normal targeted PaO_2	Hgb 85, SpO_2 100%, PaO_2 400 $\Rightarrow$ Hyperoxia, too much oxygen
$CaO_2 = (85)(1.34)(0.97) + (0.003)(100)$	$CaO_2 = (85)(1.34)(1.00) + (0.003)(400)$
$= 110.5 + 0.3$	$= 113.9 + 1.2$
$= 110.8$	$= 115.1$

Compare the calculated oxygen content (CaO_2) for both patients. The calculated CaO_2 of both of these patients are very close though they are receiving very different amounts of oxygen. This model shows that, in people with normal hemoglobin, delivering just enough oxygen for saturating hemoglobin adequately is the most important aspect of oxygenation in the blood. Over-delivering oxygen does not significantly contribute to improving the amount of oxygenation if the saturation of hemoglobin does not change. Therefore, the damage of delivering high levels of O_2 and the creation of oxygen free radicals far outweighs the benefits of dissolving more oxygen into the blood, and it should be avoided.

2.6 SUPPLEMENTAL OXYGEN AND MECHANICAL VENTILATION

Before going into the modes of ventilation, it is important to have a basic understanding of oxygen delivery and the goals of treatment. In the atmosphere, we all breathe 21% oxygen, with the balance being mostly nitrogen (approximately 78%) with some other trace gases. This level of 21% oxygen is high enough for a healthy person to take the amount of oxygen they need out of the air. This level can be increased with oxygen delivery up to 100% or pure oxygen. If the lungs are unhealthy or compromised, they need higher percentages of oxygen given to get enough oxygen into the body.

Room air is a slang medical term for the lowest level of oxygen that will ever be given to a patient. It is the composition of atmospheric air—21% oxygen.

When you give oxygen to a patient, it can be expressed in multiple ways. When the entire breath is being given via an oxygen delivery device and the patient does not breath any air other than what is given, it can be expressed as a percentage $(\%)$ from 21% to 100%, or a **Fraction of Inspired Oxygen (FiO_2)**, which is expressed as a decimal from 0.21 to 1.00. This is a pre-mixed amount of oxygen blended with regular room air to deliver the percentage you desire from the lowest (0.21 or **room air**) to pure oxygen (1.00).

Key Takeaway

Mechanical ventilation is a sealed system and every breath is fully delivered by the ventilator. You must decide the percentage of oxygen the patient needs to breathe. Oxygen is expressed in FiO_2 on ventilators and can range from 0.21 to 1.00.

When pure oxygen is given in small doses to a spontaneously breathing patient, but the patient also breathes in room air around the oxygen to make up some of their breath, it is termed **supplemental oxygen**, and delivery is usually expressed in litres per minute (Lpm) of O_2 delivery. Supplemental oxygen can be increased to almost equal 100% oxygen depending on the interface it is supplied with (nasal prong or face masks).

When a person is sick and has an acute disease process happening in their body, the oxygen demand of their cells and vital organs is increased. Often, supplemental oxygen delivery can meet this need without needing to go the mechanical ventilation route. But how do you know if your patient needs more oxygen?

The most direct way to monitor oxygen level is through the **saturation of hemoglobin in the blood (SaO_2)**. A healthy individual will have their **hemoglobin (Hb)** very close to fully saturated: which means $95 - 100\%$ attachment to oxygen. This

percent saturation reflects the total binding sites available for oxygen on the hemoglobin in the blood and compares the amount bound with oxygen to the total.

> SaO_2 or SpO_2: what is the difference? SaO_2 is the saturation of Hb in oxygenated arterial blood. It is read directly from a blood sample. SpO_2 is also the saturation of Hb, but it is read through a pulse oximeter on the finger using light refraction to estimate the binding of hemoglobin. Usually, in most cases, the SpO_2 is almost identical to the SaO_2. Most clinicians will utilize SpO_2 to monitor oxygen levels as getting an arterial sample is an invasive procedure only select medical professionals can perform. SpO_2 is a very useful non-invasive tool to monitor oxygen levels

Hemoglobin has four binding sites available for oxygen to bind, as you can see in the following video (watch video only up to 2:41):

One or more interactive elements has been excluded from this version of the text. You can view them online here: https://ecampusontario.pressbooks.pub/mcvresource/?p=48#oembed-1

Video: "Respiratory System, Part 2: Crash Course Anatomy & Physiology #32" By CrashCourse [10:23] *Transcript Available*

Many factors can affect the oxygen's ability to bind to the hemoglobin. The most important concept to remember is that increasing the amount of oxygen available to bind (increasing oxygen delivery) usually can increase the binding of the oxygen to the hemoglobin.

If SpO_2 is lower than 92%, this usually suggests the patient will require some amount of supplemental oxygen. Supplemental oxygen can be started and increased to attempt to increase the SpO_2 to above 92%. Mechanical ventilation would be a subsequent step when high levels of supplementary oxygen is not adequate to support the patient's oxygen needs, in order to prevent hypoxic failure.

Key Takeaway

If the patient's SpO_2 is less than 92%, this is evidence of lower oxygen levels in the body, and the patient may benefit from supplemental oxygen.

Total Oxygen Content in the Blood

Oxygen saturation is not the whole picture of oxygenation. The total **oxygen content (CaO_2)** in arterial blood—or, oxygen being

delivered to the vital organs—is the sum of two distinct factors. Primarily, oxygen is attached or "bound" to hemoglobin (represented by the SpO_2) and secondly, a small amount diffuses through the alveolar-capillary membrane and dissolves into the blood plasma because of the high amount of oxygen in the alveoli compared to the blood. This is the same principle of air flowing from a high pressure to a low pressure that we learned about in Chapter 1, only this time it is referring to oxygen flowing from an area of high "density" of oxygen in the alveoli to lower "density" of oxygen in the blood. This description of density is referred to as a partial pressure. It is expressed as a PAO_2 (partial pressure of oxygen in the alveoli) and PaO_2 (partial pressure of oxygen in the artery)

The formula to determine total oxygen content in the blood (CaO_2), is as follows (units are omitted for simplicity). Note: This is not math that you need to do on a regular basis. It is not essential to do this calculation to ventilate patients safely. It is more important that you understand the concepts here and be able to apply them to oxygenation.

$$\text{Oxygen Content } (CaO_2) = \underbrace{(\text{Hb})(1.34)\left(\frac{\%SaO_2}{100}\right)}_{\text{Oxygen bound to Hb}} + \underbrace{(0.003)(PaO_2)}_{\text{Diffused Oxygen}}$$

The 1.34 is constant and represents the maximum amount of oxygen that can bind to $1\ \text{gram}$ of Hb. The 0.003 represents a constant that the partial pressure of oxygen dissolved in the blood (PaO_2) is multiplied against. Normal or "targeted" PaO_2 are $80 - 100\text{mmHg}$.

Let's look at the formula in action. If Patient A has a Hgb of 120, SaO_2 of 99% and a "normal" PaO_2 of 100mmHg, here is the formula:

$$\begin{aligned}\text{Oxygen Content } (CaO_2) &= (120)(1.34)(0.99) + (0.003)(100)\\ &= 159.1 + 0.3\\ &= 159.4\end{aligned}$$

For more information on the Oxygen Content Formula, check out this video:

One or more interactive elements has been excluded from this version of the text. You can view them online here: https://ecampusontario.pressbooks.pub/mcvresource/?p=48#oembed-2

Video: "Easy Ways to Calculate Oxygen Content of Blood" By 100lyric [8:58] *Transcript Available*

Looking at the calculated oxygen content of bound oxygen and diffused oxygen, it is obvious that diffused oxygen is a negligible amount when comparing to the amount of oxygen that is bound to hemoglobin. This is true in most cases and is why SaO_2 can be used to approximate the overall oxygen content in most standard cases. However, some distinct situations where diffused oxygen might have more of an impact on overall oxygenation include cases of inadequate or abnormal hemoglobin. In these cases:

- the hemoglobin is at dangerously low levels (anemia),
- the hemoglobin is attached to carbon monoxide and not available for oxygen to attach (carbon monoxide toxicity), or
- the oxygen is literally sticking to the hemoglobin and not unloading to the tissue (shifts in the oxy-hemoglobin curve).

These situations might require high PaO_2s well above normal targeted ranges to compensate for lack of oxygen delivery via hemoglobin, allowing the body additional dissolved oxygen to be available in these rare cases until the problem is fixed. Remember, these are the exceptions and not the normal.

Other than these special circumstances, the diffused oxygen portion of the formula represents a very small portion of the oxygen content available in the blood. Therefore, for general understanding, SaO_2 can be used as a general overview of the oxygenation status of the patient. As previously discussed, SaO_2 and SpO_2 are usually the same. We will use SpO_2 as our primary method of determining oxygen requirements for the purpose of this book.

Key Takeaway

SpO_2 is usually a good indicator of SaO_2. SaO_2 is the primary impact on oxygen content for a patient. Therefore, SpO_2 can usually be used to monitor a patient's overall oxygenation status.

2.7 THE RELATIONSHIP BETWEEN PEEP AND FIO2

At this point in your learning, you should understand how increasing the FiO_2 can directly impact the amount of oxygen available to the body through the oxygen content equation—specifically hemoglobin amounts and SpO_2.

There is another factor that can affect oxygenation, and this factor becomes extremely important when it applies to ventilation and specifically ventilating potentially sick individuals with lung pathologies.

In Chapter 1, we learned that Positive End Expiratory Pressure (PEEP) describes the little bit of air that remains in the alveoli after exhalation has ended. As we begin to discuss mechanical ventilation, PEEP is also set on the ventilator. As you may have already surmised, the PEEP setting on the ventilator is meant to mimic the physiologic properties that occur when spontaneously breathing. When we move to mechanical ventilation and switch to positive pressure in the lungs, we need to ensure the lungs do not fully empty at the end of exhalation. Remembering the pressures noted in spontaneous breathing, the negative pressure in the pleural space ensures some air is maintained in the lungs to keep them inflated. Utilizing positive pressure from a ventilator eliminates this ability and must be artificially added via the ventilator.

We set the ventilator to have PEEP to replace the air that is normally left in the lungs. PEEP is, at minimum, always set to $5\ cmH_2O$. This ensures the "balloons" that are the alveoli do not fully empty, and that residual volume we talked about in Chapter 1 is left in the lungs to make them easier to blow up for the next breath.

PEEP has other functions as well. Not only does it ensure the lungs do not fully collapse and are easier to inflate from breath to breath, but this same pressure can also augment oxygenation by adding an extra "push" to get the oxygen across the alveolar-capillary membrane.

Object Lesson

Imagine you are trying to dry a wet shirt. You wrap the shirt in a towel, and you're hoping to transfer the water from the soaked shirt to the dry towel. The water is the "oxygen" and you want to get it across the "membrane" (into the towel). PEEP is equivalent to wringing/squeezing out the towel-wrapped shirt, pushing the water (oxygen) across at a faster/higher rate.

Photo by Teona Swift, Pexels Licence.

PEEP is used specifically in cases where the alveolar-capillary membrane is thickened or scarred due to pathology or disease of some kind. As inflammation or infiltrates permeate the alveolar-capillary membrane, this would impede the ability of oxygen to diffuse across the membrane. Thinking back to lung concepts and air flowing from high to low pressure, PEEP ensures that gradient exists at a higher degree and oxygen diffuses easier down the steeper gradient. As a general rule, in cases where the alveolar-capillary membrane is abnormal (i.e., non-healthy lungs), increasing PEEP will improve oxygen in the blood without having to increase FiO_2.

Object Lesson

Think of PEEP and FiO_2 as two people lifting a coffee table. They work as partners to move the table in the same way that PEEP and FiO_2 work together to improve oxygenation. If one person works harder to lift the table, the other person will not have to work as hard. Similarly, increasing PEEP should allow you to decrease the levels of oxygen that your patient requires, minimizing exposure to high levels of oxygen.

Photo by Blue Bird. Pexels Licence.

Key Takeaway

PEEP is another setting that will *always* be set on a ventilator regardless of the mode. Keeping the alveoli from fully collapsing is essential to mimicking normal physiologic processes. It can be used to improve oxygen diffusion across the alveolar-capillary membrane.

PEEP is not a fix-all. There are side effects of increasing PEEP—especially at higher levels. As you increase the PEEP, this increases the pressure in the lungs, which in turn can compress the heart and decrease venous return and blood flow to the heart. Cardiac output can be compromised if PEEP is increased too high. PEEP should be increased slowly with constant evaluation of cardiac status.

Another important side effect of PEEP is the effect on lung compliance. Remember, you can think of PEEP as the "air left in the balloon." If you have a set volume that you are inflating the balloon every time, but you increase the baseline amount of air in the balloon, eventually the balloon will either not be able to take the amount of air you want to blow in, or it will pop. This is the same for the lungs; if the lungs are more full at the start of the breath (increase in PEEP), they will be less likely to "fit" the volume of air the ventilator is about to give without exposing the lungs to high pressure or causing overdistension. In general, PEEP should be increased carefully and with close monitoring and patient assessment. These concepts will be revisited in Chapters 3 to 5 when discussing ventilation settings in more depth.

2.8 BASIC VENTILATION MODES. THE VENTILATION "FAMILY TREE"

Unless specified otherwise, in this chapter, ventilation modes will refer to modes used with invasive mechanical ventilation. A ventilation mode is a set of predetermined rules, that the ventilator follows to provide or support ventilation. If you imagine ventilation like a family tree, control and spontaneous are the main branches. Up to this point, we discussed the classification of ventilation modes based on the type of breath delivered: controlled, assisted or spontaneous. We also know that the timing of the breath delivery also makes a difference in the classification of ventilation modes. Mandatory breaths are delivered at a certain rate in continuous mandatory ventilation (CMV). Patients will take spontaneous breaths in continuous spontaneous ventilation (CSV). A third option is available as intermittent mandatory ventilation (IMV) or Synchronized Intermittent Mandatory Ventilation (SIMV), which allow controlled (mandatory) breaths, assisted breaths and spontaneous breaths, based on patients needs. This is only part of the story. For complete description of a ventilation mode, the control variable must be included.

Control modes are further split into two main sub-categories:

1. **Volume Control**— the ventilator will deliver a set volume of air to the lungs
2. **Pressure Control**—the ventilator will deliver a set pressure of air to the lungs

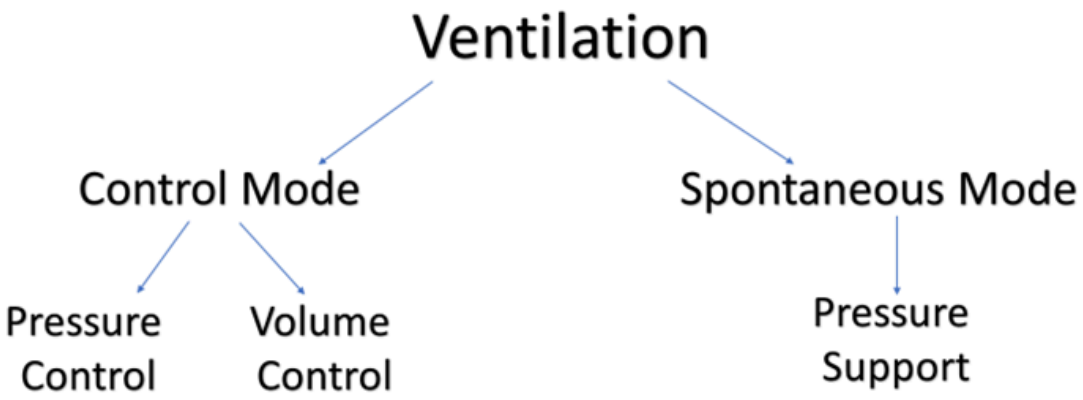


Figure 2.8.1: "Ventilation Family Tree " by Melody Bishop, CC0

Irrespective of the labelling or description of control breaths, it is important to remember that they are predetermined and delivered by the ventilator. Both categories lead to the same outcome, which is a fully "controlled" breath that the patient will receive. Essentially, every controlled or mandatory breath, whether labelled as volume or pressure breath, refers to the same process of the ventilator pushing air into the lungs. The terms "pressure" or "volume" simply describe the settings on the ventilator. Both pressure and volume modes accomplish the same objective of delivering air into the lungs based on the clinician's selected settings and allowing passive exhalation. The distinction lies in the parameter being controlled during each breath

For a patient with intact drive to breathe, taking spontaneous breaths, most commonly, breaths are delivered via **pressure support** ventilation.

Key Takeaway

Pressure or volume control modes describe the same thing: air being pushed into the lungs is set by a medical provider and does not need the patient breathing. Differentiating between pressure and volume is just how we describe the air the ventilator is pushing in.

2.9 VENTILATION MODES: "ALPHABET SOUP"

One of the toughest challenges when explaining modes of ventilation is the terminology and the common practice of using acronyms to describe modes and parameters. When it comes to the mode of ventilation used, it can often feel like you are reading random letters from a bowl of alphabet soup!

Photo by Sigmund, Unsplash License

Where things become really complicated is that every company that manufactures ventilators has slightly different names for all of their modes. When a mode is created, the manufacturer patents the specific name used. Once the patent allows for another company to utilize the intellectual principle used in that mode, other manufacturers create their own version of the mode with a slightly different name. This results in the same mode having sometimes upwards of 5 or 6 different names. For example, Volume Control can be VC, VCV, ACVC, or CMV-VC—just to name a few of the countless variations.

Try to look past the alphabet soup and identify the mode by what parameters are being set. Most modes use the same principles in their breath delivery. Examine the settings that a practitioner would set, and then classify the type of mode you are working with:

- Is the mode control or spontaneous?
- If the mode is control, is it pressure control or volume control?

Don't worry if that sounds difficult—you're about to learn more about how to develop this skill. If you can identify the mode

accurately, short-forms and acronyms won't confuse or overwhelm you. You can then apply the general knowledge from this book to any mode of ventilation you are exposed to—even adjunct or special modes not covered in this book—and have a better grasp of how to use it.

Ten maxims for understanding ventilation modes have been described. These maxims may help put some order in the alphabet soup described above.

Table 2.9.1 The 10 Maxims for Understanding Ventilation Modes Classification

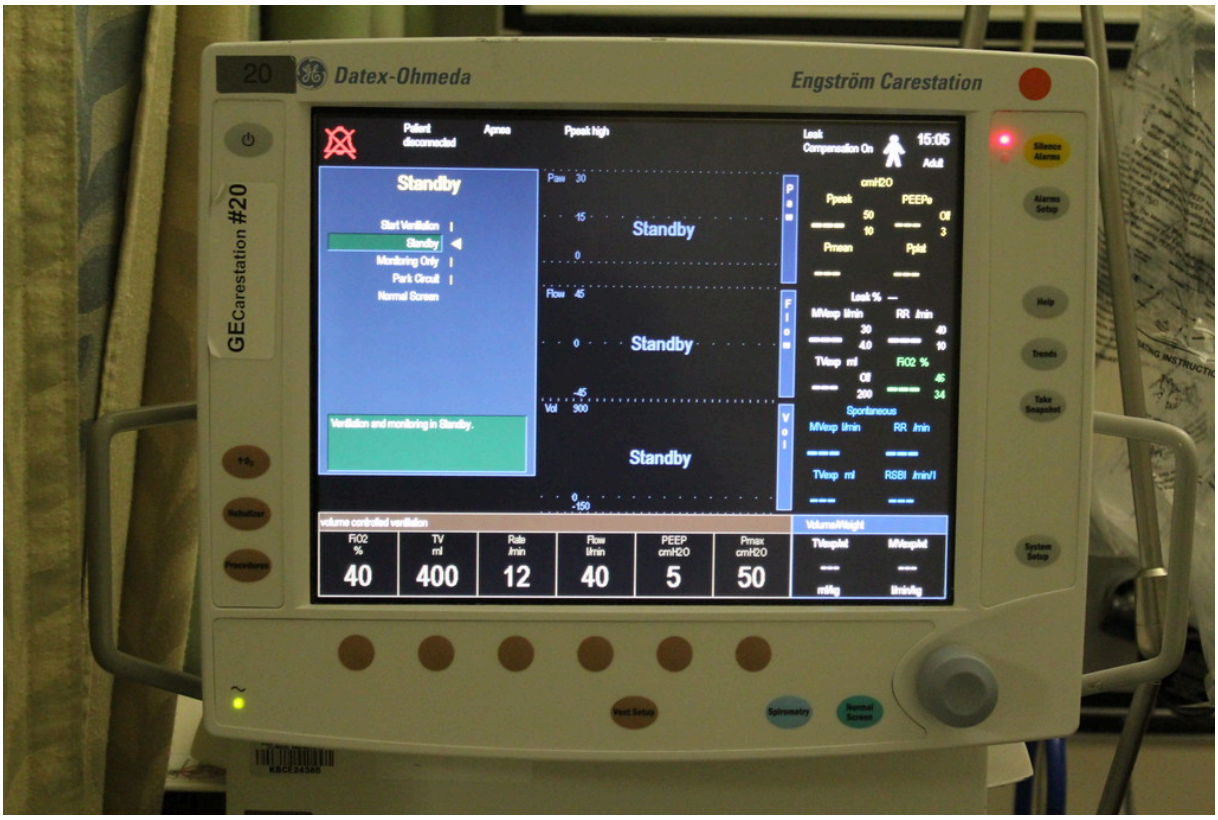
The 10 Maxims for Understanding Ventilation Modes Classification
1. A breath is described as one cycle of inspiration and expiration. With inspiratory time describing the interval from when airflow begins to move into the lungs, to when airflow changes direction at the start of expiration.
2. A breath is assisted if the ventilator provides some or all the work
3. The ventilator provides assisted breaths by using either volume control (VC) or pressure control (PC), based on the equation of motion.
4. Breaths are classified based on phase variables (trigger and cycle)
5. Trigger and cycle variables can be either patient or ventilator initiated
6. Breaths are classified as mandatory or spontaneous based on phase variables
7. There are three basic breath sequences: continuous mandatory ventilation (CMV), intermittent mandatory ventilation (IMV) and continuous spontaneous ventilation (CSV).
8. There are five basic ventilatory patterns: VC-CMV, VC-IMV, PC-CMV, PC-IMV, and PC-CSV.
9. Within each ventilatory pattern there are several types that can be distinguished by their targeting schemes (example: dual, Servo, adaptive)
10. A mode of ventilation is classified according to its control variable, breath sequence and targeting scheme.

Data Source: Chatburn et al., 2014.

2.10 SETTINGS AND DEFINITIONS

Control modes

The most important thing to remember is that control modes are designed to completely replace the physiological process of breathing. Every aspect of the breath must be set and "controlled" by the clinician. Every single breath that is delivered by the ventilator, whether initiated by the ventilator or triggered by the patient, is exactly the same and depends completely on the parameters set.



What settings do you recognize on this ventilator control panel? Photo by Quinn Dombrowski, CC BY-SA 2.0

So, what do you need to set when working with a control mode? With *any* mode of ventilation, you always set the following two settings:

1. the level of PEEP to keep the alveoli from collapsing
2. the amount of FiO_2 to adequately oxygenate your patient

In addition to these mandatory settings for all modes of ventilation, in control modes you also have to set three parameters:

1. How *often* the patient will breathe
2. How *big a breath* the patient will get

3. How *fast* the air will be given to the person

How often breaths will be delivered is usually expressed by a **Respiratory Rate (RR)**. This is the minimum number of breaths that is delivered over the course of one minute. Patients can trigger breaths above this rate, and these additional breaths will be reflected in the Total RR.

How big a breath the patient will get is usually expressed by the volume of air that is pushed into the lungs. The term for this is **Tidal Volume (V_T)**, which refers to the volume of air in mLs that is given every time a breath is delivered. The volume of air delivered can be altered depending on whether the mode is a volume or pressure control, via describing the volume itself or the pressure applied to the lungs (expressed as the **Pressure Control** or PC). You will learn this concept in detail in Chapter 4.

> You will notice that the term for the amount of pressure applied to the lungs when in a Pressure Control mode is also commonly referred to as the "pressure control." It can also be referred to as the **inspiratory pressure**. Don't get confused here. It is straightforward: if there is a "pressure control" set, then you know you are in a Pressure Control Mode!

Modes will also allow you to change how long the breath of air takes to be given. Changing the length of a breath is accomplished by changing the speed by which the air is pushed in by the ventilator. Volume and Pressure Control modes allow you to adjust the speed of air being delivered by either adjusting the **Flow Rate ($\dot{V}$)** or the time the air is delivered (referred to as **Inspiratory Time** or I_T). You can learn all about these settings in Chapter 4.

In summary, you know you are in a control mode when the following three parameters are set:

1. A Respiratory Rate
2. A Volume or Pressure applied by the ventilator
3. The Flowrate or Time the air is being pushed into the lungs

Key Takeaway

No matter the acronym you see in the mode name, if respiratory rate, volume or pressure, and flowrate or time are set, you know you are in a control mode.

Spontaneous modes

Like control modes, spontaneous modes of ventilation are also delivered via positive pressure application from a mechanical ventilator. The same mandatory settings must be set, including:

1. the level of PEEP to keep the alveoli from collapsing
2. the amount of FiO_2 to adequately oxygenate your patient

But, unlike control breaths, in spontaneous modes, the patient initiates their own breath and is responsible for deciding how many breaths to take and how often they want to breathe. You do not decide the set volume or rate that the patient will breathe. Here, you only decide how much "extra help" the ventilator will give to facilitate the breath the patient is taking in. The main setting that matters here is:

1. How much *support* we want to give to the patient

When the patient initiates a breath via their physiological trigger and a contracting diaphragm, the ventilator senses this contraction and "helps" the patient pull in air by pushing a little while the patient pulls the air in. This support lightens the respiration workload the patient has to do.

Object Lesson

Imagine you're a child outside playing on a hot summer day. You spot a garden hose nearby and decide to take a drink. Although the hose is turned off, there is cold water in the hose line. You create suction and try to pull in the water. You need to work to get the water you need. If someone were to turn on the hose, this would help you get water easier. If the water was only turned on as a "trickle", you might want more water than that and still use suction to pull more from the hose. If the hose was turned on higher, you could easily take all the water you want with no "work" from sucking on the hose.

Photo by US Environmental Protection Agency, Public Domain

In summary, you know you are in a spontaneous mode by what is *not* set. There is no set respiratory rate or set volume or flow rate. You only set a pressure to help augment the breath.

2.11 VENTILATOR SETTINGS TABLE

The main ventilator settings that are important to understand are:

Table 2.11.1: Important Ventilator Settings

Ventilator setting	Description	Unit of measurement	Used in which mode(s)?
Respiratory Rate (RR)	The number of breaths per minute. It can refer to set RR—how many times a minute the ventilator will deliver a mechanical breath; RR is set by the health care professional, and is consistent unless changed. It can also refer to spontaneous RR, which is how many times a minute the patient is breathing—decided by their individual drive to breathe and their own brain trigger.	breaths per minute (bpm)	A baseline is *set* in control modes, but patients can breathe above the set rate. *Monitored* in both mode types to see what the patient is doing.
Tidal Volume (V_{TM})	The volume of air filling the lungs during a breath.	mLs (of air)	Can be *set* on the ventilator in Control modes (specifically in Volume Control). *Monitored* in spontaneous modes to see what the patient is able to draw from the ventilator.
Pressure Control (PC) or Inspiratory Pressure (P_{insp})	The amount of pressure at which the ventilator will deliver every single breath to inflate the lungs.	cmH_2O	*Set* in Control modes (specifically in Pressure Control).
Pressure Support (PS)	The amount of "extra help" the ventilator will give whenever the patient spontaneously breathes. It is meant to help unload some of the work of breathing that the patient may have.	cmH_2O	*Set* for spontaneous modes only.
Inspiratory Time (I_T)	The amount of time air/pressure is given by the ventilator to fill the lungs for a mandatory breath.	seconds (s)	*Set* in Control modes only.
Flow ($\dot{V}$)	The maximum speed that the air can travel coming out of the ventilator when giving a breath.	liters per minute (Lpm)	*Set* in Control modes only.
Positive End Expiratory Pressure (PEEP)	Substitutes for FRC since intubating and attaching to a ventilator bypasses the normal pressures in the lungs and maintaining "physiologic PEEP." PEEP is important for stopping the alveoli from collapsing and making it easier to inflate the lungs (see Chapter 1).	cmH_2O	*Always set* whenever a person is attached to a ventilator in both control and spontaneous modes. It is necessary to try to maintain a normal physiologic process.
Fraction of inspired oxygen (FiO_2)	The percentage of oxygen the patient requires to support their oxygen needs. Normal air is approximately 21% Oxygen, so a setting of 0.21 is the absolute lowest you will ever go. The maximum amount of oxygen that can be delivered is 100% (pure oxygen)—so 1.00 is the highest possible setting.	a decimal from 0.21 to 1.00	*Always set* whenever a person is attached to a ventilator in both control and spontaneous modes.

Not all of these settings are set every single time. Depending on the mode of ventilation, different settings will be used. The following Venn diagram summarizes when settings are used:

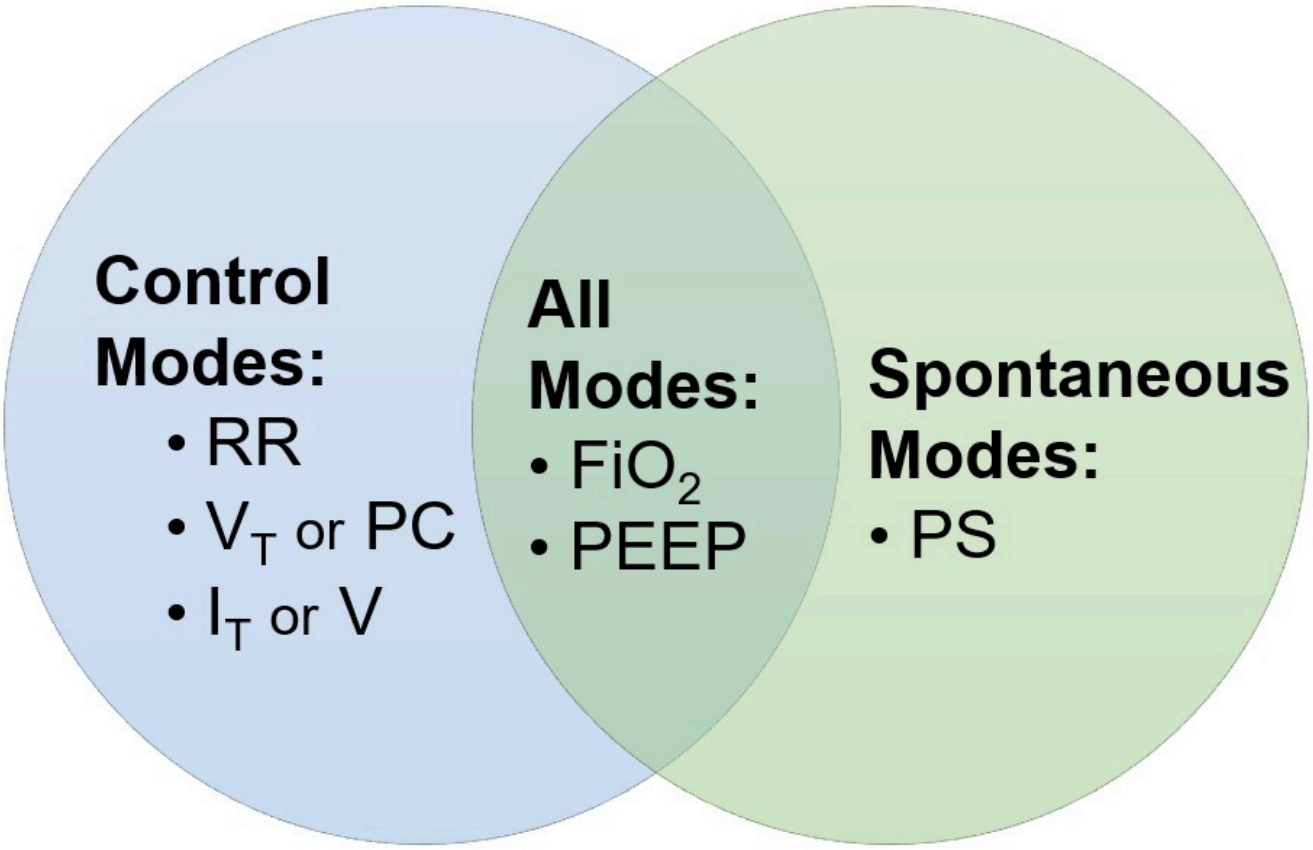


Figure 2.11.1: Ventilator Settings Organized by Mode © Melody Bishop

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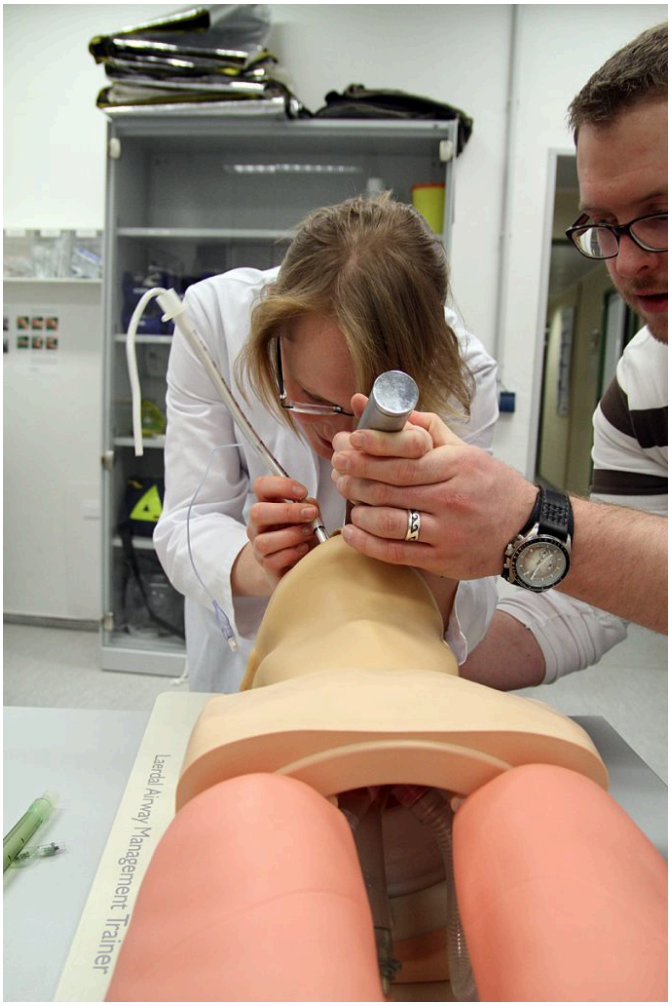
2.12 CONCLUSION

You did it! Reaching the end of Chapter 2, you should now understand the key differences between spontaneous breathing and mechanical ventilation. You also now have a strong foundation in understanding oxygenation and how best to approach hypoxia in a patient. Keep these concepts in mind as we build on them in the next chapters.

Review

Please review the following key points from this chapter:

- Mechanical ventilation is a push of air into the lungs with passive exhalation of the lungs once the breath is done being delivered.
- Oxygen content in the blood is the sum of the amount of oxygen bound to hemoglobin and the amount of dissolved oxygen in the blood. The amount of hemoglobin and percent saturated has a larger affect on oxygenation then the dissolved component.
- SpO_2 can be used to approximate the overall oxygen status of your patient
  There are two settings that are always set on a ventilator: FiO_2 and PEEP.
- FiO_2 and PEEP both have an impact on oxygenation and can be adjusted to "share the work" of improving oxygenation.
- How a breath is delivered depends on the mode type. There are two main classifications of ventilator modes: control and spontaneous.
- Control modes are completely determined by the clinician on how often and how big a breath the patient gets. Each breath is completely identical.
- Spontaneous modes are decided by the patient, but supported by some degree (decided by the clinician) by the ventilator.
- Certain ventilator settings are associated with the different ventilator modes.
- There are two settings that are always set on a ventilator regardless of mode: FiO_2 and PEEP.



Student tutor guides student in endotracheal intubation. Photo by Exoport, CC BY-SA 3.0

Now, you have been introduced to modes of ventilation as well as basic settings. Don't worry if you are still a little confused by some of these concepts. You'll get a lot more information in the next few chapters, which will explore each one these settings with a depth of detail.

If you would like to go through some of these concepts again in video format, try watching "Basic Vent Modes MADE EASY – Ventilator Settings Reviewed" by ICU Advantage.

One or more interactive elements has been excluded from this version of the text. You can view them online here: https://ecampusontario.pressbooks.pub/mcvresource/?p=84#oembed-1

Video: "Basic Vent Modes MADE EASY – Ventilator Settings Reviewed" By ICU Advantage [24:40] *Transcript Available*

2.13 SELF-CHECK

Chapter 2 Quiz

See how well you recall the concepts covered in this chapter by completing the following short quiz.

Don't forget to check your score!

An interactive H5P element has been excluded from this version of the text. You can view it online here: https://ecampusontario.pressbooks.pub/mcvresource/?p=86#h5p-3

CHAPTER 3 | VENTILATOR SETTINGS

Chapter Outline

3.0 CHAPTER OVERVIEW

In this chapter we will discuss specific features of volume and pressure control modes, and how to approach choosing initial settings for a patient when we first initiate mechanical ventilation.

> The scope of this book is adult ventilation. The settings discussed in this chapter are suggestions for adult populations only. Hospitals may have specific policies and procedures or physician orders above these recommendations. If the patient is pediatric or neonatal, similar rules do apply, but they are not fully discussed in this book.

In this chapter we will cover:

- Commonly used initial settings in control modes
- How to calculate Ideal Body Weight and implications of it in volume control and pressure control
- How to calculate safe ranges for tidal volumes and flow
- Suggested settings for ventilator alarms

Application

In most cases, arterial blood gas (ABG) results guide clinicians in making informed decisions about ventilator settings. But we need to start somewhere! Clinicians often choose initial settings by employing educated guesses regarding their patient's ventilatory needs based on how they presented prior to intubation. This chapter will help you to make these educated guesses effectively, and use this information to choose appropriate ventilator settings. Even if you have ABGs to begin with, the information in this chapter is still essential to understanding how the patient's presenting status impacts your decisions about ventilator settings.

Learning Objectives

At the end of this chapter, you will be able to:

1. Identify which initial settings are mandatory in volume control and pressure control.
2. Describe how patient status impacts decisions about ventilator settings.
3. Explain why Ideal Body Weight and the safe range for tidal volumes became standard practice for ventilation decision-making.
4. Calculate Ideal Body Weight for a patient.

5. Calculate the safe range for tidal volumes for a patient.
6. Choose recommended ventilator settings based on your mode and patient status.
7. Set ventilator alarms effectively to ensure safe monitoring of the patient when not at bedside.

Key Terms

In this chapter, you will learn about all of the following key terms. These terms will be used throughout this book, so it is important to take the time to master them and practice your recall often.

- recruiting
- titrate
- atelectasis
- tachypnea
- Arterial Blood Gas (ABG)
- Ideal Body Weight (IBW)
- Ventilator Induced Lung Injury (VILI)
- Acute Respiratory Distress Syndrome (ARDS)
- barotrauma
- volutrauma
- decelerating flow pattern
- pressure control
- volume control

Whenever these terms are first introduced in this chapter, they are bolded. However, if you need additional information about a term than what is provided here, you can research it in The Free Dictionary: Medical Dictionary.

3.1 MANDATORY SETTINGS IN CONTROL MODE: A REVIEW

As previously stated, for all mechanical ventilation modes, the clinician will always set FiO_2 and PEEP. You need to ensure the alveoli do not collapse since mechanical ventilation is bypassing the normal pleural pressures that keep them inflated (please review Chapter 1 if you need to). You also need to always set the oxygen concentration the patient is going to require to maintain their blood oxygenation, which should be $SpO_2 > 92\%$ (please review Chapter 2 if you need to).

In addition to FiO_2 and PEEP, in control modes specifically, the ventilator is driving how big a breath or how often (at minimum) the patient will breathe, so you need to set a Respiratory Rate (RR). In addition to the breathing frequency (respiratory rate), the clinician must also direct the size of the patient's breath in control modes. For volume control, this means setting a tidal volume while in pressure control, we set the pressure control applied to cause a volume. The control variables are the physical parameters that can be manipulated by the ventilator. There are three variables that the ventilator can control: pressure, volume and flow. Time, as a control variable, is implicit, because we set a respiratory rate, and we learned that by setting a certain respiratory rate per minute, we control the total cycle time. Note that the ventilator can only control one of these variable at a time.

Initial settings to be chosen by the clinician are as follows:

Table 3.1.1: Initial Settings – Volume Control & Pressure Control

Volume Control	Pressure Control
FiO_2	
PEEP	
Respiratory Rate (RR)	
Tidal Volume (V_T) and Inspiratory Flow $(\dot{V})$	Pressure control (PC) and Inspiratory Time (I_T or I_{Time})

There are other settings that you will see on the ventilator that are not listed here. One will be trigger sensitivity, a cycling mechanism that will be discussed in detail as we dive deeper into specific ventilation modes. These settings are pre-programmed with mechanical ventilators to default to numbers that work the majority of the time.

Check Your Knowledge

Remember, every ventilator is a little bit different. The initial settings are always there, but they will be labelled differently depending on the ventilator manufacturer. Challenge yourself by trying to locate the common initial settings (FiO_2, PEEP, Respiratory Rate, Pressure Control and I_{Time}) on the ventilator pictured below.

Tip: This exercise will only allow you to locate one setting at a time. After you have located a setting, if you would like to locate additional settings from the list in the activity instructions, please refresh your browser page to clear your results.

An interactive H5P element has been excluded from this version of the text. You can view it online here: https://ecampusontario.pressbooks.pub/mcvresource/?p=116#h5p-5

The Relationship Between Pressure and Volume

So, we know that pressure control and volume control are essentially the same concept explained in different ways—air being pushed into the lungs. Let's explore this concept further to explain why this is true.

When air is mechanically delivered into the lungs via a positive pressure breath, two things occur at the same time inside the lungs:

- The volume of air inside the lungs increases
- The pressure inside the lungs increases.

These effects happen because, with a positive pressure breath, it is a closed system with the patient connected to the ventilator through an endotracheal tube. The air has nowhere else to go but to inflate the lungs. In a sealed system, volume and pressure go hand in hand and have a direct, linear relationship. As one increases, the other increases as well.

As we introduce this concept, we are going to think of the lungs as an unchanging environment. We know that lungs can be damaged—due to illness or other factors, which can affect how easily lungs inflate, but let's talk about a short-term situation where the lungs themselves are not changing. We are going to look at how the ventilator settings impact each other if inflating the same set of lungs, using the following object lesson.

Object Lesson

Let's go back to that analogy of lungs as balloons to understand this concept fully. Imagine you have two identical balloons side by side. If you blew for 1 second at a soft pressure into Balloon 1 and then blew for 1 second at a hard pressure into Balloon 2, which balloon do you think would have a larger volume in it at the end? When you think of balloons, it makes it easy to see that if you blow harder, there will be more air (or volume) in Balloon 2.

Let's look at this another way, using our two identical balloons again. If you have the same amount of time to blow up both balloons, but Balloon 2 you only want to blow up to half the size of Balloon 1,

Photo by Kampus Production, Pexels License

which balloon do you think you will have to blow harder (more pressure) to achieve the volume you want in the time given? If balloon 2 only needs to get blown up half the amount, if blowing for the same length of time, you would blow much softer (less pressure).

Key Takeaway

As volume increases, if all other variables are the same, the pressure increases as well (when inflating the same set of lungs). As volume decreases, if all other variables are the same, the pressure decreases as well.

The correlation between volume and pressure is a very important principle to understand when dealing with ventilation, as it is critical to always monitor the opposite parameter (either volume or pressure) being experienced by the lungs when you do not set them. This is because the opposite variable (pressure or volume) will be affected by the volume or pressure that you set. To explain, when in a Volume Control mode, you do not set pressure directly. Instead, you set volume and the *pressure changes* as a direct outcome based on the *volume* that is set by the medical provider. In pressure control, you do not set the volume directly, but the *volume changes* as a direct outcome based on what *pressure* is set by the medical provider. Remember talking about how lungs can be damaged by high pressures or volumes? Monitoring the opposite parameter to make sure they do not go too high is essential to ensure safe ventilating practices.

Apply Your Learning

See if you can answer these two questions based on what you learning:

1. If you wanted to inflate the lungs in $1\ \text{second}$, the first time to a volume of $300\ \text{mL}$ and the second time to $500\ \text{mL}$, which time would require a higher pressure?
2. If you were inflating the lungs over $0.8\ \text{seconds}$ and were using a pressure of $15\ \text{cmH}_2\text{O}$ but then dropped the pressure to $10\ \text{cmH}_2\text{O}$ using the same inflating time, what would happen to the volume in the lungs?

3.2 CHOOSING INITIAL SETTINGS FOR A CONTROL MODE

When setting the ventilator, a "one-size-fits-all" mentality should not be used. We have talked a lot about mechanical ventilation as a trauma. Every patient is different and must be approached based on their individual needs and the pathophysiology that they are dealing with. A clinician must consider their patient and the reason for intubation when they are setting up their initial settings. Let's walk through a systematic approach to choosing your control mode settings based on your patient and their presenting illness.

PEEP and FiO_2

We have discussed in depth the importance of maintaining an SPO_2 of more than 92% but less than 100%, so we do not over-oxygenate and cause the release of oxygen free radicals (see Chapter 2). We have also discussed the relationship between PEEP and FiO_2 and how they both contribute to oxygenation (see Chapter 2). FiO_2 can be increased or decreased to deliver a higher concentration into the lungs with every breath. The higher the oxygen level being delivered, the more oxygen will be present to diffuse across the alveolar-capillary membrane into the blood. PEEP contributes by **recruiting** (opening) collapsed alveoli—allowing more lung surface to exchange oxygen with every breath and increasing the oxygen getting into the blood with every breath, as well as increasing the driving pressure to push the oxygen across the alveolar-capillary membrane. Both FiO_2 and PEEP can directly increase the amount of oxygen that gets into the blood to circulate to the vital organs. If any of this is unclear, review Chapter 2, and then come back and read this section again.

So how do you approach setting FiO_2 and PEEP? The easiest way to approach FiO_2 is to start at 0.5 (50%) or 1.00 (100%). If the patient was requiring high oxygen before intubation, start at 1.00. If the patient was not requiring a lot or any oxygen prior to intubation, then start at 0.50. But you don't stop here! Within minutes of starting ventilation, **titrate** (increase or decrease) the FiO_2 by 10% based on the SpO_2 as you learned in Chapter 2. Within 5 minutes, you should be able to settle on an FiO_2 that correlates to an $SpO_2 > 92\%$ (and $< 100\%$). Leave the FiO_2 at this level until you get arterial blood gas (ABG) results and can make further changes from there.

Key Takeaway

FiO_2 is the only setting that you will change frequently when you first set up the ventilator. It is common that a clinician could start at 1.00 and over the span of 5 min, wean the FiO_2 to 0.4 to target their SpO_2. For all other settings, in most cases, you will set them and then wait until you asses arterial blood gases (ABGs) after waiting a minimum of 30 minutes.

Initial PEEP settings should be anywhere from 5 - 10 cmH_2O. Remember, the minimum PEEP is 5 cmH_2O. It is always better to start low and go up after you take an ABG and the patient has had some time on the ventilator. People with healthy lungs should be started at a PEEP of 5 - 8 cmH_2O. Higher PEEP should be considered for, on an individual basis, as initial settings

when you have patients with known atelectasis, pulmonary edema or evidence of thickening of the alveolar-capillary membrane in their diagnosis.

Remember, increasing PEEP is not without its dangers. If PEEP is increased too much, it can decrease blood return to the heart and also decrease lung compliance. Ensure the patient's pathophysiology would benefit from PEEP prior to initiating at 8 or 10. You can always increase the PEEP after you take a blood gas. It is better to allow the patient to settle on the ventilator with lower PEEPs and gradually increase later, if you are not sure whether they would benefit from higher PEEPs.

Refer to this summary table for FiO_2 and PEEP initial settings:

Table 3.2.1: FiO_2 and PEEP Initial Settings

Setting	Patient Status	Initial Settings
FiO_2	Hypoxic prior to intubation	$1.0\ (100\%)$
	No/little need for supplemental oxygen	$0.5\ (50\%)$
PEEP	Most patients	$5 - 8\ \mathrm{cmH_2O}$
	Known atelectasis or thickening of their alveolar-capillary membrane	$>8\ \mathrm{cmH_O}$

Respiratory Rate

One of the cornerstones of both control modes—both volume and pressure—is that the clinician sets the minimum respiratory rate the patient must breathe every minute. Remember, the patient can trigger additional breaths above that set rate, but all breaths will be delivered the exact same based on what the clinician sets in the other settings.

When choosing a respiratory rate for adult patients, you always want to be within the normal physiologic respiratory rates. An adult person breathing normally at rest usually breathes $12 - 20\ \mathrm{bpm}$ when there are no issues with their lungs. When setting the respiratory rate on the ventilator, initial respiratory rates should be chosen within that range.

So how do you choose what number to actually set? The best way to do this is to look at how your patient was breathing prior to intubation and think about what physiologic process was going on. Do they have an issue with their lungs? Why did we intubate them?

If the patient was breathing normally and was only intubated for airway protection, but their respiratory rate was in the lower end of normal, this patient can safely be initiated with a lower RR (still within those normal limits).

If the patient has compromised lungs or was breathing rapidly prior to intubation and is being intubated due to oxygenation or ventilation failure, we know the chemoreceptors in the brain were stimulating them to breathe rapidly, most likely because of elevated CO_2 levels or low oxygen. They will be tachypneic, breathing at a higher RR than normal ($> 25\ \mathrm{bpm}$ usually). The patient may also be showing signs of increased work of breathing with accessory muscle use. When you note signs of tachypnea in a patient prior to intubation, they are most likely requiring the higher RR to fix an abnormality in their CO_2 or O_2 levels. Even without an ABG to confirm this diagnosis, initial settings can still be set based on this observation. We, as clinicians, need to mimic a patient's physiologic breathing.

Now, the aim of mimicking physiologic breathing does *not* mean that you should always copy a patient's RR. Some of these patients are breathing faster than $30\ \mathrm{bpm}$. With positive pressure ventilation, that is difficult to do without causing extra damage to the lungs because, remember, we are pushing the air into the lungs, and the patient is not spontaneously pulling the air in which is less traumatic to their alveoli (see Chapter 1). Therefore, do not copy a too-high rate of breathing, which would cause trauma. Instead, choose a RR on the high side of normal. So, if normal is $12 - 20\ \mathrm{bpm}$, a clinician should start at $18 - 20\ \mathrm{bpm}$ for this patient's RR.

With positive pressure ventilation, an RR of higher than 24 bpm can start causing patient asynchrony and potentially contribute to gas trapping and damage to the lungs. It takes a trained eye to look at ventilator waveforms and patient respiratory efforts to ensure this outcome is not happening. Clinicians who are not as experienced with ventilation should try to stay below 24 bpm. A physician and/or RRT should be consulted to ensure a higher RR is appropriate.

Remember, these are your initial settings only. You would start with this RR and then do an ABG to assess how well the CO_2 and O_2 levels are after 30-60 minutes on the ventilator and make changes accordingly (we will discuss this further in Chapters 8 and 9).

Table 3.2.2: Initial Settings – RR

Setting	Patient Status	Initial Settings
RR	Normal lungs/intubated for airway protection only or slow RR prior to intubation.	14 bpm
	Compromised lungs/intubated due to oxygenation or ventilation issues or tachypnea prior to intubation.	18 - 20 bpm

Key Takeaway

For both Pressure and Volume control modes, the clinician needs to set FiO_2, PEEP and RR. Additional settings will differ based on whether you have chosen Volume or Pressure Control.

3.3 IDEAL BODY WEIGHT

What is **Ideal Body Weight (IBW)**? Essentially, every body has a set weight that reflects the size of their vital organs, based on their sex and height. IBW is not about a number on a scale or body morphology; it is a unit of measurement that is used to describe the organs in the body. Do not get caught up in the "weight" term—we use IBW to ventilate properly based on the size of the patient's lungs.

Historically, clinicians used to ventilate with large volumes $(>600\ \text{mL})$ and slow $RR\ (10\text{ - }12\ \text{bpm})$. This style of ventilation resulted in significant number of patients developing lung damage after mechanical ventilation for a few days. This damage was referred to as **Ventilator Induced Lung Injury (VILI)**, and it sometimes resulted in an inflammatory process often referred to as **Acute Respiratory Distress Syndrome (ARDS)**.

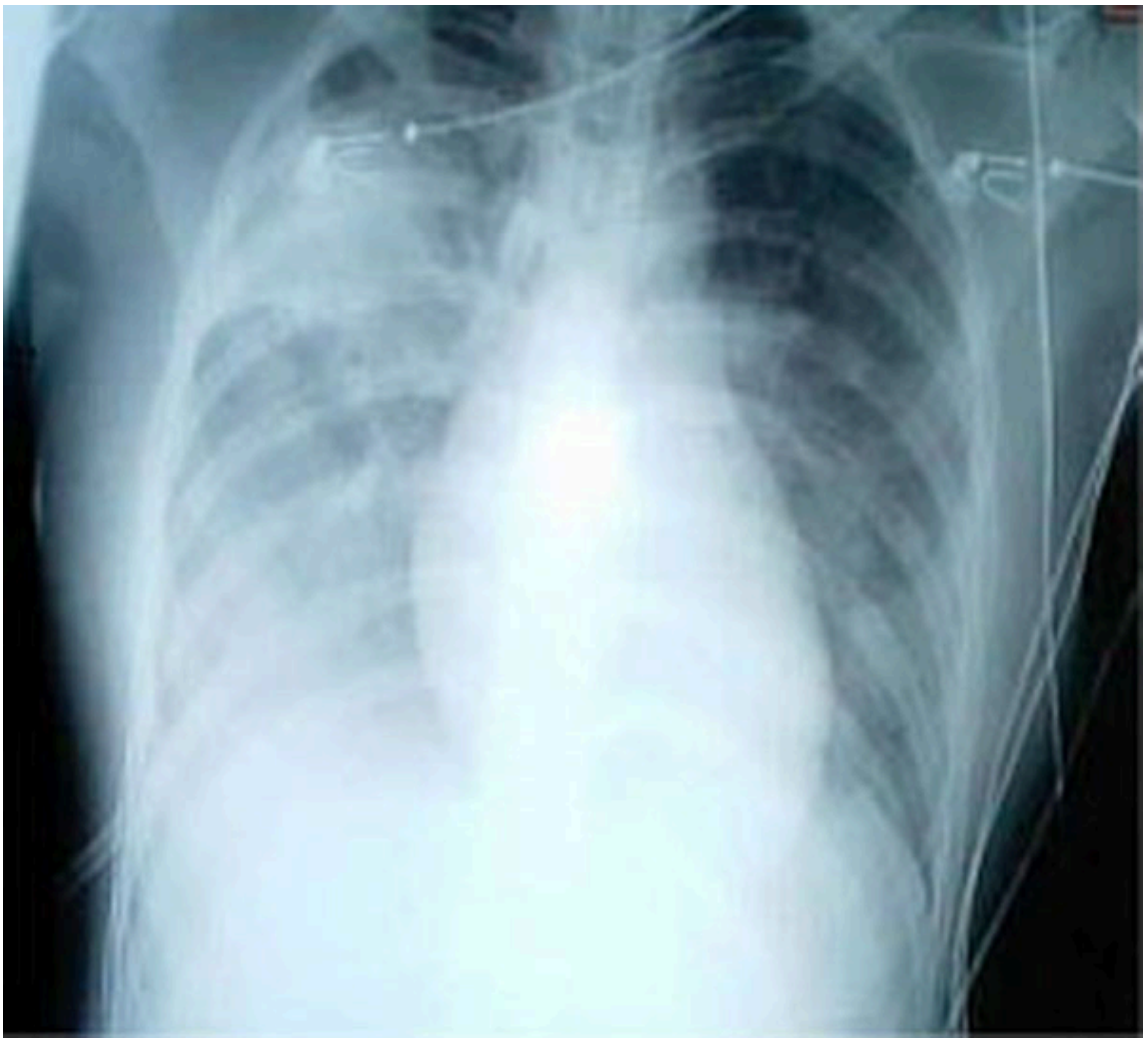


Figure 3.3.1: This x-ray shows bilateral infiltrates on both lungs. A ventilator-induced lung injury could appear similar to this. Photo by CDC, Public Domain

ARDS is a complex pathology that is recognized as diffuse bilaterally inflammation and damage to the lungs. It is usually identified on a chest x-ray after other pathophysiologies are ruled out. It can be caused by other processes in addition to VILI. These patients can become very sick and difficult to ventilate. If you would like some basic knowledge regarding ARDS, refer to these two links for a brief overview (the first link is more basic, and the second link provides more advanced information):

- ARDS (Mayo Clinic)
- Acute Respiratory Distress Syndrome: New Definition, Current and Future Therapeutic Options

ARDS ventilation will be discussed in detail in a later chapter.

When it was identified that mechanical ventilation was seeming to cause bilateral damage to the lungs, multiple studies were carried out to determine if the settings on the ventilator were contributing to this. The conclusions were very clear that using large tidal volumes directly related to increased incidence of VILI by causing **barotrauma** (too much pressure in the alveoli) and **volutrauma** (too much volume in the alveoli).

Object Lesson

The terms *barotrauma* and *volutrauma* can be used interchangeably and they mean the same thing—volume equals pressure, and pressure equals volume. Remember the balloon analogy. Think of inflating a balloon to the point that the pressure and volume are too high. What happens? It pops! The same thing can happen to the alveoli.

A weight-based approach to choosing tidal volumes started to be used. The smaller the patient, the smaller the tidal volumes and the larger the patient, the larger the tidal volumes. It was still common practice to assume patients with a higher weight required a larger volume to satisfy their ventilation needs (O_2 in and CO_2 out) because they had more habitus, meaning more cells in the body creating CO_2 and needing oxygen to function. After bariatric patients were getting VILI at a higher incidence when the larger tidal volumes were used, further investigation revealed that the size of the lungs do not alter based on body weight of the patient. A person's lung size is based on skeletal structure, which means the only relevant variables are sex and height. By ventilating heavier patients with larger volumes, clinicians were overinflating the alveoli in the lungs and causing barotrauma and volutrauma, leading to VILI.

Fun fact: If you see a full body CT scan or MRI of a bariatric patient, this concept is clear. Regardless of body habitus, a bariatric patient's lungs are not larger than another patient that is the same sex and height. The size of the lungs does not change based on the body weight of the patient.

