

# Breathing and Ventilation

The easy guide

for Nurses



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This work has been reviewed, corrected and made suggestions by:

**Ehab Daoud MD, FACP, FCCP, Critical Care Physician Founder and president of the Society and Journal of Mechanical Ventilation**  
Kaneohe, Hawaii, USA.

**Salvatore Sardo MD, Department of Medical Sciences and Public Health,**  
University of Cagliari, Monserrato, Italy.

Some images in the text did not obtain the required authorization; many of them were located in multiple jobs and sites, making the permit application complicated.  
Any problem in this sense can be communicated to the email addresses at the end of the text; I will immediately remove these images and redistribute the work with different images.

## Premise

In the introductory part of my works, I always insert the same words:

*“These pages are information that I have tried to simplify, to these I have added some ideas that could be useful... Don't blindly trust what I have written!”*

These are jobs dedicated to **newly hired colleagues or nursing students**; they are not and should not be considered reference or perfect guides.”

I do all the work myself with the help of books, articles, videos and a lot of curiosity.

I try to create an easy and understandable work and to break the rhythm also fun (I hope); understandable even by those who are completely unfamiliar with certain concepts, this inevitably leads to making the concepts "less true" spoiled by simplifications that are the basis of these works. It remains a "raw" job that has the sole purpose of giving a hand.

When I tried my hand at this job on ventilation I didn't think it could require more than five months of work, other jobs had "taken" me 20 or 30 days at most.

Ventilation and, in particular mechanical ventilation, hides a world of its own and, behind that world, yet another world! One thing is for sure, when the next nursing student says the phrase “can you explain the ventilator to me?” I'll probably headbutt him!!!

I'm joking, I will give him/her this guide, hoping that he will become passionate about these concepts.

PS I still don't understand anything about ventilation.... Poor me!

Once again, my usual advice:

**Be curious and use these pages as a basis for further investigation.”**

Criticisms and advice are welcome, I hope I have done something that will please most people

I wish you a good read,  
Gian Luigi Fadda

## Introduction

The idea behind this work is decidedly different from any conventional text out there on ventilation. First of all, it was born as a work dedicated to the intensive care sector but starting from the basics I tried to understand any tool used in a hospitalized patient. The subject I had in mind when I started the work was the nurse who had just entered the intensive care unit or those "curious" nursing students; in fact, it is to them that this text is dedicated. I had the opportunity to look at an enormous amount of work on ventilation and they all seemed unfit for purpose; some too sectoral with few notions and others too complex, I would define them "from the Big Bang to the Big Crunch". Inside you find everything and more with a level too complex for the purpose I set myself. All excellent works, complete and well done, from which I only took the "gist" of the discussion. I wanted something that was easy to understand and included everything about breathing and ventilation. That it was easy, completely and at a level appropriate to those who have to manage a patient under any form of ventilatory support. I included many concepts and excluded others. I avoided the part concerning the fine setting of the ventilatory modes and the ventilator-patient asynchronies, the first because it is up to the clinician who is responsible for setting the ventilatory therapy and the second because, in my opinion, it can only be understood

after a minimum of knowledge of mechanical ventilation. Find the different ways in which mechanical ventilation is divided, the most important aspects of the most common ventilation modes and the particular ones. From time to time I have included some "tips & tricks", some curiosities and some advice.

This work has the sole purpose of providing a starting point, do not trust what I have written and delve deeper into the topics that interest you with a "healthy distrust" of what I have written! I do all the work myself, apart from a few images that I have "stolen" (I actually asked for permission!). I provide this work completely free of charge in PDF format without any restrictions on distribution. If you need to modify it to adapt it to your needs, contact me and I will provide you with the work in editable Word format.

Enjoy the reading, *Gian Luigi Fadda*

# Acronyms

**Δ -V o P:** Variation in volume or pressure.  
**A/C:** Assisted Controlled Ventilation.  
**AAC:** Adaptive Airway Compensation.  
**ABG:** Arterial Blood Gas.  
**APRV:** Airway Pressure Release Ventilation.  
**APV:** Automatic Positive Pressure Ventilation.  
**ARDS:** Acute Respiratory Distress Syndrome.  
**ASV:** Adaptive Servo Ventilation.  
**ATC:** Automatic Tube Compensation.  
**ATM:** Atmosphere.  
**BAR:** Bar.  
**Bipap:** Bilevel Positive Airway Pressure.  
**C:** Compliance.  
**CaO<sub>2</sub>:** Arterial Oxygen Content.  
**CI:** Inspiratory Capacity, capacità inspiratoria.  
**CLiO<sub>2</sub><sup>™</sup>:** Closed Loop Controller of Inspired Oxygen system.  
**CMV:** Controlled Mechanical Ventilation.  
**CO:** Cardiac Output.  
**CO<sub>2</sub>:** Carbon diOxide.  
**COPD:** Chronic Obstructive Pulmonary Disease.  
**CPAP:** Continuous Positive Airway Pressure.  
**CT:** Computerized Tomography.  
**CSV:** Continuous Spontaneous Ventilation.  
**CVP:** Central Venous Pressure.  
**DUOPAP:** Duo Positive Airway Pressure.  
**E:** Elastance.  
**ECMO:** Extracorporeal Membrane Oxygenation.  
**EPAP:** End-Expiratory Positive Airway Pressure.  
**ERV:** Expiratory Reserve Volume.  
**ETCO<sub>2</sub>:** End-Tidal Carbon Dioxide.  
**ETS:** End Tidal Pressure.  
**ETT:** Endotracheal Tube.  
**FiO<sub>2</sub>:** Fraction of inspired oxygen.  
**FRC:** Functional Residual Capacity.  
**Hb:** Hemoglobin.  
**HEPA:** High-Efficiency Particulate Air.  
**HFO:** High-Frequency Oscillatory Ventilation.  
**HFV:** High Frequency Ventilation.  
**HME:** Heat and Moisture Exchanger.  
**HMEF:** Heat and Moisture Exchanging Filter.  
**I:E:** Inspiratory to Expiratory ratio.  
**ID:** Internal Diameter.  
**IMV:** Intermittent Mandatory Ventilation.  
**INPV:** Intermittent Negative Pressure Ventilation.  
**IPAP:** Inspiratory Positive Airway Pressure.  
**IPPV:** Inspiratory Pressure Support Ventilation.  
**IRV:** Inverse Ratio Ventilation.  
**IRV:** Inspiratory Reserve Volume.  
**MMV:** Mandatory Minute Volume.  
**MV:** Volume Minuto.  
**NAVA:** Neurally Adjusted Ventilation Assist.  
**NIF:** Negative Inspiration Force.  
**NIMV:** Non-Invasive Mechanical Ventilation.  
**NIV:** Non-Invasive Ventilation.  
**O<sub>2</sub>:** Oxygen.



**ORI:** Oxygen Reserve Index, Indice di Riserva di Ossigeno.  
**P/F:** PaO<sub>2</sub>/FiO<sub>2</sub>.  
**P:** Pressure.  
**P0.1:** Airway Occlusion Pressure 0.1 second.  
**PaCO<sub>2</sub>:** Partial Pressure of Carbon Dioxide Arterial Blood.  
**Parapa Pa Pa!!!:Ventilator Allarm Soound!**  
**Patm:** Pressione Atmosferica, Atmospheric Pressure.  
**PAV:** Proportional Assist Ventilation.  
**PAW:** Mean Airway Pressure (MAP).  
**Pc:** Pressure Control.  
**PEEP:** Positive End-Expiratory Pressure.  
**pH:** It is the scale that measures the acidity or alkalinity of a solution  
**Phigh:** Pressure High.  
**PiBinKy: From Sardinian "pibincu" or excessively precise!**  
**PIP:** Peak Inspiratory Pressure.  
**Plow:** Pressure Low.  
**Pplat:** Plateau Pressure.  
**PPS:** Pressure-Regulated Volume Control.  
**PRVC:** Pressure-Regulated Volume Control with Adaptive Support.  
**PS:** Pressure Support.  
**PSV:** Pressure Support Ventilation.  
**PTMB:** Patient Triggered Mandatory Breath.  
**Pvent:** Ventilator Pressure.  
**R:** Resistance.  
**RR:** Respiratory Rate.  
**RSBI:** Rapid Shallow Breathing Index.  
**RV:** Residual Volume.  
**SB:** Spontaneous Breathing.  
**SBT:** Spontaneous Breathing Trial.  
**SIMV:** Synchronized Intermittent Mandatory Ventilation.  
**SpO<sub>2</sub>:** Saturation Peripheral Oxygen.  
**TCT:** Total Cycle Time.  
**Te:** Expiratory Time.  
**Ti:** Inspiratory Time.  
**TLC:** Total Lung Capacity.  
**V/Q:** Ventilation/Perfusion.  
**V:** Volume.  
**VAP:** Ventilator-Associated Pneumonia.  
**VAPS:** Volume-assured pressure support.  
**VC:** Vital Capacity.  
**VC:** Volume control.  
**Ve:** Minute Volume.  
**VG:** Volume Guarantee.  
**VILI:** Ventilator-Induced Lung Injury.  
**Vt:** Volume Tidal.  
**VTMB:** Ventilator Triggered Mandatory Breath.  
**WOB:** Work Of Breathing.  
**ZEEP:** Zero End Expiratory Pressure.

## Part one: A moment to breathe!!!

In nature there are some elements without which it would not have been possible for man to live; Elements like water, food and air are the basic constituents we use to exist on this planet.

In these pages I will focus on the "air" part and will try to explain in my own way what lies behind the very natural and at the same time complex process of breathing. Let's start with.

### What is breathing?

Breathing is a concept known to everyone, especially those who work in intensive care, but here we try to explain complicated concepts to new employees or nursing students; that's why we start from the basics.

Breathing, as we understand it, is an alternation of air entering the lungs and then coming out; this movement of air we call air flow. That's all!

I talked about air to mean a person's breathing in everyday life; I also specified "flow" of air and I will explain shortly what a flow is, also because without it the air just doesn't go anywhere!

This movement of air from the outside to the inside of the lungs is called **INSPIRATION**, its return to the outside is called **EXPIRATION**.

But what do we mean by air?

Taking as an example Uncle Arcibaldo who is working in his garden, we can say with good approximation the composition of his inhaled air.

In fact, air is made up of approximately 78% nitrogen, 21% oxygen (20.9 for those who are precise) and only 0.04% carbon dioxide or CO<sub>2</sub>. There are other gases that we are not interested in.

So when we inhale we bring 21% of oxygen and 0.04% of carbon dioxide or CO<sub>2</sub> into our lungs. Inside our body, oxygen is used as "fuel" for chemical reactions in muscles and tissues and from these chemical reactions it is partly transformed into CO<sub>2</sub>.

In reality, oxygen acts as an oxidizer here too, but we'll see that later!

When we throw out the air we have inhaled it will have a different composition from before and in particular the quantity of oxygen will be decreased and the quantity of carbon dioxide will increase.

When we exhale the air will have these concentrations: oxygen 16-17% and carbon dioxide 4%.

Air entering the lungs	<b>INSPIRATION</b>
Air coming out of the lungs	<b>EXHALATION</b>

	<b>O<sub>2</sub></b>	<b>CO<sub>2</sub></b>	<b>NITROGEN</b>
Inspired air	21%	0.04%	78%
Exhaled air	16-17%	4%	78%

As you can see there are no variations in the percentage of nitrogen between inhaled and exhaled air, for this reason I will not mention it again during this short discussion. Nitrogen is an inert gas and does not participate in chemical reactions in the body; for this reason its percentage remains unchanged. However, know that its role is very important, in fact it will be the one to give way to oxygen if needed.

We now know that air moves from the outside to the inside of our lungs and vice versa; We have given names to these movements of air: inspiration and exhalation. We also give a name to an inspiration followed by an exhalation and this name is **BREATHING ACT**.

A single breath consists of an inhalation and an exhalation.

**1 breath = 1 inhalation + 1 exhalation**

Let's repeat it: Every single respiratory act is composed of two single phases, an inspiratory one, during which the air enters the lungs, and an expiratory one, which will let that air out. We consider air here as a mixture of gases and we will call the natural mixture that we find in the atmosphere "**ambient air**". When we talk about a patient who is "breathing" with a very high concentration of oxygen, we will still talk about air, because even at 99% oxygen he will still be breathing a mixture of gases. We will indicate this data to be more precise by indicating the percentage or fraction of oxygen present in the air mixture that is inhaled ( $FiO_2$ ). You will hardly hear "that patient is connected to the ventilator with air at 50% oxygen", you will hear "that patient is connected to the ventilator with 50% oxygen". This is because the percentage of oxygen will be the only effective value to vary, remember to whose detriment? Good luck with nitrogen! Now let's put everything back in order with a fact that you must not forget:

Saying that a patient is breathing in room air is equivalent to saying that he or she is breathing with 21%  $O_2$  (oxygen).

We can use the acronym AA to mean ambient air with 21% oxygen!

**AA = Ambient Air = 21% oxygen**

This famous 21% is also the **minimum** value in the settings of ventilators and any device for patient breathing or ventilation... Unless?????  
Come on, think about it for a moment, it will serve as a mental exercise for understanding these topics.  
While we're at it, tell me what we do first when we come into the world: do we inhale or exhale air?  
You will find the answers in about ten pages at the end of the guide! Maybe eleven! Twelve at most!

### Let's correct the shot!

What if I told you that everything I write about isn't so correct after all?  
Well yes, that's how it is. However, I had to start with concepts known to all of us; when we speak, we insert the term "breathing" into the common phrasebook, replacing it on several occasions with the term "ventilation".  
But in the end, do we breathe or ventilate? Good question!

If we search online, we find that **BREATHING** is the exchange of gases between the outside air and the tissues and cells of our body.

If we look for **VENTILATION**, we find that it is the supply of fresh air from outside to inside the body.

But we who are black belts in intensive care can also modify these definitions and say that:

**BREATHING** is the exchange of gases between external air, body and vice versa.  
**VENTILATION** is the flow of air from outside to inside the body and vice versa.

Small note: when we hear "fresh" air we don't mean:

“Typical patient breathing on a winter day outside”!

But we mean air rich in oxygen or ambient air and in any case not exhaled air.

So, the correct word I should have used above is ventilation.

We can use the term respiration instead of ventilation almost any time we want. One of the books that made me passionate about ventilation is "Artificial **respiration** basics and practice" by Larsen; take a look!

You must know the meaning of the two terms because, when I continue with the explanations, talking about ventilation will have a very different meaning from its "almost synonymous" breathing.

We are more inclined to talk about breathing because it is the thing we have known best since we were little. When we set foot in an intensive care unit, however, it comes naturally to us to talk about ventilation when a patient is connected to the "ventilator". Know that we can also call that machine a respirator and no one will tell us that we are wrong! A little poetic license.

In this discussion I will use breathing or ventilation without distinction without having too many problems, you will understand for yourself when the meaning needs to be more precise.

Now we know that the flow of air is called ventilation and what follows this supply of air generates chemical exchanges that are called respiration. You will have noticed that the term flow has appeared again and that I have not yet given you a definition; well, I won't do it again! Be patient.

## But why do we breathe?

The answer is as obvious as it is complicated to explain in detail; settle for a summary. We breathe because we need energy to live. To produce this energy, we use a mechanism called metabolism.

To function well, the metabolism needs a fuel and an oxidizer which is oxygen and emits waste products such as carbon dioxide. Fuels are glucose, fatty acids and amino acids.

This, of course, concerns the “respiratory” part of the metabolism process.

We can divide what happens in ventilation into four main parts:

1: Oxygen is introduced into the lungs and carbon dioxide is eliminated.

2: In the alveoli, oxygen diffuses towards the capillaries and from the capillaries, carbon dioxide diffuses into the alveoli.

3: The blood circulation carries both of these gases.

4: At the tissue level, oxygen and carbon dioxide will pass from the bloodstream to the cells and vice versa.

At points 2 and 4, the transition occurs due to the difference in gas concentrations.

Thus, the passage of oxygen from the alveolus to the capillaries is due to a different concentration of the gas between the two districts. Thus, the oxygen will pass from the point of greatest concentration, the "mister" alveolus, to the point of lower concentration, the "lord capillaries". Carbon dioxide will do the same by passing from the capillaries which contain more of it to the alveoli which contain very little.

Can you guess what happens at the tissue level? The oxygen, present in the arterial capillaries in large quantities, will go to the tissues that have a lower percentage of it and the carbon dioxide, produced by the metabolism in large quantities, will pass from the tissues to the capillaries which will take it to the lungs, in a continuous cycle.

At each of these points, the  $O_2$  and  $CO_2$  values have normal or standard values which must remain so for physiological homeostasis. When we talk about respiratory failure, we mean that we find ourselves in a condition in which we are unable to keep these  $O_2$  and  $CO_2$  values normal and stable.

## A pinch of anatomy

Just a hint about the anatomy of the respiratory system, naturally only the parts that can really interest us. We divide the airways into three zones.

Upper airway, conduction zone and respiratory zone

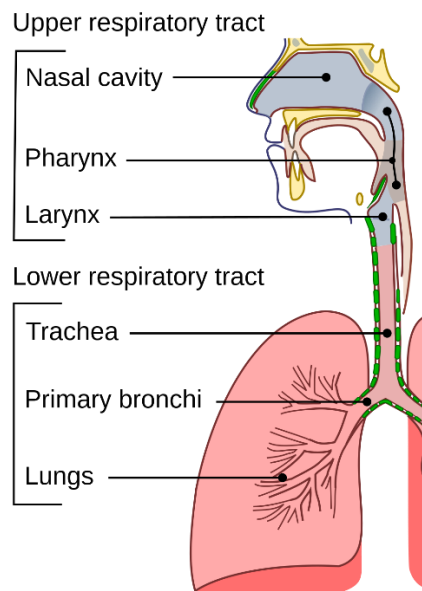
The **upper airways** are: oral cavity, nasal cavity and pharynx.

The **conduction zone** is composed of: larynx, trachea, bronchi and bronchioles.

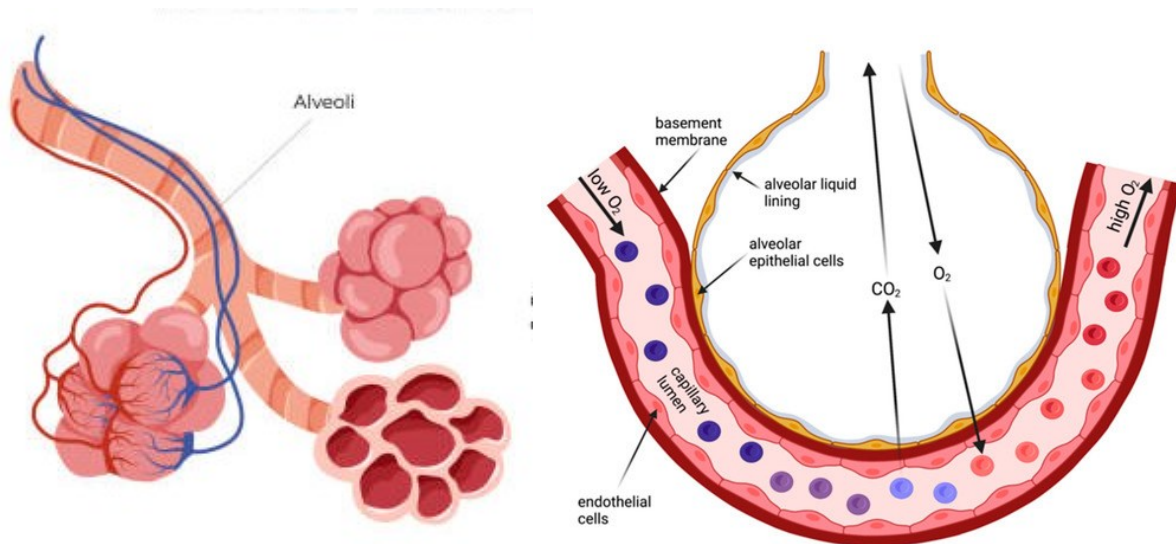
The **respiratory zone** is composed of: alveolar sacs and alveoli.

No gas exchange occurs in the upper airways and conduction zone (dead space)

Only in the respiratory zone does gas exchange occur.



Please note: In the part of the bronchial tree with large sections, the flow is turbulent. As we get closer to the alveoli, the number of branches increases, therefore the total cross-sectional area increases. This leads to a slowdown in flow and a transition to laminar flow. The slowdown allows the gases to have more time to exchange. Would you get into a moving subway?!



In the images above, the conformation of the alveolar sacs and how the exchanges between the alveolus and capillaries take place. Naturally I have reduced the information to the bare bones. Look for more complete information on the anatomy and physiology of the respiratory system, it is much more complex than that.

## Normal ventilation

In normal breathing, the contraction of the respiratory muscles, primarily the diaphragm, causes the rib cage to expand, causing the pressure of the alveoli to become negative compared to the external pressure. Therefore, with this pressure difference between the alveolar  $<$  Patmosphere, a flow of air from the external environment inside the alveoli is generated.

During exhalation, the elastic force of the respiratory system, including the rib cage and lungs, returns the system to its original position, generating a condition in which the Palveolar  $>$  Patmosphere and generating a flow from the lungs towards the outside.

Exhalation is passive and does not require energy consumption.

Normal ventilation:

Inspiration	Exhalation
Palveolar $<$ Patmosphere	Palveolar $>$ Patmosphere

Once the air has been exhaled, we are in a condition of equilibrium of the pressures which are now identical; not only is the respiratory system now in the condition called **Functional Respiratory Capacity**.

The kinetics of gases takes on a sinusoidal shape in this type of breathing.

The inhalation phase is active and requires energy and work to be accomplished.

Exhalation requires no energy or work.

The work of breathing is called **WOB** or Work Of Breathing. Physiologically, WOB is normal, but in particular pathological conditions it can become very intense for the patient, to the point where he is unable to support normal breathing with his own strength.

This fatigue develops or worsens a condition of respiratory failure; this condition can be resolved depending on the severity with small aids, such as the administration of oxygen alone, up to much more substantial aids, such as mechanical ventilation.

