Basic Principles of Mechanical Ventilation

BASIC PRINCIPLES OF MECHANICAL VENTILATION

MELODY BISHOP BSC, RRT, CCAA

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INTRODUCTION

Mechanical ventilation is one of the most confusing and often overcomplicated topics that medical professionals—such as nurses, paramedics, physicians and respiratory therapists—encounter in their education. It is very common for seasoned medical practitioners working in critical care environments to still be intimidated with how to approach ventilation and how to understand all settings and modes. Much of the available learning materials overcomplicate concepts that can be explained simply—including unnecessary calculations and advanced concepts—because they are written for respiratory therapists and respiratory physicians who are expected to hold advanced knowledge of the field. However, the reality is that ventilators are used every day by non-specialized health care workers, who lack training and resources that are appropriate to their needs. By stripping away unnecessary complexity and using common analogies, **the basics of ventilation can be taught to everyone**, and advanced settings and concepts can then be added once the general foundation is fully understood.

This open educational resource (OER) was written for a wide audience of health care professionals, including:

- Nurses, who may encounter ventilators in critical care settings such as the emergency room, the Intensive Care Unit, or operating rooms.
- Paramedics, who may transfer ventilated patients or encounter ventilators in their work environment.
- Anyone who practices flight medicine.
- Student physicians and respiratory therapists prior to additional training and education.

Don't see yourself on this list? This book can still be for you. Mechanical ventilation is a useful skill for all health care professionals who encounter ventilators in their scope of practice.

Topical Overview

This OER provides a foundation of mechanical ventilation concepts and basics. By the end, you will understand the basics of ventilation, settings, basic 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.

Additional Learning

Although this OER is open to anyone, some users may wish to deepen their learning experience. Additional exercises, application notes, case studies and live support from an experienced instructor are available to learners who enroll in the Mechanical Ventilation micro-credential course at Sault College. Students who successfully complete this online course will earn a **micro-credential certification in Mechanical Ventilation**. Please visit training.saultcollege.ca for more information or to register.

Author Biography

Melody Bishop (B.Sc.) is a Registered Respiratory Therapist (RRT) and a Certified Clinical Anaesthesia Assistant (CCAA). She has worked in diverse areas of critical care medicine for over 13 years with specialty in intensive care units, emergency medicine and the operating room. For example, her love for complex medical challenges brought her to the flight medicine world, where she worked as an emergency first responder flying into remote communities, stabilizing and transporting patients to health care centres in Southern Canada. In her roles as a Clinical Educator and a college professor, she has been involved in teaching and educating a wide range of medical professionals over the last decade at the post-secondary, post-graduate and professional level—specializing in Critical Care RN ventilator education and paramedical mechanical ventilation training. Her mission as a teacher is to simplify the "scary" topic of ventilation and present it to all medical professionals in an easy to understand and approachable way.

Editing and Instructional Design

Editorial and instructional design services were completed by **Amanda Baker Robinson**. Amanda works as an instructional designer for colleges across Ontario, including Sault College, Humber College, Canadore College, and Cambrian College.

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CHAPTER 1 | HOW THE HUMAN BODY BREATHES

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?

This chapter describes the process by which the human body takes in and expels air in a straightforward and understandable way. As the saying goes, "it's as natural as breathing," so we'll try to avoid overcomplicating things. You'll learn about breathing with the aid of simple, everyday analogies and object lessons. Consider trying these object lessons yourself as you wrap your head around the concepts you are learning.

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. Define FRC and Intrinsic PEEP and the importance to the alveoli.
4. Understand the basic pressure changes that occur in the lungs during the respiration process.

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
- Pmo
- pleural space

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.

THE ANATOMY OF BREATHING

Before you can dive in to the theory of mechanical ventilation, you should have a basic understanding of how the human body is designed to breathe. 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.

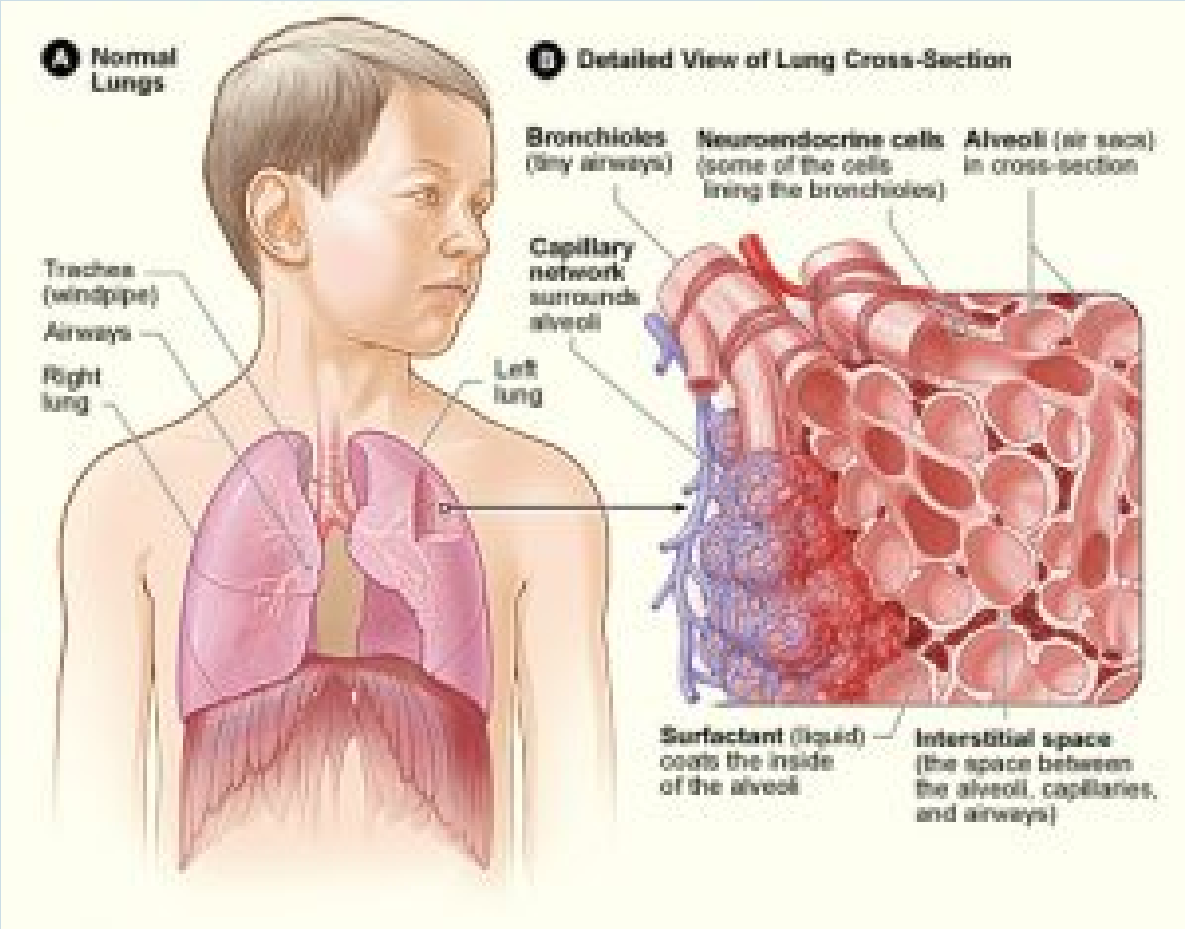
Thinking of the lung as a balloon is a great simple analogy but it is important to review the physiology of the respiratory system to fully explain how the lungs behave. The lungs are not simply one big balloon. It is like they are made up of millions of little balloons all stuck together—similar to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



Normal lungs with a detailed view of lung cross-section.

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- Normal Lung Structure © National Heart Lung and Blood Institute is licensed under a Public Domain license

THE BASIC 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 Takeaways

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.

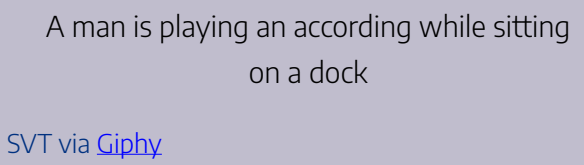
One or more interactive elements has been excluded from this version of the text. You can view them online here: https://ecampusontario.pressbooks.pub/mechanicalventilators/?p=67#video-67-1

GIF via Gfycat

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.



A man is playing an according while sitting on a dock

SVT via Giphy

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.

Boomunderground via Giphy

Key Takeaways

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.

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 Lessons

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.

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.

SVT via Giphy

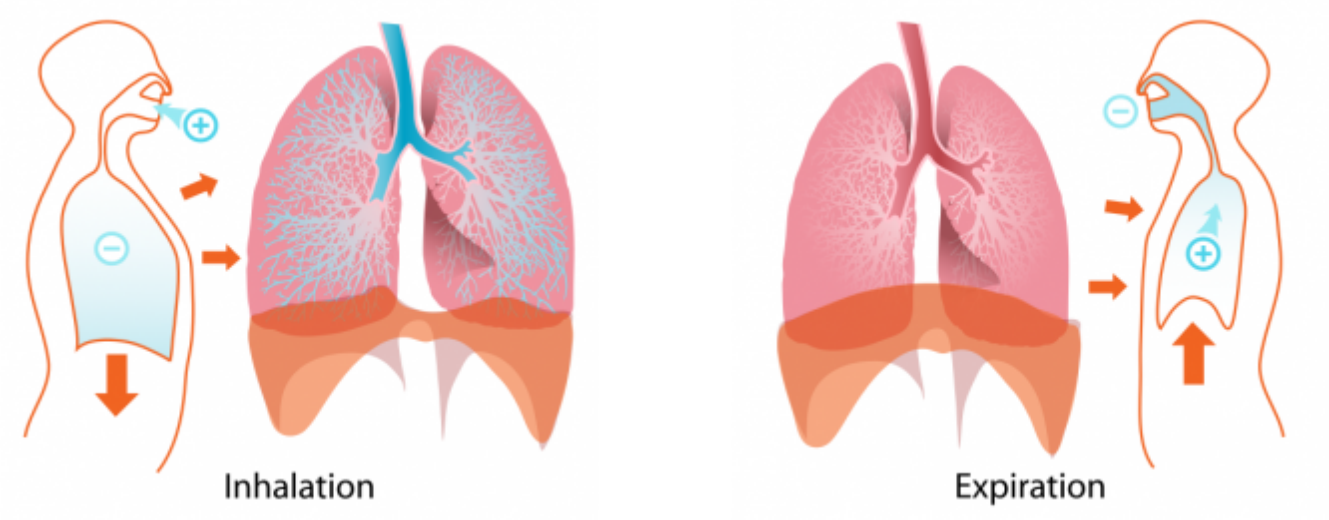
Boomunderground via Giphy

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 Takeaways

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.

To summarize, 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.



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.

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FRC AND INTRINSIC PEEP

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.

Key Takeaways

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.

Pressures

When dealing with breathing, it is important to have a basic understanding of the resting pressure changes in

the lungs during inspiration, expiration and at rest. All pressures are compared to the "baseline" of the open atmosphere. Imagine the pressure at the mouth is equal to atmosphere and is zero.

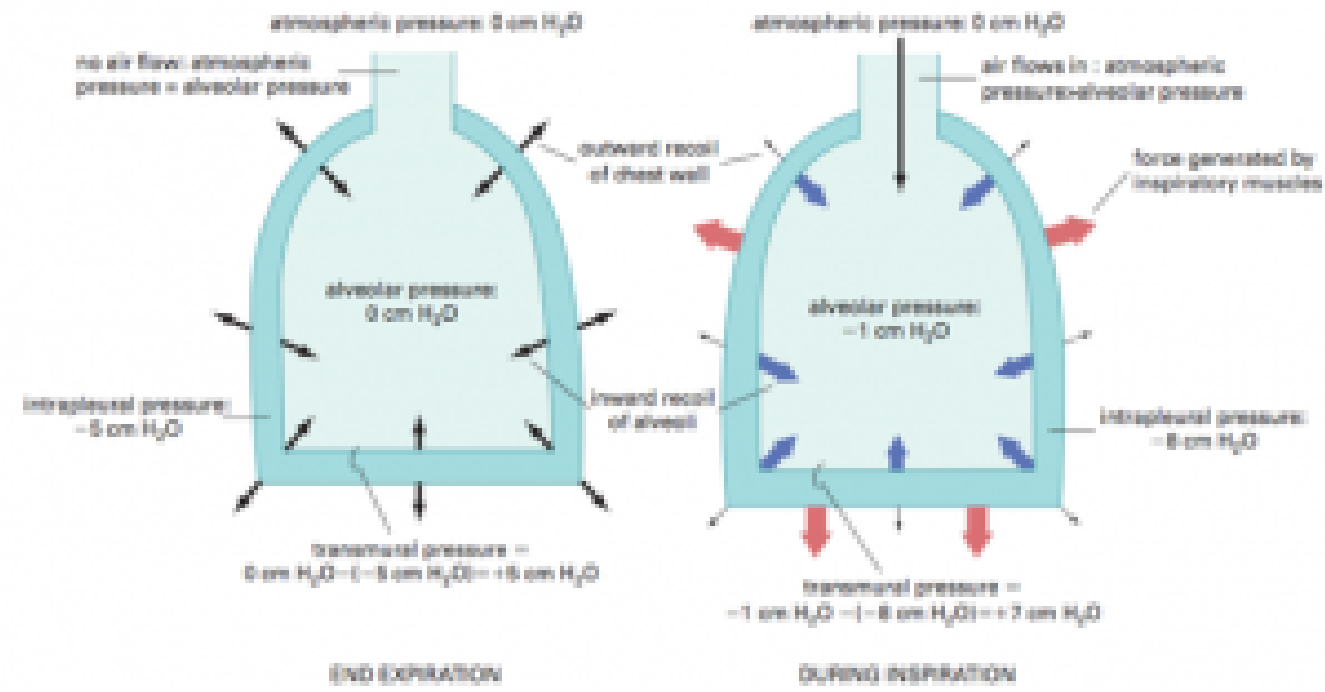
We tend to use **cm H_2O** to measure pressures in the respiratory system. As you may have already guessed, cm H_2O literally refers to the weight of a centimeter (cm) of water (H_2O) on a given spot. The atmosphere is 0 cm H_2O and any additional pressure would be above that. The other pressures that are instrumental in the pressure gradients of breathing are the **Palveoli** (pressure in the alveoli) and the **Ppleural** (pressure in the pleural space). The gradient is always between the **Pmo** (pressure at the mouth or atmosphere pressure) and the Palveoli. The pressure in the alveoli is determined by the volume of air left in the alveoli and the pressure being exerted around the alveoli—by the Ppleural or the diaphragm contracting.

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 cm H_2O) keeps a small amount of air in the lungs (equal to +5 cm H_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 Takeaways

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

As the diaphragm contracts, the negative pressure gradient from the dropping of the diaphragm creates a pressure gradient for air to flow in. 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.



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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.