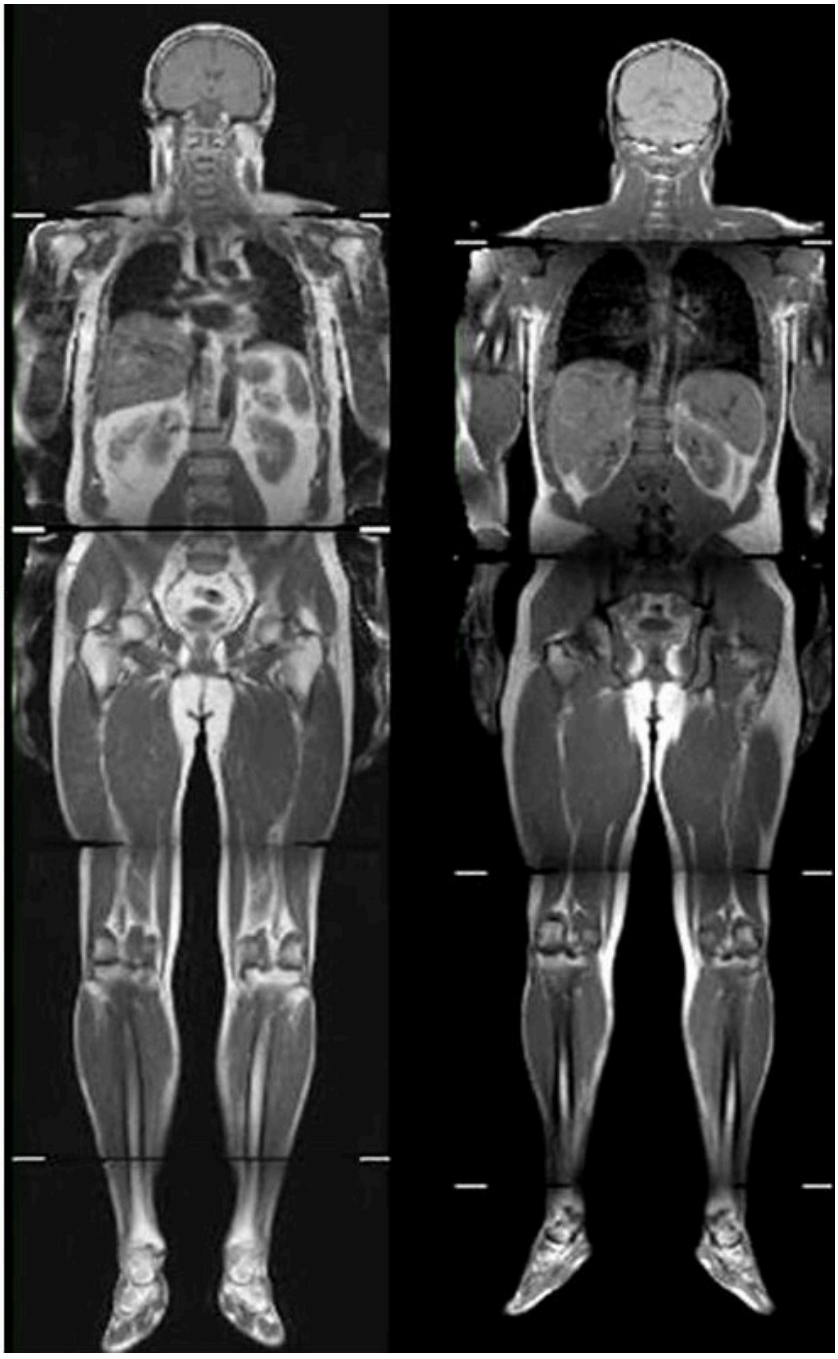


Figure 3.3.2: These two x-rays show two males of comparative age, sex and body fat, but different heights. Notice that the lung size is smaller in the shorter person on the right (there is a bit of lung collapse here as well). Photo by ImagingFat, CC BY-SA 3.0

To fix this problem once and for all, ventilation is now completely based on Ideal Body Weight (IBW). IBW is calculated based on a patient's sex and height to ensure the weight used to estimate the tidal volumes is correct based on what the patient should be getting. When setting up a ventilator for an adult patient, the only calculation you will have to do is determine their ideal body weight and then use that weight to choose a safe tidal volume for your patient.

Then, IBW depends on the sex of the patient. Here are the formulas for males and females:

Table 3.3.1: IBW Formulas

Sex	IBW Formula (Adults Only)	
Male	$\text{IBW (Kg)} = 106 + 6\ (\text{Height (in.)} - 60)$	
Female	$\text{IBW (Kg)} = 105 + 5\ (\text{Height (in.)} - 60)$	$* \left(\frac{\text{lbs}}{2.2} = \text{Kg} \right)$

Important note: For an adult patient, the minimum ideal body weight (Kg) you will use is the constant at the beginning of the equation. If you end up with a zero or negative number in the bracket, do not continue with this formula: just use $50\ \text{Kg}$ for males or $45.5\ \text{Kg}$ for females—which are the minimum sizes for adult lungs.

For a more detailed demonstration on how to calculate ideal body weight, please watch this video, which uses pounds.

One or more interactive elements has been excluded from this version of the text. You can view them online here: https://ecampusontario.pressbooks.pub/mcvresource/?p=122#oembed-1

Video: "Respiratory Therapy – Calculating IBW, Initial Vt, Initial Vent Settings" By Respiratory Coach [19:16] *Transcript Available*

Examples

Let's do a couple of calculations together!

Patient A | A male who is 6'2" (74 inches tall)

$$\text{IBW (lbs)} = 106 + 6(74 - 60)$$
$$\text{IBW (lbs)} = 106 + 6(14)$$
$$\text{IBW (lbs)} = 106 + 84$$
$$\text{IBW (lbs)} = 190 \text{ lbs}$$
$$\text{IBW (Kg)} = \frac{190 \text{ lbs}}{2.2 \text{ Kg/lb}} = 86 \text{ Kg}$$

Patient B | A female who is 5'3"

$$\text{IBW (lbs)} = 105 + 5(63 - 60)$$
$$\text{IBW (lbs)} = 105 + 5(3)$$
$$\text{IBW (lbs)} = 105 + 15$$
$$\text{IBW (lbs)} = 120 \text{ lbs}$$
$$\text{IBW (Kg)} = \frac{120 \text{ lbs}}{2.2 \text{ Kg/lb}} = 55 \text{ Kg}$$

Even with the determination of using the ideal body weight instead of actual body weight, historically, tidal volumes of 10 mL for every kilogram of IBW were often used. This approach still resulted in volutrauma and VILI. In the early 2000s, many studies were done to find the best tidal volume range based on ideal body weight to decrease the risk of VILI and ARDS. Researchers also studied patients diagnosed with ARDS to see if the amount of tidal volume impacted morbidity and mortality. The results were conclusive that using tidal volumes of 6 - 8 mL/Kg of ideal body weight directly improved morbidity and mortality. This discovery revolutionized tidal volume strategies and has become the gold standard of adult tidal volume ranges to decrease the risk of VILI from volutrauma/barotrauma. Today, 8 mL/Kg is the *hard limit* for tidal volumes to be set by clinicians in control modes. Once the IBW is calculated, it is multiplied by 6 and then 8 to get the range of safe tidal volumes for a patient on a mechanical ventilator.

Calculating Tidal Volume Range

Let's review the steps to determine a safe tidal volume for your patients.

1. Determine your patient's height and sex.
2. Use appropriate IBW formula (male versus female) to calculate your IBW.
3. Multiply your 6, 7 *and* 8 mL/Kg and write these in your chart, so you have a handy reference point for the tidal volumes you can use now and in the future.

Examples

We have already learned how to calculate IBW (steps 1 and 2), so let's try step 3 using the IBWs that were calculated earlier in this chapter. Remember, the safe range for tidal volumes is 6 - $8\ \mathrm{mL/Kg}$.

Patient A | IBW 82.2 Kg

$$\begin{aligned}\text{Tidal volume of } 6\ \mathrm{mL/Kg} &= 86\ \mathrm{Kg} \times 6\ \mathrm{mL/Kg} \\ &= 516\ \mathrm{mL} \\ \text{Tidal volume of } 7\ \mathrm{mL/Kg} &= 86\ \mathrm{Kg} \times 7\ \mathrm{mL/Kg} \\ &= 602\ \mathrm{mL} \\ \text{Tidal volume of } 8\ \mathrm{mL/Kg} &= 86\ \mathrm{Kg} \times 8\ \mathrm{mL/Kg} \\ &= 688\ \mathrm{mL}\end{aligned}$$

Therefore, the safe range of ventilation is 516 - $688\ \mathrm{mL}$ $(6$ - $8\ \mathrm{mL/Kg})$ and the respective tidal volumes for $6, 7$ and $8\ \mathrm{mL/Kg}$ are $516, 602$ and $688\ \mathrm{mL}$ respectively.

Patient B | IBW 55 Kg

$$\begin{aligned}\text{Tidal volume of } 6\ \mathrm{mL/Kg} &= 55\ \mathrm{Kg} \times 6\ \mathrm{mL/Kg} \\ &= 330\ \mathrm{mL} \\ \text{Tidal volume of } 7\ \mathrm{mL/Kg} &= 55\ \mathrm{Kg} \times 7\ \mathrm{mL/Kg} \\ &= 385\ \mathrm{mL} \\ \text{Tidal volume of } 8\ \mathrm{mL/Kg} &= 55\ \mathrm{Kg} \times 8\ \mathrm{mL/Kg} \\ &= 440\ \mathrm{mL}\end{aligned}$$

The safe range of ventilation for this patient is 330 - $440\ \mathrm{mL}$ and the respective tidal volumes for $6, 7$ and $8\ \mathrm{mL/Kg}$ are $330, 385$ and $440\ \mathrm{mL}$ respectively.

Key Takeaway

For every patient being initiated on mechanical ventilation, the clinician will determine their height, calculate their ideal body weight based on height and sex, and then multiple it by $6 - 8\ \mathrm{mL/Kg}$ to determine the safe range of tidal volumes to choose from.

When patients have very fragile and damaged lungs, some ventilation strategies will go as low as $4\ \mathrm{mL/Kg}$ to decrease the risk of barotrauma/volutrauma.

Take this information with you, as we now split to both volume control and pressure control for their individual settings.

3.4 VOLUME CONTROL VENTILATION

Volume Control

In volume control, the clinician who is setting the ventilator will dial in the set volume for each breath. The control variable, in this case is volume and this is the primary variable measured and adjusted during inspiration.

We have learned how to find a safe range for tidal volumes for our patients, but what number do you start with? It is safe to start with any number within the $6 - 8\ \mathrm{mL/Kg}$ range, for patients without ARDS.

Table 3.4.1: Initial Settings – V_T

Setting	Steps	Initial Setting
V_T	Calculate Ideal Body Weight (IBW) and multiply with $6 - 8\ \mathrm{mL/Kg}$ to get your safe range of tidal volumes	$6 - 8\ \mathrm{mL/Kg}$

In Volume Control, we also usually set the max flow of air going into the lungs. Some ventilators will ask for you to set an Inspiratory time instead (discussed below) of a flow, but the classic versions of volume control have a max inspiratory flow setting. Often the default value for an adult patient, is usually a flow of 65 liters per minute (Lpm) with a **decelerating flow pattern**. A decelerating flow pattern means that the flow peaks at initiation and then slows down as the lungs fill. This setting will work for the overwhelming majority of adult patients; however, with practice you should always try to adjust ventilation settings based on individual patient needs. The flow waveform can also take different patterns such as square (constant) and ascending. When volume is the control variable, the volume and flow waveforms are unaffected by changes in patient's lung characteristics. Because volume and flow are closely related, control of one allows the control of the other.

The pressure waveform may change depending on lung mechanics.

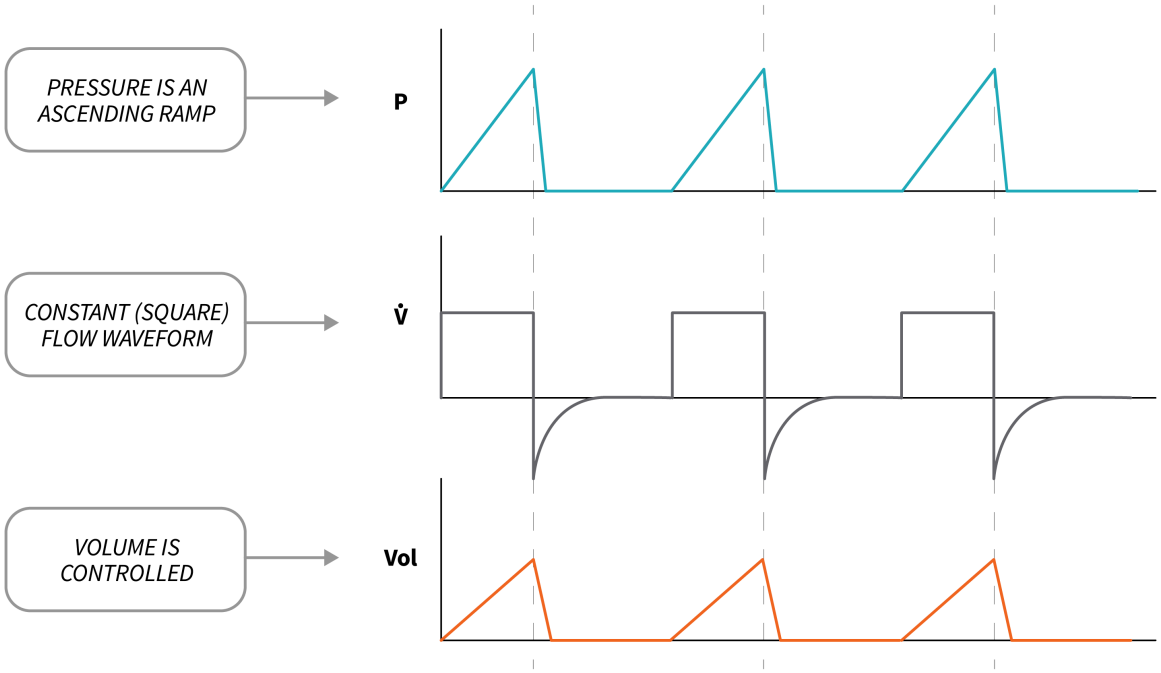


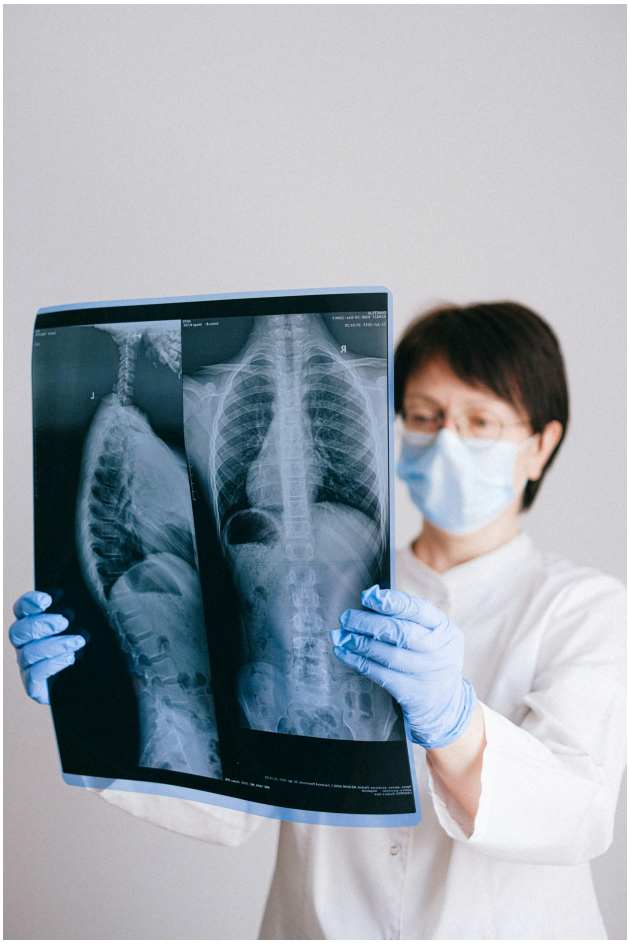
Figure 3.4.1: "Volume Control Ventilation" by Freddy Vale, CC BY-NC-SA 4.0

A word of caution: the higher the flows used, the higher the pressure you will be pushing into the lungs. Increasing the flows can cause large spikes in pressure that can cause damage to the lungs (barotrauma).

Table 3.4.2: Initial Settings – Flow

Setting	Patient Status	Initial Setting
Flow	Adult patients (except below)	$65\ \text{Lpm}$, decelerating pattern
Flow	Patients triggering additional breaths, who appear to be gasping and causing the ventilator to alarm	Titrate flow up (increase by $5\ \text{Lpm}$ at a time) while monitoring pressure.

Watch your peak pressures and keep them below $35\ \text{cmH}_2\text{O}\ (\text{PIP} < 35\ \text{cmH}_2\text{O})$.



Appropriate ventilator settings are key to avoiding damage to the lungs. Photo by Anna Shvets, CC0

3.5 PRESSURE CONTROL VENTILATION

Ventilation is considered pressure-controlled (pressure-limited), when the ventilator keeps the pressure waveform in a specific pattern. Volume and flow waveforms may change based on changes in lung mechanics. When pressure is the control variable, instead of setting the tidal volume and flow of air directly, remember that we set the pressure applied to the lungs over a specified time that causes the lungs to inflate to a certain volume. All the same rules apply as with IBW and tidal volume. You will still measure your patient's height and calculate their IBW and their tidal volume range, but instead of setting the tidal volume directly, you will set a pressure to be applied and then watch what volume you see in your lungs after about three breaths:

- Too high? Decrease the PC by $2\ cmH_2O$.
- Too low? Increase the PC by $2\ cmH_2O$ and assess.

A safe pressure control to start at is $14\ cmH_2O$. Even with a slightly higher PEEP, it will ensure your peak pressures are still well below $35\ cmH_2O$.

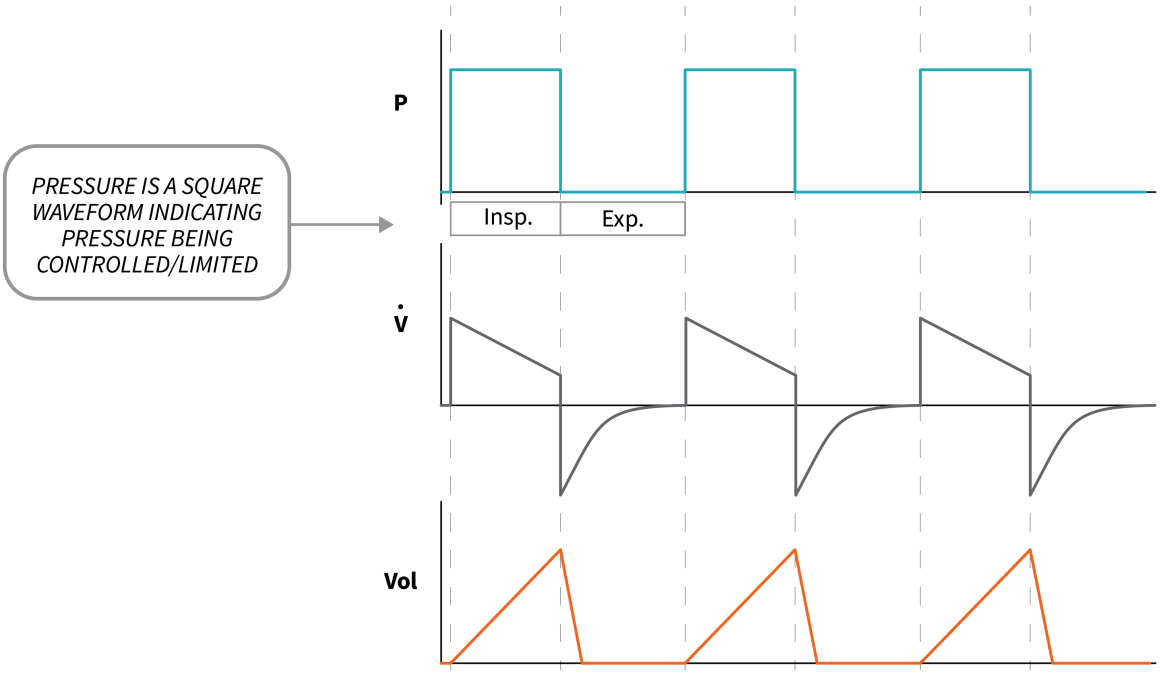


Figure 3.5.1: "Pressure Control Ventilation" by Freddy Vale, CC BY-NC-SA 4.0

Object Lesson

Remember! When the time element is the same, if you blow into a balloon harder for the same amount of time, you will blow it up bigger. A higher pressure equals a higher volume (and vice versa).

This change is very quick, and, within approximately a minute, you should be able to adjust the PC up or down to be approximately $8\ \mathrm{mL/Kg}$.

Note: Being off by approximately $20\ \mathrm{mL}$ is not an issue. You will never be exactly the same. Try to stay under the $8\ \mathrm{mL}$ instead of over. If you increased the PC by $1\ \mathrm{cmH_2O}$ and your tidal volumes go from below $8\ \mathrm{mL/Kg}$ to above, then undo that change and leave your volumes just below your maximum. Remember $8\ \mathrm{mL/Kg}$ is the upper limit, and we do not want our volumes to be above that.

The approach to setting your inspiratory time is very similar to the mentality with flow. A default Inspiratory time of 0.8 - 1.0 seconds works for most adult patients. A good rule of thumb is to use an Inspiratory time of 1.0 second. If your $\mathrm{I{:}E}$ is $1{:}1$, you can decrease your Itime by 0.1 seconds to see if you can ensure your patient has enough time to exhale. Insufficient exhalation time should not be an issue unless your patient is triggering a lot of breaths above your set respiratory rate. The only time the $\mathrm{I{:}E}$ would end up at $1{:}1$ or inverse is with high set RR (or patient triggering more breaths). Sedation could be considered in this case or a different ventilation strategy, when Itime of 0.8 - 1.0 seconds is causing issues, alarms, or an $\mathrm{I{:}E}$ that is $1{:}1$ or inverse.

Default Itimes of 0.8 - 1.0 seconds should be fine for all patients as long as the RR is less than $24\ \mathrm{bpm}$. We will learn to make adjustments, such as increasing the RR, the I-time may start to be adjusted to ensure the $\mathrm{I{:}E}$ stays greater than $1{:}1$.

Table 3.5.1: Initial Settings – Inspiratory Time

Setting	Patient Status	Initial Setting
Inspiratory Time (I-time)	Adult patient with RR set less than $24\ \mathrm{bpm}$. Consult an expert clinician if you think the Itime is not appropriate (0.8 - 1.0 seconds).	1.0 seconds

3.6 VENTILATOR ALARMS

Every ventilator has alarms that are set for the safety of the patient. We have talked a lot about the danger of applying pressure to the fragile alveoli. Using alarms on the ventilator will make sure that the pressures and volumes stay in safe ranges. After setting up your patient and initiating ventilation with your settings, go into your alarm screen and ensure your alarms are appropriate to your settings and patient.

Ventilator alarms are a useful tool for keeping you aware of your patient's status when not present at the bedside. Photo by TBIT, CC0.

The standard alarms that are set for all ventilation modes are as follows:

Table 3.6.1 Ventilator Alarms

Type of Alarm	Alert	Significance
Input Alarms	Loss of electric power	Activated when the power supply is interrupted while the ventilator is on
	Loss of pneumatic power	Activated when either the O_2 or air supply pressure is reduced below a specified point
Control Alarms	The set parameters are incompatible	Activated when operator makes errors in set up
	Part of the self-test failed	Activated during self test prior to initiating ventilation: Ex. Leak in the circuit, or failed flow sensor calibration
	There is something wrong with the ventilator control circuitry	Activated when ventilator microprocessor fails to function (usually alarms "vent inop")
Output Alarms	Volume Alarms	High and Low Expired Tidal Volume
	Flow Alarms	High and Low Expired Minute Volume
	Time Alarms	High and Low Ventilatory Frequency
		Inappropriate Inspiratory Time
		Inappropriate Expiratory Time
	Pressure Alarms	High and Low Peak Airway Pressure
		High and Low Mean Airway Pressure
		High and Low Baseline Pressure

If you would like additional information about the ventilator alarms listed above, check out Ventilator Alarms in Mechanical Ventilation.

It is important that you ensure your alarms are adequately set for all patients. Do not set your alarms too close to what your normal numbers are. These alarms are your safety net that your patient will not be exposed to volutrauma or barotrauma, but if your alarm ranges are set too tightly, it could cause any patient movement or change to trigger an alarm. Frequent alarms are disturbing for patients as well as the clinician. We also know that if things are alarming too often, it can cause alarm fatigue in healthcare workers and sometimes important alarms can be overlooked. Suggested ranges for alarm settings are below, but always defer to your health centre's guidance when it differs from these recommendations:

Table 3.6.2: Alarms and their Suggested Settings

Alarm	Suggested Setting	Rationale
High RR	30 - 35 bpm	Patient can wake up or rouse from sedation and increase their RR. This alarm is mostly used for spontaneous modes
High Pressure	35 cmH_2O (max) **or** +10 cmH_2O above your Peak Pressure	If this limit is hit, it will cut off the breath that is being delivered. This can be very uncomfortable for the patient and cause coughing and asynchrony. Monitor your pressures carefully and try to stay below 30 cmH_2O if possible. This is just the maximum limit.
Low Pressure	Set 2 cmH_2O below PEEP	Not set on every ventilator. It is good for sensing a leak or disconnect in the circuit.
High Volume	+200 mL from your target volume	Monitor your volumes breath to breath. This alarm is set wide to avoid alarm fatigue. We as clinicians want to monitor our tidal volumes tighter than this when we are present, but this alarm will come into play when clinicians are not present.
Low Volume	−200 mL from target tidal volume	Not important in control modes. Usually used for spontaneous modes
High Minute Volume	20 Lpm	Set widely on purpose. Usually used in spontaneous modes
Low Minute Volume	3 - 4 Lpm **or** −1 Lpm below the MV reading on the ventilator.	Set widely on purpose. Usually used in spontaneous modes
Apnea time	20 seconds	Standard used in adult patients.

3.7 CONCLUSION

At the end of this chapter, you should feel comfortable choosing initial settings for control modes of ventilation for adult patients. These initial settings will be reevaluated with an arterial blood gas to assess the adequacy of their ventilation for their CO_2 and O_2 levels. Changes are usually made once ABGs are obtained. We will discuss ABGs and making changes in later Chapters, but remember, you need to start somewhere! Clinicians often choose initial settings by employing educated guesses regarding their patient's ventilatory needs based on how they presented prior to intubation, and what is expected from each patient physiologically.

In summary, these are the initial settings you have learned in this chapter. Keep in mind these values are general guidelines and can be adjusted to more appropriate levels for individual patients:

Table 3.7.1: Summary of Ventilator Initial Settings

Initial Setting	Volume Control	Pressure Control	Rationale
FiO_2	1.0 or 0.5	1.0 or 0.5	Based on oxygenation status and then wean to $SpO_2 > 92\%$
PEEP	5 - 8 cmH_2O	5 - 8 cmH_2O	Based on lung physiology and whether a patient would benefit from recruitment/extra push for oxygen
RR	14 - 20 bpm	14 - 20 bpm	Choose a number based on whether the patient was breathing slower or faster before
Tidal Volume	8 mL/Kg	N/A	Use your patient's height and weight to calculate their IBW and find the safe range $(6$ - 8 mL/Kg$)$
Pressure Control	N/A	15 cmH_2O then titrate up or down to get a V_T of 8 ml/Kg	Use your patient's height and weight to calculate their IBW and find the safe range $(6$ - 8 mL/Kg$)$
Inspiratory Flow	65 Lpm	N/A	Can increase slightly if the patient is pulling faster than the ventilator (up to 80 Lpm)
Inspiratory Time	N/A	1.0 second	Can shorten slightly if the patient is trying to exhale (down to 0.8 seconds)

3.8 SELF-CHECK

Application Exercise

Chapter 3 Quiz

See how well you recall the concepts covered in this chapter by completing the following 6-question quiz.

Don't forget to check your score!

An interactive H5P element has been excluded from this version of the text. You can view it online here: https://ecampusontario.pressbooks.pub/mcvresource/?p=136#h5p-6

CHAPTER 4 | NON-INVASIVE RESPIRATORY SUPPORT

Chapter Outline

4.0 CHAPTER OVERVIEW

Non-invasive respiratory support, often called non-invasive ventilation (NIV), is the application of positive pressure to the lungs through a tight fitting mask strapped to the face.

In this chapter we will cover:

- What is non-invasive ventilation?
- CPAP and BiPAP: definitions and uses
- Indications and contraindications for NIV
- Who is the ideal NIV patient?
- Initial settings and titration for NIV
- Assessing NIV efficacy

Application

As you have already learned, invasive ventilation should be used as little as possible—only when necessary and for the shortest possible time—because it is more traumatic for the lungs. A less traumatic option for supporting normal oxygen and CO_2 levels is non-invasive ventilation—usually BiPAP. In this chapter, you will learn more about when NIV is a safe option for patients, including initial settings and common adjustments. You will also learn how to assess the degree to which NIV is working for your patient. Altogether, this information will help you use, and support the use of, NIV in your health care practice.

Learning Objectives

At the end of this chapter, you will be able to:

1. Define *non-invasive ventilation,* with examples.
2. Explain why CPAP does not qualify as ventilation.
3. Identify when to use non-invasive ventilation.
4. Implement initial settings in NIV.
5. Assess how well NIV is working for a patient.

Key Terms

- Non-Invasive Ventilation (NIV)
- Positive Airway Pressure (PAP)
- Biphasic Positive Airway Pressure (BiPAP)
- Continuous Positive Airway Pressure (CPAP)
- Expiratory Positive Airway Pressure (EPAP)
- Inspiratory Positive Airway Pressure (IPAP)
- aspiration
- airway patency

Whenever these terms are first introduced in this chapter, they are bolded. However, if you need additional information about a term than what is provided here, you can research it in The Free Dictionary: Medical Dictionary.

4.1 WHAT IS NON-INVASIVE VENTILATION?

Non-invasive respiratory support or **non-invasive ventilation (NIV)** encompasses both non invasive positive pressure ventilation (NIPPV) and continuous positive airway pressure (CPAP). NIV applies positive pressure to the lungs via a mask that seals tightly with straps on the face. It allows the delivery of positive pressure ventilatory support without the use of an artificial airway such as an ETT or tracheostomy tube. NIV is used in both the acute setting as well as the chronic settings such as patients homes and long term care facilities.

You already know that invasive ventilation is applied via an endotracheal tube and allows for a completely sealed system. Conversely, delivery of **positive airway pressure (PAP)** without an endotracheal tube is considered **non-invasive ventilation (NIV)**. NIV is not a fully sealed system. PAP is usually delivered through a ventilator to a circuit, but instead of an endotracheal tube, a tight-fitting mask with straps is the interface. Due to leaks and open areas in the circuit, it is not possible to fully dictate ventilation with a mask interface. This means that whatever the output pressure given will not necessarily act fully on the lungs. Some of it may escape due to leaks and bleeds in the circuit or mask. There are many similarities between invasive and non-invasive ventilation, and many of the same rules apply. It is important to recognize the key differences, as they play important roles in the use and application of the therapies.

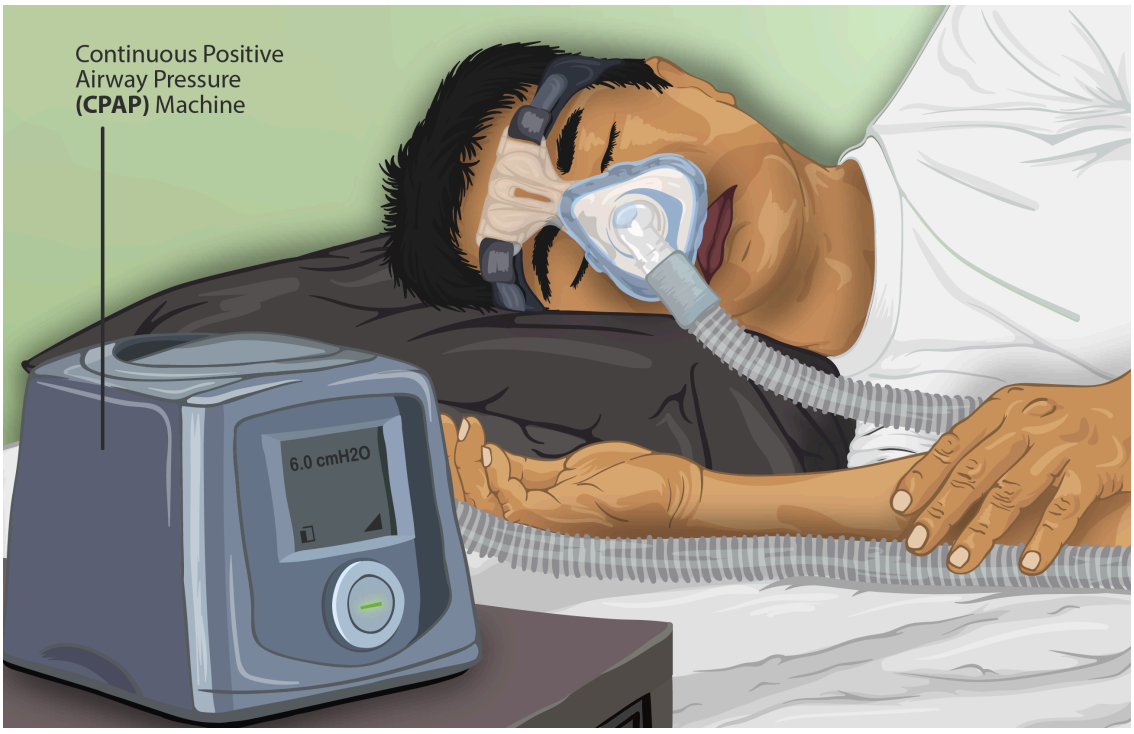


Figure 4.1.1: Photo by Dr. Nabi Darya Vali (AIIMS), CC BY-SA 4.0. Many people are familiar with CPAP machines, used commonly at home for the treatment of sleep apnea.

Return to the Ventilation Family Tree

We have already gone into detail on the invasive side of the family tree. Now, we will add a little more to the family tree so that NIV is included:

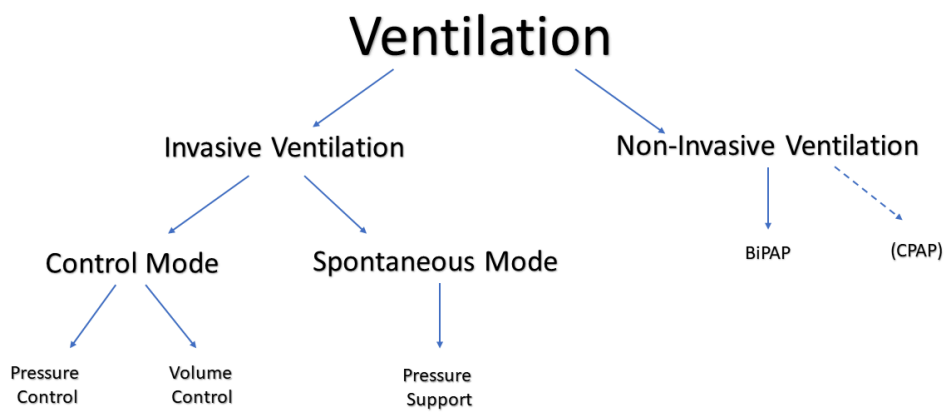


Figure 4.1.2: "Ventilator Family Tree" by Melody Bishop, CC BY-SA 4.0

Non-invasive therapy uses similar principles that follow the same rules as invasive modes. In a way, NIV is a very close relative to spontaneous ventilation, as it follows many of the same principles: it is still the application of pressure to the lungs, and it helps with the lungs inflating and deflating, assisting with CO_2 clearance. As such, NIV is still referred to as a method of ventilation. It can also help improve oxygenation.

> Non-invasive ventilation is usually referred to as Biphasic Positive Airway pressure (BiPAP) or Noninvasive Ventilation (NIV). Both terms are correct and can be used interchangeably.

4.2 INDICATIONS OF NON-INVASIVE VENTILATION

Acute Setting Indications

In the acute setting, NIV has the potential to improve survival, reduce the time a patient is mechanically ventilated, shorten hospitalization, and decrease the rate of ventilator associated pneumonia. While there are a number of different disease processes that may benefit from NIV, careful patient selection is important. This section will cover the indications for NIV in the acute care setting. It is important to recognize that each patients pathophysiology needs to be carefully considered as well as the severity of their disease process.

Indications for NIV

Table 4.2.1 Current Guidelines for Adult Noninvasive Ventilation

Patient Category	Recommendation	Certainty of Evidence
Hypercapnic COPD exacerbation	Do it*	High
Cardiogenic pulmonary edema	Do it*	Moderate
Postoperative patients	Do it	Moderate
Palliative care	Do it	Moderate
Immunocompromised	Do it	Moderate
Postextubation in patients at high risk	Do it	Low
Trauma	Do it	Moderate
Weaning patients who are hypercapnic	Do it	Moderate
Prevention of hypercapnia COPD exacerbation	Do not do it	Low
Postextubation respiratory failure	Do not do it	Low
Acute asthma exacerbation	No recommendation	
De novo respiratory failure	No recommendation	
Pandemic viral illness	No recommendation	

* Indicates a strong recommendation, all other recommendations (for or against) are conditional; further evidence may impact the certainty of effect for conditional recommendations

Data Source: Piraino, 2019; Rochwerg et al., 2017.

1. **Hypercapnic respiratory failure** due to a COPD exacerbation is one of the primary indications for NIV. There is a strong recommendation with a high level of supporting evidence. Many patients with COPD are hypercapnic at baseline and with an exacerbation develop an acute on chronic picture with a respiratory acidosis. These patients present with rapid shallow breathing, respiratory muscle and diaphragm compromise, and hyperinflation and air trapping leading to intrinsic PEEP and inadequate alveolar ventilation and respiratory acidosis. Hypercapnia in these patient can lead to CO_2 narcosis causing a decreased level of consciousness. NIV trial should still be cautiously considered in these patients with a decreased LOC for a short duration of time while closely monitored for improvement. Non invasive ventilation with low-level extrinsic PEEP

(EPAP) may help decrease intrinsic-PEEP in some patients, by splinting airways open, leading to the easier release of air from the alveoli.

2. **Acute cardiogenic pulmonary edema** patients greatly benefit from NIV over conventional mask oxygen therapy and it has been shown to reduce the rate of intubation. NIV functions as a bridging therapy treating hypoxemia and relieving respiratory distress until medical therapies such as diuretics and vasodilators can take effect. NIV provides alveolar recruitment to restore functional residual capacity reducing the intrapulmonary shunt and improving oxygenation and lung mechanics. Furthermore NIV reduces cardiac preload and afterload which can increase cardiac output and reduce congestion on the heart. CPAP is often the first line therapy while NIPPV is often used for those patient with hypercapnia or to offset work of breathing.
3. **Postoperative respiratory failure** patients who have undergone major surgery such as abdominal or thoracic surgery may benefit from NIV and reduce the need for intubation. These patient often present with atelectasis that can be reversed with positive pressure.
4. **Immunocompromised patients** in respiratory failure may benefit from NIV as a strategy to reduce intubation.

NIV application is used for three distinct reasons, which will be discussed in turn:

1. Maintaining upper airway patency
2. Lung recruitment/oxygenation
3. Correcting hypoventilation/hypercapnia

Maintaining upper airway patency

Patency refers to the condition of being open, expanded, or unobstructed. Patients with sleep-disordered breathing, like obstructive sleep apnea (OSA) do not have consistent patency of their airways, and therefore are ideal candidates for PAP therapy. During REM sleep, the upper airway can narrow or collapse fully. No air is able to pass into or out of the lungs. Normally, the brain recognizes the lack of air-exchange and forces the body out of deep REM sleep to a lighter sleep where muscle tone is restored. If OSA is not actively treated, it results in poor sleeping patterns as well as constant drops in oxygen in the blood stream. Reduced oxygen in the blood can cause increased stress on the heart and organs and can contribute to many health issues.

PAP is the main treatment method for sleep disorders that involve airway narrowing or collapse. CPAP therapy uses the pressure of air to splint the airway open so that the collapse never occurs. The pressures are usually titrated based on what the individual requires to maintain their airway.

Lung recruitment/oxygenation

Positive airway pressure (PAP) increases alveolar pressure. This treatment avoids alveoli collapse and can also recruit collapsed alveoli, improving ventilation and oxygenation in a few ways:

1. **Improving ventilation and prefusion matching:** Pathologies that cause the alveoli to collapse or fill with fluid cause poor oxygenation due to the presence of a mismatch between the areas of the lung that are being ventilated compared to the areas that are being perfused. If some alveoli are collapsed or filled with fluid, they will not be inflating and deflating with every breath; in other words, they are not participating in ventilation. Blood flow around these alveoli would still occur—they are still being actively perfused by the blood vessels. This means that there is a percentage of the alveolar-capillary membranes that cannot participate in gas exchange because the alveoli are collapsed or full of fluid. They have adequate perfusion, but are not being ventilated. PAP can reverse atelectasis and re-expand alveoli that have collapsed, allowing them to be ventilated, and

restoring gas exchange. PAP can also increase alveolar pressure and counteract extra fluid around the lungs that may have leaked into the alveoli and caused them to fill with fluid. The pressure will push the fluid from the alveoli and the interstitial space to the pulmonary circulation.

2. **Improving oxygenation with additional pressure:** We have previously talked about how PEEP—the overall constant pressure applied at the end of breaths—can help push the oxygen across the alveolar-capillary membrane. In non-invasive ventilation, this pressure is not referred to as Positive End-Expiratory Pressure (PEEP); it is referred to as Expiratory Positive Airway Pressure (EPAP). However, EPAP essentially does the same action as PEEP. Even on exhalation, a constant pressure is still applied, and it increases the pressure being felt in the alveoli. That increase in pressure helps push the oxygen across the membrane and can improve oxygenation.

> Don't get confused by the different name. The different term is just to help you differentiate between invasive and non-invasive modes: PEEP (invasive) is equivalent to EPAP (non-invasive). Remember: PEEP is the pressure that is still being applied *in between* breath delivery. This is the same thing when talking about EPAP with non-invasive ventilation.

3. **Decreasing work of breathing by restoring FRC/PEEP:** When trying to inflate a balloon, the highest pressure is required when the balloon is fully empty. Once a small amount of air is in the balloon, the pressure required to inflate it drops significantly. As discussed extensively, maintaining some end expiratory pressure in the lungs stops the alveoli from collapsing, which decreases the overall work of breathing experienced by the patient. This benefit can be extremely useful in patients that are at high-risk of ventilatory failure.

Correcting hypoventilation/hypercapnia

Correcting hypoventilation and hypercapnia is, by far, the most common use of NIV. This use of NIV does not apply to CPAP at all, as two levels of pressure are needed to augment ventilation and contribute to CO_2 clearance. By using the two levels of pressure, non-invasive ventilation can help the lungs increase how large a volume they are taking in and increase the effectiveness of each breath in, clearing CO_2. BiPAP is effective when a patient's breathing is not adequate in clearing CO_2 and the CO_2 levels start to rise. But remember, with NIV, patients need to be awake and still have an intact drive to breath.

Furthermore, the two levels of pressure contributing to larger tidal volumes decreases the muscle recruitment a patient must use to pull these tidal volumes. For patients with signs of increased work of breathing, this effect is key to increasing the effectiveness of their breathing and decreasing the chance of ventilatory failure.

Some patients are dependent on mechanical ventilation to breathe. These patients will have ventilation delivered via tracheostomy. Photo by Dan Perez, CC BY-NC 2.0

4.3 CONTINUOUS POSITIVE AIRWAY PRESSURE (CPAP)

Continuous Positive Airway Pressure **(CPAP)**—the name says it all. This mode applies one constant (or continuous) pressure that is maintained during both inspiration and expiration.

The main purpose of CPAP is to:

1. Stents airway open. This would include
2. May help offset autoPEEP
3. Recruits alveoli, increases FRC
4. Improves gas exchange
5. Improved oxygenation and decreases FiO_2

While CPAP/PEEP does not have a direct effect on ventilation and CO_2 clearance, it can indirectly help, through various mechanisms, such as:

1. Reduced atelectasis improving alveolar compliance and surface area for gas exchange increasing tidal volume and CO_2 clearance
2. Stent open upper airways in patients with obstructive sleep apnea or tracheomalacia increasing ventilation
3. Counterbalance dynamic hyperinflation (auto PEEP) in patients with COPD exacerbations increasing effective tidal volume

Key Takeaway

CPAP vs. PEEP vs. EPAP vs. IPAP. What's the difference?

Before moving forward it is important to review the terminology used in non invasive ventilation.

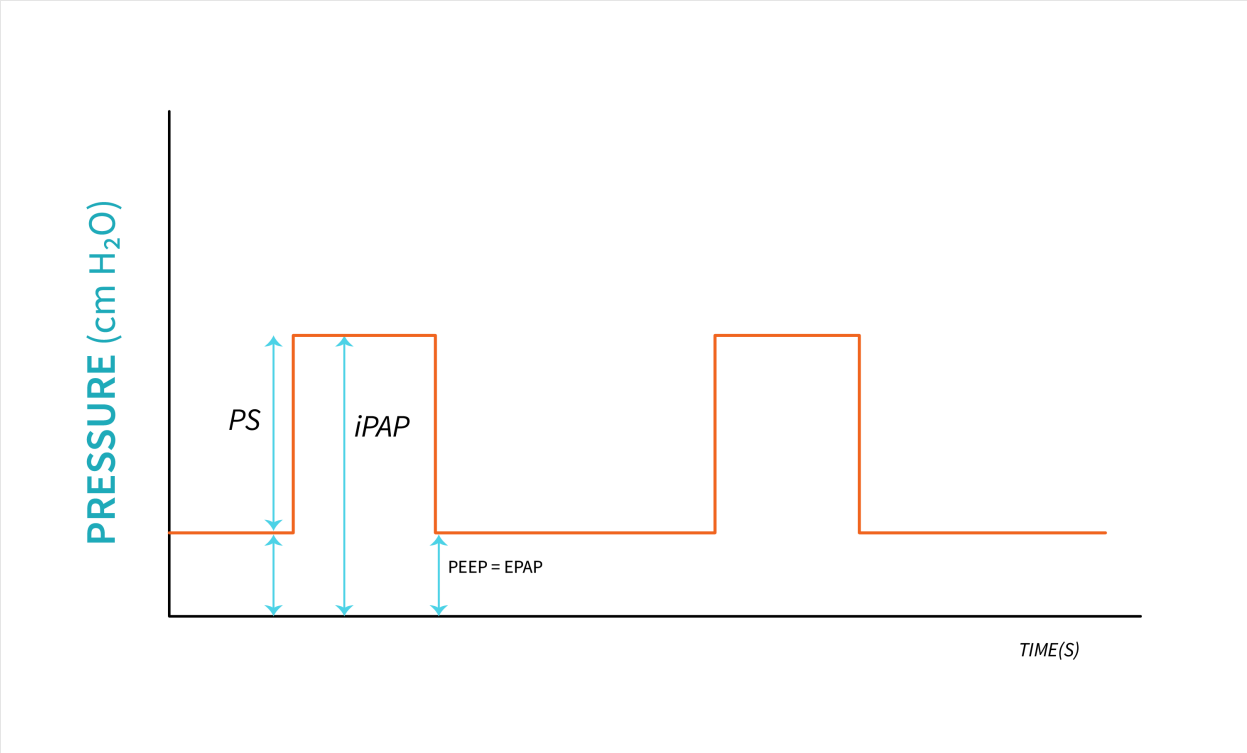


Figure 4.3.1: "PS, iPAP, PEEP, and EPAP" by Freddy Vale, CC BY-NC-SA 4.0

CPAP is the application of a continuous level of positive pressure during the **entire respiratory cycle** when the patient's drive to breathe is intact (spontaneous breathing). CPAP does not provide inspiratory assistance, and therefore, is not considered a type of mechanical ventilation.

PEEP is the pressure delivered by the ventilator during exhalation. EPAP is equivalent to PEEP, and it is the baseline pressure that every breath starts and ends at.

IPAP is the pressure delivered by the ventilator during inspiration.

The difference between IPAP and EPAP is the **Pressure support (PS)** provided by the ventilator. This gradient is equivalent with driving pressure provided by the ventilator during controlled or assisted ventilation.

When thinking of a mask interface and pressure delivered, most people will think about CPAP and CPAP machines. CPAP is usually used for home patients that have obstructive sleep apnea. Though still pressure-applied, CPAP is not considered ventilation for a one main reason: *Continuous* Positive Airway Pressure (CPAP)—the name says it all. This mode literally applies one constant (or continuous) pressure through the circuit and mask interface to be delivered to the patient. Though CPAP is discussed in this chapter as a non-invasive *therapy*, it is not a method of non-invasive *ventilation* (NIV). Ventilation at its core refers to the clearance of carbon dioxide. As we have discussed, CO_2 clearance happens during exhalation, and if you want to clear more CO_2, you need to take a bigger breath in and, most importantly, out. In order to help the lungs inflate larger, a higher pressure must be applied during inspiration. Since CPAP is one constant pressure, CPAP does not change the pressure applied to help augment tidal volumes and does not contribute to the clearance of CO_2.

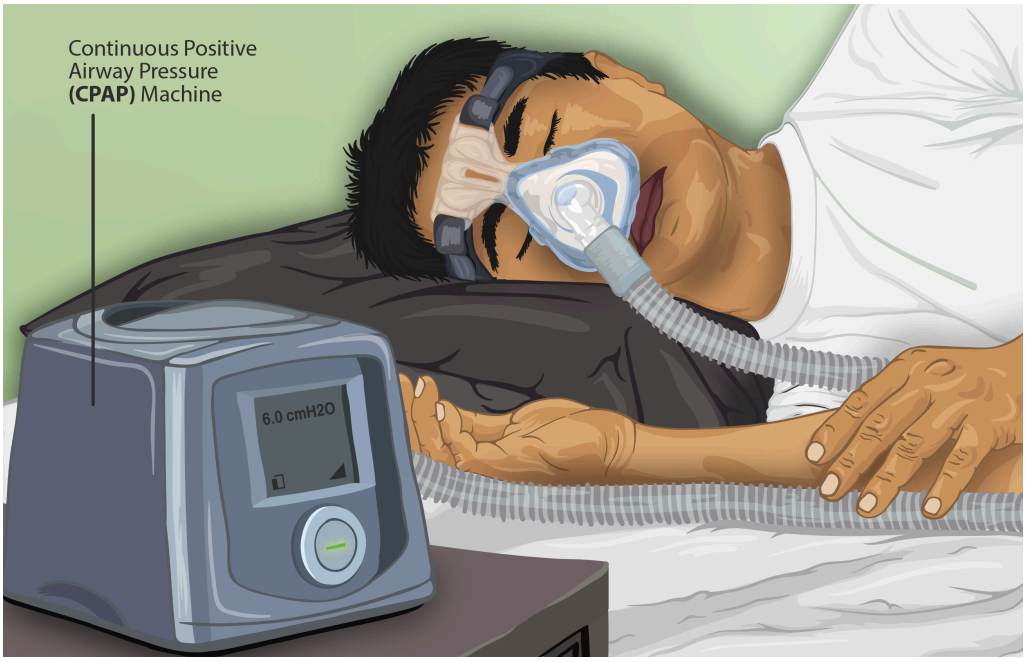


Figure 4.3.2: Photo by Dr. Nabi Darya Vali (AIIMS), CC BY-SA 4.0. CPAP machines provide constant pressure, and so they do not qualify as ventilators.

The main benefit of CPAP stems from the patient breathing spontaneously in and out against the set pressure. Exhaling against positive pressure, produces a back pressure that increases the air left in the lungs at the end of a breath. This can help open collapsed airways or alveoli and is often referred to as a **splinting effect**.

Object Lesson

To explain back pressure, imagine breathing out through a thin straw. You are pushing against a resistance. You know how you feel pressure in your lungs as you are trying to blow into this narrow straw? That increased pressure is actually happening. Breathing out against a constant pressure of air, like CPAP, would cause the same thing. That increased pressure in the lungs will hold extra air in all areas of the lungs and help blow open (recruit) any areas of collapse. This is a back pressure that causes the "splinting effect."

Photo by Monstera, CC0

This splinting effect can be useful for specific lung issues. It is beneficial in recruiting collapsed alveoli. Involving more alveoli in air exchange will improve ventilation. Another benefit of this "splinting effect" is seen with patients who have symptoms of obstructive sleep apnea. CPAP stops the soft tissue of their nasopharyngeal region from collapsing and closing off. In a way, CPAP is similar to PEEP but just called something different because we are not in an invasive ventilation mode.

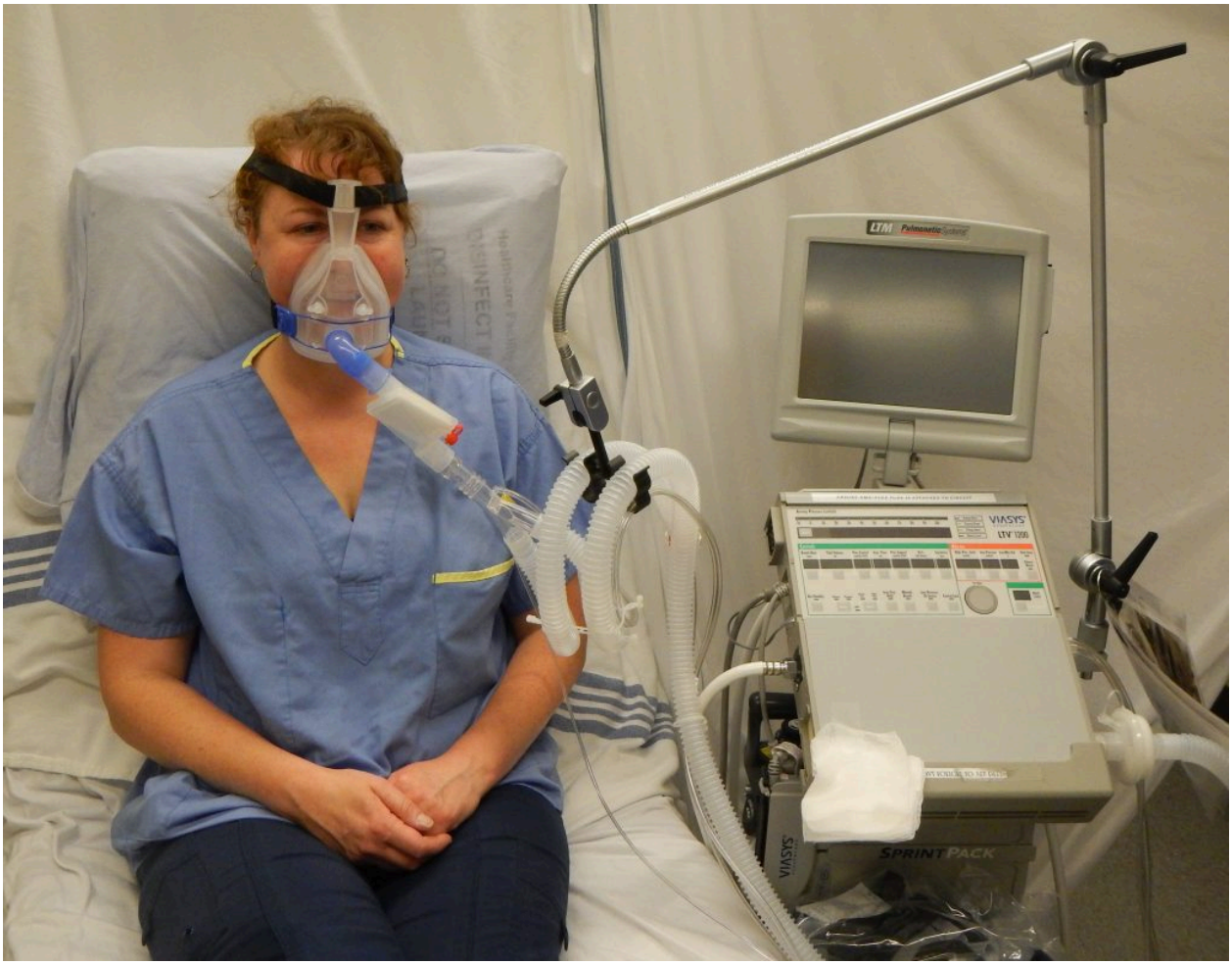
CPAP is periodically used for patients that need some help with recruiting or splinting open their airways, but in critical care and hospital settings, NIV or BiPAP is usually preferred because it allows the practitioner more control over oxygenation and ventilation. Therefore, we will focus on NIV instead of CPAP, as it is used to a much larger degree in critical care medicine.

Key Takeaway

CPAP is the application of a *constant* pressure. The pressure stays the same while the patient breathes in and out. It does not change unless the clinician increases or decreases the set pressure. CPAP may help oxygenation by methods of lung recruitment or keeping airways open but it does not aid in the clearance of CO_2 and is not considered a method of non-invasive ventilation (NIV).

4.4 BIPAP (NIV)

The term **Biphasic Positive Airway Pressure (BiPAP)** also is fairly self-explanatory. "Bi" means two, and you already know that "PAP" refers to positive airway pressure. Therefore, BiPAP is the application of two separate pressures to the patient via the circuit-and-mask interface. Just as the name suggests, it is "biphasic," or unfolding in two phases: inspiration and expiration. In BiPAP, the pressure starts at a baseline pressure (similar to CPAP) that remains throughout inspiration and expiration. Where BiPAP differs from CPAP is when the patient starts to initiate a breath: BiPAP changes to a higher pressure where CPAP stays constant.



"BiPAP using a Ventilator" by James Heilman, MD, CC BY-SA 4.0. BiPAP is the NIV method that is used in hospitals when indicated for a patient.

The most significant difference between CPAP and BiPAP centers around the supportive inspiratory pressure that BiPAP can deliver. By helping augment the patient's natural tidal volume, BiPAP allows the patient to exhale more carbon dioxide. Similar to invasive mechanical ventilation, BiPAP can aid in correcting high CO_2 from respiratory failure. Since BiPAP contributes to the clearance of CO_2, it is a method of ventilation. BiPAP can also be referred to as non-invasive ventilation or NIV.

The term CPAP is often referred to when discussing non-invasive therapies while PEEP is the similar term used for invasive mechanical ventilation.

Expiratory positive airway pressure (EPAP)

BiPAP or NIV senses that the patient is starting their inspiration and bumps up the pressure to a higher set pressure. At the end of inspiration, as the patient exhales, the pressure drops back down to the baseline lower pressure. Similar to CPAP, the patient breathes against the lower pressure, benefiting from the "splinting" effect and contributing to lung recruitment. The lower pressure is referred to as the **Expiratory Positive Airway Pressure (EPAP)**. The inspiratory phase with the higher pressure helps facilitate the delivery of a larger breath than the patient would have been able to take on their own—it helps "push" the air into the patient's lungs.

Key Takeaway

Application of pressure to the lungs is the same as applying a volume of air. Increasing the pressure at the higher level will help the lungs inflate to a larger volume and help with the clearance of CO_2.

NIV is similar to pressure support (PSV) during invasive ventilation. The higher pressure is referred to as **Inspiratory Positive Airway Pressure (IPAP)**. The higher the pressure delivered, the larger augmentation that is seen in the patient's breath. In a way, BiPAP/NIV can be seen as the non-invasive version of PSV. Many of the same rules apply. A patient must have an intact drive to breathe and must be actively involved in triggering a breath so NIV can kick up to the higher pressure. PSV helps augment the patient's normal tidal volume, but allows the patient to control how long they breathe in and the ending of inspiration. NIV is the same way.

Let's compare PSV and NIV, with the following table:

Table 4.4.1: PSV and NIV Comparison

	PSV	**NIV**
Invasive/Non-invasive	Invasive (ETT)	Non-Invasive (mask)
Needs an intact drive to breath	Yes	Yes
Patient triggered assisted breaths	Yes	Yes
Constant pressure at the end of the breath delivered	PEEP	EPAP
Pressure delivered when breath initiated	PS	IPAP
Patient controls the length of inspiration and breath cycling	Yes	Yes

Key Takeaway

BiPAP is the application of *two different levels* of pressure. There is a lower pressure that is there during expiration (EPAP) and then the pressure increases to a higher level during inspiration (IPAP). These two pressures remain constant and vary up and down based on the ventilator sensing inspiration and expiration. The IPAP and EPAP will not change unless the clinician increases or decreases the pressures set. The EPAP may help oxygenation by methods of lung recruitment or opening collapsed airways. The IPAP is able to help improve the clearance of CO_2 by augmenting each breath the patient is taking and by increasing the tidal volume, allowing more CO_2 clearance with every breath.