## Mechanical ventilation

Mechanical ventilation can be achieved in three different ways.

- **PPV:** Positive Pressure Ventilation whether invasive or non-invasive.
- **NPV:** Negative Pressure Ventilation via negative pressure (iron lung).
- **HFOV:** High Frequency Oscillatory Ventilation.

In our discussion I will talk exclusively about PPV or Positive Pressure Ventilation systems.

### PPV = Positive Pressure Ventilation

In mechanical ventilation, the ventilator generates enough pressure to overcome the resistance of the airways and create a pressure difference between the machine and the alveoli. The greater pressure on the machine side generates a flow of gas that moves a certain volume ( $V_t$  or Tidal Volume\*) inside the lungs. Once inhalation has ended and the desired volume has been reached, we find ourselves in a condition in which the Palveolar  $>$  Pventilator and, simply, the ventilator opens the expiratory valve. Also in this case, the outgoing flow is generated by the elastic force of the respiratory system, so **also in this case exhalation is a passive phenomenon**.

Write it carefully in your notes:

**exhalation with mechanical ventilation is usually passive**

But sorry, Gi, isn't it always passive even in normal breathing?

No! Not always, because you can force the exhalation by making it active!

\* $V_t$  = tidal volume = tidal volume I will shortly explain what this means.

Now, in true Gi style, I also tell you that there are actually two "breaths".

NO, I can't help but mention them, but I promise no scheme or chemical formula will be sacrificed for this moment!

We usually divide breathing into two different parts depending on where the gas exchanges take place. We have external breathing and internal breathing.

## External and internal breathing

**External respiration** is the exchange of gases that occurs at the alveolar level between the air present in the alveoli and the blood capillaries.

**Internal respiration** occurs in mitochondria, which are cellular organelles.

Mitochondria use oxygen to oxidize nutrients, such as glucose, producing energy in the form of ATP. Carbon dioxide is a waste product of combustion and is released into the environment.

In external respiration, the oxygen present in the inhaled air passes from the alveoli to the bloodstream and, at the same time, the carbon dioxide to be eliminated passes from the bloodstream to the alveolar air, which will now be exhaled and eliminated.

At the tissue level, oxygen will pass from the capillaries to the tissues, and CO<sub>2</sub> will pass from the tissues to the capillaries to be transported to the lungs and eliminated through external respiration.

If you want more details, read the details in my little job! on blood gas analysis.

Remember: Oxygen = O<sub>2</sub>, carbon dioxide CO<sub>2</sub>

Internal respiration = Exchange of gases at the tissue level

External respiration = Exchange of gases at the alveolar level

Internal breathing	Fabrics
External breathing	Alveoli

## The inevitable boring part. Pressures and volumes

In the ventilatory process, there are pressures and volumes at play that we need to know.

### The complete respiratory cycle

The respiratory act is actually composed of 3 phases. Of these, we have already seen inhalation and exhalation, but there is a short pause between one and the other; this pause is also important. Let's make a small reference to respiratory physiology, just to understand the dynamics of its functioning.

Between the lung and the chest wall there is a virtual space called the pleura; this is composed of two parts, one visceral and one parietal. Between them there is a liquid which carries out the task of lubricating these two surfaces, thus allowing them to slide and therefore allowing us to breathe. Within this space, which we will call **PLEURAL SPACE**, there is a certain pressure for each moment of breathing, we will call this **INTRAPLEURAL PRESSURE** or **PLEURAL PRESSURE**.

The alveoli also have a pressure called **ALVEOLAR PRESSURE**.

The alveolar pressure minus the intrapleural pressure gives us a third pressure called **TRANSPULMONARY PRESSURE**.

$$(\text{Alveolar pressure} - \text{Pleural pressure}) = \text{Transpulmonary pressure}$$

The measurement of these pressures is measured in centimeters of water (cmH<sub>2</sub>O) because it is a linear scale that is easy to read and interpret. Atmospheric pressure is 760 mmHg, which is equal to 1033.23 cmH<sub>2</sub>O. But by convention, we consider atmospheric pressure equal to 0 cmH<sub>2</sub>O, we do this to relate it to the other pressures measured. In practice, that value of 0 that we give it is because we consider it a reference value.



## Pressures are measured in cmH<sub>2</sub>O

### The atmospheric pressure is 0 cmH<sub>2</sub>O

Air can only move between points with different pressures. Put more simply, for there to be a movement of a certain volume of air, there must be a different pressure between two districts. This movement of air always occurs from the district with higher pressure towards the one with lower pressure.

This movement of air is called **FLOW**.

By flow we mean the movement of a certain volume of air in a certain time.

### Pressure, Flow and Volume

But here are three very useful definitions for understanding respiratory mechanics and which we will find again in mechanical ventilation.

**Volume:** quantity of air measured in ml or liters.

**Flow:** speed of movement of a volume of air measured in ml or liters per second

**Pressure:** it is the force exerted by a certain volume of air and measured in cmH<sub>2</sub>O

Different measurement methods are used for pressures, but for the respiratory system the cmH<sub>2</sub>O method remained, inherited from those who carried out the first studies and who, for practicality, used water column manometers.

Air enters the lungs because they have a lower pressure than the atmosphere. If our beautiful lung is connected to a ventilator we take as reference the pressures not of the atmosphere but of the ventilator; in this way inspiration will occur because the pressures of the ventilator will be greater than those present in the lung.

The air flow is therefore nothing more than a movement of air and to generate this flow a pressure difference between two compartments is needed; you can also find this pressure difference referred to as **PRESSURE GRADIENT**.

Therefore, an air flow is generated when a pressure gradient exists between two districts, this flow has a direction that goes from the area with higher pressure towards the one with lower pressure. This is one of those pieces of information that we must consider certain because it is described by the physical laws of gases.

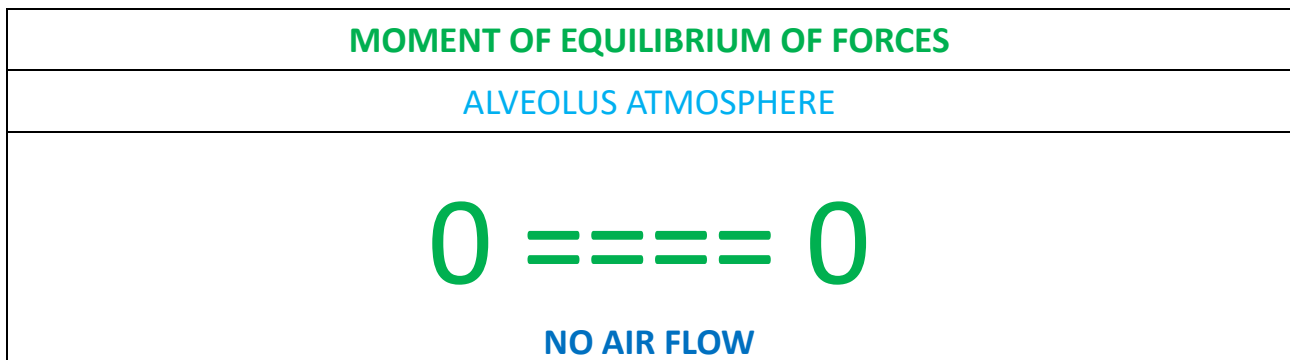
<b>Pressure difference = Pressure gradient</b>
--

The direction of an air flow goes from the compartment with higher pressure towards the one with lower pressure.

## Dynamics of the flows and pressures involved

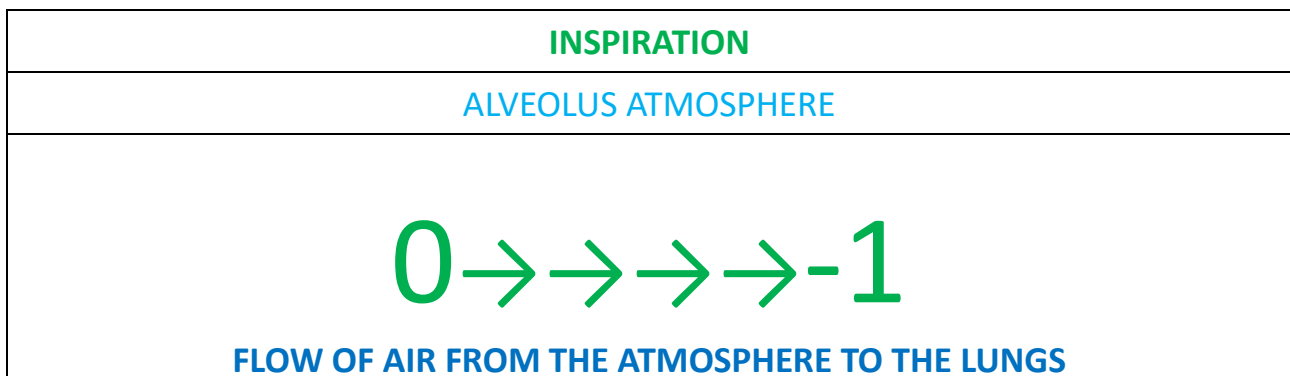
Another interesting concept to know about respiratory dynamics is that the thoracic part has the tendency to want to "pull" outwards while the lungs try to collapse on themselves, these two forces tend to balance in the moment of respiratory pause. At this precise moment the intrapleural pressure will be  $-5 \text{ cmH}_2\text{O}$  and the pressure inside the alveoli will be  $0 \text{ cmH}_2\text{O}$ , a perfect balance of the previous forces.

Since the alveolar pressure is 0 like atmospheric pressure there will be no pressure difference and there will be no air flow, easy right?



Now, watch out! Let's see what happens when the diaphragm and the rib cage decide to become part of this ventilation "game". The diaphragm contracts as it lowers and the rib cage expands. This brings the pressure inside the alveoli to  $-1 \text{ cmH}_2\text{O}$ , generating a difference with atmospheric 0 and allowing a flow of air towards the lungs.

We call this flow **inhalation**.



The fact that the rib cage expands outwards causes the intrapleural pressure to drop to a value of  $-7.5 \text{ cmH}_2\text{O}$ .

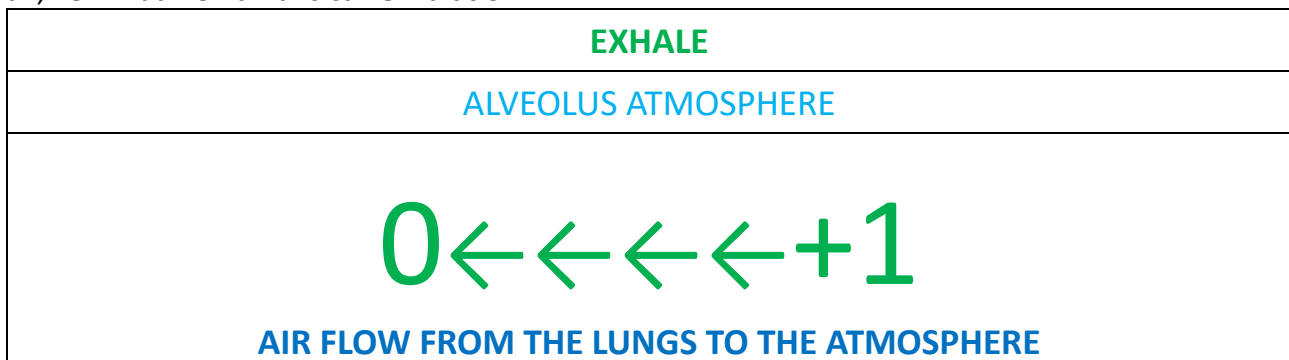
In a normal person weighing around 70kg, the flow displaces a volume of air of around 500ml. Do you know what this volume is called?  $V_t$ , or tidal volume.

Now comes the best part: When inhalation ends and those 500ml of air are inside our lungs, the lung-atmosphere system returns to equilibrium with an alveolar pressure of  $0 \text{ cmH}_2\text{O}$ . And here you will say that you have watched all the National Geographic documentaries: how the hell does the air come out if you have just said that without a pressure gradient there is no flow and therefore no movement?

And here I wanted you! The value 0 alveoli and 0 atmosphere remain only for a moment, when the elastic forces will try to bring the rib cage back to a state of rest. In doing this, they will apply force to the lungs, bringing the pleural pressure back to -5 cmH<sub>2</sub>O at the end of exhalation. But more importantly...

Be careful here, because if you miss this step, you will miss the ending!

...most importantly, the force exerted by the chest wall will bring the pressure inside the alveoli to +1 CmH<sub>2</sub>O, reversing the pressure gradient between the inside of the respiratory system and its outside. We now have +1 in the lungs and 0 in the atmosphere, thus generating an outward flow of air, i.e. what we humans call **exhalation**.



Remember that we also talked about transpulmonary pressure?

This is given by the difference between the alveolar and pleural pressure, and throughout the respiratory cycle it remains positive, +5 in the pause, +7.5 in inspiration and +5 in exhalation. If this pressure were to become negative it would indicate a collapsed lung, just so you know.

Now we know how respiratory mechanics works in broad terms and we have laid a minimum foundation to continue.

Up to this point we have talked almost exclusively about pressures and flow but remember that flow moves a certain volume of air. Now we must also talk about the volumes involved.

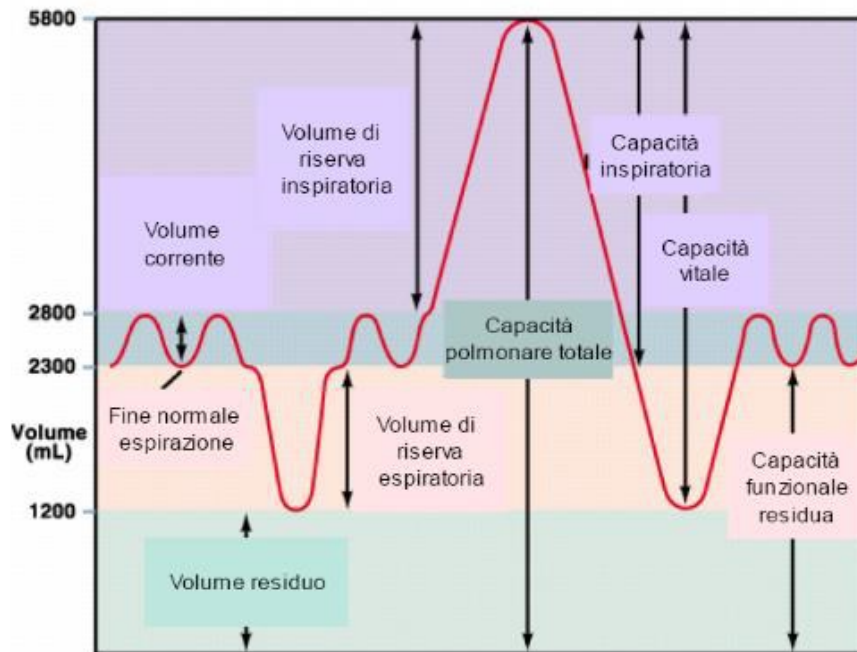
Fasten your seatbelts! Ready Set Go!

### These blessed Volumes and Capacities!

First a quick premise, by volumes we mean a quantity of space in ml or liters, the concept of capacity refers instead to two or more of these volumes added together.

<b>Volume is the space occupied by a certain amount of air</b>
<b>Capacity is the sum of two different volumes</b>

I am inserting an image here that will be used for the definitions that follow, I will try to talk to you only about the most important things just to understand what happens in the mechanics of human breathing.



If we want to be concise, we consider four main volumes:

- **Tidal volume (VT) 500mL**
  - Volume of air moved in a normal inhalation or exhalation
- **Inspiratory Reserve Volume (IRV) 3000mL**
  - Volume of a forced inspiration above Vc
- **Expiratory Reserve Volume (ERV) 1100mL**
  - Forced exhalation volume above normal exhalation
- **Residual Volume (VR) 1200mL**
  - Volume of air present after forced exhalation

Here are the four capacities, which are added volumes. Are these:

- **Functional Residual Capacity (FRC) 2300mL**
  - Volume given by ERV + RV
- **Inspiratory Capacity (IC) 3500mL**
  - Volume given by IRV + VT
- **Vital Capacity (CV) 4500mL**
  - Volume given by VT + IRV + ERV
- **Total Lung Capacity (TLC) 5800mL**
  - Volume given by VC + RV

A normal breath moves 500ml of air. A forced inspiration moves a larger volume which over 500ml is defined as VRI; a forced exhalation over 500ml is called ERV.

These are the volumes present in the respiratory system. In addition to volumes, there are lung capacities which are given by multiple volumes added together.

Thus, the inspiratory capacity is given by IRV+VT and has a value of 3500ml given precisely by a deep inhalation.

The vital capacity (VC) is given by a deep inhalation followed by a deep exhalation and reaches the considerable volume of 4500ml.

The ultimate capacity is given by the expiratory reserves and the reserve volume reaches 2300ml. This capacity is called FRC or functional respiratory capacity; this is all the volume of air that remains in the lungs during normal breathing without forcing exhalation.

Lastly, the total lung capacity (TLC) which is the sum of the 4 volumes indicated reaches 5800ml. In women the values are lower by between 20 and 30%. You should know that in spirometry the reserve volume cannot be measured. Find out how you can measure it, curious people! Therefore, functional residual capacity is the amount of air that remains in the bronchi, bronchioles and alveoli and is not eliminated with normal exhalation.

When we inhale or exhale normally, we generate a flow of air that enters and exits our lungs. This flow of air generates a certain volume which we will call Tidal Volume. The tidal volume is indicated as Tidal Volume (VT) or  $V_t$  and corresponds to a volume equal to 500ml of air. Each inspiration in normal conditions introduces 500ml of air into the lungs. These 500ml of air will exit the lungs with the next exhalation and so on, the cycle continues almost endlessly!

However, our lungs are not completely empty and there is a certain amount of air that remains inside them even when we have sent out those 500ml. This amount of air is called FRC, which stands for functional residual capacity.

FRC is the volume of air present within the lungs after a **normal** exhalation.

We, who are curious children, know that we don't always breathe in a "normal" way and, if we want, we can force both the inhaled and exhaled volumes, making that "normal" value of 500ml change a lot. Thus, if we decide to force exhalation, we can still throw out a lot of air present in that FRC volume, managing to throw out the expiratory reserve volume. Now comes the interesting part: FRC value is very important to us. The larger it is, the more margin we have in case of need. The lower it is, the closer we are to the limit of our respiratory system. The FRC is also important because it establishes the balance between the two thoracic and pulmonary forces, a balance that is lost in the case of pneumothorax when the lung collapses on itself.

If we force exhalation and throw out the expiratory reserve volume, we reach a point beyond which we can no longer let out air. At that moment, however, a certain amount of air remains inside our lungs, called residual volume (RV).

What we have done with exhalation, forcing it, we can also do with inspiration, going well beyond that value of 500 ml of a normal inhalation. All the volume that we can fit beyond those 500ml is called inspiratory reserve volume.

We can force breathing acts voluntarily or involuntarily whenever it is necessary to give more "fuel" to our little body!

*...The ventilator is a tool that does all this and even more. IPPV, CPAP and PEEP are just noises that intubated patients make!!!...*

We have seen how respiratory mechanics works and partly how gas exchanges occur in breathing. Let's remember that we have only seen the parts that interest us up to this moment, we will talk in more depth about some concepts as the discussion becomes "full-bodied".

We know what is meant by respiratory act and what is meant by Tidal Volume. Now let's get to know one of the most important volumes in this field and an equally important characteristic of ventilation.

## The respiratory rate

If we count all the complete breaths we perform in a minute, we obtain a value called Respiratory Rate, abbreviated as RR.

<b>RESPIRATORY RATE = RESPIRATORY ACTIONS PERFORMED IN ONE MINUTE</b>
---

Respiratory rate indicates the number of breaths performed in one minute. Once we know this data, we can also calculate how much air our breathing moved in a minute.

## The minute volume

This data is called **Minute Volume** or **Vm** or **Ve** and is a very important parameter in the world of ventilation, particularly in mechanical ventilation.

<b>MINUTE VOLUME = TIDAL VOLUME x RESPIRATORY RATE</b>
--

<b>VM =VT x RR</b>
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The minute volume is calculated by multiplying the tidal volume by the respiratory rate. So if a patient has a respiratory rate of 20 breaths per minute and a tidal volume of 500ml we would have a minute volume equal to:

$20 \times 500\text{ml} = 10000\text{ml}$  or 10 liters

The minute volume is indicated in liters/minute so the patient above will have a minute volume of 10L/minute.

## Frequency variations and various definitions

A healthy person at rest has a frequency that varies between 12 and 20 acts per minute. When these acts are fewer in number, we speak of **bradypnea**, when these are in greater number we speak of **tachypnea**.

Both bradypnea and tachypnea can be physiological and ensure adequate cellular oxygenation. I'll try to explain myself better.

During an afternoon nap, a healthy person lowers his breaths per minute and therefore his respiratory rate to 5-6 breaths/minute; this happens because at that moment the body's need for oxygen is reduced.

On the other hand, a person who goes jogging... jogging jogging uffff... who goes jogging! He has a higher oxygen requirement, so his breathing rate increases, allowing more air to participate in the exchanges. In both cases it is a physiological mechanism. An athlete can even reach 40 breaths/minute, to compensate for an effort. It's a different story for a bedridden patient who has such a high respiratory rate at rest, in this case the compensation mechanism that increased the respiratory rate has something pathological behind it.

The variation in respiratory rate also depends on age. A newborn can easily have a normal range between 30 and 60 breaths per minute and a toddler between 21 and 30. In normal adults we have these conditions:

**Eupnea:** normal respiratory rate 12-20 breaths per minute

**Bradypnea:** respiratory rate less than 12 breaths per minute

**Tachypnoea:** respiratory rate greater than 20 breaths per minute

There are also other terms to indicate changes in breathing rates, I am inserting them just for the record, I don't think you will ever hear them elsewhere:

**Polypnea:** increase in the frequency and volume of each individual breath.

**Hyperpnea:** increased frequency and/or volume of breathing due to increased metabolism, for example during physical exercise, is a form of polypnea.

**Hyperventilation:** increased respiratory rate and/or volume without increased metabolism, for example due to emotional causes or while inflating a balloon.