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 Ppleura 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 medical professionals working with ventilators understand that this trauma can occur if they are not careful, it allows them to approach ventilation with the respect that this kind of care needs. 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 you understand the natural state of breathing, you should also understand how important it is to match the natural breathing process as much as possible with your 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/mechanicalventilators/?p=28#oembed-1

If you require a transcript for this video, please click Watch on YouTube to view the video source. Then, you can access the written transcript from this page.

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*

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CHAPTER 1 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!

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https://ecampusontario.pressbooks.pub/mechanicalventilators/?p=103#h5p-1

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CHAPTER 2 | OXYGENATION AND THE FUNDAMENTALS OF MECHANICAL VENTILATION

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

Application

Understanding how mechanical ventilation works will equip you, the health care practitioner, to protect the lung from unwanted damage. You will also learn the basics of selecting appropriate oxygenation for a ventilator patient.

Learning Objectives

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

1. Discuss the concept of mechanical ventilation as a sealed system
2. Specify healthy oxygen saturation levels and how to titrate supplemental oxygen optimally
3. Explain how PEEP and oxygen work together to aid in oxygenation
4. Explain why lung protective strategies are necessary in mechanical ventilation

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
- hypercapnia
- ineffective drive to breathe
- lung protective strategies
- 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

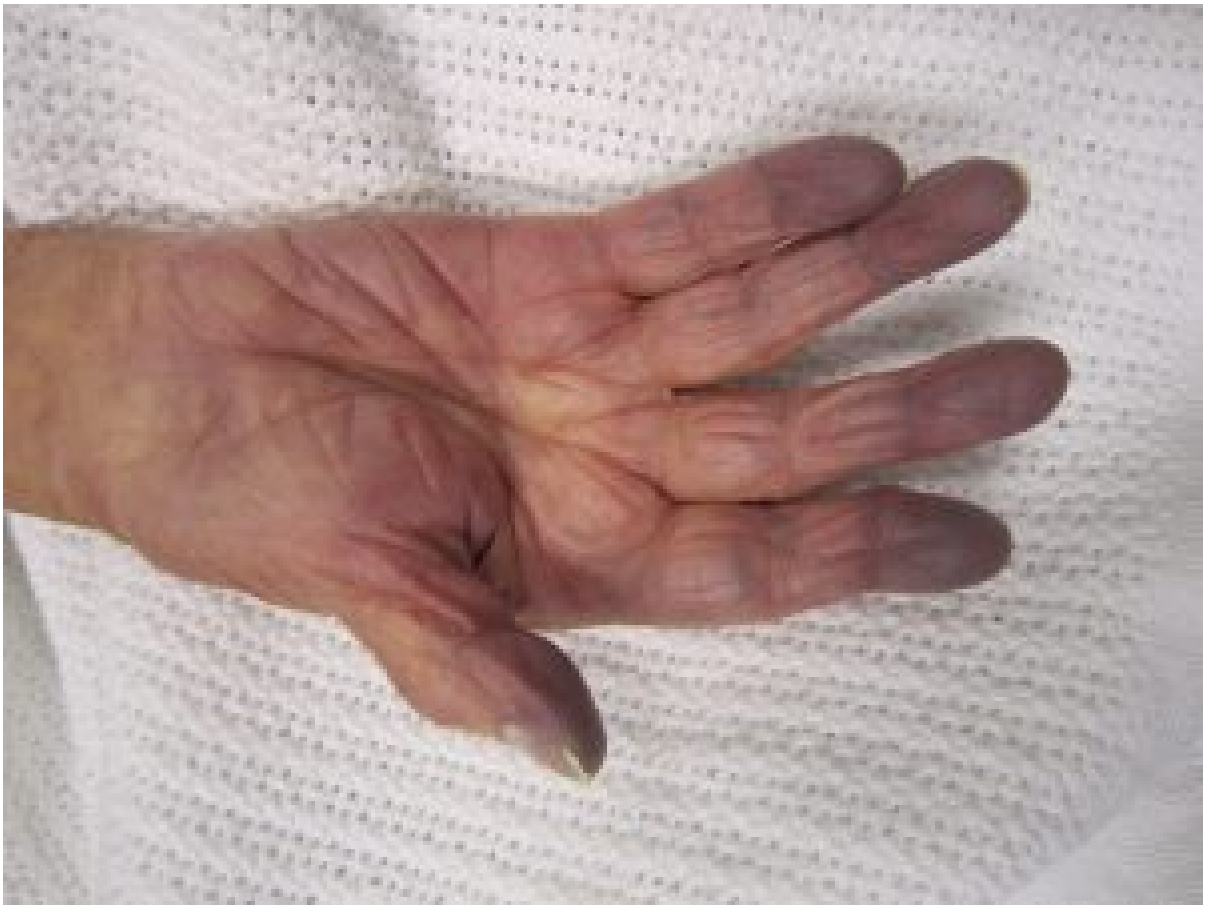
Whenever these terms are first introduced in this chapter, they are bolded. However, if you need additional information about a term than what is provided here, you can research it in The Free Dictionary: Medical Dictionary.

WHY DO WE NEED MECHANICAL VENTILATION?

Reasons for mechanical ventilation can fall into three main categories:

1. **Hypoxic failure** (also called hypoxia) 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. **Hypercapnia**, or high CO_2 levels, 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.
3. An **ineffective drive to breathe** can occur if there is an injury to the brain or neurological control to breathing. 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 will always require mechanical ventilation to take over the impaired drive to breathe.

Remember when you 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.



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.

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 modes are continually introduced. This online resource will in no way be able to comprehensively explain every parameter, mode, trigger or method of ventilation. Instead, what this will do is explain the core concepts of the types of breath delivery to give a basic understanding of ventilation. Pairing this information with a general understanding of lung mechanics, a health care professional will have 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 modes and give a health care professional a basic understanding to tailor to their medical practice and point to directions for further learning.

Key Takeaways

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.

Media Attributions

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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

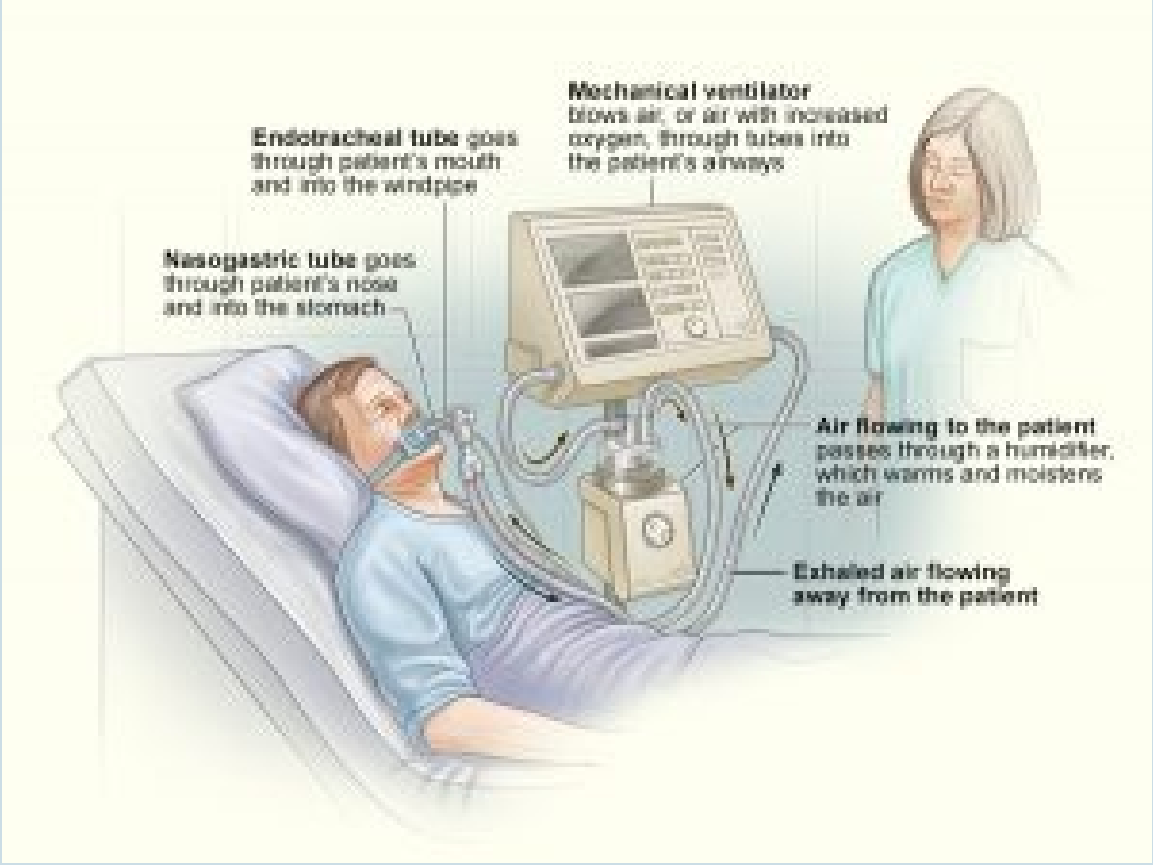
As you learned in Chapter 1, lungs act like balloons. Now, imagine a balloon sealed to the end of a straw. When you blow air through the straw, it must go into the balloon. The balloon will inflate when the pressure you blow is hard enough to overcome the strength of the rubber. If you fill the balloon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

Adapted from Margz Adventure via YouTube

In the case of mechanical ventilation, inspiration occurs by air being pushed from the ventilator into the lungs. 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). Exhalation remains a passive process. When full, the inflated lungs have a higher pressure than the outside atmosphere once the driving pressure stops from the ventilator—and, as you remember from Chapter 1, air always flows from higher to lower pressure. During exhalation, a valve opens and the ventilator allows the air to vent 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 (Pmo). 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. 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.



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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 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. The medical provider must decide the percentage of oxygen the patient needs to breathe. Oxygen is expressed in FiO2 on ventilators and can range from 0.21-1.00.

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

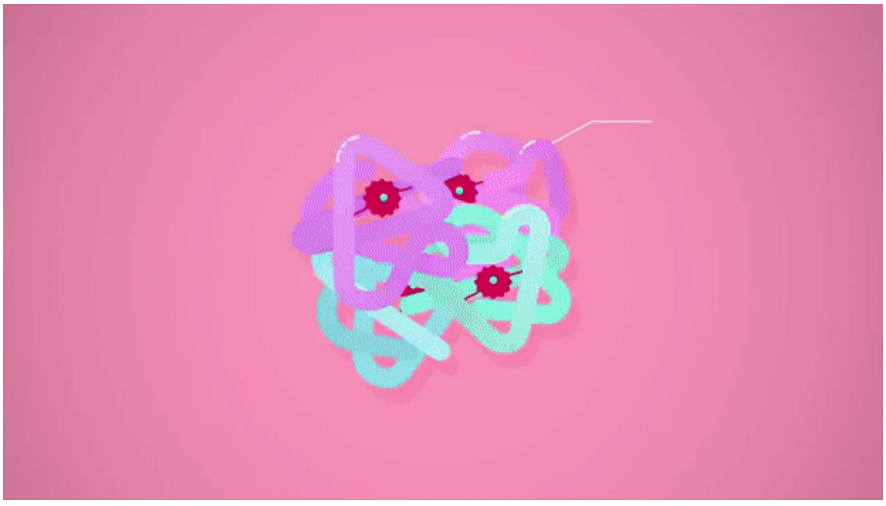
is usually expressed in liters per minute (Lpm) of O_2 delivery. Supplemental oxygen can be increased to almost equal 100% oxygen depending on the interface it is supplied with (nasal prong or face masks).

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 animation:



Vlog Brothers via GfyCat

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 SpO2 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 Takeaways

If the patient's SpO2 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.

Oxygen Content (CaO_2)	=	$(Hb)(1.34)(\%SaO_2/100)$	+	$(0.003)(PaO_2)$
		Oxygen bound to Hb		Diffused Oxygen

The 1.34 is constant and represents the maximum amount of oxygen that can bind to 1 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 80-100mmHg.

Let's look at the formula in action. If Patient A has a Hgb 120, SaO2 99% and a "normal" PaO_2 of 100mmHg, here is the formula:

Oxygen Content (CaO_2) = (120)(1.34)(0.99) + (0.003)(100)
= 159.1 + 0.3
= 159.4

For more information on the Oxygen Content Formula, check out this video: "Easy Ways to Calculate Oxygen Content of Blood."

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.

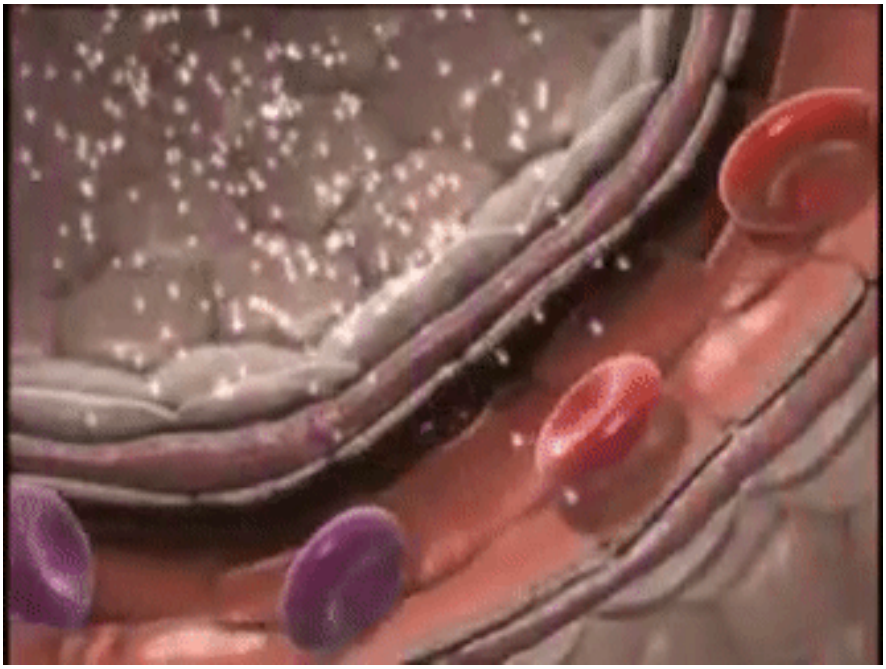
OXYGEN ON A VENTILATOR: SETTING THE FIO2

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 learned in this chapter can be applied when adjusting settings on the ventilator. If the $Sp0_2$ 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 increase 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 (see the following animation):



Lonely Teeming Billygoat via GfyCat

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. 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

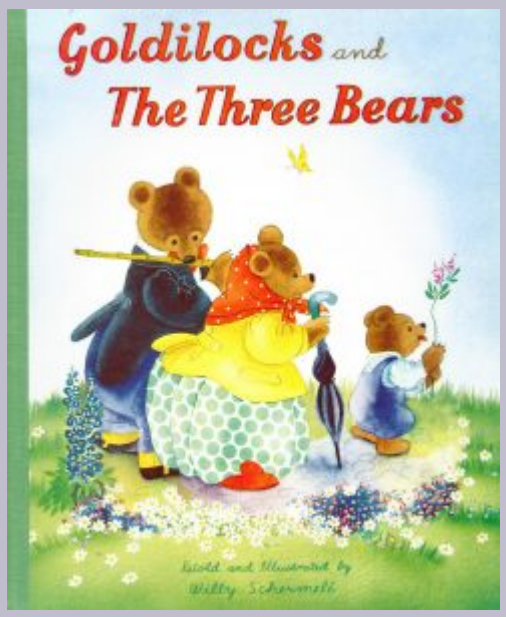
much slower process. It is essential to understand that you must give more oxygen if levels are low and *do not withhold oxygen* if your patient is showings signs of lower SpO_2. Just avoid delivering higher oxygen than you need to for extended periods of time.