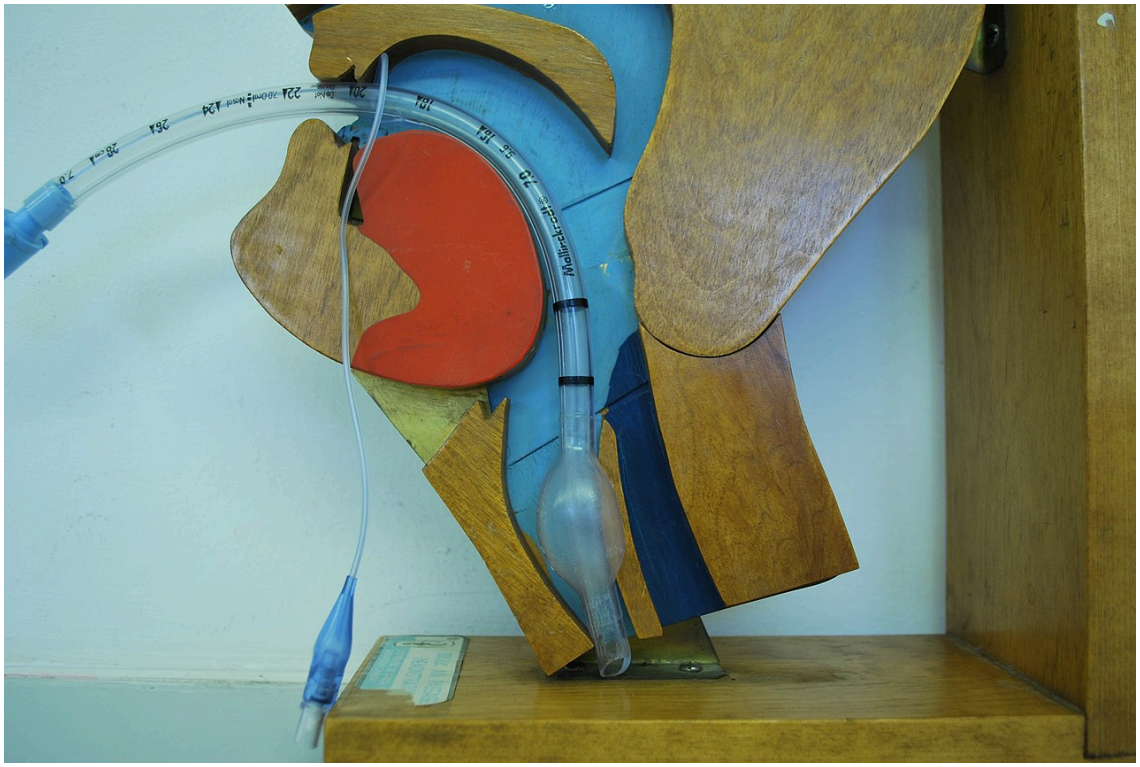
4.5 CONTRAINDICATIONS OF NON-INVASIVE VENTILATION

The main contraindications of non-invasive ventilation (NIV) stem from the interface being a tight-fitting mask that does not seal the airway. They include:

- Decreased or altered level of consciousness
- Full stomach/risk of aspiration
- Altered drive to breathe
- Trauma to the face or inability to create a mask seal

If the patient is unable to protect their own airway (by keeping their airway open, coughing or swallowing when needed), they cannot be placed on BiPAP. NIV pushes air into a patient's upper airway instead of directly into a sealed trachea, like with an endotracheal tube with a balloon to seal. When air is pushed into the upper airway, although most air will go into the lungs, some will go down the esophagus and into the stomach. Air in the stomach increases the risk of vomiting and aspiration. **Aspiration** is when vomit or stomach contents come up into the oropharynx and fall into the respiratory tract, ending up in the lungs. Patients on NIV are at high risk of aspirating vomit or other foreign objects.

NIV therapies are delivered via a tight-sealing face mask instead of an endotracheal tube that passes through the cords. An endotracheal tube (ETT) is able to create a perfect seal by way of passing through the cords, while an ETT cuff is inflated that seals off the trachea.



This image shows correct endotracheal tube placement, with the balloon cuff inflated. Photo by Dr. Lorimer, CC BY-SA 4.0

Conversely, a mask interface—though normally secured very tightly to the face, is not a perfect seal. There are always small gaps, and any movement of the face can cause leaks. Another critical concept is that the mask does not allow for complete "protection" of the airway. The trachea is not closed off and is in communication with the oropharynx, but it would be sealed off with the cuff on an endotracheal tube (ETT) if one was in place.

This open communication between the esophagus and the trachea when using NIV and a mask interface is the main concern, as the application of pressure will push vomit/stomach contents down into the lungs if the mask is kept in place when a patient vomits. Risk of aspiration is very high with non-invasive therapy. If the patient vomits while NIV is in place, aspiration is almost unavoidable. Non-invasive ventilation is not indicated with altered level of consciousness since it (a) can impact the patient's gag reflex, and (b) may impact their ability to remove the mask if they feel they need to vomit. Since aspiration is such a large concern with NIV, this therapy is not used on patients with full stomachs, and patients are usually fasted while using this therapy.

An altered drive to breathe is also an issue with NIV. BiPAP can only be used to augment spontaneous breaths that the patient is already triggering. It cannot deliver a manual breath, as it is not a sealed system. If the patient is not reliably breathing on their own, they cannot be put on NIV. Two classic examples of inappropriate drive to breathe include agonal respirations or a neurologic injury that disrupts spontaneous breathing.

One or more interactive elements has been excluded from this version of the text. You can view them online here: https://ecampusontario.pressbooks.pub/mcvresource/?p=183#oembed-1

Video: "Agonal Respiration (Not Breathing Normally)" By ProCPR [1:34] *Transcript Available*

Inability to create an effective mask seal is a major concern with non-invasive ventilation. We have already discussed about the issue with leaks and the need for a tight seal with non-invasive interfaces. When air is pushed into a patient's nose and mouth through a mask, it is mandatory that a tight seal is created. Any situation that would impact the ability of the mask to seal on the patient's face will be an issue. Any facial trauma that would render a mask seal inadequate or that can be damaged by a tight sealing mask can make a patient unsuitable. Even beards, small chins or large noses can pose difficulties in creating a tight seal. Also, bleeding in the upper airway or trauma to the upper airway that would necessitate the bypassing of the upper airway would necessitate endotracheal intubation.

Photo by Sgt. Jessi McCormick via Wikimedia Commons, Public Domain. U.S. Army Spc. Leroy Granado, a medic with the 56th Infantry Brigade Combat Team, examines a wounded Afghan soldier in Uruzgan province, Afghanistan, Feb. 20, 2013. This patient is ventilated using an ETT because his facial trauma disqualifies him for NIV.

4.6 THE IDEAL NON-INVASIVE VENTILATION PATIENT

The most important step to initiating BiPAP or CPAP is in identifying the ideal patient population, and the reason NIV is being initiated.

NIV is not equal to invasive mechanical ventilation. NIV has its own distinct ideal patient population. Similar to spontaneous modes of ventilation, all patients need to be breathing on their own. They must also be alert enough to protect their own airway and not be at risk for aspiration.

Other than these baseline requirements, the ideal non-invasive patient can be selected based on the previously mentioned "indications for NIV". Patients who would be ideal candidates either need additional pressures applied to help:

- recruit alveoli and/or improve oxygenation, or
- augment their own breathing noticed through elevated CO_2 or increased work of breathing.

To simplify it even further, they need either distending pressure (EPAP/PEEP) or they need supportive pressures (IPAP/PS).

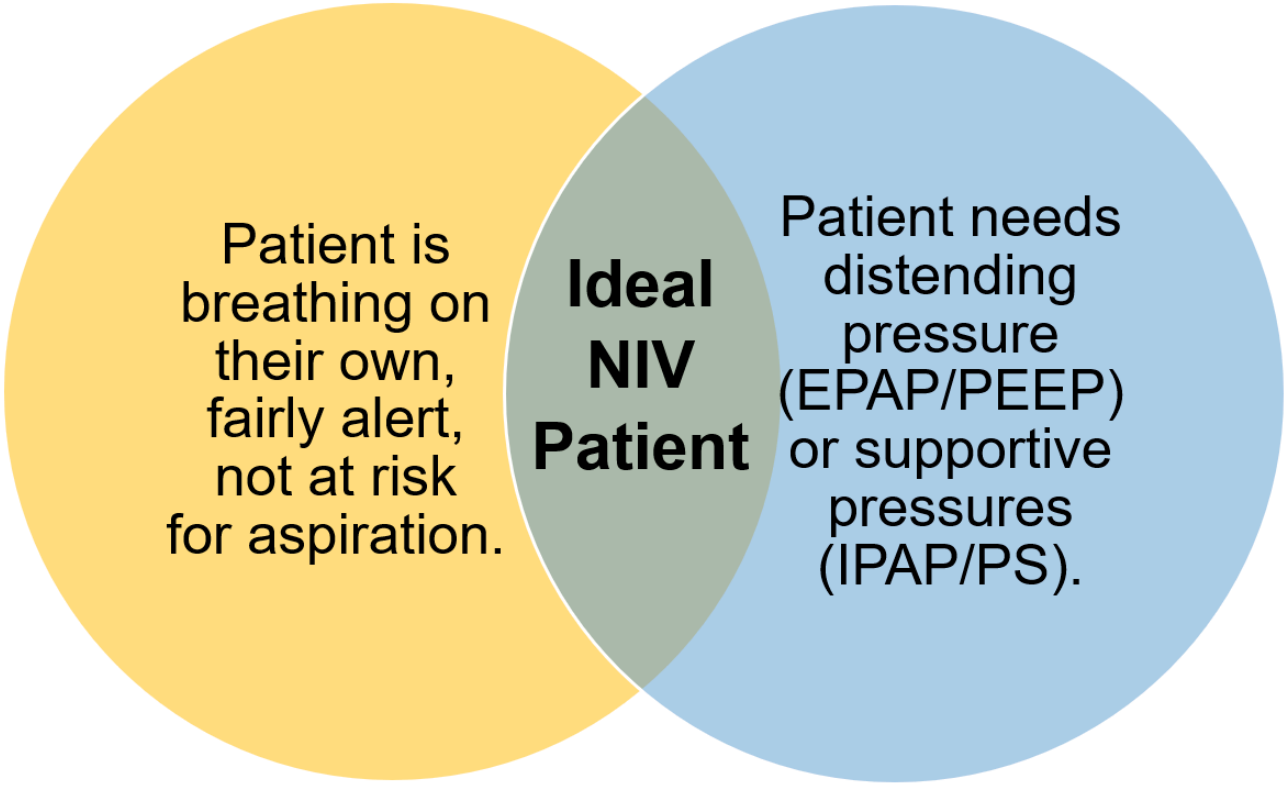


Figure 4.6.1: "Ideal NIV Patient" by Amanda Robinson, CC BY-SA 4.0

Identifying the reason that NIV is going to be used will benefit your patient in two ways. It will

- aid the practitioner in knowing whether NIV will be successful, and

- drive the settings chosen when setting a patient up on NIV.

4.7 NIV MACHINES AND INTERFACES

Non invasive ventilation can be provided through a number of different devices. There are specifically designed NIV machines that can only provide NIV and not invasive ventilation in the hospital environment. An example of these machine is the Philips Respironics V60 and V680. In the past, these machines provided superior NIV due to complex leak detection and synchronization algorithms. Nowadays most ICU ventilators and transport ventilators can provide NIV with enhancing software and algorithms as well as invasive ventilation.

Additionally there are home CPAP units that are primarily used to provide CPAP to OSA patients. There are also home devices that can be used to provide NIV (BIPAP) to patients at home.

There are a number of different NIV mask interfaces. There are those that cover just the nose such as the nasal mask or nasal pillows/prongs. These masks are primarily used for patients on home CPAP with OSA.

NIV Mask Interfaces

- Nasal Mask
- Nasal Pillows/Prongs
- Oral Mask
- Oral – Nasal
- Full Face Masks
- Total Face Masks
- Helmets

When selecting the NIV device and mask, it is essential that the correct interface with specific characteristics are chosen.

Ventilator NIV with Dual Limb Circuit

When providing NIV through a ventilator with a dual limb circuit (inspiratory and expiratory) limb. A non vented full face mask that covers both the mouth and nose is recommended. In this setup, expiration occurs through the expiratory valve in the ventilator and the mask **must not** have a vent or anti-asphyxia valve built in. A vent or leak in the mask or circuit is not needed as the patients exhaled volume will travel to the expiratory limb and out the ventilators expiratory valve. An anti-asphyxia valve is not required as the gas flows in a series from inspiratory to expiratory limb and therefore does not require the anti-asphyxia safety system built in. Additionally these patients are more likely to be in a closely monitored ICU bed.

NIV Machine with Single Limb Circuit

When providing NIV with a full face mask and a specially NIV machine such as the Philips Respironics V60 and a single limb circuit is used, it is important to select a mask that has an anti-asphyxia valve built in. An anti-asphyxia valve is a safety feature on a single limb circuit that opens if the pressure in the ventilator circuit falls below a predetermined level (usually $3\ \mathrm{cmH_2O}$). This may

occur if the machine malfunctions or the circuit becomes detached. The purpose of this anti-asphyxia valve is to allow the patient to exhale and breathe without having to breathe through the entire length of the single limb circuit that could cause CO_2 re-breathing. The anti-asphyxia valve opens to ambient and would allow the patient to exhale and also breath in room air gas. During regular NIV, the anti-asphyxia valve flap is pushed closed due to the pressure inside the circuit and it would only open to ambient if you pressure in the circuit decreased.

One or more interactive elements has been excluded from this version of the text. You can view them online here: https://ecampusontario.pressbooks.pub/mcvresource/?p=876#oembed-1

Video: "Intersurgical's FaceFit™ NIV mask with anti-asphyxiation valve" By intersurgical [0:33] *Transcript Available*

Anti-asphyxia Valve / Safety Valve

- Loss in pressure causes valve to fall during exhalation to facilitate CO_2 removal and prevent rebreathing
- Required for vented and non vented masks used with single limb circuits
- Also called an anti-asphyxiation valve
- Basic design features
- Vented
- Holes in the mask
- Non-vented
- Leak is in the circuit
- Whisper swivel
- Exhalation port
- Safety/anti-asphyxiation valve
- Not used with dual limb circuits (ventilators)

The next important feature when using a NIV machine with single limb circuit is the location of the exhalation valve or sometimes called a leak valve. When a **Vented Mask** is used, the exhalation port is built into the mask. A vented mask will have holes that allow for fixed resistance to flow during exhalation. Because the resistance is fixed, CPAP is increased by increasing flow from the ventilator through the circuit.

When a **Non-vented mask** is used, a fixed/intentional leak valve must be added to the circuit, which contains holes that allow fixed resistance to flow during expiration.

4.8 INITIATION AND TITRATION OF NIV/BIPAP SETTINGS

Initiating non-invasive ventilation is very similar to how you approach setting up PSV for a spontaneously breathing patient. There are three main settings that need to be adjusted as well as one additional "back-up" setting. First, you will set the oxygen delivery (FiO_2), a distending pressure to help recruit alveoli (EPAP), and a high pressure to augment the patient's normal breath (IPAP). The back-up setting is a basic RR (remember, this setting does not replace the patient spontaneously breathing).

Therefore, the settings to be set for NIV are:

1. FiO_2
2. EPAP pressure (similar to PEEP)
3. IPAP pressure (Δ Pressure, similar to pressure support)
4. RR

These settings will be discussed in detail below.

FiO_2

FiO_2 is mandatory to set and should be titrated based on what the patient needed before non-invasive application. If on high oxygen, start FiO_2 at 1.00 and then wean to SpO_2. If the patient does not require a lot of oxygen, starting at 0.50 and weaning within a few minutes to target $\mathrm{SpO_2} > 92\%$ is ideal.

EPAP

EPAP pressure is similar to PEEP. It is the distending pressure that helps recruit alveoli and help with oxygenation. Where it differs from invasive ventilation is using an interface of a face mask instead of intubating and sealing the lungs to a ventilator. The normal pleural pressures that exist with spontaneously breathing patients are still present (see the discussion of lung pressures in Chapter 1). This means that the distending pressures of EPAP can be lower than the minimum PEEP you must set. EPAP settings are usually started from $4 - 8\ \mathrm{cmH_2O}$.

IPAP

IPAP is the high pressure NIV will cycle up to when the patient initiates a breath. The difference between the EPAP and IPAP is the delta pressure or change in pressure. The change in pressure is the same as a pressure support or additional pressure given to help augment a person's breath. Think of the change in pressure between the EPAP and the IPAP (Δ pressure) as the push to get up

to that high level. This is identical to the Pressure Support in PSV but described a slightly different way in NIV. Think back to the waveform and mountain analogy

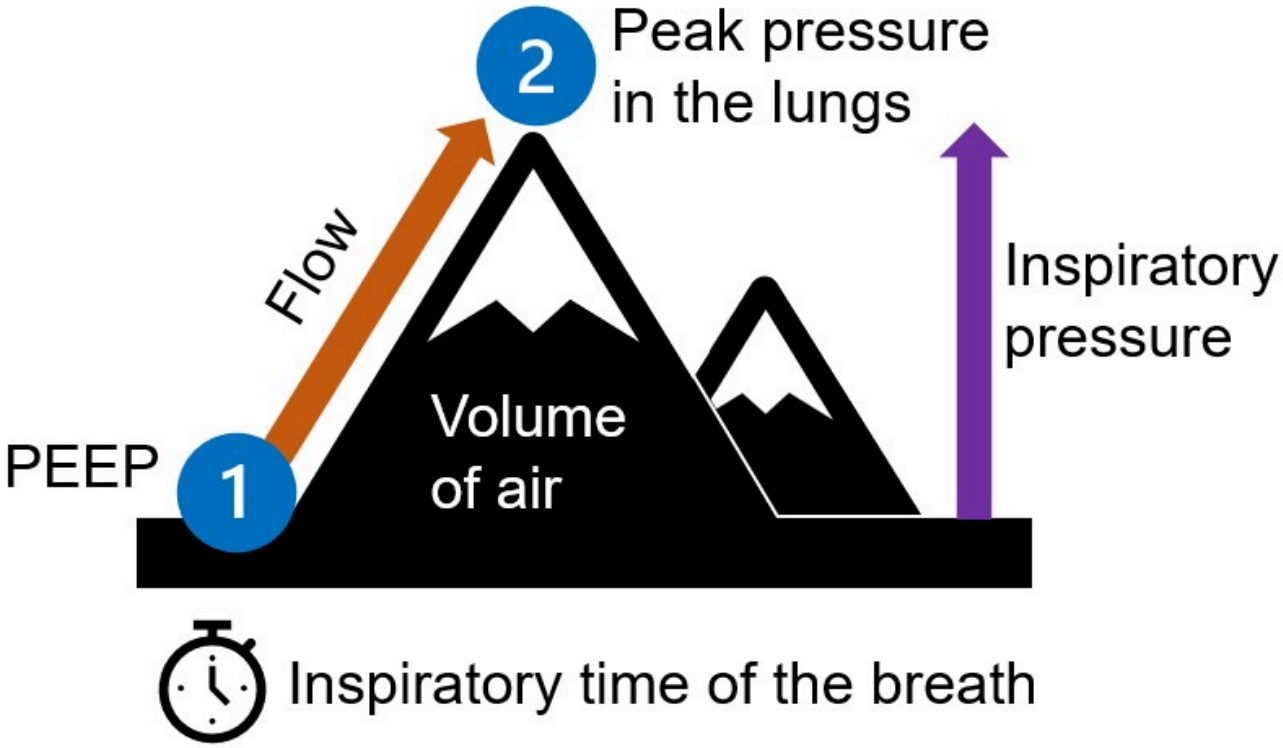


Figure 4.8.1: "Mountain Analogy Infographic" by Amanda Robinson, CC BY-SA 4.0

If the difference between the EPAP and IPAP is bigger, that is a larger push of pressure to get to the higher level.

Examples

Example A:

$\text{EPAP 6 and IPAP 10} \rightarrow \Delta\frac{P}{PS}$ of 4 (difference between EPAP and IPAP)

This is a Pressure Support of $4\ \text{cmH}_2\text{O}$.

Example B:

$\text{EPAP 6 and IPAP 12} \rightarrow \Delta\frac{P}{PS}$ of 6 (difference between EPAP and IPAP)

This is a Pressure Support of $6\ \mathrm{cmH_2O}$.

In Example B, this is a higher support, or "push," to give the patient a bigger assist for a larger tidal volume.

IPAP is usually set $+4$ - $10\ \mathrm{cmH_2O}$ above the EPAP and then adjusted to make sure the patient is taking adequate tidal volumes. Increase the IPAP to help the patient take bigger breaths or if they need more support due to increased work of breathing (WOB).

Two words of caution on pressures...

EPAP and IPAP can be increased higher than these initial ranges as needed based on the patient—namely their WOB and issues with CO_2 and oxygen. Pressures should be started lower first and then adjusted after watching the patient for about 30 minutes and checking the effect with ABGs and overall WOB. Don't start higher than these levels unless directed by a physician or a practitioner experienced with NIV.

Keep in mind as well: the higher the pressures that are used, the tighter the patient's mask needs to be. A tight mask means additional pressure that is being pushed into your patient's face and may mean that NIV is not tolerated for a conscious patient. Pressures should be initiated on the lower side and increased as needed over time to promote patient comfort and decrease the risk of failure of therapy due to patient's refusal to wear the mask. Staying within these starting ranges with slow increases over time will help promote patient tolerance.

RR stands for Respiratory Rate. But, how can you set a respiratory rate for a non-invasive mode where the patient must have an intact drive to breathe? Though it is termed an "RR," NIV does not use a true respiratory rate. As you have already learned, the patient must be triggering breaths on their own. Therefore, you can think of the RR as a mandatory breath cycle that can be used as a back-up to ensure the patient continues to trigger breaths at a certain rate. Usually, the RR is set much lower than the patient's intrinsic RR. For NIV, it is normally set at 12 - $16\ \mathrm{bpm}$, though most patients on NIV are usually tachypneic due to increased WOB or oxygenation issues.

When setting up a patient on NIV, the best guide for choosing your initial numbers originates in the reason the patient would benefit from NIV. As stated earlier, patients usually fall into two categories:

- difficulty oxygenating/requiring lung recruitment
  or
- high CO_2 or hypercapnia/increased WOB.

Patients needing lung recruitment or help with oxygenating will benefit from higher EPAPs and not as large of a $\Delta \frac{P}{PS}$—resulting in a lower IPAP setting. Patients with high CO_2/increased WOB do not need as much distending pressure (EPAP), but need their IPAPs set higher to ensure a larger $\Delta \frac{P}{PS}$ to help augment their normal tidal volume.

Occasionally, you may have a patient that is a combination of the two situations. These patients will require a higher EPAP as well as a high IPAP with a large ΔP.

Let's break it down in this table, which shows recommended initial settings according to the type of patient you are dealing with:

Table 4.8.1 NIV Recommended Initial Settings

Type of Patient	Setting Starting Point				
	EPAP	IPAP	RR	FiO_2	Charting Notation
Type 1: Needs oxygenation help and distending pressure	8	12 - 14	12 - 14	1.0	RR 12 12/8 (IPAP/EPAP) FiO_2 0.8
Type 2: Needs help clearing CO_2 or with increased WOB	4 - 6	12 - 16	16	0.5 - 1.0	RR 16 16/6 (IPAP/EPAP) FiO_2 0.5
Type 3: Combo of type 1 and type 2	6 - 8	14 - 18	16	1.0	RR 16 18/8 (IPAP/EPAP) FiO_2 1.0

If you are confused, just remember that EPAP helps those lung "balloons" from deflating fully, and that increasing that low pressure might help splint open a few more of the alveoli. A pressure increase will also help push oxygen across the alveolar-capillary membrane. Remember PEEP helping like wringing out a towel? EPAP does the same thing. IPAP is the high pressure the mode will cycle up to. The difference between the EPAP and the IPAP is going to be the amount of support or help to assist with increased WOB and also a push to augment the patient's breath to get rid of more CO_2.

4.9 CHECKING TO SEE IF NIV IS WORKING

Similar to invasive ventilation, after choosing initial settings on a mode of ventilation, watch the patient's breathing, oxygen status and tidal volume size. Small changes can be made in the first few breaths. One main difference with BiPAP versus invasive ventilation is the targeted tidal volume and RR.

Remember that NIV is used for patients that are conscious and have an intact drive to breathe. If they are tachypneic on BiPAP, this is *not* necessarily a sign of failure. As long as you are supporting adequate tidal volumes, let the patient breathe at whatever rate they want. Do not sedate them or give them sedation of any kind. This action can decrease their level of consciousness and decrease their drive to breathe, essentially making them no longer a candidate for BiPAP. Also, you can allow the patients to breathe at slightly higher tidal volumes than when doing invasive ventilation, as the patient is pulling these volumes spontaneously and the risk of barotrauma/volutrauma is a lot lower when we are not pushing the volumes into the lungs without the patient actively being involved.

After approximately 30 minutes on your initial NIV settings, do an arterial blood gas (ABG) to assess if the therapy is working. As long as the ABGs are trending in the right direction (CO_2 decreasing/oxygenation increasing), your treatment is a success! If the ABGs are still the same or getting worse (CO_2s increasing/oxygenation decreasing), then the patient may need to be intubated and fully ventilated with a control mode of ventilation to correct their abnormal CO_2/O_2 levels.

> Don't worry! ABGs are covered in detail in Chapter 8.

This automated BiPAP machine is much simpler than the ventilators we have been looking at so far, as it allows patients to use BiPAP at home. Photo by Scott Mindeaux, CC BY-NC-ND 4.0

4.10 CONCLUSION

NIV, or BiPAP, is a common therapy in critical care medicine. In the right patient population, it can be used instead of invasive ventilation and keep patients off mechanical ventilators. It is important to ensure you are picking the right patient as NIV in the wrong setting will not only be unsuccessful but can also put the patient at risk for aspiration. The classic NIV patient is alert with an intact drive to breathe. They either need some help with distending pressure or assistance with augmenting their tidal volumes with some extra support. Choosing initial settings on BiPAP is straightforward once you identify the therapy that is most beneficial to your patient.

Review

Non-invasive ventilation (NIV) is preferred to invasive ventilation, but only when the conditions are met. An ideal NIV patient is breathing on their own, fairly alert, and not at risk for aspiration. At the same time, the patient must have a need for distending pressure (EPAP/PEEP) or supportive pressures (IPAP/PS). When NIV is used in hospital, that means BiPAP, as CPAP is not a true form of ventilation.

Initiating non-invasive ventilation is very similar to how you approach setting up PSV for a spontaneously breathing patient. There are three main settings that need to be adjusted as well as one additional "back-up" setting. First, you will set the oxygen delivery (FiO2), a distending pressure to help recruit alveoli (EPAP), and a high pressure to augment the patient's normal breath (IPAP). The back-up setting is a basic RR (remember, this setting does not replace the patient spontaneously breathing). Then, the efficacy of NIV is checked after 30 minutes with an arterial blood gas.

4.11 SELF-CHECK

Chapter 4 Quiz

See how well you recall the concepts covered in this chapter by completing the following 6-question quiz.

Don't forget to check your score!

An interactive H5P element has been excluded from this version of the text. You can view it online here: https://ecampusontario.pressbooks.pub/mcvresource/?p=195#h5p-8

CHAPTER 5 | VENTILATION MODES - CONTROL VENTILATION

Chapter Outline

5.0 CHAPTER OVERVIEW

In Chapter 2 and 3, we discussed the basics of mechanical ventilation, identified the basic categories of control and spontaneous modes of ventilation and the differences between the two. It is time to build on that foundation and dive into the specifics of control modes of ventilation and the differences between pressure and volume control.

In this chapter we will cover:

- Overview of volume control and pressure control and derivative modes.
- The relationship between flow, volume, pressure and time.
- How lung compliance and resistance will impact the delivery of air into the lungs when various ventilation modes are applied.

Application

In order to use control modes of ventilation in practice, it is important to understand the significance of setting a ventilator according to pressure and according to volume. When additional factors, such as time, flow, and lung compliance are including in this understanding, you are better equipped to use control modes effectively for a specific patient's condition.

Learning Objectives

At the end of this chapter, you will be able to:

1. Explain the relationship between flow, pressure and volume.
2. Differentiate between set parameters in volume control, pressure control and derivative modes
3. Describe the impacts of changing settings in control modes
4. Describe how lung changes will impact volumes and pressures in the lungs

Key Takeaways

In this chapter, you will learn about all of the following key terms. These terms will be used throughout this book, so it is important to take the time to master them and practice your recall often.

- Inspiratory Time (I_T)
- Flow
- I:E
- gas-trapping (or Auto-PEEP)
- lung compliance
- lung Resistance
- synchrony

If you need additional information about a term than what is provided here, you can research it in The Free Dictionary: Medical Dictionary.

5.1 USING CONTROL MODES OF VENTILATION

When mechanical ventilation is indicated, it is normally because the patients are not oxygenating or ventilating appropriately. Indications for mechanical ventilation include: hypoxic failure, hypercapnia (or respiratory failure) and ineffective drive to breathe. When patients are acutely ill and experiencing indications for ventilation, their body cannot effectively balance their oxygenation or CO_2 clearance needs without intervention. They are intubated and ventilated to take over control of these imbalances. A control mode of ventilation is the obvious choice, as there is a need to fully take over how much and how often the patient is breathing, since they are ineffective in fixing it on their own.

Key Takeaway

When patients experience conditions such as hypoxia, hypercapnia, or a compromised drive to breathe, their breathing needs to be supported, and a control mode is utilized to address the underlying ventilation issue. It's important to note that while mechanical ventilation does not cure the underlying illnesses causing respiratory problems, it serves to supplement patient effort and reduce respiratory muscle workload, enabling the body to allocate more energy towards the healing and repair processes.

We have already discussed that there are two categories of control modes. One that is described via the *volume* of air delivered and one described by the *pressure* applied to the lungs. Let's narrow our focus to the control mode branch of the ventilation "family tree:"

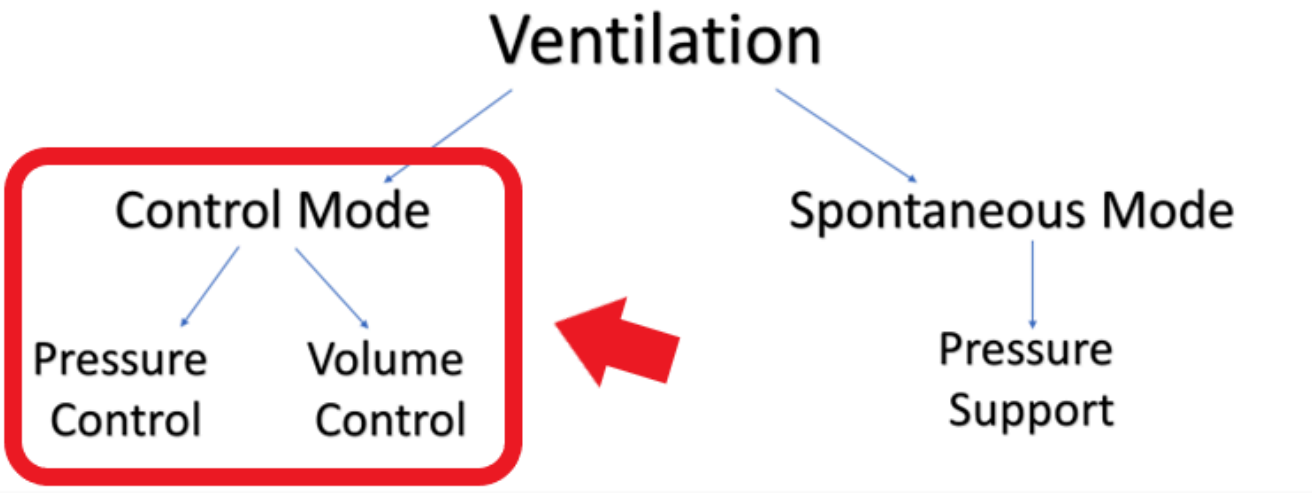


Figure 5.1.1: "Family Tree Control" by Melody Bishop, CC BY-SA 4.0

So you have identified the need to control ventilate your patient, but now we have a choice to make: do we use pressure control or volume control? Which one is better?

We previously discussed that both of these modes describe the exact same thing—air being pushed into the lungs. Realistically, once you understand the premise of these modes, you would be able to ventilate essentially the same in pressure or in volume by manipulating all of the settings on the ventilator. Though described in different ways, they are two sides of the same coin. Whether it be pressure or volume, both control modes are simply a set amount of air being pushed in at a set rate.

Key Takeaway

Take a look at the following acronyms which are commonly used by clinicians when talking about volume or pressure control:

- **A/C** = assist/control (used to refer to control mode in general)
- **PC** or **PCV** = pressure control or pressure control ventilation
- **VC** or **VCV** = volume control or volume control ventilation

This might look a bit like "alphabet soup" to you, but don't be anxious. Just translate these short-forms to *control mode*, *pressure control* or *volume control*, and you will understand perfectly. Over time, you will become more familiar with these acronyms and begin to use them yourself.

We have now gained enough knowledge about breath control and delivery to be able to define a ventilation mode based on the type of breath delivered, the control variable for each breath and the timing of breaths. The specific phase variable (trigger, limit, cycle, baseline) depends on the mode selected.

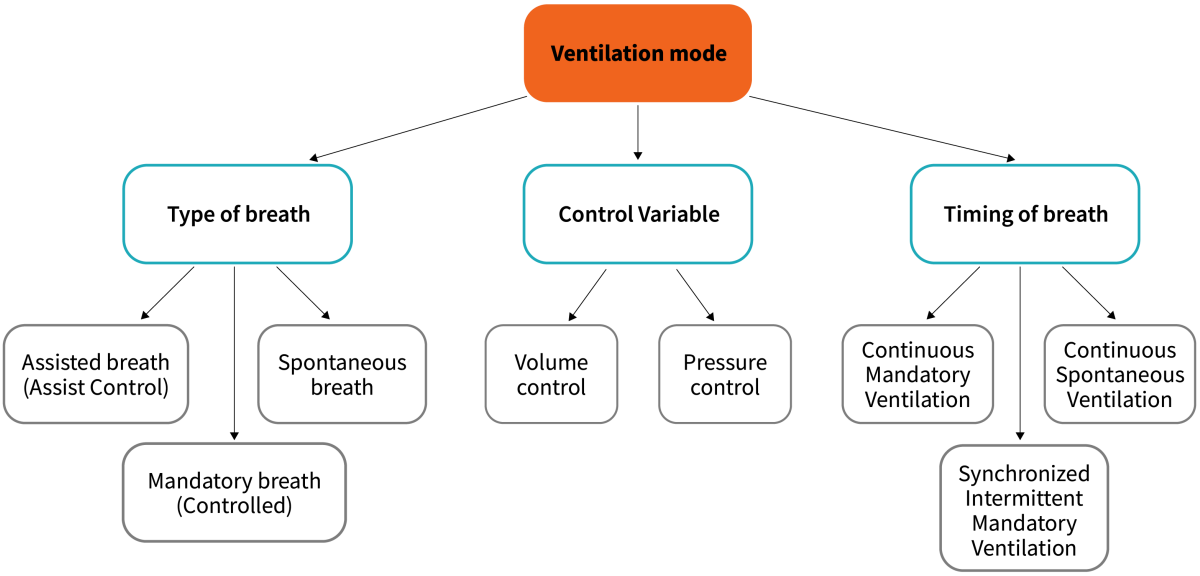


Figure 5.1.2: "Ventilation Modes" by Freddy Vale, CC BY-NC-SA 4.0

With controlled ventilation, all breaths are mandatory (controlled), meaning they are delivered independent of patient effort and are *time triggered.*

Example

For a respiratory rate of 15 bpm, a breath is delivered every 4 seconds. The limit and cycle variable for mandatory breaths depend on the control variable used (volume, flow, pressure). This type of ventilation mode is used when full ventilatory support is required in patients unable to make respiratory efforts.

Fully controlled ventilation mode is mostly a thing of the past as most ventilators today provide Continuous Mandatory Ventilation (CMV) also known as **Assist Control Ventilation.** With Assist Control (A/C) ventilation, breaths can be time triggered or *patient-triggered*. This mode is sensitive to patient effort, allowing patient triggering based on the sensitivity set by the clinician. The limit and cycle variable depend on the control variable used (volume, flow, pressure). The assist control mode offers an advantage by ensuring a minimum rate, which proves beneficial when patients are unable to initiate respiratory efforts. Simultaneously, it allows patients to trigger breaths beyond the minimum set rate according to their individual needs. When patients initiate a breath, the ventilator detects the trigger and delivers a mandatory breath based on the limits, cycle, and baseline set by the clinician. Consequently, in assist control ventilation, all breaths (both time-triggered and patient-triggered) exhibit uniform characteristics including the same limits, cycle, and baseline, with the trigger variable being the only distinguishing factor.

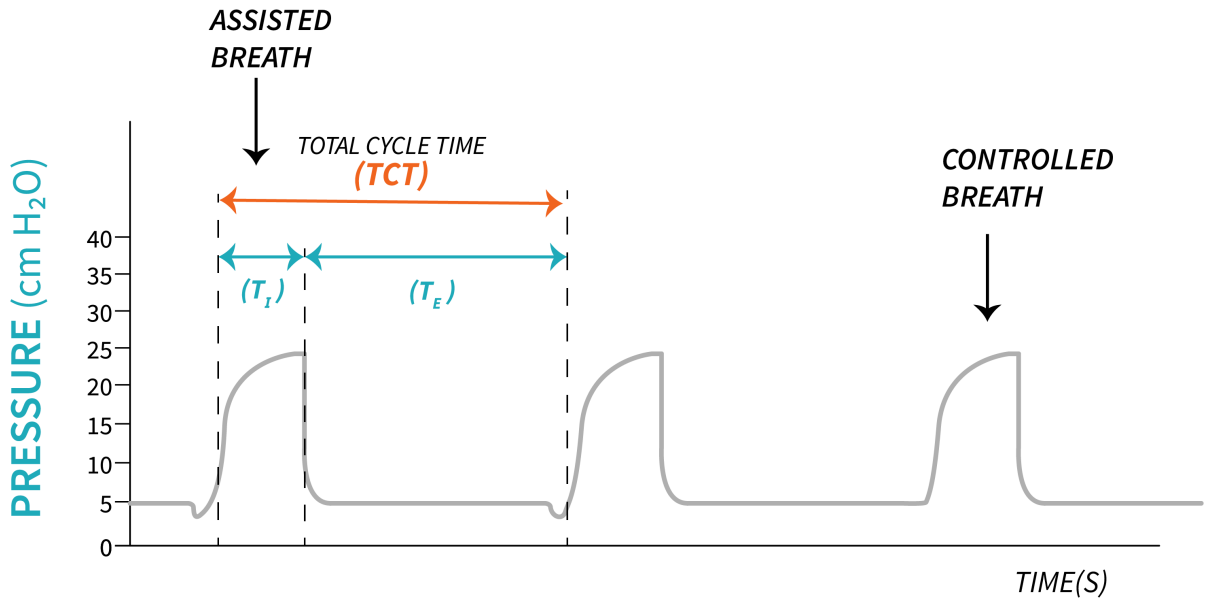


Figure 5.1.3: "Assist Control Ventilation" by Freddy Vale CC BY-NC-SA 4.0

Example

RT set a rate of 12. The patient will receive a breath every 5 seconds.

If patient requirements increase, and they start triggering the ventilator above the set rate of 12.

The ventilator will recognize those triggers, and deliver as many breaths as the patient requires. All breaths will have the same limit, cycle and baseline (all breaths will look the same)

Intermittent Mandatory Ventilation (IMV) is another ventilation strategy determined by the timing of breaths delivery. Breaths are delivered at present time intervals. This is a type of partially supportive mode as it allows patients to take spontaneous breaths in between mandatory breaths. This feature is different than Assist Control ventilation where the patient may trigger a breaths but the ventilator takes over and delivers a mandatory breath. When the patient triggers a breath during IMV, in between mandatory breaths, that breath will be spontaneous and can be supported or unsupported (more on this when we discuss spontaneous ventilation modes). Because there is no synchronization between ventilator delivered breath and patient's own spontaneous breaths, this mode may lead to breaths stacking when a mandatory breath is delivered on top of a spontaneous breath or the patient takes a spontaneous breath on top of a mandatory breath.

Most ventilators today are capable of synchronization and offer **Synchronized Intermittent Mandatory Ventilation (SIMV)**.

There is still a set rate in this mode, but the patient can dictate their own respiratory rate, as they can take spontaneous breaths in between mandatory breaths. This mode allows three different types of breaths: mandatory, assisted and spontaneous breaths. An assisted breath is delivered in SIMV, when the patient triggers the ventilator (makes an inspiratory effort) during the trigger *window*. This so called window represents a short period of time, just before a mandatory breath is delivered, based on preset time, when the ventilator becomes sensitive to any inspiratory effort (trigger), and will deliver an assisted breath. This is similar to the assist control mode described above. However, if the patient makes an inspiratory effort outside of the trigger window, then the patient will receive a spontaneous breath. If patient makes no effort in that trigger window then the ventilator will initiate a time triggered mandatory breath.

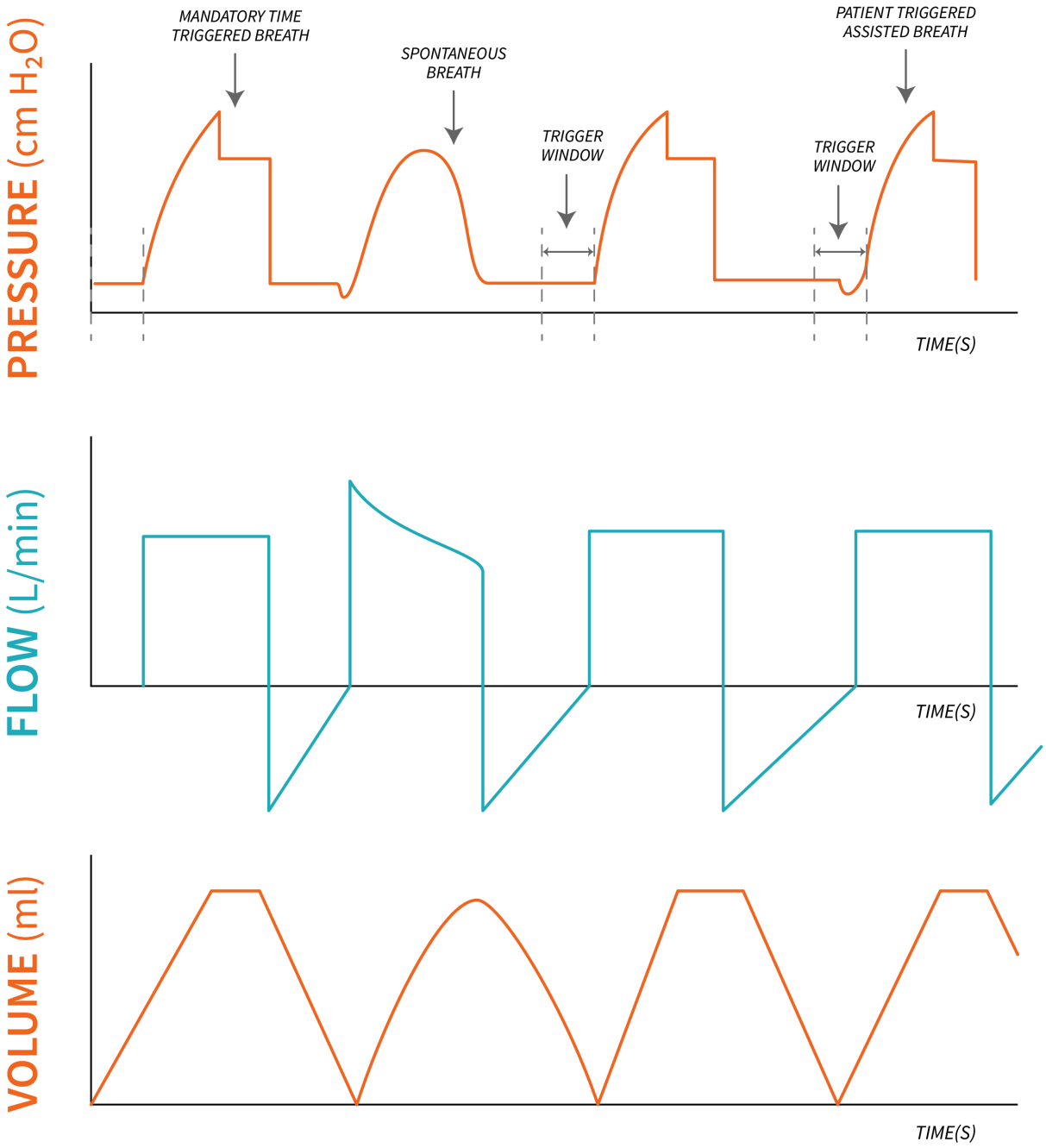


Figure 5.1.4: "Synchronized Intermittent Mandatory Ventilation" by Freddy Vale, CC BY-NC-SA 4.0

Key Takeaway

The trigger window can be likened to a key that unlocks the door to a breath. Just like a key activates the mechanism to open a door, the trigger in SIMV initiates the delivery of a assisted breath. It allows the patient to have control over when they need assistance with their breathing. Once the trigger has sensed by the ventilator, it responds by providing an assisted breath, ensuring that the patient's respiratory needs are met. Similar to how a key triggers the unlocking process, the trigger window in SIMV acts as a gateway, enabling synchronized and coordinated ventilation for the patient's comfort.

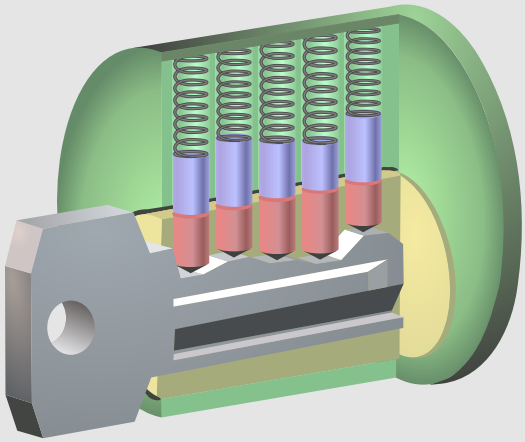


Figure by Wapcaplet, CC BY-SA 3.0

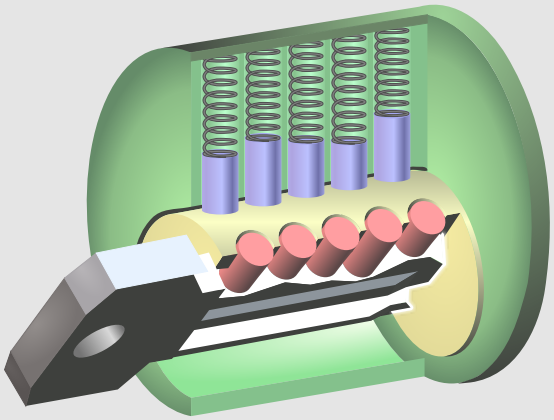


Figure by Wapcaplet, CC BY-SA 3.0

Example

With a set rate in SIMV of $10\ \text{bpm}$—the ventilator delivers $1\ \text{breath}$ every $6\ \text{seconds}$.
If the trigger window is $0.5\ \text{seconds}$, then at $5.5\ \text{seconds}$ from the onset of the last mandatory breath the ventilator becomes sensitive to any spontaneous effort, and will deliver an assisted breath. In the absence of a spontaneous effort, the ventilator delivers a time triggered, mandatory breath.

SIMV can be a challenge to understand, as it is usually a combination of control, assisted and spontaneous breaths. This mode is not used to a large degree in most Intensive Care Units due to large amounts of research that have identified inherent issues in asynchrony and increased length of ICU stays. With more adaptive modes available today, this ventilation mode is losing popularity. However, you may still encounter it, and use it in certain patients for weaning purposes where variable amounts of work of breathing are required.

5.2 THE TIME VARIABLE

The time variable is an important parameter in all ventilation modes. We have just discussed how volume and pressure have a linear and direct relationship if all other variables are the same—meaning that if the speed and time that the air is being delivered are the same and the lungs (balloons) have not changed. Now let's discuss these "other variables" we have alluded to—time and speed of air.

All control ventilation has a "time" element that is set, which refers to the speed by which air is delivered. At its most basic explanation, mechanical ventilation is the application of a positive pressure of air *over a period of time* to a closed system. This time aspect can be expressed in a few different ways; we will discuss **inspiratory time** and **flow**.

Inspiratory Time

The most direct way of manipulating how long the air is pushed into the lungs is by setting an inspiratory time (I-time, I_T). I_T is most commonly used in Pressure Control ventilation, but since it is more intuitive than other "time" parameters, it is becoming more common in volume control as well. You should already understand that if the pressure of air to a closed system is applied for a longer time (longer I_T) this will result in higher volumes.

Object Lesson

Think about balloons again. If you blew into two different balloons with the same force (pressure), but you only blew into Balloon 1 for 1 second, but then you blew into Balloon 2 with the same force for 3 seconds, Balloon 2 would be more full.

Now, consider this the other way around: If you wanted to hit a specific volume, but Balloon 1 had 1 second to achieve the volume, while Balloon 2 had 3 seconds to get the volume in, which balloon would require a higher pressure to hit that same volume? Balloon 1 needs more force to push the air in faster to hit the volume needed in the shorter time.

Photo by karosieben, Pixabay Licence.

In this way, the I_T can have a direct impact on many factors of ventilation—impacting the volume delivered, or the peak pressures experienced by the lungs. Here, the clinician will not set a flow rate of the air. The ventilator can change the flow rate to achieve the set pressure and inspiratory time that the clinician has set.

Object Lesson

Yet again, we have two identical balloons (which will represent two copies of the same lungs). Let's think about blowing up Balloon 2 twice as big (double the volume) as Balloon 1. What if we didn't want to blow too hard (high pressure) because we were worried about damage to the balloon? What if, instead, we use the same blowing force that we used to inflate Balloon 1, but just blew for a longer time? We would be able to inflate Balloon 2 to twice the volume by just blowing longer (longer I-time) at the same pressure.

Manipulating how long the air is being pushed into the lungs for can directly affect the volume without having to change the pressure. Remember: volume delivered is just air being pushed into the lungs for a set amount of time—regardless of whether you are in pressure control or volume control.

Patient comfort should also be observed while setting the inspiratory time. If the patient has any spontaneous drive, try to match the patient's spontaneous inspiratory time by watching chest-rise and respiratory efforts.

Flow as Related to the Time Variable

Some modes of ventilation do not utilize I-time (I_T). Instead, the time in inspiration is affected by the peak flows that are utilized to deliver the breaths. Most commonly, this is seen with Volume Control Ventilation. We know that flow is basically a representation of volume over time. The clinician will set the peak flow of air the ventilator will deliver when giving the set volume. How does this information relate to the time needed for inspiration? Returning once again to the balloon analogy, if a volume of $100\ \mathrm{mL}$ is the overall target, Balloon 1 has a peak flow of $60\ \mathrm{Lpm}$ vs Balloon 2 has a peak flow of $30\ \mathrm{Lpm}$. Obviously, the balloon with the air filling it at a faster rate will achieve the target volume in less time and at a lower pressure. The clinician will not set an I-time here. The ventilator I_T will depend on the set parameters of the volume and flowrate of the air.

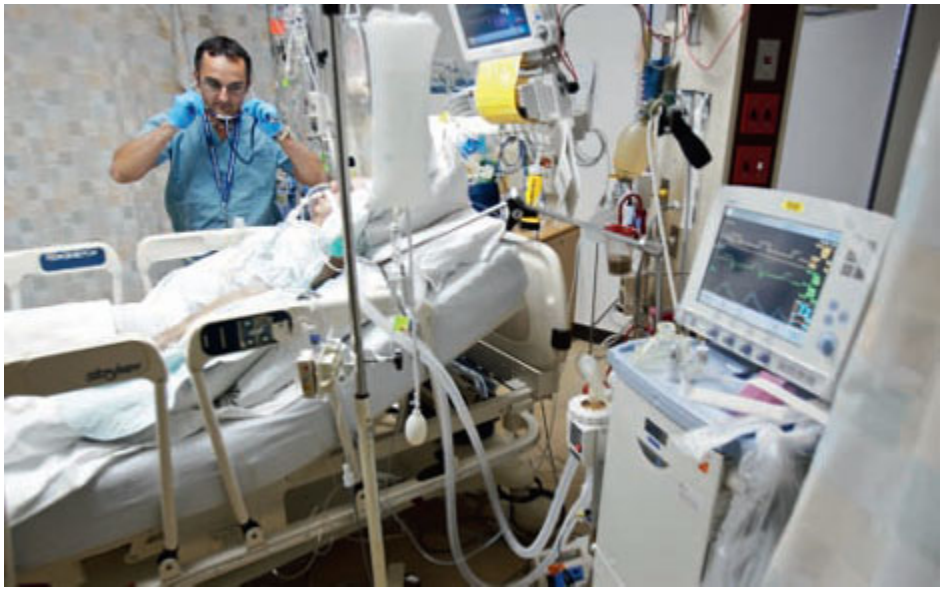
5.3 I:E RATIO

Let's review what we know about I:E Ratio from physiology and apply that to mechanical ventilation. Regardless of what setting the ventilator employs to affect time, it is important to ensure the breath delivery includes adequate time to exhale. Normal inspiratory to expiratory ratios (I:E) on spontaneously breathing patients are usually around 1:3 to 1:5. Meaning, the ratio of time in expiration is 3 to 5 times longer than the ratio of time in inspiration.

> Think logically about the time you take when you breathe to inhale and exhale. In fact, pay attention to your breathing right now. Try timing your average length of inhalation. Then, try timing your average length of exhalation. You should notice that your natural breath patterns will fit into an I:E of 1:3 to 1:5.

Why do we need longer to exhale than to inhale? If the time to exhale was shortened considerably to the point that you were spending almost the same or more time breathing in instead of out, there would be a risk that you would not have enough time to breathe out fully. You would therefore increase the baseline amount of air left in the lungs before the next breath in.

The I:E ratio is especially true in mechanical ventilation, as inspiration is an active process of the ventilator pushing air into the lungs, while expiration is a passive act with the normal elastic recoil of the lungs causing the air to flow out of the lungs. If the exhale time continued to remain too short to exhale fully, a small amount of air would continually be added to that baseline amount of air in the lungs before breathing in again. Eventually, your lungs will be too full to take the volume in without reaching dangerously high volumes and high pressures in the alveoli.



Respiratory Therapists work carefully to develop ventilator settings that will respond effectively to a patient's needs. Photo by Rcp.basheer, CC BY-SA 3.0

The I:E ratio can be directly affected by the clinician in control modes of ventilation by using I-time or flow, or respiratory rate settings. Let's take a closer look...

Time element of Inspiratory time (T_i)

If a shorter T_i or a higher flow is set, the inspiratory phase would be shorter to inflate the lungs to the set amount. For example, if you set the T_i to 0.8 seconds versus 1.2 seconds, how would that affect your patient's $\mathrm{I{:}E}$ ratio? (Before you look at the answer below, please note that this scenario will assume that the respiratory rate (RR) is maintained at 15 bpm; therefore, each inspiratory/expiratory phase is 4 seconds.)

- If T_i is 0.8 seconds, then T_E is 3.2 seconds. Therefore, $\mathrm{I{:}E}$ is $1{:}4$
- If T_i is 1.2 seconds, then T_E is 2.8 seconds. Therefore, $\mathrm{I{:}E}$ is $1:2.3$

In conclusion, a longer T_i will increase the $\mathrm{I{:}E}$ ratio, giving more time for inspiration, but less time to exhale. Longer T_i is associated with improvement in oxygenation. However, keep in mind that decreasing T_E may lead to air trapping if not enough time is given for exhalation.

For a good summary of the time element, watch this video:

One or more interactive elements has been excluded from this version of the text. You can view them online here: https://ecampusontario.pressbooks.pub/mcvresource/?p=99#oembed-1

Video: "Understanding I time, E time, TCT, and I:E ratio" By Respiratory Coach [20:48] *Transcript Available*

Respiratory Rate

As the respiratory rate increases, the $\mathrm{I{:}E}$ ratio increases—more breaths per minute means more of the time ratio spent in the inspiratory phase. This is assuming that the inspiratory time was not changed and that the only parameter changing is respiratory rate. Let's look at another comparison scenario: an RR of 10 bpm versus an RR of 20 bpm (The T_i is set at 1.0 second for both of these scenarios).

- If the RR is $10 \text{ bpm} = 6$ seconds per total cycle time (TCT), that's inspiratory + expiratory phase. With a T_i of 1.0 second, the T_E would be 5 seconds. Therefore, $\mathrm{I{:}E}$ is $1{:}5$.
- If the RR is $20 \text{ bpm} = 3$ seconds TCT, keeping T_i at 1.0 second, the T_E would be 2 seconds. Therefore, $\mathrm{I{:}E}$ is $1{:}2$.

In conclusion, a higher RR will increase the $\mathrm{I{:}E}$, giving less time to exhale.

In order to ensure adequate time for exhalation, the $\mathrm{I{:}E}$ ratio must be greater than $1{:}2$, $1{:}3$. An $\mathrm{I{:}E}$ ratio less than $1{:}2$, $1{:}1$, or inverted, such $2{:}1$, $3{:}1$, refers to more time in inspiration than expiration, which means that air trapping is almost a certainty. Again, air trapping refers to a little extra air remaining in the lungs with every single breath as the lungs do not have time to fully exhale. This remaining air is more than should be left in the lungs with PEEP and it can cause the lungs to inflate more and more, eventually leading to over-inflation and lung damage. Remember, the term air-*trapping* is referred to as **Auto-PEEP**.

Conventional ventilation strategies suggest $\mathrm{I{:}E}$ ratios of around $1{:}2$—meaning expiration should be twice as long as inspiration. Lower $\mathrm{I{:}E}$s (shorter T_i, longer T_E) could be beneficial in certain pathologies prone to airway collapse, such as some patients with

COPD or asthma where the airways may restrict the flow of air going out of the lungs, requiring more time for the lungs to passively deflate. In certain cases where patients are difficult to ventilate, I:E ratios of 1:1 or even inverse ratio are used, with very careful monitoring that gas trapping is not occurring. This strategy will be covered in detail in a later chapter.

5.4 PUTTING IT ALL TOGETHER: VOLUME, PRESSURE AND TIME

Imagine you are walking a mountain range. We will use your path across the mountains as an analogy in which each mountain represents a breath that is being delivered by the ventilator.

The patient's progression through their ventilated breathing can be likened to traversing up and down a range of mountains. Photo, PickPik Licence.

When you climb up the mountain, that is your inspiration and when you climb down the mountain, that is your expiration. You start walking at the bottom of the mountain. You are not at sea level; you are already at a set altitude before you even start climbing the mountain. This baseline altitude represents the PEEP—the lungs are never empty, even before a breath is delivered.

Once you climb to the top, the peak of the mountain represents the peak pressures experienced in the lungs. The difference in height that you climbed from the start represents the pressure control (Inspiratory pressure). The time it took to reach the top represents the Inspiratory time of the breath. The entire size of the mountain, if you broke down the amount of rock and measured the weight of it, represents the volume of air. The speed of your climb represents the flow.

Review the following visual representation of this analogy carefully:

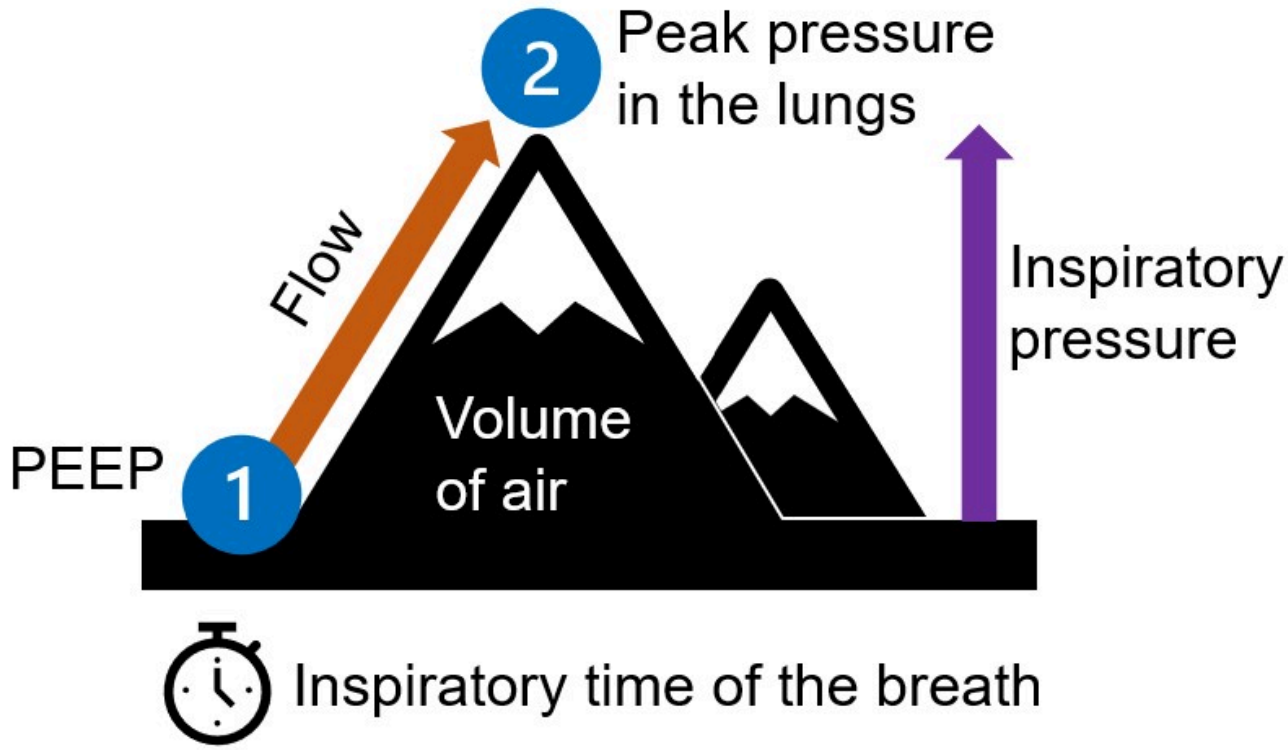


Figure 5.4.1: "Mountain Analogy Infographic" by Amanda Robinson, CC BY-SA 4.0

Advance your understanding in this analogy by deepening your thinking. Consider the following two points:

- Peak heights are a factor of the altitude you start plus how high you climb (PEEP + inspiratory pressure): If you start at a lower altitude (PEEP 5) and walk up a steep mountain (your set pressure is PC 10) you could reach the same peak height (peak pressure), as if you started at a higher altitude (PEEP 8) and walked up a shorter mountain (PC 7).
- The size of the mountain in amount of rock (volume) can still be larger if you climb longer but not as high: a high narrow mountain (high pressure and short inspiratory time) might be less rock mass then a wide, moderately high peak (moderate pressures and long inspiratory time).