**Hypoventilation:** reduction of alveolar ventilation in shallow breathing, asthma or restrictive lung disease.

**Dyspnea:** difficult breathing, caused by pathologies or intense physical activity.

**Apnea:** interruption of breathing, voluntary or due to pathologies

In these first pages we have seen what is meant by breathing, we have explored some concepts that will also be useful to us later and in doing so we have almost always considered a healthy subject who breathes normally without any type of problem. We have seen concepts such as tidal volume, minute volume and respiratory rate. We have only scratched the surface of what is a huge topic, but without this foundation we could not continue. Some American respiratory therapists say, without any doubts or buts, that if you don't know the relationship between  $V_t$ , RR and MV you cannot talk about mechanical ventilation.

Now let's continue the discussion and raise the bar a little by defining complex concepts that will help us in the future. One of these "dark characters" is the concept of **compliance**. To make it easy I looked at more than 30 works, looked up definitions, watched videos and finally decided to put it all through my complicated concept grinder and got an easy definition, easy to understand. Let's go!

## Concepts of compliance, elastance and resistance

Before starting with the detailed definitions, you need to sit through a "philosophical" discussion regarding compliance and elastance.

In the medical field, the term "elastance" is increasingly used as a replacement for "compliance". The two terms are similar in that they refer to a material's ability to deform under the application of a force. However, there are some key differences between the two.

**Elastance** is defined as the ratio of the applied force to the resulting deformation.

**Compliance**, on the other hand, is defined as the ratio between the deformation and the applied force.

Elastance and compliance have an inverse relationship where:

$$E = 1/C \text{ and } C = 1/E$$

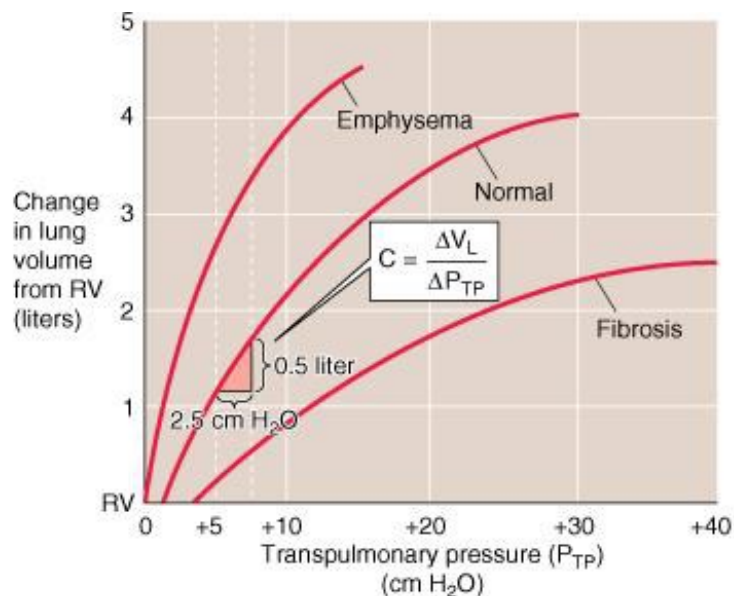
## Compliance

In this section I will use the stick first and then the carrot.

Compliance is given by the ratio between the change in volume divided by the change in pressure. This is written like this:

$$\text{COMPLIANCE} = \Delta V / \Delta P$$

You don't understand an alveolus, do you? Let's put up an image and then move on to the carrot!



In physics use the symbol  $\Delta$  indicates a variation and is pronounced DELTA; therefore, compliance is nothing more than the result of the change in volume divided by the change in pressure.

Still dark? Ok, fine, I'll save you.

Compliance is the ability of the lungs to expand, and that relationship between variation in volume and pressure tells us how easy it is to expand the lung.

So, if I apply a little pressure and get a normal change in volume, that lung will have normal compliance. If I have to apply very high pressure to achieve small lung expansion or volume, I will have low lung compliance.

In the image, it is indicated by the lower curve with a fibrotic lung, therefore hard and difficult to expand. Very high pressures will be needed to ventilate this type of lung.

The more vertical curve indicates an emphysematous lung. In this case, with low pressure we will obtain a large expansion and therefore a large lung volume.

If you look carefully at the image, in the abscissa or x line, in short, the horizontal line, it indicates the pressure and in particular what we take into consideration is the transpulmonary pressure.

That's why I included it in the "important" pressures!

On the ordinate or Y axis we have the volume indicated in mL or liters.



Compliance is a unique characteristic of the pulmonary system for each individual and for this reason it is the **variable component** of the respiratory system of each of us.

<b>NORMAL COMPLIANCE</b>	<b>NORMAL LUNG</b>
<b>LOW COMPLIANCE</b>	<b>LUNG DIFFICULT TO VENTILATE</b>
<b>HIGH COMPLIANCE</b>	<b>LUNG EASY TO VENTILATE</b>

**COMPLIANCE IS UNIQUE FOR EACH INDIVIDUAL**

But not only is it unique to each of us, it can also vary over time!

Let's go back to the concept. I know you are shocked by the simplicity of this definition. After a review, I will add some compliance information that you can't ignore.

The lung is a balloon that lives inside our chest. In everyday life, breathing normally, I can make it inflate without problems. This is because I have normal compliance. I must use an inhalation with normal pressure for this balloon to reach its correct tidal volume of 500ml; then **it reaches its normal volume by applying normal pressure.**

My uncle, who worked in the mine, has such a fibrotic lung that if you Google "fibrosis" his name comes up. He doesn't even reach that famous 500ml volume and to do so he has to make an enormous effort. He therefore applies a frightening pressure to have an almost normal tidal volume!

**His lungs are fibrotic and therefore have very low compliance.**

The wife, my aunt, smoked so much that she has dual nationality, Italian and Turkish. She has developed emphysema so severe that with only one nostril, when she sleeps, she develops 3000 tidal!

**My aunt's emphysema gives her very high lung compliance.**

I'll try to explain myself even better. Do you know at your nephew's birthday when you arrive 5 minutes early and they make you inflate 2000 balloons?

The normal ones to inflate have normal compliance. The easy ones that even children inflate have high compliance, little pressure and large volume!

Then there are the thin and long ones that are very difficult to inflate. Those are the balloons with low compliance, for them it takes enormous pressure and effort to be able to inflate them.

They are those balloons that when you are finished you have to put one eye back into the socket due to the force you used!

We can add that compliance measures the distensibility of an elastic structure starting from its original position.

Compliance is therefore the change in volume for each centimeter of H<sub>2</sub>O applied as pressure. If you have a compliance of 40mL/cmH<sub>2</sub>O it means that for every centimeter of water applied under pressure that volume will vary by 40ml. Easy, right?

Below I insert a table that shows us which tidal, in the colored columns, develops a lung based on its compliance.

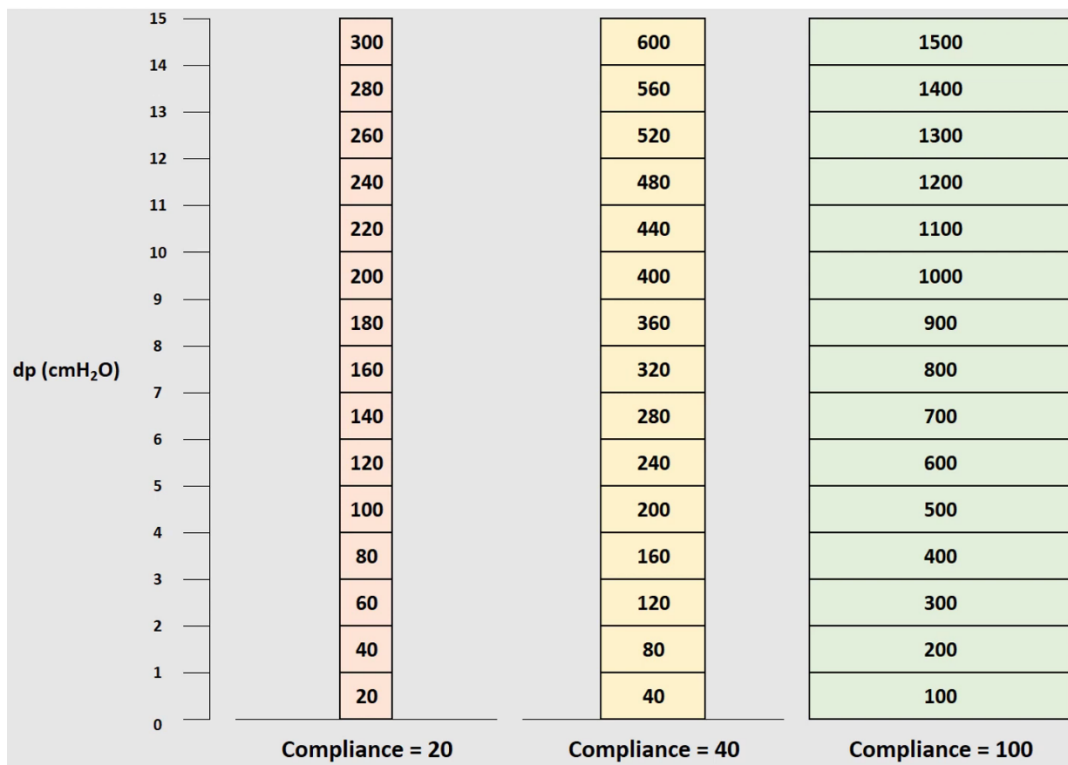
The force used, or rather the pressure, is indicated in cmH<sub>2</sub>O. Next to it is the **DP** which indicates the driving pressure.

I will introduce you to driving pressure in detail when we talk about mechanical ventilation. For now, I'll just tell you that she is a "Cinderella" who has had little luck in the past. Lately, driving pressure has been given ever greater importance. Thanks to its correct management it is possible to improve the outcome of patients who have been on mechanical ventilation.

As you say, what does outcome mean? But do I have to tell you everything?

By outcome in medicine, we mean the outcome of a therapy. In this specific case we mean the increase in survival in a patient who has been on mechanical ventilation.

To put it down to earth we can translate it like this: come=come - out=out, come out.... Live hopefully!



## COMPLIANCE

It is the measure of **distensibility** of the respiratory system

**V = litres**

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**P = cmH<sub>2</sub>O**

I'm not done with compliance! I want to give you a small list of values so you can get an even more precise idea.

If you search online, you will find values indicated in different ways depending on whether liters or milliliters are used in the equation. The normal compliance value of a healthy lung is 200mL/cmH<sub>2</sub>O. Values above 200 indicate increased (high) compliance and below 200 indicate reduced (low) compliance.

Compliance	Value
LOW	<200 mL/cmH <sub>2</sub> O
NORMAL	200 mL/cmH <sub>2</sub> O
HIGH	>200 mL/cmH <sub>2</sub> O

Two other concepts to know are those of static and dynamic compliance.

**Static compliance:** Static compliance is measured when there is no airflow, so it is an indication of the general flexibility of the lungs and their structures. In simpler terms, you can think of static compliance as the "softness" of the lungs when there is no air movement.

**Dynamic compliance:** This term refers to how easily the lungs expand during incoming or outgoing airflow. Since there is air movement involved, dynamic compliance considers the dynamic aspect of the breathing process. Simply put, dynamic compliance takes into account how easy it is for the lungs to expand as air moves in or out.

## The elastance

The inverse of compliance is elastance, which measures the ability of an elastic body to return to its original position once the force that caused its distension has ended.

Thus, a patient who has emphysema will have high compliance and low elastance; on the other hand, a patient with ARDS will have low compliance and high elastance.

Elastance represents the increase in pressure for a 1 liter increase in volume.

## ELASTANCE

It is the measure of **rigidity** of the respiratory system

$$P = \text{cmH}_2\text{O}$$

-----

$$V = \text{litres}$$

# ELASTANZA

E' la misura della rigidità del sistema respiratorio

$$\frac{P_{EL}}{V} = \frac{\text{cmH}_2\text{O}}{\text{litri}} = \text{cmH}_2\text{O} \cdot \text{litri}^{-1}$$

$$\text{cmH}_2\text{O} \cdot \text{l}^{-1}$$

**Elastance is the ability of the lung to return to its resting volume after it is stretched**

In emphysema, elastance decreases and a conscious effort is needed to exhale

But what is elasticity?

We have already said it, and if we apply the concept to the respiratory system, it is that characteristic that allows the lungs to return to their starting size once the air inside them is eliminated. The elasticity of the pulmonary system is given by elastic fibers present in the parenchyma, and without them it would not be possible to breathe optimally. In fact, if we consider a patient with emphysema, we discover that he has a breakdown of these elastic fibers. This allows the lung to expand very easily but makes it difficult to eliminate the inspired air due to the lack of elasticity of the entire system.

On the other hand, if we are faced with a patient with pulmonary fibrosis, the elastic system is stiffened by "healing" of the lung tissue. This results in an increase in lung stiffness, making this lung less compliant and therefore very difficult to ventilate.

When we talk about lung elasticity, we must point out that it is the result of two characteristics of the lung itself. These two characteristics, or components, are one parenchymal and one alveolar, both of which contribute to total elasticity.

The parenchymal component is given by the physical characteristics of the lung tissue, composed of elastin fibers which are distensible and less distensible collagen.

The alveolar component is given by the surface tension of the part responsible for exchanges, composed of an interface between liquid and air.

Very briefly, the parenchymal component behaves no more and no less like the elastic described above. The mechanism of the alveolar component is different: in fact, in it the liquid contained inside it, due to its surface tension, will try to take up as little space as possible, due to its physical characteristic which tends to make it become a sphere.

Do you know the water droplets that appear in the absence of gravity? Well, those droplets are the liquid's attempt to occupy as little space as possible, precisely by exploiting surface tension, that is, the strongest bond given by its molecules on the surface. It is precisely this surface tension that will cause the alveolus to collapse, and the alveolus will tend to "take with it" its container, the lung!

The presence of surfactant in this context blocks the action of surface tension, reducing elasticity and increasing compliance.

Compliance or capacitance is directly proportional to elasticity.

I try to give a simple definition of these three components:

Compliance: How easily a lung can be distended

Elasticity: The tendency of a lung to return to its original shape once it has been stretched

Elastance: the inverse of compliance

## The resistance

Resistance is nothing other than the pressure necessary for flow to occur in the airways and can be defined as the obstacle opposed by the airways to the flow of air.

The resistance is given by the change in pressure divided by the change in flow:

$$R = \Delta P / \Delta V$$

We can define airway resistance as the measure of the impedance or difficulty to the flow of air and is given by the difference between alveolar and atmospheric or external pressure.

Resistance increases in the presence of secretions, bronchospasm, foreign body, tumor, condensation in the ventilator circuit, endotracheal tube and many other factors.

Airway resistance is inversely proportional to airway diameter.

This means that if the section of the airway decreases, its resistance to the flow of air increases and vice versa.

The flow is given by the change in pressure divided by the resistance.

When the pressure increases the flow always increases if the resistance remains stable.

Therefore, **flow and pressure are directly proportional.**

If resistance increases, flow decreases. If resistance decreases, flow increases.

So, **the flow is indirectly proportional to the resistance.**

Remember that gases diffuse from an area of high pressure to an area of low pressure.

The first parts of the respiratory tree have a certain section, the further we go the larger this section becomes, effectively reducing the speed of the flow. Up to the respiratory bronchioles and alveoli, where the flow is practically stopped. This slowdown allows more time for respiratory exchanges.

Remember that in ventilation, if you increase the flow, you will also increase the resistance due to various turbulences. The size of the tube is important: the bigger the tube, the lower the resistance. The length of the duct is also important: the more the length increases, the more the resistance increases. Thus, in a patient with an 8 tracheal cannula or 8 tube, we have greater resistance with the tube, because the greater length corresponds to greater resistance.

## Dead space

Dead space is the part of the airways that does not participate in gas exchange.

There are three types of dead space: **anatomical, alveolar and physiological.**

The **anatomical dead space** is that part of the conduction pathways that does not participate in gas exchange.

If we consider a normal tidal volume ( $V_t$ ) of 500 ml, the real amount that reaches the alveoli for alveolar ventilation is only 350 ml. The other 150 ml is the amount of air that remains inside the airways in spaces that cannot participate in gas exchange. Thus, from the nose or mouth to the alveoli, we have a volume of air that cannot be exchanged: this volume is the dead space.

In particular, it is called anatomical dead space.

Now, considering a respiratory rate of 12 breaths per minute and multiplying it by the  $V_t$ , we obtain a minute volume ( $V_m$ ) of 6 liters/minute. But now we know that not all of those 6 liters participate in gas exchange. To find the exact quantity of gas participating in gas exchange, we must modify the multiplication in this way:

$$350\text{ml} \times 12 = 4200\text{ml/minute.}$$

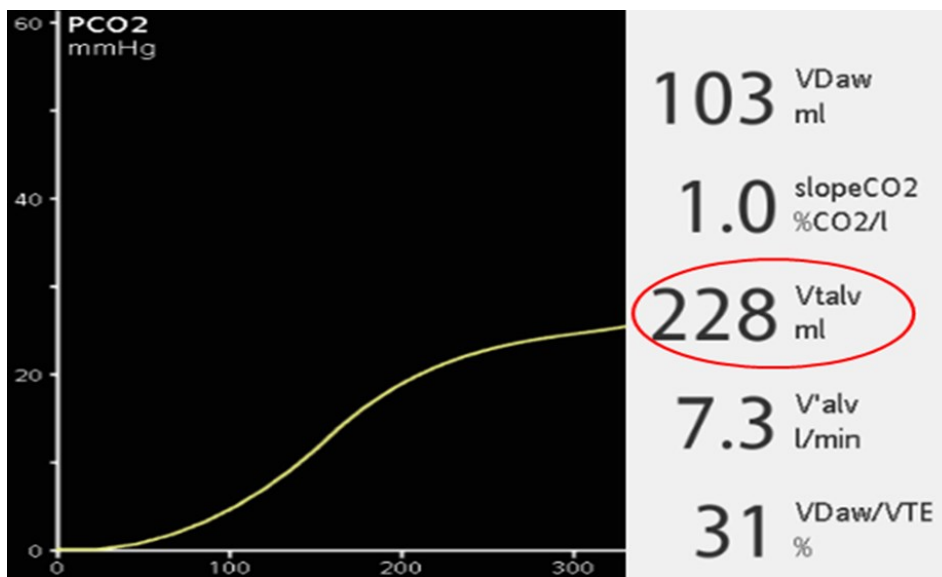
In this way we can also know the quantity of air that does not participate in the exchanges:

$$150\text{ml} \times 12 = 1800\text{ml/minute.}$$

We can say that of 6 liters per minute, only 4.2 liters are really useful.

**Alveolar dead space** represents alveolar space that is ventilated but is not perfused and therefore does not participate in gas exchange. This may be due to embolism, decreased cardiac output, anemia, or atelectasis.

If we add the anatomical dead space and the alveolar dead space, we obtain the **physiological dead space**.



Volumetric capnometry curve in the left side (tidal volume on x-axis), exhaled pressure of CO<sub>2</sub> (PeCO<sub>2</sub>) on y-axis. Alveolar tidal volume V<sub>Talv</sub>: 228 ml (red circle), Anatomical dead space: V<sub>Daw</sub> 103, Dead space to tidal volume ratio: V<sub>Daw</sub>/V<sub>TE</sub>

## Respiratory failure

When everything goes smoothly, we are in a normal condition. But what happens when this gas exchange is compromised? We are in a state of respiratory failure.

But what do we mean by respiratory failure?

We mainly have two types of respiratory failure:

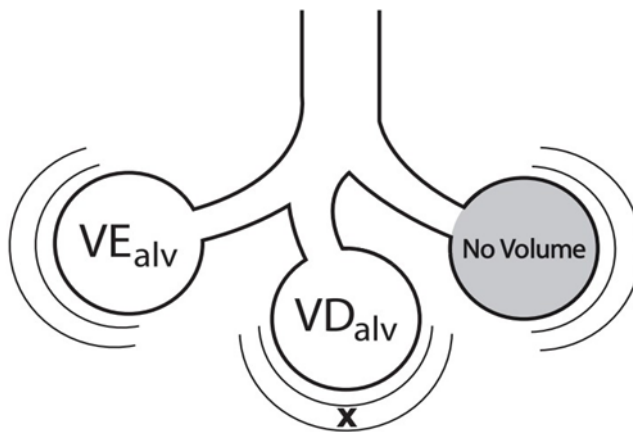
Hypoxemic and hypercapnic..... and mixed!

...hey Gi, did you notice that there are three? Yes, yes, but there are two main components, be careful!

## Hypoxemic respiratory failure

In this condition, gas exchanges do not allow adequate oxygenation of the blood, with low levels of oxygen ( $O_2$ ) causing hypoxemia. The  $CO_2$  value remains normal or slightly lower than normal. It may be due to:

- Reduction of inspired oxygen, for example if we are at high altitude.
- Hypoventilation, reduced minute ventilation in cases of apnea or hypopnea
- Problem with oxygen diffusion at the alveolar level, for example in the presence of liquid due to pneumonia.
- V/Q (ventilation/perfusion) mismatch, with adequate oxygenation but poor perfusion or vice versa.
- Shunt, when alveolar consolidation happens so there is no transfer of oxygen and carbon dioxide from the capillaries and alveoli and blood goes back into the arteries unoxygenated.



Schematic drawing showing 3 different alveoli with their corresponding capillaries. On the left: well perfused and ventilated alveolus with effective alveolar volume ( $VE_{alv}$ ), the middle is a ventilated but not perfused alveolus and is considered as alveolar dead space ( $VD_{alv}$ ), and the right one is an alveolus that is perfused but filled with fluid or collapsed (shunt).

In this case, the partial pressure of oxygen in the arterial blood ( $PaO_2$ ) is less than 60 mmHg, so there is not enough oxygen to adequately perfuse the tissues.

## Hypercapnic respiratory failure

In this condition, two conditions occur: a reduction in oxygen supplied to the blood and inadequate elimination of carbon dioxide ( $CO_2$ ).