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.



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 80-100mmHg 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 Pa02. As stated, the normal is 80-100mmHg. A person with an SpO_2 of 100% could have an PaO_2 of 100mmHg or upwards of 400mmHg. PaO_2 that are higher than 100mmHg are indicative of over oxygenation, or hyperoxia. High levels greater than 100mmHg 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_2 100.

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 100mmHg. 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.

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

Media Attributions

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.

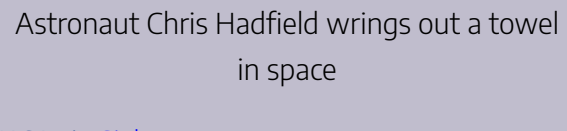
In Chapter 1, we learned that Positive End Expiratory Pressure (PEEP) describes the little bit of air that remains in the alveoli after exhalation has ended. As we begin to discuss mechanical ventilation, PEEP is also set on the ventilator. As you may have already surmised, the PEEP setting on the ventilator 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 5cmH20. 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.



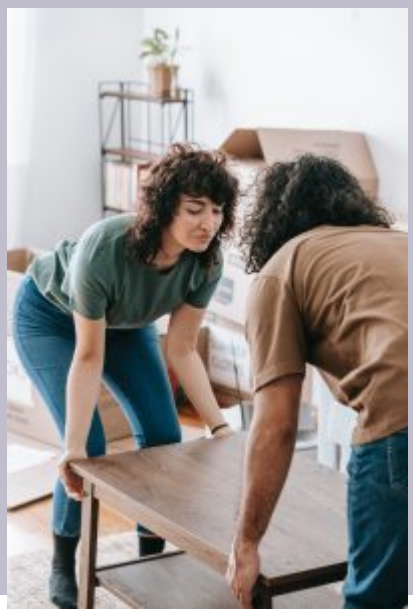
Astronaut Chris Hadfield wrings out a towel in space

NASA via Giphy

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.



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 always follow physician orders. These concepts will be revisited in Chapters 3 to 5 when discussing ventilation settings in more depth.

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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.

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.

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.

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CHAPTER 2 SELF-CHECK

Chapter 2 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/mechanicalventilators/?p=261#h5p-2

Advance Your Learning

Additional exercises, application notes, case studies and live support from an experienced instructor are available to learners who enroll in the Mechanical Ventilation micro-credential course at Sault College. Students who successfully complete this online course will earn a micro-credential certification in Mechanical Ventilation. Please visit training.saultcollege.ca for more information or to register.

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CHAPTER 3 | BASIC VENTILATOR MODES AND SETTINGS

Overview

In Chapter 2, you gained an introduction to mechanical ventilation as a sealed system and some of the basics of delivering oxygen to a patient—whether breathing spontaneously or on a ventilator. Now it is time to dive into mechanical ventilation and begin to discuss the different modes a ventilator can deliver to help a patient breathe.

In this chapter we will cover:

- Basic ventilation mode categories
- Basic ventilation settings and what they mean

Application

It is essential that any health care practitioner who is working with ventilators understand the two main modes of ventilation and be familiar with all of the main 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. Differentiate control and spontaneous ventilation modes
2. 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 (Pinsp)
- Pressure Support (PS)
- Inspiratory Time (IT)
- Flow (.V)
- Positive End Expiratory Pressure (PEEP)
- Fraction of inspired oxygen (FiO_2)

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.

MODES OF VENTILATION: THE BASICS

Basic modes of ventilation can be mostly categorized into two separate groups:

- Control modes
- 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 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.

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.

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A BRIEF HISTORY OF CONTROL AND SPONTANEOUS MODES

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/mechanicalventilators/?p=283#oembed-1

If you require a transcript for this video, please click Watch on YouTube to view the video source. Then, you can access the written transcript from this page.

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.

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).

Media Attributions

- PB 7200 © Kacmarek (2011)

CONTROL VS. SPONTANEOUSLY INITIATED BREATHS

Control breaths are the breaths delivered according to the set respiratory rate on the ventilator. For example: if the respiratory rate is set at 15bpm, 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.

We have just outlined that, 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*.

So how does a ventilator "sense" a patient?

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. This 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 health care provider 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.

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.

> An experienced provider 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 is outside the scope of the course, but when it comes to patients triggering breaths in control modes, all you need to remember is 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 15bpm and you notice the patient is breathing 18bpm in total, it is obvious that the patient is triggering breaths above the set respiratory rate.

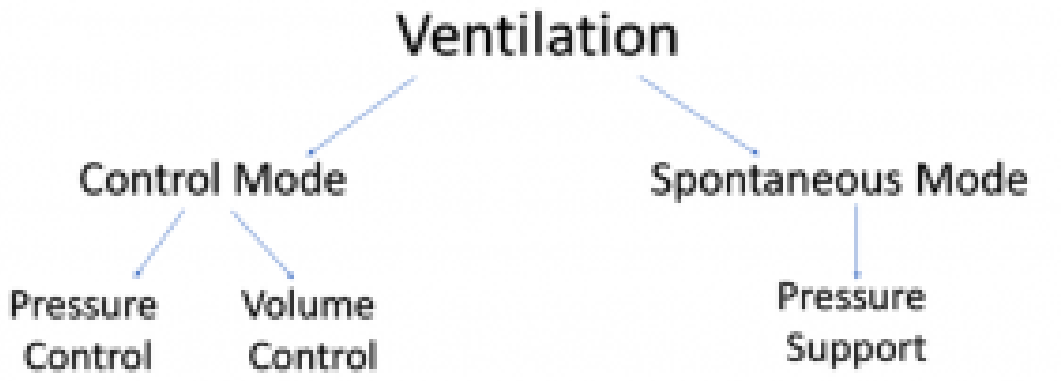
Media Attributions

- Lt. Cmdr. Michael Heimes checks on a patient connected to a ventilator at Baton Rouge General Mid

THE VENTILATION "FAMILY TREE"

If you imagine ventilation like a family tree, control and spontaneous are the main branches. 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



Regardless of how the control breaths are labelled or described, keep in mind that control breaths are set and delivered by the medical provider on the ventilator, and either category gives you the same outcome—fully "controlled" breaths that a patient *must* take. Essentially, every controlled or mandatory breath—regardless of whether it is considered a volume or pressure breath—is still describing the same thing. Air is being *pushed* into the lungs by a machine. This designation of "pressure" or "volume" refers to how the breath is *described* via settings on the ventilator. Both pressure and volume modes do the exact same thing: push air into the lungs according to the clinician's chosen settings and then let the lungs exhale passively.

Spontaneous breaths, or assisted breaths a patient chooses to take, are overwhelmingly 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.

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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!

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.

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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?

So, what do you need to set when working with a control mode? As outlined in Chapter 2, 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 (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 Takeaways

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:

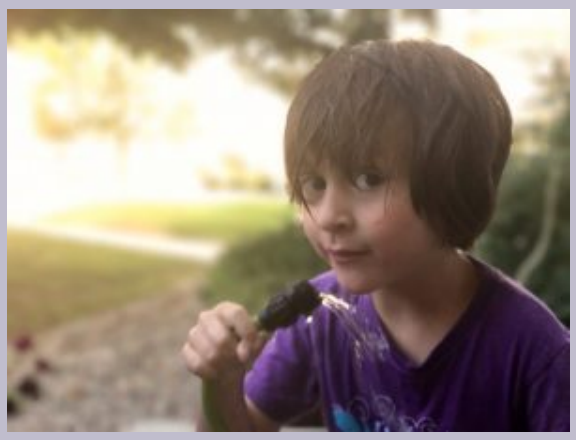
1. the level of PEEP to keep the alveoli from collapsing
2. the amount of FiO_2 to adequately oxygenate your patient

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.

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.

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- Ventilator control panel © quinn.anya is licensed under a CC BY-SA (Attribution ShareAlike) license
- Summertime Drinking from the Hose © nekosoft is licensed under a CC BY-SA (Attribution ShareAlike) license

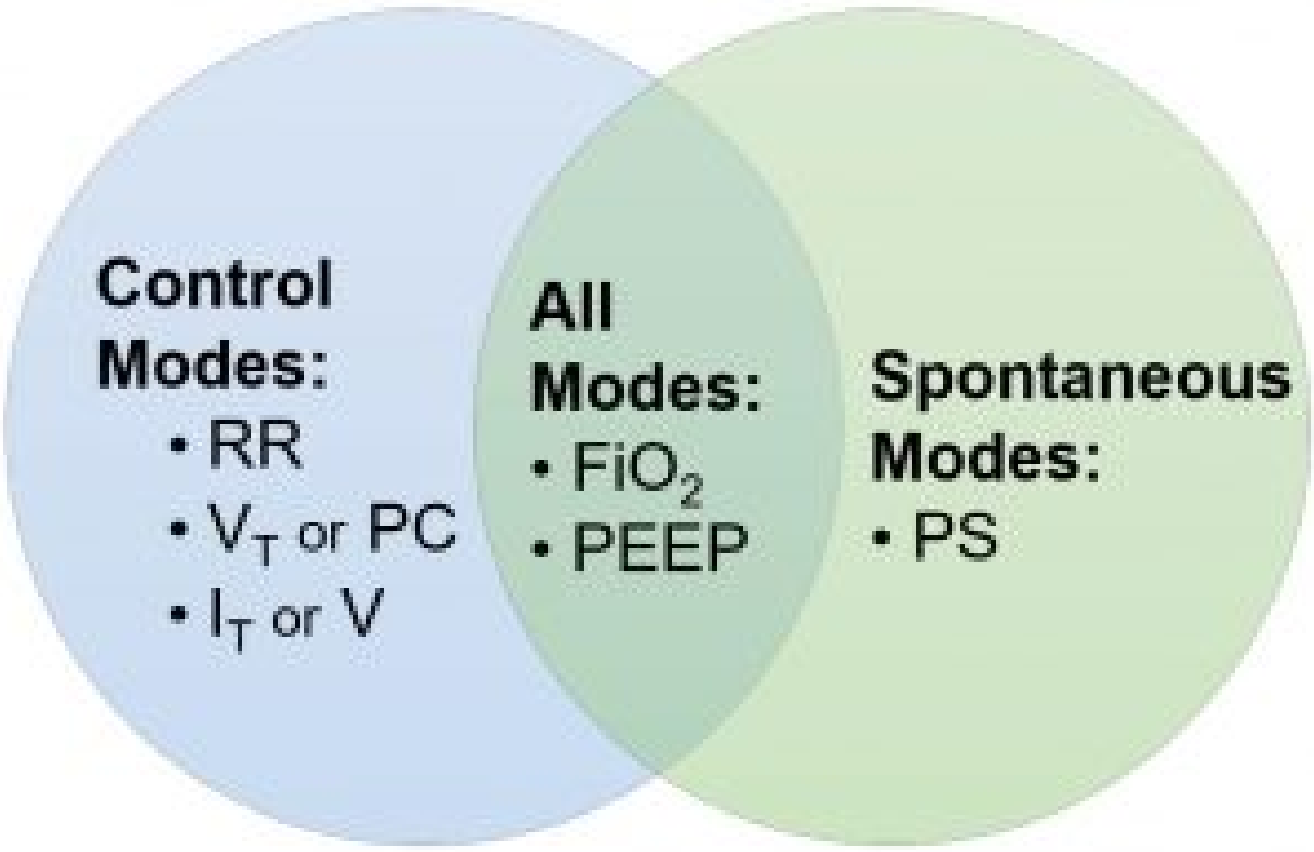
VENTILATOR SETTINGS TABLE

The main ventilator settings that are important to understand are:

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 (Pinsp)	The amount of pressure at which the ventilator will deliver every single breath to inflate the lungs.	cmH20	*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.	cmH20	*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).	cmH20	*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:



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- Ventilator Settings Organized by Mode © Melody Bishop

CONCLUSION

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.

Please review the following key points from this chapter:

- 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 health care provider 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 health care professional) 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.

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/mechanicalventilators/?p=322#oembed-1

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CHAPTER 3 SELF-CHECK

Chapter 3 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/mechanicalventilators/?p=331#h5p-3

Advance Your Learning

Additional exercises, application notes, case studies and live support from an experienced instructor are available to learners who enroll in the Mechanical Ventilation micro-credential course at Sault College. Students who successfully complete this online course will earn a micro-credential certification in Mechanical Ventilation. Please visit training.saultcollege.ca for more information or to register.

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CHAPTER 4 | CONTROL MODES

Overview

In Chapter 2, 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
- The relationship between volume, pressure and time
- How lung compliance and resistance will impact the delivery of air into the lungs

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

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.

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

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.

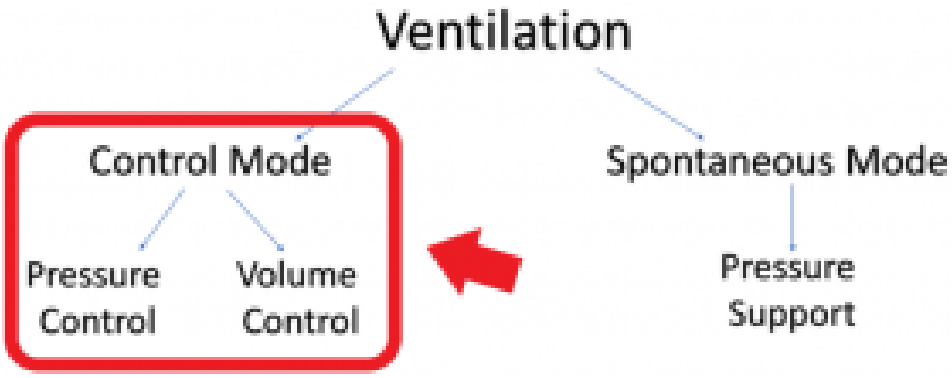
USING CONTROL MODES OF VENTILATION

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 (See Chapter 2). 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 are intubated due to issues like hypoxia, hypercapnia or an ineffective drive to breathe, their breathing needs to be taken-over and a *control mode* is used to fix the presenting reason for ventilation.

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 (see Chapter 3). In this chapter, we will be narrowing our focus to the control mode branch of the ventilation "family tree:"



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 have discussed in Chapter 2 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. Either pressure or volume, both control modes are simply a set amount of air being pushed in at a set rate.

This chapter will introduce both types of control modes, including differences and similarities. Though we cannot teach all of the subtleties of the parameter differences, at the end of this book, you should be able to comfortably set up volume control or pressure control to facilitate basic ventilation strategies for a patient.