Now, consider a few applications to ventilation using this same analogy:

- **Volume control (VC):** If you want to climb a bigger mountain in the same amount of time, you have to walk faster. In the same way, in the context of volume control, the more volume, the higher the flow.
- **Pressure Control (PC):** If you climb to a higher peak, the mountain is larger. In the context of pressure control, when a higher pressure is applied, this equates to a bigger volume.
- **I-time (I_T):** If you climb longer at the same speed, but to the same peak altitude, this means that you're climbing a wider mountain, which is still a bigger mountain. When the Itime is lengthened, at the same flow and to the same peak pressure, this means that you will have higher volume than before.

5.5 VOLUME CONTROL VENTILATION

We have overviewed the two branches of control ventilation: pressure and volume. Now lets dive into the two types of control modes a little deeper. Volume Control (ACVC, VCV, CMV-VC) is usually a more straightforward and easy-to-understand mode for anyone new to the world of mechanical ventilation. The clinician sets how big (tidal volume) a breath and how often the breath is delivered. As the name suggests, the volume of breath delivered is exactly the same every single time; it is constant and does not vary. The ventilator will deliver whatever pressure is needed to achieve the tidal volume in the set time that each breath must be delivered in. Volume is the control variable here. Once the set volume is delivered, the ventilator will either cycle into exhalation, or hold an inspiratory pause, as set by the clinician. In the later case, the ventilator will time cycle into exhalation at the end of the inspiratory pause (inspiratory hold).

We learned that flow is an expression of volume over time. Flow remains constant until the volume is delivered. Thus the flow curve does not change if the volume is the controlled variable, and there is a set respiratory cycle time. Usually, in volume control the set time is controlled by a flowrate, but some ventilators will let you set an inspiratory time instead.

The clinician sets on the ventilator the amount of air to be pushed into the lungs and the number of times the lungs will inflate per minute (tidal volume and respiratory rate). Also set is the max flow rate of the air being delivered—think of it as the "speed" of the air being pushed into the lungs. With constant volume, the Inspiratory time will change as a byproduct of the max flow rate you set on the ventilator. The higher the flow rate, the shorter the inspiratory time of the breath to deliver the set volume.

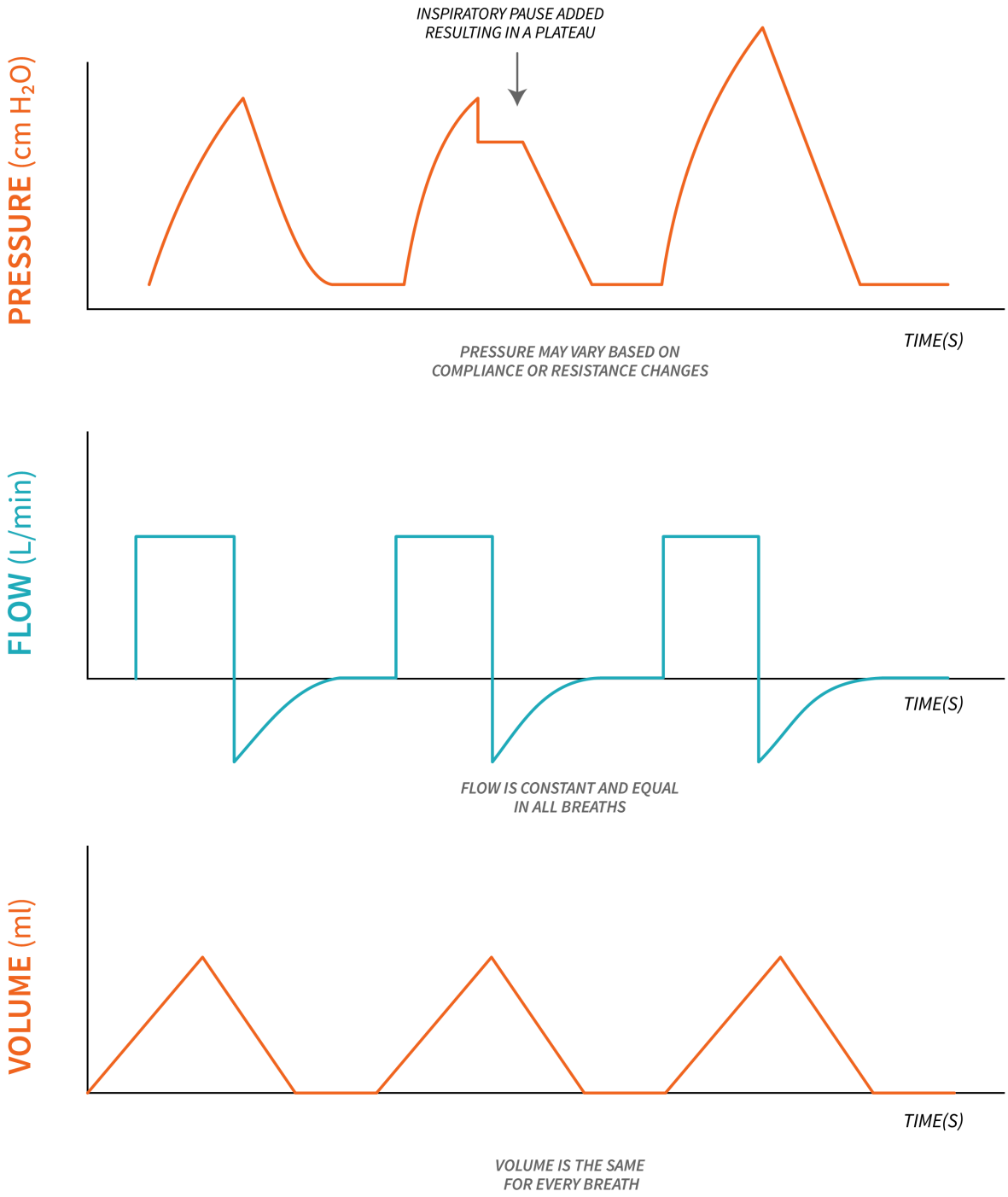


Figure 5.5.1: "Volume Control Diagram" by Freddy Vale, CC BY-NC-SA 4.0

One parameter that you do not set in volume control is pressure—specifically peak pressures or distending pressures (pressure control or inspiratory pressure). Remember that ventilators deal with the application of positive pressure to fragile alveoli in the lungs. If not done carefully, the alveoli will act like any balloon. Balloons, if overinflated, will pop. Alveoli, if inflated too much, will become overdistended and can be irrevocably damaged. Have a look at the graphic above, pressure increased on the third breath due to a change in either compliance or resistance in order to deliver the set tidal volume.

Also take note on the above graphic that an inspiratory pause (inspiratory hold) can be added to the inspiratory time, resulting in a plateau on the pressure-time waveform. During this inspiratory pause, plateau pressure can be measured.

With constant volume, the Inspiratory time will change as a byproduct of the max flow rate you set on the ventilator.

$$\text{Flowrate} = \frac{\text{Volume}}{\text{Time}}$$

PEEP is always present in the lungs and contributes to the peak inspiratory pressure, but does not impact distending pressure experienced by the alveoli. PEEP is ensuring the lungs do not collapse.

So how do you limit the pressure being experienced in the lungs when you are in volume control mode? You cannot directly set it—you set the volume instead. Pressure is monitored as a direct relationship based on the volumes and flows set. This means that you need to watch the pressure the ventilator is needing to inflate the lungs at the set volume and time you are asking it to achieve. If the pressure you are seeing in the lungs is too high, you may need to decrease the volume you are targeting or slow down the flowrate (increase the inspiratory time) to decrease the pressures experienced by the lungs.

Key Takeaways

- Pressure and volume have a linear relationship. If you decrease the volume, you are inflating the lungs too. This will decrease the pressure.
- Taking longer to inflate the lungs will allow you to "blow softer" or a lower pressure to achieve the same volume. Slowing the flow rate down or increasing the Inspiratory time will help with this.

While in volume control ventilation, the flow available to the patient is limited, a ventilator can use different patterns to deliver the flow of air. Other than constant waveform (rectangular waveform), you may see the following patterns:

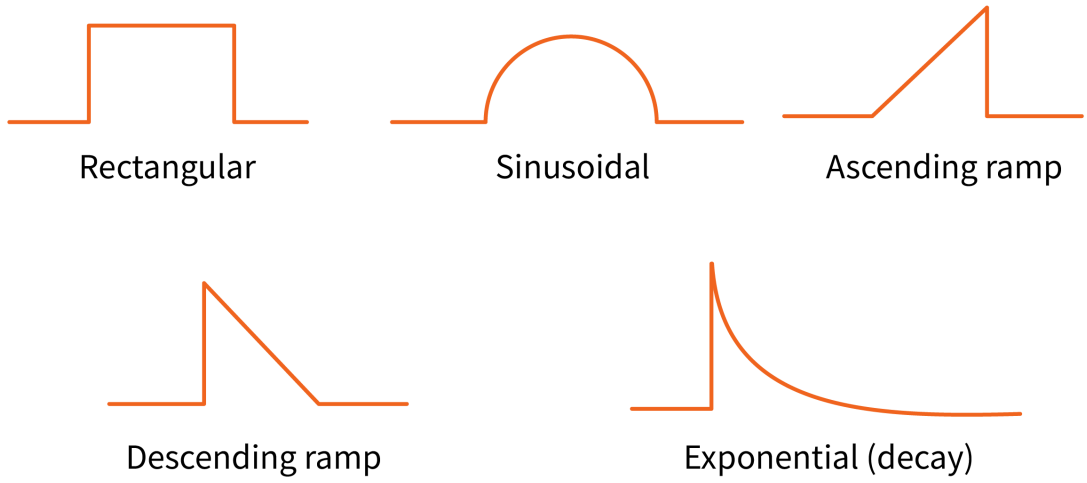


Figure 5.5.2: "Flow-Time Waveforms" by Freddy Vale, CC BY-NC-SA 4.0

Test Your Knowledge

Consider volume control ventilation and what are the breath variables for this ventilation mode: Trigger, limit, cycle and baseline.

Table 5.5.1 Summary of Basic Settings in A/C – VCV

Summary of Basic Settings in A/C – VCV	
In A/C volume control, you will directly **set**:	In A/C volume control you will **observe** but not directly set:
• Respiratory Rate (RR) • Tidal Volume (V_T) • Max flow rate (V) or Inspiratory Time (I_T) • PEEP (every ventilator, every mode) • FiO_2 (every ventilator, every mode)	• Inspiratory Pressure (PC) • Peak Inspiratory Pressure (PIP) • Inspiratory Time (I_T) would not be set if the ventilator lets you set flow. In this case, observe Itime.

Lung Compliance and Resistance

We have now reviewed and reinforced the relationship of volume and pressure, and we have also discussed that in volume control the achieved volume will always be delivered by the ventilator with whatever pressure is needed. The volume is constant unless the clinician decides to change it based on what they are noting in terms of the patient's condition. But what if the lungs themselves start to change? We know that many illnesses and pathologies can change the status of the lungs: they can make the lungs stiffer (less compliant) or restrict the air's ability to flow through the airways (increased resistance).

Let's explore lung compliance and resistance a little further and how these factors impact volume control ventilation.

Lung compliance is a measurement of how easily the lungs can expand and stretch. More compliance would refer to lungs that stretch much easier than lungs with lower compliance. If lungs are less compliant, they will be harder to inflate and they will need higher pressures to overcome the rigidity of the lungs. Think about blowing up a regular balloon versus a tire. Both inflate and expand, but a tire is much more stiff and less compliant than a regular balloon so air must be delivered at high pressures, such as by an air compressor. In the same way, any lung condition that affects the stretch of the lungs will cause a lowering of compliance, and higher pressures will be needed to inflate the lungs.

Lung resistance refers to obstructions along the pathway that air will take into the lungs. Air flows through the ventilator circuit, through the endotracheal tube into the trachea, bronchus, bronchioles and then to the alveoli. The amount of force needed to start to move air through these tubes is termed resistance. Think about blowing into a straw vs. a paper towel roll. You need to blow harder to get air passing through the straw compared to the paper towel roll because it is more narrow. The narrower the tube, the higher the resistance to air movement. Any narrowing of the airways will cause a resistance to airflow, and the air will not be able to enter the lungs as easily. A good analogy for lung resistance is lane closures on a highway. Not as may cars get through and it will slow traffic down. Lung resistance will decrease the amount of air entering the lungs for every breath and will require a higher pressure as well.

As you can see, both of these factors—lung compliance and lung resistance—impact the pressures needed to deliver the set volume in volume control.

Object Lesson

Imagine blowing up two very different balloons with the exact same volume over the same amount of time. Balloon 1 is a regular birthday balloon that has been blown up many times, but Balloon 2 is a long skinny "balloon animal" balloon that has never been blown up before. Obviously, the stiffer and longer Balloon 2 will be harder to inflate and it will definitely need a higher pressure to achieve the set volume, than Balloon 2, which is looser and inflates more easily.

The same can be said for ventilating lungs. If you deliver an equal volume of air at the same flow to normal or "healthy" lungs versus damaged or "stiff" lungs, the pressures required to inflate those lungs would be different. Stiffer lungs would require a higher pressure to achieve the same volume.

"Balloon Animals" by DHORA S.R.L. IMPRESA SOCIALE, CC BY-ND 2.0

We have spent a large amount of time highlighting the relationship between pressure and volume—specifically how the pressures will increase as the volume is increased. This correlation is noticed when the clinician is making changes to the settings. In contrast, the changes noted due to compliance and resistance happen *without any changes being made on the ventilator*. Instead, these changes occur breath-to-breath over time as the lung condition changes. For example, if a patient has asthma, the resistance of their lungs increases as their airways constrict, or tighten, during an asthma attack. Air can't get into the lungs as easily.

Remember, in volume control, the ventilator will adjust the pressure needed to reach the volume that is set. When the resistance increases, or if compliance drops, in volume control ventilation, this will cause an increase in the peak pressures to overcome the changes in the lungs and deliver the set volume. With this in mind, when ventilating in volume control, the pressures required by the ventilator to deliver the set volume can give you information about the status of the lungs. In a way, monitoring the pressures over time can give you another vital sign to monitor and can sometimes give you direct information on if the lungs are improving or getting worse. This information may help direct care or prompt the need for medical treatment, depending on the lung pathology, to help improve the compliance and resistance.

Let's review the pulmonary pressures and their definitions to better understand the impact of changes in compliance and resistance on volume control ventilation.

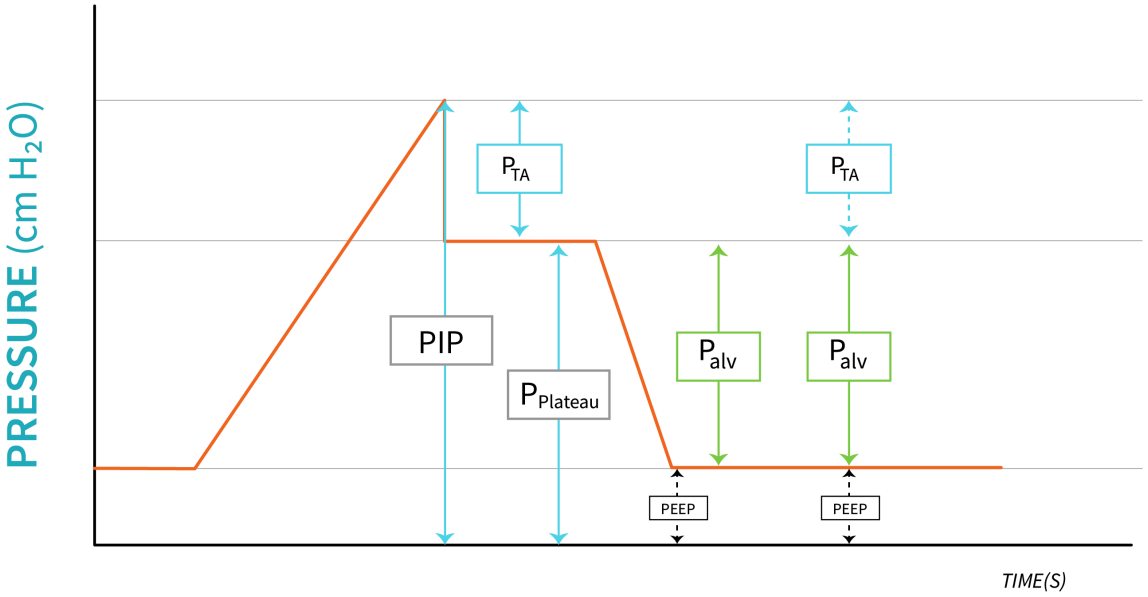


Figure 5.5.3: "Pulmonary Pressures" by Freddy Vale CC BY-NC-SA 4.0

Remember from Chapter 1 that a change in lung compliance will be reflected on the alveolar pressure (P_{alv}), which in term will affect plateau pressure (P_{plat}). Plateau pressure is often measured and monitored during volume control ventilation to identify changes in compliance. A change in airway resistance will be reflected on the transairway pressure (P_{TA}).

Key Takeaway

To visualize the impact of changes in compliance and resistance on pulmonary pressures and implicitly on volume control ventilation, observe the Pressure-Time waveform above, and note that we can express peak inspiratory pressure, mathematically as:

$$\text{PIP} = \text{PEEP} + P_{alv} + P_{TA}$$

We can also rewrite this equation as:

$$\text{PIP} = \text{PEEP} + \left(\frac{V_T}{C_s}\right) + (\dot{V} \times R)$$

An increase in any one of the pulmonary pressures will cause an increase in peak pressure, which is what the ventilator

will recognize and alert the clinician with an alarm. It is then the clinician's responsibility to identify which component of the total pressure caused it to increase.

Example

A decrease in compliance will cause plateau pressure to increase. This, in turn, will cause the peak pressure to increase in order to deliver the same volume. The ventilator will alarm High Pressure to alert the clinician who will identify the increased plateau pressure by performing an inspiratory pause.

To conclude, in volume control, the volume delivered is constant, but there will be changes in peak pressures (PIP or P_{peak}, which refers to the highest pressure experienced by the alveoli during a breath) to achieve the set volume for both the following situations:

1. the clinician changing the set parameters (volume, flow/time).
2. breath-to-breath changes based on the compliance and resistance of the lungs.

Impacts to peak pressures should be considered by the clinician when making changes, and lung pathology should be considered if changes are noted in the pressure when no adjustments to settings are made. Peak pressures should not be allowed to go too high. Remember that high pressures can cause damage to the alveoli of the lungs. Ideally, pressures should be kept below $30\ \mathrm{cmH_2O}$, with a maximum of $35\ \mathrm{cmH_2O}$, to ensure the alveoli do not get damaged. We will discuss this concept further in Chapter 5 when we discuss Volume Control settings.

Key Takeaway

Any time you connect a patient to a ventilator, you always set FiO_2 and PEEP. In A/C (control mode) volume control, in addition to these settings, you must decide how often the patient is breathing as you are taking over control of the breath initiation. Therefore, the Respiratory Rate (RR) must be set. In A/C volume control, you set the volume of air delivered with every single breath. This amount is always the same and will not change unless you, the clinician, change it. Pressure is not set: it is an outcome based on the volume and flow/time of the breath and the state of the lungs you are ventilating. Target plateau pressure lower than $30\ \mathrm{cmH_2O}$ with peak pressure of $35\ \mathrm{cmH_2O}$.

5.6 PRESSURE CONTROL VENTILATION

Now that we have fully discussed volume control ventilation, we will move on to pressure control. All of the principles we have learned so far still apply. The main difference between the two categories of ventilation is that in volume control, we set the volume, but in pressure control, we do not directly set the volume. Instead, in pressure control, we set the pressure.

Pressure Control Ventilation (A/C-PC, PCV, CMV-PC) is a full control mode of ventilation—the ventilator will control almost all phases of the breath delivered based on the set parameters. The word "almost" all phases, is intentional, to allow inclusion of patient triggering a breath. In the absence of patient inspiratory effort, the ventilator controls ALL phases of the breath. Exhalation is still passive. The clinician sets a minimum respiratory rate and sets the positive pressure applied (pressure control) to the lungs that causes the lungs to inflate with volume. The pressure delivered is constant and does not change breath to breath unless the clinician changes it.

Apply Your Learning

So, if the pressure is what is constant here, what will change with changes in lung compliance or airway resistance? Think back to the relationship of pressure, volume and time. If you increase the pressure applied to a balloon, how will the inflation change? (If you guessed the volume of inflation would increase, you got it!)

Volume will vary based on changes to the set pressure control. The higher the pressure delivered, the higher the volume. But just like volume control where the pressures can change breath to breath based on lung condition, we see the same thing in pressure control with the volume changing breath to breath based on the compliance and resistance of the lungs. If the compliance increases, the same pressure control will give you a larger volume. If the resistance increases, this will decrease the volume into the lungs.

Think back to the analogy of the balloon versus the tire for compliance. If no change in pressure is applied, which object will inflate larger with the same pressure?

Now, recall the analogy of lane closures on a highway for resistance in the airways. How would opening a lane back up—decreasing resistance—affect the volume of traffic getting through?

With the same amount of pressure given, the balloon would inflate quicker and easier than a tire, resulting in a higher volume inside the balloon. The same thing occurs with lungs. With the same pressure control settings, "stiffer" lung tissue that has poor compliance or increased resistance will exhibit smaller volumes than "healthy" lungs with normal compliance and resistance.

Just as tires require air compressors to inflate because they offer high resistance, lung tissue can be stiffer in patients with certain conditions. Photo by Maxime Agnelli, Unsplash Licence.

Let's apply this concept to our asthma patient. During an acute asthma exacerbation, their airway has increased resistance to air coming in. Any pressure being pushed in would conclude with less volume in the lungs compared to a healthy lung with normal airway resistance. Asthmatic airways decrease the flow of air getting into the lungs and resist inflation. If bronchodilators were given and the airways started to open, the lung volumes would start to increase as the airway resistance starts to return to normal.

Apply Your Learning

Consider the case of pressure control ventilation with no change in the set pressure. What would happen to the volume delivered if the compliance of the lungs decreased and they became much stiffer and less stretchy?

In pressure control, the volume being delivered over time is an additional vital sign that you can trend to see whether the lungs are improving or getting worse. Volumes must be continuously monitored to ensure they are enough to effectively ventilate your patient, but not too high that they cause damage. Pressure control (Inspiratory Pressure) should be limited to deliver tidal volumes within a safe range for the patient's lungs.

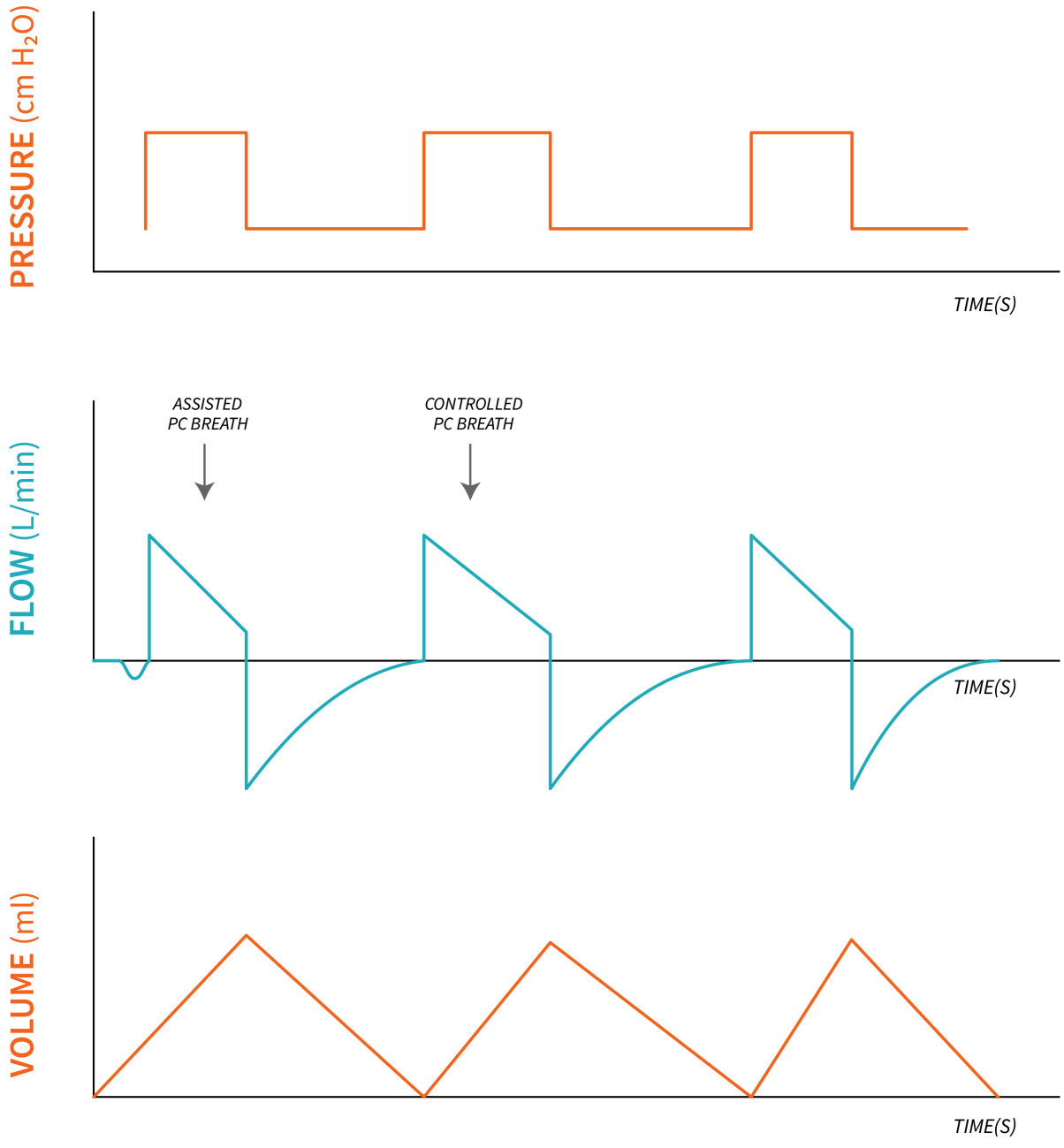


Figure 5.6.1: "PCV Waveforms" by Freddy Vale, CC BY-NC-SA 4.0

Key Takeaway

Overdistension and damage to alveoli doesn't just occur with high pressures; the same can occur with high volumes. Pressure control (Inspiratory Pressure) should be limited to deliver tidal volumes within a safe range for the patient's lungs.

Table 5.6.1 Summary of Basic Settings in A/C – PC

Summary of Basic Settings

In A/C pressure control, you will directly **set**:

- Respiratory Rate (RR or f)
- Pressure Control (PC)
- Inspiratory Time (I_T)
- PEEP (every ventilator, every time)
- FiO_2 (every ventilator, every time)

In A/C pressure control, you will **observe** but not directly set:

- Tidal Volume (V_T)
- Inspiratory flow ($\dot{V}$)
- Peak Inspiratory Pressure (PIP or P_{peak})
- I:E

The clinician will set how often a minute the breaths are being delivered. Also set is the driving pressure of air into the lungs, as well as the amount of time the pressure is applied for (T_i). What is not set in Pressure Control Ventilation (A/C-PC) is the specific volume being given. The volume is a direct product of the amount of driving pressure (PC), and the time that pressure is applied for (T_i). In other words, the pressure and time delivered would be set, and the volume would be an outcome of how long and how hard the ventilator pushes air into the lungs.

Inspiratory Flow is not a set parameter either. The clinician sets the Inspiratory time (T_i) that the breath is delivered for, but the patient can alter the amount of flow that is being given by the ventilator, pulling whatever flow they require for the duration of time indicated in the set inspiratory time.

The fact that patients can modify the flow is significant to remember for any patients who have an intact drive to breathe that exhibit symptoms of "air-hunger" (e.g., COPD exacerbations, Kussmaul respirations, Cheyne-Stoke Respirations). With abnormal neuro-driven respiratory patterns, if the patient's inspiratory demand is really high, fixed inspiratory flows could be uncomfortable for them. They could outstrip the flow that is being offered by the ventilator causing double triggering of breaths, asynchrony, and ventilator alarms. A pressure control mode with a flowrate that adjusts to the patient demand can in these cases be slightly more comfortable without having to make ventilator changes to try to mimic the patient's demands.

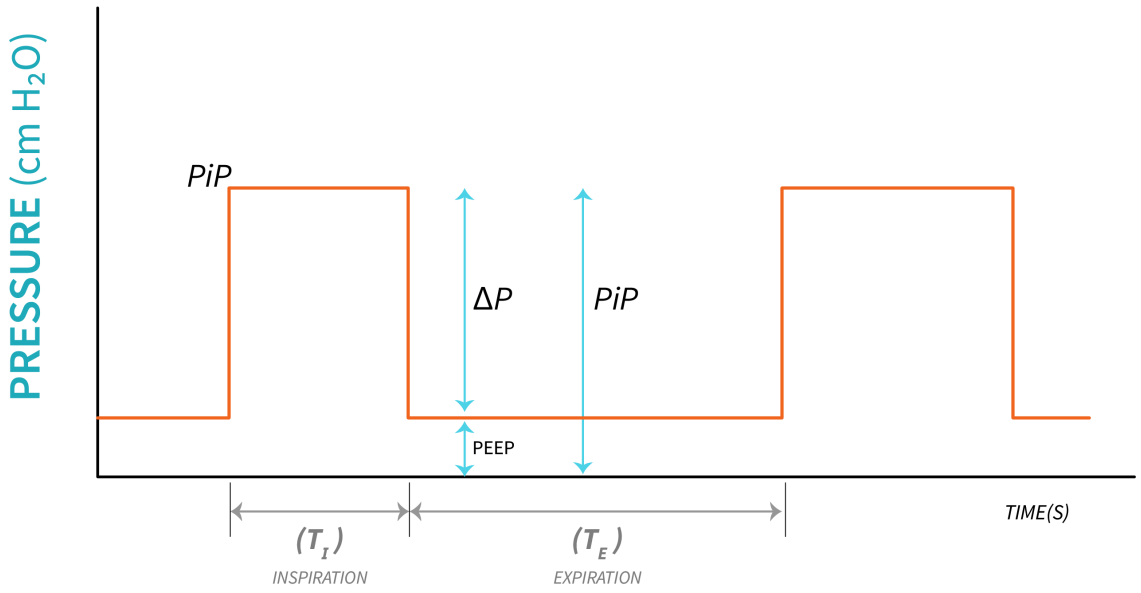


Figure 5.6.2: "Pressure – Time in PCV Waveform" by Freddy Vale, CC BY-NC-SA 4.0

Depending on the ventilator, the clinician sets Peak Inspiratory Pressure in some occasions, and in others, driving pressure (PC or ΔP). When Peak Inspiratory Pressure (PIP) is not directly set by the clinician (ΔP is set instead), it remains very controllable in pressure control ventilation as it is the sum of all pressures experienced in the lungs $(\text{PIP} = \text{PEEP} + \Delta P)$. The PIP is the highest pressure being delivered to the alveoli. Since the pressure being delivered (ΔP) is set, the PIP is the ΔP plus whatever baseline you were at (PEEP). Think back to the mountain range analogy. Standing at the bottom of a mountain, you are not at sea-level altitude. How high is the peak? It's not just "how high the mountain is". The altitude at the peak would be the total altitude above sea level, which would be the altitude at basecamp (PEEP), plus the height of the mountain (ΔP). Take look at these two examples:

- $\text{PEEP } 5 + \Delta P\ 10 = \text{PIP } 15$
- $\text{PEEP } 8 + \Delta P\ 12 = \text{PIP } 20$

Apply Your Learning

Now you try! Your ventilator settings are: $\text{PEEP } 6, \Delta P \ 15$. What is your PIP? What would your PIP be if you decrease ΔP to 12?

What will your PIP be, if you increase PEEP to 8?

When Peak Inspiratory Pressure (PIP) is directly set on a ventilator, the baseline PEEP will be included in that setting. PIP is still the sum of $\text{PEEP} + \Delta P$, however, ΔP is determined by the PIP and PEEP settings $(\text{PIP} - \text{PEEP} = \Delta P)$.

For example, if we set $\text{PIP} = 20$ and $\text{PEEP} = 5$, ΔP is $20 - 5 = 15$. Tidal volume will be determined by how much air can be pushed into the lungs with a pressure of 15. If we want to increase tidal volume, we would want to increase ΔP, but this ventilator does not offer that feature directly. Instead we can increase PIP to increase the difference between PIP and PEEP, which is ΔP.

Apply Your Learning

With a PEEP of 6 and PIP of 22. What is ΔP?

What will ΔP be, if you increased PEEP to 8?

The I:E—as previously explained—is very important to ensure the patient has adequate time to exhale and decrease the risk of auto-PEEP (air-trapping). The clinician sets the Inspiratory time and the respiratory rate, but not the I:E directly. The I:E is the outcome of those settings and will remain constant as long as the RR and inspiratory time remain unchanged. If the patient starts to trigger breaths above the set RR, the I:E will change as the total RR increases with patient-triggered breaths.

Remember, T_i is important to ensure the I:E is not an inverse ratio. There should always be more exhalation time than inspiration. If needed, the RR or T_i can always be adjusted to ensure adequate expiration time.

Key Takeaway

Any time you connect a patient to a ventilator, you always set FiO_2 and PEEP. Therefore, in A/C pressure control, those would be set by the health care provider. In a control mode, you must decide how often the patient is breathing, as you are taking over control of the breath initiation. Therefore, the Respiratory Rate (RR) must be set. In A/C pressure control, you set the pressure of air delivered with every single breath. The pressure is always the same and will not change. Volume is not set: it is an outcome based on the pressure of air delivered and the lung mechanics of the lungs you are ventilating.

When thinking about the impact that pressure and volume have on each other and how lung compliance will affect the pressures or volumes in the lungs, always think about balloons. When thinking about how a settings change will affect the opposite, think of two identical balloons with the change to how you are blowing into the balloon (the ventilator pushing the air based on the settings). When thinking about how lung compliance and resistance affects the pressure or volumes, think about the balloon and the tire and remember what settings are constant (volume or pressure) depending on what mode you are in. From there, consider how you would need to change the set parameters to achieve the inflation you want.

Knowing that pressure is the control variable and it stays constant for every breath in pressure control ventilation, consider the impact of changing lung compliance or airway resistance on ventilation. In pressure control ventilation, with constant pressure, when compliance decreases, less tidal volume will be delivered to the lungs. The same will happen when airway resistance increases.

Now, what about P_{TA} and P_{plat} in pressure control ventilation? The still play important roles. When analyzing the pressure-time waveform, it may seem that P_{TA} is equivalent to P_{plat} due to their square shape. However, this assumption is incorrect. P_{plat} is actually measured under conditions of no flow, where only elastic pressure is evaluated.

PIP is determined by the combined effect of both elastic and resistive pressures acting on the lungs. To identify the specific component causing low volume, an inspiratory hold can be performed. This allows us to differentiate between resistive P_{TA} and elastic P_{plat}. By evaluating the elastic pressure that distends the lung under zero flow conditions, we gain insights into the lung's distensibility.

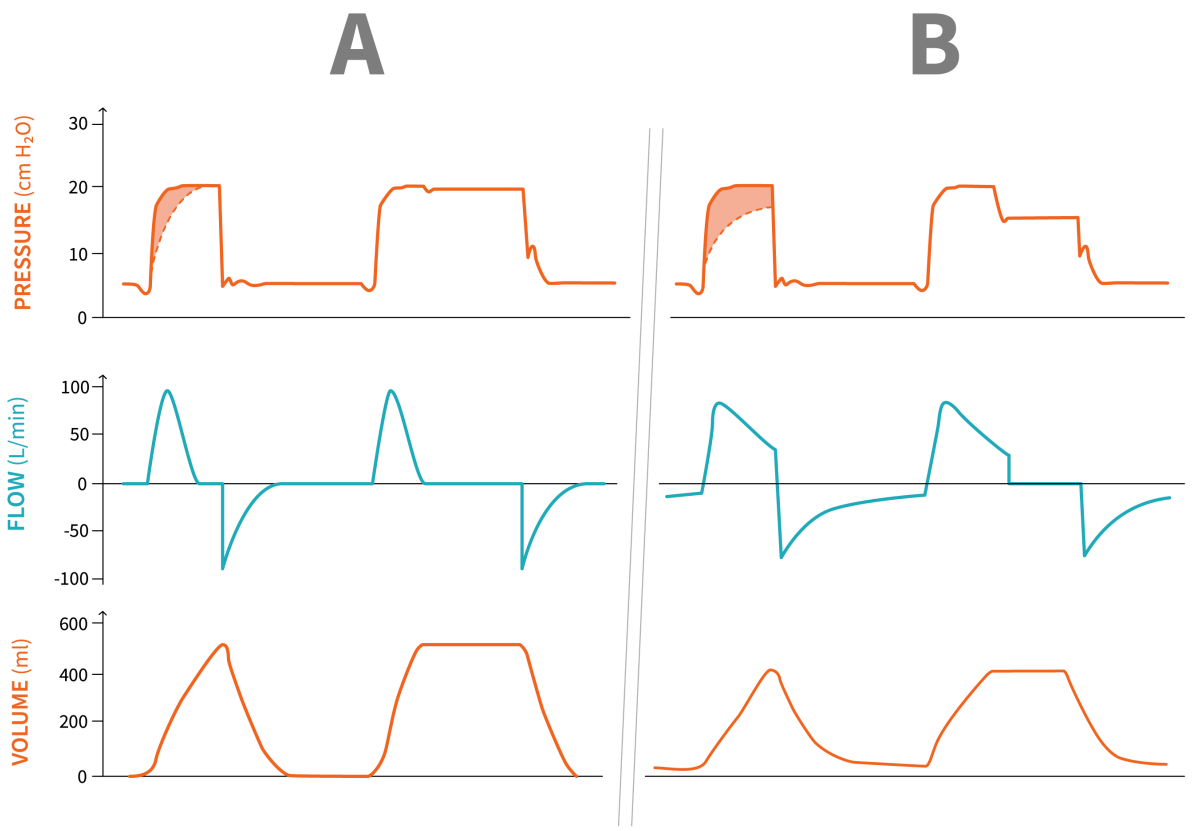


Figure 5.6.3: "Plateau pressure during pressure control ventilation" by Giacomo Bellani, adapted by Freddy Vale, CC BY-NC 4.0

Let's examine the diagrams provided. In subject A, where resistance is low, flow reaches zero at the end of inspiration. Consequently, PIP is very close to or equal to P_{plat}. On the other hand, subject B, with increased resistance, experiences incomplete flow cessation at the end of inspiration resulting in a lower P_{plat} compared to P_{peak}. This indicates that more of the total pressure generated by the ventilator is utilized to overcome the resistance to flow.

Please refer to the accompanying diagrams for a visual representation of these concepts.

I hope this clarifies the distinction between P_{TA} and P_{plat} in pressure control ventilation. If you have any further questions or require additional clarification, please feel free to ask.

For more information on this topic, read this excellent article on the topic:

Plateau Pressure During Pressure Control Ventilation

Regardless of whether you ventilate in pressure control or volume control, it is important to constantly monitor the volumes being delivered and the peak pressures being experienced by the lungs. Both of these values, if left unchecked, can cause overdistention

of the alveoli and result in damage. High pressures can result in barotrauma while high volume causing damage is referred to as volutrauma.

When would you use Pressure Control or Volume Control? Realistically, both modes can achieve similar outcomes as long as the clinician is setting patient-oriented ventilator settings and ventilator outcome parameters closely to ensure lungs are not exposed to over distention via barotrauma or volutrauma. Patient comfort should also be closely monitored, and settings such as RR, flow, and Itime should be changed to achieve optimal matching of patient's demands. This approach will improve comfort and ensure ventilator **synchrony**—where breath delivery is matching the timing of the patient's physiologic effort.

5.7 OTHER VENTILATION MODES

As previously mentioned, the basic modes of ventilation are volume and pressure. All other modes are derivatives of the basic modes. Many of these modes use a closed loop feedback algorithm where the ventilator has the ability to interpret data measured from the patients and make adjustments to target certain parameters.

Pressure Regulated Volume Control Ventilation (PRVC)

PRVC can be described as a mode in which breaths are patient or time triggered, pressure regulated, volume targeted, and time cycled. In this mode, the clinician establishes a target tidal volume based on the patient's ideal body weight. The ventilator will start ventilation with a volume controlled breath during which it measures lung compliance and the pressure required to inflate the lung. For subsequent breaths, the ventilator then measures the delivered tidal volume and compares it to the set target. If there is a discrepancy, the ventilator automatically adjusts the pressure to achieve the desired volume. You may find this mode with different names on different ventilators. In clinical setting, ensure that you familiarize yourself with each ventilator and available modes. PRVC is also known as: Adaptive Pressure Ventilation (APV) Volume Control + (VC+).

To ensure patient safety, it is crucial to set an appropriate upper pressure limit when using PRVC. Most ventilators have safeguards in place to prevent pressure adjustments from exceeding $5\ \mathrm{cmH_2O}$ below the defined upper pressure limit.

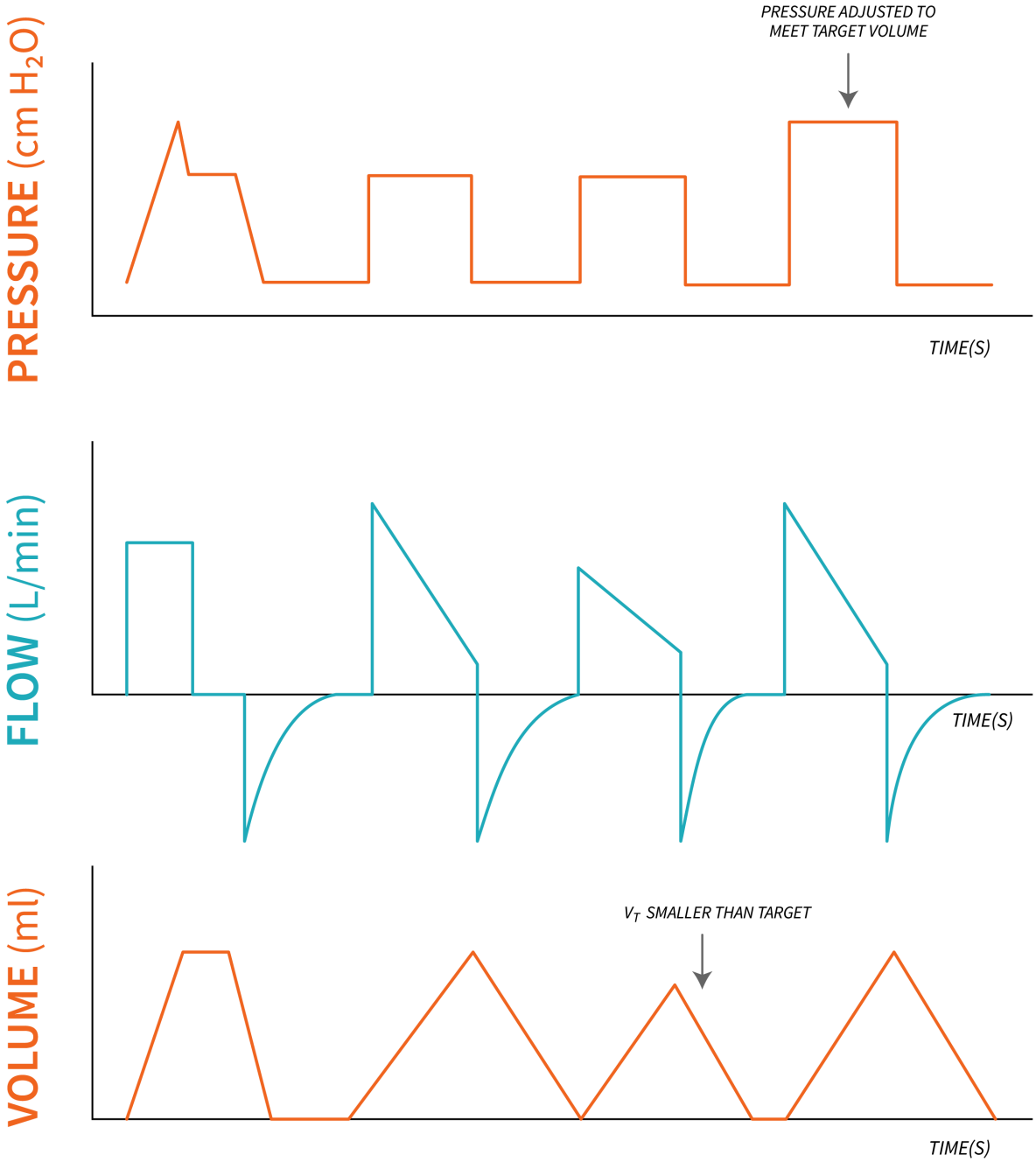


Figure 5.7.1: "PRVC Diagram" by Freddy Vale CC BY-NC-SA 4.0

PRVC has been described as an adaptive targeting mode in which the ventilator uses a feedback mechanism on a breath-by-breath

basis to dynamically regulate the delivered pressure and achieve the desired tidal volume target. PRVC attempts to combine the advantages of variable flow from pressure control mode with the guaranteed minute ventilation of VC volume control mode.

Table 5.7.1 Basic Settings in PRVC

Summary of Basic Settings in PRVC

In PRVC, you will directly **set**:	In PRVC, the ventilator will **set/ determine**:
• Ideal Body Weight and Sex • Respiratory Rate • Tidal Volume Target • T_i • FiO_2 • PEEP • Maximum Pressure Limit • Alarms	• Flow • Driving Pressure

For more information on PRVC, watch this video:

One or more interactive elements has been excluded from this version of the text. You can view them online here: https://ecampusontario.pressbooks.pub/mcvresource/?p=1445#oembed-1

Video: "Modes of mechanical ventilation: Pressure Regulated Volume Control (PRVC)" By Society of Mechanical Ventilation [17:56] *Transcript Available*

Adaptive Support Ventilation (ASV)

The **Adaptive Support Ventilation (ASV)** mode allows the clinician to set a target minute volume based on the patient's ideal body weight.

Minute volume, IBW, sex (the ventilator uses this information to determine IBW range), FiO_2, PEEP, Maximum pressure limit. Normal Minute volume is on average, $10\ mL/Kg$ IBW. With this information from the clinician, the ventilator then determines the minimum respiratory rate and tidal volume required to meet the target minute volume and deliver the necessary pressure or breaths to achieve that.

Table 5.7.2 Basic Settings in ASV

Summary of Basic Settings in ASV

In ASV, you will directly **set**:	In ASV, the ventilator will **set**:
• Ideal Body Weight and Sex • Minute Volume Target • FiO_2 • PEEP • Maximum Pressure Limit • Alarms	• Respiratory Rate • Tidal volume • I:E • Driving pressure

ASV combines and alternates a control mode with pressure with a spontaneous mode with pressure based on the patient's level of consciousness and ability to trigger and maintain breaths to ensure minimal work of breathing is required from the patient.

One or more interactive elements has been excluded from this version of the text. You can view them online here: https://ecampusontario.pressbooks.pub/mcvresource/?p=1445#oembed-2

Video: "INTELLiVENT-ASV explained from setup to ventilation, on a real patient" By Hamilton Medical [12:12] *Transcript Available*

Mandatory Minute Ventilation (MMV)

Mandatory Minute Ventilation (MMV) is another example of a closed loop feedback ventilation mode intended to guarantee minimum minute ventilation in which the ventilator recognizes patient effort and contributes with whatever part the patient is unable to maintain, either the respiratory rate or tidal volume. It combines spontaneous breaths initiated by the patient and supported with pressure by the ventilator with volume controlled mandatory breaths.

Mandatory breaths are provided by the ventilator when spontaneous breaths are insufficient and fall below the set minute volume. As spontaneous breathing increases, fewer mandatory breaths are provided. With this ventilation mode, the patient always receives at least the set minimum minute volume (Remember, $V_E = V_T \times RR$). In this mode, pressure and flow are adjusted so that tidal volume is reached with the minimum necessary pressure for the volume controlled breaths based on changes in lung mechanics (compliance and resistance).

Below is a screenshot from a Draeger ventilator showing MMV with volume control and Autoflow feature (Figure 5.7.2).

Figure 5.7.2: VC-MMV + AF with mandatory breath and spontaneous breathing, © Drägerwerk AG & Co. KGaA. Used under fair dealing. All Rights Reserved.

Automode

Automode is available on some ventilators such as the Servo I and Servo U, this ventilation mode introduces an interactive strategy, where the ventilator can switch between pressure control and pressure support, as well as volume control and volume support, based on the patient's needs.

When the patient's respiratory drive decreases and/or mechanics indicate the need for full ventilation with pressure control, the ventilator will deliver breaths in pressure control mode providing a set pressure with each breath. This helps ensure consistent and controlled ventilation when needed, while allowing the patient to breathe spontaneously when they are able. Conversely, when the patient's respiratory mechanics allow for volume control, the ventilator will operate in volume control mode, delivering a set tidal volume with adjusted pressure support. The ability of AutoMode to switch between pressure control and pressure support, as well as volume control and volume support, allows for adaptive respiratory support based on the patient's lung characteristics and changing respiratory needs.

Note: there are other ventilation modes used in clinical practice often classified as Advanced Ventilation Modes, such as Bilevel Ventilation, Airway Pressure Release Ventilation (APRV), High Frequency Oscillation ventilation (HFOV). We will discuss these modes in more detail in a later chapters.

5.8 CONCLUSION

Volume and pressure have a direct relationship. Whichever parameter you adjust will affect the opposite value. When all other settings are kept the same and the lungs are the same, the following statements are true:

Table 5.8.1: Adjusting Volume and Pressure (Other Settings are Kept the Same; Lungs are the Same)

In Volume Control	In Pressure Control
As the volume set is increased, the pressure increases.	As the set pressure is increased, the volume will increase.
If the Pressure reached in the lungs is too high (>30 - $35 cmH_2O$), dropping the volume will help.	If the volume in the lungs is too high, dropping the pressure will help.
If more volume is needed, increase the volume and expect the pressures to climb.	If more volume is needed, increasing the pressure will increase the volume delivered.

Inspiratory time and flow can also impact the pressure and volumes experienced by the lungs. When all other settings are kept the same, the following statements are true:

Table 5.8.2: The Effects of Inspiratory Time and Flow on Volume and Pressure (Other Settings are Kept the Same)

In Volume Control (constant volume to be delivered)	In Pressure Control (constant pressure and/or same target volume)
If the flow is increased, the time to deliver the breath is shorter.	If the inspiratory time is increased, the volume delivered will increase.
If the flow is decreased, the time to deliver the breath is longer.	If the inspiratory time is decreased, the volume delivered will decrease.
If the flow is increased and the inspiratory time is shorter, the pressure needed to hit the same volume will be higher.	If the inspiratory time is increased, the set pressure can be decreased to hit the same volume.
If the flow is decreased and the inspiratory time is longer, the pressure needed to hit the same volume will be lower.	If the inspiratory time is decreased, the set pressure will need to be increased to hit the same volume.

And if the settings on the ventilator do not change, but the lung compliance or resistance in the lungs changes:

Table 5.8.3: The Effect of Changing Lung Compliance and Resistance on Volume and Pressure (No Change in Ventilator Settings)

In Volume Control (no change to settings)	In Pressure Control (no change to settings)
If the compliance of the lungs increases (more stretchy), the pressure used to hit the volume decreases.	If the compliance of the lungs increases (more stretchy), the volume delivered with the set pressure will increase.
If the compliance of the lungs decreases (less stretchy), the pressure used to hit the volume increases.	If the compliance of the lungs decreases (less stretchy), the volume delivered with the set pressure will increase.
If the resistance of the lungs increases, the pressure used to hit the volume increases.	If the resistance of the lungs increases, the volume delivered with the set pressure will decrease.
If the resistance of the lungs decreases, the pressure used to hit the volume decreases.	If the resistance of the lungs decreases, the volume delivered with the set pressure will increase.

5.9 SELF-CHECK

Chapter 5 Quiz

See how well you recall the concepts covered in this chapter by completing the following short quiz.

Don't forget to check your score!

An interactive H5P element has been excluded from this version of the text. You can view it online here: https://ecampusontario.pressbooks.pub/mcvresource/?p=113#h5p-4

CHAPTER 6 | VENTILATION MODES - SPONTANEOUS VENTILATION

Chapter Outline

6.0 CHAPTER OVERVIEW

We have now overviewed control modes of ventilation and talked about when and how to control a patient's breathing. In previous chapters, we discussed how spontaneous modes allow a patient to be much more in control of their breathing, including how often they breathe and how big a breath they take. In this chapter, we will look more closely at spontaneous modes.

This chapter covers:

- Spontaneous modes: Overview and description of pressure support ventilation (PSV), volume support (VS) and other spontaneous modes
- When we use spontaneous modes
- Spontaneous modes settings
- How we choose the correct settings when initiating spontaneous modes
- Review of SIMV, in the context of spontaneous breathing, and why it is not a preferred mode

Application

As we have already learned, spontaneous modes are used whenever a patient has an intact drive to breathe because spontaneous ventilation helps to avoid muscle wastage and asynchrony. This chapter will help you to use spontaneous mode effectively, including choosing appropriate ventilator settings. In addition, this chapter describes Synchronized Intermittent Mandatory Ventilation (SIMV), which was once the preferred approach to ventilation, and explains why it is not used in typical ventilation practice today. Since SIMV is still used by some healthcare centers, it is important that you are aware of this mode and the reasons that it is no longer used so that you are well positioned to advocate for your patients.

Learning Objectives

At the end of this chapter, you will be able to:

1. Identify when to use spontaneous modes
2. Describe typical settings for spontaneous modes
3. Determine appropriate settings based on assessment of a patient.

Key Terms

- muscle atrophy
- weaning
- Pressure Support Ventilation (PSV)
- Volume Support Ventilation (VSV)
- Pressure Augmentation
- Spontaneous Breathing Trial (SBT)
- Minimal settings
- Extubation
- Work of Breathing (WOB)
- Arterial Blood Gas (ABG)
- Endotracheal Tube (ETT)
- Synchronized Intermittent Mandatory Ventilation (SIMV)
- minute volume

Whenever these terms are first introduced in this chapter, they are bolded. However, if you need additional information about a term than what is provided here, you can research it in The Free Dictionary: Medical Dictionary.

6.1 SPONTANEOUS MODES: A REVIEW

Now that we have learned all about control modes, it's time to shift to the other branch of the ventilation "family tree": spontaneous mode:

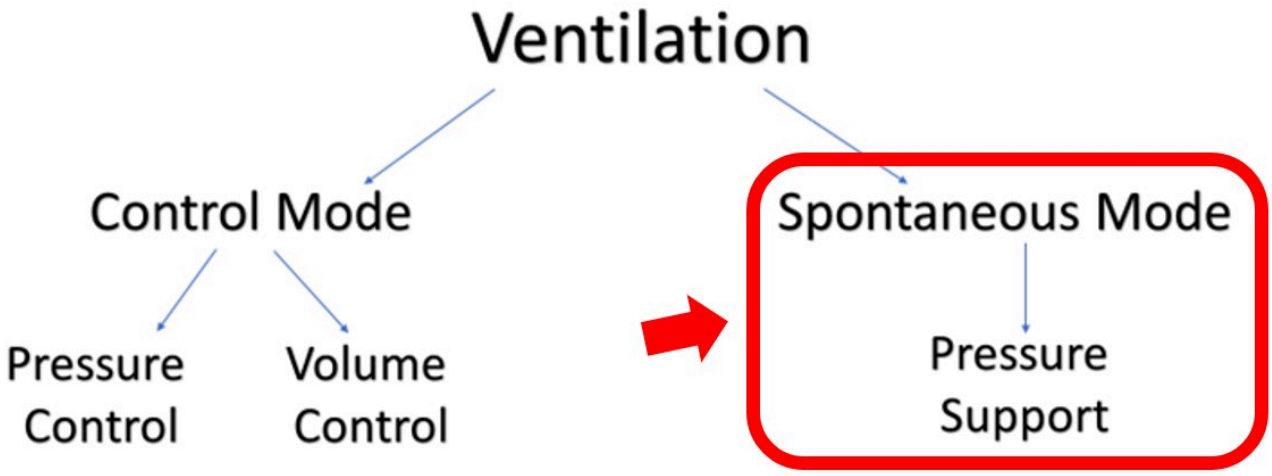


Figure 6.1.1: "Family Tree – Spontaneous" by Amanda Robinson, CC BY-SA

The importance of spontaneous modes should be clear to you after a brief overview of what we have learned so far about mechanical ventilation, including the following key concepts:

- Positive pressure applied to the lungs is damaging
- Mimicking physiologic breathing is much better for the body
- Patient-ventilator asynchrony can happen in control modes because the ventilator is delivering breaths based on what is set and not the patient

In this chapter, we will discuss these points in detail, as well as highlight additional benefits of spontaneous modes.

6.2 SPONTANEOUS MODES VERSUS CONTROL MODES

In a spontaneous mode, the patient will initiate a breath from the ventilator and then the ventilator will "support" the patient. It can help eliminate some of the work that the patient is doing to breath by the application of additional pressure to overcome how much the patient is working. Work of breathing can be assessed subjectively by looking at how fast they are breathing and if they are using accessory muscles to breathe.

A health care worker calibrates a ventilator on the USNS Mercy. Photo by U.S. Pacific Fleet, CC BY-NC 2.0

The key to this process is that the patient must *have an intact neurological drive to breathe*. They trigger their own breaths and have their diaphragm involved in pulling the breath into the lung and then exhaling. Patients also decide the frequency of each breath completely on their own. The ventilator will sense the patient-initiated efforts with a flow or pressure trigger, and deliver a set amount of air to augment the patient's own breath. If the patient's internal respiratory rate is not adequate to manage their CO_2 and oxygen levels, they do not have a fully intact drive to breathe and may need a control mode or they will deteriorate.

Normal physiologic breaths are not all uniform. Most people take a variety of breaths every minute, including frequent sigh breaths, or have variability in their respiratory rate. Control modes do not allow for these variations, and the size of the breath or frequency only changes when the clinician changes the settings. This inflexibility can quickly lead to asynchrony discomfort for the patient. In spontaneous modes, however, the diaphragm is active in triggering, expanding the lungs and relaxing for exhalation once

the breath is the desired volume. These variables are controlled by the neurotransmitters in the brain and can change from breath to breath if needed.

Spontaneous modes of ventilation allow for a return of some of the normal physiologic breathing processes. The chemoreceptors of the brain are active at triggering the initiation of breaths and the diaphragm is triggered to contract. This approach maintains some work in the respiratory muscles and decreases the risk of muscle wasting.

The risk of muscle wasting—or **muscle atrophy**—is key to the necessity of spontaneous modes. Spontaneous modes of ventilation are beneficial as they help maintain involvement of respiratory muscles such as the diaphragm. Remember Chapter 1? We discussed the key involvement of the diaphragm in creating the negative pressure that pulls the breath into the lungs. The diaphragm is the most important muscle involved in a person who is spontaneously breathing. Like all muscles, if not used, the diaphragm starts to weaken.