Then, we see a decrease in oxygen ( $O_2$ ) concentration and an increase in  $CO_2$  concentration, or hypercapnia.

It may be due to:

Airway obstruction, such as chronic obstructive pulmonary disease (COPD).

Problems with respiratory rate (RR), as in respiratory depression.

Neurological problems, such as paralysis of the breathing muscles.

The partial pressure of  $CO_2$  in arterial blood ( $PaCO_2$ ) is greater than 50 mmHg and the pH is less than 7.30. The change in pH is caused by the presence of  $CO_2$  in excessive quantities. We have patients such as COPD who have high  $PCO_2$  values but are compensated with a normal pH.

## Mixed respiratory failure

In mixed respiratory failure we have a problem with hypoxemia and hypercapnia.

## Acute and chronic respiratory failure

We can also divide respiratory failures based on their time of onset.

So, we can have acute, chronic and acute on chronic respiratory failure.

**Acute respiratory failure** is a condition in which breathing is unable to meet the body's metabolic needs. It occurs suddenly.

**Chronic respiratory failure** is a condition in which breathing is impaired over time. It develops gradually.

**Acute-on-chronic respiratory failure** is a condition in which acute respiratory failure occurs in a patient with pre-existing chronic respiratory failure. It is a serious condition that can be fatal if left untreated.

## Mild, moderate and severe respiratory failure

We therefore have a respiratory system that to define as complex is limiting. A system that works great until there are problems that we identify as respiratory failure.

This respiratory failure can be of different types and can have different levels of severity.

Thus, we define as mild an impairment in which a patient cannot manage minimal effort, such as climbing stairs or a prolonged walk. To reach moderate conditions that require external aid. In moderate cases, non-invasive aids and interfaces will be sufficient. We then come to the severe forms in which we can still make use, quickly, "early" as the English would say, of non-invasive sealed systems such as CPAP or BiPAP, but which more often require invasive methods such as intubation of the patient or tracheostomy and use of mechanical ventilation. So:

### Mild respiratory failure

Characterized by relatively mild symptoms, such as:

Shortness of breath, cough, chest pain, nausea and vomiting.

Respiratory function is still sufficient to meet the body's needs.

### Moderate respiratory failure

Characterized by more serious symptoms, such as:



Severe shortness of breath, cyanosis (bluish discoloration of the skin and mucous membranes), mental confusion, inability to speak  
Pulmonary function is compromised, and the patient may require ventilatory support.

### Severe respiratory failure

Severe respiratory failure is a life-threatening condition. Symptoms are very serious and can include:

Unbearable shortness of breath, severe cyanosis, coma

In this case, the patient requires mechanical ventilation and is likely to need to be admitted to intensive care.

## What is cyanosis and what does it indicate

Cyanosis is a condition in which the skin and mucous membranes take on a bluish color.

This happens because the blood is not sufficiently oxygenated.

It can be central if the hypoxemia is systemic or peripheral, i.e. affecting only the peripheral districts. Cyanosis usually appears with saturation values lower than 85%. In case of anemia or shock, cyanosis may appear with higher saturation values.

Cyanosis is therefore given by the amount of hemoglobin that is not oxygenated or deoxygenated.

The normal amount of hemoglobin in men is between 13 and 17 g/dL, in women between 12 and 15 g/dL. If deoxygenated hemoglobin is greater than 5 g/dL, then cyanosis may occur.

People with chronic anemia deserve a separate discussion, as they can reach significant levels of desaturation and, in some cases, close to 60% before cyanosis occurs.

Last curiosity:

"Cyan-" comes from the ancient Greek "kyanós" (κυανός), meaning "dark blue" or "bluish", cyan is in fact a color.

## Main pathologies and problems of the respiratory system

In the first part of this manual, I have deliberately avoided describing the respiratory system at an anatomical and physiological level unless necessary; I consider this information within everyone's reach and easily available.

What I wanted to do is leave some space, otherwise I would run out of pages of paper! to describe the most frequent pathological conditions in the patients we encounter in intensive care.

Pathologies that generate respiratory failure in brief:

### COPD (Chronic Obstructive Pulmonary Disease)

It represents a series of progressive respiratory diseases, emphysema and chronic bronchitis are included among COPD. Not all patients with emphysema have COPD.

In COPD the patient is unable to let the air out causing it to become trapped (air trap).

COPD is a chronic obstructive disease caused by inflammation of the narrowing airways. Many people have asthma and COPD at the same time.

Caused by smoke, toxic substances and irritating dust. At the beginning the symptoms may be absent or very mild, as the disease progresses, we have shortness of breath, productive cough, chest tightness, difficulty breathing, easy fatigue, weight loss.

Usually the airways are elastic, in COPD the airways narrow due to chronic inflammation and the production of mucus increases. This reduction in the section of the airways makes it difficult to breathe by increasing the work of breathing (WOB).

In patients with COPD, the alveolar sacs have broken alveoli with larger chambers, where gas exchange is reduced. In these pockets the supply of fresh air and emptying are extremely slowed down. In fact, it is precisely the expiratory phase that is most difficult for these patients. This pathology is more frequent in men but has a higher mortality rate in women.

Chronic inflammation of the tissues causes the destruction of the alveolar sacs, which effectively become emphysematous. In fact, often the combination of these two factors, emphysema and chronic bronchitis, leads to COPD.

There is no cure for COPD, but symptoms can be relieved. Bronchodilators, steroids, elimination of cigarette smoke and oxygen administration, respiratory physiotherapy are used. In severe cases, thoracic surgery or lung transplant is required.

### **ARDS (Acute Respiratory Distress Syndrome)**

It is a pathological condition caused by an inflammatory lung lesion with fluids accumulating in the alveoli, it can be caused by pneumonia, sepsis, covid 19, serious trauma, burns and other causes. The impossibility of exchanges generates even severe hypoxia, leading to hospitalization in intensive care for support with mechanical ventilation. This hypoxic condition quickly affects all organs.

As symptoms we have severe shortness of breath, breathlessness, severe tiredness, confusion, rapid heartbeat, cyanosis of lips and nails, cough and chest pain.

If sepsis is present, we also have hypotension and fever.

We carry out a diagnosis via chest x-ray, ABG, echocardiogram, CT scan, bronchial culture tests. In mild cases, oxygen therapy can be instituted, but the use of mechanical ventilation is usually required. Complications such as pneumothorax, deep vein thrombosis, confusion, muscle weakness, anxiety and depression, pulmonary fibrosis may appear.

It does not affect the conducting airways but only the alveoli, with leakage of liquids that fill the alveolus causing non-ventilation and atelectasis (Shunt). This compromises gas exchange. ARDS causes hypoxemia that is refractory to oxygen administration. What will correct this hypoxemia is positive pressure, alveolar recruitment, re-establishing a normal exchange.

The real problem is the inflammation that includes the alveolar and capillary membrane, partially or completely compromising the exchange of respiratory gases.

In an ARDS condition we check the  $\text{PaO}_2/\text{FiO}_2$  ratio (P/F) on the blood gas and three scenarios can arise:

A mild form with P/F values between 200-300 has a mortality of 27%

A moderate form with P/F values between 100-200 has a mortality of 32%

The severe form with a P/F less than 100 has a mortality of 45%

When they require mechanical ventilation, these patients must be ventilated with low Vt because there is less parenchyma to ventilate and high PEEP, chosen based on the driving pressure which should be kept lower than 15cmH<sub>2</sub>O.

## **Emphysema** it is a form of COPD

It is the enlargement of the alveoli with rupture and formation of large air pockets in the lungs, it is called pulmonary emphysema.

The alveoli broken during exhalation trap air, not allowing the exchange between “old” air and “fresh” air also known as Auto-PEEP.

In most emphysematous patients there is chronic bronchitis, hence the presence of persistent cough.

The bubbles present are, to all intents and purposes, non-ventilating areas (V/Q mismatch). Emphysema and chronic bronchitis are a form of COPD, treatment can improve the condition but cannot reverse the damage.

Elastin and collagen are destroyed by chronic inflammation, making the alveoli stiff and delicate. It is precisely the lack of elastin that causes the collapse of the terminal airways. For the same reason, the terminal airways become much more compliant, generating real pockets during inspiration, pockets that narrow during exhalation.

Symptoms can be:

Cough, wheezing, shortness of breath, tightness in the chest, there may be anxiety, depression and easy fatigability. Caused mainly by cigarette smoke and chemical dust and fumes, but also present in non-smokers. Its evolution leads to COPD.

A normal person exhales about 5 liters per minute, 4 liters of which in the first second.

Then exhale 80% of the total in just one second. A patient with emphysema can expel only 4 liters per minute and only 2 liters in the first second, reaching 50% of the total exhaled in the first second. The values are really reduced compared to the norm. This causes a serious condition of respiratory failure.

## **Pulmonary fibrosis**

Pulmonary fibrosis is a serious, lifelong disease caused by scarring and tissue thickening that reduces lung compliance, making it difficult to breathe. Fibrosis is a terminal disease that ultimately leads to death. Depending on the etiology and severity, it has a very rapid prognosis or one that can last years, but this is not predictable. Pulmonary fibrosis is not a form of COPD, as it is an interstitial which affects the thin membrane between the cells of the pulmonary alveoli and blood vessels, causing them to thicken. Cigarette smoking can cause fibrosis but in the majority of cases the cause remains unknown (idiopathic), toxic fumes, silica, asbestos, coal.

Symptoms include shortness of breath, dry cough that doesn't go away, fatigue, shortness of breath, unexplained weight loss.

In advanced disease there may be clubbing, with fingertips that appear wider and rounder, cyanosis.

The diagnosis is made with chest x-ray, CT scan, and oxygen desaturation study.

The therapy is based on drugs capable of slowing down healing, oxygen therapy and respiratory physiotherapy.

## **Asthma**

Asthma is a lung disease in which the airways become narrowed, swollen and blocked by excess mucus. Asthma, which is a chronic disease, can cause bronchospasm due to the muscles narrowing the airways, due to this the air cannot flow freely.

There may be inflammation, always with narrowing of the airways.

During the attack, even more mucus is produced, causing obstruction.

In an asthma attack, as you exhale, your breathing may wheeze.

Asthma can be allergic or non-allergic; the non-allergic one can be triggered by exercise or stress. It can also be caused by environmental factors, respiratory infections and genetics.

Symptoms are chest tightness, cough, shortness of breath, wheezing.

The diagnosis can be made via spirometry and a chest x-ray may be requested.

It is treated through bronchodilators, anti-inflammatories or biological therapies.

## Pneumonia

It is a disease that generates inflammation and collection of fluids in the lungs, caused by bacteria, viruses or fungi. It can affect only one or two lungs, in this case it is called bilateral.

Bacterial pneumonia is usually more serious than viral pneumonia and requires treatment with antibacterials and hospitalization. The viral one, if not in severe form, gives the symptoms of a flu and resolves without treatment.

Pneumonia acquired outside of a hospital is called community-acquired pneumonia.

Hospital-acquired pneumonia is called nosocomial.

Those caught in hospital are usually more serious because they are caused by antibiotic-resistant bacteria, such as methicillin-resistant *Staphylococcus aureus* (MRSA).

Pneumonia associated with mechanical ventilation is called Ventilator Associated Pneumonia (VAP).

We can have pneumonia from inhalation of gastric contents, called "aspiration".

Symptoms of bacterial pneumonia include high fever, tiredness, shortness of breath, rapid breathing, sweating and chills, fast heartbeat, loss of appetite, chest pain, confusion and altered mental status, cyanosis of lips and nails.

Symptoms of viral pneumonia include dry cough, headache, muscle pain, extreme tiredness or weakness.

Fungal pneumonia is not contagious.

The diagnosis is made with chest x-ray, pulse oximetry and sputum examination, blood gas analysis, bronchoscopy.

Treatment includes the use of antibiotics, antivirals and antifungals, oxygen therapy, IV fluids, and fluid drainage in case of pleural effusion.

## Pleural effusion

By pleural effusion we mean the accumulation of fluid between the pleura and the lung. It may be due to viruses, pneumonia, or heart failure. If mild, it may be asymptomatic, otherwise it may cause chest pain, dry non-productive cough, dyspnea, requiring orthopnea (forced breathing in a straight sitting position or standing position).

It can also be caused by pulmonary embolism, cirrhosis, nephropathy, cancer, inflammatory disease, tuberculosis, autoimmune disease.

The diagnosis is made with chest x-ray, CT, chest ultrasound, thoracentesis.

It can be treated with anti-inflammatories, diuretics, chest drainage. It causes difficulty in lung expansion. In the pleura the lung is able to move thanks to a minimal quantity of lubricating liquid composed of proteins such as albumin. This space between visceral and parietal pleura is practically a virtual space in a normal individual. Normally the capillaries present in the two membranes convey liquid into the pleural space, this liquid is evacuated by the lymphatic vessels. An accumulation of fluid occurs when we have excessive production such as transudate or exudate

or when the lymphatic vessels are unable to drain it adequately. Fluid accumulation may result from an increase in hydrostatic pressure or a reduction in oncotic pressure. An increase in hydrostatic pressure may be due to heart failure. If we have low oncotic pressure due to liver failure or nephrotic syndrome we will have a passage of fluids into the pleura.

Even inflammation of the capillaries can cause a pleural effusion as the walls of these capillaries become much more permeable. This may be due to a tumor, inflammation, pneumonia or trauma. Depending on its extent, it can be asymptomatic or cause breathing difficulties and pain. In severe cases the lung is completely compressed, and the trachea shifts to the side of the effusion. To evacuate excess fluid, which could also be blood (Hemothorax) or pus (Empyema), a thoracentesis is necessary. Through this it is also possible to analyze the liquid and therefore make a targeted diagnosis. Treatment includes diuretics, chest drainage, or surgery.

## Pulmonary edema

Pulmonary edema is a collection of fluids in the lungs, it can be chronic or acute. One of the main causes is congestive heart failure. It causes shortness of breath and difficulty breathing. You may have wheezing and a cough with foam.

Acute pulmonary edema is life-threatening and requires immediate treatment.

Pulmonary edema can be cardiogenic and non-cardiogenic. In the non-cardiogenic one, the blood vessels become inflamed and damaged, causing fluid leakage (ARDS).

Causes of pulmonary edema are pneumonia, sepsis, trauma, pancreatitis, liver disease, drugs.

We perform a diagnosis with chest x-ray, CT, ECG, echocardiogram, pulse oximetry, cardiac catheterization. In case of acute pulmonary edema, the treatment is urgent and includes high flow oxygen up to mechanical ventilation and diuretics to evacuate excess fluids.

The cardiac cause is due to a failure of the left heart, thus generating an accumulation of blood in the left atrium, pulmonary veins and pulmonary capillaries with pulmonary hypertension.

The balance between fluids produced and removed is lost and the increased capillary pressure causes increased passage of fluid into the lungs.

This phenomenon can also occur in the presence of severe hypertension with 180 systolic and 110 diastolic.

Non-cardiogenic causes include lung infections, chest trauma, inhalation of toxic substances. These are causes that act directly on the alveoli and their membrane, thus inflammation increases capillary permeability, favoring the passage of fluids into the interstitial space. Another cause may be sepsis which can promote the accumulation of fluids throughout the body including the lungs. Low oncotic pressure also generates pulmonary edema. For example, in the presence of reduced albumin, due to malnutrition, liver failure or nephrotic syndrome.

Pulmonary edema makes it difficult for gases to pass through an interstitial space enlarged by the presence of excess fluid.

The orthopneic position makes breathing easier as in the supine position we have greater pulmonary congestion. Treatment includes giving oxygen and treating the causes.

## Atelectasis

A condition called atelectasis is when the alveolar sacs cannot inflate properly.

Non-expansion of the alveoli can be caused by compression of liquids, air or blood, obstruction to air flow or caused by lung scarring. It can occur in small areas or affect the entire lung.

We have pneumothorax when air escapes from the lung causing it to collapse, in this case the entire lung is atelectatic.

If we find ourselves in severe conditions of atelectasis with large affected areas, we may have symptoms such as breathing difficulties, cough, chest pain, tachypnea, cyanotic lips and nails. COPD, mucus plugs, inhaled objects, pleural effusion, pneumothorax, tumors and lung scarring can also cause atelectasis. The diagnosis can be made with chest x-ray, CT and bronchoscopy. Atelectasis causes low blood oxygen, pneumonia, and/or respiratory failure. An atelectatic area is an area that does not participate in gas exchange, generating or worsening a picture of respiratory failure.

## Part two: A little help, thanks!!!

In the first part I talked about how a normal person breathes; I tried to describe the respiratory mechanics as a whole in an easy way. In doing so, I deliberately avoided complex discussions or state equations that would only complicate the discussion. There will be ways and opportunities to include and describe the concepts listed above in more depth. But if it is true that the breathing or ventilation of a healthy patient begins and ends with the description of "how he breathes", it is also true that when a patient begins to have different needs, a whole series of aids come into play that we need to know.

These aids, or devices for those who speak English, help the patient in case of hypoxia and reduce respiratory work and cardiac work.

Hypoxia leads these patients to an increase in breathing rate and heart rate in a desperate attempt to recover the necessary amount of oxygen. Work of breathing (WOB) is also increased. We can have cases that need light help and cases that need very high oxygen support.

You will find below the definition of hypoxia and work of breathing; the latter is often referred to as WOB, an acronym for Work of Breathing.

In this second part I will focus on all those devices that help the patient feel better, devices that provide, in the majority of cases, an increase in oxygen.

So, let's talk about masks, nasal cannulas and similar.

**For these devices to work, the patient must be able to breathe spontaneously.**

Hypoxia: is a condition in which tissues do not receive enough oxygen necessary for normal metabolism. As an indicative reference, we can consider a patient in hypoxia with an oxygen saturation in the arterial blood ( $\text{SaO}_2$ ) equal to or lower than 90%.

You will find more details on hypoxia and hypoxemia later.

### Basic respiratory equipment

#### Nasal cannulas



One of the most used devices in intensive care with patients who only require oxygen support are nasal cannulas. They are composed of a small tube that carries oxygen to two perforated prongs that are inserted into the nostrils.

They are often called “nose pads” or “goggles”.

The oxygen supply that this device can provide is limited and certainly, if our patient does not have the strength to breathe effectively, they will be of little use.

They are fixed to the face by passing them around the ears and fixing everything under the chin. You need to be careful not to overtighten the nasal cannula, which could be uncomfortable. They are very practical compared to other devices, because they allow the patient to eat or drink freely. They can be used with an oxygen flow between 1 and 6 liters/minute, thus reaching an oxygen percentage between 24 and 44%.

The operating limit of 6 liters/minute is due to the section of the cannula; in this case, increasing the liters per minute will not lead to a real increase in  $FiO_2$ .

We must pay particular attention to the humidification of the oxygen delivered. Remember that when we are faced with a device that delivers a flow greater than 4 liters/minute of oxygen, humidification of the airways must be envisioned.

The use of nasal cannulas is not recommended in patients who breathe mainly through the mouth, even if in some texts you find that this is not a limit or a problem.

Sensation of dryness and irritation are always present in case of inadequate oxygenation of the gases delivered. I remind you that pure oxygen in a cylinder or that of centralized distribution is always very dry.

It should be the first device to be used if oxygen-only support is needed for patients in intensive care, except in cases where it is chosen for its practicality and ergonomics.

1-6 liters/minute nasal cannulas 24-44% $FiO_2$
---

Above 4 liters/minute humidifying the airways is a good strategy!

## Simple face mask





The second device is the simple facial mask, composed of a mask that covers the nose and mouth, with an elastic fastening that is blocked behind the head and a small tube that blocks the oxygen from the flow meter to the patient; Furthermore, in some models there is an aluminum part in the upper part of the nose which allows the mask to be adapted to the patient's anatomy.

With the simple face mask, we can deliver oxygen flows between 6 - 12 liters/minute, equivalent to the amount of oxygen between 35% and 50%.

Naturally, all the devices taken into consideration can be used with lower quantities of oxygen.

Thus, it is not uncommon to find a patient with a face mask or nasal cannulas with only 2 liters of oxygen or even just 1 liter.

The simple face mask is recommended as oxygen support in post-operative patients or those who do not require invasive ventilation. It is especially suitable for patients with forced oral breathing due to the presence of a nasogastric tube or nasal swabs.

Some of these masks allow you to connect to the aerosol administration system, in other cases there are dedicated masks with the ampoule included in the kit.

The face mask has holes, usually in the sides, that allow room air to mix with the oxygen delivered. This added to the fact that the patient exhales into the space of the mask, leads us to the conclusion that the patient who is breathing with 6 liters of oxygen is not really receiving the correct or expected amount of oxygen.

In fact, the quantity will be lower: instead of a range between 44% and 68% for 6-12 liters/minute we have the percentage written above 35%-50%. The percentage of oxygen lost will be replaced by the ambient air that has mixed with the oxygen supplied, through those famous side holes.

This phenomenon is truer the lower the flow of oxygen delivered. This is why this device should be used with flows of at least 6 liters/minute. Furthermore, these blessed minimum flows of 6 to 12 liters/minute also have another very important task:

evacuate as much exhaled air as possible from the mask, to reduce the **rebreathing phenomenon!** Interesting right?

## Rebreathing

Rebreathing means breathing the same air that was exhaled. This causes the concentration of oxygen delivered to drop, in some cases even significantly.

In an ideal world it would be necessary to avoid this phenomenon, but we humans experience it every day without even knowing it. If you think about it, a portion of the air we exhale is brought back into the lungs at the next inhalation, this is the air that remains in our dead space.

If you've been paying attention, I already talked about dead space a few pages earlier!

Remember those holes on the sides? Well, in some cases they may have integrated valves. These valves open when the patient is exhaling, allowing air to escape, and close when inhaling, reducing the amount of ambient air that will mix with the patient's inspiration. The presence of these valves slightly improves oxygen delivery performance. This is because this type of mask, not being airtight, allows air to enter throughout their perimeter. It is true, however, that they allow you to breathe with higher percentages of oxygen.

Simple face mask 6-12 liters/minute 44-68% theoretical FiO <sub>2</sub> !
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The real value drops to 35-50%
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Above 4 liters/minute humidifying the airways is not a bad idea!