Key Takeaways

Take a look at the following acronyms which are commonly used by practitioners when talking about volume 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.

Media Attributions

- family_tree_control © Melody Bishop

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 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, 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)

Photo by Kampus Production from Pexels

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 second, the first time to a volume of 300mls and the second time to 500mLs, which time would require a higher pressure?
2. If you were inflating the lungs over 0.8 seconds and were using a pressure of 15cmH_20 but then dropped the pressure to 10cmH20 using the same inflating time, what would happen to the volume in the lungs?

THE TIME VARIABLE

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 (Itime, 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.

Image by karosieben from Pixabay

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 Itime) 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

Some modes of ventilation do not utilize Itime (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. 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 100mls is the overall target, Balloon 1 has a peak flow of 60Lpm vs Balloon 2 has a peak flow of 30Lpm. 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 Itime here. The ventilator I_T will depend on the set parameters of the volume and flowrate of the air.

I:E RATIO

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.

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

Time element of Itime (or flow)

If a shorter Itime 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 Itime to 0.8 seconds versus 1.2 seconds, how would that affect your patient's 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 15bpm; therefore, each inspiratory/expiratory phase is 4 seconds.)

- If Itime is 0.8seconds, then Etime is 3.2 seconds. Therefore, I:E is 1:4
- If Itime is 1.2seconds, then Etime is 2.8 seconds. Therefore, I:E is 1:2.3

In conclusion, a longer Itime will shorten the I:E ratio, giving less time to exhale.

Respiratory Rate

As the respiratory rate increases, the I:E ratio narrows—more breaths per minute means more of the time ratio spent in the inspiratory phase. Let's look at another comparison scenario: an RR of 10bpm versus an RR of 20 bpm. (The Itime is set at 1.0 seconds for both of these scenarios.)

- If the RR is 10bpm = 6 seconds per inspiratory/expiratory phase. With an Itime of 1.0 second, then the Etime would be 5 seconds. Therefore, I:E is 1:5.
- If the RR is 20bpm = 3 seconds per inspiration/expiration phase. With an Itime of 1.0 second, then the Etime would be 2 seconds. Therefore, I:E is 1:2.

In conclusion, a higher RR will shorten the I:E, giving less time to exhale.

In order to ensure adequate time for exhalation, the I:E ratio must never be allowed to invert. Inverted I:E refers to more time in inspiration than expiration. If this inversion were to occur, **gas trapping** is almost a certainty. Gas 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. The term *gas-trapping* is sometimes referred to as **Auto-PEEP**.

Conventional ventilation strategies suggest I:E ratios of around 1:2—meaning expiration should be twice as long as inspiration. Higher I:Es 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 will be shorted to a 1:1 ratio with very careful monitoring that gas trapping is not occurring.

A shorter I:E can be monitored by skilled clinicians in the ventilator waveforms. This skill is beyond the scope of this course, but remembering the key concepts of keeping Etime longer than Itime and targeting an I:E of 1:2 will allow you to avoid gas-trapping and ventilate effectively for the vast majority of patients. In rare cases of needing 1:1 I:E ratios, speak to your physician or respiratory therapists, and refer to any physician orders you receive. Monitor your patient closely for signs of discomfort or hemodynamic instability.

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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.

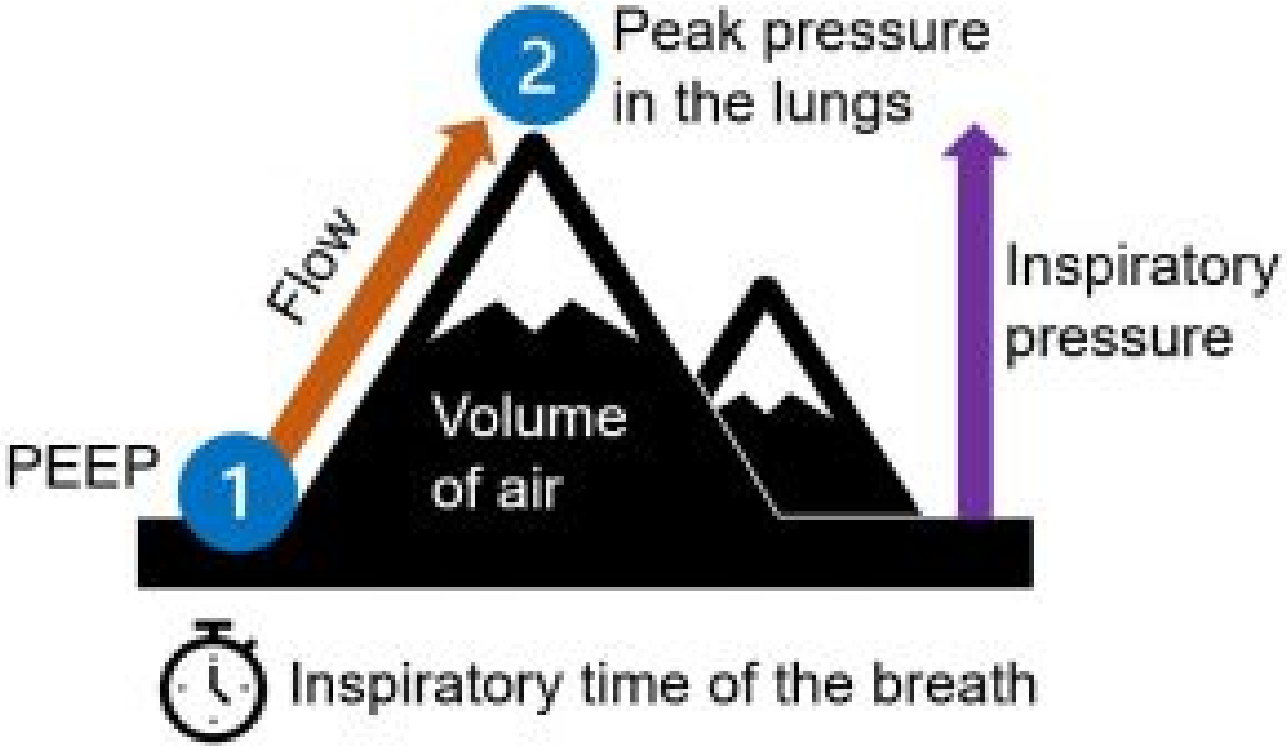
The patient's progression through their ventilated breathing can be likened to traversing up and down a range of mountains.

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:



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.

- **Itime (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.

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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 healthcare professionals who are new to the world of mechanical ventilation. The clinician sets how big (tidal volume) a breath and how often the breath is delivered. 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.

Usually in volume control the set time is controlled by a flowrate, but some ventilators will let you set an Itime instead. For the purposes of this book, we will use flowrate as the primary time variable in volume control, as it is the most utilized time variable for most volume control modes in different ventilators.

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. 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. 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.

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.

> PEEP is not the same as pressure. PEEP is ensuring the lungs do not collapse. It does not refer in any way to the distending pressures experienced by the alveoli.

So how do you limit the pressure being experienced in the lungs when you are in a "volume" 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 felt by the lungs.

Key Takeaways

- Pressure and volume have a linear relationship. If you decrease the volume, you are inflating the lungs to, 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.

Summary of Basic Settings

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

- Respiratory Rate (RR)
- Tidal Volume (Vt)
- Max flow rate (V) or Inspiratory Time (I_T)
- PEEP (every ventilator, every mode)
- FiO_2 (every ventilator, every mode)

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

- 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.

Damanhur, Federation of Communities, via CC Search

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.

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.

To conclude, in volume control, the volume delivered is constant, but there will be changes in peak pressures (PIP or Ppeak, 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 30cmH_20, with a maximum of 35cmH_20, to ensure the alveoli do not get damaged. We will discuss this concept further in Chapter 5 when we discuss Volume Control settings.

Key Takeaways

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 peak pressures lower than $30cmH_20$, with a hard maximum of $35cmH_20$.

Hospitals tend to have preferences for how ventilators will be used, based on policies and procedures. Please refer to any hospital-specific policies for preferred modes at your specific place of work.

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, you set the volume, but in pressure control, you do not directly set the volume. Instead, in pressure control you set the pressure.

Pressure Control Ventilation (ACPC, PCV, CMV-PC) is a full control mode of ventilation—the ventilator will control all phases of the breath delivered based on the set parameters. Exhalation is still passive. The clinician sets a minimum of how often breaths will be delivered 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 if the pressure changes? 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.

Let's apply this concept to our asthma patient. During an acute asthma exacerbation (asthma attack), 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 volumes 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. We will discuss this concept further in Chapter 5 when we outline pressure control settings.

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.

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 (Vt)
- Inspiratory flow (V)
- Peak Inspiratory Pressure (PIP or Ppeak)
- 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 (It). What is not set in Pressure Control Ventilation (ACPC) 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 (It). 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 (V) is not a set parameter either. The clinician sets the Inspiratory time (Itime) 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 Itime.

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.

Peak Inspiratory Pressure (PIP) is not directly set by the clinician, but it remains constant and very controllable in pressure control ventilation, as it is the sum of all pressures experienced in the lungs (PEEP + PC). The PIP is the highest pressure being delivered to the alveoli. Since the pressure being delivered (PC) is set, the PIP is the PC 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 (PC). Take look at these two examples:

- PEEP 5 + PC 10 = PIP 15
- PEEP 8 + PC 12 = PIP 20

Apply Your Learning

Now you try! Your ventilator settings are PEEP 6, PC 15. What is your PIP? What would your PIP be if you decrease your PC to 12?

The I:E—as explained in the previous chapter—is very important to ensure the patient has adequate time to exhale and decrease the risk of auto-PEEP (gas-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 Itime 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, I_T 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 Itime 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.

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.

Hospitals tend to have preferences for how ventilators will be used, based on policies and procedures. Please refer to any hospital-specific policies for preferred modes at your specific place of work.

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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 statement are true:

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 (>30-35cmH20), 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:

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:

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.

CHAPTER 4 SELF-CHECK

Chapter 4 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:
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CHAPTER 5 | CONTROL MODES: INITIAL VENTILATOR SETTINGS

Overview

We have discussed in detail control modes of ventilation and differentiated between volume and pressure control. In Chapter 4, we highlighted the settings that need to be set for control modes. In this chapter we will discuss how to approach choosing initial settings for a patient when you first initiate mechanical ventilation.

> The scope of this book is adult ventilation. The settings discussed in this chapter are suggestions for adult populations only. Please refer to your 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. Refer to your ordering physician or registered respiratory therapist.

In this chapter we will cover:

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

Application

Once arterial blood gas (ABG) results come in, clinicians can make better informed decisions about ventilator settings, but you need to start somewhere! Clinicians 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

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.

MANDATORY SETTINGS IN CONTROL MODE: A REVIEW

As previously stated, for all mechanical ventilation modes, the clinician will always set FiO2 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.

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

Volume Control		**Pressure Control**
	FIO_2	
	PEEP	
	Respiratory Rate (RR)	
Tidal Volume (V_T) and Inspiratory Flow (V̇)		Pressure control (PC) and Inspiratory time (IT or I_{Time})

There are other settings that you will see on the ventilator that are not listed here. One will be trigger sensitivity (i.e., sensing a patient-initiated breath), as well as some secondary cycling settings, or, potentially, flow of air if the clinician is in volume control. These settings are pre-programmed with mechanical ventilators to default to numbers that work the majority of the time.

Because mechanical ventilators will default to a sensitivity trigger that will work for 99.9% of patients, medical providers who are not skilled in mechanical ventilation should focus on determining the initial settings listed above, and *do not adjust the trigger sensitivity*. There are very rare times that adjustment is needed, but it is best to leave adjustments of trigger sensitivity to advanced clinicians. Inappropriate adjustments are far more harmful to the patient than sticking with the default settings.

Practice Makes Perfect

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/mechanicalventilators/?p=488#h5p-5

CHOOSING INITIAL SETTINGS FOR A CONTROL MODE

When setting the ventilator, a "one-size-fits-all" mentality cannot 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 SpO2 >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 Concept

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, you will set them and then wait until you get arterial blood gases (ABGs) after waiting a minimum of 30 minutes. ABGs will be discussed in detail in Chapters 8 and 9.

Initial PEEP settings should be anywhere from 5-10 cmH_20. Remember, the minimum PEEP is 5 cmH_20. 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 cmH_20. For clinicians without advanced ventilator training, PEEPs of 8 or 10 should only be considered for initial settings when you have patients with known **atelectasis** (collapse of some alveoli), pulmonary edema or evidence of thickening of the alveolar-capillary membrane in their diagnosis.

Remember, increasing PEEP is not without its dangers (see Chapter 2). 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:

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 cmH_20
	Known atelectasis or thickening of their alveolar-capillary membrane	8-10 cmH_20

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 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 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 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 bpm, a clinician should start at 18-20 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).

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 Concept

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.

Mode specific settings: Tidal Volume or Pressure Control

We have reviewed the settings for PEEP, FiO_2 and also the frequency of breath delivery (RR). The final setting the clinician needs to decide on in control ventilation is how big of a breath the patient will need. You need to control the tidal volume (VT) that will be delivered with every breath.

Chapter 4 covered how the tidal volume is physically set in volume control, but in pressure control, the volume delivered is controlled by the pressure applied over a set amount of time. Remember the balloons object lesson? If you blow air for a set time, the balloon will inflate to a set volume. If this concept is a little fuzzy, go back to Chapter 4 and review the relationships between volume and flow, pressure and Itime for breath delivery.

We'll talk about appropriate volumes first, on the next page, because this will dictate what you will set—either tidal volume or pressure control and Itime.

This ventilator is ready for the clinician to input initial settings. Notice that the word "Standby" appears where we would normally see patients' breaths (waveforms).