Control modes of ventilation do not allow the diaphragm to be fully involved in breathing and can cause the diaphragm to atrophy and weaken. This muscle weakening and loss can contribute to patients having difficulty taking over their breathing and being strong enough to breathe on their own. The quicker a health care practitioner can allow a patient to start using their own respiratory muscles to breathe, the better. A fast turnaround will decrease the muscle atrophy that can occur. Spontaneous modes are the number one way that diaphragm use can be maintained and patients will have an easier time taking over their breathing needs without the support of a ventilator.

Though unable to fully eliminate the need for mechanical ventilation, the creation of spontaneous modes of ventilation was key in helping address these concerns.

Key Takeaway

Spontaneous modes are key to minimize damage from positive pressure ventilation, by reducing asynchrony and mimicking a more natural pattern of breathing the patient can control. Spontaneous modes are also instrumental in maintaining respiratory muscle strength, allowing patients to get off the ventilators easier.

Summary of Control vs. Spontaneous Modes

The following table summarizes the key differences between control modes and spontaneous modes:

Table 6.2.1: Key Differences – Control Modes & Spontaneous Modes

	Control Modes	Spontaneous modes
Drive to breathe	No drive to breathe needed	Needs to be breathing spontaneously
Oxygen	Can help give additional oxygen	Can help give additional oxygen
Elevated CO_2 levels	Used to fix abnormal gases	Can only augment normal breathing; not indicated for very high CO_2 levels
Sedation level	Usually requires more sedation	Much more comfortable, less sedation
Work of Breathing	Patient is sedated and ventilator completely overtakes the muscles—they are not actively working	Can give extra support to unload the work of breathing, but the muscles still work a little
Maintaining muscle strength	The diaphragm is not involved in the creation or delivery of the breath	The diaphragm must contract and be involved. Helping maintain muscle strength and decrease muscle wasting
Level of Invasiveness	More invasive, less physiologic	More physiologic; spontaneous modes are used during the transition off of ventilation (weaning)

A pervasive thought in critical care exists that "resting" patients in a control mode is essential to their recovery and is ideal for the first few days of ventilation once the patient has failed enough to require mechanical ventilation. However, more and more research is pointing to the fact that this is not the case. Putting a patient on a control mode often requires large amounts of sedation and in some cases paralytics. Every day that the diaphragm is not working directly contributes to muscle loss and atrophy. Allowing patients to maintain some work in the breathing process has a large impact on decreasing ventilation days and decreasing length of stay in the hospital. As more and more research is being done, control modes are used for increasingly short durations, and spontaneous modes are becoming the standard of care earlier and earlier in the ventilatory process.

6.3 INDICATIONS AND CONTRAINDICATIONS FOR SPONTANEOUS MODES

Indications

The most important indication for a spontaneous mode is a spontaneously breathing patient. Once that baseline has been satisfied, spontaneous modes can be used in two different ways:

1. **Weaning**: A way to decrease ventilator support to work towards getting the patient off mechanical ventilation, the weaning process is started as soon as the patient is starting to improve. Sedation is decreased and patients are encouraged to start triggering breaths to change to a more spontaneous mode. This weaning will continue with slow decreasing of settings until the patient is on minimal settings. Once the patient is on minimal settings, they will be removed from the ventilator and the endotracheal (breathing) tube will be removed. This is referred to as **extubation**.

2. **More comfortable ventilation**: Spontaneous ventilation is a less invasive ventilation mode that will allow for better mimicking of physiologic breathing that the patient can control. This is a less common and more secondary indication for a spontaneous mode.

A spontaneous mode, because it is patient-driven, is not as good as control modes are at fixing imbalances in CO_2 or oxygen levels. Control modes are used to "fix" these imbalances prior to weaning. Therefore, CO_2 and O_2 levels are the primary indications for a spontaneous mode.

Key Takeaway

When indicated, spontaneous modes are preferred to control modes because they more closely mimic natural breathing and keep the diaphragm actively engaged—allowing the patient to do more of the work of breathing and help facilitate getting them off the ventilator. However, spontaneous modes can only be used when a patient has a fully intact drive to breathe and when the patient is stable.

Contraindications

The most important consideration to remember is that most patients that require ventilation are very sick. Their oxygenation is low, and they are not getting rid of CO_2 well enough, causing their organs to start shutting down. When patients are that sick, they need to be stabilized first with a lot of support and treatment before they can start taking over their breathing and breathing on their own again. Spontaneous modes only "help" patients take in breaths on their own terms. If the patient is not stabilized and has abnormal arterial levels of CO_2 or O_2 (described more in Chapter 8 and 9) they are not a candidate for a spontaneous mode.

A good rule of thumb for mode choice is to stabilize them first on control modes, and then, once the patient is stable, decrease sedation enough for them to trigger breaths consistently, and change them over to spontaneous modes.

The presence of any one of the following conditions precludes the use of spontaneous mode:

1. No drive to breathe.
2. Very poor oxygen status/high CO_2 levels requiring full control of the breathing.
3. Unstable vitals requiring advanced medical support—patient should be sedated and control ventilated.

Assessing **work of breathing (WOB)** is a key factor in ventilation. Practicing this skill will improve your eye for key factors to assess work of breathing. WOB is mostly a subjective assessment based on how a patient looks when they breathe, trying to answer questions like:

- How fast are they breathing?
- Are they taking smaller breaths than they should?
- Can you see muscles contracting with every breath?

A helpful mnemonic to assess difficulty breathing is **D**iap**HR**a**GM** (Diaphoresis, Hypoxia, Respiratory rate, Gasping, accessory Muscle use).

Normal breathing is very rhythmic and gentle with very few external changes to a person other than chest and/or belly expansion. In patients who are working, you will see visual signs of distress: diaphoresis, increased respiratory rate, small or sharp breaths similar to gasping, and additional or accessory muscle use for every breath. Additional muscles can be recruited by the patient to try to assist them in taking breaths in. This is a key finding if they are working to breathe.

Increased WOB is noted if the patient's RR is above physiologic normal (usually $>28\ \text{bpm}$ for an adult) and they have *any* of the following:

1. intercostal indrawing
2. substernal retractions
3. scalene muscle use
4. sternocleidomastoid muscle contractions
5. paradoxical breathing—belly and chest moving in a see-saw pattern

To learn more about spotting accessory muscle use for respiration, watch "Using Accessory Muscles of Respiration" from Doctor's Hub:

One or more interactive elements has been excluded from this version of the text. You can view them online here: https://ecampusontario.pressbooks.pub/mcvresource/?p=145#oembed-1

Video: "Using Accessory Muscles of Respiration" By Doctors Hub [3:07] *Transcript Available*

Start watching people breathing around you. Watch your patients! More practice will help you recognize increased WOB immediately.

Case Studies

We have discussed some indications and contraindications for spontaneous mode use. Now, let's put it into practice with a few examples where spontaneous modes would likely be used.

Examples

For each example, ask yourself whether spontaneous modes are being used for (1) weaning or (2) increased comfort.

1. **Patient A** has been ventilated on volume control for three days. His vitals are stable, the health care providers have started decreasing his sedation, and he is breathing above the set Respiratory Rate (RR) on the ventilator. His bloodwork shows normal CO_2 levels. The doctor wants to start weaning, or decreasing ventilation support, and work towards extubating him. **Weaning or comfort?**

    Solution
    A spontaneous mode is indicated here for weaning. This is the most common reason a spontaneous mode is chosen.

2. **Patient B** was intubated yesterday, and her CO_2 levels and vital signs are within normal limits. She has started waking up and is asynchronous with the ventilator in pressure control mode. You tried to change the settings in pressure control, but cannot fix the patient-ventilator asynchrony, with the patient fighting the ventilator and looking very uncomfortable. **Weaning or comfort?**

    Solution
    A spontaneous mode could be used here for both weaning and comfort. Vitals and bloodwork is good. There is no reason to sedate this patient for a control mode. Change to spontaneous mode and start weaning!

3. **Patient C** is awake and alert, but tachypneic (or labouring) with increased work of breathing shown with accessory muscle use while breathing. Blood work is showing slightly elevated CO_2 levels in the blood. Patient C might be a candidate for a spontaneous mode of ventilation since they are breathing on their own but need a little extra "help" to take away that increased work of breathing. If the blood work did not improve with a spontaneous mode, then a control mode might need to be used. **Weaning or comfort?**

Solution

A spontaneous mode could be tried here for comfort. This patient may not be stable enough on a spontaneous mode. Monitoring and bloodwork should be done, and if the results are worse, the patient will need to be sedated and fully ventilated on a control mode.

6.4 TYPES OF SPONTANEOUS MODES

Just like control modes, there are several spontaneous modes available on the market, but unlike control modes, one has gained more popularity over other, and it is mainly used in current practice. **Pressure Support Ventilation (PSV)** is by far the most common support mode utilized in critical care. **Volume Support Ventilation (VSV)** does exist, but it is not often used—most likely due to physiologic variability in volumes being a cornerstone of spontaneous breathing. We will focus on Pressure Support Ventilation as the main spontaneous mode.

We already discussed modes such as Synchronized Intermittent Mandatory Ventilation (SIMV), and Adaptive Support Ventilation, considered "mixed" modes—control and spontaneous.

Another spontaneous mode that is up-and-coming in the respiratory world is **Proportional Assist Ventilation (PAV).** PAV is still a relatively "new" mode, but a lot of ongoing research is increasing its use in the critical care world. PAV utilizes unique breath-initiation and breath-delivery software that has promising implications for how patients breathe on a ventilator.

The COVID-19 pandemic saw a massive increase in ventilated patients, with make-shift ICUs and new clinicians recruited to meet the need. Photo by Mohsen Atayi via Wikimedia Commons, CC-BY 4.0

Pressure Support Ventilation (PSV)

In PSV, every time a patient initiates a breath, the ventilator will push air to reach a set pressure into the patient's lungs while they spontaneously inhale. The patient plays the only role in deciding how often to breathe and can also vary their volumes based on what they need—for example, if they want to take a long and large breath, they can do this at any point. They can also breathe at a more rapid pace with smaller breaths if desired. The only fixed setting that will not change is the amount of support given to them by the ventilator. They cannot get more than the set "help pressure" unless the clinician comes and changes it.

PSV Basic Settings:

- Pressure Support (PS)
- PEEP (every ventilator, every time)
- FiO_2 (every ventilator, every time)

What is NOT set:

- RR (patients must trigger all breaths)
- Flow or I-time (patients can take air in at whatever speed they want and end the breath when they want to. The vent will sense when inspiration and expiration start)

FiO_2 and PEEP are set on every ventilator—it is no different in spontaneous modes. In addition, in PSV, the amount of pressure support (PS) is the singular central setting that a clinician must set. The pressure support should be set at an amount that will allow the patient's work of breathing to remain within normal limits. If they are getting worse, then they are "failing" and may need more support or a change back to a control mode of ventilation.

During pressure support ventilation, each breath will be patient triggered, pressure limited and flow cycled. When we observe the waveforms in pressure support ventilation, notice some similarities with pressure control ventilation. The difference is that every breath in pressure support is patient triggered and the cycle variable is flow, as opposed to time as in pressure control.

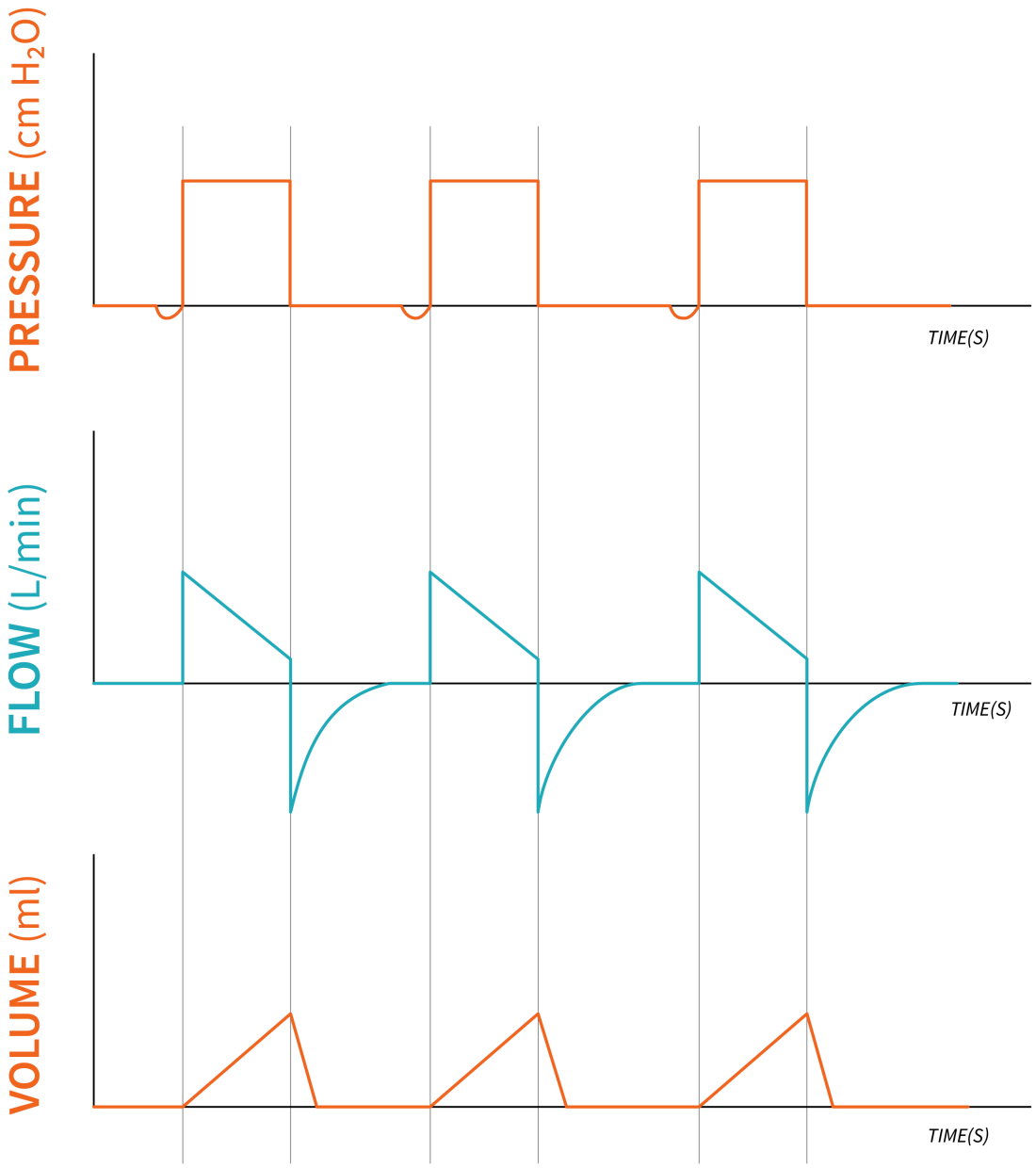


Figure 6.4.1: "Pressure Support Ventilation Diagram" by Freddy Vale, CC BY-NC-SA 4.0

Key Takeaway

Any time you connect a patient to a ventilator, you always set FiO_2 and PEEP. In a spontaneous mode, the patient is in control of how often they breathe, therefore no RR is set. In PSV, you set a "helping" pressure that is delivered with every patient-triggered breath. A volume is not set: the patient pulls whatever volume they want/can based on their strength and pathophysiology. Under-support can cause high respiratory rates and small volumes. Over-support can cause low respiratory rates and high volumes.

Watch this short video for summary of features and graphics Pressure Support Ventilation.

One or more interactive elements has been excluded from this version of the text. You can view them online here: https://ecampusontario.pressbooks.pub/mcvresource/?p=148#oembed-1

Video: "Pressure Support Ventilation Curves" By The Critical Care Practitioner [2:30] *Transcript Available*

6.5 PRESSURE SUPPORT: SETTINGS AND HOW TO SET THEM

Normal pressure supports fall between 5 - $20\ cmH_2O$ depending on the patient's needs. In most cases, the pressure support is never lowered below $5\ cmH_2O$. The minimum of $5\ cmH_2O$ is widely considered as necessary to overcome the resistance that has been added artificially by the endotracheal tube.

Object Lesson

Being intubated is akin to "breathing through a straw". Imagine trying to breathe for longer than a couple minutes—in and out—through a straw. It might become difficult after a while to get the air in fast enough to fill your lungs adequately.

Photo by Monstera, CC0

The added pressure support of $5\ cmH_2O$ gives a little "push" to the air to help get the air in quicker and negate the added difficulty the endotracheal tube adds. This low limit is often termed "minimal settings" and therefore, we do not go below $PS\ 5\ cmH_2O$ unless instructed by a physician.

Since PSV is meant to allow the patient to initiate their own breaths at their own speed, monitoring how they are breathing is the first step in monitoring tolerance. As patients are tiring out or experiencing increased WOB, their respiratory rates normally increase and their tidal volumes may be insufficient for their physiologic needs. These effects can be seen quite soon after any changes are made. If the respiratory rate climbs outside the physiologic averages $(12$ - $25\ bpm)$, this may be indicative of a patient not tolerating the support level given. They may benefit from increasing the set pressure support.

In addition, look at the tidal volumes the patient is getting. If they are a lot lower than the normal tidal volumes $(6$ - $8\ mL/Kg)$ for their ideal body weight, this also is indicative of needing additional support. Additional indicators that the patient may require more support from the ventilator are the heart rate and blood pressure. If there is an increase by 20% from the initial baseline when the change is made, this may indicate the patient is not tolerating the change.

It is important to remember that some patient populations have higher respiratory rates that fall higher than

textbook normals. There may be specific cases where patients are allowed to maintain high respiratory rates.

Once the pressure support is increased, the patient can be observed for improvements in their work of breathing, a slowing of their respiratory rate or an increase in their tidal volumes. ABGs should also be considered to ensure that the patient is improving.

Conversely, if the pressure support is higher than needed, this can be seen in respiratory rates that may be too low (less than $12\ \text{bpm}$) or tidal volumes that fall above safe volume ranges of $4 - 8\ \text{mL/Kg}$. Refer to ideal body weight (IBW) calculations to check appropriate tidal volumes. Pressure support should be titrated up or down quickly within a few minutes until tidal volumes are within the normal range for the patient's IBW and respiratory rates are within normal limits. Pressures should not be left higher than necessary for a long time, as this can lead to over-support, or in extreme cases could result in asynchrony or missed breaths.

Note: If the patient is pulling large volumes and you decrease the pressure support, do not go lower than the set minimum of $5\ \text{cmH}_2\text{O}$. Once you get this far, leave it. The pressures experienced by the lungs are very low and safe. Even if the tidal volume is still too high, this is an indication the patient may be ready for extubation, removal of the endotracheal tube (ETT), and discontinuation of mechanical ventilation.

Pressure Support Initial Settings

Table 6.5.1: Pressure Support Initial Settings

Setting	Starting Point	Additional Instructions
PEEP	Use the same PEEP that was utilized on the previous control mode. or Start at PEEP $5\ \text{cmH}_2\text{O}$ and titrate up if needed to assist with oxygen delivery by $1 - 2\ \text{cmH}_2\text{O}$. ***NOTE:*** *$5\ cmH_2O$ is the minimum, PEEPs higher than $10\ cmH_2O$ should be cleared with a physician.*	Changes to PEEP should be done in tandem with FiO_2 and approximately every 30 minutes. PEEP changes take time for the impacts to be seen, as changes to distending pressure recruit collapsed alveoli over time.
FiO_2	Use the same FiO_2 as the previous control mode. **or** Start at 100% and wean down quickly to SpO_2.If the patient did not require high oxygen before initiation, start at 50% and titrate up or down to target SpO_2 $92 - 99\%$.	Changes to FiO_2 can be done within 3-4 minutes after watching SpO_2. You want to give the minimum amount of FiO_2 possible to target SpO_2 within the normal range.
PS (Pressure Support)	A starting point of $10\ \text{cmH}_2\text{O}$ is reasonable with titrating up or down within a few minutes after watching the RR and V_T the patient is getting and ensuring they are within normal limits as described above.	*Note: minimum PS of 5 to overcome ETT resistance. Do not go below this level unless instructed by a physician.* Always check a PS change with an ABG within 30-60 min to ensure the patient is tolerating it.
Trigger, exp %, other settings	25 by default. Can be adjusted to allow for shorter or longer exhalation	These settings are defaulted to fit 95% of patients.

Let's look at some cases together!

Case Study A

A patient has been ventilating on AC/VC (volume control). The clinicians have been weaning the sedation and want to try changing the patient to a spontaneous mode to start weaning the patient off mechanical ventilation. The volume control settings are as follows:

- $RR = 14$ bpm (patient breathing above at a rate of 18 bpm total)
- $V_T = 380$ mL $(6$ mL/Kg$)$
- $\text{PEEP} = 8$ cmH_2O
- $\text{FiO}_2 = 0.40$

Is this patient a candidate for PSV?

Solution

Yes! They are triggering above the set RR and have an intact drive to breathe. Before switching to PSV, the practitioner should ensure the patient's CO_2 levels are within normal limits by checking the ABG. If the ABG is normal and the patient has stable vital signs, then the patient is deemed a candidate.

What settings do you start with?

Solution

Change the patient to pressure support ventilation by switching the mode. Before accepting changes, select the chosen settings for PEEP, FiO_2 and PS. For this patient, initial PSV settings could be as follows:

- $\text{PEEP} = 8$ cmH_2O (same as previous control mode)
- $\text{FiO}_2 = 0.40$ (same as previous control mode)
- $\text{PS} = 10$ cmH_2O.

Don't forget: Every patient has their own "normal." Before changing to PSV, look at their WOB, RR $(18$ bpm$)$ and vital signs (heart rate and blood pressure) for a baseline.

What do you do now?

Solution

Watch the patient for about 5 minutes. What is the RR? What are the tidal volumes? What is their WOB? What are their vitals?

The clinician should target RR 12 - 25 bpm, tidal volumes: around the same as they had on AC/VC. Watch for signs of increased WOB and watch the vitals for any changes 20% or greater than baseline. If patient is getting worse, increase the PS by 1 - 2 cmH_2O and watch again if an improvement. If their tidal volumes are higher than when in AC/VC or their RR is on the low range of normal, try decreasing the PS value by 1 - 2 bpm and assess the same

things above. Remember! We want to set the lowest tolerated PS that keeps the patient breathing comfortably without increased WOB.

The patient seems to be tolerating the change. What now?

Solution

Repeat an ABG after 30 minutes and if everything looks good, consider decreasing the PS within a few hours until you get to the minimum settings—at which point the patient may be a candidate for extubation.

Case Study B

A patient with profound hypoxia is being intubated electively due to increasing oxygen requirements. After initial intubation, before being put on the ventilator, they are waking up and triggering breaths. The respiratory therapist (RT) tried to put the patient on pressure control ventilation (ACPC), but the patient is double triggering and uncomfortable. Instead of fully sedating the patient, the RT attempts to put them on Pressure Support Ventilation to see if they can keep breathing on their own.

Is this patient a candidate for PSV?

Solution

Maybe. They have a drive to breathe and would probably be more comfortable on pressure support, but their hypoxia might mean they will not be stable on a spontaneous mode. ABGs and oxygenation status must be monitored closely and if they deteriorate, the patient should be sedated and fully ventilated for better control over their ventilation.

What settings do you start with?

Solution

Since the patient is being intubated for mostly oxygen issues, FiO_2 should be started at 1.0 with a higher PEEP—if acceptable by the physician—probably $10\ \mathrm{cmH_2O}$ to start. The PS can be started at $10\ \mathrm{cmH_2O}$ and then adjusted up if the RR is high or the tidal volume is low.

What do you do now?

Solution

FiO_2 can be titrated down by watching the SpO_2 over the next few minutes and the patient's vitals and RR and WOB should be monitored to see if higher pressure support is needed. If their volumes are too high and they are tolerating PS, the PS can be decreased until the volumes are within normal $4 - 8\ \mathrm{mL/Kg}$, as long as they do not show signs of intolerance such as increased WOB (remember the DiapHRaGM mnemonic).

The patient seems to be tolerating. Now what?

Solution

ABGs should be checked for overall tolerance. If there are any changes to patient status, settings should be increased to the previous better-tolerated level; or, if there is a *significant* change, consider sedation and full control ventilation if the ABGs are poor.

It is important to remember that, because pressure support is a patient-triggered breath, changing the level of pressure support will not necessarily change the volumes or respiratory rate of the patient. Sometimes, the patient's neurological control to breathe will remain the same. Here are two common situations that fit this scenario:

Example #1: Diabetic Ketoacidosis (DKA)

These patients tend to be tachypneic—breathing rapidly—with a high **minute volume** (amount of air exhaled in a total minute, as calculated by multiplying the tidal volume by the respiratory rate). Some clinicians might see tachypnea and assume the patient has an increased work of breathing. Increasing the pressure support will not decrease the respiratory rate at all. These patients will most likely remain tachypneic no matter what mode or setting you have them in. This is not a sign of failure. Instead, the tachypnea is a symptom of the pathophysiology of their illness.

Example #2: Oversedation/Waking up from anaesthesia

Patients that have had a large amount of sedation or narcotics usually breathe very slow with large tidal volumes. At times, their respiratory rates can be lower than $10\ \text{bpm}$ with tidal volumes well above their IBW range. Decreasing the pressure support to minimum pressures can sometimes still reveal tidal volumes that are too large and respiratory rates that are below normal range. These effects will normally resolve when sedation wears off. Consider decreasing sedation further if able, or allow the patient to wake up more.

Back Up Ventilation!

For safety reasons, in PSV ventilation (or any other spontaneous mode), back up ventilation must be available in case the patient stops breathing. This is often called Apnea Ventilation on most ventilators and entails a controlled mode that the ventilator will initiate if the patient does not trigger a breath in a specific time interval. By default, most ventilators use a time interval of 20 seconds.

6.6 A WORD OF CAUTION ON PSV

There are two "flaws" in pressure support ventilation that may lead to difficulty weaning patients. These flaws exist in how the breath is triggered, or the "on-switch", and in the "off-switch" for the pressure delivery.

Once the ventilator senses a patient-triggered breath, the pressure support turns on and pushes air into the lungs at the set "help" pressure. The trigger is usually a set threshold of flow that patient must pull in to trigger a breath. A common flow trigger is anywhere from 2 - 5 Lpm. When you break that amount of flow down to the fraction of a second it takes for the ventilator to sense a breath, it is essentially mere milliliters of air that the patient needs to pull in. Very little effort is needed to cause the pressure to start delivering.

This pressure then continues to be delivered until the "off-switch" is activated to tell the ventilator that the patient is done breathing. Most variations of PSV use a flow decay percentage to trigger the ventilator to stop delivering pressure. This means that the ventilator must sense a decrease in the air being delivered to the lungs. Normally, this decrease occurs naturally as the diaphragm is at its lowest point and the lungs are filling. As they fill, the air the patient is pulling in naturally slows down. This slowdown would stop the pressure and then the lungs would passively exhale. Remember, pressure delivered to a closed system always equals volume delivered.

Because of this fact, there are some instances where patients are able to "ride" PSV like a control mode. They trigger the vent with a small pull to start the pressure support but then allow the vent to deliver the air with the set pressure "help" without needing to pull any air themselves. The pressure will continue to push air in—similar to a controlled breath—until that flow decay is reached. Because the lungs will spontaneously decrease in compliance as they fill, this means that at the top of filling, the compliance pushes back to the point that it will naturally slow the air coming in. This triggers the expiratory phase on its own without the patient driving the breath. Essentially, the patient only has to satisfy a small inspiratory trigger or mere milliliters of air to essentially get "free pressure" until their lungs fill enough to terminate the breath on their own. High pressure supports are more likely to cause this scenario.

Object Lesson

Think of this scenario like a continuous motion ball pendulum, commonly called a Newton's Cradle. With very little effort at the beginning, a movement is generated that continues with no effort.

One or more interactive elements has been excluded from this version of the text. You can view them online here: https://ecampusontario.pressbooks.pub/mcvresource/?p=155#oembed-1

Video: "Newton's Cradle – Incredible Science" By IncredibleScience [0:52] *Transcript Unavailable*

One easy way to decrease the risk of pressure support acting like a control mode is to minimize the support pressure to ensure over-support is not possible. If the pressure support is kept on the lower side, then the patient will not be able to get an adequate tidal volume by passively allowing the pressure to push air into their lungs. Then, they will have to actively pull air in themselves to achieve the tidal volume their neurological drive to breathe demands. You can target minimum pressure support by ensuring the patient's RR is not too low (less than 12) and that the volumes they are pulling in are within their normal range (no more than $8\ \mathrm{mL/Kg}$ of IBW).

6.7 VOLUME SUPPORT VENTILATION

Volume Support Ventilation is another spontaneous mode available on some ventilators. The principle of this mode is similar to that of pressure regulated volume control (PRVC). However, to use volume support, the patient must have an intact drive to breathe. With this mode, pressure support is provided with a target volume set. Based on phase variables, a volume support breath can be described as follows: patient triggered, volume targeted and flow cycled.

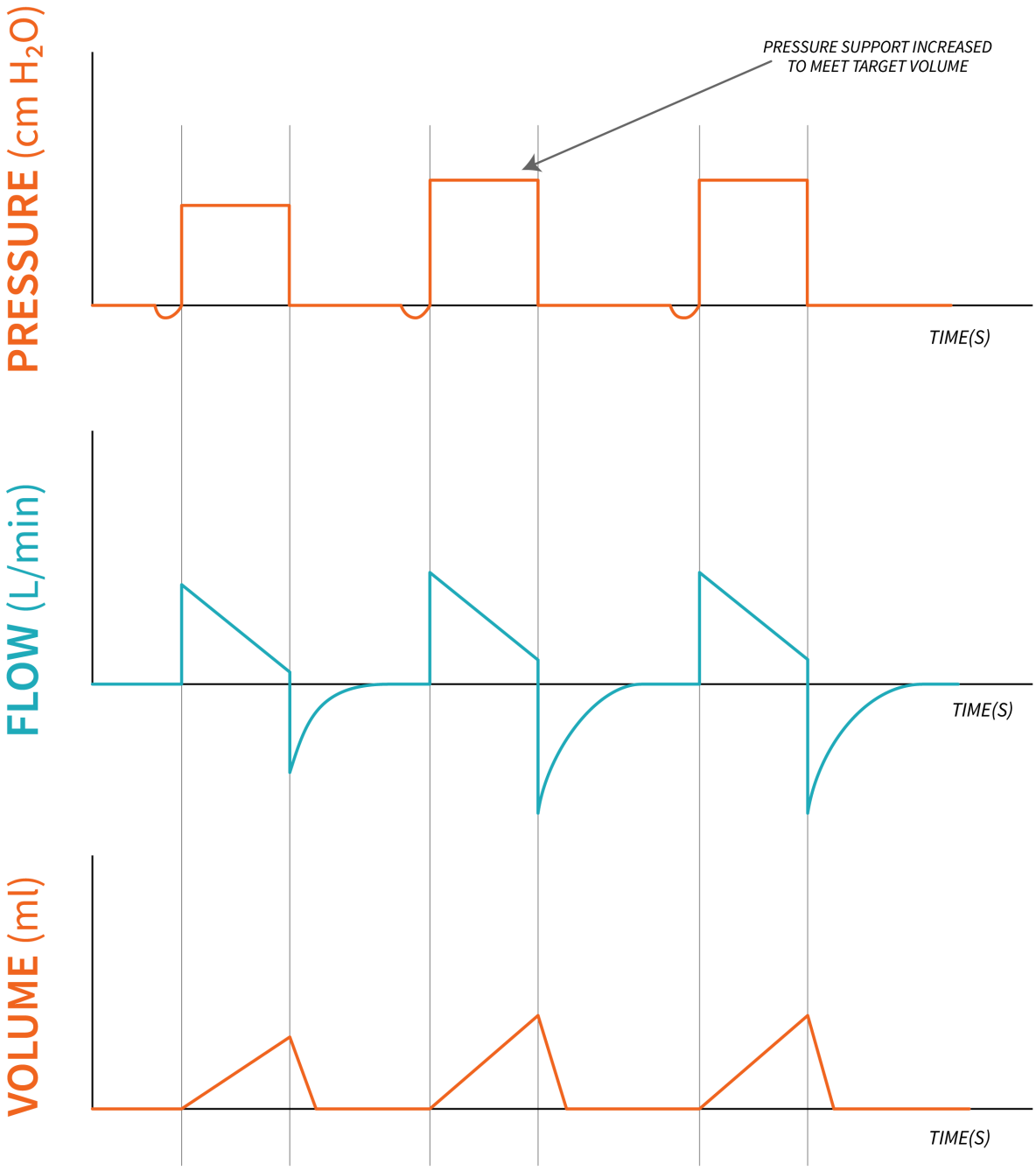


Figure 6.7.1: "Pressure Support with Target Volume" by Freddy Vale, CC-BY-NC-SA

The ventilator will adjust pressure over several breaths in order to achieve the target volume set. For example, if the tidal volume measured is below the target volume set, the ventilator will increase pressure.

As with any spontaneous mode, back up ventilation must be set appropriately.
Watch this video for a review on volume support ventilation principles and settings:

One or more interactive elements has been excluded from this version of the text. You can view them online here: https://ecampusontario.pressbooks.pub/mcvresource/?p=1588#oembed-1

Video: "Volume Support – Basics" By Mike's RT [5:23] *Transcript Available*

6.8 VOLUME ASSURED PRESSURE SUPPORT/ PRESSURE AUGMENTATION

Volume Assured Pressure Support

Volume Assured Pressure Support (VAPS) is described as a dual control mode within a breath, and may also be called Pressure Augmentation depending on the ventilator. We are discussing this mode under spontaneous modes, as a key component of using it in practice, as the patient has the ability to trigger breaths and has a consistent drive to breathe.

Ventilation starts with a patient-triggered pressure support breath, but volume is targeted, so pressure will be adjusted to achieve the target volume. Breaths are patient-triggered, volume targeted, pressure supported and flow cycled. The difference between this mode and other volume targeted dual modes, is that the ventilator makes adjustments within the same breath, as follows: the clinician typically sets a V_T target, a minimum RR, set pressure support, inspiratory flow and sensitivity.

If V_T is met before inspiratory flow drops to the preset level for cycle, then the breath is simply a PSV breath. If monitored V_T is less than the target volume, the ventilator maintains the set flow until the volume is delivered and the ventilator switches from flow cycle to volume cycle.

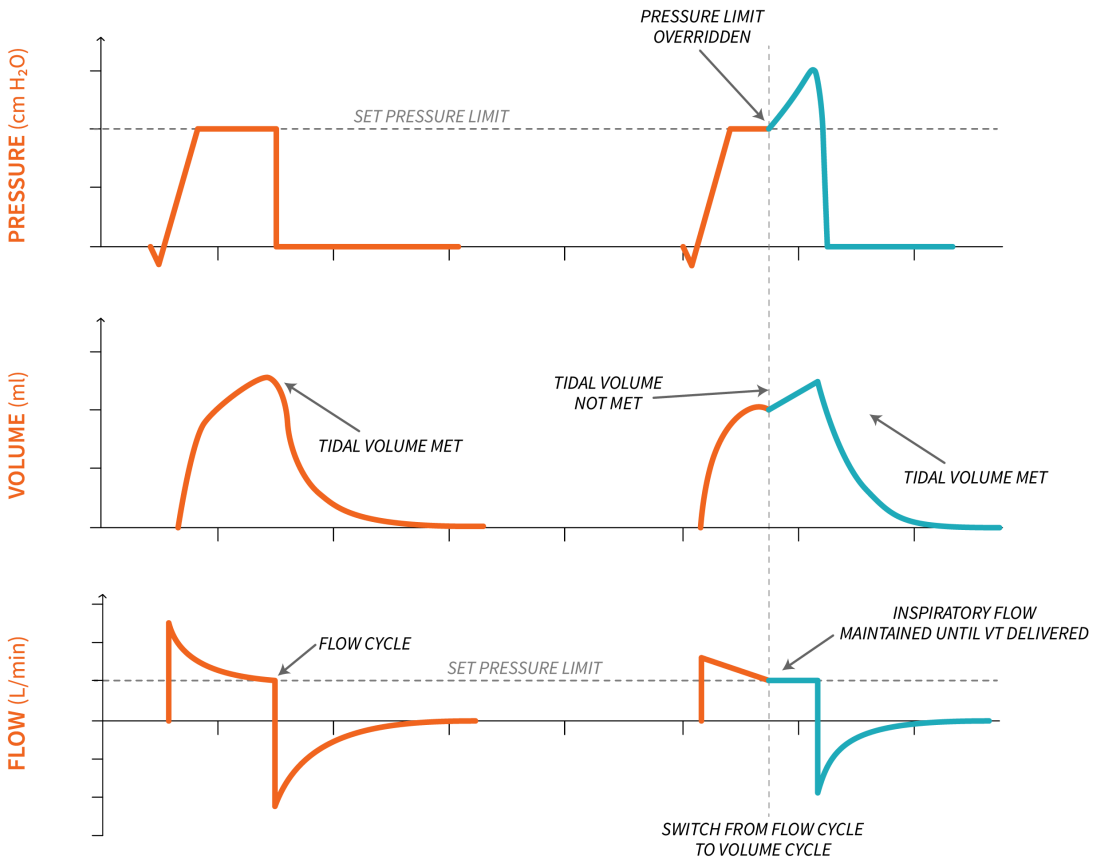


Figure 6.8.1: "Volume Assured Pressure Support" by Freddy Vale, CC BY-NC-SA 4.0

6.9 PROPORTIONAL ASSIST VENTILATION

Proportional Assist Ventilation (PAV) is another ventilation mode that relies on the patient's ability to initiate breaths. With this approach, support provided by the ventilator is adapted according to the patient's effort. The clinician establishes the level of assistance (ranging from 5% to 95%) that the ventilator will provide. This assistance represents the work performed by the ventilator.

During PAV, the patient's generated flow and pressure are continuously measured and assistance is provided in proportion to their effort. Additionally, lung mechanics, such as resistance and compliance, are monitored and taken into account when delivering support. The ventilator employs an algorithm based on the equation of motion to calculate the appropriate pressure required for assistance.

Key Takeaway

PAV adjusts the level of assistance based on the intensity of patient inspiratory effort, in real time, which may prevent or reduce patient-ventilator asynchrony.

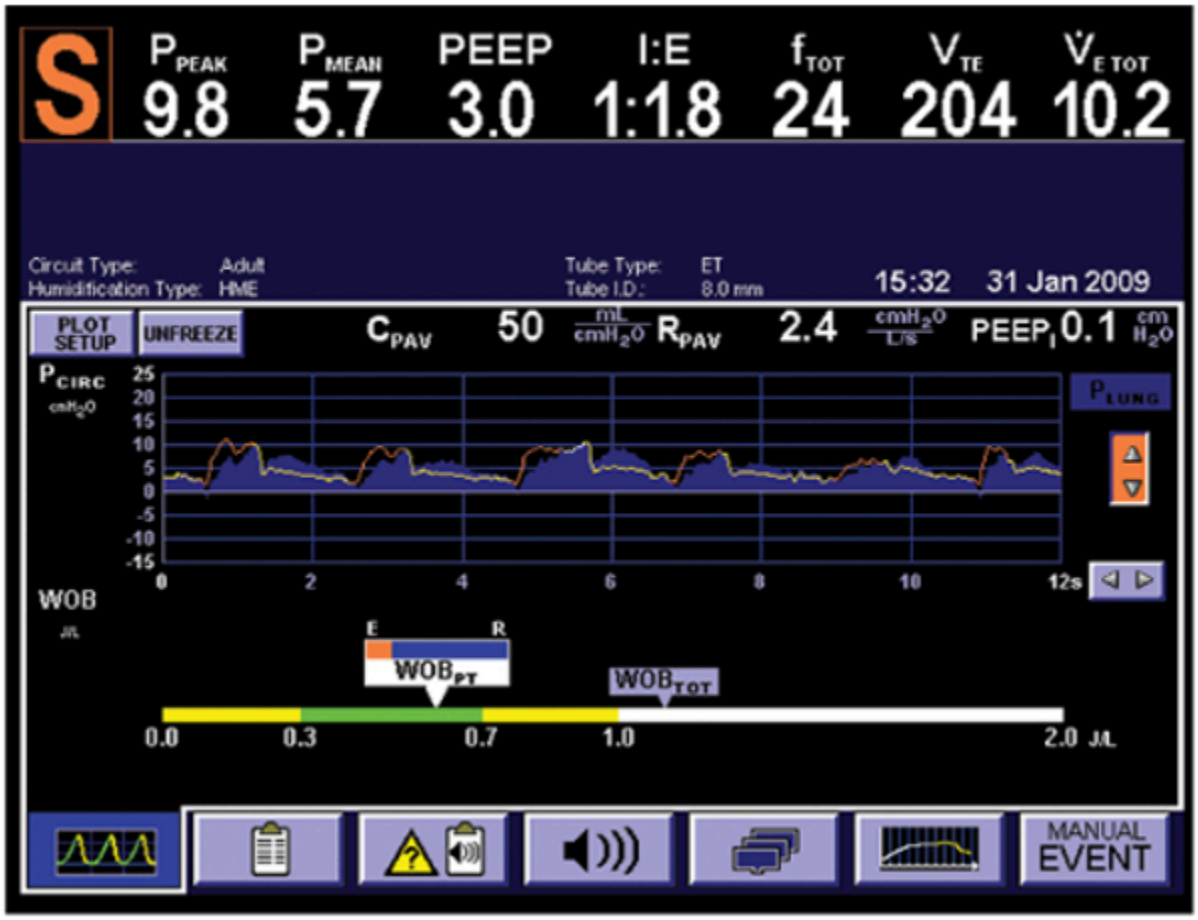


Figure 6.9.1: Puritan Bennett PAV+ Software by Medtronic. Used under fair dealing. All Rights Reserved.

6.10 CONCLUSION

Now that you have reached the end of Chapter 6, you should understand spontaneous modes in much more depth, including when they are used. You should be able to describe pressure support ventilation (PSV) and understand why it is the most popular spontaneous mode, as well as Volume support ventilation and other spontaneous modes.

Three nursing students practice intubation. These students understand that mechanical ventilation and other respiratory treatments are critically important in health care and save countless lives. Intubation by UMDNJ School of Nursing, CC BY-NC 2.0

Review

Please review the following key points from this chapter:

- Spontaneous modes are always used for patients who have a spontaneous drive to breathe
- Patient populations for which spontaneous modes are used include:
  - Tachypneic or tired patients that may require ventilatory support to unload their work of breathing, and
  - Patients who are weaning from the ventilator.
- PSV can range from 5 - $20\ cmH_2O$, and the setting is consistent for every breath unless changed by the health care professional.
- PS of approximately $5\ cmH_2O$ is considered to be “minimal settings.”
- Other spontaneous modes available.

6.11 SELF-CHECK

Chapter 6 Quiz

See how well you recall the concepts covered in this chapter by completing the following 5-question quiz.

Don't forget to check your score!

An interactive H5P element has been excluded from this version of the text. You can view it online here: https://ecampusontario.pressbooks.pub/mcvresource/?p=164#h5p-7

CHAPTER 7 | MECHANICAL VENTILATION INDICATIONS

Chapter Outline

7.0 CHAPTER OVERVIEW

Overview

Mechanical ventilation is supportive therapy in patients with respiratory failure, a life-saving intervention that provides respiratory support to patients who are unable to maintain adequate oxygenation and ventilation on their own or with other supportive therapies including supplemental oxygen. By understanding the indications for mechanical ventilation in respiratory failure, respiratory therapists can accurately assess patients, make informed decisions, and provide appropriate care.

In this chapter, we will discuss why and when mechanical ventilation is needed, as well as assessment criteria to determine this need. We will explore supplemental oxygen requirements and its impact on the body. The content of this chapter briefly describes causes of respiratory failure in the context of mechanical ventilation requirements, but it is not meant to replace a detailed pathophysiology textbook or resource

Application

Understanding how mechanical ventilation works will equip us as respiratory therapists to protect the lung from unwanted damage. We will also learn the basics of selecting appropriate oxygenation for a ventilated patient.

Here are some key reasons why understanding the indications of mechanical ventilation is important:

Patient Safety: Identifying the appropriate indications for mechanical ventilation ensures that patients who require respiratory support receive it promptly. It helps prevent delays in initiating mechanical ventilation, which can be detrimental to patients with severe respiratory failure.

Optimal Ventilation and Oxygenation: Respiratory failure can result in inadequate oxygenation and ventilation, leading to organ dysfunction and life-threatening conditions. Understanding the indications for mechanical ventilation allows us, as respiratory therapists, to provide the appropriate level of support to optimize gas exchange and maintain adequate oxygenation.

Proper Patient Selection: Not all patients with respiratory failure require immediate mechanical ventilation. Understanding the indications helps respiratory therapists differentiate between patients who may benefit from non-invasive ventilation, supplemental oxygen, or other respiratory interventions, and those who require invasive mechanical ventilation.

Monitoring and Assessment: Respiratory therapists play a crucial role in monitoring patients on mechanical ventilation. Understanding the indications helps them assess the patient's response to therapy, make necessary adjustments, and detect potential complications associated with mechanical ventilation.

Collaborative Decision Making: Respiratory therapists work as part of a multidisciplinary team, including physicians, nurses, and other healthcare professionals. Understanding the indications for mechanical ventilation allows them to actively contribute to discussions and collaborate effectively in making decisions regarding the initiation, management, and weaning of mechanical ventilation.

Optimizing Patient Outcomes: Proper use of mechanical ventilation based on appropriate indications can improve patient outcomes, including reduced morbidity, mortality, and complications associated with respiratory failure.

Learning Objectives

At the end of this chapter, you will be able to:

1. Describe different types of respiratory failure and need for mechanical ventilation
2. Describe various causes of respiratory failure
3. Specify healthy oxygen saturation levels and how to titrate supplemental oxygen optimally

Key Terms

In this chapter, you will learn about all of the following key terms. These terms will be used throughout this book, so it is important to take the time to master them and practice your recall often.

- hypoxic failure
- hypoxemia
- hypercapnia
- ineffective drive to breathe
- Fraction of Inspired Oxygen (FiO_2)
- room air
- supplemental oxygen
- saturation of hemoglobin in the blood (SpO_2)
- hemoglobin
- oxygen content (CaO_2)
- oxygen free radicals
- titrate

Whenever these terms are first introduced in this chapter, they are bolded. However, if you need additional information about a term than what is provided here, you can research it in The Free Dictionary: Medical Dictionary.

7.1 WHY DO WE NEED MECHANICAL VENTILATION - RESPIRATORY FAILURE TYPE I

Respiratory failure occurs when the cardio-respiratory system is unable to provide adequate tissue oxygenation and/or CO_2 removal. This condition can become life threatening, and it is important for respiratory therapists to recognize impending respiratory failure and take appropriate action. Respiratory Failure can be classified as acute or chronic.

Key Takeaway

Going back to the analogy of lungs as balloons, it is a simple concept that too much volume or pressure applied to a balloon can over stretch or pop the balloon. Keep this concept in mind when learning the basics of mechanical ventilation. Lung protective strategies will be explored further in later chapters.

Acute Respiratory Failure (ARF) has a sudden onset and may present with hypoxemia alone, or hypoxemia may be accompanied by hypercapnia. Acute Respiratory Distress Syndrome (ARDS) is a severe form of Respiratory Failure, and will be discussed later. Chronic Respiratory Failure is often caused by chronic lung disease or neuromuscular disease.

Reasons for mechanical ventilation due to *respiratory* failure can fall into two main categories:

1. **Hypoxemic respiratory failure** (also called hypoxia or Type I respiratory failure) is the inability to oxygenate the body adequately. Issues with the lungs, circulating blood, or the heart can result in inadequate oxygen being delivered to the organs and tissue. If not corrected, this low oxygen can cause organ failure and irreversible damage.
2. **Hypercapnic respiratory failure** (also called pump failure or Type II respiratory failure) is characterized by high CO_2 levels and can occur because of compromised breathing or other illnesses in the body. If CO_2 increases to a level the body cannot clear by increasing breathing, mechanical ventilation is indicated. An **ineffective drive to breathe** can also occur if there is an injury to the brain or neurological control to breathing, leading to respiratory failure. It can occur with spinal or nerve injuries as well, where the triggers of ventilation do not result in an intact signal to breathe. These cases may require mechanical ventilation to take over the impaired drive to breathe.

Respiratory Failure

Acute Respiratory Failure is usually sudden in onset and is caused by temporary conditions such a pneumonia.

Chronic Respiratory Failure is caused by chronic disease such as Chronic obstructive Pulmonary Disease.

Chronic Respiratory Failure can also be caused by neuromuscular diseases or chest wall deformity.

Integrity of the complex system that is required for breathing and maintenance of normal O_2 and CO_2 levels compatible with metabolic demand is essential to all organ functions. Imagine this complex interaction between respiratory and cardiovascular systems as a chain with multiple links. Problems can arise at any level of this chain. Failure of one or more of links in the chain can lead to development of ARF.

The first three components of this chain represent the "ventilatory pump" and are responsible for alveolar ventilation. A problem with any of these components results in CO_2 retention which will eventually compromise oxygenation, limiting alveolar gas exchange. Problems with the next components, airway and lung tissue, result in inadequate oxygenation but normal CO_2 elimination, assuming the ventilatory "pump" function is maintained. The last link in the respiratory system chain, representing gas transport in circulation, can result in inadequate O_2 delivery to the tissues and CO_2 removal. Cases of shock severe anemia and/or hemoglobin dysfunction could also cause ARF. Often, multiple chain links are broken at the same time, resulting in ARF of multiple causes. For example, a patient who suffered a head injury, (involving the central nervous system) lung contusion (involving lung tissue) and hemorrhagic shock (involving circulation) may develop ARF from multiple causes without appropriate intervention.

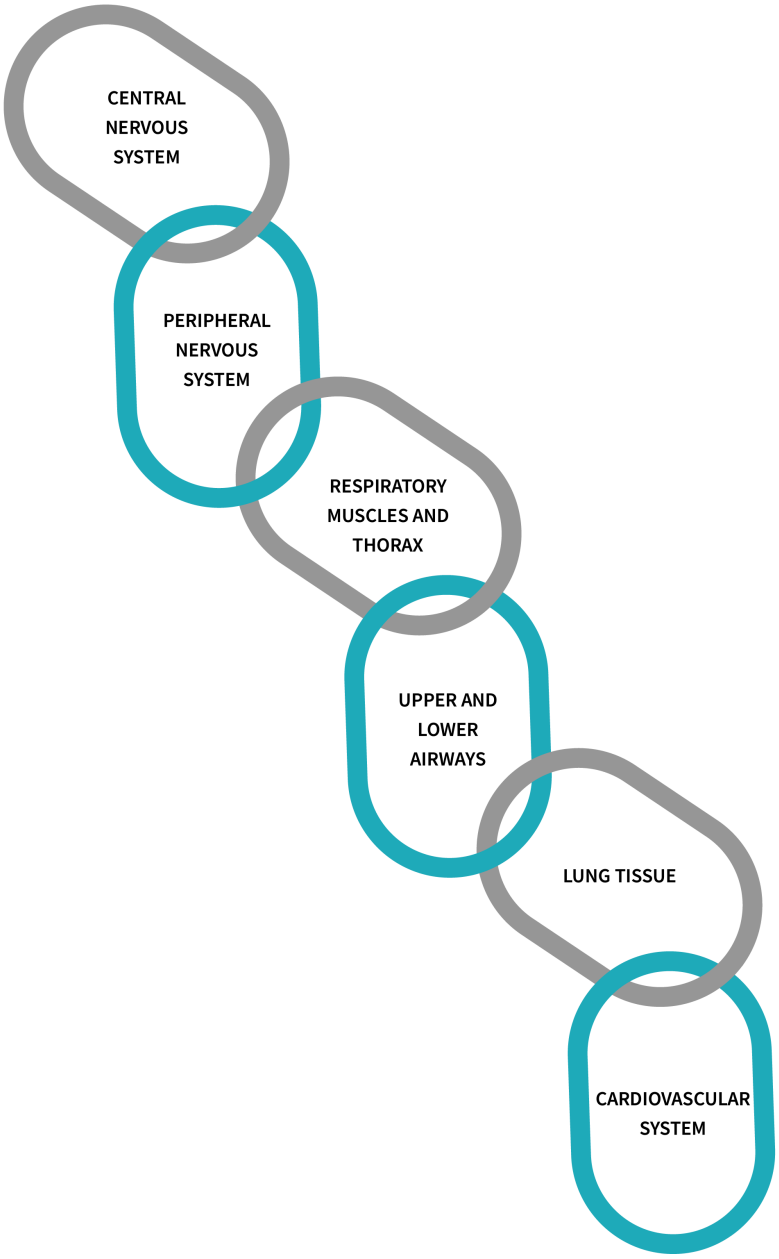


Figure 7.1.1: "Respiratory System Chain" by Freddy Vale, CC BY-NC-SA 4.0

Remember when we learned about those chemoreceptors in the brain which are sensing oxygen and CO_2 levels? Anything that interrupts the signal from the brain to the lungs, or if the signals or subsequent breaths are not adequate for what the body needs, will require ventilation. The body naturally wants to keep the CO_2 levels within a desired range and wants to take in enough oxygen to feed the body. When a patient gets sick or has any type of organ dysfunction, this can quickly cause a disruption in the balance of

the body by increase the oxygen consumption or increasing the CO_2 production of the cells. When this happens, the body will try to compensate, but this compensation can only go so far. As patients get sicker, there may come a point where the oxygen levels are too low (hypoxic failure) or CO_2 levels are too high for the lungs to compensate (hypercapnia), and mechanical ventilation is necessary to help restore healthy levels of CO_2 or oxygen.

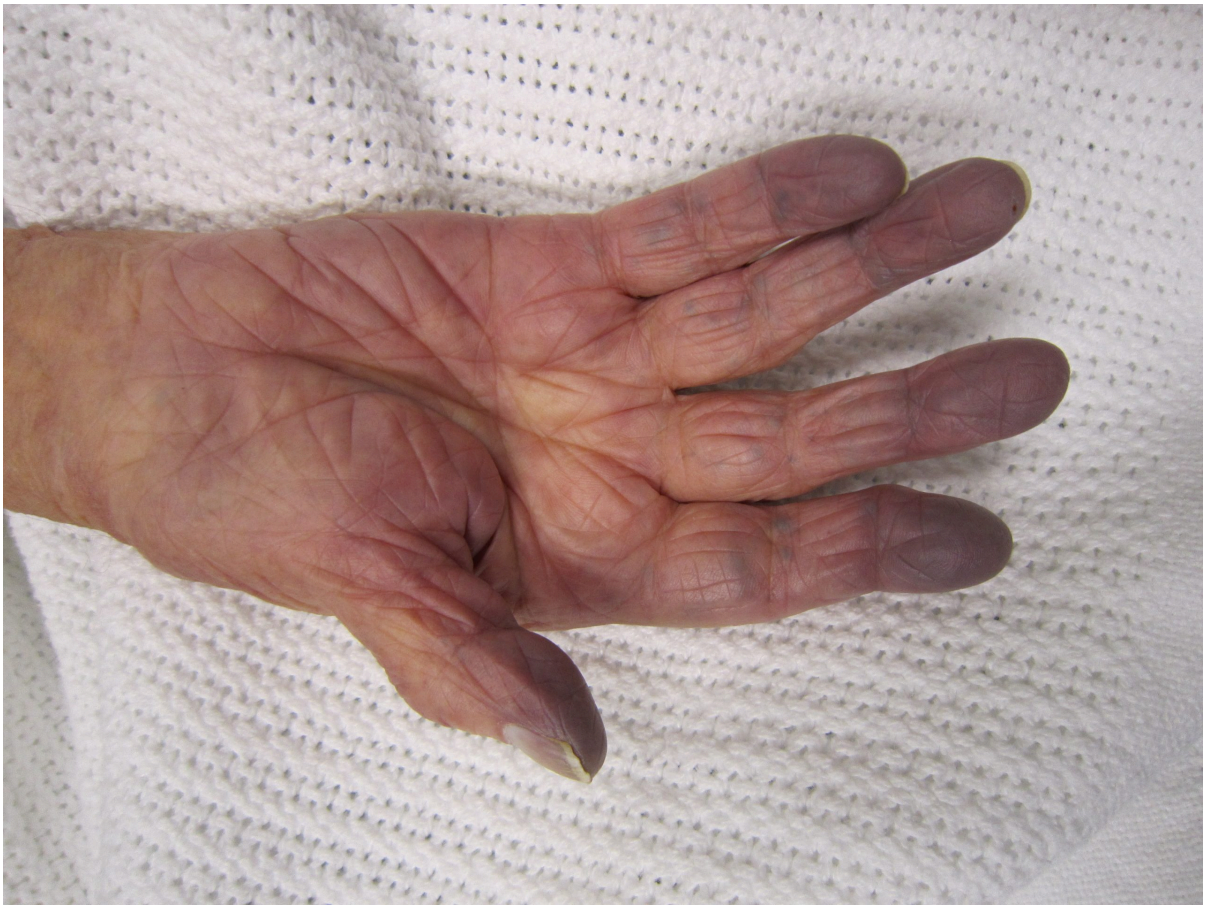


Figure 7.1.2: Photo by Doc James, CC BY-SA 3.0

Cyanosis will occur if hypoxic failure is left untreated. Mechanical ventilation will prevent oxygen levels from falling to this low level. The health care provider must always act quickly to treat hypoxic failure.

Hypoxemic Respiratory Failure or Type I Respiratory Failure

The main causes of Hypoxemic respiratory Failure (Type 1) are Ventilation/Perfusion (V/Q mismatch) or shunt, Alveolar hypoventilation, diffusion impairment, and decreased inspired oxygen.

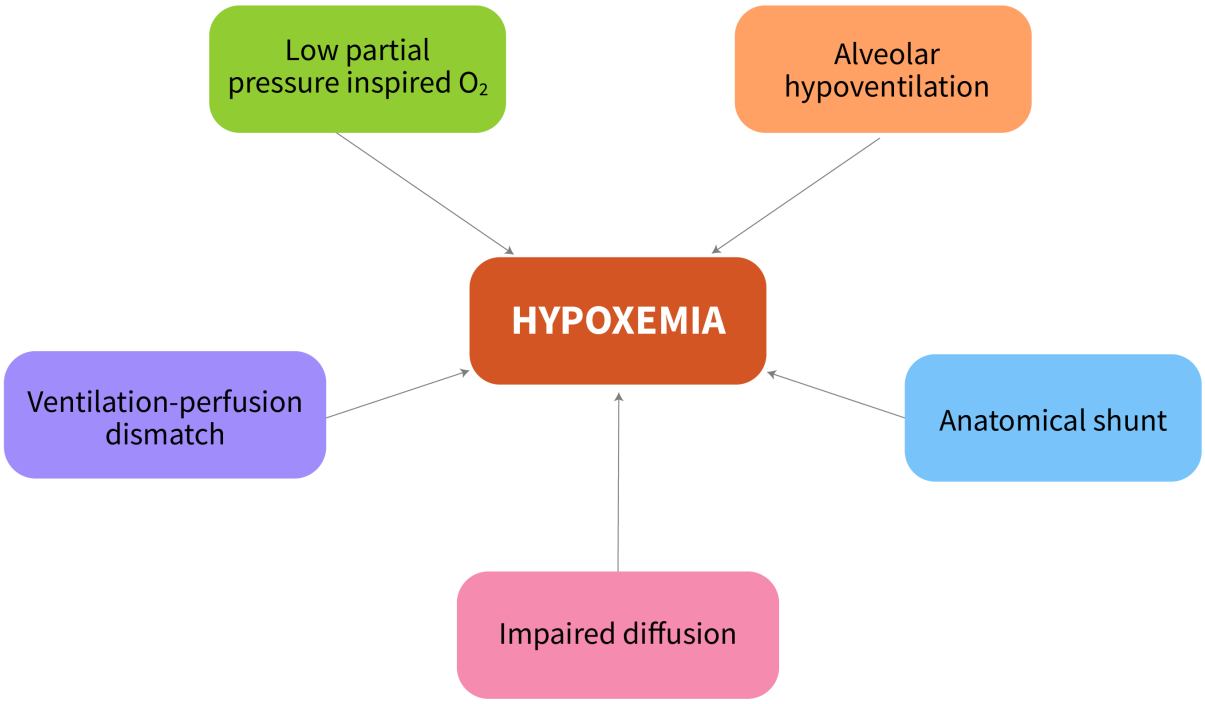


Figure 7.1.3: "Causes of Type 1 ARF" by Freddy Vale, CC BY-NC-SA 4.0

Ventilation/Perfusion Mismatch

Ventilation/Perfusion mismatch ($\frac{V}{Q}$ mismatch) is often a difficult concept to understand, so let's review it as a cause of hypoxemic respiratory failure. The optimal $\frac{V}{Q}$ ratio is 1, when alveolar ventilation matches blood flow available for gas exchange. However, ventilation and perfusion are not evenly distributed and matched in all areas of the lung. Under normal physiological conditions, in the upright individual, there is more ventilation than blood flow at the apex of the lung and more blood flow than ventilation at the base due to gravity $\left(\frac{V}{Q} > 1\right)$. In an individual in supine position, there will be more blood flow to the posterior areas of the lung (dependent areas, $\frac{V}{Q} < 1$).