## Face mask with reservoir



It is a device for administering oxygen completely similar to the normal mask, which has a tank at the base that is filled with pure oxygen. When the patient inhales, he will take in a higher quantity of oxygen from this tank than the normal mask can guarantee.

It is a type of non-rebreathing mask, this allows the patient to receive a higher percentage of oxygen thanks to the fact that it does not allow mixing between exhaled air and "fresh" inhaled air. It should be used with a flow of 8-10 liters/minute and guarantees a percentage of oxygen available for the patient above 60%. In some cases, manufacturers indicate a maximum percentage close to 90%.

Before allowing the patient to use it, it is necessary to ensure that the reserve tank is filled. If you notice that the reservoir is emptying during inhalation, you will need to increase the incoming oxygen flow. This will allow the reservoir to best perform its function.

The reservoir should empty in approximately 1/3 of the time with each breath and no more.

The large flow of very high oxygen associated with the reserve tank allows for a "boost" at the moment of inspiration and to wash away the portion of the patient's exhaled breath. The presence of a non-return valve between the reservoir and the mask does not allow the exhaled air to mix with that present in the reservoir, which is why we can reach such high percentages.

This mask on the side also has holes equipped with one-way valves, this is because the exhaled air will exit calmly and will not allow ambient air to enter and thus reduce the oxygen concentration.

Always remember to humidify the oxygen using a bubbler and sterile water always above 4-6 liters/minute. Furthermore, being a mask that does not have a perfect seal, the delivery percentage depends on its correct positioning. To be clear, if the patient moves the mask and places it on his forehead, we can give all the oxygen we want but, apart from the blonde hair, we would not have the desired benefits!

There is a model called partial rebreathing, the same as the previous one but with holes instead of valves. In this case, the percentage of oxygen supplied drops considerably and with 6-10 liters/minute it reaches 35-60% of O<sub>2</sub>.

Mask with non-rebreathing reservoir of 8-10 liters/minute 60-90% FiO <sub>2</sub>
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Mask with partial-rebreathing reservoir of 6-10 liters/minute 35-60% FiO<sub>2</sub>

Above 4 liters/minute humidifying the airways could be a trick!

In this table the percentage of oxygen delivered per liters/minute.

As you have seen, **this is an indicative percentage** and varies depending on the device used, so it can be more precise for nasal cannulas and less so for a simple rebreathing type facial mask.

Liters/minute	0	1	2	3	4	5	6	7	8	9	10
% oxygen	21	24	28	32	36	40	44	48	52	56	60

### The Venturi mask



What you see in the photo is a venturi mask with different nozzles for each percentage of oxygen that can be delivered. Each nozzle must be mounted according to the patient's needs and will provide a percentage of oxygen very close to that indicated. To do this, the flow of oxygen to be delivered must be suitable. So, if I want to give my patient 31% oxygen, he will have to mount the orange nozzle and adjust the flow meter to a delivery equal to 6 liters / minute.

This is what we need to do to ensure a correct percentage. The Venturi mask is the ideal aid in intensive care when we want to administer a precise percentage of oxygen.

Also, in this case with flows greater than 4-6 liters per minute, we can consider the possibility of humidifying the flow of oxygen administered.

Do not take into consideration the values I have entered in the photo because the color code is not a standard and you may find different settings in different brands of the available products.

Check the description sheet on the mask every time.

Now, since things are not always easy, I must tell you that there is also a Venturi mask with a single adjustable nozzle, this one here:



Even with this mask, once you have set the nozzle to the desired percentage, you will have to adjust the flow meter to the corresponding liters.

This is the "pro" version of a normal oxygen mask and differs from it because it is able to have greater precision in the delivery of oxygen to be administered to the patient. The fixing system is the same via elastic and has holes in a lateral position. The tube between the mask and the nozzle, when present, has the sole purpose of allowing a homogeneous distribution of the oxygen flow. In the kit you will also find a nozzle cover which usually remains in the kit packaging; its importance is not recognized by most, who consider it a waste of money and plastic! However, this is not the case! And believe it or not, I'm about to tell you what that "piece of plastic" considered so useless is for.

He protects the nozzle and in particular the parts from which it takes the ambient air from accidental closure, for example from the sheet or from the patient's arm. Due to the way it is designed, it protects the nozzle 360°, allowing the air to flow into the system in any case. The Venturi system in fact works by exploiting the incoming oxygen flow and through its valve causes this flow to carry with it a certain quantity of ambient air. This well-calibrated and measured system allows for the precision we talked about before, which is why it is important to adjust both the oxygen flow and the type of nozzle or its opening.

Now I'll respond to those who are so curious they're biting their fingers!

They will have noticed that in the first photo we have two nozzles with different oxygen percentages but the same liters/minute; the "Mr. Yellow" 35% and the "Mr. Red" 40% both require 8 liters/minute. This is because in this case the size of the nozzle window that allows the ambient air to enter the system will vary. By varying the air-oxygen mixture with the variation in the quantity of ambient air dragged by the oxygen flow, ehh... yes, the "window" that lets the ambient air pass through the yellow one will be smaller! Well done!

...but sorry, couldn't they have done it for the others too?

...and who am I Mr. Venturi? Ask him!

Venturi masks typically range from 4 to 12 liters/minute and provide 24 to 60 percent oxygen

Color	Liters/minute	O2 percentage
Blue	2	24%
White	4	28%
Orange	6	31%
Yellow	8	35%
Red	8	40%
Purple	12	50%
Green	15	60%

The color code is not standard, refer to the instructions for your device!  
Above 4 liters/minute humidifying the airways could be a trick!

### The respiratory enhancer



When a patient remains connected to the ventilator, he or she experiences progressive muscle depletion. This loss of function of the respiratory muscles is all the more important the longer the patient has remained dependent on mechanical ventilation; furthermore, it depends on the type of ventilation used. In this context, controlled ventilation causes worse muscle atrophy (Diaphragmatic dysfunction) than assisted or spontaneous ones. Once we have weaned the patient and removed that annoying tube, we need to get him back on track as quickly as possible. To help the patient recover muscle function, we must make him work on the respiratory muscles; to do this, we use a device called a respiratory incentive.

There are different types of incentives, but the operating principle is always the same: rehabilitating the respiratory muscles through the application of forced inspiration. This forced inhalation has the task of moving indicators, usually colored plastic balls, from one point down to another. Incentives usually have three different sectors where the indicators are moved with an increasing level of effort. The aim of this type of exercise is to bring all the indicators to the top.

In addition to being a respiratory enhancer, it can also be called a respiratory exerciser. The models are different in type, quantity of compartments, shapes and colors. The one I've included in the photo has three compartments for the colored marbles to go through, each of which requires more effort. So, first you have to raise the red colored marble, which requires an effort capable of moving 600 mL. When the first one has reached the top, an inhalation of 900 mL is needed to make the yellow ball reach the "top". It can be considered an excellent result when the patient manages to lift all three balls reaching a notable tidal of 1200 mL. All this, as already mentioned, exclusively with the use of inspiratory force. You should know that if a patient cannot even move the first ball with inhalation, it is possible to make him work on exhalation. In fact, many patients manage to blow in the first phase after extubation, and therefore exhale forcefully, rather than inhale. To do this, simply put the incentive the other way around and invite the patient to blow. The system with the colored balls helps people manage their own respiratory rehabilitation and gives the patient greater safety, allowing them to regain their autonomy.

## The flow meter

This in the photo is a flow meter and a bubbler:



Each intensive care unit has more than one oxygen connection. These connections allow the use of oxygen which is made available centrally and have the characteristic of reaching considerable pressures which allow flows of up to 15 or 30 liters/minute, sometimes even higher.

When we need to supply oxygen to a patient wearing a mask, nasal cannula, Venturi or other we can use this socket via a flow meter which regulates the flow in liters/minute.

The flow meters have a cone connection to be able to connect the tubes of devices such as masks, nasal cannulas or a "to and fro".

This cone fitting can be removed to insert a bubbler; in this case it will be the bubbler that has the cone connection for the various devices.

The bubbler must be filled with sterile double-distilled water and allows humidified oxygen to be given to the patient; this is because the oxygen that is distributed is dry, and this can be a problem for patients who ventilate with very high oxygen percentages.

When the amount of oxygen is low, less than 4 liters/minute, it is mixed with the ambient air and our respiratory system is able to manage humidification better.

The rear part of the flow meter is the one that will go to the centralized oxygen connection; in the lower part we find a thread to which the bubbler will be attached which allows the humidification of the oxygen flow sent to the patient.

I'll tell you something obvious that might escape most people: up to this point what we deliver with a flow meter is 100% pure oxygen, which is why I haven't talked about air.

The flow meter allows adjustment via a knob and the value in liters of oxygen per minute is indicated by a metal dot or a floating cone that is positioned near the regulated value. If we have the cone available, refer to its upper part as an index of the flow delivered. If it is a metal sphere, in theory the exact center would be the point to use as a reference, but if in doubt, close the flow meter and check that point of the ball is zero!

## Oxygen

From the discussions made earlier we understood that a healthy person, from a respiratory point of view, is able to be completely autonomous. When insufficiencies or more serious pathologies arise, the first help we can give is an increase in oxygen. This argument is valid both in routine conditions such as in a post-operative patient, and in serious conditions such as patients in intensive care.

Oxygen, it should be remembered, is a drug. As a drug it must be prescribed exclusively by the doctor who defines the method of administration and the quantity to be administered. Let's remember that oxygen also has a cost and that this is not exactly cheap; leaving a flow meter open when a patient is not using it is a waste that we should avoid.

In an intensive care unit and in normal hospitalizations, oxygen arrives via dedicated sockets directly from the unit's distribution center.

This power station can be equipped with a single large tank or a system of large cylinders in quantities sufficient to meet any request from the operating units.

In this, the Covid19 health emergency has highlighted infrastructure deficiencies in many facilities, in several cases forcing operators to use portable oxygen cylinders.

For information only, our department has been tested for continuous delivery with all flow meters set at 15 liters/minute open simultaneously without experiencing any drop in pressure in the

distribution system. This is a test that is often performed to certify an adequate flow rate of the "oxygen" system.

If there is no oxygen available from the distribution system sockets, also called centralized oxygen, we can connect a large cylinder, usually 50 liters, to the department's distribution system, thus making up for the lack of oxygen.

There are therefore two ways to have oxygen available in our department:

- Centralized
- Portable cylinders

If we want to be precise, oxygen is also available in ambient air, but let's not be precise otherwise they will tell us that we are PiBinKy (search the acronyms).

Portable cylinders are smaller in size and are available with variable volumes of 3, 5, 7 and 10 liters. The capacity in liters is called "water capacity" in this type of cylinder, which indicates how much water in volume would be needed to fill them.

An oxygen cylinder has an upper part, called a nose cone, which is always white. White is the color that identifies oxygen in medical gases according to workplace safety standards. It is equipped with a pressure gauge which indicates the internal pressure in Bar. In fact, it contains oxygen inside it in the form of a gas compressed to many atmospheres, usually the maximum limit is 200 atmospheres.

In the pressure gauge there may be the acronym **ATM** to indicate atmospheres, you can also find **bar** as a unit of pressure measurement.

Consider a bar equivalent to an atmosphere:

### 1 atmosphere = 1.013 bar

The cylinder then has a shut-off tap, a pressure reducer with a delivery regulator. The pressure reducer takes care, through the regulator, of bringing the total internal pressure of the cylinder to a lower level, providing an oxygen flow between 0.5 and 15 liters/minute.

Delivery can take place via a standard conical-type connector which allows the attachment of classic oxygen tubes of devices such as masks, nasal cannulas or "back and forth".

Alternatively, some cylinders have a standard DIN type connection the same as those positioned in the hospital room and used for connection to mechanical ventilators.

I insert here some images that can complete the description just made:





On the left the white color of the oxygen cylinder nose cone, on the right the green symbol indicates a non-toxic and non-flammable bottled gas, the yellow symbol indicates that it is an oxidizer, i.e. it can accelerate a fire.



There are dozens of different pressure gauges, they usually also have a colored pressure indication in order to instantly give an indication of the availability of oxygen. In the image above, up to 40 atmospheres the cylinder is considered usable.

Let's now see how to calculate the autonomy of a cylinder quickly and easily. It is a calculation that you will have to make every time you have to use a cylinder, whether for transport or for a bedridden patient who must remain in the room.

Important note: if you have a patient in bed and use a cylinder for oxygen administration if you do not carefully evaluate its duration and autonomy **when the oxygen runs out there will be no alarm to alert you**, that patient will simply no longer have oxygen available, be careful!

Calculation of the autonomy of an oxygen cylinder

- 1 We need to know the size of the oxygen cylinder in liters
- 2 We need to check his available pressure in the pressure gauge
- 3 We need to know how many liters/minute are needed for the patient.

We find the size written in the characteristics of the cylinder and, usually, in the nose part the values are 3, 5, 7, 10 liters.

The pressure gauge, through the position of the pointer, will tell us how many atmospheres or bars are present inside the cylinder.

We can see the useful liters/minute for the patient in the medical record or ask the doctor. If the patient is connected to centralized oxygen, we will take liters/minute here as a value to calculate the autonomy of the cylinder.

Once we have all this data, we first need to calculate how many liters of oxygen are available and deliverable from the cylinder.

## 1 How many liters does the cylinder contain?

To do this, we need to know that a full 5 liter cylinder has a pressure of 200 atmospheres. The compressed oxygen inside the cylinder will occupy those famous 5 liters of volume, but once past

the pressure reducer, the oxygen gas will occupy much more volume. And we want to know how much volume.

In short, how many liters of oxygen can this tank give me in total? Easy

We multiply the size of the cylinder by the atmospheres indicated.

Therefore: 5 liters x 200 Atmospheres, thus obtaining the total liters of the cylinder.

So,  $5 \times 200 = 1000$  liters

We now know that a 5 liter cylinder at its maximum operating pressure, i.e. 200 atmospheres, contains 1000 liters of oxygen in the form of gas at atmospheric pressure that we will deliver to the patient.

What if the 3 liter cylinder had always been full? Let's see it together:

$3 \times 200 = 600$  liters.

What if the cylinder was 7 liters with 80 atmospheres? More liters x pressure:

$7 \times 80 = 560$  liters.

Now we know how to calculate the total available liters and therefore we know the autonomy in liters of the cylinder, but we must find out the autonomy in time; How long will this blessed cylinder last?

This is the value we really need.

## 2 How long will the cylinder last?

To do this, we need to know how many liters per minute our patient uses.

So, if we use the liter values found above (1000, 600 and 560) and the patient has a face mask with oxygen at 3 liters/minute, the duration will be this:

$1000 \text{ liters} / 3 \text{ liters total} = 333 \text{ minutes}$ . In hours =  $333/60 = 5 \text{ hours and } 33 \text{ minutes}$

$600 \text{ liters} / 3 \text{ liters total} = 200 \text{ minutes}$ . In hours =  $200/60 = 3 \text{ hours and } 20 \text{ minutes}$

$560 \text{ liters} / 3 \text{ liters total} = 186 \text{ minutes}$ . In hours =  $186/60 = 3 \text{ hours and } 6 \text{ minutes}$

So, let's take the size of the (tank x pressure) / (liters/minute) = minutes

To transform minutes into hours, we need to divide by 60..... Almost!

Some examples to understand better.

7 liter cylinder with 120 atmospheres, patient with Venturi mask 35% oxygen, I have to go to Magnetic Resonance. Will he have the necessary supply of oxygen?

First step, how many liters does the cylinder have:  $7 \times 120 = 840$  liters total

Second step, how much oxygen does the patient need in one minute = Venturi 35% = 8 liters/minute

Third step, how many minutes of battery life:  $840/8 = 105$  minutes

Fourth step how many hours can I stay outside: 1 hour and 45 minutes

Now it's up to you to calculate whether the 120 atmosphere tank is the right choice or whether it would be better to get one with more oxygen. Also consider the fact that during resonance you must necessarily connect the patient to centralized oxygen, remember that:

**The cylinder just can't fit there;** while in CT or other places it is always possible to leave the patient connected to the cylinder you brought with him. My advice is to always connect the patient to centralized oxygen when present and keep the autonomy of the cylinder as high as possible.

So, what do we do? Are we going to resonate? I would say yes!

I hope these examples are not too boring, I think they can be useful.

Let's change the scenario:

Patient who must go to CT, 7 liter cylinder with 150 atmospheres, ventilated with "back and forth" with 12 liters/minute of oxygen. On with the calculations:

First pass:  $7 \times 150 = 1050$  liters

Second pass: 12 liters/minute

Third pass:  $1050/12 = 87$  minutes

Fourth step: Time?  $87/60 = 1$  hour and 27 minutes

In this case our autonomy will be less, but it should still guarantee us a round trip by CT without problems. But if I have to transport the same patient to another facility in the same conditions, things change a lot.

In this case it is good practice to take the cylinder fully charged. It is true that the ambulance has its own oxygen supply system, but we must be as cautious as possible.

**Never risk having to return to the ward with the "back and forth" that loses its source of oxygen.**

Now a little curiosity: the oxygen contained in the cylinders is not just oxygen, there is a margin of 0.5% on the total quantity. So out of 1000 liters there could be 5 liters "missing"; add to this the fact that the pressure gauge has a tolerance.

So when you do the calculations, take some margin to be safe!

The formula to follow to calculate the autonomy of a cylinder is this:

$$\frac{\text{Cylinder capacity (liters)} \times \text{Pressure indicated by the pressure gauge (atm or bar)}}{\text{Flow to be delivered (liters/minute)}}$$

This formula gives us the minutes of autonomy.

$$\frac{\text{Capacity} \times \text{Pressure}}{\text{Flow}} = \text{Minutes of battery life}$$

## What does the oximeter read and why is it overrated!

Oxygen saturation ( $SpO_2$ ) measures the amount of oxygen that has saturated the hemoglobin in arterial blood. This is a measurement in a peripheral district and for this reason can be influenced by various factors.

The oximeter has two LEDs that emit a different light, this light is then captured by sensors on the opposite side. Visible red light (660 nm) and infrared light (950 nm) are emitted; based on the amount of oxygen present in the arterial blood we obtain the saturation value.

Thus, when blood is oxygenated, it absorbs less infrared light and more red light.

When the blood is poor in oxygen the opposite will happen, it will absorb more infrared light and less red light. These values, appropriately evaluated by a measurement algorithm, determine the patient's saturation level.

Why is it overrated? Because despite being a very important value it can easily be deceived, either by underestimating or overestimating the real value.

Thus, hypotension, hypoperfusion, hypothermia, edema, enamel, restless patients, carbon monoxide intoxication can distort the measurement.

In an ideal condition in which we can trust the reading of the oximeter alone, values from 95-96 and above are to be considered good, between 94 and 90, they should alert us and let us verify the matter with other methods. A saturation lower than 80, if real, is an indication of severe hypoxia and, if it is lower than 70, it is to be considered a real emergency.

What should you evaluate instead of saturation? Not "instead of saturation", but with saturation it is important to evaluate a blood gas analysis whenever the respiratory failure situation requires it.

With this exam you will evaluate PaO<sub>2</sub>, PaCO<sub>2</sub> and the P/F index; these, together with the value given by the oximeter, offer the complete picture of the state of a patient with respiratory failure.

Do not check the patient only by observing the oximetry, it could be a big mistake.

A little curiosity: in the case of carbon monoxide (CO) poisoning, the oximetry value can be distorted and, in some cases, can reach values higher than 100% saturation. In this case, the saturation measurement is completely unreliable.

This is why some monitors allow setting alarms above 100%!

Then there are those patients who think it's a stress reliever and make it ring every 5 minutes happy to see you arrive to fix that beautiful red light!

## **Blood gas analysis the litmus test of ventilation!**

Blood gas analysis (EGA or ABG) is the tool with which the clinician can evaluate how the implemented ventilatory therapy is progressing, whether it is NIV ventilation or mechanical ventilation. In the same way that the titration of a norepinephrine infusion receives confirmation from the blood pressure values, the values offered by the ABG will allow us to adapt the ventilation strategy.

Especially when a patient is on mechanical ventilation it is necessary to have the trend of his PaO<sub>2</sub>, PaCO<sub>2</sub>, pH, P/F values and not only these. Seeing a patient who acquires the ability to trigger spontaneous breaths, and then deliver them autonomously, does not tell us much; seeing this progress and with it an ABG that improves and returns to normal ranges, for that patient is something completely different.

With the ABG we have complete monitoring, easy to perform, always available and fast, which allows very accurate management of mechanical ventilation and more. Always remember to observe the patient, the ventilator curves, the ABG values, the saturation, the radiological checks and the patient's state of wakefulness; with all these indicators it is possible to start weaning as early as possible in a timely manner. Early weaning reduces the days of mechanical ventilation, reducing the days of mechanical ventilation means reducing risks and with them mortality, think about it!

Remember that the clinician, by controlling specific mechanical ventilation settings, is able to modify the values "almost" at will, reversing acidosis or alkalosis, recovering hypoxia or hypercarbia. All this thanks to the knowledge of the values extrapolated from this exam.

In the management of mechanical ventilation, ABG plays a very important role not only for the clinician but also for those who have to monitor these patients; I am referring precisely to the nursing role. If you have no idea how to "read" an ABG, take a look at my work, I explain the basics in a very simple way.

## Part three: Excuse me! I need more help!!!

Up to this point we have discussed ventilation and help given by increasing the  $\text{FiO}_2$  administered to the patient. In many cases this is the only help needed and is indicated as an adjuvant factor in mild respiratory failure.

But mind you, we only limited ourselves to giving oxygen, we didn't act on anything else. The time has come to increase that help also from a pressure point of view. If you have been attentive, you will surely have noticed that, up to this point, the systems used, the devices used, or the interfaces used were not watertight.

Nasal cannulas, masks, Venturi mask: none of them guarantee a perfect seal with the respiratory system; in addition to this, the flows are so low that they do not affect the airway pressure level in any way. All the previous systems simply give oxygen, but do not increase pulmonary pressure and simply put, do not cause alveolar recruitment.