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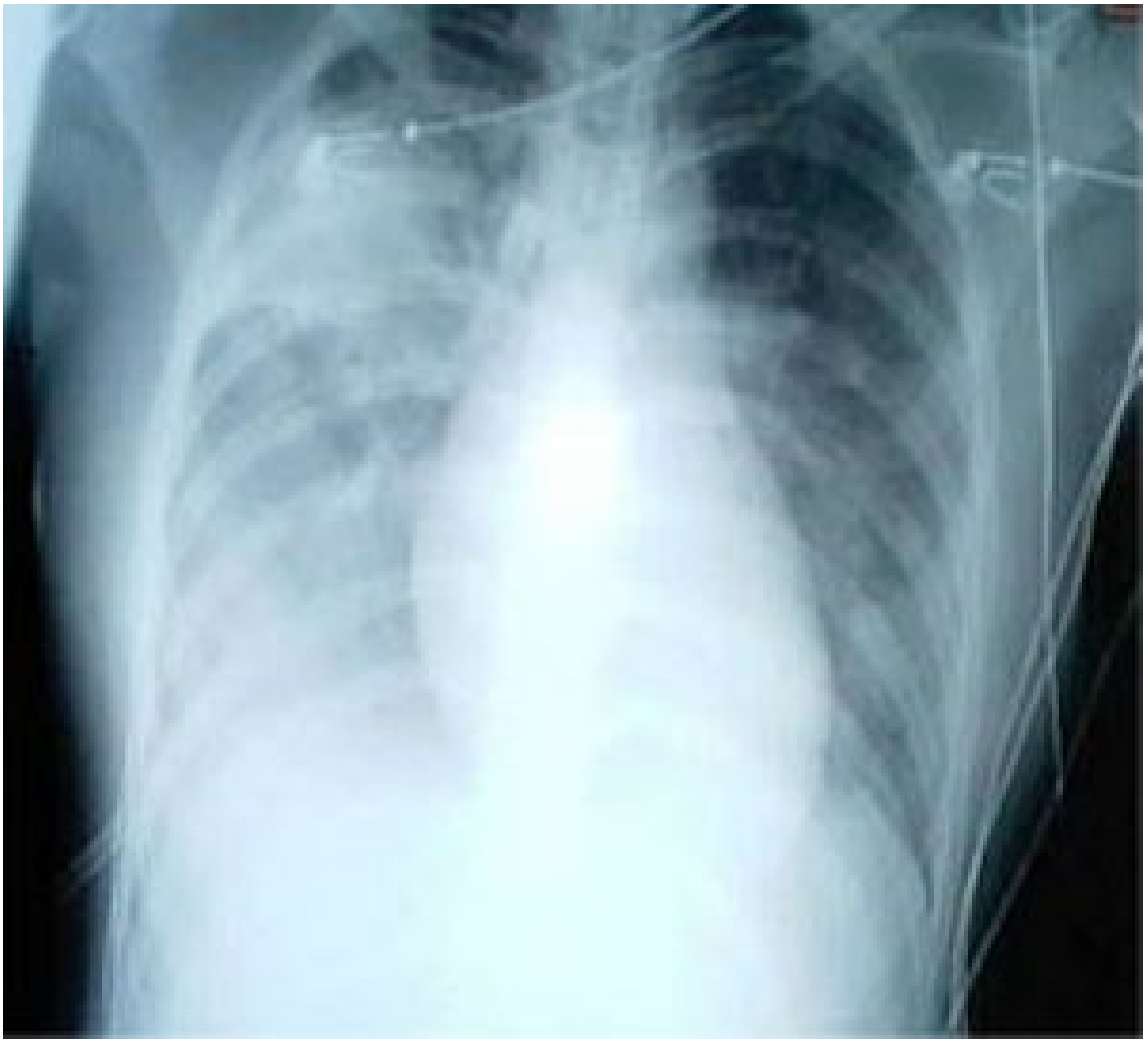
IDEAL BODY WEIGHT

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 ml) and slow RR (10-12 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)**.



This x-ray shows bilateral infiltrates on both lungs. A ventilator-induced lung injury could appear similar to this.

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

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). Health care providers were directly harming their patients with this high volume/low RR ventilation strategy.

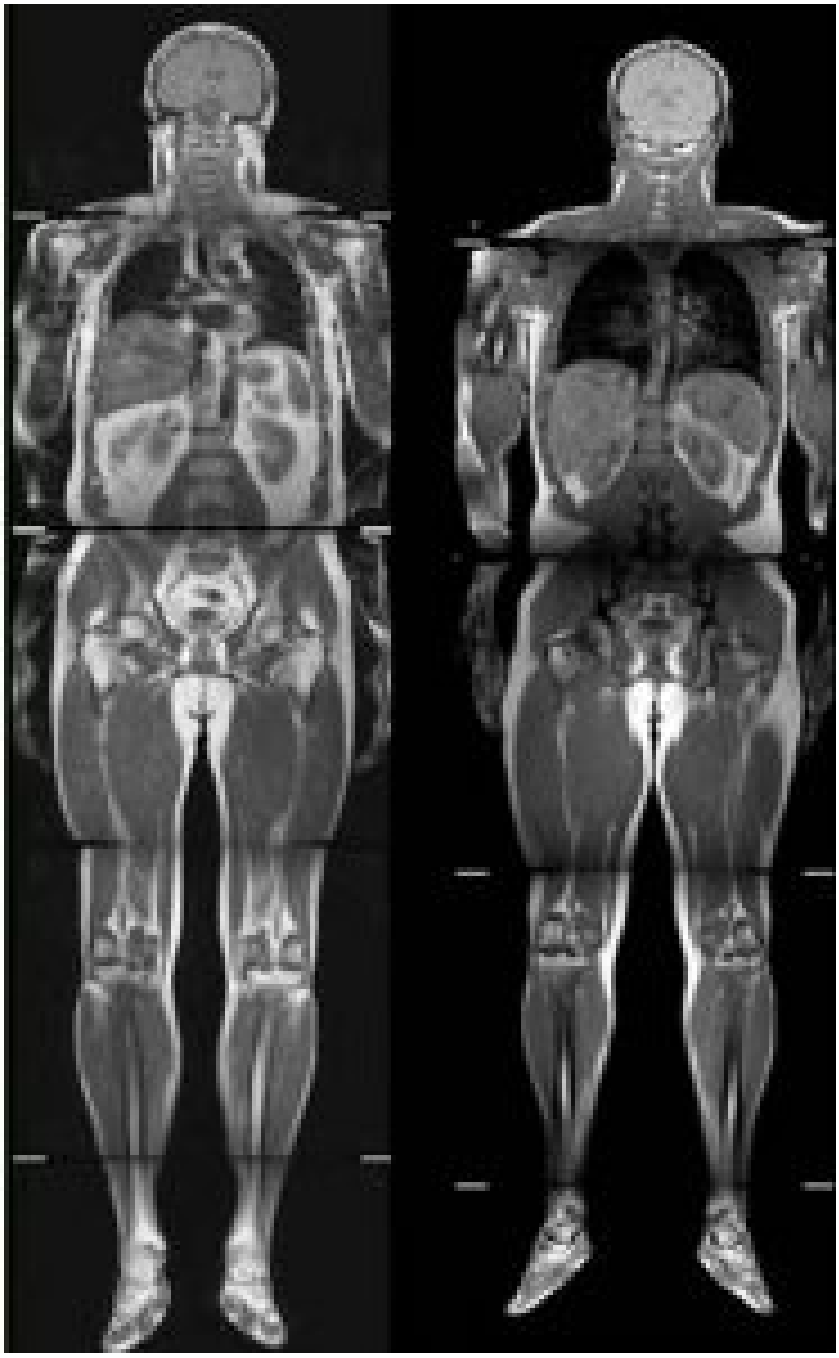
Object Lesson

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.



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).

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:

Sex	**IBW formula (adults only)**
Male	IBW (kg) = 50 + 2.3 (height in inches – 60) or IBW kg) = 50 + 0.91 (height in centimeters – 152.4)
Female	IBW (kg) = 45.5 + 2.3 (height in inches – 60) or IBW (kg) = 45.5 + 0.91 (height in centimeters – 152.4)

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 kg for males or 45.5 kg for females—which are the minimum sizes for adult lungs.

RRT Hack: If you know a patient's height in "feet and inches," instead of converting this number to inches, you can save yourself a step. 60 inches is 5 feet tall. You are "minusing" 5 feet from their height in the formula, so a handy shortcut is to just write how many inches tall they are above 5 feet in that bracket (e.g., if a person is 5 foot 6 inches, you would just use the number 6 in brackets, which you will then multiply by 2.3 and add to the base number).

> If you live in the United States and utilize pounds to express a person's weight, there is a version of this formula that uses pounds and then converts the results to kilograms. If it sounds like this formula would be helpful to you, please watch this video, which uses pounds.

Let's do a few calculations together!

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

IBW (kg) = 50 + 2.3(74-60)

IBW (kg) = 50 + 2.3(14)

IBW (kg) = 50 + 32.2

IBW (kg) = 82.2 kg

Now, let's try using the shortcut described above. You know they are 6'2" so this is 1 foot (12 inches) plus 2

inches above 5 feet tall (60 inches) = 14 inches. Just plug 14 into the bracket. Look! You are already on step 2 of the formula! Mental math skips the step of having to figure out how many inches if you do not have it handy.

Patient B | A female who is 5'3"

IBW (kg) = 45.5 + 2.3(63-60)

IBW (kg) = 45.5 + 2.3(3)

IBW (kg) = 45.5 + 6.9

IBW (kg) = 52.4 kg

Now, let's use the shortcut... the patient is 3 inches taller than 5 feet. We subtract 60 inches (5 feet) from their height to do the calculation. Therefore, we take how much taller she is above 5 feet, and that number of inches goes in the bracket. Therefore 3 goes in the bracket to multiply with 2.3 and then add to the female constant of 45.5.

Patient C | A male who is 175 cm

IBW (kg) = 50 + 0.91(175 – 152.4)

IBW (kg) = 50 + 0.91(22.6)

IBW (kg) = 50 + 20.566

IBW (kg) = 70.566 kg

Because this formula uses centimeters instead of inches, the "shortcut" does not apply.

Media Attributions

IBW AND THE RELATIONSHIP TO TIDAL VOLUME

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. Note: you will always round your result up or down to match the settings on the ventilator (usually, they use whole numbers only and count by 5s).

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 ml/kg.

Patient A | IBW 82.2 kg

Tidal volume of 6ml/kg = 82.2 kg x 6 ml/kg

= 493.2 ml

= approx. 490ml

Tidal volume of 7ml/kg = 82.2 kg x 7 ml/kg

= 575.4 ml

= approx. 575 ml

Tidal volume of 8ml/kg = 82.2 kg x 8 ml/kg

= 657.6 ml

= approx. 660ml

Therefore, the safe range of ventilation is 490-660ml (6-8ml/kg) and the respective tidal volumes for 6,7,8 ml/kg are 490, 575 and 660 ml respectively.

Patient B | IBW 52.4 kg

Tidal volumes 52.4 x 6 = approx. 315 ml

52.4 x 7 = approx. 365 ml

52.4 x 8 = approx. 420 ml

The safe range of ventilation for this patient is 315-420ml and the respective tidal volumes for 6, 7, and 8 ml/kg are 315, 365 and 420 ml respectively.

Key Concept

For every patient being initiated on mechanical ventilation, the health care provider will determine their height, calculate their ideal body weight based on height and sex, and then multiple it by 6-8ml/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 ml/kg to decrease the risk of barotrauma/volutrauma. Refer to your health care facility and physician orders for your low limit based on patient pathophysiology, but 8 ml/kg is the absolute maximum you will every use for a tidal volume. You will not go above this number unless specifically ordered by a doctor. The 8 ml/kg number is a hard limit, and exceeding this limit is never accepted in critical care medicine since the studies came out proving how harmful it is to the patient.

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

VOLUME CONTROL SPECIFIC SETTINGS: TIDAL VOLUME AND FLOW

In volume control, the clinician who is setting the ventilator will dial in the set volume for each breath. Remember, in volume control we will set the volume, but in pressure control, the volume will be based on the pressure and Itime that is set by the clinician.

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 6-8ml/kg range. For people that are new to ventilation, starting at 8 ml/kg of IBW is the best recommendation for adult ventilation strategies. Why is this? Because most patients who are being intubated have compromised ventilation, and their CO_2 levels are not being maintained with changes to how they are breathing. Giving the patient the largest "safe" breath you can will ensure the patient is breathing in as much oxygen as possible and exhaling as much CO_2 as possible per breath. You can always come lower on your tidal volume later after follow-up bloodwork. Start with 8 ml/kg and then reassess after that.

Setting	Steps	Initial Setting
VT	Calculate Ideal Body Weight (IBW) and multiply with 6-8 ml/kg to get your safe range of tidal volumes	8 ml/kg

In Volume Control, you also usually set the max flow of air going into the lungs. Some ventilators will ask for you to set an Itime instead (discussed below) of a flow, but the classic versions of volume control have a max inspiratory flow setting. For users who are not experienced with ventilators, the default for an adult 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.

The only time where you might consider increasing the flow above 65 lpm is if you have a patient who is triggering breaths and gasping or pulling the air in beyond what the ventilator is supplying. Usually, when this happens, the ventilator alarms are activated. You can try increasing the flow to 70 or 75 lpm until you satisfy their air flow needs. The max flow you should ever set is 80 lpm, but this setting would be used for a very small number of patients. They will be spontaneously triggering breaths, and if you are struggling, you may consider trialing a spontaneous mode (to be discussed in the next chapter) or more sedation if small increases in flow do not satisfy the patient's need.

In the beginning, leave the flow at the default of 65 lpm with a decelerating flow pattern and do not change

this unless you really think the patient is pulling air quicker than the ventilator. When in doubt, sedate your patient and continue to ventilate with the advised flows of 65 lpm.

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). This is why flows should only be adjusted if the patient is definitely outstripping the ventilator's delivered flows. Watch your peak pressures and keep them below 35 cmH_20 (PIP <35cm H20).

Setting	Patient Status	Initial Setting
Flow	All adult patients (except below)	65 lpm, decelerating pattern
	Patients triggering additional breaths, who appear to be gasping and causing the ventilator to alarm	Titrate flow up to 80 lpm (increase by 5 lpm at a time)

Appropriate ventilator settings are key to avoiding damage to the lungs.

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PRESSURE CONTROL SPECIFIC SETTINGS: THE PRESSURE CONTROL AND INSPIRATORY TIME

When in a pressure control mode, 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_20.
- Too low? Increase the PC by 2 cmH_20 and assess.

A safe pressure control to start at is 14 cmH_20. Even with a slightly higher PEEP, it will ensure your peak pressures are still well below 35 cmH_20.

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 ml/kg.

Note: Being off by approximately 20 ml is not an issue. You will never be exactly the same. Try to stay under the 8mls instead of over. If you increased the PC by 1 cm H_20 and your tidal volumes go from below 8 ml/kg to above, then undo that change and leave your volumes just below your maximum. Remember 8 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 I:E is 1:1 (remember Chapter 4? If needed, go back and review), 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 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; consult your physician or RRT if the default Itime of 0.8-1.0 seconds is causing issues, alarms, or an I:E that is 1:1 or inverse.

Default Itimes of 0.8-1.0 seconds should be fine for all your patients as long as the RR is less than 24bpm. If directed by a physician or RRT to increase the RR, the Itime may start to be adjusted to ensure the I:E stays greater than 1:1. For the purposes of this course, we are discussing overall ventilation strategies that will work for most patients. For difficult-to-ventilate patients, refer to your physician or RRT.

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

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.

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

- High Respiratory Rate
- High Pressure Limit
- High Volume
- Low Volume

- High Minute Volume
- Low Minute Volume

> 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:

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_20 (max) or +10 cmH_20 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 cmH20 if possible. This is just the maximum limit.
Low Pressure	Set 2 cmH_20 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.

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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 Chapters 8 and 9, but remember, you need to start somewhere! Clinicians choose initial settings by employing educated guesses regarding their patient's ventilatory needs based on how they presented prior to intubation.