Represented in Figure 7.1.4 blow:

Key Takeaway

The greatest amount of perfusion and ventilation is in the dependent areas of the lungs.

At the apex of the lung = VA/Q ratio is increased

At the middle of the lung= VA/Q = 1

At the base of the lung = VA/Q ratio is decreased

- Zone 1: At the top of the lung = VA/Q ratio is increased
- Zone 2: At the middle of the lung= VA/Q = 1
- Zone 3: At the bottom of the lung = VA/Q ratio is decreased

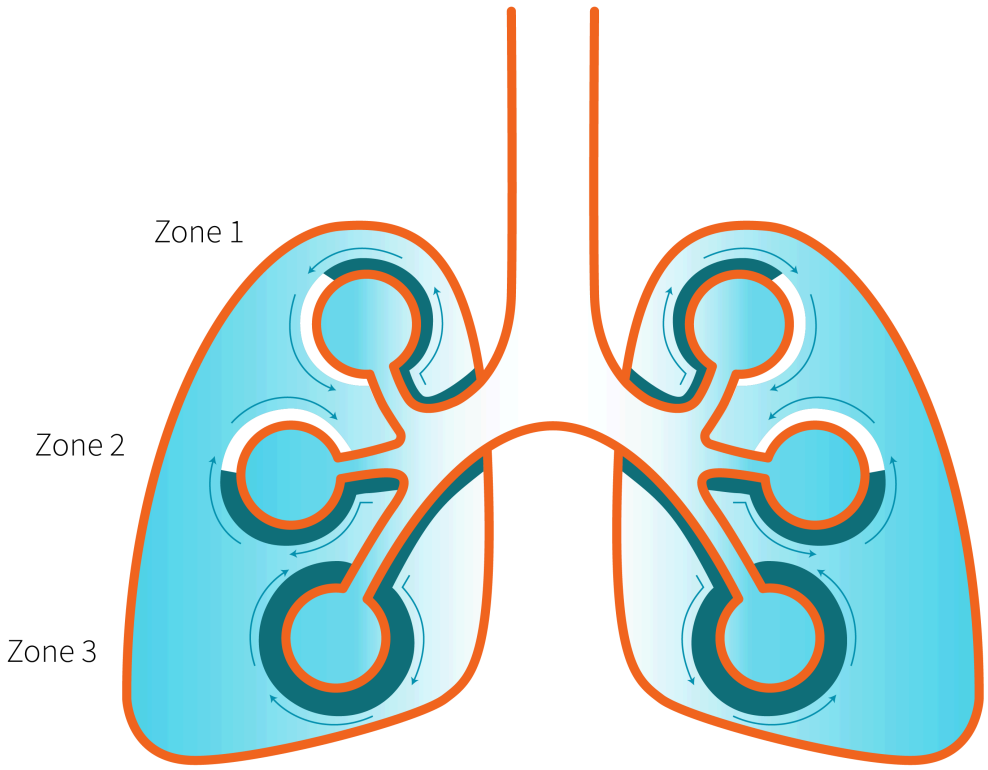


Figure 7.1.4: "Lung Zones" by Freddy Vale, CC BY-NC-SA 4.0

Disease process often disrupts this physiological balance, leading to hypoxemia. $\frac{V}{Q}$ varies based on lung zone and body position.

This is often used to improve oxygenation in severe ARDS, where patients are placed in prone position, so that anterior areas of the lungs become dependent and available to increased blood flow for gas exchange.

A **shunt** is an extreme form of $\frac{V}{Q}$ mismatch, when there is no ventilation to match blood flow. $2\% - 3\%$ of the blood supply is normally shunted (anatomic shunt). Shunt increases, in certain abnormal conditions such as right to left shunting of blood flow though cardiac septal defects. Physiologic shunt leads to hypoxemia in abnormal conditions such as atelectasis or pulmonary edema.

Shunt does not respond to supplemental oxygen, because gas exchange is not available $(\frac{V}{Q} = 0)$.

Object Lesson

Ventilation/perfusion mismatch usually responds to O_2 therapy, while shunt does not.

Watch this video for a quick review of $\frac{V}{Q}$ mismatch:

One or more interactive elements has been excluded from this version of the text. You can view them online here: https://ecampusontario.pressbooks.pub/mcvresource/?p=43#oembed-1

Video: "V-Q Mismatch" By macrophage [4:23] *Transcript Available*

Alveolar Hypoventilation

While alveolar hypoventilation is discussed mostly in the context of hypercapnic respiratory failure, it can also lead to hypoxemia. Alveolar hypoventilation refers to a decrease in the amount of fresh air reaching the alveoli, resulting in reduced O_2 intake and increased CO_2 retention.

When alveolar hypoventilation occurs, the exchange of oxygen and carbon dioxide in the alveoli is compromised. This can lead to a decrease in the O_2 content within the alveoli, resulting in lower O_2 levels available for transfer into the bloodstream. As a result, the O_2 saturation of arterial blood can decline, leading to hypoxemic respiratory failure.

Diffusion Limitations

Diffusion is defined as the movement of air and oxygen across the alveolar capillary membrane along a pressure gradient. Diffusion limitation is seen in conditions affecting gas exchange, such as interstitial lung disease, where destruction of interstitium leads to impaired gas exchange. Destruction of alveolar tissue can also impair diffusion. Abnormalities in pulmonary circulation may lead to impaired gas exchange due to reduced blood flow available.

Decreased Inspired Oxygen

Hypoxic respiratory failure caused by decreased inspired oxygen occurs when the body oxygen requirements are greater than available oxygen. Knowing that the concentration of oxygen is the same anywhere on the planet, 21%, this low inspired oxygen

is seen at high altitude due to decreased atmospheric pressure, or in situations where the body's increased oxygen requirements go unrecognized for prolonged periods of time without appropriate intervention (i.e. provide O_2 at higher FiO_2).

Check Your Knowledge

An interactive H5P element has been excluded from this version of the text. You can view it online here: https://ecampusontario.pressbooks.pub/mcvresource/?p=43#h5p-11

7.2 WHY DO WE NEED MECHANICAL VENTILATION? - RESPIRATORY FAILURE TYPE II

Hypercapnic Respiratory Failure or Type II

Let's review the analogy of respiratory system as a chain.

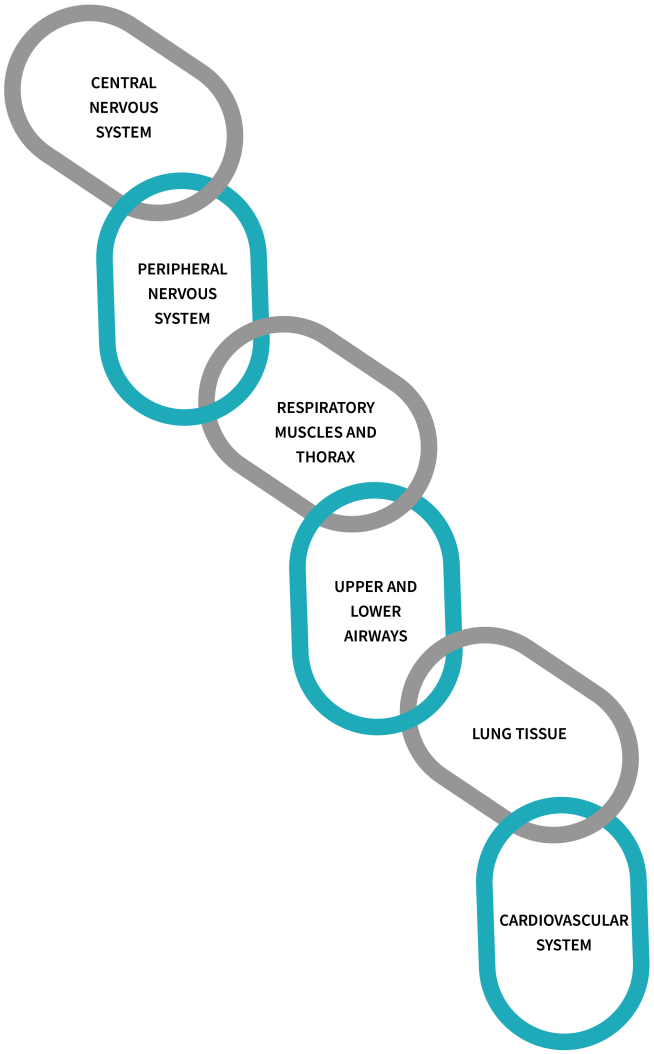


Figure 7.2.1: "Respiratory System Chain" by Freddy Vale, CC BY-NC-SA 4.0

A problem with any of the first three components of this chain results in CO_2 retention. In hypercapnic respiratory failure, the lungs fail to remove an adequate amount of CO_2 due to inadequate alveolar ventilation, leading to an imbalance in the blood gases. Hypercapnic respiratory failure, also known as Type II ventilatory failure or pump failure, can be acute or chronic and is characterized by increased levels of CO_2 in the blood, leading to respiratory acidosis.

Ventilation Calculation Example

For normal values:

$RR = 12 - 20/\text{min}$ (avg: $12/\text{min}$)

$V_T = 400 - 700$ ml
OR $(6 - 8\text{ml/Kg})$;(avg: 500 ml)

$$V_E - V_T \times RR$$
$$V_E = 500 \text{ ml} \times 12 \text{ /min}$$
$$V_E = 6000 \text{ ml/min}$$
$$V_E = 6 \text{ L/min}$$

In a healthy respiratory system, the lungs help eliminate CO_2 by exchanging it with oxygen during breathing. Ventilation refers to the movement of air in (O_2) and out of the lungs (CO_2). A detailed patient assessment will identify problems with ventilation. Parameters used to identify ventilatory problems will be described later in this chapter. Ventilation is simply described as the product of tidal volume and respiratory rate.

$$V_E = RR \times V_T \text{ where}$$
$$V_E = \text{Minute Ventilation},$$
$$V_T = \text{Tidal Volume},$$
$$RR = \text{Respiratory Rate (Frequency)}$$

Object Lesson

Arterial level of CO_2 ($PaCO_2$) and Alveolar ventilation are inversely related. Higher alveolar ventilation leads to increased oxygen concentration and decreased CO_2 concentration in the alveoli. Conversely, lower alveolar ventilation results in decreased oxygen concentration and increased CO_2 concentration in the alveoli.

* *$PaCO_2$ is the single best index of alveolar ventilation.*

Check Your Knowledge

What causes Hypercapnic respiratory failure?

Answer

The main causes of hypercapnic respiratory failure are impairment of the central nervous system, including neuromuscular transmission, mechanical defect of the thorax, and respiratory muscle fatigue.

Alveolar Ventilation and Dead Space

While minute volume provides an overall estimate of ventilation, alveolar ventilation provides information on the effectiveness of ventilation. Alveolar ventilation is determined by substracting physiological dead space (V_{Dphys}) from tidal volume:

$$V_A = (V_T - V_{Dphys}) \times RR$$

V_A = Alveolar Ventilation, V_T = Tidal Volume, V_{Dphys} = Physiologic dead space, RR = respiratory rate (or frequency)

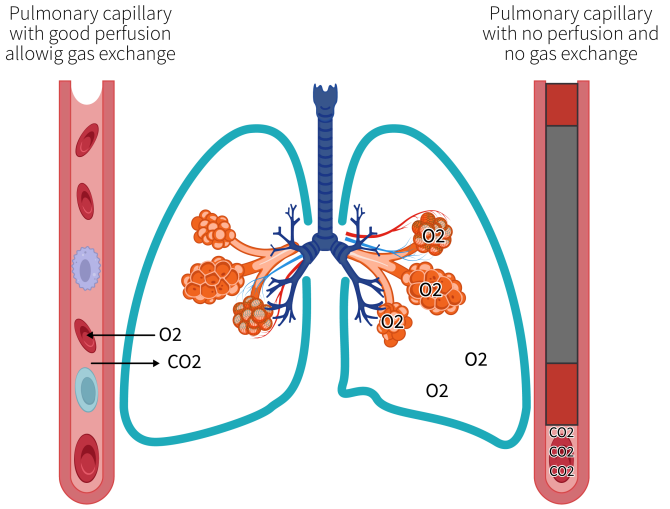


Figure 7.2.2: "Dead Space Ventilation Diagram" by Yvonne Drasovean, CC BY-NC-SA 4.0

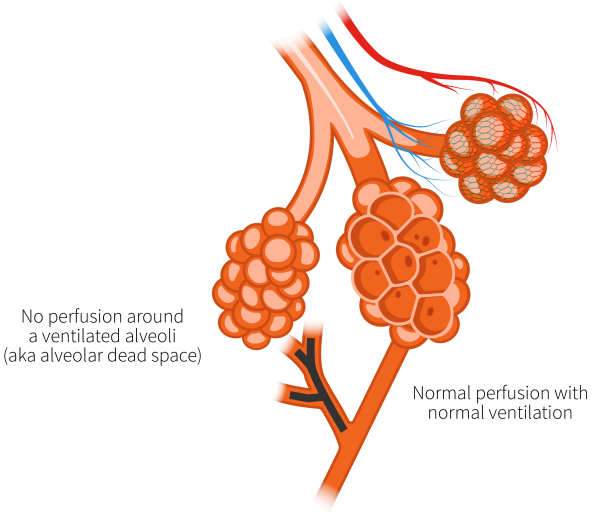


Figure 7.2.3: "Alveolar Dead Space" by Yvonne Drasovean, CC BY-NC-SA 4.0

Object Lesson

Recall from Chapter 1, physiologic dead space is the sum of anatomic dead space and alveolar dead space. Under normal circumstances, anatomic dead space is almost the same as physiologic dead space. In dead space causing diseases, however, physiologic dead space increases.

**Dead space refers to ventilated alveoli, that are not perfused.*

When an increase in dead space ventilation is suspected, the dead space to tidal volume ratio (V_D/V_T) can be determined with a simple calculation at the bedside using the Bohr-Enghoff equation. V_D/V_T is a physiological measurement that assesses the efficiency of gas exchange in the lungs. It represents the proportion of each breath that is occupied by dead space, which is the volume of air that does not participate in gas exchange. This calculation requires collection and analysis of arterial blood gases and measurement of exhaled CO_2.

$$\frac{V_D}{V_T} = \frac{(\mathrm{P_aCO_2} - \mathrm{P_ECO_2})}{\mathrm{P_aCO_2}}$$

Where:

$\frac{V_D}{V_T}$ represents the physiological dead space to tidal volume ratio.

$PaCO_2$ is the partial pressure of carbon dioxide in arterial blood.

P_ECO_2 is the partial pressure of carbon dioxide in the mixed expired gas.

$\frac{V_D}{V_T}$ Example

Normal $\frac{V_D}{V_T}$ varies between $20\text{-}40\%$

Inserting normal values in the equation:

$$\frac{V_D}{V_T} = \frac{(\mathrm{P_aCO_2} - \mathrm{P_ECO_2})}{\mathrm{P_aCO_2}}$$

$$\frac{V_D}{V_T} = \frac{40 - 28}{40}$$

$$\frac{V_D}{V_T} = 0.30 \text{ or } 30\%$$

Check Your Knowledge

Can pulmonary embolism contribute to dead space ventilation?

Answer

Yes. When a pulmonary artery becomes obstructed by a blood clot, the blood flow to that specific lung region is compromised, leading to a ventilation-perfusion (V/Q) mismatch with ventilated alveoli that are not perfused, and increased dead space.

Significant reduction in alveolar ventilation, with subsequent hypercapnia, can result from impairment of central nervous system, such as drug overdose, tumors, intracranial hemorrhage with increased intracranial pressure (ICP), or traumatic brain injury. As previously discussed, unless supplemental oxygen is administered, patients experiencing hypoventilation will present with hypoxemia as well. Under normal circumstances, the body has the ability to respond to hypercapnia and hypoxemia by increasing respiratory drive (increasing alveolar ventilation). When the central nervous system is compromised, this response is reduced. These patients will require supplemental oxygen and ventilatory support. Neuromuscular disorders often lead to hypercapnic respiratory failure due to respiratory muscle impairment caused by problems with nerve damage, neuromuscular junction, or muscle damage.

7.3 WORK OF BREATHING AND THE NEED FOR MECHANICAL VENTILATION

In physics, **Work** is described as the force required to move an object over a certain distance.

$$\text{Work} = \text{Force} \times \text{Distance}$$

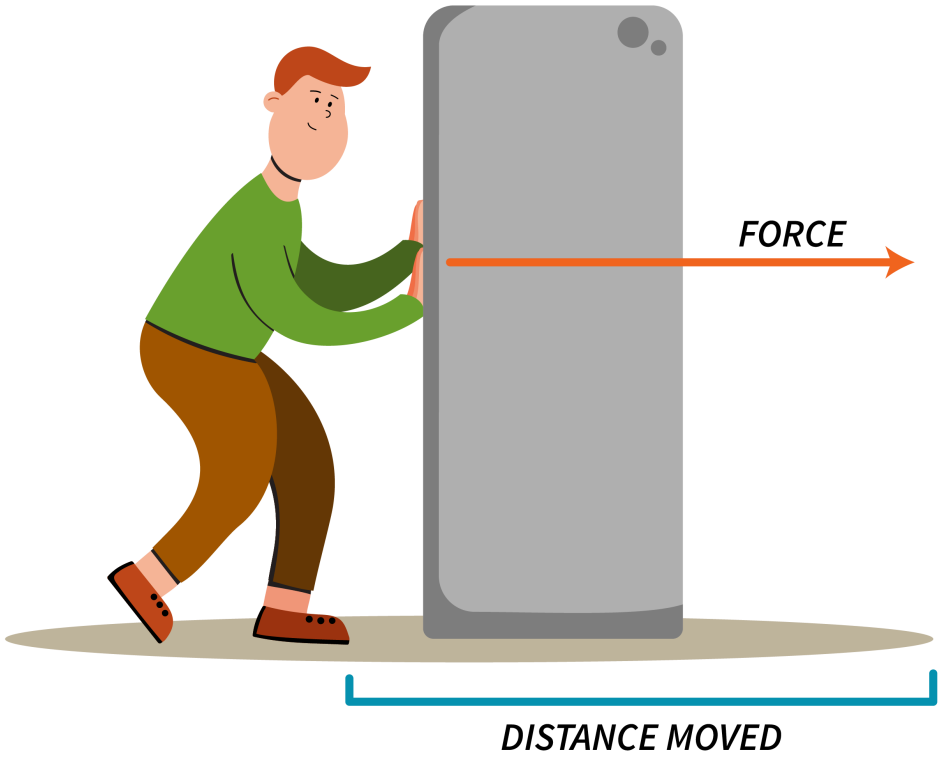


Figure 7.3.1: "Work Demonstration" by Freddy Vale, CC BY-NC-SA 4.0

In respiratory physiology, **work of breathing** (WOB) is performed by the respiratory muscles. WOB can be determined by the pressure required to move tidal volume during the breathing process.

$$\text{WOB} = \text{Pressure} \times \text{Volume}$$

$$\text{WOB} = P \times V$$

The equation above represents the mechanical work performed by the respiratory muscles. The pressure represents the force generated by the muscles, and the volume represents the amount of air moved in each breath.

The respiratory muscles need to overcome elastic and frictional forces that oppose lung inflation (distension), which requires energy. This energy is provided through metabolic work of breathing, which is the amount of energy or oxygen consumption needed by the respiratory muscles to produce sufficient ventilation and meet the metabolic demands of the individual. WOB depends on breathing patterns, both under normal circumstances or disease process.

A graphic representation is helpful in assessing work of breathing. Shaded area represents the work of breathing.

Example: WOB and Breathing Pattern

During exercise, when higher flow is required, an increase in respiratory rate leads to increased frictional forces, and increased WOB.

In individuals with restrictive lung disease (stiff lung) elastic WOB is increased in an attempt to distend the lung. These individuals often adopt a rapid shallow breathing in order to reduce WOB.

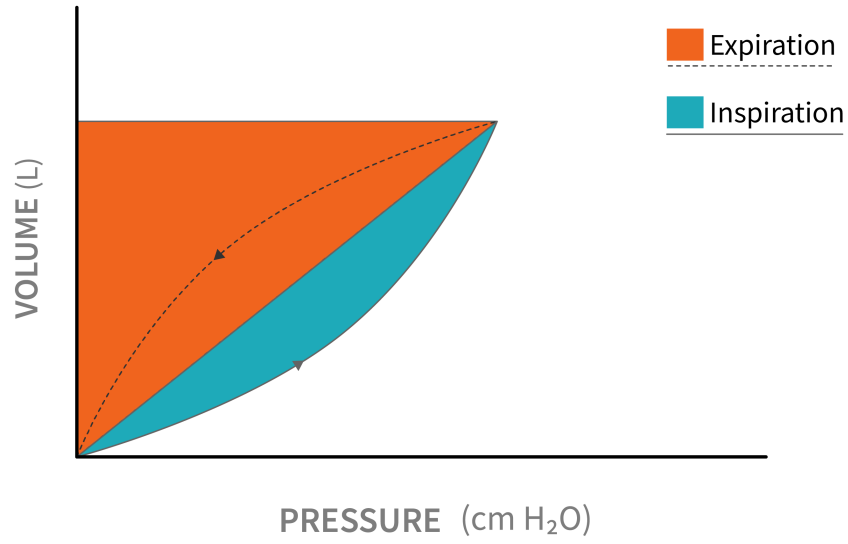


Figure 7.3.2: "Work of Breathing" by Freddy Vale, CC BY-NC-SA 4.0

Key Takeaway

Work of breathing is determined by the pressure-volume characteristics (compliance and resistance) of the respiratory system. Work of breathing will increase in diseases that increase resistance or decrease compliance or when respiratory rate increases.

An increase in work of breathing can lead to respiratory failure secondary to respiratory muscle fatigue and weakness. As respiratory therapists, it is extremely important that we recognize signs of increased work of breathing. Several assessment measurements are available to assess work of breathing and respiratory muscle strength and are discussed below.

Object Lesson

There are several factors that can influence WOB.

- Metabolic requirements. As the body requires more energy, WOB must increase in order to ensure sufficient ventilation.
- The effort required by the respiratory muscles to overcome airflow resistance, enabling air movement in and out of the airways and compliance, to maintain appropriate lung volumes.
- The respiratory rate and pattern. The intensity and speed of contraction in the respiratory muscles determine WOB.
- Presence of V/Q. Mismatching and impaired diffusion resulting from various diseases.

How do we determine increased work of breathing and respiratory muscle fatigue?

Respiratory muscle weakness and fatigue are commonly observed in patients with neuromuscular disease, as well as chronic respiratory conditions like COPD or restrictive lung disease. While this is often a sign of disease progression, it is important to recognize it and promptly address any increase in work of breathing caused by muscle weakness to prevent complications.

The following physiological measurements are commonly used to assess respiratory muscle strength, and degree of work of breathing.

Maximum inspiratory pressure (MIP) is the lowest pressure generated during inspiration with the airway occluded.

Remember, the pressure measured during inspiration is considered negative.

This measurement is sometimes called negative inspiratory force (NIF).

This measurement can be performed at the bedside using a manometer.

Normally, MIP ranges between -50 to $-100\ \mathrm{cmH_2O}$. Generally, an MIP of at least -20 is required to inhale an adequate tidal volume.

A higher than $-20\ \mathrm{cmH_2O}$ MIP signals inspiratory muscles weakness.

Object Lesson

Measuring **MIP** is equivalent with gauging the strength of a person's lung muscles through a fitness test. Just as a fitness test measures the maximum force exerted by muscles, measuring maximum inspiratory pressure assesses the maximum force generated by the respiratory muscles during inhalation.

Photo by Jonathan Borba, Pexels Licence.

Maximum Expiratory Pressure (MEP), sometimes called peak expiratory pressure, is similar to MIP and is a measurement used to assess the strength of the expiratory muscles. It determines the maximum force generated by these muscles during forced expiration. Normal value for MEP is $100\ \mathrm{cmH_2O}$.

Object Lesson

MEP similar to an athlete's final sprint in a race, demonstrating the athlete's leg muscles power and endurance.

Photo by Keith Johnston, Pixabay Licence.

Vital Capacity (VC) is the volume of air that can be exhaled following a maximal inspiration. Similar to MIP and MEP, VC provides information on a person's ability to inhale a large tidal volume in order to produce a strong cough to clear the airway.

Photo by Autumn Mott Rodeheaver, Unsplash Licence

Peak Expiratory Flow provides information on airway resistance and a person's ability to maintain airway patency.

Tidal volume, respiratory rate and minute ventilation are also used often as indicators of respiratory muscle strength. A respiratory rate greater than 30 - 35 bpm is usually an indication of increased work of breathing. Without intervention, it will lead to respiratory failure.

7.4 CONCLUSION

Respiratory failure is a complex and critical condition that occurs when the respiratory system fails to adequately fulfill its primary function of providing oxygen and eliminating carbon dioxide. We have examined the two main types of respiratory failure: hypoxemic and hypercapnic. Hypoxemic respiratory failure is characterized by low oxygen levels in the blood, often due to ventilation-perfusion mismatch or diffusion abnormalities. Hypercapnic respiratory failure, on the other hand, is characterized by elevated carbon dioxide levels, typically resulting from inadequate alveolar ventilation.

We have discussed various underlying conditions that can lead to respiratory failure, including chronic lung diseases, acute respiratory distress syndrome (ARDS), neuromuscular disorders, and chest wall abnormalities. Recognizing the signs and symptoms of respiratory failure is crucial for prompt diagnosis and initiation of appropriate interventions.

The management of respiratory failure involves addressing the underlying cause, ensuring adequate oxygenation, and optimizing ventilation. Oxygen therapy, non-invasive ventilation, and mechanical ventilation may be utilized at different stages depending on the severity and specific needs of each patient. Close monitoring, frequent assessment and recognition of increased work of breathing, and ongoing adjustment of treatment strategies, ranging from oxygen therapy to mechanical ventilation, are essential to optimize patient outcomes.

Review the following measurements discussed in this chapter:

Table 7.4.1 Physiological Measurements

Physiological measurement	Normal Value	Intervention required
$PaCO_2$ (mmHg)	35 - 45	> 55
pH	7.35 - 7.45	< 7.20
Tidal Volume (mL/Kg IBW)	6 - 8	< 5
Vital capacity (mL/Kg)	65 - 75	< 10
Respiratory rate (br/min)	12 - 20	> 35
Maximum Inspiratory pressure (MIP)	-180 - 100	> 20
V_D/V_T	0.25 - 0.40	> 0.6

7.5 SELF-CHECK

Chapter 7 Quiz

See how well you recall the concepts covered in this chapter by completing the following short quiz.

Don't forget to check your score!

An interactive H5P element has been excluded from this version of the text. You can view it online here: https://ecampusontario.pressbooks.pub/mcvresource/?p=59#h5p-13

CHAPTER 8 | PATIENT MONITORING DURING MECHANICAL VENTILATION

Chapter Outline

8.0 CHAPTER OVERVIEW

In the world of critical care, the health care team faces a crucial task—watching over seriously ill patients who need mechanical ventilation. To do this well, we must understand how the body works and how to use monitoring equipment. This knowledge is gained throughout various courses in the respiratory therapy program.

In this chapter, we will explore the ins and outs of this essential process that reflect the efficiency of mechanical ventilation. Our main focus will be on monitoring blood gases and how patients interact with ventilators. Get ready to dive into the heart of patient care, as we uncover the vital aspects of critical care monitoring.

Photo by Alan Levine, CC BY 2.0

Arterial blood gases (ABGs) are the cornerstone of assessing the respiratory status of a patient that may require mechanical ventilation or is already being mechanically ventilated. It is important that all health care providers that use mechanical ventilators have an understanding of ABGs and how to interpret them, in order to identify the issues that the patient is experiencing and know how to fix them. This chapter will introduce ABGs, how to approach an interpretation of ABGs, and the issues that are occurring based on the blood gas.

Patient-Ventilator interaction is another important aspect of the complex monitoring of ventilated patients and it should be closely observed by respiratory therapists.

This chapter will focus on:

- The theory behind ABGs
- Normal ABG values
- How to interpret ABGs
- Assess ventilator graphics
- Assess patient- ventilator interactions and asynchrony

Application

ABGs are a key piece in ventilation, as they are used to identify issues that require ventilation-based treatments, and they are used to check on how well a patient is responding to ventilation. Interpreting ABGs is essential to identifying the issues that a patient is experiencing.

Learning Objectives

At the end of this chapter, you will be able to:

1. Relate ABGs to the acid-base relationship in the body.
2. Identify normal ABG values.
3. Interpret ABGs.
4. Comment on oxygenation status.
5. Identify patient-ventilator asynchrony

Key Terms

- Arterial Blood Gases (ABGs)
- Homeostasis
- Acidic pH
- Alkaline/Basic pH
- Neutralization/Buffering

- HCO_3 or bicarbonate
- Respiratory failure/ventilatory failure
- Hypercarbia
- Hypocarbia
- Alkalosis
- Acidosis
- pCO_2
- Hypoxemia
- Asynchrony

Whenever these terms are first introduced in this chapter, they are bolded. However, if you need additional information about a term than what is provided here, you can research it in The Free Dictionary: Medical Dictionary.

8.1 THE THEORY BEHIND ACID-BASE AND ABGS

On an ongoing basis, the human body is engaged in a complex balancing act—the pH of the body and blood. All of our cells and organs operate best at a specific pH. This level is referred to as the body's **homeostasis**. There are two byproducts produced by different systems in the body that affect the pH of the body: carbon dioxide (CO_2) and bicarbonate (HCO_3). CO_2 and HCO_3 are monitored and altered by the body to insure a balanced pH. The brain oversees this balancing act by turning up or down specific processes in the body to regulate the levels of CO_2 and HCO_3 to try to get back to "normal" or balanced pH. Therefore, testing the blood levels of CO_2 and HCO_3 and checking the pH of the blood is a very quick and effective way of assessing how well the body is doing. Abnormal levels are often one of the first signs of deterioration and can help clinicians know whether they may need to intervene and support a patient to prevent respiratory or organ failure.

Acid-base balance and ABGs is a huge topic that could be an entire book. This chapter will focus on the main concepts you need to know and why the acid-base balance is important when it comes to how humans, specifically your patients, need to breathe. Let's dive into this concept of body pH, CO_2 and bicarbonate levels a little more...

What is normal? The body is in homeostasis and happy with pH levels of $7.35 - 7.45$. This range is considered normal. If the pH of the body goes lower than 7.35, the body pH is **acidic**. If the pH goes higher than 7.45, the body pH is **basic** or **alkaline**.

Look at the following pH scale for a visual model of the spectrum from acidic (left) to basic (right) with a healthy and balanced homeostasis between them.

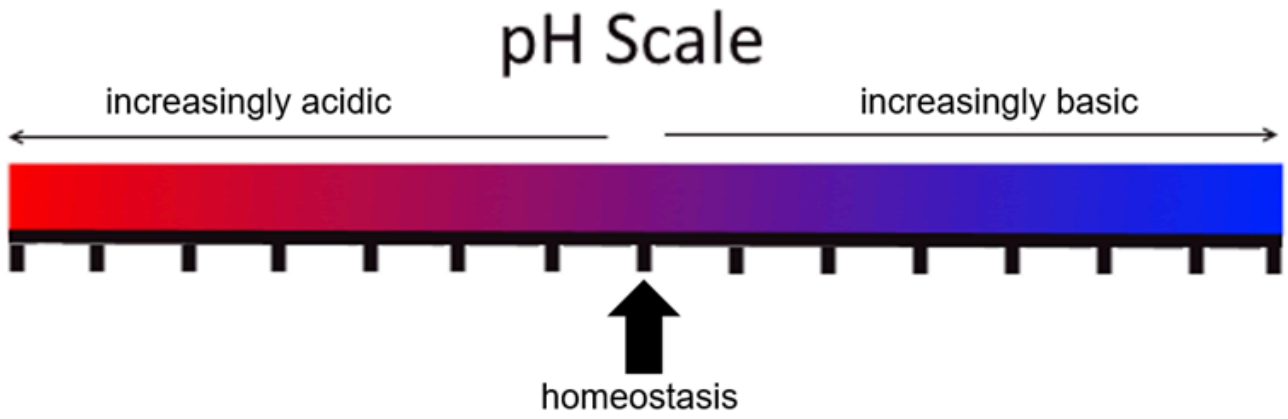


Figure 8.1.1: pH scale by Christinelmiller adapted by Amanda Robinson, CC BY-SA 4.0

Carbon dioxide—the waste of every cell on a molecular level—is a potent acid. All cells create CO_2 in the process of cellular respiration. Remember high school biology? You probably learned that CO_2 is created as a waste product during the Krebs cycle. If left unchecked, the excess CO_2 production would leave the body in a highly acidic environment.

Object Lesson

Photo by Laurence Facun, CC BY 2.0

How well do you think the cells and organs in the body would fare if they were floating in a highly acidic environment? Think of how much it hurts if vinegar comes in contact with a papercut. It stings, right? It is the same thing for your body when there is too much CO_2.

This acid (CO_2) must be buffered—neutralized—by the body and then cleared out of the system. The entire focus of the acid base status is to neutralize the amount of CO_2 present until it can be cleared by the lungs. Remember, we exhale CO_2.

So how does neutralization happen, and how does the body regulate the pH without becoming acidic? Regulating pH can happen by two methods:

1. Neutralization (buffering the acid)
2. CO_2 Clearance (acid removal)

Neutralization

The kidneys play the main role in neutralizing and regulating the pH of the body. Cellular processes can create acids at high rates and to combat this effect, the kidneys are able to regulate an important electrolyte that buffers, or neutralizes, any acid that is present. Bicarbonate is a negative ion that can bind to acidic Hydrogen (H+) ions in the body to get rid of circulating acid. In essence, bicarbonate (HCO_3) "buffers" or neutralizes the circulating acid to stabilize the pH of the body. HCO_3 is the most important electrolyte when it comes to the body's regulation of pH. The combined compound carbonic acid (H_2CO_3) circulates in the body until it reaches the lungs where it breaks down into CO_2 and is exhaled. The kidneys play the main role in neutralizing the acids by excreting acid in the urine while conserving or eliminating bicarbonate to ensure the perfect amount is present in the body to neutralize all the CO_2 that is present. This process is referred to as the **metabolic** component of the ABG.

The constant buffering process looks a little like this:

$$CO_2 + H_2O \leftrightarrow H_2CO_3 \leftrightarrow H+ + HCO_3-$$

CO_2 Clearance

The other method of maintaining homeostasis is by CO_2 clearance. CO_2 is cleared out of the body through the lungs. CO_2 is carried in the blood stream to the alveolar-capillary membrane where it diffuses into the alveoli to be exhaled with every breath. This process is referred to as the **respiratory** component of an ABG.

Any alterations to normal physiology or any disease process can cause changes to CO_2 levels. These changes could occur in two ways:

1. Changes in CO_2 production of the cells, causing higher or lower levels to be circulating in the body (e.g., illness, increased metabolism, exercise)
2. Changes to how much CO_2 is eliminated by the alveoli—changes in gas exchange or increased/decreased CO_2 amounts being exhaled every minute (e.g., changes to the respiratory rate or volume of breathing; impaired lungs that cannot contribute to gas exchange)

Either way, if either of these changes occur, the level of CO_2 in the body can quickly start to increase or decrease. Remember: if you think of CO_2 as an acid, any increase in CO_2 would cause the pH to become acidic, while decreases in CO_2 (acid) would cause the pH to become more alkaline (basic). Chemoreceptors in the brain—the same ones we talked about in Chapter 1—sense this change and try to drive the body back to homeostasis via two mechanisms:

- by altering the breathing pattern of the body, and
- by changing the levels of bicarbonate in the body.

> Think back to Chapter 1 and how we talked about the chemoreceptors in the brain that can sense the levels of O_2 and CO_2 and trigger us to breathe! An ABG is a point-of-care test that can help you see the CO_2 and pH numbers the chemoreceptors are reacting to. If you start to look at ABG values with your patients, you will see this in action. Higher CO_2 levels will be seen in patients breathing quickly. Their brain is "triggering" them to breath at an elevated rate because of that high CO_2 level. To be more specific, it isn't the CO_2 the chemoreceptors are reacting to as much as the pH of the blood.

The first line of defense for restoring acid-base balance is the respiratory system and altering the breathing pattern of the body, as this adjustment can be done immediately with effects being seen within minutes. As discussed in Chapter 1, higher levels of CO_2, or more specifically a decrease in pH (becoming more acidic), will trigger the brain to stimulate the patient to breathe more to help clear more CO_2. More importantly, increased breathing drives the pH back to a normal level because that is where the body functions best at.

The second line of defense for restoring acid-base balance is the metabolic process of the kidneys and changing bicarbonate levels. This process takes more time as HCO_3 is either excreted in the urine or reabsorbed in the urinary system and needs time to be reabsorbed by the body. The metabolic process takes upwards of 24 to 48 hours to correct an imbalance. Though slower, bicarbonate levels can also regulate pH levels back to physiologic normal (7.35-7.45), so the respiratory system does not have to continue to do all the "work".

The difference in correction time is an incredibly important differentiation to understand. Respiratory changes happen within minutes, while metabolic changes take 24 hours to start working. If for any reason a person's drive to breathe is altered due to injury or illness, the ability of the brain to correct acid-base imbalances in the short term immediately disappears. Small alterations in pH can cause very large changes in level of consciousness and can be life threatening very quickly. If left uncorrected, the bicarbonate may not have enough time to change before the balance becomes life-threatening. This condition is termed **respiratory failure** (or **ventilatory failure**), and it requires some type of ventilatory support—in most cases mechanical ventilation. Mechanical ventilation is a clinician's way to impact and change the patient's current acid-base status when their own physiologic ability has become compromised or is deemed inadequate.

Don't sweat it if you don't really get it. The main takeaway messages are that the body wants to stay at a pH of $7.35 - 7.45$ for vital organ and cellular functions to occur optimally. It achieves this by regulating the amount of CO_2 and bicarbonate in the body. Remember:

- Bicarbonate = alkaline
- CO_2 = acidic

As long as you have enough bicarbonate in the body to equal out the CO_2 amount, the body will stay in balance.

Key Takeaway

Acid-base (pH) levels in the body are sensed by the chemoreceptors in the brain. Any issue with CO_2 levels is a respiratory issue, and any HCO_3 issue is a metabolic issue. The quickest way to restore homeostasis in acid-base levels is by changing the breathing pattern to alter the speed in which CO_2 is expired out of the human body. Breathing-pattern changes can alter CO_2 levels within minutes. A secondary mechanism to control acid-base status is a metabolic route by controlling the amount of bicarbonate available to be used as a buffer in the body. This process is controlled by the kidneys and can take 24 hours or more to fix acid-base imbalances.

Learn More

If you want more details about what you learned on this page, further reading on this topic can be found here:

- Acid-Base Balance – Anatomy and Physiology (opentextbc.ca)
- Disorders of Acid-Base Balance – Anatomy and Physiology (opentextbc.ca)

8.2 ARTERIAL BLOOD GASES

Now that we reviewed the basics of acid-base balance and pH in the body, let's talk a little more about **Arterial Blood Gases (ABGs)**. Arterial blood is being pumped from the heart after getting oxygenated by the lungs, and is being delivered to the organs. This blood has not dropped off the oxygen at all the cells yet and therefore, the levels of CO_2 and O_2 are reflective of how well the lungs have been able to clear the CO_2 and add oxygen after going through the lungs.

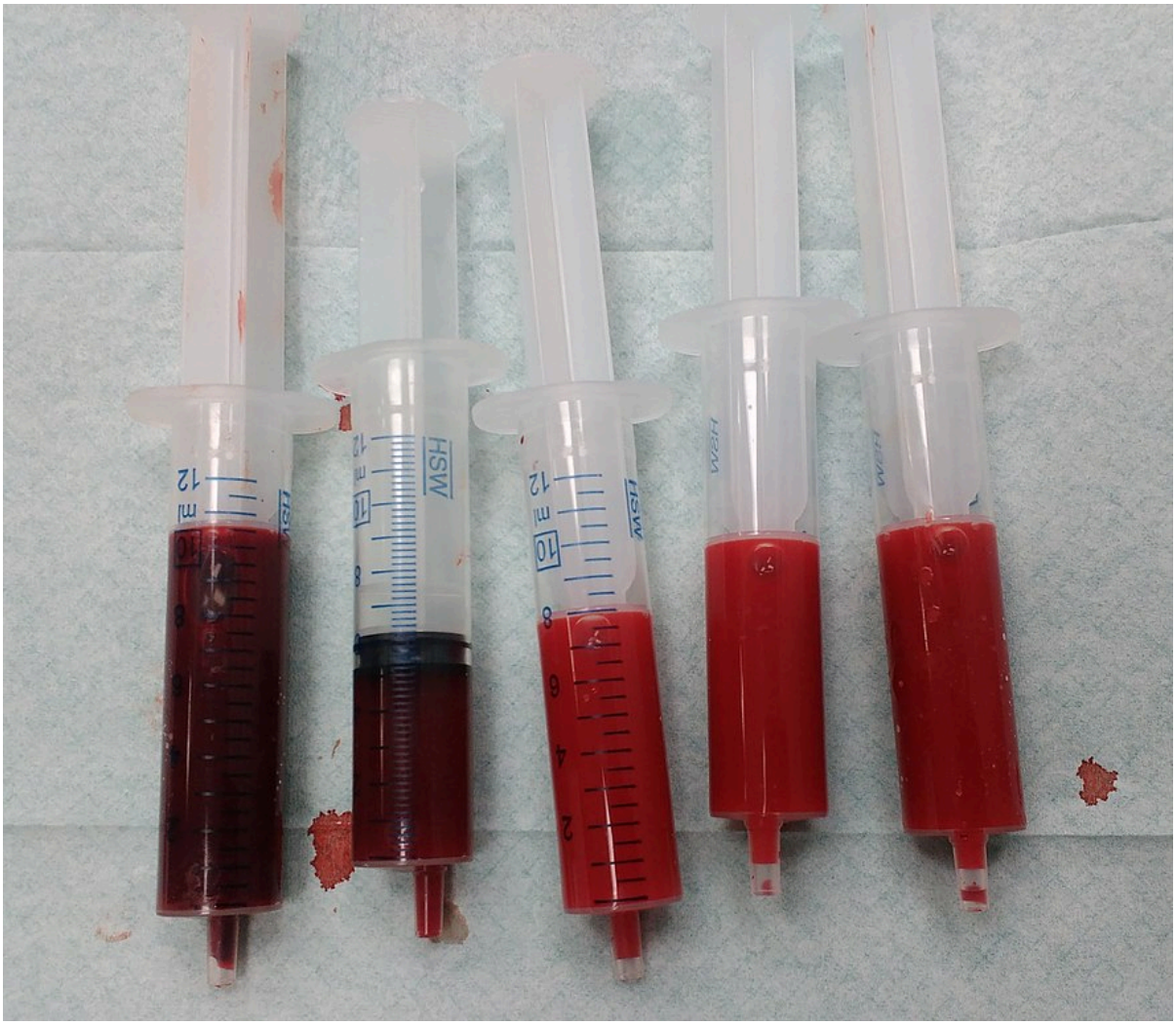


Figure 8.2.1: The difference between the oxygen-rich arterial blood and the oxygen-depleted venous blood is clear on sight. Can you tell which is which? Photo by Wesalius, CC BY 4.0

ABGs convey four different values (often Base Excess is added as the fifth value):

- pH
- pCO_2
- pO_2

- HCO_3

You will notice there is a "p" in front of the CO_2 and O_2 in an ABG. This is because the levels of CO_2 and O_2 are expressed as a partial pressure. Don't get caught up in that. It is just a way of giving the levels units that the numbers can be expressed in. It is still reflective of the levels of CO_2 and O_2 in the blood, and we will go through how to read them.

Normal Values

Take a look at this brief table which presents the normal ranges for all of the values in the ABG results. When results are outside of these ranges, they tell you that something needs to be done for your patient. Each of these normal ranges and what they mean is discussed in more detail below.

Table 8.2.1: ABG Normal Ranges

Normal Values	
pH	7.35 - 7.45
pCO_2	35 - 45 mmHg
pO_2	80 - 100 mmHg
HCO_3	22 - 28 mmol/L

As previously discussed, the pH of the blood should be between $7.35 - 7.45$ to be considered within normal range. If the pH is below 7.35, it is considered acidic; and if the pH is above 7.45, it is considered alkaline.

Revisit the visual pH scale now, but notice that the numerical ranges have been added:

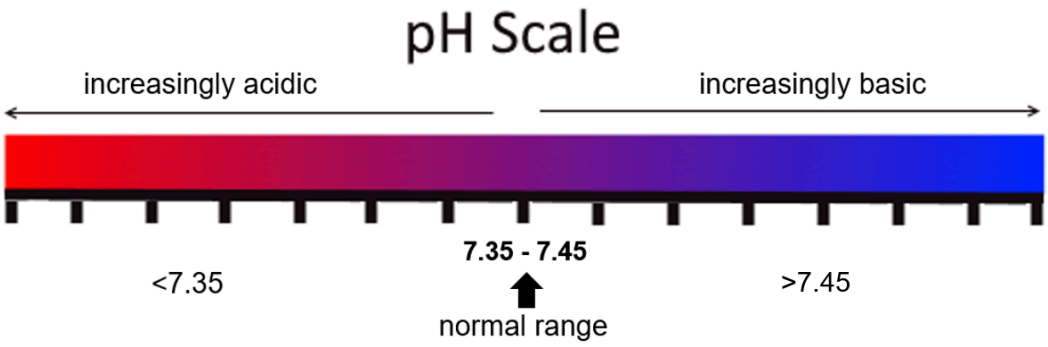


Figure 8.2.2: pH Scale Ranges by Christinelmiller adapted by Amanda Robinson, CC BY-SA

The partial pressure of CO_2 in the blood is considered normal at $35 - 45$ mmHg. If the CO_2 is higher than 45 mmHg, this is termed **hypercarbia** (or hypercapnia). If the CO_2 is less than 35 mmHg this is **hypocarbia** (hypocapnia). Another term that is used to discuss this type of reading is CO_2 retention.

Key Takeaway

Remember! CO_2 = acid; therefore, low CO_2 = **alkalosis** (alkaline pH), and high CO_2 = acidosis (acidic pH).

The partial pressure of oxygen in the blood should be $80 - 100\ mmHg$ when at normal levels. This number is read off the blood gas, but is separate from the acid-base process. The pO_2 reading is not part of your determination of acid-base and is generally looked at after your ABG interpretation to assess the oxygenation status. You will learn more about pO_2 later in the chapter after ABG interpretations (see Oxygenation Status: The Final Piece).

The concentration of HCO_3 in the blood is somewhere around $22 - 28\ mmols/L$ (each textbook/hospital may vary slightly in their normal range—for the purpose of this book, this will be our normal range). Bicarbonate levels higher than 28 will shift the body into a state of alkalosis. Low bicarbonate levels less than 22 will not be able to buffer free H+ ions, allowing the body to go into a state of acidosis.

Key Takeaway

Remember! HCO_3 = base; therefore, low HCO_3 = acidosis and high HCO_3 = alkalosis.

When interpreting an ABG, the values are normally reported in this order:

1. pH,
2. pCO_2,
3. PO_2 and then
4. HCO_3.

ABG shorthand is often written with the numbers in that order with backslashes between them ($pH/pCO_2/pO_2/HCO_3$). Don't let this confuse you. Just remember the order and you will look like a pro!

Let's uncomplicate this. Before jumping into how to start interpreting blood gases, we are going to go through an analogy to help you understand the science behind what is going on when it comes to ABGs. Truly understanding the concept is always better than trying to memorize patterns. Take a look at the following object lesson.

Object Lesson

Think of homeostasis as a massive game of Tug O' War. The CO_2 family (Team Acid) and the Bicarbonate Family (Team Base) are locked in an epic battle that never seems to end. Normally, the CO_2 family has 35-45 members (normal pCO_2 levels) while the Bicarbonate Family has 22-28 members (normal HCO_3 levels in the body). At these levels, the teams are evenly matched and no one is winning. At this balanced or "normal" level, the pH in the body is 7.35 - 7.45.

Photo by saxonrider, Pixabay Licence.

If Team Acid is winning, they have been able to pull the rope to their side, pulling the pH rope past the 7.35 mark—any pH under 7.35 is classified as an acid. If Team Base is winning, they have been able to pull the pH rope past the 7.45 mark—any pH over 7.45 is considered base. If the game of Tug O' War is perfectly matched with 35-45 pCO_2 on one side and 22-28 HCO_3 on the other side, changes in the number of players on either side will have direct impact on which team is winning.

If the CO_2 family calls their friends in to join the team (more CO_2) the pH/rope is going to start to move toward the acid side. Conversely, if the CO_2 side is at their normal player numbers, but the bicarbonate family calls for extra teammates to come join, the pH/rope will pull towards the base side. It is also very important to realize that if both teams are at their normal amounts, any loss of players on either side will cause the opposite side to start to pull the pH/rope their way. If the HCO_3 family loses some of their teammates, then the rope starts to go more toward Team Acid, or if the CO_2 family loses members, then Team Base will start to win the battle of the pH and pull the rope above 7.45 pH)

Here is the most important fact to remember: given the opportunity, the human body always wants to return to homeostasis (pH 7.35 - 7.45). As pCO_2 and HCO_3 start to change, the human body will start to compensate by altering the opposite side to try to even out the imbalance. Think of this as the CO_2 and HCO_3 family wanting a fair fight. So as teammates are lost on one side, then the other side will drop teammates as well because they want to beat the other team "fair and square." Also, as one side adds players, the other side will also add players, if given the chance, to compensate and even out the game.

Keep this Tug O' War analogy in mind when interpreting ABGs, and you will never go wrong.

8.3 A COMPLETE ABG INTERPRETATION

There are four parts to a complete ABG interpretation. The pH, pCO_2, and HCO_3 are used to determine the overall acid-base status of the blood. The pO_2 is used to assess the oxygenation level of the blood. A clinician must determine if the blood sample is:

- **Uncompensated, partially compensated, or fully compensated:** Remember, the human body wants to be in homeostasis with a pH that is within normal range $(7.35 - 7.45)$. If either the CO_2 or HCO_3 is abnormal, the brain will try to even out the imbalance. Are the scales completely uneven or has the body started to even things out by "adding or subtracting weight" to the other side? This is easy to see by looking at the CO_2 and HCO_3 values. Is only one outside of normal ranges or are both outside of normal ranges? If only one value is abnormal, it is uncompensated and the body has not started changing the other value to normalize the pH. If both CO_2 and HCO_3 are abnormal, then the body is trying to compensate. Determining whether these levels are only partially or fully compensated depends on whether the pH has returned to normal.

- **Respiratory or metabolic:** Acidosis or alkalosis has to have a cause. Is it because of the CO_2 (respiratory) or the HCO_3 (metabolic)? What is tipping the scale and causing the body to go off balance?

- **Acidosis or alkalosis:** Is the body sitting with a pH lower or higher than normal? Or is it normal? At the end of the day, a blood gas can only be unbalanced or balanced. Even if the pH is balanced, the ABG may not be "normal". It is easy to see if other levels are off that the brain has already fixed with up- or down-regulating CO_2 or bicarbonate via breathing and the kidneys. That blood gas, though balanced, is still an acidosis/alkalosis that is fixed. It is not normal.

- **Hypoxemic, hyperoxic, or normal:** After deciding on acid-base status, look at the pO_2 level and decide on the oxygen level. Is the oxygen within normal range? Too low (hypoxemia)? Too high (hyperoxia)?

Though this is the order the interpretation is written down in, deciding acid-base status is easiest when done a little out of order by looking at the pH, pCO_2 and HCO_3 together. An easy approach is to fill in the blanks as you find the information. Which of the following choices is it?

_____	_____	_____	*with*	_____
(un/partially/fully compensated)	**(respiratory/metabolic)**	**(acidosis/alkalosis)**		**(mild/ moderate/ severe hypoxemia)**

Here are the rapid steps to success when interpreting an ABG:

1. Classify if it is an acid or alkaline gas

Look at the pH and determine where it falls. Ask who is winning the Tug O' War game: Team Acid (acidosis), Team Base (alkalosis), or are they evenly matched (normal or fully compensated)?

→ *fill out either the acidosis/alkalosis or the fully compensated blank*

2. Classify the pCO_2 and HCO_3

Look at the pCO_2 and HCO_3, and determine if any are abnormal and if so, is it only one (uncompensated) or both values? (partially or fully compensated). Look at your Tug O' War teams' CO_2 and HCO_3 members—do they have their normal member numbers? Or have they lost/added teammates.

→ *fill out the compensated blank*

3. Determine the cause and type of change to the pH

Ask which abnormal value is causing the change in pH, and determine if it is a respiratory or metabolic change to the pH. In other words, what change in the Tug O' War team members up and down is causing the abnormal pH? Example: If team CO_2 is winning and it is an acid, and both sides have lost players, which change would cause team acid to win? It wouldn't be losing CO_2 that would cause them to win, so it is because of the loss of HCO_3; therefore, this is a metabolic change.

→ *fill out the respiratory/metabolic blank*

4. Comment on the oxygen status

Look at the oxygen status and then make a comment on the degree of hypoxia.

→ *fill out the oxygenation blank*

Feeling a bit lost at this point? Don't worry! This is just an overview of the general approach to ABG interpretation. On the next two pages, you'll see more in-depth discussion of each step mentioned here.

A device similar to this will analyze the arterial blood sample and produce the values of the ABG–all ready for your interpretation! Arterial Blood Gas Device by PhilippN, CC BY-SA 4.0

8.4 EXAMPLES OF REAL-WORLD ABG INTERPRETATION

Let's look at some common gases and what they would look like in real-world practice. We will start easy and slowly increase the difficulty. If you're still having trouble understanding ABGs at this point, try working through the first example or two and see if things "click." For the purposes of these examples, we will comment on the acid-base status only and fill in the first three "blanks" of an ABG interpretation. After you have a good understanding of this concept, you will learn how to comment on the oxygenation status.

As you work through this page, you will need to refer back to the normal ranges of ABG values to figure out your answers. Here are those values again now:

Table 8.4.1: ABG Normal Ranges

Normal Values	
pH	7.35 - 7.45
pCO_2	35 - 45 mmHg
pO_2	80 - 100 mmHg
HCO_3	22 - 28 mmol/L

If you can, take a moment to jot these numbers down on paper, or save it in a handy file on your computer so you can easily refer to these ranges when needed.

Level 1: Uncompensated gases

The easiest ABGs to interpret are uncompensated ABGs. In this case, an illness or problem is causing a swing in the pH out of normal range. The body has not had time to try to even out the imbalance yet. Therefore, only *one* of either the CO_2 or HCO_3 is outside of the normal range. These uncompensated gases are easy to identify because the abnormal pH is caused by *one abnormal* value (CO_2 OR HCO_3).

Let's see this ABG type in practice! Remember, ABGs are often written with just the values pH/pCO_2/pO_2/HCO_3. We will be filling out only the acid-base portion of the ABG interpretation so that we can focus on acid-base interpretation, so there are three blanks to fill:

_____	_____	_____
(un/partially/fully compensated)	**(respiratory/metabolic)**	**(acidosis/alkalosis)**

Since we are leaving out the oxygen level for now, the placeholder text "oxygen level" will appear in each of the following examples. Oxygen level will be covered on the next page.

Patient A | 7.31/57/Oxygen level/24

Steps to complete:

1. Start with the pH and determine if it is abnormal or normal (or fully compensated).

Solution

In this example, the pH is less than 7.35. This is considered an acid. This ABG is an acidosis:

(un/partially/fully compensated) (respiratory/metabolic) (**acidosis**/alkalosis)

2. Look at the pCO_2 and the HCO_3. How many are abnormal?

Solution

In this example, only the pCO_2 is outside of normal ranges. The HCO_3 is normal. There is too much CO_2. It is higher than normal. Only one is abnormal. There is no compensation with the other value starting to change too. This gas is uncompensated.

(**un**/partially/fully **compensated**) (respiratory/metabolic) (**acidosis**/alkalosis)

3. Determine which change (in CO_2 and HCO_3) is causing the acidosis/alkalosis.

Solution

In this case, we know the CO_2 is too high. We have already learned that $\mathrm{CO_2} = \mathrm{acid}$ and the more we have, the more acidic the pH will be. This makes sense. We know CO_2 relates to the respiratory component and HCO_3 relates to the metabolic component. This abnormality is of the CO_2, therefore the cause of the acidosis is respiratory.

(**un**/partially/fully **compensated**) (**respiratory**/metabolic) (**acidosis**/alkalosis)

If you use the Tug O' War Analogy: In this case, the rope has been pulled past 7.35 so Team Acid is winning! Why are they winning though? Is it because the CO_2 side added players? Or because the HCO_3 side lost players? In this case, the CO_2 side added players. They are the reason for the change, so it is a respiratory acidosis.

ANSWER: $7.31/57/\mathrm{Oxygen\ level}/24$ is an **Uncompensated Respiratory Acidosis**

Practice Makes Perfect

You try one! Take a look at the following ABG values fill in the three blanks yourself (remember, you can skip oxygen level for now).

$$7.53/37/\text{Oxygen level}/33$$

Solution
Uncompensated Metabolic Alkalosis

Level 2: Partially compensated gases

After an illness or physiologic process causes an acid-base imbalance, the body will start to try to "fix" the imbalance by altering the levels of the opposite value—increasing or decreasing the amounts of the opposite variable to compensate for the change in the other value. The pH will partially correct but has not returned back to normal, so the body's compensation is only *partially* done.

Key Takeaway

With a partially compensated gas, the pH is still *abnormal*, but there are two abnormal sides of this: both CO_2 and HCO_3 are outside of the normal ranges.

The trickiest part of this interpretation is deciding what the problem is—is it respiratory or metabolic? To decide this, identify whether the pH is high (alkalosis) or low (acidosis) and whatever altered value—CO_2 or HCO_3—would drive the pH that way. The other value would be the one to try to pull the pH back to normal.

Let's see this ABG type in practice! Remember, ABGs are often written with just the values $pH/pCO_2/pO_2/HCO_3$. We will be filling out only the acid-base portion of the ABG interpretation so that we can focus on acid-base interpretation, so there are three blanks to fill:

_____	_____	_____
(un/partially/fully compensated)	**(respiratory/metabolic)**	**(acidosis/alkalosis)**

Patient B | 7.32/30/Oxygen level/18

Steps to complete:

1. Start with the pH and determine if it is abnormal or normal (or fully compensated).

Solution

In this example, the pH is less than 7.35, so this is an acidotic pH.

(un/partially/fully compensated) (respiratory/metabolic) (**acidosis**/alkalosis)

2. Look at the pCO_2 and the HCO_3. How many are abnormal?

Solution

In this example, both pCO_2 and HCO_3 are outside of the normal limits. We still have an abnormal pH, but both values have moved from "normal"—one causing the acidosis, and one trying to fix the acidosis. It is definitely a compensated-type gas. But is it fully or partially compensated? Since the pH is still abnormal, the imbalance is only *partially* fixed.