But we saw what alveolar recruitment is in the first part. Here we briefly remind you that if the respiratory insufficiency is of a medium-severe type, to the benefit of an increase in  $\text{FiO}_2$  we can add the benefit of making the closed alveoli ventilatable. To do this we must "recruit" them and we can only do this by increasing pressure; the increase in alveolar pressures can only be obtained through a sealed interface.

### Mapleson C and Ambu Ball



The Mapleson C circuit, commonly called "Come and Go," is used to ventilate a patient through a tight facemask, tracheostomy tube, endotracheal tube, or laryngeal mask airway.

It is made up of a tube that must be connected to the flow meter or oxygen cylinder using an adapter available in the assembly kit, a balloon that inflates with the oxygen flow, a special valve called APL (Adjustable Pressure Level) or relief valve overpressure, from the fitting which must be connected to the previously indicated devices.

The bag is 2 liters in size, more than necessary to deliver adequate tidal volumes, in an adult patient. The pediatric equivalent is smaller than the size suitable for the subject.

The adjustable valve allows you to choose the amount of oxygen that will fill the balloon and the amount that is discharged outside. When this valve is completely closed, all the flow of oxygen will fill the bag and, having no other possible escape routes, will generate pressure in the lungs. In

these cases, even if the oxygen flow is very low, there is a risk for the patient of barotrauma and subsequent pneumothorax.

The only reason why that valve must remain completely closed and at the initial moment, before ventilating an emergency patient, is only if it is necessary to fill the bag quickly.

In fact, it is the bag that, filled with oxygen, will allow the patient to be ventilated. Remember that this is a "passive" type bag that requires a source of oxygen to fill. Very different from its cousin the "Ambu bag" which, being self-expandable and more rigid, does not require an external gas source. As soon as the correct quantity of oxygen inside the bag has been reached, the valve must absolutely be adjusted so as to allow ventilation of the patient with an adequate time for filling the bag. This time must allow the bag to fill quickly to allow rapid ventilation and therefore with a high respiratory rate.

The correct technique is precisely to find a balance between the opening of the APL valve and the liters/minute delivered with the flow meter. It is not uncommon in emergency cases to put the flow meter at maximum flow rate and in any case above 10 liters/minute.

Remember that the Mapleson C circuit balloon is built with precise data relating to its resistance, in fact it must explode before the patient's poor lung does.

Despite this, you must not exaggerate the pressures and the quantity of air vented from the valve must be adequate. Remember that this is a non-rebreathing ventilation system, and that the patient's exhaled air comes out near the valve.

For this reason, it is good practice to place an antibacterial filter in the expiratory outlet connection, in order to protect all nearby personnel from the patient's exhaled breath.

## High flows

Among the sealed interfaces we also include high flows, even if they do not have a real seal, but they are able to maintain a certain pressure within the ventilatory system, favoring recruitment, oxygenation and "washing" of CO<sub>2</sub>.

When I talk about CO<sub>2</sub> "washing", I am referring to the English term, "wash out". In this case, "washing" should be understood as a system capable of eliminating CO<sub>2</sub> from the anatomical dead space.

A system that is significantly more efficient than methods with a leaky interface.

High flow ventilatory therapy is increasingly recommended in that type of patient who is borderline for NIV or invasive treatment.

The system works by administering a high flow of air (60-80 Liters/minute) and variable oxygen percentages.

This system usually uses nasal cannulas to administer the flows; these flows are very high, heated and humidified. They are able to resolve situations of respiratory failure in patients with maintained spontaneous breathing, who require a high percentage of oxygen administered. The presence of such a high flow guarantees good oxygenation even in patients who tend to keep their mouth open.

What does pharyngeal dead space washout mean?

Through the high flow system, the CO<sub>2</sub> present in the pharyngeal dead space is replaced by oxygenated air. This does not happen with any other device and favors a much faster recovery in case of hypercapnic respiratory failure.



Airvo™ 2, Fisher & Paykel Healthcare™

This in the image is one of the high flow systems, which has among its advantages that of generating a PEEP within the respiratory system.

The system is made up of a device to be connected to the flow meter and of course to the electrical current, a tube that is connected to the nasal cannula.

The nasal cannulas are of different sizes so that they can be adapted to any patient.

A tank is filled with sterile water, which has the function of humidifying and heating the flow sent to the patient.

This device in particular is referred to as a humidifier with an integrated flow generator that delivers heated and humidified high-flow respiratory gases.

It is used in spontaneously breathing patients. The flow can vary from 2 to 80 liters/minute.

The flow is delivered via nasal cannulas and generates a positive pressure directly proportional to the flow delivered.

The device has a water chamber that is filled via the sterile water bottle positioned at least 20 centimeters above the chamber.

The machine has a temperature that can be set to 31, 34 and 37°C.

The system can also be used via tracheostomy and facemask adapter.

It requires a flow meter that allows adjustment up to 30 liters/minute.

Oxygen regulation occurs via the flow meter and the value delivered is indicated directly by the machine.

So far, we have seen non-sealed oxygen administration systems, from now on we will see the sealed ones which will be our bridge towards invasive ventilation.



## Boussignac cylinder or mask



The Boussignac cylinder CPAP system is used in patients with respiratory failure.

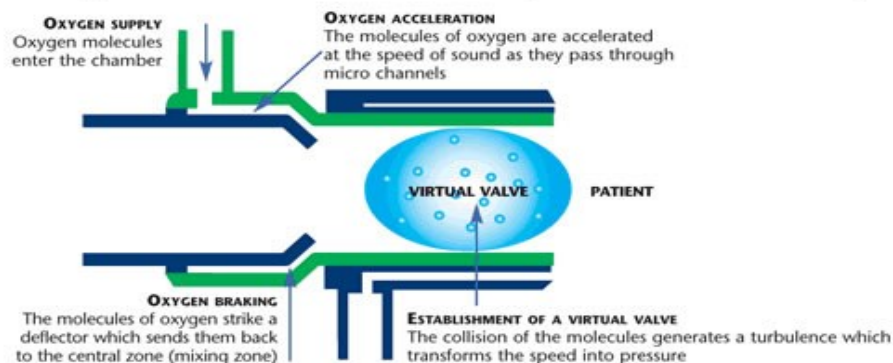
The improvements of the CPAP system are to increase functional residual capacity (FRC), reduce work of breathing (WOB), and improve oxygenation of arterial blood. It can be adapted to both masks and endotracheal tubes or tracheostomy tubes. It is composed of a small open hollow cylinder. (5.5cm x 1.3cm). When air passes through thin tubules to create CPAP it creates turbulence; it is this turbulence that generates a sort of virtual valve similar to a PEEP valve. The amount of this PEEP is regulated by the oxygen-air flow administered. You can view the amount of PEEP from the pressure gauge in the system.

It allows you to manage particular situations such as moderate hypoxic respiratory failure, atelectasis without having to resort to intubating the patient.

The operation is very simple: the gas particles that pass through these tubules are accelerated up to the speed of sound, thus generating turbulence. It is these turbulences that create a diaphragm or virtual valve between the patient and the outside. This valve allows the establishment of a CPAP system both in the expiratory phase and, thanks to the high flows involved, during the inspiratory phase.

The Boussignac system is a low dead space system because it is an open system, this allows the insertion of suction tubes and nasogastric tubes without reducing flows.

### **Boussignac CPAP works the same way as the turbines of a jet engine.**



Here you can see the valve and cylinder in section.

The Boussignac pressure must be monitored continuously and is dependent on the patient's ventilation and the flow of gas administered.

The system requires a flow meter that reaches up to 30 liters/minute which allows PEEP with values of 9-10 cmH<sub>2</sub>O.

The flow meters usually used reach a maximum of 15 liters/minute, allowing a PEEP of 5 cmH<sub>2</sub>O to be obtained.

Using only oxygen results in high FiO<sub>2</sub> greater than 60-70%, contraindicated in patients with chronic hypercapnia. Furthermore, it is not possible to correctly and continuously measure the administered FiO<sub>2</sub>. With a PEEP of 10 you get to a FiO<sub>2</sub> of 100%, in reality this is precisely the purpose of this system, to administer high percentages of oxygen. If necessary, smaller quantities of oxygen can be delivered using separate oxygen and compressed air flow meters. This is possible with two administration routes or via an AT fitting to be placed between two different flow meters. The oxygen consumption of this system is lower than similar systems that use the Venturi system. In any case, the oxygen delivered by the flowmeter will be the total value used by the patient. In these types of oxygen supply, if a mask is used as an interface, it is necessary to ensure its correct seal.

The Boussignac system does not use electrical or mechanical components, and this makes it very practical.

**The Boussignac system is an open system** able to maintain a PEEP even when using a fiberoptic. In this you will find that it is different from a similar system which uses the Venturi system in which a gas, usually oxygen, is accelerated in progressively thinner tubules until the pressure in the tubules is lower than ambient pressure, thus drawing in air from outside. In a Venturi system this air enters from the outside which generates another flow of oxygen-air gas, reaching flows exceeding 130-140 liters per minute conveyed into the mask or tube system. The internal pressure is regulated by the incoming gas and regulated by a spring valve.

**The venturi system is a closed system** with a large dead space.

You will find in some online works that the Boussignac "mask" is not a Venturi type system but uses the Bernoulli principle. To avoid any misunderstanding, let's immediately explain that the principal Bernoulli's theorem is a rule applicable to fluids. This rule generates the Venturi effect. We can conclude that both systems follow Bernoulli's theorem, but Boussignac does not use a Venturi-type valve, but a system more similar to aircraft jet engines.

The Boussignac cylinder, through the possibility of using oxygen or oxygen and compressed air, allows a very wide variety of oxygen/PEEP settings.

Always refer to the operating instructions of the devices available in your department.

## EasyVent Dimar type mask

An example of a high flow mask that exploits Bernoulli's theorem, but generating a Venturi effect, is the mask called EasyVent™ by Dimar™.



The Venturi effect is given by the property of a fluid, in this case oxygen, to accelerate its motion when the section through which it passes becomes thinner. But when the section of the duct becomes thinner, the flow becomes faster but the pressure is lowered. When this pressure becomes sub-atmospheric, if the duct is put in communication with the ambient air, the ambient air will be "called" towards that flow; the extent of this phenomenon is all the more important the greater the depression generated by the flow. By increasing the pressure of the inlet flow we would have an increase in speed in the thin part of the duct, the higher the speed the lower the pressure, the lower the pressure the greater the flow of ambient air will be attracted from the outside.

It is a system that applies a CPAP to the patient. A CPAP or Continuous Positive Airway Pressure system ensures that the patient is always ventilated in conditions of pressure higher than atmospheric pressure. In this case we obtain a level of PEEP, Positive, End Expiratory Pressure, higher than the external pressure always present throughout the respiratory cycle. This type of ventilatory approach will allow the patient's "ventilating" alveoli to be increased, keeping them open and will increase the patient's FRC, improving their respiratory performance.

This will allow the patient to reduce muscular effort and therefore the WOB or work of breathing. For the same reason, by increasing the number of open alveoli, we improve the patient's oxygenation and gas exchange, thus improving his ABG.

We must take into account three main parameters with this ventilation, pressure, flow and oxygen.

The pressure must be continuous throughout the respiratory cycle and stable

The flow must be high enough to compensate at any time for the negative flow generated by the patient during inspiration, this will be necessary to avoid alveolar collapse.

It is recommended to maintain a flow rate never lower than 60 liters/minute

In any case, a lower flow is acceptable if we obtain a stable PEEP.

Oxygen is a parameter that must be decided by the doctor and is adjusted to the patient's conditions and needs.

Since we are using a Venturi type system, these parameters will be linked together. Thus, a high PEEP will mean greater resistance and therefore less flow; a reduced flow can give an unstable PEEP with increased FiO<sub>2</sub>.

This system can deliver a flow of 120 liters/minute starting from just 15 liters/minute at the flow meter. The outgoing flow is usually 7-8 times greater than the incoming flow.

In this mask it is possible to adjust the PEEP via the PEEP valve and the FiO<sub>2</sub> following the indications in the table supplied and printed on the Venturi valve.

For this system we must use flow meters with a full scale of 15 or 30 liters/minute.

The system is equipped with a mask, a silicone mask holder, the oxygen connection tubes, a PEEP pressure gauge, the PEEP valve and the Venturi valve. The two gas connection pipes are named A in blue and B in white.

Usually, it is necessary to connect tube A to the flow meter set to 6 liters, place the mask on the patient with the PEEP valve completely open, then adjust the PEEP valve by checking the pressure gauge until the desired value that remains stable is reached.

In these conditions the patient receives, regardless of PEEP and flow, an  $FiO_2$  of 33%. By connecting the second tube, called B, we can enrich the  $FiO_2$  administered up to 100%.

Following the indications in the table, to obtain a  $FiO_2$  of 50% we must connect both tubes to two different flow meters with these settings: Tube A 10 liters/minute and Tube B at 14 liters/minute. With this system the losses are not so important, it is more important to be able to maintain a stable PEEP during the treatment.

This is the table with the settings to use when connecting two flow meters:

Conc. $O_2$ % approx.		40%	50%	60%
FLOWS $O_2$ Lt/min.		A + B	A + B	A + B
PEEP cm/ $H_2O$	5	7 10	6 18	5 26
	10	9 8	8 16	7 26
	15	11 6	10 14	9 26
OBTAINED FLOW approx.		60 Lt/min.		

## The helmet for CPAP



The CPAP helmet is used when the patient's condition does not yet require a type of invasive ventilation with an endotracheal tube. It is a system that we have seen often during the covid 19

epidemic, in a desperate attempt to help patients in their condition of respiratory failure. When we decide to put a patient on NIV with a helmet or diving suit, we must take into account the rebreathing phenomenon that is always present with this type of ventilation. To reduce this phenomenon, the flow to be used must be very high, usually greater than 30 liters/minute. High flows, however, bring with them a high noise that is very annoying for the patient, we are talking about levels above 90db. To give better comfort to the patient, we can use earplugs. Furthermore, if possible, we use smooth connection pipes inside to reduce turbulence and noise formation. It is possible to connect the CPAP helmet to dedicated devices or to a mechanical ventilator. Not all mechanical ventilators are capable of generating such high flows; for this reason, specialized companies provide systems to be connected to the oxygen distribution system, capable of generating flows of up to 180 liters/minute. One of these is the Harol™ 9293/D system



This system uses a Venturi valve which, through a reduced flow of oxygen, manages to generate a flow that is more than sufficient for correct ventilation with a helmet.

Here we will dedicate ourselves to use via a mechanical ventilator. Usually, this type of interface is well tolerated by the patient, but it is necessary to stay as close to him as possible, especially in the initial phase of ventilation. With this type of ventilation, the patient is able to speak and look around himself without major difficulties, this makes it more bearable than a face mask throughout the ventilation period. There are different types of CPAP helmets with different characteristics, but they are all equipped with a transparent plastic container, the actual helmet or diving suit. Then there is the lower diaphragm which has the task of adhering perfectly to the lower part of the neck, it is he who guarantees the tightness of the system; It is elastic so as not to allow air leaks. The entire system has attachments to which straps must be attached which must pass under the patient's armpits to keep the helmet in the correct position. The latter, especially in the phase of inspiratory delivery by the ventilator, will tend to go upwards. In some helmets there is an inflatable cushion to be positioned around the patient's neck. This cushion holds the helmet during the lifting phase, allowing the axillary straps to be released and giving more relief to the patient. In fact, even if well padded, the straps can cause irritation or injuries. The helmet also has an attachment for the ventilatory circuit. Here too we can have helmets with a single attachment and a dedicated expiratory valve positioned in another hole in the same helmet or helmets that

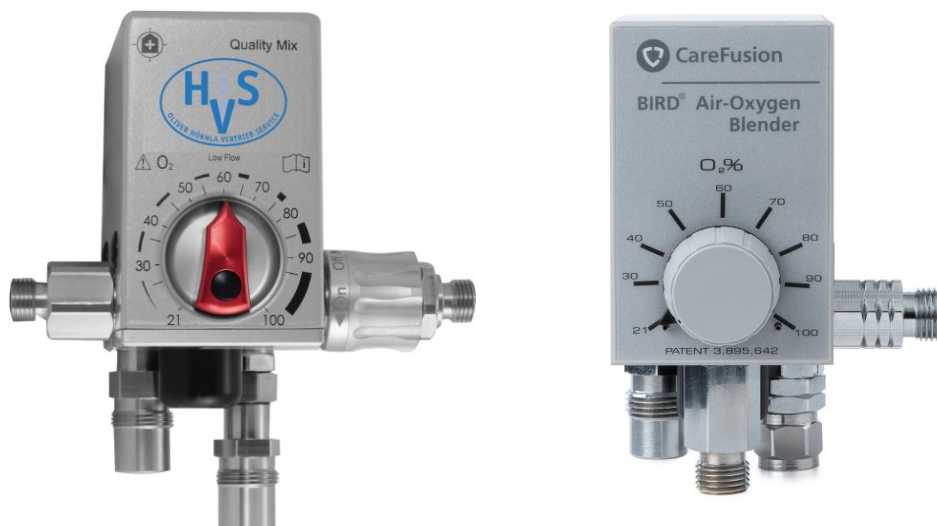
allow connection to circuits with inspiratory input and expiratory output. There are auxiliary holes through which to pass the nasogastric tube and, in some models, actual portholes that allow ventilation to be suspended for a moment and allow the patient to drink or freshen his face. Usually, the helmet is thought of as a second choice device compared to an NIV with a mask. But the data tells a different story: according to these data, NIV with a helmet performs slightly better when it comes to survival in patients. Probably the fact that the mask is less tolerated also leads to reduced ventilation times or discontinuous ventilation.

The helmet is therefore a type of interface that allows both an increase in the administered  $\text{FiO}_2$  and the generation of a respectable positive pressure with PEEP; Furthermore, at adequate flows, it is not affected by the phenomenon of  $\text{CO}_2$  accumulation, contrary to what one might think. Furthermore, it has on its side the greater tolerability even for patients who cannot tolerate other devices, such as a face mask.

### CPAP with pressurized gas

On some occasions, CPAP delivery systems are used without a ventilator or auxiliary equipment, but only via a simple pressure and oxygen regulator. In some cases, they allow the use of oxygen and compressed air, managing to mix the two gases to obtain, using a simple knob or flowmeters, the desired  $\text{FiO}_2$  and PEEP. They are very practical and easy to use, so they can be simply attached to an IV pole. For this reason, they have their greatest use in normal inpatient departments. They must be used with a tight face mask of any model. There are high flow or low flow models.

I insert two images of products of this type:



They had their golden age and fame, but the lack of alarms, and therefore the need for continuous monitoring of patients, relegated them to certainly not being the first choice in the management of respiratory failure responsive to CPAP.

They can be used with different types of interfaces, particularly when there is no ventilator available.

## NIV

### Tight face mask and CPAP systems



Facial mask



Total face mask



CPAP Mask



CPAP Nasal

These above are some of the models of full-seal CPAP masks, they are interfaces that must be adapted to the person's face. For this reason, they exist in different sizes; the choice is made using

a template usually present in the packages or by bringing the mask closer to the face, choosing the most suitable one. The choice of size should not be underestimated: it is one of the necessary precautions to obtain an optimal result during ventilation.

Too small will be annoying for the patient and poorly tolerated; too large will offer too much room for air leaks, thus invalidating the maintenance of positive pressure within the airways.

The purpose of these masks is precisely to maintain a positive pressure throughout the ventilatory cycle, favoring alveolar recruitment.

Usually, simple face masks are fixed behind the neck using an elastic called "nuchal". The adhesion of the mask must be perfect, for this reason they are equipped with an inflatable cushion that is placed between the rigid part of the mask and the patient's face. Even if regulated and managed correctly, they can cause injury to the patient, which relegates them to a secondary choice. In fact, over time, the various companies producing the devices have indulged in trying to solve some of these problems, marketing dedicated and certainly more tolerated masks, and total face systems that are much less "demanding" for the patient.

Remember that these are sealed systems, and the exhaled gases are eliminated through the same hole through which the "fresh" air arrives. This generates a certain amount of rebreathing that we must take into account. They are usually connected via catheter mount to an antibacterial filter and humidifier and then to the circuit.

To use these devices the patient must be particularly cooperative, otherwise we only risk wasting time. Unfortunately, finding yourself with a mask tight on your face is not the best and often this type of ventilation can only last a few hours. Precisely because it is a non-bloody ventilation, it is called non-invasive ventilation.

In English the term is Non Invasive Ventilation or NIV

## But what is the NIV really?

By NIV we mean non-invasive ventilation. First, however, we need to say what we mean by "invasive". We perform invasive ventilation using an endotracheal tube, a tracheostomy tube or a laryngeal mask.

You would think that everything else falls into the category of NIV, face mask, CPAP mask, helmet, high flow etc.

I think a small clarification is in order:

In the material you find around, books, research, sites, videos and chat discussions on ventilation, NIV systems are often considered and categorized in different ways.

For example, you find it written that the Venturi mask generates positive pressure, that CPAP is only NIV, or that a type of NIV ventilation can also include nasal cannulas.

In this context, it is difficult to give a univocal and precise definition, and it is certainly more important to understand the general concept than to waste time understanding whether a device has been precisely categorized.

We find ourselves in a field in which "poetic license" also plays an important role.

I won't tell you what you will find by looking at the ventilation modes; You'll really be tearing your hair out there! You, certainly not me who no longer have any!

Apply this concept to all these pages and, when in doubt, go and check. I don't have to do everything?!



In reality, even among professionals there is a certain uncertainty about this definition. In fact, you will find manufacturers of high flow machinery labelling their system as an NIV system.

If we go backwards, we find that for NIV we must consider any device that provides ventilatory support to the patient. The term NIV, in fact, derives from the less used **NIMV**, where the “M” indicates mechanics. Therefore, the term NIV indicates **non-invasive mechanical ventilation**, this would exclude all high flow systems, but also all systems dedicated to sleep apnea which are not real ventilators. Yet all these systems are always placed in a vast NIV world. If we really have to insert a limit that defines what is meant by NIV, we can on the one hand insert intubated patients, with tracheostomy tube or laryngeal mask. On the other, all those systems that allow maintaining a positive pressure during the ventilatory cycle.