In summary, these are the initial settings you have learned in this chapter:

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_20	5-8 cmH_20	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	14 cmH20 then titrate up or down to get a VT 8ml/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 seconds	Can shorten slightly if the patient is trying to exhale (down to 0.8 seconds)

CHAPTER 5 SELF-CHECK

Application Exercise

Chapter 5 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/mechanicalventilators/?p=555#h5p-6

Advance Your Learning

Additional exercises, application notes, case studies and live support from an experienced instructor are available to learners who enroll in the Mechanical Ventilation micro-credential course at Sault College. Students who successfully complete this online course will earn a micro-credential certification in Mechanical Ventilation. Please visit training.saultcollege.ca for more information or to register.

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CHAPTER 6 | SPONTANEOUS MODES AND SIMV

We have now overviewed control modes of ventilation and talked about when and how to control a patient's breathing. Remember in Chapter 3 when we talked about the differences between Control and Spontaneous Modes? 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)
- When we use PSV
- PSV settings
- How we choose the correct settings when initiating PSV
- What is SIMV, and why it is not a preferred mode

Application

As you 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 should not be used in typical ventilation practice today. Since SIMV is still used by some out-of-date practitioners and 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 PSV.
2. Describe typical settings for PSV.
3. Determine appropriate PSV settings based on assessment of a patient.
4. Explain why SIMV should not be used as routine practice, in order to advocate effectively for your patient.

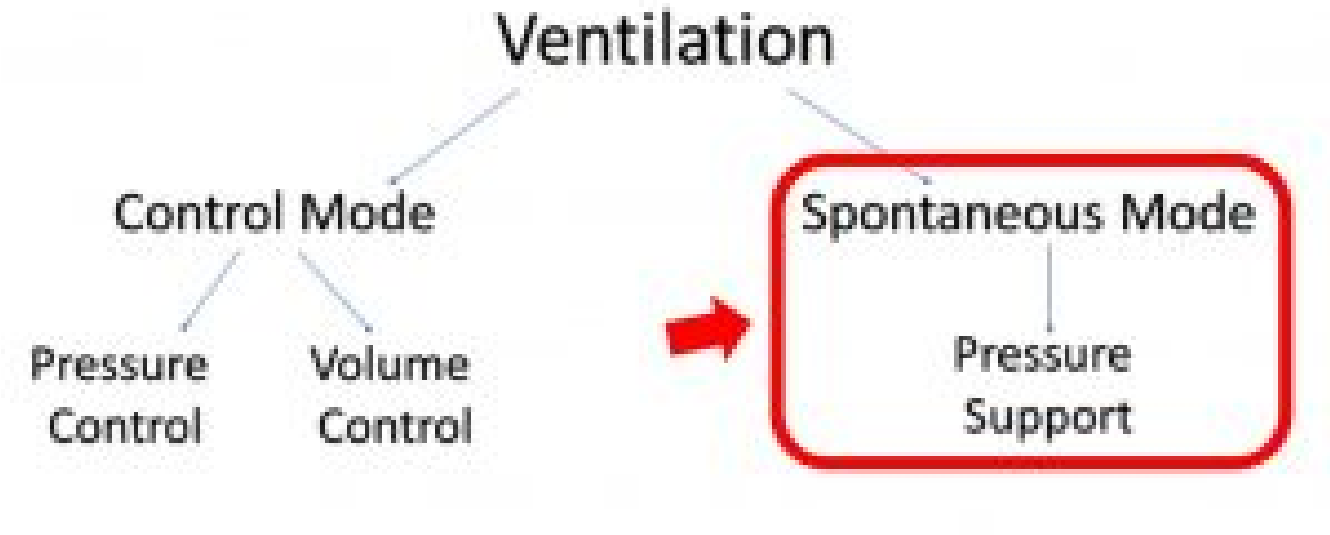
Key Terms

- muscle atrophy
- weaning
- Pressure Support Ventilation (PSV)
- Volume Support Ventilation (VSV)
- 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.

SPONTANEOUS MODES: A REVIEW

Now that you have learned all about control modes, it's time to shift to the other branch of the ventilation "family tree": spontaneous mode:



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 (Chapter 1)
- Mimicking physiologic breathing is much better for the body (Chapter 1)
- Asynchrony can happen in control modes because the ventilator is delivering breaths based on what is set and not the patient (Chapter 3)

In this chapter, we will discuss these points in detail, as well as highlight additional benefits of spontaneous modes.

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SPONTANEOUS MODES VERSUS CONTROL MODES

As we have described in Chapter 3, 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.

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 (see Chapter 3) 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 any of 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 it helps 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 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 Concept

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:

	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 CO2 levels	Used to fix abnormal gases	Can only augment normal breathing; not indicated for very high CO2 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.

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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 for 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 is 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 Concept

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 is ventilation. Many healthcare practitioners probably are already skilled in this area. 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 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 "Use 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/mechanicalventilators/?p=585#oembed-1

If you require a transcript for this video, please click Watch on YouTube to view the video source. Then, you can access the written transcript from this page.

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. 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? 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. The respiratory therapist has tried to change the settings in pressure control, but cannot fix the patient double-triggering breaths and fighting the ventilator.

Weaning or comfort? 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 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? 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.

TYPES OF SPONTANEOUS MODES

Just like control modes, there are several spontaneous modes available on the market, but unlike control modes, there is really only one that is mainly used. **Pressure Support Ventilation (PSV)** is by far the most common support mode utilized in critical care. **Volume Support Ventilation (VSV)** does exist, but it never gained traction in the market—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.

Later in this chapter, **Synchronized Intermittent Mandatory Ventilation (SIMV)** will also be discussed. Thought of as a "mixed" mode—control and spontaneous, it is no longer a mode that has a significant place in ventilation. A brief discussion will be included regarding why SIMV is no longer used in intensive care units across North America.

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 revolutionary implications for how patients breathe on a ventilator. PAV will not be covered at length in this book. However, supplementary information on PAV is available for students of the Basic Principles of Mechanical Ventilation course at Sault College.

The COVID-19 pandemic saw a massive increase in ventilated patients, with make-shift ICUs and new clinicians recruited to meet the need.

Pressure Support Ventilation (PSV)

In PSV, every time a patient initiates a breath, the ventilator will push a set pressure of air to the patient while they spontaneously breathe in. The patient plays the only role in deciding how often to breathe and can also vary their volumes based on what they desire—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 Itime (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

Key Concept

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.

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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 quick as possible based on patient status and tolerance of changes.

Minimal settings refers to minimal pressure support as well as minimal PEEP (PEEP of 5 cmH_20 as discussed in previous chapters). Once minimal settings are reached, this is termed a **Spontaneous Breathing Trial (SBT)**. SBTs remain the standard in most ICUs as one of the definitive tests to assess if the patient is ready for discontinuation of 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 5-7 cmH_20). 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.

Don't worry! ABGs will be covered in more detail in Chapter 8.

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:

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If you require a transcript for this video, please click Watch on YouTube to view the video source. Then, you can access the written transcript from this page.

For additional information about post-ventilator recovery, please see the following links:

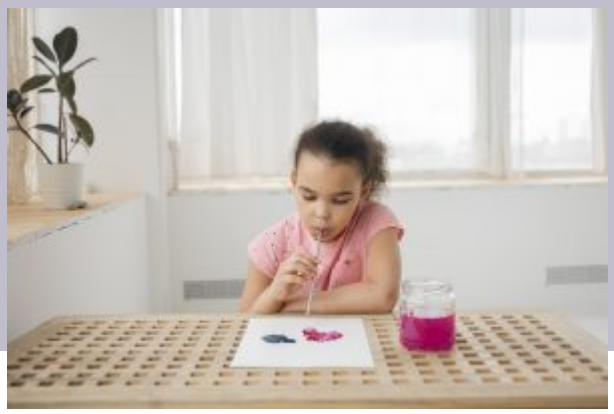
- Life After a Ventilator (UNC Health Talk)
- Ventilator Weaning and Spontaneous Breathing Trials; an Educational Review

PRESSURE SUPPORT: SETTINGS AND HOW TO SET THEM

Normal pressure supports fall between 5-20 cmH_20 depending on the patient's needs. In most cases, the pressure support is never lowered below 5 cmH_20. The minimum of 5 cmH_20 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.



The added pressure support of 5 cmH_20 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_20 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 (as discussed in Chapter 5), 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. Please refer to physician specific directives for your specific patient populations.

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 bpm) or tidal volumes that fall above safe volume ranges of 4-8 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 5cm H_20. 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

Setting	Starting Point	Additional Instructions
PEEP	Use the same PEEP that was utilized on the previous control mode. or Start at PEEP 5 cmH_20 and titrate up if needed to assist with oxygen delivery by 1-2 cmH_20*NOTE: 5 cmH_20 is the minimum, PEEPs higher than 10 cmH_20 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 cmH_20 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 30-60min to ensure the patient is tolerating it.
Trigger, exp %, other settings	No changes from default unless instructed by a physician or advanced ventilation practitioner.	These settings are defaulted to fit 95% of patients. For basic ventilation strategies, they do not need to be adjusted. Advanced providers may utilize these settings.

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)
- VT 380 ml (6 ml/kg)
- PEEP 8 cmH_20
- FiO_2 0.40

Is this patient a candidate for PSV?

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?

Change the patient to pressure support ventilation by switching the mode. Before accepting changes, select the chosen settings for PEEP, FiO2 and PS. For this patient, initial PSV settings could be as follows:

- PEEP 8 cmH_20 (same as previous control mode)
- FiO_2 0.40 (same as previous control mode)
- PS of 10 cmH_20.

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?

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_20 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?

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?

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?

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 cmH_20 to start. The PS can be started at 10 cmH_20 and then adjusted up if the RR is high or the tidal volume is low.

What do you do now?

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 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?

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 health care professionals might see the 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 10bpm 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.

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A WORD OF CAUTION

There are two flaws in PSV. They 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/mechanicalventilators/?p=610#video-610-1

GIF via Gfycat

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 ml/kg of IBW).

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. This mode is not discussed fully in this book, as it is not currently considered "best practice" for mechanical ventilation.

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.

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.

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 10bpm, 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.

Due to the considerations mentioned above, SIMV settings and use will not be reviewed in this book. Please refer to hospital specific policies and procedures if this mode is utilized in your health center.

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?

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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 opposed to a method like SIMV, which is mostly disused. Finally, you can describe the role of an SBT in weaning a patient off a ventilator.

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.

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_20, and the setting is consistent for every breath unless changed by the health care professional.
- PS of approximately 5 cmH_20 is considered to be "minimal settings."
- An SBT is done to check if a patient is a candidate for extubation.
- An SBT is minimal Pressure Support and minimal PEEP.
- SIMV is no longer the "gold-standard" in weaning patients due to issues with asynchrony and increased work of breathing.

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CHAPTER 6 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/mechanicalventilators/?p=636#h5p-7

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CHAPTER 7 | NON-INVASIVE VENTILATION

By definition, any application of positive airway pressure (PAP) to the lungs is considered ventilation. You have learned about both types of invasive ventilation—control and spontaneous. Now you will discover non-invasive ventilation (NIV).

This chapter covers:

- 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 CO2 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.

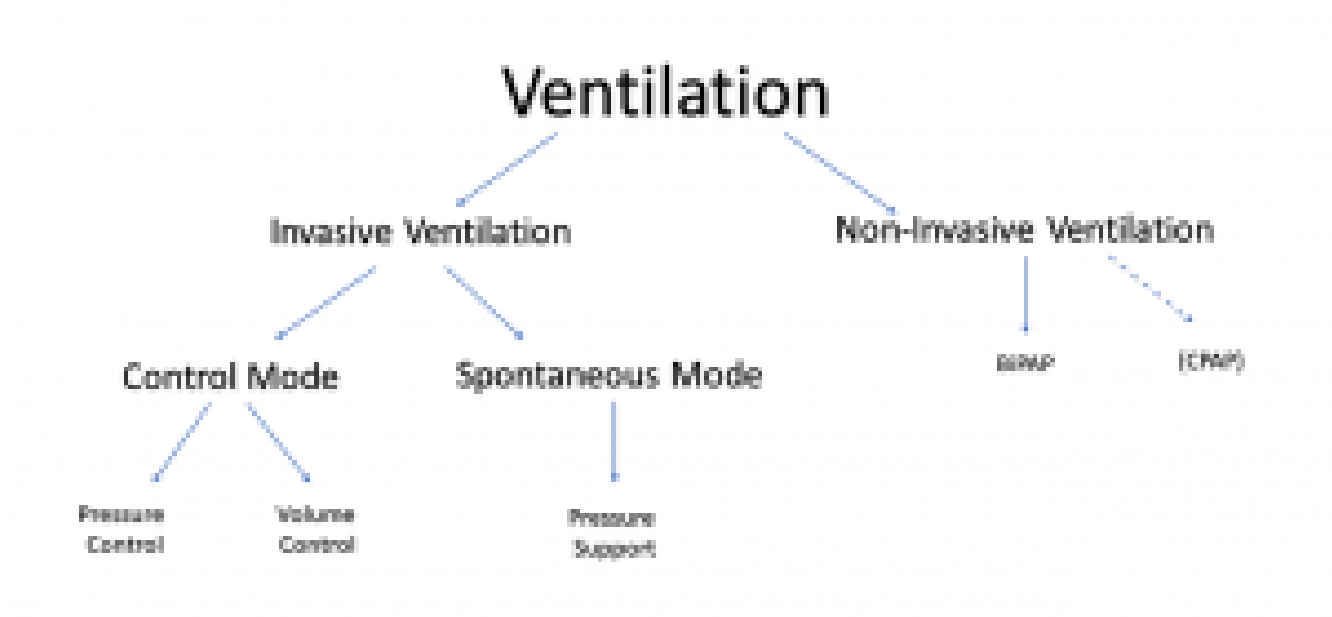
WHAT IS NON-INVASIVE VENTILATION?

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 necessary 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.

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:



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.