(un/**partially**/fully **compensated**) (respiratory/metabolic) (**acidosis**/alkalosis)

3. Determine which change (in CO_2 and HCO_3) is causing the acidosis/alkalosis.

Solution

In this example, we have low CO_2 and low bicarbonate. Which one of these would cause an acidic situation? Remember $\mathrm{CO_2} = \mathrm{acid}$ and $\mathrm{HCO_3} = \mathrm{base}$. Low CO_2 would not cause an acidosis but low HCO_3 (not enough base) would! HCO_3 is the metabolic component, so this is a metabolic acidosis.

(un/**partially**/fully **compensated**) (respiratory/**metabolic**) (**acidosis**/alkalosis)

If you use the Tug O' War analogy, in this ABG, one side is winning Tug O' War. The pH/rope has been pulled lower than 7.35, so Team Acid is winning at this time. Why are they winning—did team CO_2 gain players? No, it looks like the CO_2 is lower. This should mean they would be losing, but both teams have changed numbers! The HCO_3 has lost players as well. That would definitely cause Team Acid to be winning. Losing CO_2 is the attempt to try to even out the game of Tug O' War, but it is not enough to fully bring it back to an even game. Is it respiratory or metabolic? Remember if the pH rope is pulled to the acidosis side, whatever player number changes that would cause Team Acid to win is the primary mechanism and any other player number changes are the body's response, and if you think about it, that change would cause the opposite reaction.

Practice Makes Perfect

You try one! Take a look at the following ABG values fill in the three blanks yourself (remember, you can skip oxygen level for now).

$$7.47/30/\text{Oxygen level}/20$$

Solution
Partially compensated Respiratory Alkalosis

Level 3: Fully compensated

Abnormalities that cause pH imbalances will, over time, be normalized with normal compensation of the body. We have described early in this chapter how the brain can regulate the CO_2 levels by triggering breathing differently, and it can conserve or eliminate HCO_3 to help even out pH. When an illness causes an issue with the pCO_2 or HCO_3 levels, the body will drive a change for the other value to cause the opposite effect. Is the pCO_2 too high, causing an acidotic gas? The body will conserve HCO_3 to add more base to the body. This will drive the HCO_3 up to help bring the pH back to normal range. You can recognize a fully compensated blood gas by a normal pH, but both the pCO_2 and HCO_3 levels are *abnormal*.

The trickiest part of a fully compensated gas is determining what the problem originally was that caused the imbalance. Textbook theory states that the clinician would look at what side of the normal range the pH is on. Meaning, if a normal range is $7.35 - 7.45$, the middle is 7.4. anything $7.35 - 7.39$ is acid "ish" and anything $7.41 - 7.45$ is alkaline "ish." Then, you would look at which abnormal value would cause that imbalance between the pCO_2 level and the HCO_3 level.

Let's see this ABG type in practice! Remember, ABGs are often written with just the values $pH/pCO_2/pO_2/HCO_3$. We will be filling out only the acid-base portion of the ABG interpretation so that we can focus on acid-base interpretation, so there are three blanks to fill:

_____	_____	_____
(un/partially/fully compensated)	**(respiratory/metabolic)**	**(acidosis/alkalosis)**

Patient C | 7.37/27/Oxygen level/16

Steps to complete:

1. Start with the pH and determine if it is abnormal or normal (or fully compensated).

Solution

In this example, the pH is between $7.35 - 7.45$. This is a normal pH. But is it a normal gas? Move on the next step to see if the rest of the values are within normal ranges.

(un/partially/fully compensated) (respiratory/metabolic) (acidosis/alkalosis)

Nothing selected because we are not sure yet. Remember, the blood gas could be totally normal, unless you see something off in the other values.

2. Look at the pCO_2 and the HCO_3. How many are abnormal?

Solution

In this example, both pCO_2 and HCO_3 are outside of the normal limits, but we have a normal pH. Both CO_2 and HCO_3 values have moved from "normal". Low HCO_3 would cause acidosis, and low CO_2 would cause alkalosis. It is definitely a compensated-type gas. But is it fully or partially? Since the pH is still normal, it is fully fixed.

(un/partially/**fully compensated**) (respiratory/metabolic) (acidosis/alkalosis)

3. Determine which change (in CO_2 and HCO_3) caused the imbalance in the first place.

Solution

In this example, we have low CO_2 and low bicarbonate. First, decide if the pH is more acidic or alkaline. Since the pH is less than 7.4, it is more acidic. This must be a compensated acidosis. Between the CO_2 and HCO_3 levels, an elevation in which one would cause an acidic situation? Remember $\mathrm{CO_2} = \mathrm{acid}$ and $\mathrm{HCO_3} = \mathrm{base}$. Low CO_2 would not cause an acidosis, but low HCO_3 (not enough base) would! HCO_3 is the metabolic component so this is a metabolic acidosis.

(un/partially/**fully compensated**) (respiratory/**metabolic**) (**acidosis**/alkalosis)

One of the easiest ways to classify a fully compensated gas is to look at the patient's diagnosis. If they are in for respiratory failure, that most likely is not a metabolic issue. It would be a respiratory issue. But if the patient was septic or on chronic diuretic treatments, that would not be respiratory. If I told you the above ABG was for a patient in DKA (diabetic ketoacidosis), this is definitely not a respiratory issue. It is easy to classify this gas as a fully compensated metabolic acidosis.

Practice Makes Perfect

You try one! Take a look at the following ABG values fill in the three blanks yourself (remember, you can skip oxygen level for now).

7.38/56/Oxygen level/34

Solution
Fully compensated Respiratory Acidosis

If you didn't get all three "Practice Makes Perfect" answers correct, try watching the following video which presents the ROME method for ABG interpretation. Some people prefer to use this method, as it makes more sense to them. After watching the video, try all of the exercises on this page again, and see how you do.

One or more interactive elements has been excluded from this version of the text. You can view them online here: https://ecampusontario.pressbooks.pub/mcvresource/?p=210#oembed-1

Video: "ROME Method ABGs (Arterial Blood Gases) Interpretation: Compensated vs Uncompensated Nursing" By RegisteredNurseRN [16:41] *Transcript Available*

8.5 OXYGENATION STATUS: THE FINAL PIECE

A complete ABG interpretation also includes a comment made on the oxygenation status. This is the easiest part. Hypoxia is graded in stages. Since this low-oxygen reading is coming from arterial blood, instead of hypoxia, it is referred to as **hypoxemia** (hypoxia in the blood). "Hypox" means low oxygen; "emia" means in the blood.

Hypoxia versus *hypoxemia*? Don't get confused here. Both terms essentially mean the same thing. The only difference between these words is where the low oxygen state was read from. Does the patient have low oxygen that is read off of the SpO_2 and clinical picture of the patient? The patient is hypoxic. If you took a sample of arterial blood and tested it and found low oxygen, then you would say the patient has hypoxemia (low oxygen in the blood).

Whatever the PO_2 is, look at where the number falls on the hypoxia description. At the end of your complete ABG interpretation, you add your comment on oxygenation, referring to the following ranges:

Table 8.5.1: Comments for Different pO_2 Values

If PO_2 is...	Comment
80 - 100 mmHg	Normal Oxygenation
60 - 79 mmHg	Mild Hypoxemia
40 - 59 mmHg	Moderate Hypoxemia
$<$ 40 mmHg	Severe Hypoxemia
$>$ 100 mmHg	Hyperoxemia

Don't feel you have to memorize the levels. As long as you know the normal levels, and severe is < 40 mmHg, then make each stage 20 mmHg above. It is easy to remember, as each stage represents 20 mmHg.

Now, let's look at what the comments will look like based on what we have learned by looking at three examples:

1. pO_2 of 62? Add "with mild hypoxemia" to the end of your ABG interpretation.
2. pO_2 of 49? Add "with moderate hypoxemia" to the end of your ABG interpretation.
3. pO_2 of 93? Add "with normal oxygenation" to the end of your ABG interpretation.

A clinician completes patient documentation in an ICU. Photo by Mohsen Atayi , CC BY 4.0

8.6 PATIENT-VENTILATOR INTERACTION & GRAPHIC REPRESENTATION OF VENTILATION

When monitoring patient-ventilator interaction, beginning with ventilation parameters appears to be a straightforward and universally adopted practice across all ventilators currently in use. The following table presents a list of common ventilation parameters that provide clinicians with valuable insights into how the patient responds to the ventilator.

Table 8.6.1: Basic Ventilation Parameters

Ventilatory Parameters	Respiratory Rate	Average Normal Value
Lung Volume	Tidal Volume	5 - 6 mL/Kg
	V_D/V_T	20 - 40%
	I:E	1:2
Airway pressures	P_{peak}	< 35 cmH_2O
	$P_{plateau}$	< 30 cmH_2O
	ΔP (driving pressure)	< 15 cmH_2O
	PEEP and auto-PEEP	Based on patient requirements
Lung mechanics	Compliance	50 - 100 mL/cmH_2O
	Resistance	0.5 - 2.5 $L/cmH_2O/s$ (up to 6 $cmH_2O/L/s$)

Patient-ventilator asynchrony is a significant concern in critically ill patients receiving mechanical ventilation. It can prolong mechanical ventilation, increase the risk of complications, and contribute to patient discomfort and distress. Close monitoring, adjustment of ventilator settings, and consideration of patient-specific factors are essential in minimizing asynchrony and optimizing patient-ventilator interaction. Asynchrony is sometimes obvious, as it causes patient discomfort, and other times it can be very subtle and difficult to notice.

Patient-ventilator asynchrony refers to a mismatch or lack of coordination between the patient's respiratory effort and the assistance provided by a mechanical ventilator. It occurs when the timing, flow, or volume of the ventilator's assistance does not align properly with the patient's own breathing patterns and needs. This lack of synchronization can result in discomfort for the patient, compromised ventilation, and potential complications. The most common methods of detecting asynchrony is analyzing the ventilator graphics.

Types of Asynchrony

1. Trigger Asynchrony

Trigger asynchrony occurs when the ventilator fails to detect the patient's effort to initiate a breath, or when the patient initiates a breath at a different time than the ventilator is delivering a breath. This lack of coordination can result in discomfort for the patient, increased work of breathing, and may affect the effectiveness of mechanical ventilation.

Key Takeaway

To minimize trigger asynchrony, the sensitivity settings must be set appropriately.

When the ventilator is not sensitive enough to patient effort, it results in increased work of breathing and potentially missed breath as the ventilator will not recognize patient effort. This is known as **missed trigger asynchrony.** Watch this brief video to better visualize missed trigger asynchrony.

One or more interactive elements has been excluded from this version of the text. You can view them online here: https://ecampusontario.pressbooks.pub/mcvresource/?p=1704#oembed-1

Video: "Patient Ventilator Asynchrony: Missed Trigger" By FREMITUS [0:54] *Transcript Available*

Auto triggering, also known as auto-cycling or self-cycling, is a phenomenon that can occur during mechanical ventilation where the ventilator is triggered to deliver a breath even when the patient has not initiated any respiratory effort. In other words, the ventilator senses a "false" respiratory effort and delivers a breath even though the patient did not intend to take one.

Auto triggering can happen due to various reasons, including:

- Sensitivity Settings: If the sensitivity of the ventilator's trigger is set too low, it may interpret non-respiratory signals (e.g., patient movement, cardiac oscillations) as spontaneous breaths and initiate a mechanical breath.
- Circuit or Tubing Movement: Movement or vibrations in the ventilator circuit, tubing, or connections can be misinterpreted as patient respiratory efforts by the ventilator.
- Airway Secretions or Water: Presence of excessive secretions or water in the airway can stimulate the

ventilator's trigger, leading to auto triggering.
- Patient-ventilator Interaction: In some cases, the patient's breathing pattern may become unintentionally synchronized with the ventilator's settings, causing auto triggering.

Auto triggering can be detrimental to the patient because it can lead to excessive ventilation, which may cause discomfort, worsen lung injury, or induce respiratory alkalosis. Additionally, it can lead to asynchrony between the patient and the ventilator, affecting the effectiveness of mechanical ventilation and potentially increasing the patient's work of breathing.

To prevent auto triggering, clinicians need to carefully adjust the ventilator settings, particularly the sensitivity level of the trigger, to ensure that it responds only to the patient's true respiratory efforts. Regular monitoring and assessment of the patient-ventilator interaction are essential to identify and address any auto triggering issues promptly. Ensuring proper sedation and analgesia for the patient can also help reduce the risk of auto triggering and improve overall patient comfort during mechanical ventilation. Watch this brief video to better visualize auto triggering.

One or more interactive elements has been excluded from this version of the text. You can view them online here: https://ecampusontario.pressbooks.pub/mcvresource/?p=1704#oembed-2

Video: "Ventilator Asynchrony: Auto Trigger" By FREMITUS [0:53] *Transcript Available*

Double Trigger Asynchrony

Double-triggering, also known as **breath-stacking**, refers to a distinctive pattern where the patient triggers two consecutive ventilator breaths and the expiratory time between them is less than half of the mean inspiratory time. This second triggering or breath stacking is often caused by a mismatch between the set inspiratory time and the patient's desired inspiratory time, which can be described as cycle asynchrony. Watch this brief video to better visualize double triggering.

One or more interactive elements has been excluded from this version of the text. You can view them online here: https://ecampusontario.pressbooks.pub/mcvresource/?p=1704#oembed-3

Video: "Patient Ventilator Asynchrony: Double Trigger" By FREMITUS [0:55] *Transcript Available*

2. Cycle Asynchrony

As we recall from chapter 2, the Cycle variable plays a crucial role in determining the opening of the exhalation valve and concluding the inspiration phase. **Cycle asynchrony** arises when there is a disparity between the inspiratory time of the ventilator and that of the patient. For example, if the inspiratory time set on the ventilator is shorter than what the patient requires (patient's desired T_i), it leads to a cycle asynchrony, resembling double triggering (this is sometimes called premature cycling).

Let's take a look at the diagram below for a better understanding: during ventilation, the ventilator-set T_i concludes, but the patient's inspiratory muscles are still contracting, resulting in a drop in pressure. Should this drop in pressure reach the trigger sensitivity level, it will prompt the ventilator to deliver another breath, causing the appearance of double triggering.

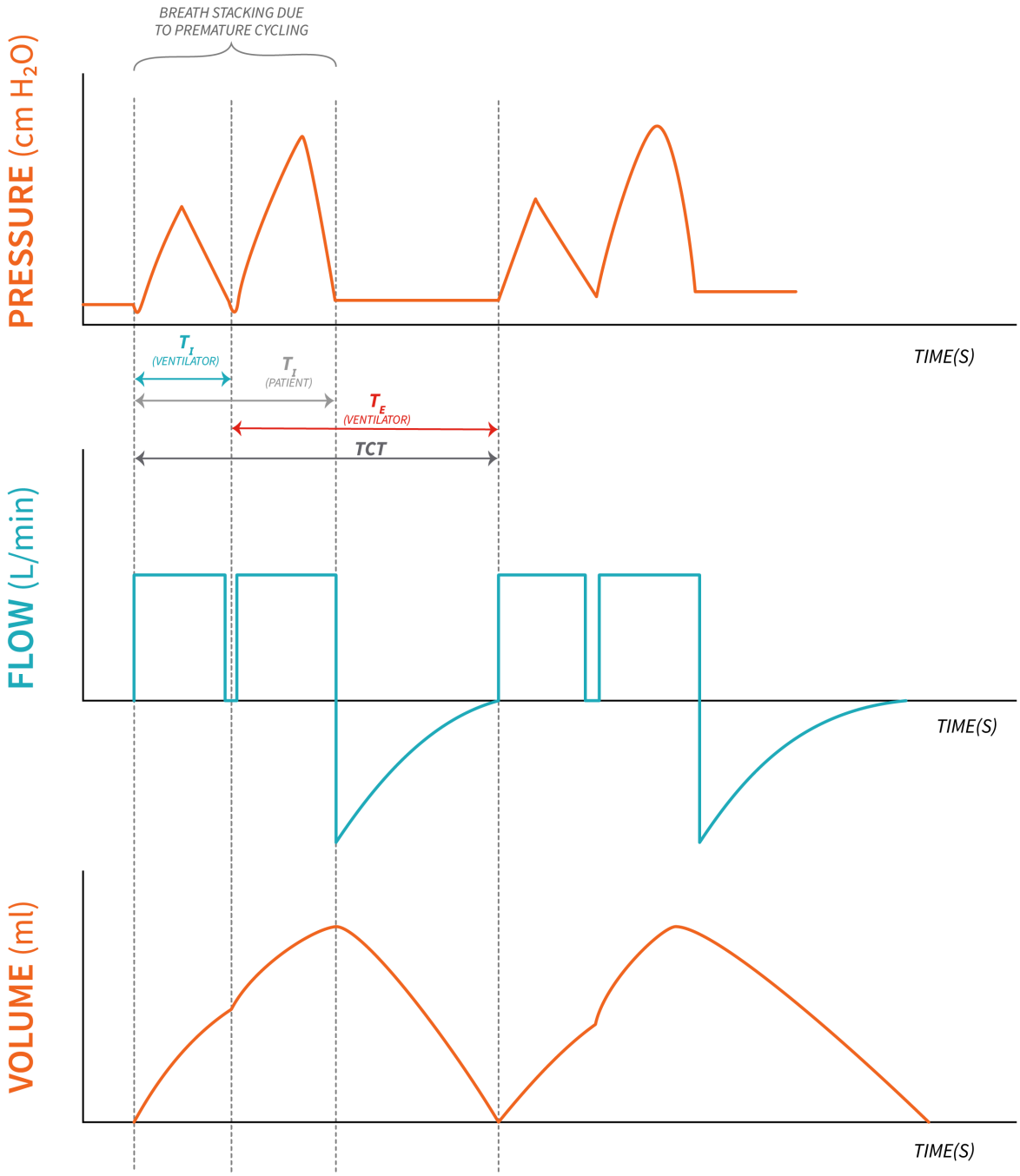


Figure 8.6.1: "Cycle Asynchrony" by Freddy Vale, CC BY-NC-SA 4.0

When the set inspiratory time is longer than the patient's desired inspiratory time, the patient may instinctively attempt to exhale,

resulting in an increase in pressure within the ventilator circuit. This increased pressure can reach the upper limit, triggering an audible alarm (this is sometimes called delayed cycling).

3. Flow Asynchrony

This type of asynchrony occurs mostly during ventilation modes where flow is set inadequately or on patients with high inspiratory flow demand. However, it can be seen in pressure control modes where inspiratory rise time is inadequate for the patient. **Flow asynchrony** can be corrected either by increasing flow in volume control ventilation or adjusting the rise time in pressure control ventilation. Watch this video explanation of flow asynchrony.

One or more interactive elements has been excluded from this version of the text. You can view them online here: https://ecampusontario.pressbooks.pub/mcvresource/?p=1704#oembed-4

Video: "Flow Asynchrony on Volume Controlled Mechanical Ventilation" By ICU REACH [2:56] *Transcript Available*

4. Mode asynchrony

It goes without saying that selecting an inappropriate ventilation mode for a particular patient can lead to discomfort and increased work of breathing. If **mode asynchrony** goes unnoticed, it can often result in unnecessary over-sedation as clinicians may misinterpret the patient's attempt to synchronize with the ventilator as resistance or "fighting" against it. This over-sedation, in turn, prolongs the time the patient spends on the ventilator, giving rise to complications associated with extended ventilation.

To prevent mode asynchrony and its potential consequences, it is crucial to regularly assess patients for signs of spontaneous breathing and opportunities for weaning and liberation from mechanical ventilation. By proactively monitoring and addressing any asynchrony, we can optimize patient comfort, enhance the weaning process, and minimize complications related to prolonged ventilation duration.

Ventilation Graphics – Loops

Until now, our primary focus has been on monitoring ventilation graphics represented through waveforms or scalars, such as pressure-time, flow-time, and volume-time graphs. However, it's important to highlight that all ventilators are equipped with the ability to display pressure-volume or flow-volume loops, which offer invaluable insights into patient-ventilator interaction. These loops, also known as curves, serve as useful tools providing essential information that helps us better understand and optimize the dynamics between the patient and the ventilator, specifically the patient's lung mechanics.

The Pressure-Volume Loop

First, let's identify the type of breath on a pressure-volume loop (PV loop).

A PV loop is drawn in a counterclockwise direction for a controlled PPV breath and in a clockwise direction for spontaneous breaths. Compare the three diagrams below from a controlled breath (Figure 8.6.2) and a spontaneous breath (Figure 8.6.3). An assisted breath (seen in Figure 8.6.4) will look like a combination of a spontaneous and a controlled breath (Figure 8.6.2 and

Figure 8.6.3). The breath starts with a patient effort (spontaneous), with the flow moving in the direction of a spontaneous breath (clockwise direction) followed quickly by a change in direction of flow, counterclockwise, as the ventilator takes over.

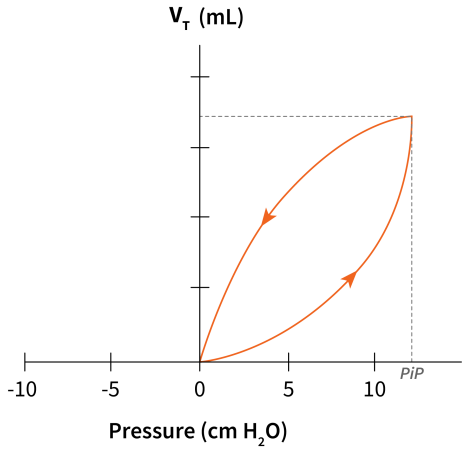


Figure 8.6.2: "PV Loop – Controlled Breath" by Freddy Vale, CC-BY-NC-SA 4.0

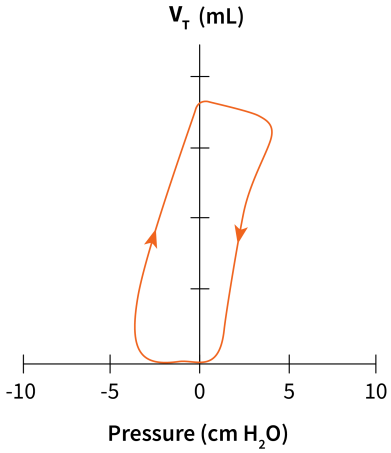


Figure 8.6.3: "PV Loop – Spontaneous Breath" by Freddy Vale, CC-BY-NC-SA 4.0

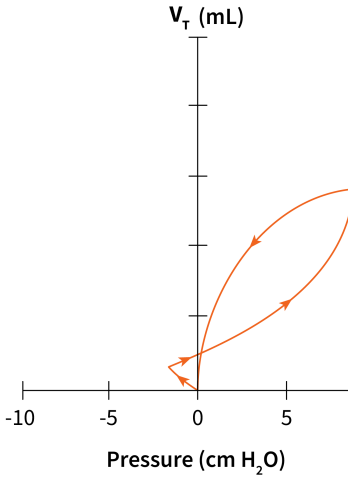


Figure 8.6.4: "PV Loop – Assisted Breath" by Vale, CC-BY-NC-SA 4.0

Now, let's review the components of a pressure-volume loop (PV loop) on a volume controlled breath with constant flow for a healthy lung.

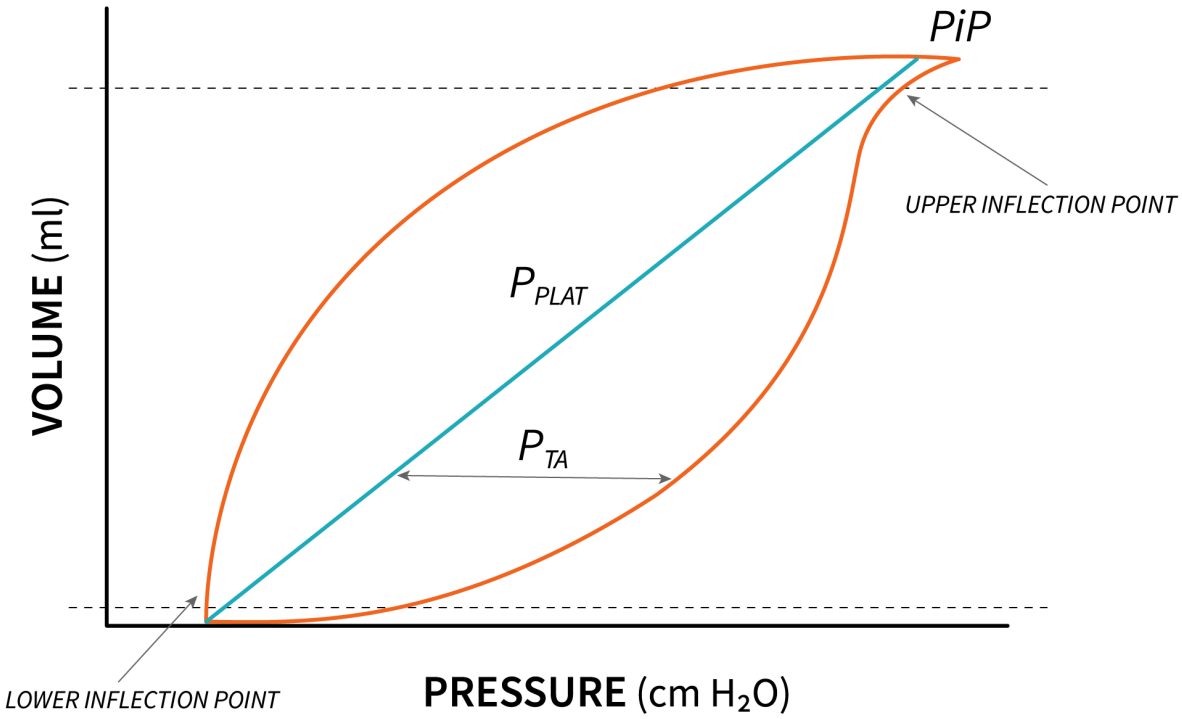


Figure 8.6.5: "PV Loop – Healthy Lung" by Freddy Vale, CC BY-NC-SA 4.0

Note that the breath begins from the set baseline (PEEP). As alveoli open at the beginning of inspiration, there is a sharp increase in pressure to the point where the alveoli begin to open. This point is known as the **Lower Inflection Point**. After this point, the pressure rises smoothly up to peak inspiratory pressure (PIP). Pressure increases in the lungs, increasing tidal volume in the same time, as long as compliance allows distention. Increasing pressure past the compliance threshold leads to overdistension. This point is known as the **Upper Inflection point** and it can be identified on the PV loop as the point past which increasing pressure does not result in an increase in volume. In fact, past this point, the risk of barotrauma increases.

The above diagram also displays a static PV loop, represented by a solid straight line, showing the pressure needed to open the lungs under static conditions known as plateau pressure. P_{alv} (alveolar distending pressure) can be determined by subtracting PEEP from this plateau pressure, and its value is influenced by lung compliance, indicating the lung's expandability.

During inhalation, the solid line of the inspiratory curve corresponds to the pressure at the airway opening when delivering a tidal volume (V_T). At the onset of each breath, the ventilator must apply adequate pressure to open collapsed alveoli, resulting in a noticeable lag time (CLICK7) before lung inflation and volume increase.

To overcome airway resistance and initiate lung opening, transairway pressure comes into play. The area between the inspiratory and expiratory curves illustrates hysteresis, which describes two associated phenomena that do not occur simultaneously. Specifically, there is a difference between the inspiratory and expiratory curves, with the lung's elastic recoil being lower during expiration compared to inspiration for any given lung volume.

Key Takeaway – Lung Overdistention

Careful assessment of PV loops is a useful tool in identifying overdistension. Additional pressure applied past the Upper inflection Point will over distend the lung without any additional volume gain. This is often described on the PV loop as "beaking" because the PV loop at the top inspiration takes on the shape of a penguin beak.

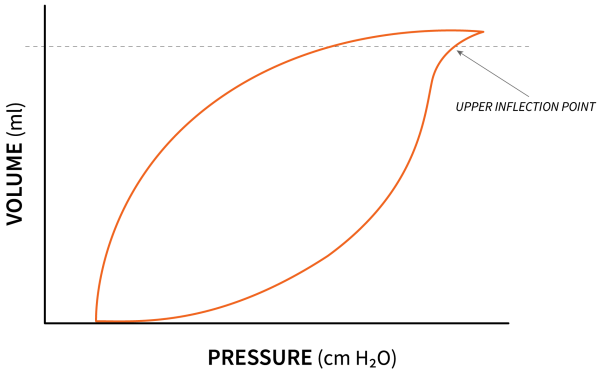


Figure 8.6.6: "Beaking PV Loop – Lung Overdistention" by Freddy Vale, CC BY-NC-SA 4.0. "Emperor Penguin jumping!" by Christopher Michel, CC BY 2.0 has been incorporated into the figure for comparison purposes.

Object Lesson

Why do we need to learn the components of the PV loop? To better understand risks posed by mechanical ventilation, and that for optimal ventilation we should be keeping pressure between the Lower inflection point and upper inflection point.

The shape and orientation of the PV loop often provides the first indication of decreasing lung compliance. For example, in the following diagram representing pressure control breaths, where pressure stays the same from breath to breath, note the difference

between the PV loop of breaths A and the PV loop of breath B. The second PV loop shows the same pressure being used to ventilate, but a significantly lower tidal volume is delivered due to the lung compliance decreasing.

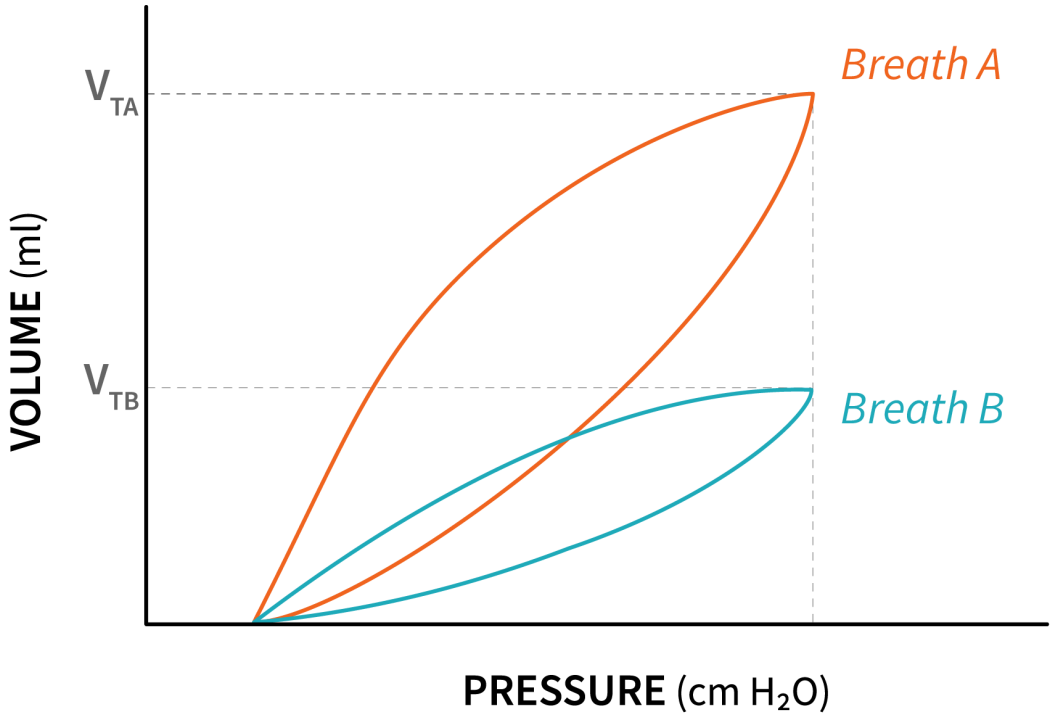


Figure 8.6.7: "PV Loop Comparison – Same Pressure with Decreasing Lung Compliance" by Freddy Vale, CC BY-NC-SA 4.0

In volume control ventilation, where the set volume remains constant with each breath, a reduction in lung compliance will result in higher pressures being applied to achieve the desired volume. On the following diagram, compare breath A to breath B where compliance decreased.

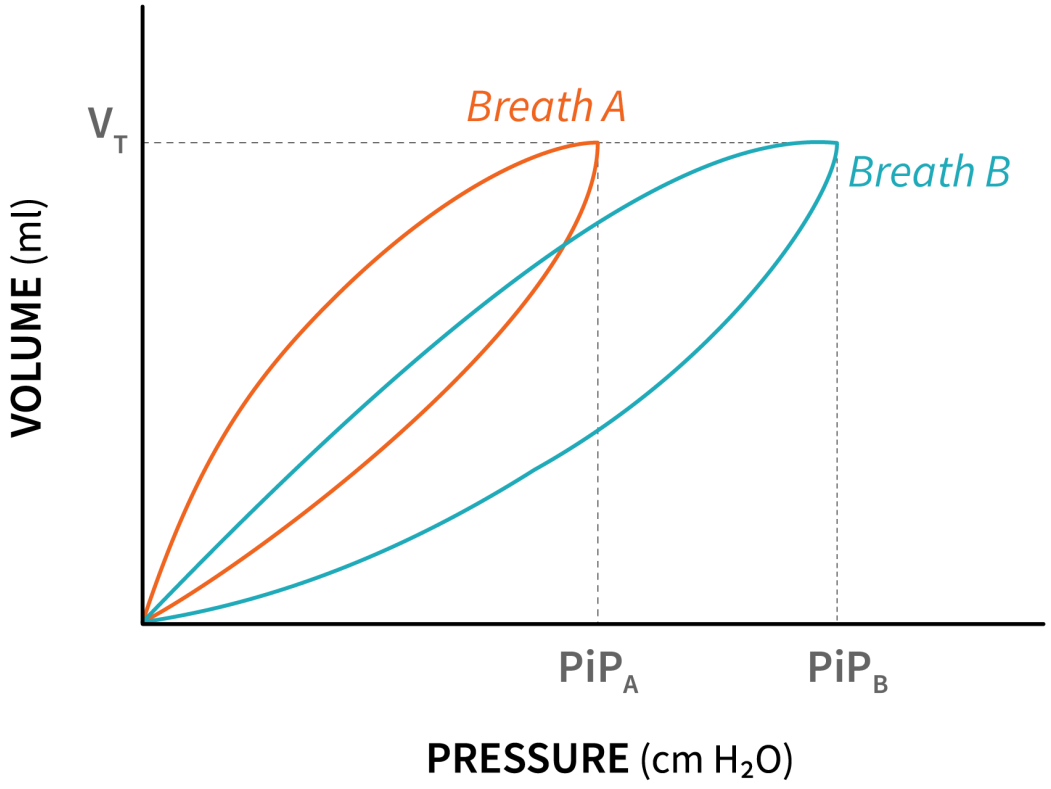


Figure 8.6.8: "PV Loop Comparison – Constant Set Volume with Decreasing Lung Compliance" by Freddy Vale, CC BY-NC-SA 4.0

Airway resistance can also be observed on a PV loop. When there is an increase in resistance to airflow, the loop becomes wider on either the inspiration or expiration side, depending on which phase experiences the higher resistance. This is more commonly seen with increased expiratory resistance, such as in conditions like Asthma or COPD.

Increased inspiratory resistance can be noticed on the PV loop. This may be caused by factors like a small-sized endotracheal tube, a kinked tube, the patient biting the tube, or the presence of large airway tumors. In such cases, the pressure can change from breath to breath as it tries to overcome the increased resistance.

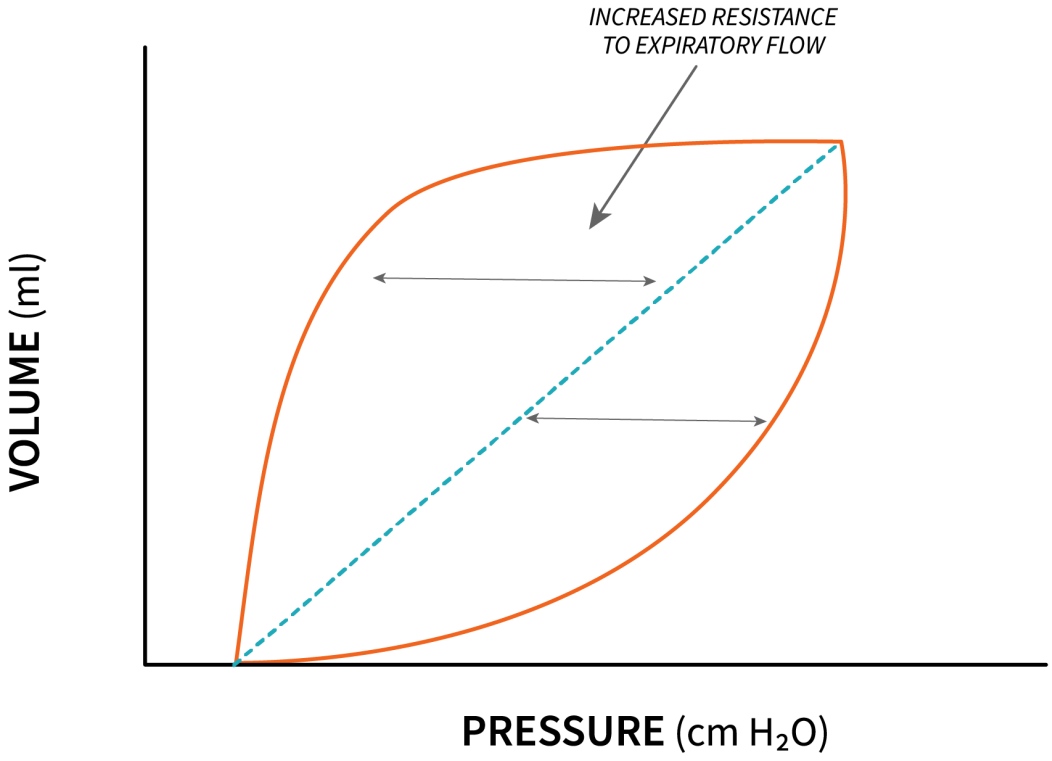


Figure 8.6.9: "PV Loop – Increased Inspiratory Resistance" by Freddy Vale, CC BY-NC-SA 4.0

The Flow-Volume Loop

Flow Volume loops (FV loops) are useful in identifying changes in airway resistance. Let's look at the flow-volume loop of a volume controlled breath. The inspiratory limb of the FV loop is controlled and constant, so it will not show any significant changes with changes in airway resistance. However, the expiratory limb of the FV loop will change with changing airway resistance. As resistance increases, the peak expiratory flowrate decreases. Sometimes, in the presence of obstruction in the airway, the expiratory limb of the FV loop takes a concave shape, giving it a scooped-up appearance.

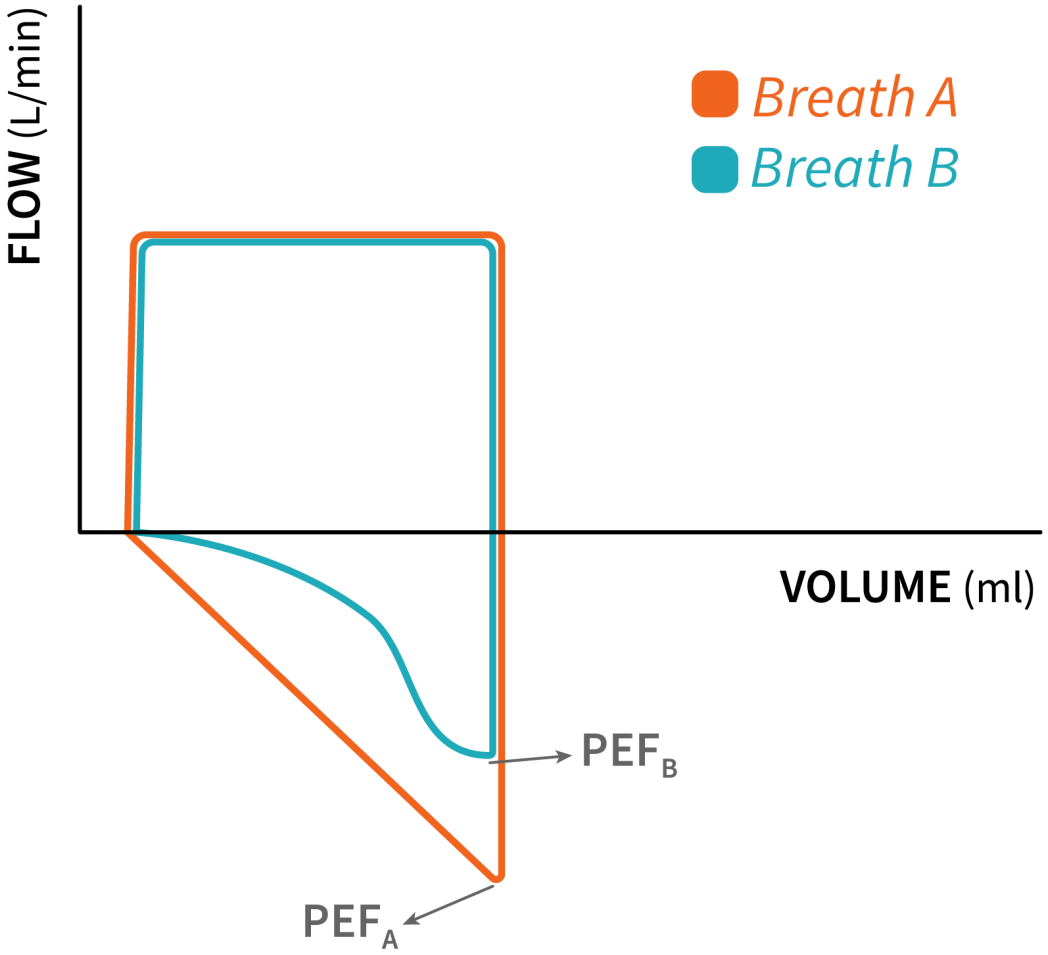


Figure 8.6.10: "Flow-Volume Loops" by Freddy Vale, CC BY-NC-SA 4.0

Key Takeaway

Because they reflect changes in airway resistance, Flow-Volume Loops are often used to assess patient's response to bronchodilator therapy.

8.7 CONCLUSION

We have now discussed the basics of ABG interpretations. It is best to try to practice as much as you can. It will become second nature the more you do it. The best way to approach ABG interpretation is to understand the "why" and not just memorize the patterns. In the next chapter, we will take these skills and start to make changes on ventilation settings, so make sure you understand the relationships between CO_2, HCO_3 and pH.

A final word of caution with ABG interpretations

Sometimes when looking at ABGs is easy to miss a "normal" gas. Clinicians can get so caught up with diagnosing a problem, that they may miss a normal reading. If all values are within normal limits, that ABG is normal and no other interpretation is needed. Normal means the body is perfectly balanced with no extra acid or base anywhere. All values are normal—there is a perfectly matched game of Tug O' War with the pH/rope in the normal position and with normal team numbers on both sides.

Review

ABG interpretation is an essential part of effective ventilation, as the information you learn will inform initial settings (when possible) and adjustments. In this chapter, you learned about the theory behind arterial blood gases: the acid-base "Tug O' War" in the body. Then, you learned what an ABG is, and the values that it produces: pH, pCO_2, pO_2, HCO_3. Finally, you learned how to answer the four main questions that underly the ABG interpretation:

- Uncompensated, partially compensated or fully compensated?
- Respiratory or metabolic?
- Acidosis or alkalosis?
- Hypoxemic, hyperoxic, or normal?

Effective patient-ventilator interaction significantly impacts the duration of ventilation and the length of stay in the intensive care unit, directly influencing the occurrence of complications. To reduce these risks, patient assessment plays an important role in promptly identifying any signs of asynchrony and implementing appropriate corrective measures. By prioritizing patient monitoring and intervention, clinicians can foster improved outcomes, reduce ventilation time, and enhance the overall quality of ICU care.

What's next?

In the next chapter, we will learn what to do with ABG results, and patient – ventilator interactions, in the context of choosing and adjusting ventilator settings.

8.8 SELF-CHECK

Chapter 8 Quiz

See how well you recall the concepts covered in this chapter by completing the following 6-question quiz.

Don't forget to check your score!

An interactive H5P element has been excluded from this version of the text. You can view it online here: https://ecampusontario.pressbooks.pub/mcvresource/?p=216#h5p-9

CHAPTER 9 | VENTILATORY ADJUSTMENTS FOR OXYGENATION AND VENTILATORY SUPPORT

Course Outline

9.0 CHAPTER OVERVIEW

As you have worked your way through this book, you have overviewed the differences between spontaneous breathing and mechanical ventilation, and learned that ventilation is *not* a "one size fits all" approach. To ventilate as safely and effectively as possible, as clinicians we need to choose our mode and ventilation settings based on our patient and what they need. You have learned about:

- Control modes, including initial settings,
- Spontaneous modes, including initial settings,
- Non-invasive ventilation, and
- Arterial blood gas (ABG) interpretations.

Finally, you already understand that, once initial settings are chosen, these settings will be evaluated for effectiveness by testing an ABG of the patient. But what do you do to the ventilation settings when you have an ABG in front of you? How do you know what to change and what to keep the same? In this chapter, we will overview:

- The goals of making changes to ventilation
- What values can you fix on an ABG with ventilation
- Which ventilator settings impact CO_2 and O_2 levels
- Identify one or more issue(s) in the ABG
- Identify *all* changes that could improve the ABG, and choose which setting would be most appropriate
- What is the appropriate degree of adjustment in order to correct ABG issues
- How to ventilate patients with severe respiratory failure such a ARDS.

Application

This chapter builds on everything you have learned in this book to put the entire picture together: from determining that a patient needs ventilation using ABGs, to choosing the correct mode for the patient, to selecting appropriate settings. Then, you will understand that you must redo ABGs to check on how the patient is doing on their treatment, and know how to use this information to adjust the ventilator settings. By working through the case study provided in this chapter, you will move with a patient through the full progression of ventilation from initial assessment to weaning, seeing how all of the pieces of the ventilation puzzle fit together. At the end of the chapter, you should feel confident to work with ventilators and ventilated patients in your clinical placement and beyond.

Learning Objectives

At the end of this chapter, you will be able to:

1. Articulate the goals of making changes to ventilation.
2. Identify what values you can fix on an ABG with ventilation.
3. Relate ventilator settings to CO_2 and O_2 levels.
4. Identify one or more issue(s) in the ABG.
5. Evaluate *all* changes that could improve the ABG to select the setting that would be most appropriate.
6. Determine the appropriate degree of adjustment that is required for a patient.

Key Terms

- blowing off CO_2
- intrathoracic pressure
- venous return
- ejection fraction
- open lung ventilation
- lung recruitment maneuver
- lung protective strategies
- esophageal manometry

Whenever these terms are first introduced in this chapter, they are bolded. However, if you need additional information about a term than what is provided here, you can research it in The Free Dictionary: Medical Dictionary.

9.1 ADJUSTMENTS TO VENTILATION PARAMETERS : THE GOALS

As clinicians, remember our guiding goal is to decrease the trauma of ventilation. We have discussed the impacts that mechanical ventilation can have on the lungs—application of positive pressure and the risk of VILI, barotrauma and volutrauma. Also, we have discussed the concerns of respiratory muscle atrophy if the patient is on the ventilator for too long. The best paths to minimizing these negative effects are:

- tailoring the ventilation settings to match your patient's needs, and
- shortening the duration of mechanical ventilation and decreasing settings as quickly as possible.

These two guiding considerations need to be at the forefront of all approaches to ventilation. As clinicians, we need to ensure we are correcting issues with mechanical ventilation, but also, when able, constantly moving to decrease ventilation settings and progress our patients towards extubation. But, don't forget: you cannot push towards extubation until the patient's presenting issue is resolving and the arterial blood gases (ABGs) are corrected.

Key Takeaway

The goals of making changes to mechanical ventilation are:

1. Correcting imbalances or issues in the body—often seen on ABGs
2. Progressing the patient towards extubation via decreasing settings, moving to spontaneous modes or getting to minimal settings

Now that their surgery is complete, this OR patient is being suctioned prior to extubation. Suctioning Prior to Extubation by Rafael Ortega, M.D., Christopher Connor, M.D., Ph.D., Gerardo Rodriguez, M.D., & Caresse Spencer, M.D. Used under fair dealing. All Rights Reserved.

9.2 VENTILATION TO CORRECT ABNORMAL ABGS: KEY QUESTIONS

After initiation of ventilation, ABGs are used to assess the effectiveness of the current settings. When approaching ventilation changes to fix an ABG, a clinician has to ask themselves six separate questions:

1. What issues can I fix with ventilation?
2. Which ventilation setting affects which ABG value?
3. What are the problems with the blood gas (ABG interpretation comes in handy here), if any?
4. What settings could I change to fix the problem?
5. Which change(s) would be *most* correct for my patient?
6. How *much* should I change my settings by?

Questions 1 and 2 will help you when you are starting out, but once you master these concepts, you will no longer need to ask them every time. But Questions 3, 4, 5 and 6 are *always* asked for each mechanically ventilated patient.

Over the next several pages, we will walk through each question in detail, with reference to an example patient, to look at how to put all your knowledge together and ventilate a patient effectively.

The capable clinician can put all the pieces together to achieve their goals for their ventilated patient. Photo by outsideclick, Pixabay Licence.

9.3 WHAT CAN I FIX WITH VENTILATION?

Let's review what we have discussed in previous chapters. We know that with mechanical ventilation, we often have to intubate patients who are having difficulty regulating their CO_2 and O_2 levels. Patients can be intubated due to ventilatory failure (increasing CO_2, hypercarbia) or due to hypoxia (decreasing oxygen levels). We have discussed how changing CO_2 levels in the body can affect the overall pH of the body, which we can measure using an ABG. Often, the decision to intubate a patient is directly due to the ABG results that are being seen.

In an ABG, we know that we have values for pH, pCO_2, pO_2 and HCO_3. Which values can be affected by ventilation and which ones cannot?

Apply Your Learning

Think about how the body normally tries to fix acid-base imbalances. Which of the compensation mechanisms is related to breathing? Which value is not related to breathing? If you have difficulty answering, please refer to Chapter 8. Then, read on for the answer...

You guessed it! CO_2 is directly affected by ventilation, while HCO_3 cannot be changed by ventilation and must be regulated by the kidneys. Therefore, the pH can be affected in ventilation only by affecting the CO_2 and not the "bicarb" (bicarbonate). In addition, the pO_2 can also be directly impacted by affecting how much oxygen we are delivering effectively to the lungs.

Key Takeaway

For ABGs with acid-base imbalances, remember that HCO_3 cannot be affected by ventilation. CO_2 is the main component that is affected by breathing and mechanical ventilation. The ultimate goal of fixing ABG acid base issues is to maintain a normal pH. Sometimes you will drive your CO_2 slightly outside of normal ranges to correct an abnormal pH.

During the COVID-19 pandemic, many health care workers who are not respiratory therapists needed to gain increased familiarity with ventilation. Photo by HelenJank, Pixabay Licence.

For most patients, the goal is to normalize $PaCO_2$ in the range of $35 - 45\ mmHg$.

Iatrogenic Hyperventilation

In certain clinical situations, usually involving traumatic brain injury with increase intra-cranial pressure or other neurological pathology, you may encounter the term **iatrogenic hyperventilation.** This is intentional hyperventilation and the idea behind it is that reducing $PaCO_2$ may result in cerebral vasoconstriction and reduced intracranial pressure (ICP). However this vasoconstriction also reduced cerebral blood flow resulting in potential hypoxic injury to the brain. For this reason, this medical strategy is considered controversial and, while it may provide a temporary solution to increased ICP, more robust research is needed to better guide the clinical use of iatrogenic hyperventilation.

Hyperventilation Article

Hyperventilation in neurological patients: from physiology to outcome evidence

Key Takeaway

While **iatrogenic hyperventilation** may provided a temporary solution to increased ICP, $\mathrm{PaCO_2} < 25\ \mathrm{mmHg}$ or during the first 24 hours after traumatic brain injury is not recommended. $PaCO_2$ falls and pH rises resulting in a respiratory alkalosis. Low $PaCO_2$ may lead to unwanted physiological side effects and should be used with caution.

Permissive Hypercapnia

Permissive Hypercapnia is a unique ventilatory strategy that moves away from routine clinical practices. By using small tidal volumes and lower ventilation pressures, this approach aims to minimize alveolar overdistention, reducing the risk for potential complications. The deliberate elevation of $PaCO_2$ levels and the subsequent decline in pH leads to respiratory acidosis. While you may see this strategy used in clinical practice, current evidence shows that hypercapnia may lead to unwanted physiological side effects and should be used with caution.

ARDS Article

Formal guidelines: management of acute respiratory distress syndrome

Key Takeaway

Permissive Hypercapnia allows temporary respiratory acidosis to develop, with pH no less then 7.15 to 7.20, in clinical situations where tidal volume of $6\ \mathrm{mL/Kg}$ IBW or less must be maintained. Hypercapnia may lead to unwanted physiological side effects and should be used with caution.

9.4 WHICH VENTILATION SETTING AFFECTS WHICH ABG VALUE?

At this point in your learning, the terms *ventilation* (the exchange of pCO_2) and *oxygenation* (pO_2) are probably very familiar to you. Remember, pCO_2 and pO_2 are values in an ABG reading. Let's relate these concepts to settings on the ventilator, and learn which specific setting affects ventilation or oxygenation.

We have repeatedly discussed how ventilation refers specifically to the exchange of air in and out of the lungs. In terms of blood gas effects, ventilation directly refers to the removal of CO_2. So, what ventilator settings would affect this? What settings directly impact the amount of air going in and out of the lungs? If you guessed the tidal volume, you are correct! The size of breath will directly impact the amount of air going in and CO_2 coming out of the lungs.

There is another setting that will impact the amount of CO_2 clearance. What do the chemoreceptors in the brain trigger if the CO_2 levels start to rise? If you answered the respiratory rate (RR), then you are remembering correctly! The RR also has direct impact on the amount of CO_2 leaving the lungs over time. If you breathe faster, you are getting rid of CO_2 more often and this will help drive CO_2 levels down.

Increased CO_2 clearance is commonly referred to as **blowing off CO_2**.

Key Takeaway

To affect ventilation, the RR and the tidal volume are the two settings with direct impact on the amount of air into the lungs and CO_2 out of the lungs. Remember $V_T \times RR = V_E$.

What about oxygenation? Which ventilator settings directly impact the patient's oxygenation status? This concept should not be new to you. We have talked extensively about FiO_2 and PEEP as working together to deliver oxygen into the body. The FiO_2 can be increased to deliver higher amounts of oxygen to the lungs, while PEEP can push the oxygen across the alveolar-capillary membrane.

Please note that this is a slight simplification of these concepts. There is some crossover between the ventilator settings that affect oxygenation and ventilation. But for beginner learners, it is better to keep oxygenation and ventilation and the settings that affect them separate in your minds without crossover. With practice you will be able to see the big picture and even make predictions on what order to use ventilation settings.

Take a look at the following table, which summarizes the mechanical ventilation settings that affect oxygen versus ventilation (keep in mind that V_T can also be affected by other settings on a ventilator):

Table 9.4.1: Settings that Affect Oxygenation and Ventilation

Settings that affect Oxygenation	Settings that affect Ventilation
FiO_2	RR
PEEP	V_T

If you ever get the chance to work in flight medicine or medevacs, you may be the sole or lead care provider for a ventilated patient and need to know how to make appropriate decisions. Photo by ArtisticOperations, Pixabay Licence.

9.5 WHAT ARE THE PROBLEMS WITH THE BLOOD GAS?

When you interpret ABG results, you determine whether the pH is balanced and if you have abnormal CO_2, O_2 and HCO_3 levels. The most important thing to remember here is that, as the ultimate goal, you want to leave the body with a normal pH. We cannot cause problems by fixing CO_2 levels when that action will shift the pH further away from normal. Sometimes we need to allow for abnormal CO_2 levels as long as we maintain a correct pH.

Let's look at an example ABG and identify the problems in the gas, if there are any. Then, we'll connect these findings to what we can fix with mechanical ventilation:

Patient | 7.31/57/68/24

Remember, the ABG for this patient is expressed as: $pH/pCO_2/pO_2/HCO_3$. Take a second and interpret this ABG. Remember, you need to comment on:

1. the compensation,
2. whether it is a respiratory or metabolic issue,
3. whether it is acidosis or alkalosis, and
4. the oxygenation status.

Apply Your Learning

How would you interpret this ABG? Do not keep reading until you have made a guess at the answer.

Solution

If you got the answer **uncompensated respiratory acidosis with mild hypoxemia**, you are right!

So, what are the problems in this gas?

- The pH is low and the body is in an acidosis state.
- The pCO_2 is too high and causing the acidosis.
- The pO_2 is low and the patient is hypoxic.

The bicarbonate is normal, so there is no problem here.

Remember, with mechanical ventilation, we can change the O_2 and CO_2 directly, and indirectly affect the pH by changing the CO_2 levels. We cannot affect the HCO_3; that change is done by the body over time.

This is a perfect ABG to affect with mechanical ventilation because we can improve the patient's oxygenation, change how much CO_2 is being exhaled over time and then normalize the pH by that change in CO_2.

Mechanical ventilation is essential to safe surgeries for many. Photo by sasint, Pixabay Licence.

Which settings could I change to fix the identified issue?

The next step to ask yourself is what changes to which settings will improve the gas. You have just identified whatever issue is in the ABG, the next step is to ask yourself what possible changes to each setting could you make?

We have talked about how ventilation and oxygenation both have two distinct settings that can impact CO_2 and O_2 levels, respectively. Let's review:

Table 9.5.1: Settings that affect Oxygenation and Ventilation

Settings that affect Oxygenation	Settings that affect Ventilation
FiO_2	RR
PEEP	V_T

Based on the issues you have identified in the ABG, what direction would you want to move these settings?

Let's look at the settings that affect ventilation first. We know the only change that we can do on a ventilator to impact the overall pH of the patient is their CO_2 level. We have talked about the RR and the V_T as options to change the pCO_2 level and therefore the pH of the patient.

Object Lesson

The best analogy to keep in mind to understand what changes you would make to your RR or V_T in order to adjust CO_2 is to think about being in a boat with a hole in the bottom in the middle of a lake. The water would start rushing into the boat. You would need to start bailing the water out of the boat to decrease the water level and stop the boat from sinking. In this analogy, think of the lungs as the boat and the water level as the CO_2 level in the body. How fast and to what degree you are bailing refers to the removal of CO_2 with every exhalation.

If the water was rising in the boat, you can impact the level of water directly by two options:

- bailing faster, or
- bailing at the same speed but with a bigger bucket.

Let's think back to the lungs here. If you increased your bailing speed, that is the same as breathing at a faster rate. You are exhaling more CO_2 (removing more water) by breathing faster. Conversely, if you maintained the same breathing rate (speed of bailing), you could get rid of more CO_2 by getting rid of more air per breath (a bigger bucket). This would refer to taking a bigger tidal volume with every breath.

Keep this analogy in mind when we start going through examples.

As you learned in the object lesson above, you can decrease the CO_2 by increasing RR or V_T. Conversely, if the CO_2 level is too low, the reverse is true: you can decrease RR or set a smaller V_T.

Now let's look at oxygenation. This one is pretty intuitive: If the patient's oxygen level is too low, giving them a higher FiO_2 of oxygen or a higher PEEP to push the oxygen across the alveolar-capillary membrane will improve their oxygen level. Conversely, if their oxygen level is too high, decreasing the oxygen level supplied (never below 0.21) and decreasing the PEEP can decrease their oxygen level in their blood.

Let's continue to look at our example patient...

Patient | 7.31/57/68/24

We have identified the problems in the ABG as follows:

- pH is acidotic—this is the primary goal to fix
- pCO_2 is too high—we will change this to fix the pH
- pO_2 is too low

In this blood gas, we have a ventilation problem (high CO_2 leading to an acidotic pH) as well as an oxygenation problem (low pO_2). So, which changes *could* be made to fix the issues?

The body is not clearing enough CO_2 with the patient's current breathing. Think back to the analogy of bailing out the sinking boat. In this situation, the water level in the boat is too high. What can you do to your bailing? Increase the frequency of bailing, or get a bigger bucket. Therefore, our possible ventilation changes are as follows:

- you could increase the RR, or
- you could increase the tidal volume to clear more CO_2.

Now, let's look at the pO_2 issue with this patient's ABG results. The oxygen level is too low, and the patient has a mild hypoxia. You need to get more oxygen into the blood either by delivering a higher amount of oxygen or by pushing it harder across the alveolar-capillary membrane to get more into the blood per breath. Therefore, your possible oxygenation changes are:

- you could increase the FiO_2 delivered, or
- you could increase the PEEP to help push the oxygen across the membrane and recruit other alveoli.

Now that you have identified all of your options, you are able to narrow your focus to the best choices for your specific patient. Time to move on to your next question...