Another unclear thing is the association that is often made between NIV and CPAP. While it is true that CPAP can be considered NIV, when performed via an endotracheal tube it is no longer so! So, the answer is NO: CPAP is not NIV and NIV is not CPAP, they are not synonymous or even related!

Up to this point, we have helped (I hope!) patients who are able to take spontaneous breaths in complete autonomy and, although helped, they carry out the main respiratory work.

## Part Four: Things Get Serious!!!

In the first parts of this "little job", we first analyzed breathing in a normal subject: in the second part, the help we can provide with only oxygen and suitable aids; and, in the third part, to that help we added positive pressure and therefore the recruitment of the alveoli, managing to resolve medium-level respiratory failure.

When a patient benefits from a non-invasive treatment, we have obtained an **excellent result**. The achievement of this result passes through different paths that must come together to arrive at a positive result. Thus, the rapid "early" approach to NIV ventilation, the collaboration of the patient, the correct management of the parameters, the devices to be used and last but not least the careful monitoring of the patient are all links in a chain that will lead to potential success. What I didn't tell you in the previous part concerns the patient: he is the most unpredictable parameter, sometimes very collaborative, sometimes with an oppositional attitude. Often, with this type of patient, we have a first phase in which respiratory fatigue makes itself felt and puts the subject in a position to "accept" any compromise. This is due to the conditions which are not good and which he himself perceives as no longer tolerable.

Something not so rare happens later: the same patient as before, who would have accepted anything as soon as he improves, changes his attitude. If it is true that tolerating NIV in case of exhaustion requires making enormous sacrifices, as soon as NIV begins to have positive effects, the patient, having regained a minimum of functionality, implements this attitude of renunciation and abandonment. Everything that previously "had to be done" becomes superfluous, their reasoning is easily understandable, breathing better than before, they think they can dismantle the mess and the puppets and return to a normal life... but that's not the case!

Try to follow me carefully because here the doctor and the nurse in particular intervene who, having transformed into the patient's "personal trainer", must encourage him to continue that therapy, explaining its importance and above all that it is not the first signs of improvement that determine the end of the NIV.

You must persist and be adamant. If necessary, you can make an NIV "gentler" by asking your doctor to change the settings. To be effective, ventilation in NIV must be of appropriate duration and without continuous interruptions which would compromise all the work done on alveolar recruitment.

I would like to explain how I organized this part:

This part concerns a very important and at the same time interesting part.

It took me a lot of time just deciding the order, what to include and what not to include.

I checked other texts and works on ventilation, but the classical academic approach is just not my style and certainly not within my reach!

Based on what I chose, here's what awaits you in this section:

- First, I define invasive ventilation and briefly explain the types of mechanical ventilator, how they produce the air flow to generate positive pressure; nothing complicated, don't worry.
- I then explain how ventilators are able to deliver the right amount of oxygen and how they receive it.

- Afterwards, I will show you how the ventilator "talks" to us through the display and shows how we breathe; furthermore, let's see the pressure, flow and volume curves together, just a pinch though!
- Through the curves we can analyze concepts such as the pressures involved, PIP, average pressure, Plateau pressure, driving pressure and PEEP, rise time. We see them one by one and break them down into their important parts, inserting some curiosities and why they are so useful.
- Now that we know those curves and what their angles, lines and inclinations indicate, let's do a little gossip about their ratios and what they indicate.
- After these concepts, I briefly explain what mechanical ventilation is and how we can divide it and therefore:
  - Amount of assistance.
  - Volume or pressure control
  - Open or close loop
  - How to divide the ventilatory modes and why all these damned acronyms exist!
  - Chatburn's taxonomy
  - Let's talk about the main ventilation modes
  - Weaning

Don't worry, we've finished everything in another three pages!

## Invasive mechanical ventilation

In these two pages we must necessarily talk about ventilators, giving a small overview of their characteristics. I preferred to insert this part here, even if I could have talked about it in the section dedicated to non-invasive ventilation. In fact, many of the devices listed above, such as helmets and face masks, can also be used via a mechanical ventilator.

It is precisely this "may" that made me inclined to include the topic here, where I will talk exclusively about mechanical ventilation.

We decide to place a patient on mechanical ventilation following numerical data that may raise alarm bells, such as:

- **Respiratory rate > 30 breaths/minute**
- **Arterial oxygen saturation < 90% with FiO<sub>2</sub> > 0.60**
- **A pH < 7.25**
- **PaCO<sub>2</sub> > 50 mm Hg, unless chronic and stable**

But the decision to start with mechanical ventilation is not based only on numerical criteria, but on clinical judgment that considers the patient's entire condition; this task falls to the doctor.

Now, as I like it, let's start from scratch by explaining what is meant by Mechanical Ventilation.

To distinguish it from normal ventilation, the name mechanical is given because mechanical equipment is used to generate a flow of "air" in and out of the respiratory system.

Are mechanical ventilation and automatic ventilation the same thing? No.

You will often hear these two ways of defining ventilation and, to most, they may seem synonymous, but this is not the case. The correct wording of the machine is "mechanical ventilator" and it is he who can provide automatic ventilation by executing the set parameters.

Always refer to it as mechanical ventilator and mechanical ventilation.

In reality the ventilator, especially the recent ones, is a mix of mechanics and electronics, in this sense it would be more correct to define it as a "mechatronic" type device. By mechatronics we mean a system or device that combines mechanical and electronic components.

...But I really can't see you writing that the patient is connected to the "mechatronic ventilator"!...

A ventilator has the ultimate purpose of moving air and therefore generating flow, thus creating artificial inhalation and exhalation.

To do this it requires internal components that must generate this flow and sensors, regulators, displays and safety systems that allow this flow to be obtained safely for the patient.

The way they generate this flow is one way to divide ventilators into different categories; so we have mechanical ventilators used in intensive care, portable ventilators and portable ventilators that use a gas as a system to generate this famous flow.

Let's look at these systems briefly.

## How the ventilator work

Intensive care or hospital ventilators use the pressure of a gas to carry out their work and therefore generate this blessed flow. To do this correctly they use electricity.

The main force used to work is the pressure of the compressed air and oxygen they are connected to. They need high and stable pressure, and this is guaranteed by the centralized oxygen and compressed air distribution system. Usually, the "driving force", i.e. the force that moves the mechanical part responsible for generating the flow, is given by the pressure of the compressed air. If this is missing, the ventilator can carry out its work using the pressure given by the oxygen.

Therefore, oxygen is usually only responsible for delivering the set  $FiO_2$ . In the event of a lack of compressed air, it performs both the work of "driving force" and the supply of the required  $FiO_2$ .

And electricity? You may say, a ventilator of this type can hardly ventilate a patient in the absence of medical gases. Electricity has the task of managing the entire control and management part, sensors and everything that guarantees correct operation.

This type of ventilator generates flow via a pneumatic system.

A pneumatic system is a system that uses compressed air (or other gas) as a means of transmitting energy to operate machinery and devices.

They are ventilators dependent on medical gases.

Then there is portable ventilator, which use electricity as a "driving force" through the use of a turbine. In this case, by turbine we mean a system capable of taking appropriately filtered ambient air and transforming it into the flow necessary for patient ventilation. The oxygen in these ventilators is supplied via a suitable frusto-conical connection and has no role in the flow generation, but only in the value of  $FiO_2$  administered.

Some define the turbine as the driving force of these ventilators, for us it is not important to define exactly who does what. Without electricity that turbine doesn't move.

They are ventilator dependent on electricity, it is in fact what makes the turbine work. They are used in domestic or hospital environments, where there is no centralized medical gas connection.

In a separate category there are portable ventilators for local use, such as the Oxylog<sup>TM</sup> from Dräger. It is a hybrid, as it uses the pressure of an oxygen source as a driving force, be it a centralized connection or a cylinder with a suitable connection. The electrical part only deals with controls, sensors etc. In practice, it is a hospital-type ventilator without a compressed air connection, but also portable. Or rather, **portable**, because with it you will always have to carry an oxygen tank with you!

TYPE	FLOW GENERATOR	EMPLOYEE
Hospital	Compressed air or oxygen	Compressed air, oxygen and electricity
Portable	Electricity	Electricity
Oxylog	Oxygen	Oxygen and electricity

As you can see, none of these ventilators can work without electricity!

They all have internal batteries that allow operation for even long but not unlimited times. I forgot, in the event of a power cut they simply don't work.

...The only “mechanical” ventilator that works without electricity is the Ambu bag or the “back and forth”! Here, the “driving force” is you!

## The regulation of FiO<sub>2</sub>

After this basic description of how ventilator work and in particular how they generate flow, let's try to understand how they regulate FiO<sub>2</sub>.

Ventilators that have access to a high pressure oxygen supply, be it a cylinder or centralized oxygen, via the high pressure DIN connection, always have all the oxygen they need available. An internal mechanism takes care of mixing this pure oxygen with the ambient air to generate the desired FiO<sub>2</sub>. These ventilators can deliver FiO<sub>2</sub> from 21% up to 100%; it is the ventilator that can regulate the percentage of oxygen delivered.

**The pressure connection ventilator can ventilate 100% oxygen, he can adjust this percentage according to our choices!**

Here are the most popular connections for oxygen:



## UNI 9507 COUPLING AFNOR NF S 90-116 COUPLING

There are also different fittings scattered around the globe!

The situation changes for ventilator s that have a non-pressure connection; these are "given" oxygen via a normal truncated cone fitting like this:



This is a low pressure oxygen adapter. Called 1/8" male to 1/4" hose adapter.

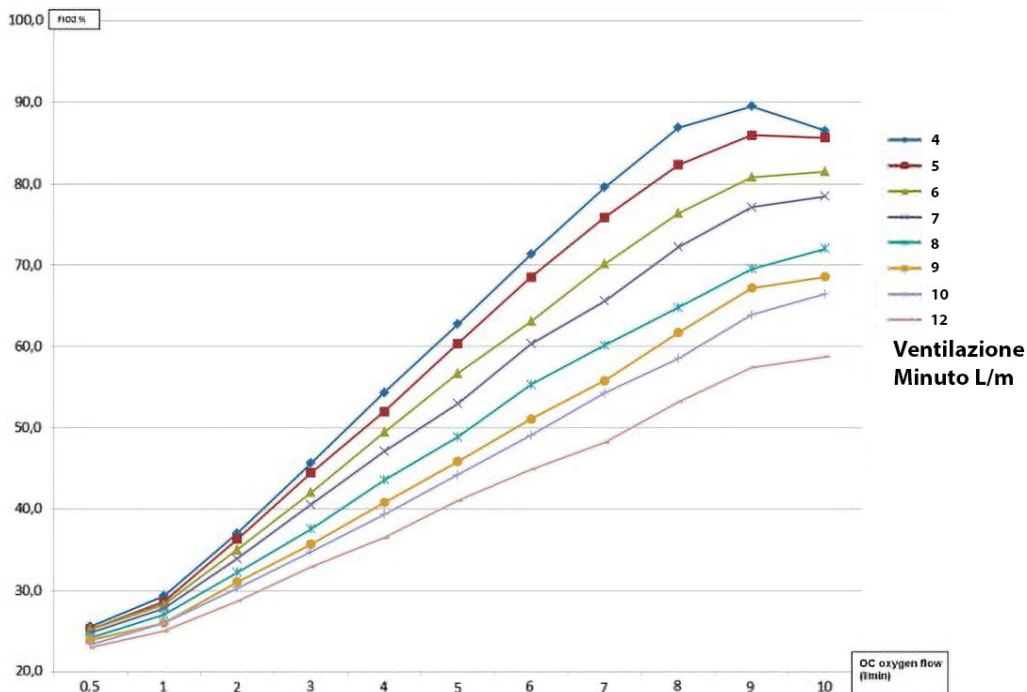
In this case, it is not the ventilator that regulates the percentage of oxygen delivered, but the operator by regulating the flow in liters/minute using a flow meter.

Some of these models allow a real-time reading of the FiO<sub>2</sub> delivered to the patient, others do not have this functionality. In this case, how do we define the percentage of oxygen delivered?

The rule of 2 liters = 28% still applies, No! In this case it doesn't apply, it could be an approximation, but not even that, it could be a **big mistake!**

Each device differs from the others in the oxygen enrichment mode. It is therefore not certain that a portable ventilator with 3 liters of oxygen delivers the same FiO<sub>2</sub> as another model of ventilator with the same liters/minute of oxygen.

This is a diagram showing how FiO<sub>2</sub> rises based on the liters per minute administered into portable ventilators:



As you can see, we do not have a single liters/minute FiO<sub>2</sub> correlation delivered, but several lines. This is because everything depends on the minute ventilation that the patient develops.

I'll make it short for you: if I have a patient with a minute volume of 6 liters and we give 4 liters of oxygen per minute, we are approximately close to 50%  $\text{FiO}_2$ .

If minute ventilation increases,  $\text{FiO}_2$  decreases.

Therefore, the  $\text{FiO}_2$  administered to the patient in these ventilators is dependent both on the set inlet flow but also on the minute volume developed by the patient.

Now one would think that a patient who has a minute volume of 6 liters, if we input 6 liters of oxygen, will have an  $\text{FiO}_2$  of 100%! Yes, yes, believe it, and I'm skinny!

Absolutely not! The theory in this case is very different from reality and, as you can see in the graph, it will have an  $\text{FiO}_2$  of perhaps 55%, in some models a little more.

Why does this happen? For the air-oxygen mixing system which always takes ambient air, any flow is delivered.

Remember that this is a diagram for only one ventilator model. Other models may also differ quite a bit.

Secondly, if you have an oximeter available, use it to check the  $\text{FiO}_2$  actually delivered.

**Ventilators with a non-pressure oxygen connection do not regulate the percentage of oxygen. We do it via the flow meter!**

Even at very high flows with low minute volumes, 100%  $\text{FiO}_2$  is not achieved!

A little advice: each portable ventilator has in its characteristics the maximum flow, expressed in liters/minute, which can be administered via that device. Always follow the directions.

Be careful, because some models have different indications if used inside the transport bag!

Reading the manual is always a good idea, believe me!

What is  $\text{FiO}_2$

...hey, did you notice that you wrote a lot of inaccurate things? Did you reread your "little work" before distributing it?

I know what you're referring to, it's a technique I often use in my "jobs" to see if you've been paying attention!... In fact:

Indicating  $\text{FiO}_2$  as the percentage of oxygen is an unforgivable mistake, perhaps!

A percentage indicates how much oxygen is present compared to a maximum which is precisely 100%. So, in the ambient air we have 21% oxygen.

A ventilator that works with only ambient air, therefore without an increase in oxygen, works with 21% oxygen. If we want to describe this quantity with a system other than the percentage, we must also adapt the numerical data.

Thus, the  $\text{FiO}_2$  indicates the **inspired Fraction of Oxygen**, since as a fraction its maximum value is to be considered 1. In this way if I want to indicate the  $\text{FiO}_2$  of the ambient air I would have to write a  $\text{FiO}_2$  of 0.21.

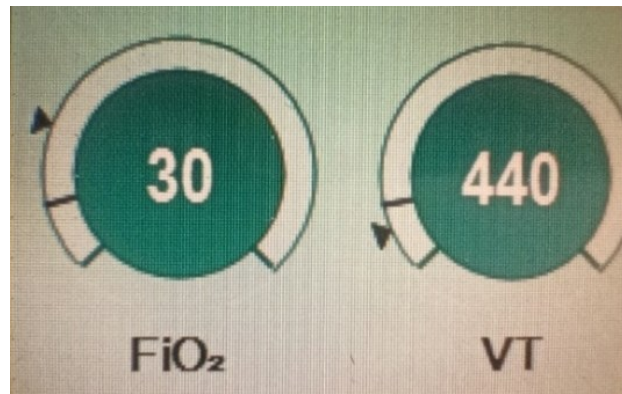
I deliberately made this mistake so that it is clear what one value indicates and what the other indicates.

So, when I ventilate a patient with 100% oxygen I am giving them an  $\text{FiO}_2$  of 1.

At 60% oxygen it will have an  $\text{FiO}_2$  of 0.6. Now that the concept is clearer, you will realize that many ventilators indicate a suitable value for the percentage under the wording  $\text{FiO}_2$ . We can consider it an error if you want but know that it is not uncommon to find  $\text{FiO}_2$  written and the % symbol.

It will not be difficult to understand that 60 will indicate 60% oxygen or 0.6  $\text{FiO}_2$ .

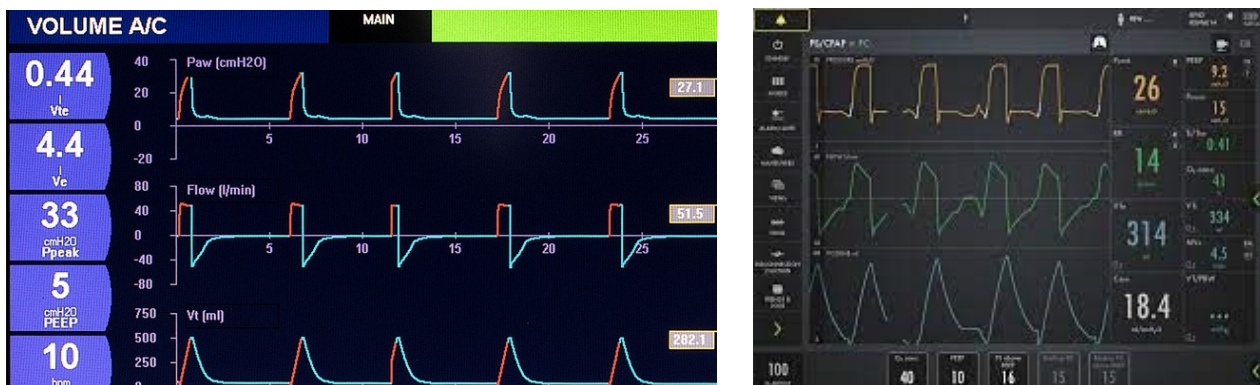
This below is a Dräger™ in my department, the first one I've found!



As we wanted to demonstrate,  $FiO_2$  30! I repeat, this is not a mistake! The important thing is to understand each other!

## The ventilator Curves!

But what did you understand? I'm not referring to the design work of the ventilator and their sinuous shapes! I'm referring to these:



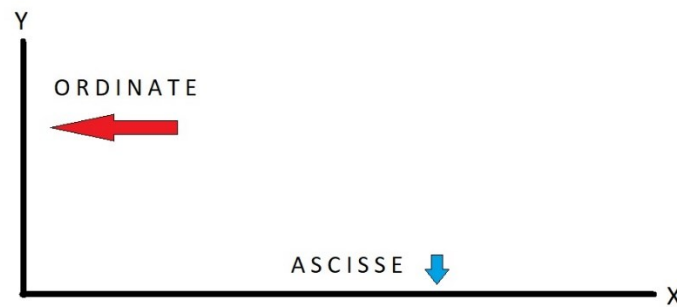
In order from top to bottom, the first refers to pressure, the second to flow, and the third to volume.

To a non-expert look, these lines, or waveforms for those who know English, mean nothing at all, looking at them or at the washing machine spinning makes no difference!

Each of those curves represents the patient's breathing, and they do it accurately. Thus, on the ventilator display you will find both the graphical representation and the numerical representation of the useful values. The clinician knows well what the numerical values indicate and knows how to interpret the waveforms equally well in any ventilatory mode. Through these it is possible to see if the given settings are having the desired effect or if there are problems.

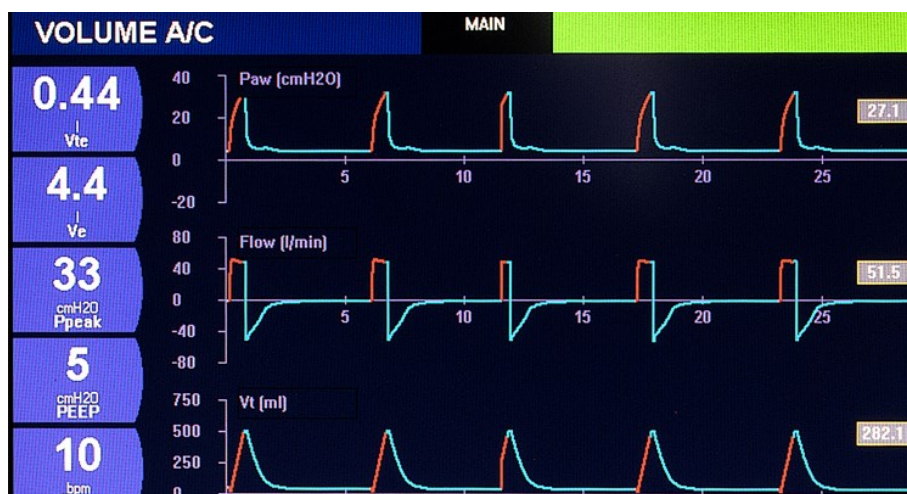
Thus, one can notice any machine-patient asynchronies and problems relating to the ventilatory system. In this case, by ventilatory system, I mean everything, from the ventilator to the gas transport system from the ventilator to the patient, to problems that affect the patient himself. The graphical representation is based on a system that describes the measured values on a time scale. Just to clarify this below:





Therefore, the time value is indicated on the abscissa line, the value depending on the type of curve is indicated on the ordinate line. Thus, the first curve, the pressure curve, will have the pressure value expressed in cmH<sub>2</sub>O in its ordinates. The second, or central one, the flow one, will have the flow value indicated in liters/minute in the ordinates. Furthermore, it is also the only one of the three that also works in the negative part of the ordinates; it therefore has positive and negative values. The other two indicate only positive values....almost always! The last curve has the volume value indicated in ml in its ordinate. Each of these values is indicated as a function of time. Each of the instantaneous values indicated, at any point of the three curves, can be represented by a numerical value.