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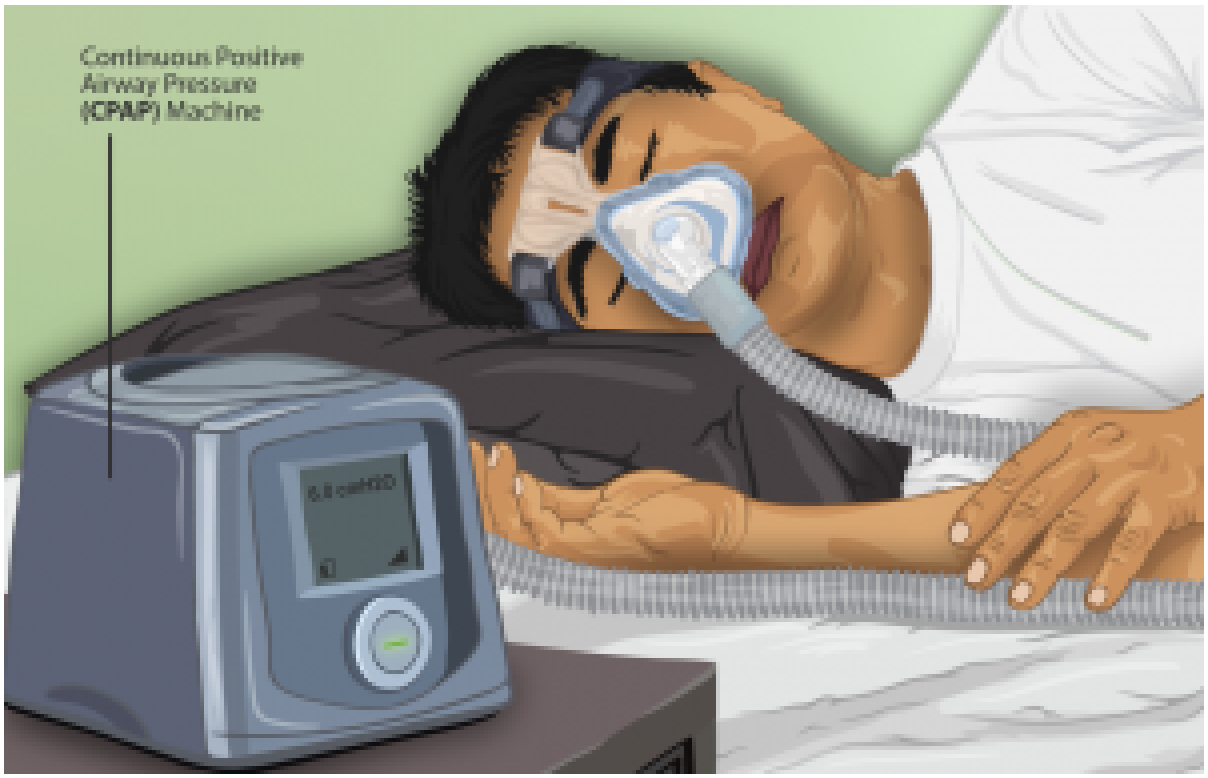
- ventilator family tree © Melody Bishop is licensed under a CC BY-SA (Attribution ShareAlike) license

CONTINUOUS POSITIVE AIRWAY PRESSURE (CPAP)

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.



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 backpressure that causes the "splinting effect."

This splinting effect can be useful for specific lungs 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 of 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 Concept

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).

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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 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.

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, benefitting 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 Concept

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:

	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 Concept

BIPAP is the application of a *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.

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INDICATIONS OF NON-INVASIVE VENTILATION

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.

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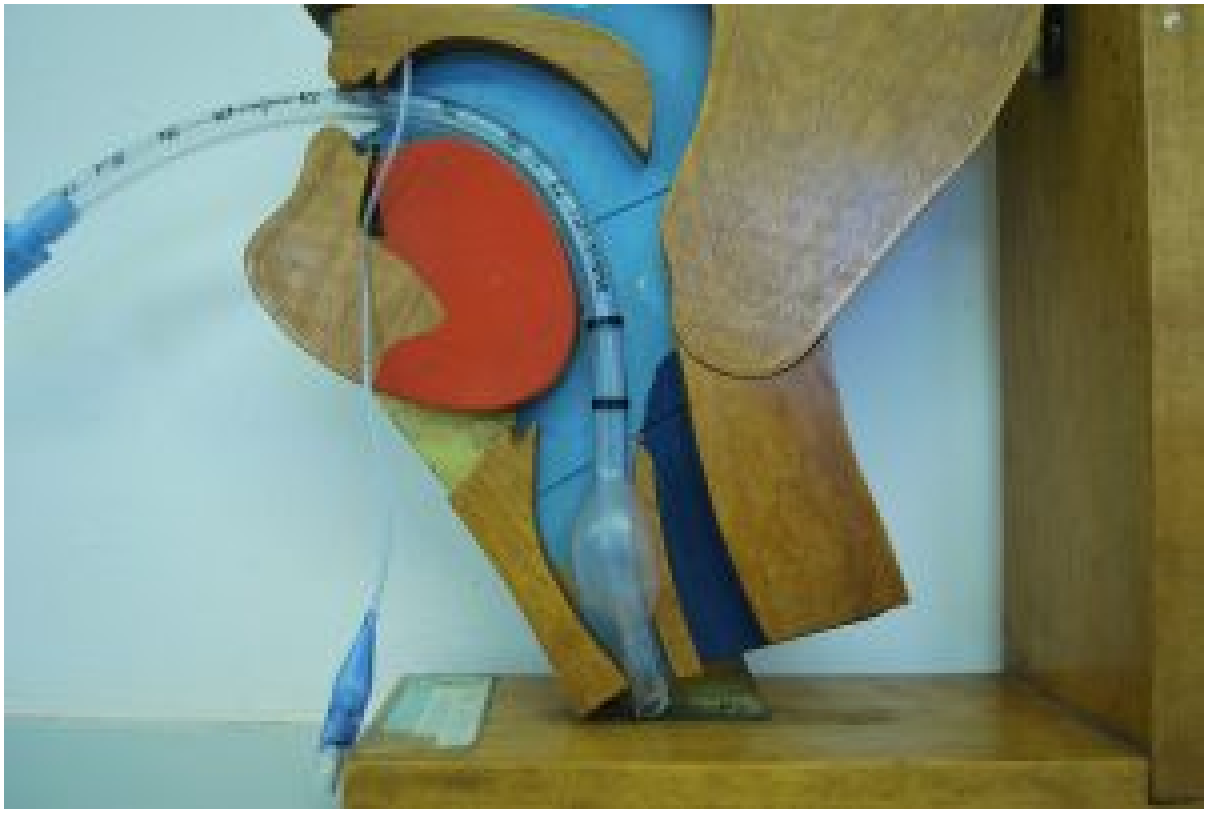
CONTRAINDICATIONS OF NON-INVASIVE VENTILATION

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.

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 only can 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.

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.

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.

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- Facial Trauma © U.S. Army photo by Sgt. Jessi McCormick/Released

THE IDEAL NON-INVASIVE VENTILATION PATIENT

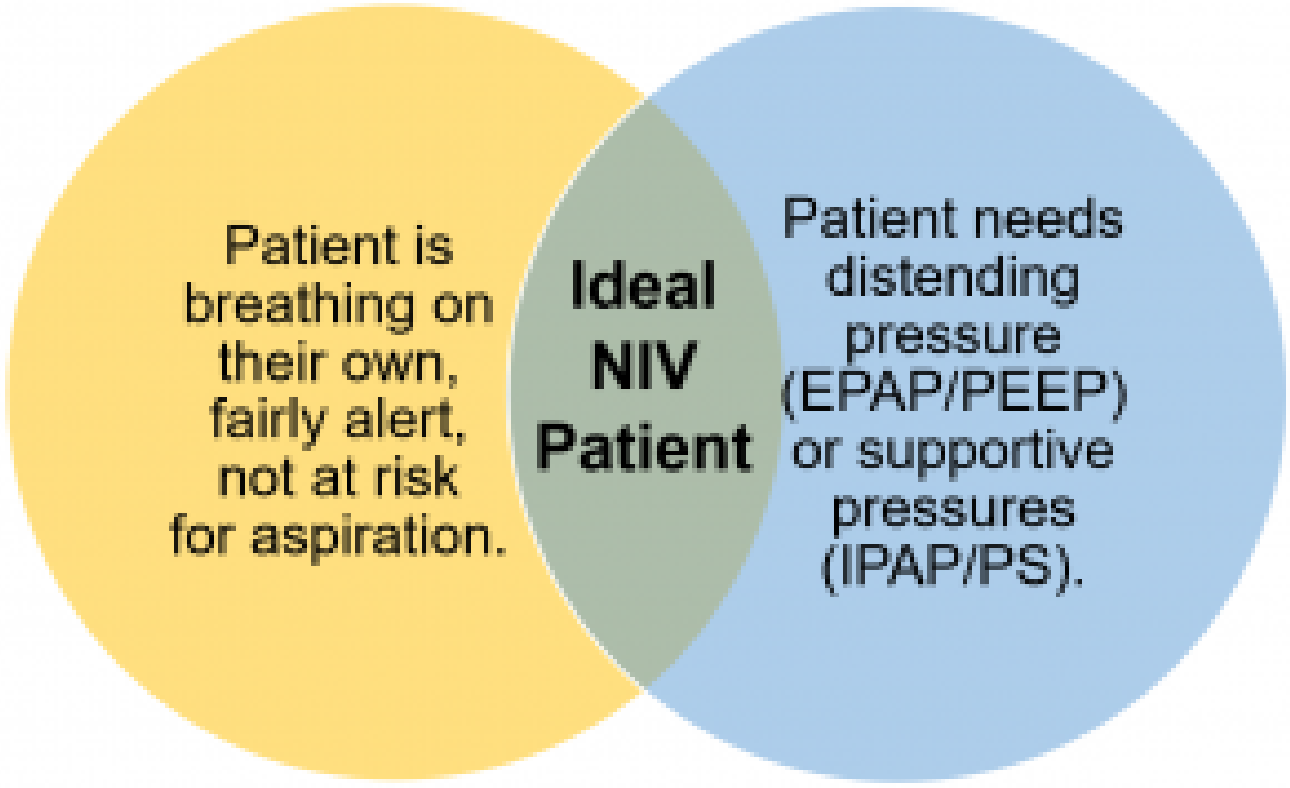
The most important step to initiating BiPAP or CPAP is in identifying the ideal patient population, and the reason NIV is being initiated.

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).



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.

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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 (delta Pressure, similar to pressure support)
4. RR

These settings will be discussed in detail below.

FiO_2

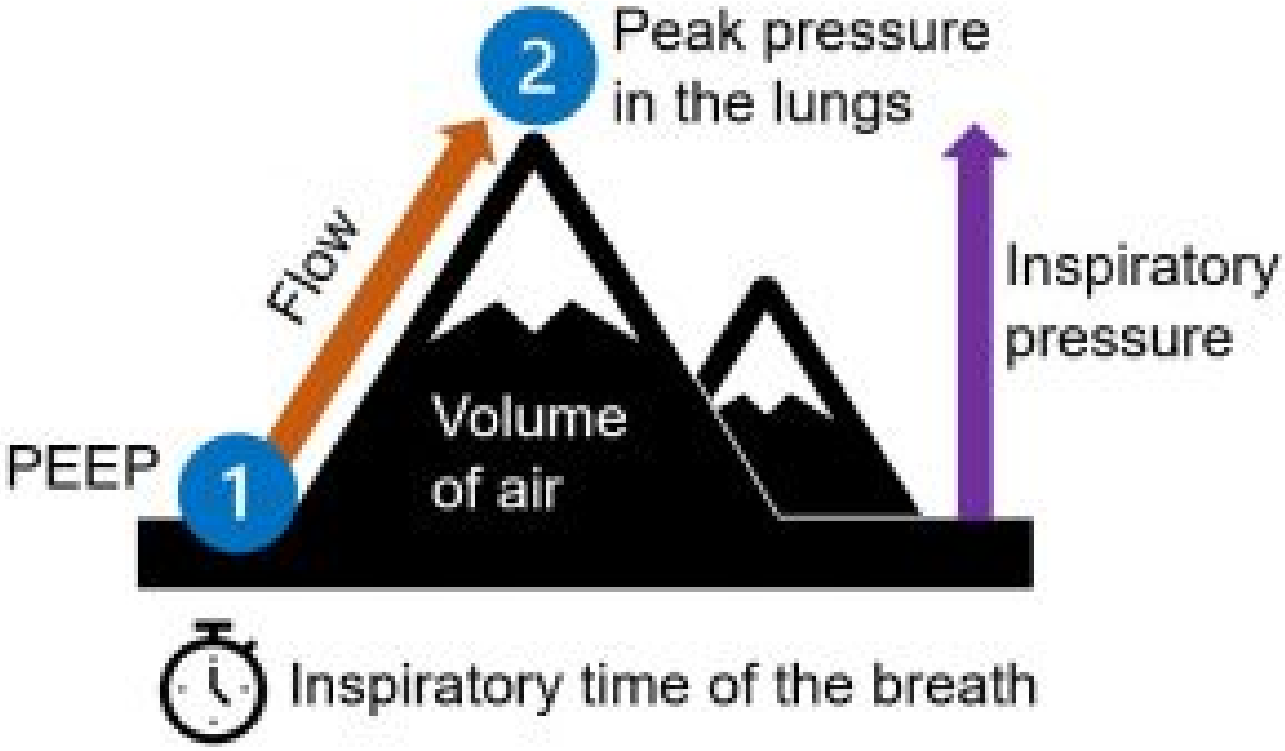
FiO_2 is mandatory to set and should be titrated based on what the patient needed before non-invasive application. If on high oxygen, start FiO_2 at 1.00 and then wean to SpO_2. If the patient does not require a lot of oxygen, starting at 0.50 and weaning within a few minutes to target SpO_2 >92% is 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 cmH_20.

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 (delta 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 from Chapter 4:



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

Example A:

EPAP 6 and IPAP 10 → Delta P/PS of 4 (difference between EPAP and IPAP)

This is a Pressure Support of 4 cmH_20

Example B:

EPAP 6 and IPAP 12 → Delta P/PS of 6 (difference between EPAP and IPAP)

This is a Pressure Support of 6 cmH_20.

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 cmH_20 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

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 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 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 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 delta P.

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

	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	RR12 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.

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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.

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- Not a CPAP, a BiPAP Machine © Foodie in Disguise is licensed under a CC BY-NC-ND (Attribution NonCommercial NoDerivatives) license

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.

CHAPTER 7 SELF-CHECK

Chapter 7 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/mechanicalventilators/?p=711#h5p-8

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CHAPTER 8 | INTRODUCTION TO ABGS AND INTERPRETATION

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.

This chapter covers:

- The theory behind ABGs
- Normal ABG values
- How to interpret ABGs, with examples
- How to comment on oxygenation status

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.

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

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.

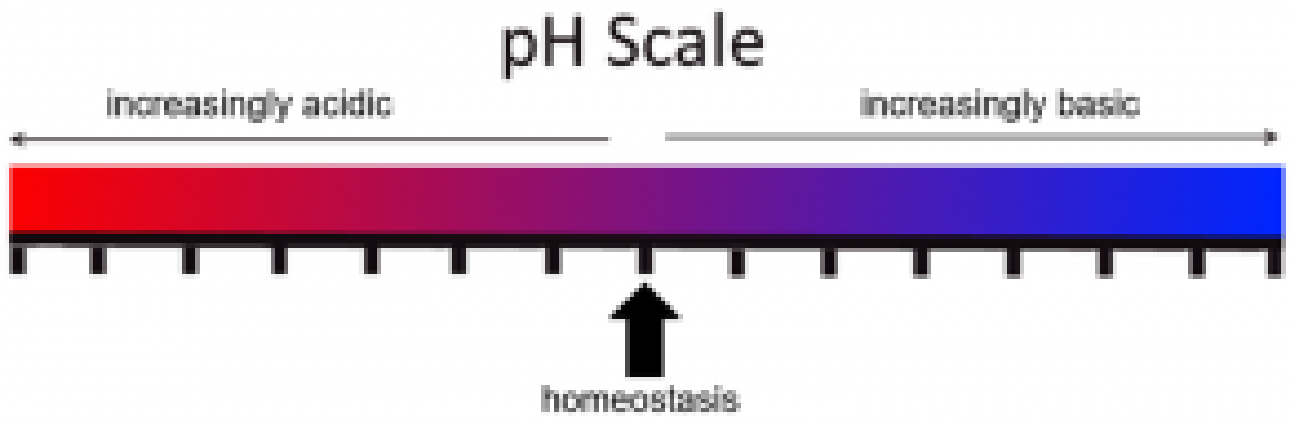
THE THEORY BEHIND ACID-BASE AND ABGS

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 and 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.