9.6 WHICH CHANGE(S) WOULD BE MOST CORRECT FOR MY PATIENT?

For oxygenation and ventilation issues, you usually have two different settings that could fix whatever problem you have identified in the ABG. Usually, when making changes on the ventilator, a clinician will choose to change just *one* of the two options and then recheck the ABG before making additional changes. So, here is the most important question to ask yourself: which setting is the *most* appropriate to change?

Are both settings an option? Rarely, either setting could be chosen, but in most cases, changing one setting makes more sense than adjusting the other. Through this book, you have learned all of these situations and it is time to put your knowledge together to learn how to make informed decisions for your patient.

Ventilation issues: RR or V_T

If the pH is abnormal and you are going to try to normalize it by increasing or decreasing the pCO_2 level, you know V_T and RR are both options to impact the amount of CO_2 being exhaled every breath—but which change is most correct?

The answer depends on where you are currently with your ventilator settings. Remember the safe ranges for RR? Medical providers with basic ventilator knowledge are encouraged to start with an RR between $14 - 18\ \text{bpm}$, with a caution to not go above an RR of $24\ \text{bpm}$ without guidance from physicians or RRTs due to the impact on adequate time to exhale and the danger of causing patient asynchrony. If the RR is above $14\ \text{bpm}$ and below $18\ \text{bpm}$, you have flexibility to go up or down safely within the range of $10 - 24\ \text{bpm}$ without causing issues.

Now think about your tidal volume ranges. This parameter is much more definitive with its allowances. The safe tidal volume ranges for medical providers with basic ventilator knowledge is $6 - 8\ \text{mL/Kg}$. Remember, we do not go above $8\ \text{mL/Kg}$—that is an absolute maximum. Using the knowledge you have of ideal body weight (IBW) and calculated safe tidal volume ranges, you would compare the tidal volume your patient is getting to your calculated ranges. Do you have room to move to correct the problem? If you do, this would be an option to take. However, if you are at either limit, then changing the tidal volume would not be an option to correct the problem.

Key Takeaway

Do you choose V_T or RR? If you are at the low ends of your safe ranges for both, then either RR or V_T can be adjusted. If you are at the higher ends of either one, then use the other setting instead. If you are at the high end of both RR and V_T, remember that V_T is a hard limit, while RR can still be adjusted carefully with physician or RRT input. If you are already at the low ends of V_T and RR, and you need to move your patient even lower, instead, consider moving to the next step towards weaning (See Chapter 10.1).

Oxygenation issues: FiO_2 or PEEP?

When it comes to changing either FiO_2 or PEEP, you need to think about the impacts of either of these values on the body. Remember, high levels of oxygen can cause lung damage, and we are always targeting the lowest FiO_2 to maintain $SpO_2 > 92\%$ and a pO_2 at normal ranges $(80 - 100\ mmHg)$. FiO_2s of higher than 0.5 can lead to oxygen damage to the lungs (See Chapter 2).

For PEEP, there is a hard minimum of $5\ cmH_2O$—you will never decrease the PEEP below that number. Conversely, increasing PEEP too high can start to negatively impact the body. Remember Chapter 2? We talked about how high levels of PEEP can negatively impact the compliance of the lungs—just like balloons that are already inflated with pressure could lose elasticity and not be able to inflate as easily. High PEEPs will also increase the pressure in the alveoli that, when added to the additional volume or pressure applied with every breath, could increase your patient beyond safe pressure levels and put the lungs at risk of barotrauma. We need to maintain pressures of less than $35\ cmH_2O$—and, ideally, the lower, the better.

PEEP also increases the pressure in the thorax (chest) of the patient. Other than the lungs, this cavity also houses your heart and important vessels like the aorta and vena cava. Increased **intrathoracic pressure** will increase the pressure on these vessels as well. This pressure could squeeze the heart, decreasing the blood flow back into the heart and the pumping effectiveness of the heart. The medical terms for these situations are **venous return** and **ejection fraction** of the heart. Both conditions will show an impact in the blood pressure of the patient.

Blood pressure is one way to assess issues like venous return and ejection fraction of the heart. Photo by Netha Hussain, CC0

When selecting FiO_2 or PEEP as the setting to change, a good rule of thumb when treating hypoxia is to increase FiO_2 until you approach $FiO_2\ 0.50$, and then slowly increase PEEP by $1 - 2\ cmH_2O$ until you get to a moderate level $(10 - 12\ cmH_2O)$ to facilitate oxygenation. If your patient is still hypoxic, continue to increase FiO_2. Remember to consult a physician or the most responsible health care professional if requiring higher PEEPs and $FiO_2 > 0.50$.

When weaning FiO_2, go in reverse. Wean FiO_2 until you are below 0.5; then, slowly decrease PEEP until you reach approximately $8\ cmH_2O$ before weaning FiO_2 again.

At any time, if increasing PEEP causes a decrease in blood pressure below acceptable levels (20% below starting levels), return it to the previously documented amount and titrate FiO_2 to achieve SpO_2.

Let's go back to our example patient...

Example – Patient | 7.31/57/68/24

Let's say your patient is being ventilated in volume control.
Their settings are: $RR = 16\ \text{bpm}, V_T = 420\ \text{mL}, \text{PEEP} = 5, \text{FiO}_2 = 0.5$. Their IBW is calculated at $52\ \text{Kg}$.

What settings would be most correct to adjust to fix the issues of acidotic pH, high pCO_2 and low O_2?

Looking at their RR, you definitely have room to adjust it up as the patient is only at $16\ \text{bpm}$, and you can safely increase the RR up as high as $24\ \text{bpm}$ with minimal concerns. This would be an appropriate change.

Looking at their V_T of $420\ \text{mL}$, compare it to the safe V_T range of $6\text{ - }8\ \text{mL/Kg}$ of IBW. The safe tidal volume range for a $52\ \text{Kg}$ person is $312\text{ - }416\ \text{mL}$. You are already at the high range for this patient, and are ventilating at $8\ \text{mL/Kg}$. In this case, you cannot increase the V_T to help blow off the CO_2 and correct the ABG. This would be an inappropriate change.

What about the low pO_2? The patient is getting a $\text{PEEP } 5$ and $\text{FiO}_2\ 0.5$. PEEP is at the minimum, and we definitely have the ability to increase that number to assist. Before you decide to take this action, it's a good idea to check the FiO_2.

What about the FiO_2? Though we can increase the FiO_2 up to 1.0, we know that oxygen damage can occur at levels higher than 0.50. So, in this case, a change to PEEP would be a better option.

A word of caution here: We know that severe hypoxia needs to be fixed. All of our cells need oxygen to survive. FiO_2 has a rapid effect within a few breaths, while PEEP can take up to one hour to start to fully work. If there is moderate to severe hypoxia (pO_2 is less than $60\ \text{mmHg}$). Increasing the FiO_2 to get immediate results while increasing PEEP, and then dialing back the FiO_2 when SpO_2 recovers would be the *most* correct. In this case, the hypoxia is only mild $(60\text{ - }79\ \text{mmHg})$, so increasing the PEEP only might be enough as long as the patient maintains $\text{SpO}_2 > 92\%$. If needed, you can also make a small increase to FiO_2 while the PEEP increase is starting to work, and then wean the FiO_2 back down when the SpO_2 improves enough.

Making sense so far? There are only two more question to work through...

9.7 HOW MUCH DO I CHANGE VENTILATION SETTINGS BY?

We usually choose one value to change at a time and then test the ABGs and SpO_2 again to assess the changes. But, how much do we change each setting by to see changes?

Remember the factors affecting alveolar ventilation are: respiratory rate, tidal volume and dead space.

$$V_A = (V_T - V_{Dphys}) \times RR$$

adjustments to ventilation.

Example

$\text{Known PaCO}_2 = 50 \text{ mmHg}$,
$\text{Known } V_E = 7 \text{ Lpm}$

$\text{Desired PaCO}_2 = 40 \text{ mmHg}$

What V_E is required to achieve a $PaCO_2$ of 40 mmHg?

Solution

$$\text{Desired } V_E = \frac{\text{Known PaCO}_2 \times \text{Known } V_E}{\text{Desired PaCO}_2}$$

$$\text{Desired } V_E = \frac{50 \times 7}{40}$$

$$\text{Desired } V_E = 8.75 \text{ Lpm}$$

Arterial partial pressure of CO_2 ($PaCO_2$) is considered the best indicator of ventilation. As we previously learned, there is an inverse relationship between alveolar ventilation and $PaCO_2$.

As alveolar ventilation increases, $PaCO_2$ decreases.

For most patients, the goal is to normalize $PaCO_2$ in the range of $35 - 45 \text{ mmHg}$.

Assuming, there is no change in a patient's CO_2 production and dead space, and we have full control of ventilation, the following equation can be used to quantify the change in ventilation parameters, to target a specific $PaCO_2$. This equation is especially useful as we navigate this chapter and practice making

$$\text{Known PaCO}_2 \times V_E = \text{Desired PaCO}_2 \times V_E$$

Note that this equation provides us with the value of minute volume required, however, to make ventilation adjustments on the ventilator, we must change either tidal volume (V_T) or respiratory rate (RR), as most ventilators and modes don't have a minute volume setting. Which one do we choose: tidal volume or respiratory rate?

Changing Tidal Volume

Keep in mind that under normal circumstances we are targeting a tidal volume of $6 - 8 \text{ mL/Kg IBW}$. If the known V_T is 6 mL/Kg, we may consider increasing the tidal volume as long as plateau pressure does not increase above $30 \text{ cmH}_2\text{O}$.

Changing Respiratory Rate

If tidal volume is already on the high side, at 8 mL/Kg, and/or plateau pressure is greater than $30 \text{ cmH}_2\text{O}$, perhaps a better choice is to increase the respiratory rate to achieve desired minute volume. Whenever rate is increased, care must be taken to allow patient sufficient expiratory time to avoid air trapping.

Key Takeaway

Strategies to Increase/Decrease V_T

In Volume Control Ventilation, set V_T. Consider other factors that might affect V_T depending on ventilator features for volume control (Flow and Inspiratory time may have to be adjusted to achieve desired V_T).

In Pressure Control Ventilation, target tidal volume can be achieved (increased or decreased) by adjusting ΔP or inspiratory time.

Strategies to Increase/Decrease RR

Change the set RR.

Change Inspiratory time or $\mathrm{I{:}E}$ ratio.

With practice and experience, ventilation adjustments are often done by using simple logic in clinical practice, often when other factors impact acid-base balance, including CO_2 production and dead space. A good approach to stepwise changes is the following:

Table 9.7.1: Guidelines for Stepwise Changes

Setting	Guideline of each stepwise change	Limits and Cautionary Ranges
RR	± 2 bpm	10 - 24 bpm (higher if needed, with caution)
V_T	± 1 mL/Kg (between 6, 7, 8 mL/Kg)	Min 6 mL/Kg, Max 8 mL/Kg
$\mathrm{FiO_2}$	± 0.1 or 10% $\mathrm{O_2}$	Max 1.0 (use caution over 0.5)
PEEP	± 1 - 2 $\mathrm{cmH_2O}$	5 - 12 $\mathrm{cmH_2O}$ (higher if needed, with caution)

Let's return once more to the example patient.

Example – Patient | 7.31/57/68/24

Vent settings: $RR = 16$ bpm, $V_T = 420$ mL, $\mathrm{PEEP} = 5$, $\mathrm{FiO_2} = 0.5$

Known information: $\mathrm{IBW} = 52\ \mathrm{Kg}$. When calculated using the safe V_T range of $6\text{ - }8\ \mathrm{mL/Kg}$ equals a safe tidal volume range of $312\text{ - }416\ \mathrm{mLs}$ for this patient.

When answering the previous questions, you already decided that you needed to fix the pH by blowing off more CO_2. Though the two options would be increasing the RR or increasing the tidal volume (think of the analogy of the rising water and needing to bail either faster or with a bigger bucket), you determined you cannot increase the V_T since the patient is at the maximum tidal volume of $8\ \mathrm{mL/Kg}$. Therefore, the only change you can make is increasing the RR to decrease the pCO_2 and correct the pH. A practitioner would most likely choose to increase the $RR + 2\ \mathrm{bpm}$ and set it at $18\ \mathrm{bpm}$.

In addition, you already know that your patient has mild hypoxia, and you decided that although you can increase either PEEP or FiO_2 to fix this issue, since we are already at an FiO_2 of 0.5, it might be worthwhile to just increase the PEEP as long as the $\mathrm{SpO_2} > 92\%$. A practitioner would most likely increase the PEEP to $7\ \mathrm{cmH_2O}$. If the SpO_2 is less than 92%, the FiO_2 might be increased to 0.6 for about 30 minutes and weaned as soon as the PEEP change starts to impact the patient.

You have just learned the typical amounts each setting is adjusted to impact a change on an ABG. Sometimes, ABGs will show mild imbalances, while other times, the issues are quite significant. If the numbers on the ABG are profoundly off, it might be worthwhile to do two steps of changes.

When completing two stages of changes, the same rules still apply—only some changes will be appropriate for your patient. What this approach looks like in practice is changing two settings (if able) or doing two-step changes to the same settings. For example, a significant issue would be if your pH is less than 7.3 or greater than 7.5 and your pO_2 is less than 55. In these cases, if the RR was the only change you could make, you might consider changing it to $20\ \mathrm{bpm} (16 + 2 + 2 = 20\ \mathrm{bpm})$, and you would definitely increase your $\mathrm{FiO_2}\ +0.1$ and consider your $\mathrm{PEEP}\ +2$ as well. Remember, PEEP is slower to work, and the hope is you would bring the FiO_2 back down as soon as PEEP starts to work.

This RN is proud of the skills that allow her to improve patient outcomes. Photo by Laura James, Pexels Licence.

9.8 HOW MUCH DO I CHANGE OXYGENATION SETTINGS BY?

Acute Respiratory Distress Syndrome (ARDS) poses the most extreme oxygenation challenge in the domain of mechanical ventilation. An extensive body of literature on oxygenation support during ventilation predominantly centers around addressing the complexities of ARDS. This critical condition demands special attention and strategic management to optimize oxygenation and improve patient outcomes. As such, the focus on ARDS in ventilation literature reflects the significance of this condition and the ongoing efforts to enhance therapeutic approaches.

Let's first review ventilation settings that affect oxygenation, mainly FiO_2 and PEEP.

Adjustments of FiO_2 to Improve Oxygenation

Depending on the cause of hypoxia, it may take some time for a patient to respond to oxygen therapy. For example in hypoxemia caused by circulatory problems or anemia, supplemental oxygen may be necessary until the underlying cause is resolved.

In the realm of oxygenation assessment, a plethora of parameters exists, each influenced by the specific clinical site protocols and patient oxygenation requirements. The table below lists common parameters useful in assessing oxygenation status. However, across all practice settings, a rapid and universally embraced bedside evaluation of oxygenation status can be conducted employing the following key parameters: PaO_2, SpO_2 and P/F ratio (the ratio of PaO_2 to FiO_2).

Table 9.8.1: Commonly Used Oxygenation Indices

Commonly Used Oxygenation Indices		
Arterial partial pressure of O_2	PaO_2	80 - 100 mmHg
Arterial oxygen saturation	SaO_2	$>92\%$
PaO_2/FiO_2	P/F	380 - 475 (when PaO_2 is normal and FiO_2 is 0.21)
Alveolar partial pressure of oxygen	PAO_2	100 - 673 mmHg (when FiO_2 is 0.21 to 1.0)
Alveolar-arterial oxygen tension gradient	$P(A\text{-}a)O_2$	5 - 10 mmHg on FiO_2 0.21 30 - 60 mmHg on FiO_2 1.0
Mixed venous partial pressure of O_2	PvO_2	40 mmHg
Saturation of mixed venous O_2	SvO_2	75%

Similar to adjusting ventilation parameters, arterial blood gases are initially used to determine presence and level of hypoxia, based on PaO_2 and SaO_2. When the patient's cardiovascular status is relatively stable, there is a linear relationship between FiO_2 and PaO_2. This relationship can be used as a guideline to determine necessary FiO_2 adjustment based on the following equation:

$$\frac{\text{Known } PaO_2}{\text{Known } FiO_2} = \frac{\text{Desired } PaO_2}{\text{Desired } FiO_2}$$

$$\text{Desired } FiO_2 = \frac{\text{Known } FiO_2 \times \text{Desired } PaO_2}{\text{Known } PaO_2}$$

This method offers a simplified approach for determining FiO_2 when the patient's condition is stable. Nonetheless, it's essential to acknowledge that oxygenation is frequently a complex and challenging process often requiring multiple adjustments, typically in increments of $10\% - 20\%$, to achieve the desired outcome. Additionally, identifying the root cause of hypoxia is crucial in effectively managing oxygenation issues.

The harmful effects of prolonged FiO_2 levels exceeding 0.60 are widely recognized, as they can lead to oxygen toxicity. Therefore, whenever a patient's oxygen requirements surpass this threshold, or even as a preventive measure, it is crucial to consider adjusting Positive End-Expiratory Pressure (PEEP). By exploring PEEP adjustments, we can effectively manage and optimize oxygenation while minimizing the risk of oxygen-related complications.

Adjustment of PEEP to Improve Oxygenation – Optimal PEEP

Optimal PEEP is defined as the level at which the majority of alveoli remain inflated at the end of expiration. This critical point represents the PEEP setting that provides the maximum beneficial effects, including increased Functional Residual Capacity (FRC), enhanced alveolar recruitment, and optimal static compliance. At this specific PEEP level, the respiratory system achieves its best performance and efficiency ensuring improved lung function and oxygenation.

A number of different methods are currently used in practice, depending on the clinical site protocol and resources, you may encounter all or some of the following during your clinical placement:

- Incremental or Decremental PEEP studies
- Esophageal pressure monitoring
- Pressure Volume Curves
- PEEP – FiO_2 tables

Regardless of the method used, setting optimal PEEP is often challenging and it should be set based on individual patient lung condition and disease process.

Incremental or Decremental PEEP Studies

During a PEEP study, it is essential to exercise caution as the end-expiratory pressure is incrementally increased. Diligent monitoring of the patient's hemodynamic status becomes imperative to prevent potential cardiovascular compromise that may arise from elevated pressures in the lungs. At a minimum, continuous monitoring of the patient's blood pressure is warranted, as a PEEP study may lead to a reduction in cardiac output with subsequent drop in blood pressure.

In addition to careful attention to hemodynamics, comprehensive monitoring of the patient's ventilatory status is equally important. Monitoring parameters such as peak inspiratory pressure, plateau pressure, tidal volume, and arterial blood gases allows

for a thorough assessment of the patient's response to the PEEP study. Other oxygenation and cardiovascular indices can be monitored, when resources are available.

To perform an incremental PEEP study, PEEP is increased in $2\ \mathrm{cmH_2O}$ increments every 15 minutes, while estimating static compliance and monitoring selected ventilation and hemodynamic parameters to assess the adequacy of oxygenation and the function of the cardiovascular system.

To perform a decremental PEEP study, as the name suggests, we start with a higher PEEP obtained with a **lung recruitment maneuver**. From there, PEEP is reduced to $25\ \mathrm{cmH_2O}$ and then decreased in increments of $2\ \mathrm{cmH_2O}$.

A decrease in compliance or oxygen saturation at a certain level of PEEP implies decruitment and that PEEP should be increased to the previous level.

Esophageal Pressure Monitoring to set Optimal PEEP

Esophageal manometry allows measurement of esophageal pressure which indirectly reflects pleural pressure. You will remember from previous chapters that the pressure responsible for alveolar inflation is the transpulmonary pressure $(P_{alv} - P_{pl})$. The most common way to measure Pes is using an air-filled balloon integrated into an esophageal catheter that is placed in the esophagus. Knowing pleural pressure, we can set PEEP accordingly to achieve a transpulmonary pressure of zero. Watch this video for a demonstration of using transpulmonary pressure to set optimal PEEP.

One or more interactive elements has been excluded from this version of the text. You can view them online here: https://ecampusontario.pressbooks.pub/mcvresource/?p=1787#oembed-1

Video: "Accurately setting PEEP with transpulmonary pressure" By Hamilton Medical [5:09] *Transcript Available*

Pressure Volume Curves (Loops) to set Optimal PEEP

Ventilators equipped with this feature perform a respiratory mechanics maneuver by delivering a quasi-static PV (pressure/volume) curve at a low flow rate, typically less than $6\ \mathrm{Lpm}$. As we previously learned, the components of a PV curve can help us improve ventilation. It allows for determination of the lower and upper inflection points. When this method is employed, PEEP is set $2\ \mathrm{cmH_2O}$ above the lower inflection point. Many clinicians recommend that PEEP be set on the exhalation limb of the PV loop, as more volume is present in the alveoli during exhalation for a certain pressure compared to the inspiratory limb. What stays open during exhalation will be easier to inflate during the next inhalation, requiring less driving pressure. Research is continuously being done to determine best practices on setting optimal PEEP.

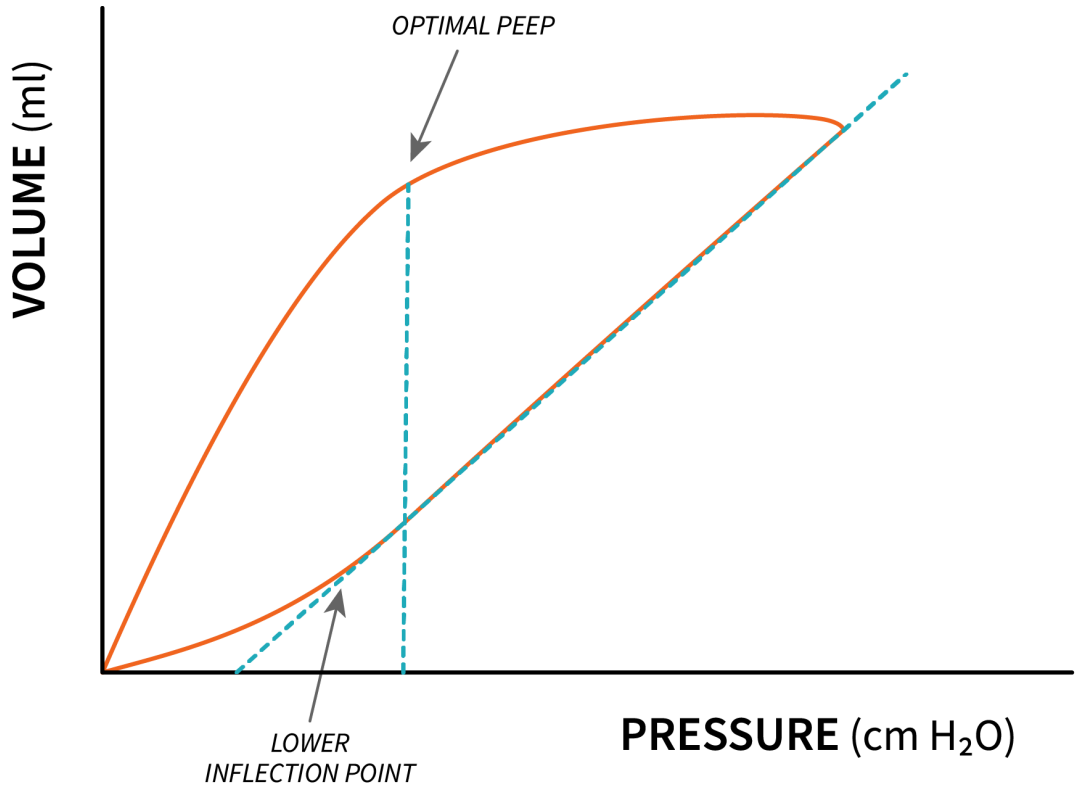


Figure 9.8.1: "PV Curve for Optimal PEEP" by Freddy Vale, CC BY-NC-SA 4.0. Low Flow (<6 Lpm) ensures a PV Curve close to a static curve.

The shape of PV loops can also be used to determine if lungs are recruitable. For a lung to be deemed recruitable, presence of hysteresis and gain in volume is required. A large hysteresis indicates potential for recruitment, while a small or absence of hysteresis indicates the lung is not recruitable.

A **lung recruitment maneuver** (LRM) is another tool to improve oxygenation, and it involves an increase in pressure in the lung for a longer period of time than a normal respiratory cycle. Common approaches to LRM is $30\ cmH_2O$ for $30\ seconds$ or $40\ cmH_2O$ for $40\ seconds$. Due to the risk of increasing intrathoracic levels, careful monitoring of patients during this procedure is required, and the maneuver should be aborted at the first sign of cardiovascular compromise.

Electrical impedance tomography (EIT) is available to titrate PEEP to achieve the greatest volume in the lung (seen as greatest area of air in the lung with EIT), however, this method may not be available in all institutions.

Finding Optimal PEEP Article

This article provides a concise summary of the history of research on finding optimal PEEP and the need for further research.

In search of the Holy Grail: identifying the best PEEP in ventilated patients

9.9 VENTILATION IN ACUTE RESPIRATORY DISTRESS SYNDROME

Acute Respiratory Distress Syndrome (ARDS) is a complex clinical syndrome that can become a severe and life-threatening condition that affects the lungs, leading to acute respiratory failure. It is characterized by rapid and profound inflammation of the alveoli, which makes it difficult for oxygen to enter the bloodstream and carbon dioxide to be eliminated properly. While there is extensive literature available on the pathophysiology of ARDS, we will focus on ventilation management strategiesin this book .

Ventilating patients with ARDS represents one of the biggest challenges for clinicians in the ICU. Even tough advances in mechanical ventilation and critical care medicine have been made in the past years, mortality due to ARDS in hospitals is still high. The intricate nature of maintaining adequate oxygenation in these patients has spurred extensive research into achieving optimal PEEP, with the ultimate goal of opening the lung and improving respiratory function. The concept of "**open lung ventilation**" is sometimes intertwined with ARDS ventilation, as some clinicians strive to maximize lung recruitment and enhance gas exchange in these critically ill patients. Emphasizing this close relationship between open lung ventilation and ARDS management underscores the vital importance of refining ventilation strategies to enhance patient outcomes. To differentiate ARDS from other pathologies with similar presentation, an international consensus group of critical care experts has established the most up to date definition of the disease. Because this group met in Berlin, Germany, this definition is known as the Berlin definition of ARDS, 2012.

Table 9.9.1: ARDS Berlin Definition

	ARDS Berlin Definition
Timing	Within 1 week of original insult or new or worsening respiratory symptoms
Chest Imaging	Bilateral opacities not fully explained by effusions, lobar/ lung collapse, or nodules.
Origin of edema	Respiratory Failure not fully explained by cardiac failure or fluid overload. Need objective assessment to exclude hydrostatic edema if no risk factors are present
Oxygenation	**Mild:** $200 \text{ mmHg} < PaO_2/FiO_2 \leq 300 \text{ mmHg}$ with PEEP or CPAP $\geq 5 \text{ cmH}_2O$ **Moderate:** $100 \text{ mmHg} < PaO_2/FiO_2 \leq 200 \text{ mmHg}$ with PEEP or CPAP $\geq 5 \text{ cmH}_2O$ **Severe:** $PaO_2/FiO_2 < 100 \text{ mmHg}$ with PEEP or CPAP $\geq 5 \text{ cmH}_2O$

Data Source: The ARDS Definition Task Force, 2012.

The initial management of ARDS, of course, involves addressing and stabilizing the underlying disease that triggered its onset. Current guidelines from the ARDS network recommend a cautious approach in managing the injured lung, utilizing ventilation techniques characterized by low tidal volume and pressure known as **Lung Protective Ventilation**.

Key Takeaway

The goals of mechanical ventilation in the management of ARDS are to support oxygenation, avoid circulatory compromise, and avoid ventilator-induced lung injury. Lung protective ventilation includes guidelines that attempt achievement of these goals.

The therapeutic approach is based on the severity of lung damage and injury. As soon as lung injury is suspected, low tidal volume should be considered (on average $6\ \mathrm{mL/Kg}$, with a range from $4 - 8\ \mathrm{mL/Kg\ IBW}$). As the disease progresses, and oxygenation worsens, it becomes crucial to find optimal PEEP and titrate FiO_2 to maintain $PaO_2 > 55\ \mathrm{mmHg}$. For more information, please refer to the previous section, covering methods for finding optimal PEEP. The low volumes and pressure used with lung protective ventilation can become uncomfortable for patients, leading to patient-ventilator asynchrony. This ventilation strategy may also lead to respiratory acidosis with higher than normal $PaCO_2$, often leading clinicians to allow permissive hypercapnia in certain cases.

As clinicians learn more about management of ARDS, research brings new evidence into practice, so guidelines and recommendations change often, and the definition of ARDS is being challenged to include patients treated with high flow nasal cannula prior to intubation. The European Society of Intensive Care Medicine has recently published the latest guidelines.

ESICM 2023 Recommendations for ARDS Ventilation

- Use low tidal volume ventilation $(4 - 8\ \mathrm{mL/Kg\ IBW})$
- Do not use prolonged high-pressure recruitment maneuvers
- Use prone position to reduce mortality
- Do not routinely use continuous infusions of neuromuscular blockade
- Refer patients who meet criteria for ECMO (extracorporeal membrane oxygenation) to ECMO centers

Please read the following article for more information on ESICM guidelines for ARDS:

ESICM guidelines on acute respiratory distress syndrome: definition, phenotyping and respiratory support strategies

Prevention really is the best medicine ! Whenever possible, intubation and ventilation should be avoided. Early recognition and diagnostic is important. For non-intubated patients with acute hypoxemic respiratory failure not due to cardiogenic pulmonary edema or chronic lung disease, High-flow nasal oxygen (HFNO) is recommended versus conventional oxygen therapy to reduce risk of intubation.

So, how do we ventilate ARDS patients?

There is no strong evidence to support one ventilation mode versus the other. However, in order to follow the recommended guidelines for lung protective ventilation and observe patient safety, the ventilation mode employed for each patient must deliver the tidal volume recommended of 4 - 8 $\mathrm{mL/Kg\ IBW}$ with minimal pressure while ensuring adequate gas exchange. Refer to Chapter 5 for a review of basic ventilation modes.

Airway Pressure Release Ventilation (APRV)

One ventilation mode we have not yet covered, often referred to as an advanced mode, plays a role in ventilating patients with ARDS— Airway Pressure Release Ventilation (APRV). This mode has shown promising results in managing patients with oxygenation challenges, as it facilitates alveolar recruitment, thereby promoting the "open lung concept."

APRV's efficacy stems from its ability to enhance alveolar recruitment, allowing for the optimal expansion of collapsed lung areas. By periodically releasing airway pressure, APRV permits the lungs to reach a more natural equilibrium, promoting better gas exchange and oxygenation.

The evidence supporting the success of APRV in ARDS patients is substantial. This ventilation mode has proven effective in enhancing oxygenation and respiratory compliance, ultimately contributing to improved patient outcomes.

The terminology we are accustomed to using when describing ventilation modes changes slightly in APRV.

APRV can be described as a pressure controlled mode that uses inverse ratio ventilation. While up to this point we discussed $\mathrm{I{:}E}$ ratio of $1{:}2$ or $1{:}3$, in APRV, we are looking at $\mathrm{I{:}E}$ ratio of $4{:}1$ or higher. Now, if you try to follow this pattern for your own breathing, inhale for 5 seconds and exhale for 1, then inhale again for 5 seconds and repeat, you will soon become uncomfortable. Advances in technology allowed ventilator manufactures to add features that allow patients to breathe spontaneously when this mode is used.

Table 9.9.2: Ventilation Settings in APRV

Ventilation Settings in APRV
P_{high}: 25 - 35 $\mathrm{cmH_2O}$ (keep $<$40 $\mathrm{cmH_2O}$)
P_{low}: 0 - 5 $\mathrm{cmH_2O}$
T_{high}: 5.2 - 5.6 s
T_{low}: 0.5 - 1.0 s
$\mathrm{FiO_2}$ based on $\mathrm{SpO_2}$, $\mathrm{PaO_2}$, P/F

Figure 9.9.1: PC-APRV with spontaneous breathing on Phigh level by Drägerwerk AG & Co. KGaA. Used under fair dealing. All Rights Reserved.

The concept behind this approach is to elevate the mean airway pressure to open the lung effectively. With this ventilation mode, the ventilator establishes a higher pressure level, which serves as the baseline for all breaths. This baseline pressure, known as P_{high}, plays a crucial role in increasing mean airway pressure by extending the inspiratory time.

To facilitate gas exchange and CO_2 removal from the lungs, the ventilator allows a controlled release of the high pressure to a predetermined lower pressure for a brief period, referred to as P_{low}. In this context, we replace conventional ventilation settings such as inspiratory time (T_i) with Time high (T_{high}) and expiratory time with Time low (T_{low}).

You will remember from discussing conventional ventilation modes, such as pressure control and volume control, that the length of the expiratory time was set so to avoid air trapping (intrinsic PEEP). If you look closely at the above diagram depicting the pressure-time waveform of APRV, you will realize that time for expiration (release) is very short, likely causing air trapping. APRV, in fact, relies on intrinsic PEEP to maintain alveolar recruitment (open lung).

It is worth noting that this mode does not rely on setting a specific respiratory rate. Instead, it allows patients to take spontaneous breaths during both T_{high} and T_{low}, thereby contributing to the elevation of mean airway pressure. This approach particularly benefits patients who are capable of spontaneous breathing. However, in cases where patients are unable to breathe spontaneously, the respiratory rate is determined by the settings of T_{high} and T_{low}.

There is also no setting for tidal volume in APRV. Similar to any pressure controlled mode, tidal volume is determined by pressure—the setting (ΔP).

Example

For a patient who is unable to take spontaneous breaths, respiratory rate is determined by T_{high} and T_{low} settings:

$T_{high} = 5.5 \text{ s and } T_{low} = 0.5 \text{ s} \quad \text{TCT} = T_{high} + T_{low} = 6 \text{ s}$

We know $\text{TCT} = \dfrac{60}{RR}$

$$\text{TCT} = \frac{60}{RR}$$
$$RR = \frac{60}{\text{TCT}}$$
$$RR = \frac{60}{6}$$
$$RR = 10 \text{ bpm}$$

Here is a video demonstration of APRV ventilation:

One or more interactive elements has been excluded from this version of the text. You can view them online here: https://ecampusontario.pressbooks.pub/mcvresource/?p=1789#oembed-2

Video: "APRV Lung Demonstration" from Charles Bruen on Vimeo [0:31]

Object Lesson

Imagine the movement of air in and out of the lungs like the ocean with a high tide and low tide. Start with P_{high} as the high tide. At certain time intervals, the tide retreats briefly to a lower level (P_{low}). During this "low tide" phase, gas exchange and CO_2 removal occur, giving the lungs a chance to "breathe."

Just as the tide constantly shifts, APRV continues this rhythmic cycle of elevated pressure (P_{high}) followed by a brief

release (P_{low}), encouraging spontaneous breaths like waves lapping at the shore. This natural flow, driven by patient effort, further contributes to optimizing lung function and increasing mean airway pressure.

One or more interactive elements has been excluded from this version of the text. You can view them online here: https://ecampusontario.pressbooks.pub/mcvresource/?p=1789#oembed-1

Video: "APRV Lung Demonstration" by Niccolò Turrioni [0:31]

Ventilation Adjustments and Troubleshooting in APRV

Just like in conventional ventilation, the primary objectives of maintaining ventilation with APRV are twofold: to restore a balanced acid-base status and to ensure the comfort of the patient. Naturally, the ultimate aim remains to liberate the patient from mechanical ventilation as swiftly as is feasible.

To manage oxygenation problems, consider increasing mean airway pressure (MAP). Refer to the pressure-time waveform for a visual understanding of parameters that impact MAP.

To improve *oxygenation*:

Key Takeaway

Increasing MAP recruits more alveoli, opening up the lung. This can be achieved by increasing the area under the pressure-time waveform.

- Increase ΔP, by increasing P_{high}
- Decrease P_{low} (if not already at zero)
- Increase T_{high}
- Decrease T_{low}
- Decrease sedation to allow spontaneous breathing

FiO_2 should be kept at minimum; however, this parameter can be manipulated when MAP cannot be increased safely.

As oxygenation improves, consider decreasing MAP, in a "drop and stretch" style approach. "Drop" (decrease) ΔP and "stretch" (increase) T_{high} to reduce the number of releases. Consider changing the mode to a spontaneous mode such as pressure support ventilation when P_{high} is around 15 - $16\ cmH_2O$.

Ventilation ($pACO_2$) is influenced by minute volume, which is determined by tidal volume and respiratory rate. To make adjustments that impact ventilation in APRV, consider changing:

- Tidal volume moved with pressure releases (ΔP) (Ex. if $PaCO_2$ is high, consider increasing ΔP)
- Number of pressure releases
- Allow spontaneous breaths (to add to minute volume)

Note that spontaneous breaths can be supported with pressure support ventilation.

Additional Supportive Therapy in ARDS

The heterogeneity of ARDS with the subsequent impairment in gas exchange, especially oxygenation, often requires additional measures to supporting ventilation.

Prone ventilation is a technique that has been shown to improve oxygenation and outcomes in certain cases of respiratory failure. It involves placing the patient in a face-down (prone) position on the bed, as opposed to the usual supine (face-up) position.

Figure 9.9.2: CCTC team members work together to pronate a ventilated COVID-19 patient to assist with breathing. Prone Position in ICU by London Health Sciences Centre, used under fair dealing. All Rights Reserved.

The primary goal of prone ventilation is to enhance oxygenation and gas exchange by redistributing the ventilation and perfusion patterns in the lungs. Prone positioning helps to recruit under-ventilated lung regions and improve the matching of lung ventilation with blood flow, thus increasing oxygen uptake and reducing shunting. For a quick review of Ventilation/perfusion patterns in the lungs, see Chapter 7.1.

By enhancing gas exchange, prone positioning can help alleviate ventilator-induced lung injury and decrease the risk of complications associated with prolonged mechanical ventilation.

Prone ventilation requires specialized training and expertise to perform safely and effectively. It necessitates close monitoring of the patient's vital signs, airway, and pressure points to prevent complications such as pressure sores, endotracheal tube displacement, or hemodynamic instability. Additionally, patient sedation and paralysis may be required during the positioning procedure to ensure patient safety and tolerance. This technique is considered a valuable adjunct therapy in the management of severe respiratory failure and has demonstrated positive outcomes in selected patient populations.

Inhaled vasodilators may be used as supportive therapy in an attempt to increase perfusion and blood flow around the lungs to improve gas exchange. In severe cases, Extracorporeal Membrane Oxygenation (ECMO) may be indicated. For details on Inhaled

Vasodilators and ECMO, please refer to specialized respiratory care textbooks, such as the latest edition of Egan's Fundamentals of Respiratory Care.

9.10 PUTTING IT ALL TOGETHER: A CASE STUDY

You receive your patient at the start of your shift. They are intubated and ventilated on Pressure Control Ventilation (PCV). Their ventilator settings are

$$\begin{aligned} RR &= 20 \text{ bpm} \\ \text{PC} &= 16 \text{ cmH}_2\text{O} \\ I_T &= 1.0 \text{ sec} \\ \text{PEEP} &= 10 \text{ cmH}_2\text{O} \\ \text{FiO}_2 &= 0.4 \end{aligned}$$

When giving you her report, the nurse tells you she just received the results from an ABG that she took 10 minutes ago, and the results are $7.47/38/115/30$. Let's work though this one together.

Photo by Official U.S. Navy Imagery, CC BY 4.0

Question 1: What can be fixed on an ABG with mechanical ventilation?

Your primary acid base goal is to restore a normal pH. We can only affect the CO_2 levels with ventilation. The HCO_3 is done by the kidneys "behind the scenes."

Question 2: Which ventilation setting affects which ABG value?

You already know FiO_2 and PEEP affect pO_2, and that RR and V_T affects CO_2 directly and pH indirectly.

Question 3: What are the problems with the ABG, if any?

Looking at the ABG, you can see that your patient has an abnormal pH, normal pCO_2 and abnormal HCO_3. The oxygen is also abnormal. You can't do anything about the HCO_3 levels, but you can focus on normalizing the pH by altering the CO_2 levels and also correct the oxygen levels back to normal.

Question 4: What settings could you change to fix the problem?

Let's look at the ventilation issues first. The pH is alkalotic due to elevated bicarbonate. You need to normalize the pH, and the only way to do so with a ventilator is to adjust how much CO_2 you are removing to move the pH from alkaline to normal. You don't want more base; you need to add more acid. Think of the analogy of the water in the boat, and remember, $\mathrm{CO_2} = \mathrm{acid}$. You need to let the CO_2 level (acid level) rise to move the pH back to normal. How would you let the water level rise in the boat? Bail slower or use a smaller bucket. So, you can decrease the RR or decrease the tidal volume on the ventilator.

Remember, though, you are currently ventilating in Pressure Control! How do you adjust the tidal volume when in Pressure Control? How does pressure relate to volume? If you are thinking you would decrease the pressure applied to result in a lower tidal volume, you are correct! Remember, that if you blow up a balloon with a lower pressure of air, it will be inflated to a lower volume. $\mathrm{Pressure} = \mathrm{Volume}$ when all other settings remain the same.

So, to fix the ventilation issues (pH correction by adjusting pCO_2), you could decrease the RR or decrease the pressure control.

Now, let's look at the oxygenation issues. Your patient has a pO_2 of $115\ \mathrm{mmHg}$. Remember that normal is $80\text{ - }100\ \mathrm{mmHg}$. Your patient has too much oxygen in the blood. The two settings that affect oxygen levels are FiO_2 and PEEP, so you could decrease either one to decrease oxygen levels.

Question 5: Which change(s) would be most correct?

Let's look at the options you identified above, and decide which change would be the most correct to make for the ABGs.

The ventilator settings are:

$$\begin{aligned} RR &= 20\ \mathrm{bpm} \\ \mathrm{PC} &= 16\ \mathrm{cmH_2O} \\ I_T &= 1.0\ \mathrm{sec} \\ \mathrm{PEEP} &= 10\ \mathrm{cmH_2O} \\ \mathrm{FiO_2} &= 0.4 \end{aligned}$$

What information are you missing here to assess whether to change the RR or the PC? If you guessed the tidal volume and patient information, you are correct!

Let's say you have already done your IBW and safe $V_T (6\text{ - }8\ \mathrm{mL/Kg})$ calculations. You know your safe V_T levels are as follows:

- $6\ \mathrm{mL/Kg}$ is $420\ \mathrm{mL}$
- $7\ \mathrm{mL/Kg}$ is $490\ \mathrm{mL}$
- $8\ \mathrm{mL/Kg}$ is $560\ \mathrm{mL}$

You look on your ventilator screen and see that your tidal volumes are around $550\ \mathrm{mL}$. The patient is within the safe RR range of $10\text{ - }24\ \mathrm{bpm}$. Both RR and V_T could be decreased safely here. But remember, you are only going to pick one change out of the two options.

When keeping in mind the dangers of VILI and the goals of decreasing barotrauma and volutrauma, the most correct change would be to decrease the V_T from where it is currently (approximately$8\ \mathrm{mL/Kg}$). To do this, you would decrease your pressure control.

What about the high oxygen levels? With the current vent settings, the patient has a PEEP of 10 and an FiO_2 of 0.4. The patient is below the threshold of FiO_2 0.5, but at the higher levels of PEEP. Although either setting could be adjusted here, because your patient is at the higher level of PEEP, and below the threshold of FiO_2 of 0.5, a clinician would most likely opt to decrease PEEP.

There is one additional option here. If your patient was triggering breaths above the set RR and vitally stable and not requiring large amounts of medication or infusions, they might be stable enough to attempt a change to PSV (spontaneous) instead of staying in PCV (control) and just decreasing settings. Remember, the ABG is over ventilated. If their disease process is resolving and they appear to have a drive to breath, consider decreasing sedation enough that they would be consistently triggering breaths, and consider attempting a change to PSV. Stay at the bedside for approximately 10 minutes and ensure the patient is breathing at minimum $14 - 20\ \mathrm{bpm}$ with tidal volumes that are appropriate for their ideal body weight. If they are not, change back to the previous control mode and make your changes there. Consider decreasing sedation a little bit and trying again later. If they appear to be breathing with a RR and V_T that are adequate for their needs, recheck an ABG in 30 minutes and confirm it is still normal or over vented—never let it go underventilated or allow CO_2 levels start to rise after a change like that.

Question 6: How *much* should I change my settings by?

The two changes that you have decided are the most correct to make are to

- decrease V_T by decreasing PC, and
- decrease PEEP.

Tidal Volume Decrease

Your patient is currently at approximately $8\ \mathrm{mL/Kg}$. You want to move down one step to target a tidal volume of $7\ \mathrm{mL/Kg}$. Try decreasing pressure control by $2\ \mathrm{cmH_2O}$ and assess the tidal volumes your patient is getting. If needed, you can adjust the pressure control up or down to hit a V_T of $7\ \mathrm{mL/Kg}$.

PEEP Decrease

Most likely, a clinician would decrease the PEEP by $2\ \mathrm{cmH_2O}$ at this point, and then potentially decrease FiO_2 as well, if SpO_2 or pO_2 remains higher than targeted ranges. PEEP changes are usually done only once a day because it takes so long for them to work (and then reassess). This is why FiO_2 adjustment will be your second move if needed—FiO_2 changes can be made frequently as needed.

9.11 CONCLUSION

Practice makes perfect! The more you see it and do it, the better you will become at looking at patients, choosing your initial ventilation strategies and altering them based on ABG assessments and interpretations. There is so much more to learn and do, but with this information, you can be confident you are safely initiating and altering ventilation for adult patients.

ARDS Ventilation Summary

Mechanical ventilation in ARDS is a complex and essential component of patient care. By employing a lung-protective ventilation approach, clinicians aim to minimize ventilator-induced lung injury and optimize gas exchange. Tailoring ventilation settings based on the severity of ARDS, individual patient needs, and response to therapy is crucial for achieving improved outcomes.

The use of low tidal volumes, appropriate PEEP, and consideration of plateau pressure help mitigate further lung damage and enhance patient recovery. Careful monitoring, frequent assessment, and adjustments in ventilation settings ensure a delicate balance between providing adequate oxygenation and avoiding complications associated with mechanical ventilation.

While mechanical ventilation is a life-saving intervention, it is not without challenges. Healthcare teams must remain vigilant in managing potential complications, such as barotrauma, ventilator-associated pneumonia, and patient-ventilator asynchrony.

As our understanding of ARDS and its management continues to evolve, ongoing research and advancements in mechanical ventilation techniques will pave the way for further improvements in patient outcomes. With a multidisciplinary and patient-centered approach, ARDS ventilation remains an integral part of comprehensive care, contributing significantly to the recovery and well-being of critically ill patients with this challenging condition.

9.12 SELF-CHECK

Chapter 9 Quiz

See how well you recall the concepts covered in this chapter by completing the following short quiz.

Don't forget to check your score!

An interactive H5P element has been excluded from this version of the text. You can view it online here: https://ecampusontario.pressbooks.pub/mcvresource/?p=248#h5p-10

CHAPTER 10 | MECHANICAL VENTILATION WEANING AND DISCONTINUANCE

Chapter Outline

10.0 CHAPTER OVERVIEW

Weaning: Ventilation Settings to Progress towards Extubation

The weaning process from mechanical ventilation involves gradually reducing ventilatory support, allowing patients to take on a more significant portion of their respiratory effort.

In the world of mechanical ventilation, a normal ABG is considered wean-able! A normal ABG, as well as an appropriate level of consciousness and progress toward resolution of underlying disease, are good criteria for clinicians to consider decreasing sedation and ventilator settings to facilitate spontaneous breathing.

We treat a normal ABG the same as an over-vented ABG (removing too much CO_2) as long as the patient is vitally stable enough to decrease the support, allowing them to start doing more work on their own. If you notice your patient is triggering breaths above the set rate and vitally stable, this is also the time that you could consider changing from a control mode to a spontaneous mode—ABGs would still be used to assess the effectiveness of this change, and then PSV settings would be decreased down to minimal settings to work towards extubation.

In this chapter, we will discuss strategies commonly used in the weaning process and how they are applied at the bedside, including ventilation modes and protocol driven strategies.

Learning Objectives

At the end of this chapter, you will be able to:

1. Describe the process of decreasing the pressure support (PS) in PSV until “minimal settings” are reached.
2. Explain why SIMV should not be used as routine practice, in order to advocate effectively for your patient.
3. Identify when alternative weaning strategies like Automatic Tube Compensation (ATC) and Tracheostomy Mask Trials (TMT) should be implemented.

Key Terms

- Weaning
- T-piece

- Trach mask trial
- Spontaneous Breathing Trial
- Artificial Intelligence

Covid-19 SP: Full ICU on worst phase by Gustavo Basso, CC BY-SA 4.0

10.1 PSV FOR WEANING PATIENTS FROM MECHANICAL VENTILATION

The transition to a spontaneous mode is the most important step that most patients need to complete prior to being weaned off the ventilator and extubated. Weaning remains the number one reason for PSV to be used in the clinical setting. As long as a patient is triggering breaths on their own, they could be trialed on spontaneous mode.

In PSV, the pressure support (PS) would be set at a level to achieve adequate tidal volumes for the patient's ideal body weight and ensure a reasonable respiratory rate. We only set the amount of support that is needed for this. Set the minimum pressure support to maintain the RR and V_T within those parameters.

Once on PSV, the pressure support is decreased as tolerated until "minimal settings" are reached. A clinician will know it is tolerated by watching a patient's RR and overall WOB (Remember the DiapHRaGM mnemonic?) and assessing their bloodwork after all decreases in support. Usually, decreases in support are done 1-2 times a day depending on patient status but should never be delayed if a patient can support their breathing with less support. The ultimate goal is decreasing support to "minimal settings" as quickly as possible based on patient status and tolerance of changes.

Minimal settings refer to minimal pressure support as well as minimal PEEP (PEEP of $5\ \mathrm{cmH_2O}$ as discussed in previous chapters). Once minimal settings are reached, this is termed a **Spontaneous Breathing Trial (SBT)**.

SBTs continue to be the gold standard in most Intensive Care Units for assessing a patient's readiness to be taken off mechanical ventilation. Protocol-driven weaning strategies, such as SBTs, have demonstrated superiority over weaning approaches based solely on clinical judgement. Their success largely stems from eliminating the guesswork in setting ventilator parameters and instead offering clinically tested steps to follow, ensuring a more systematic and reliable process for weaning patients from mechanical ventilation.

When performing an SBT, patients are decreased to the minimal settings (slight variations of pressure support amount can be seen when comparing hospital to hospital, but most are around $5\text{ - }7\ \mathrm{cmH_2O}$). The patient's RR, **work of breathing (WOB)**, and vitals are monitored for the duration of the test. If these vitals remain within normal limits after approximately 30 minutes, an **arterial blood gas (ABG)** is usually taken as well to ensure they remain normal. If the ABG results are good, the patient is considered to pass the SBT and can be considered as a candidate for extubation if all other criteria are met and the physician gives the order.

Life After the Ventilator

What is life like for patients who were ventilated, weaned and are now in recovery? The COVID-19 pandemic gave us a tremendous opportunity to gather data about post-ventilation patient experiences. Watch *"COVID-19 Survivors Explain What It's Like To Recover From Being On A Ventilator"* from NBC News NOW to learn more:

One or more interactive elements has been excluded from this version of the text. You can view them online here: https://ecampusontario.pressbooks.pub/mcvresource/?p=150#oembed-1

Video: "COVID-19 Survivors Explain What It's Like To Recover From Being On A Ventilator | NBC News NOW" By NBC News [3:20] *Transcript Available.*

For additional information about post-ventilator recovery, please see the following links:

- Life After a Ventilator (UNC Health Talk)
- Ventilator Weaning and Spontaneous Breathing Trials; an Educational Review

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10.2 SYNCHRONIZED INTERMITTENT MANDATORY VENTILATION (SIMV)

Regardless of your exposure to ventilation, at some point during your education or career, the mode **Synchronized Intermittent Mandatory Ventilation (SIMV)** will be mentioned in the context of weaning. This mode is not currently considered "best practice" for mechanical ventilation.

As much as it may be tempting to not discuss SIMV at all, it is still discussed in most ventilation textbooks and taught in some health professions as a current ventilation strategy. If you would like to learn more about the initial theory that prompted the rise of SIMV, as well as the disadvantages and current research identifying the flaws inherent with the mode in its current form, read on...

A health care worker prepares a new ventilator to be used. Photo by UNDP Ukraine, CC BY-ND 2.0

A History Lesson

Historically, when being weaned from the ventilator, patients would be woken up while on full control ventilation, sedation would be weaned and as soon as patients roused enough to start fighting the ventilator, they were extubated. There were no supportive modes, and weaning was not as well understood. As you may imagine, this abrupt approach suffered from major issues ranging from the effects of muscle wastage to patient PTSD.

In the 1970s, a weaning mode was hypothesized that would allow for a steady decrease in the mandatory rate with time in between for the patient to breathe spontaneously on their own. This mode was termed Intermittent Mandatory Ventilation, and it became the gold standard of ventilation from 1980s-2000s. Clinicians would set a minimum mandatory rate, and above that, the patient would be able to spontaneously breathe whatever volume they pulled off the bias flow. The mandatory rate would be steadily decreased until the patient was breathing completely spontaneously. This gradual transition to spontaneous breathing was hypothesized to increase the patient's work of breathing slowly in a controlled step-by-step manner.

It is important to understand that this mode was not synchronized with the patient at all. If the mandatory rate was set at 10 bpm, every 6 seconds, the ventilator would deliver a full control breath as set by the clinician regardless on whether the patient was halfway through a breath already or if they needed it or not. The ventilator was totally "blind" to the patient.

Researchers and clinicians alike soon saw the asynchronies and breath-stacking inherent with this mode and came up with a solution to synchronize the ventilator-delivered control breaths with the patient. They programmed an approximate 0.5 second window around the scheduled mandatory breath that would allow any patient triggered breath in the window to be a fully assisted controlled breath. If no patient triggered breath was sensed, a full control breath was delivered. Thus "Synchronized" Intermittent Mandatory Ventilation, or SIMV, was born.

In recent decades, continued research has highlighted issues with weaning using SIMV. Studies started to show that sequential decreasing of the mandatory rate was not allowing the patient to gradually overtake their work of breathing. Instead, studies were pointing to the inability of the body to adapt its effort based on the amount of support available. In other words, the patient's work of breathing was based on how hard they worked with the unsupported spontaneous breaths. These spontaneous breaths were not supported in any way. Patients would breathe without any pressure support—having to overcome the resistance of the ETT on their own. Therefore, even though every few breaths was fully supported, this was not able to unload the respiratory muscles. Even fully supported breaths showed the same high WOB levels, as the neurological drive to breathe was expecting the work of breathing of the un-supported spontaneous breaths and not able to adapt to the variable support levels.

Ventilator manufacturers again attempted to fix these issues by adding additional support to SIMV. Pressure support was added to the spontaneous breaths to attempt to unload the respiratory fatigue that was being seen. Current SIMV modes all now operate as a true mixed mode—oftentimes flipping between controlled and supported modes like PCV and PSV based on the time window set by the minimal rate. But again, the mode still has its struggles. No benefits have been identified in using SIMV over standard control modes followed by a transition to spontaneous modes.

Adding a further nail in the coffin, current trending in medical research is focusing the direct correlation between high rates of ventilator asynchrony and increased ventilator days. SIMV remains a main culprit, with a high incidence of asynchrony and multiple studies identifying a potential delay in the weaning process and increased ventilator days.

Every second that a patient is on a ventilator needs to be treated as incredibly important. We have already seen in this chapter how impactful being on a ventilator can be for a patient. Using a mode that can potentially delay weaning or cause dysfunction is not necessary if there is another option that can potentially work as well with fewer disadvantages.

Though once the most widely used mode of ventilation, SIMV use has dropped off precipitously in current critical care practice. Some studies have shown that SIMV use by experienced clinicians with careful patient selection can result in safe and effective use. It is still used in neonatal populations as well as post-operatively with lingering sedation.

Though SIMV is not commonly used, the basic principle has not been abandoned. Derivatives of this mode can be seen in specialty modes of many forms. And, as we have already learned, the field of mechanical ventilation is always evolving. Future developments could possibly bring this mode back to the cutting edge of medicine—though more likely it will be rebranded with a different name.

Know better; do better...or so the saying goes. You may, at some point, find yourself in a situation where SIMV is being routinely practiced, as the outdated mode is still used by practitioners who are unaware of the most recent research. Now that you have learned more about why SIMV is not the best choice for patient respiratory care, consider advocating for your patient by sharing what you have learned.

Part of effective advocacy is being able to refer to credible and up-to-date research to back up your claims. For example, the following two resources provide information about why the use of SIMV should be discontinued:

- Synchronized Intermittent Mandatory Ventilation: Time to Send This Workhorse Out to Pasture
- Should Intermittent Mandatory Ventilation Be Abolished?

10.3 ADDITIONAL WEANING STRATEGIES

In Chapter 5, we explored several ventilation modes that enable patients to have spontaneous breaths and ensure a seamless transition from control ventilation to spontaneous supported ventilation. Depending on the available ventilator options and site protocols, we have discussed the following modes that can aid in facilitating the weaning process from mechanical ventilation: Mandatory Minute Ventilation (MMV), Automode, Adaptive Support Ventilation (ASV), Proportional Assist Ventilation (PAV) and Volume Support (VS). These ventilation modes offer valuable choices for tailoring the weaning approach to each patient's needs, supporting a smoother transition towards independent breathing and eventual liberation from mechanical ventilation.

Automatic Tube Compensation (ATC)

In addition, it is worth mentioning another ventilation strategy available on some ventilators, **Automatic Tube Compensation (ATC).** This is a feature designed specifically to overcome resistance imposed by the endotracheal tube. ATC can be combined with all conventional ventilatory modes when resistance through the endotracheal tube is high. This seems to work especially in cases of difficult to wean patients who may not have an appropriate size artificial airway (airway too small).

When ATC is used, the ventilator monitors patient inspiratory flow, and based on flow pattern and requirements and size of artificial airway, pressure is increased during inspiration and decreased during expiration to overcome the resistance and decrease work of breathing. A percentage of support can be set on the ventilator from $1 - 100\%$ compensation.

Tracheostomy for Weaning

Patients who face challenges with weaning from mechanical ventilation are often offered a tracheostomy tube instead of an endotracheal tube for various compelling reasons. This shift is motivated by a range of benefits, including enhanced comfort for the patient, reduced reliance on sedation, easier oral hygiene management, and the flexibility of being able to disconnect from ventilation temporarily and reconnect if necessary, all without requiring reintubation. The presence of a tracheostomy, allows weaning through **Tracheostomy Mask Trials (TMT).**

A TMT involves a carefully planned daily period during which ventilatory support is temporarily removed. This trial serves as a progressive step towards the patient's independence in supporting their own breathing. Each day, the duration of ventilatory support removal can be gradually extended, allowing the patient's respiratory function to strengthen until they can breathe independently without assistance. During TMT, oxygen and humidity is usually provided to ensure patient comfort.

10.4 CONCLUSION

Weaning strategies for mechanical ventilation may vary based on the available resources and ventilator capabilities in an intensive care unit. Typically, a gradual approach is favored, but there are instances where an abrupt removal of support may be more suitable. It is widely agreed that the weaning process should not be unnecessarily prolonged as prolonged mechanical ventilation increases the risk of complications for the patient. Striking the right balance between a carefully paced weaning process and timely liberation from the ventilator is crucial to optimize patient outcomes and minimize potential risks associated with prolonged ventilation.

You did it! You have taken all of the knowledge you have learned throughout this course and applied it to mechanical ventilation—with the final piece being altering your ventilation settings based on ABGs.

Congratulations on completing the Basics of Mechanical Ventilation online resource! Photo by Anton Uniqueton, Pexels Licence.

10.5 SELF-CHECK

Chapter 10 Quiz

An interactive H5P element has been excluded from this version of the text. You can view it online here: https://ecampusontario.pressbooks.pub/mcvresource/?p=2106#h5p-12

VERSIONING HISTORY

This page provides a record of edits and changes made to this book since its initial publication. Whenever edits or updates are made in the text, we provide a record and description of those changes here. If the change is minor, the version number increases by 0.1. If the edits involve a number of changes, the version number increases to the next full number.

The files posted alongside this book always reflect the most recent version.

Version	Date	Change	Affected Web Page
1.0	August 31, 2022	First Publication	N/A

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