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

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 $_{CO2}$ 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:

$$\mathbf{CO_2 + H_2O \leftrightarrow H_2CO_3 \leftrightarrow H+ + HCO_3-}$$

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. $CO2$ 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 Concept

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)

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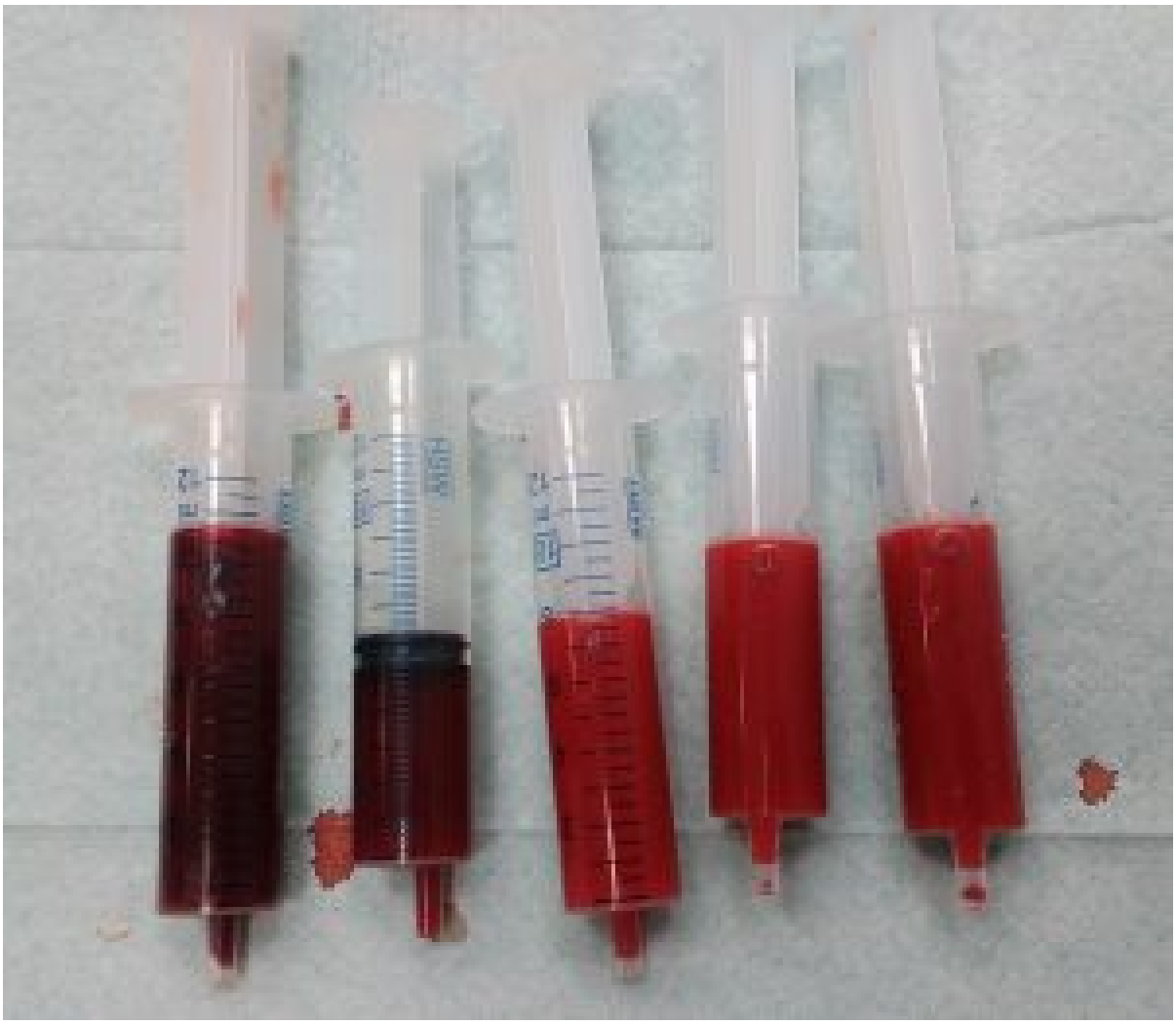
ARTERIAL BLOOD GASES

Now that you understand about the basics of acid-base balance and pH in the body. Let's talk a little more about **Arterial Blood Gases (ABGs)**. ABGs are a blood sample that is taken directly from the artery. This sample is different than normal blood work, which is taken from a vein. 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.



The difference between the oxygen-rich arterial blood and the oxygen-depleted venous blood is clear on sight. Can you tell which is which?

ABGs convey four different values:

- 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.

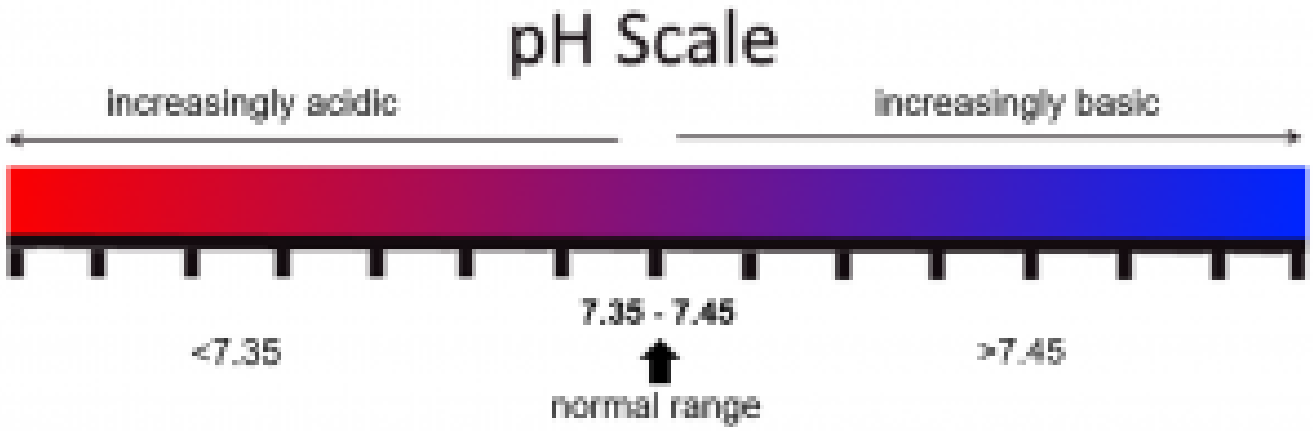
Normal Values

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.

	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 acidotic; 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:



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 Concept

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 Concept

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/pCO2/pO2/HCO3). 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.

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.

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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!

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- Arterial Blood Gas Device © PhilippN is licensed under a CC BY-SA (Attribution ShareAlike) license

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:

	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 a sticky note, 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).

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?

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.

In this case, we know the CO_2 is too high. We have already learned that CO_2 = 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/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). The answer is given at the end of this page.

7.53/37/oxygen level/33

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 Concept

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).

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?

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.

In this example, we have low CO_2 and low bicarbonate. Which one of these would cause an acidic situation? Remember CO_2 = acid and HCO_3 = 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 CO2 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). The answer is given at the end of this page.

7.47/30/oxygen level/20

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).

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?

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.

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 CO_2 = acid and HCO_3 = 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). The answer is given at the end of this page.

7.38/56/oxygen level/34

Answer Key

1. Uncompensated Metabolic Alkalosis
2. Partially compensated Respiratory Alkalosis
3. Fully compensated Respiratory Acidosis

If you didn't get all three 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/mechanicalventilators/?p=767#oembed-1

If you require a transcript for this video, please click Watch on YouTube to view the video source. Then, you can access the written transcript from this page.

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:

If PO2 is...	Comment
80-100mmHg	Normal Oxygenation
60-79mmHg	Mild Hypoxemia
40-59mmHg	Moderate Hypoxemia
<40mmHg	Severe Hypoxemia
>100mmHg	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. pO2 of 62? Add "with mild hypoxemia" to the end of your ABG interpretation.
2. pO2 of 49? Add "with moderate hypoxemia" to the end of your ABG interpretation.
3. pO2 of 93? Add "with normal oxygenation" to the end of your ABG interpretation.

A clinician completes patient documentation in an ICU.

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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?

What's next?

In Chapter 9, the final chapter of this book, you will learn what to do with your ABG interpretation in the context of choosing and adjusting ventilator settings.

CHAPTER 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/mechanicalventilators/?p=785#h5p-9

Advance Your Learning

Additional exercises, application notes, case studies and live support from an experienced instructor are available to learners who enroll in the Mechanical Ventilation micro-credential course at Sault College. Students who successfully complete this online course will earn a micro-credential certification in Mechanical Ventilation. Please visit training.saultcollege.ca for more information or to register.

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CHAPTER 9 | VENTILATION CHANGES BASED ON ABGS

You have reached the final chapter! 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 CO2 and O2 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

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 chapter end, you should feel confident to work with ventilators and ventilated patients in your professional role.

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 CO2
- intrathoracic pressure
- venous return
- ejection fraction
- over-vented

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.

CHANGES TO VENTILATION: THE GOALS

As clinical providers, 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 (see Chapter 5). Also, we have discussed the concerns of respiratory muscle atrophy if the patient is on the ventilator for too long (see Chapter 6). 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 a clinician, 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 Concept

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.

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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, 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.

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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 Takeaways

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 needed to gain increased familiarity with ventilation. Perhaps you were one of them!

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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**. Try out this term when talking to ventilation clinicians to look like a real expert.

Key Concept

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.

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 (see Chapter 2). 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 effect 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.

Take a look at the following table, which summarizes the mechanical ventilation settings that affect oxygen versus 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.

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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.

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 effect 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.

Media Attributions

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:

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 a 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.

via Giphy

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
- pCO2 is too high—we will change this to fix the pH
- pO2 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 last question...

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 VT

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 bpm, with a caution to not go above an RR of 24 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 bpm and below 18 bpm, you have flexibility to go up or down safely within the range of 10-24 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 ml/kg. Remember, we do not go above 8 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 Concept

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 6).

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_20—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_20—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.

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_20 until you get to a moderate level (10-12 cmH_20) 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_20 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...

Patient | 7.31/57/68/24

Let's say your patient is being ventilated in volume control. Their settings are: RR 16 bpm, V_T 420 ml, PEEP 5, FiO_2 0.5. Their IBW is calculated at 52 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 bpm, and you can safely increase the RR up as high as 24 bpm with minimal concerns. This would be an appropriate change.

Looking at their V_T of 420 ml, compare it to the safe V_T range of 6-8 ml/kg of IBW. The safe tidal volume

range for a 52kg person is 312 ml – 416 ml. You are already at the high range for this patient, and are ventilating at 8 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 PEEP 5 and 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 FiO2? 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 takes approximately an hour to start to fully work. If there is moderate to severe hypoxia (pO_2 is less than 60 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 (60-79 mmHg), so increasing the PEEP only might be enough as long as the patient maintains 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 is only one more question to work through...

If you're struggling to keep up, don't forget that there is guidance and extra support available for students of the Basic Principles of Mechanical Ventilation micro-credential offered online by Sault College.

You're not alone! Help from an experienced instructor is available.

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HOW MUCH DO I CHANGE THE 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? Many ventilation textbooks will suggest using formulas to calculate changes. In actual practice, these calculations can mostly be done by using simple logic. A good approach to stepwise changes is the following:

Setting	Guideline of each stepwise change	Limits and Cautionary Ranges
RR	+/- 2 bpm	RR 10-24 bpm (higher with physician input)
V_T	+/- 1 ml/kg (between 6, 7, 8 ml/kg)	Min 6 ml/kg, Max 8 ml/kg
FiO_2	+/- 0.1 or 10% O2	Max 1.0 (use caution over 0.5)
PEEP	+/- 1-2 cmH20	5-12 cmH_20 (use caution over 10, get physician input over 12)

Let's return once more to the example patient.

Patient | 7.31/57/68/24

Vent settings: RR 16bpm, V_T 420mL, PEEP 5, FiO_2 0.5

Known information: IBW 52 kg. When calculated using the safe V_T range of 6-8 ml/kg equals a safe tidal volume range of 312-416 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 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 bpm and set it at 18 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 SpO_2 is >92%. A practitioner would most likely increase the PEEP to 7 cmH_20. 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 bpm (16 +2 +2 =20 bpm), and you would definitely increase your FiO_2 +0.1+0.1 and consider your 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.

Weaning: Ventilation settings to progress towards extubation

What if your ABG is normal? In the world of mechanical ventilation, a normal ABG is considered weanable! You would consider decreasing your sedation and ventilator settings utilizing the same rules as above.

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—utilizing the initial settings described in Chapter 6. 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.

> More information on the weaning process is provided in the Sault College micro-credential course. Please go to training.saultcollege.ca to get more information or to register.

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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

- RR 20 bpm
- PC 16 cmH_20
- I_T 1.0 sec
- PEEP 10 cmH_20
- FiO_2 0.4.

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.

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, CO_2 = 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. Pressure = 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 cmH_20. Remember that normal is 80-100 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:

- RR 20 bpm
- PC 16 cmH_20
- I_T 1.0 sec
- PEEP 10 cmH20
- FiO_2 0.4

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 (6-8 ml/kg) calculations. You know your safe V_T levels are as follows:

- 6 ml/kg is 420 mls,

- 7 ml/kg is 490 mls
- 8 ml/kg 560 mls

You look on your ventilator screen and see that your tidal volumes are around 550 ml. The patient is within the safe RR range of 10-24 bpm. Both RR and VT 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 8ml/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 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 ml/kg. You want to move down one step to target a tidal volume of 7 mL/kg. Try decreasing pressure control by 2 cmH_20 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 7mL/kg.

PEEP decrease. Most likely, a clinician would decrease the PEEP by 2 cmH_20 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.

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- Lt. Cmdr. Michael Heimes checks on a patient connected to a ventilator at Baton Rouge General Mid City campus © Official U.S. Navy Imagery is licensed under a CC BY (Attribution) license

CONCLUSION

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.

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.

Congratulations on completing the Basics of Mechanical Ventilation online resource!

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CHAPTER 9 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/mechanicalventilators/?p=842#h5p-10

Advance Your Learning

Do you want to dive deeper or showcase what you have learned? Additional materials and support from an experienced instructor is available to learners who enroll in the Mechanical Ventilation micro-credential course at Sault College. Students who successfully complete this online course will earn a micro-credential certification. Please visit training.saultcollege.ca for more information or to register.

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