This is a typical ventilator screen:



Usually, the screen of any ventilator looks like this: three graphs that always have the same layout. From top to bottom, they indicate the trend via a pressure, flow and volume curve. Here you see them referred to as:

PAW	expressed in cmH <sub>2</sub> O	Pressure
Flow	expressed in liters/minute	Flow
Vt	expressed in ml	Volume

You will find these names or symbols on the ventilator:

Pressure = **P**

Flow =  $\dot{V}$

Volume =  $V$

Please don't confuse the curves  $\dot{V}$  of flow and  $V$  of volume.

Pressure	PAW	P
Flow	Flow	$\dot{V}$
Volume	Vt	V

In the vast majority of cases, the pressure wave is positive, rarely falling below zero.

As you can see from the graph, we are in the presence of a cyclical system due to the alternation of individual respiratory acts.

A single breath is this one below:



The red part up to the top indicates the inspiratory phase, the cyan part indicates the expiratory phase.

These curves indicate exactly:

Pressure: Indicates the pressure that is used to move the volume of air.

Flow: Indicates how this amount of air enters and exits.

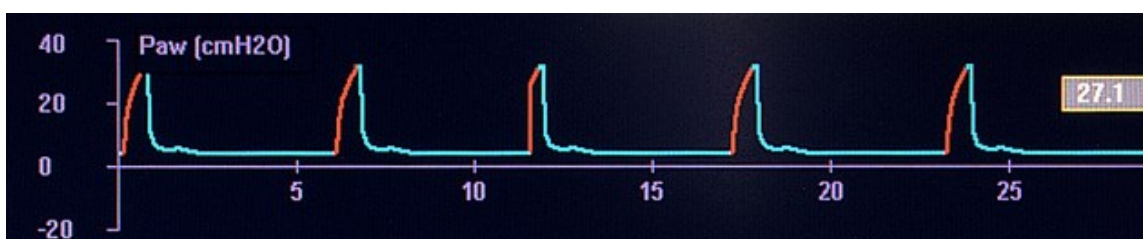
Volume: indicates how much air enters and exits the lungs.

Now that this "washing machine" seems a little clearer to us, let's try to describe the shape of each individual sector, starting from that of the pressure, what that shape indicates and why it is so important to have them available.

I forgot to tell you that this type of curve in a Cartesian system, i.e. made up of abscissae and ordinates, are defined as scalar.

Below is a brief description of the curves and how they can appear in different ventilation conditions. You can find the details of these topics later where I talk about pressures and all their related things!

### The pressure curve

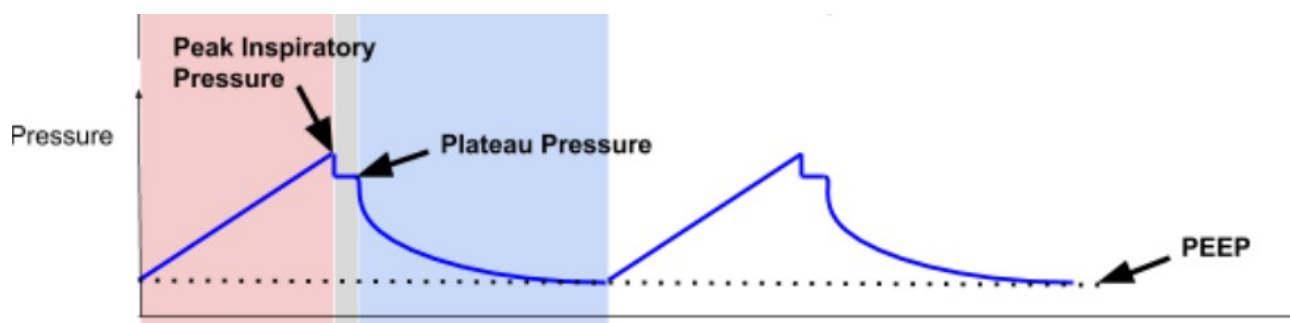


The curve representing the pressure, which in case you hadn't understood, is the first at the top "tells us" many things about the characteristics of breathing and indicates: the peak inspiratory pressure, the plateau pressure (when present or visible), the PEEP, the Driving Pressure and the mean pressure.

The shape of the wave indicates how this pressure was delivered by the ventilator and how it was able to propagate based on the pulmonary characteristics of compliance and resistance.

Put like this, it seems like a walk in the park, but there are so many parameters at play to consider that without the help of increasingly advanced systems it would be impossible to effectively manage a patient on mechanical ventilation.

Let's take this curve:



Here you can clearly see the pressures indicated by a pressure curve. The highest point reached is the PIP, the transition between inhalation and exhalation is a moment in which we have no air flow and is the plateau pressure (not always visible).

The lowest point represents the set PEEP and is also called the base line or baseline.

Here is a more detailed explanation:

**Peak inspiratory pressure(PIP):** it is the maximum pressure reached in the airways during inspiration. It is determined by the pressure set by the ventilator, the resistance of the airways and the compliance of the lungs.

**Plateau pressure(plateau):** it is the pressure reached in the alveoli at the end of inspiration. It is determined by the pressure set by the ventilator, and the compliance of the respiratory system (chest wall and lungs).

**Relaxation pressure:** it is the difference between PIP and plateau. It is determined "indicatively" by the resistance of the airways.

**Driving pressure:** is the difference between the plateau pressure and the PEEP.

**Positive end-expiratory pressure(PEEP):** it is the pressure applied in the airways at the end of exhalation. It is used to keep the lungs open and prevent respiratory failure.

In a patient with high airway resistance, the difference between PIP and plateau will be greater. This is due to the fact that the ventilator must apply greater pressure to overcome airway resistance and achieve the alveolar pressure necessary to ensure the desired tidal volume.

Still in the pressure line, if we see a small deflection below the PEEP value, we know that the patient is triggering a spontaneous breath. It is easier to see with the pressure trigger than with the flow trigger. If we have a deflection not followed by a full breath, the ventilator has not

responded adequately to the patient's request. It is a missed trigger problem, its sensitivity may not be adequate for the patient's effort.

In the pressure curve, "activity" usually does not drop below zero and usually stays up to the PEEP value (unless very excessive patient effort, also known as work shifting).

All that is "included" within that curve is the mean airway pressure.

### The flow curve



If you look at the flow line, you will see the inspiratory quota at the top and the expiratory quota at the bottom.

If you see in the flow line that there is a gap on the baseline and the flow does not return to zero, then it means that your patient is trapping air (air trapping or auto-PEEP).

Here you can reduce the frequency by also giving more time to the expiratory act, you can change the I:E ratio. If you choose to decrease  $V_t$  or RR you also reduce ventilation and therefore the removal of  $CO_2$ . Increasing the flow usually solves the problem.

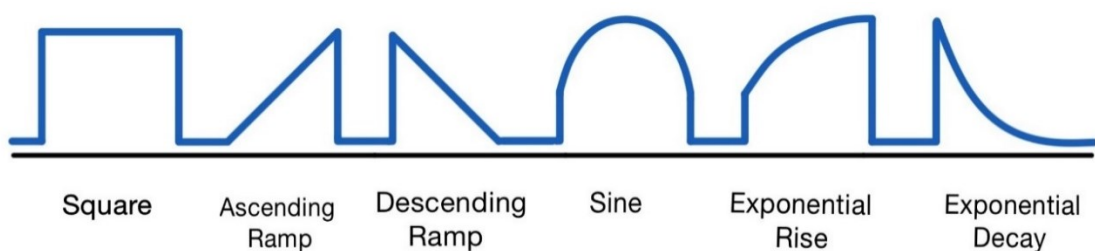
### The volume curve



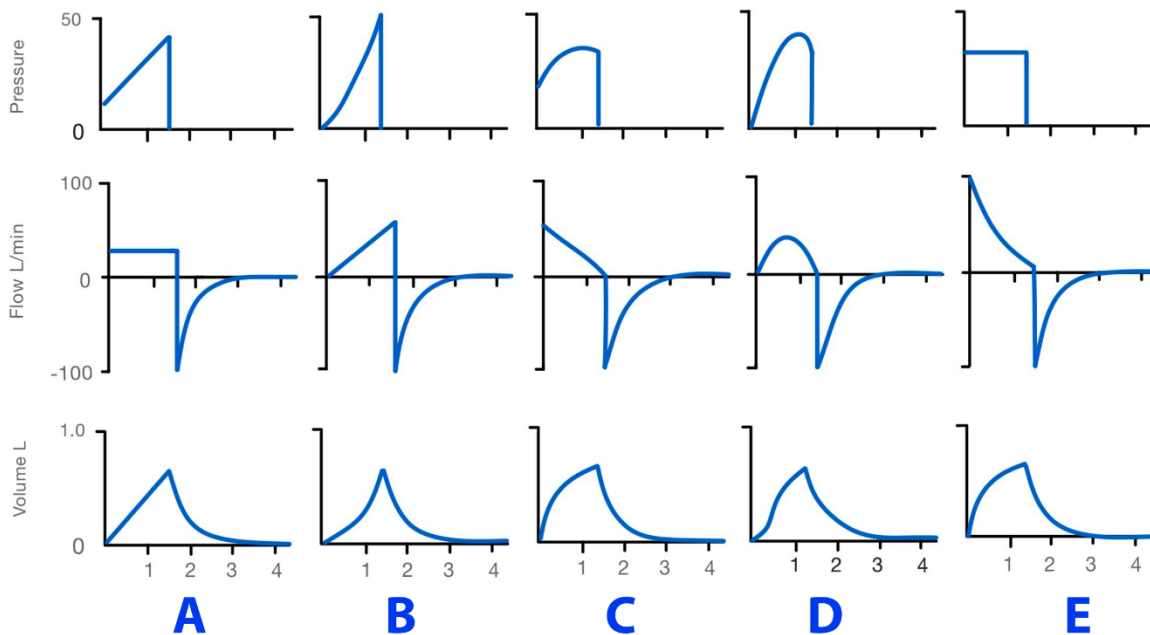
The third line, the volume line, is only positive and the peak indicates the  $V_t$  which then returns to zero.

If instead we have a problem in the volume line, and it does not return to zero. In this case you may have an air trap or have a leak in the system. However, if you look at the flow line and it returns to zero then it is not an air trap! Easy, right?

Waves can take different shapes, but their basic characteristics refer to these:



Square, ascending, descending, sine, exponential rise, fall or exponential decay.



These, very briefly, are the types of curves that we can have on display in the ventilator. They are usually visible in the pressure and flow part, the volume part is often like the one shown above. They indicate how that specific volume of gas is introduced into the pulmonary system. Let's look at them briefly:

The square shape indicates that the set value is reached almost instantly. So if we set a certain pressure, the peak represented by the upper part will be reached immediately and then decay equally instantaneously.

The same goes for flow. In fact, if we use a pressure-controlled ventilation mode, we will see a square-type curve in the part relating to pressure.

In volume control ventilation (volumetric or flowmetric), we would have the square curve in the second section of the display relating to flow.

Thus, square indicates a rapid rise, a stabilization at the required value and a rapid release of flow or pressure. It is a system that is not very comfortable for the patient, but it is also one that guarantees higher peak airway pressure but less mean airway pressure than pressure controlled modes.

When the shape of the curve takes on the shape shown in the image, it is defined as an "ascending ramp". In this case, the pressure or flow target is reached gradually over time. We therefore see the values rise to the peak in a more gradual manner. We decide how gradual this climb is by modifying the ramp values or the inspiratory time.

We can virtually have ascending curves with any possible slope.

The third curve in the photo, or "descending ramp", indicates a way of reaching a set pressure or flow quickly, and then reducing immediately but gradually. The same thing applies as before, we can also modify the level of decay or reduction of their values here. The fourth curve, or "sine", indicates a sinusoid shape and represents the patient's typical spontaneous breathing. As you can see in this case, we have an exponential increase with a mirror descent. This is absolutely the most physiological type of curve possible, but it is also difficult to implement using a mechanical

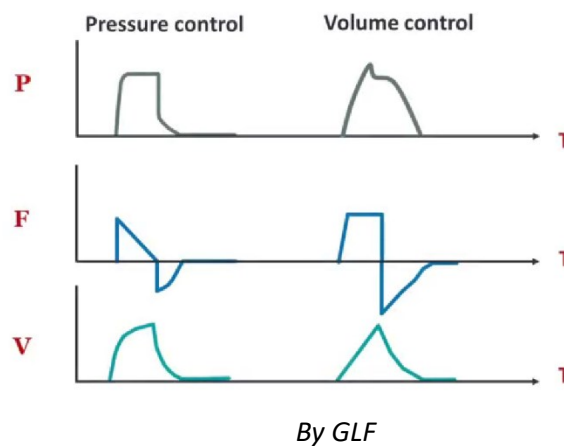
ventilator, there are too many differences between spontaneous and mechanical ventilation, remember?

The last two curves, “exponential rise” and “exponential decay”, represent a middle ground between an ascending wave and a spontaneous exponential system.

In this section of the curves, we are focusing only on the inspiratory part of the flow curve, therefore its upper part.

Small obvious curiosity, but it's better to never take anything for granted! Those are ideal curves, and you will rarely encounter them. In reality they will be similar to those but spoiled by many elements.

Just look at any ventilator in any mode to notice the difference. They will be similar, sometimes very similar, but not perfectly identical. Here is an example:



## The loops

Loops are graphs that represent the relationship between two mechanical ventilation variables, typically pressure and volume. They can be used to evaluate lung function and to monitor the effectiveness of ventilation.

The most common loops are:

**Volume-pressure loop (VP):** represents the relationship between airway pressure and tidal volume. It is the most important loop, as it provides information on lung compliance, flow resistance and plateau pressure.

**Flow-volume loop (FV):** represents the relationship between inspiratory flow and tidal volume. It may show a flow limitation, such as an airway obstruction.

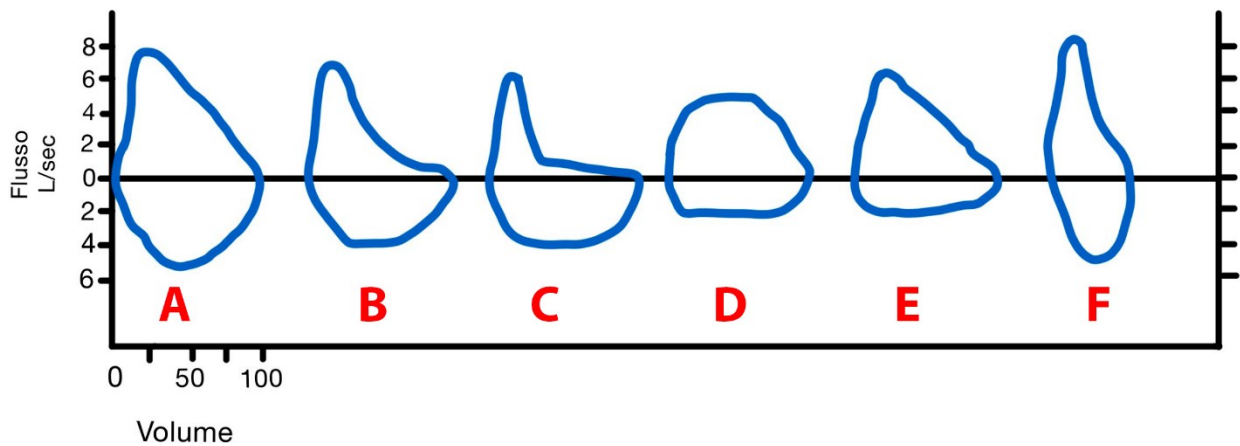
**Pressure-flow loop (PF):** represents the relationship between airway pressure and inspiratory flow. It is useful for evaluating flow resistance.

Loop analysis can provide important information about patients on mechanical ventilation.

For example, a VP loop that shows reduced lung compliance may indicate lung disease or lung damage. A VF loop showing flow limitation may indicate airway obstruction.

Loops can also be used to monitor ventilation effectiveness. For example, a VP loop that shows an increase in plateau pressure may indicate that the patient requires an increase in PEEP.

In this image below how, a loop can vary based on the patient's condition.



By GLF

- A:** Normal
- B:** Mild obstruction
- C:** Chronic obstructive disease
- D:** Major obstruction of airways
- E:** Variable airway obstruction
- F:** Restrictive pathology

The small curiosities that I have included in the description of the curves have the sole purpose of making you hungry for knowledge and to make you understand the power of the ventilator visualization system.

### How the air gets from the ventilator to the patient and how it comes back

Each ventilator has two connections, I am not considering monotubes here, one is the one that sends the air to the patient and the other recovers it from the patient. They are usually distinguished with lungs drawn in which an arrow is indicated entering towards the lungs or inspiratory route and one exiting from the lungs or expiratory route. The inspiratory route is the one from which the air comes out under pressure, the expiratory route has no recovery pressure and works only through the elastic force of the respiratory system. The ventilator circuit is connected to these two ways, which has two 22 mm female connections. We insert one in the inspiratory way and one in the expiratory way. We can insert filters called machine filters between the circuit and the ventilator. These filters have the task of protecting the ventilator and the patient. If we need to use a ventilator used with a previous patient, we place a filter in the inspiratory port. This is usually not necessary, except in this case. We almost always fit a filter in the expiratory connection to protect the ventilator, in particular the expiratory head and the flow sensor, if present, from particles such as those emitted by the patient or aerosol residues. These filters, called machine filters, have a filtering capacity of 99.999%, but have poor humidifying capacity. Continuing, these are followed by the actual circuit, often amicably called "corrugated". It is made up of two tubes, one for the inspiratory route and one for the expiratory route. These two tubes join at the end forming a real Y. The tubes in question can be completely smooth on the inside and

are recommended for ventilation with a helmet. By reducing turbulence, they are particularly quiet and comfortable for the patient.

Most circuits have a "corrugated" interior. This does not create major ventilation problems but makes them very practical and ergonomic to use. We can have circuits with obligatory or non-obligatory direction. In those with non-obligatory direction, it is possible to use one or the other tube for inspiratory or expiratory route. One-way traffic lights usually have one of the two parts marked; the reason for this distinction lies in the type of tube used which is usually larger in the inspiratory line to maintain the adequate gas temperature. Then there are circuits that have the tubes arranged coaxially, one inside the other.

In this case the internal tube is always the one that goes into the inspiratory line.

In this way, the still warm air exhaled by the patient will act as an "insulator" for the inhaled air which, in this way, will not reach the patient too cold.

At the end of the circuit, we find a 22 mm female or male connection to which a "patient" filter will be attached, which will both filter and maintain the humidity downstream of the system and therefore towards the patient. We connect a catheter mount to the filter, which has the sole task of being able to better manage the connection with the ventilator tube, without excessively twisting the tube. The positioning of the circuit also requires attention, go and look online for the most correct positioning.

The catheter mount has a 15mm or 22mm connection on the part that goes to the circuit and a 15mm female connection on the part that goes towards your tube or the cannula.

The circuit is usually equipped with a luer type fitting to be able to perform eTCO<sub>2</sub> measurements.

The catheter mount has a double cap in the part close to the patient. The larger cap allows you to perform an emergency aspiration in case the catheter mount and tube have been tightened too firmly, or to pass a fiberscope. The smaller hole has the same purpose, which allows the same procedures, making everything almost airtight.

Now that the air has reached this point, it can pass through the cannula or endotracheal tube, arriving nice and "fresh even if heated!" to the patient. From there he will go away taking with him the waste gases emitted by our lungs. This entire system, if properly set and connected, guarantees mechanical safety, leak tightness and complete infection protection.

Let's not forget that we have also protected our ventilator, costing tens of thousands of euros, which will thus be able to help many other patients.

## The endotracheal tube and cannula

Your endotracheal tube (ETT) has a 15mm male catheter mount attachment. It is held in position by the external fixation and partly stabilized by the cap. The cuff does not have the task of anchoring the tube, but only of separating the airway from the rest of the anatomical compartments. It plays the role of closure, allowing positive pressure ventilation to perform its function correctly, without air leaks. It also does not allow the entry of matter coming from above it, such as secretions, gastric material or material coming from the oropharynx. We remember that the presence of the tube makes us lose the important role played by coughing in normal people, which guarantees the elimination of foreign bodies or fluids that may enter the airways and guarantees the correct evacuation of the secretions produced.

For this reason, it is so important to maintain correct separation of the airways downstream of the cuff from those upstream. This is why it is so easy for a ventilated patient to experience unpleasant complications such as ventilator associated Pneumonia (VAP).



Searching through books and studies is a little confusing for any concept regarding ventilation. We are, so to speak, in an embryonic age despite 50-60 years of mechanical ventilation. Thus, we find different values, different names and different attitudes in any context. Over time we have gone from routine replacement of the ventilator circuit every 7 days to 30 days, up to never!

For the headset we have precise indications. These indications are more correct the more the size of the tube is suitable for the patient. If these conditions are respected, the cuff must have an ideal pressure between 15 and 25 cmH<sub>2</sub>O. However, a pressure lower than 30 cmH<sub>2</sub>O is accepted. Even with a pressure of 40 cmH<sub>2</sub>O we have an increased risk of tracheal necrosis. With 80 cmH<sub>2</sub>O tracheal necrosis begins after just 40 minutes!

Cuff pressure checks should be performed on every shift. If you have leak problems, even more often. If in doubt, it is advisable to seek advice from another colleague or a doctor. A non-functioning or leaking cuff that does not guarantee its correct function requires replacement of the endotracheal tube or endotracheal cannula.

At the correct pressure, we should not hear noises coming from the airways, gurgling or wheezing. Such an event is indicative of inadequate "cuff pressure". Consider that over time the balloon, but also the trachea, could present deformations which could affect correct closure of the airways. Two quick things, just to clarify. The evaluation method of compressing the pilot balloon between the thumb and index finger without manometric feedback is not correct (unless you are the son of a tire dealer!). This is a control that can give us only two pieces of information and in two very distinct and distinguishable situations:

The cuff is so deflated that even the pilot balloon is practically empty!

The cap is so inflated that it is almost incompressible.

Personally, as soon as I approach the patient, I always evaluate the cuff in this way, if I find myself in one of these conditions I must immediately restore a correct cuff, otherwise I can do it later during routine checks.

Incorrect cuffing also favors the extubation of the patient, because it will cause coughing and agitation in the patient. This increases the chances of extubation, especially if we are not there at the time.

Second curiosity: There is no correlation between cap and 10 or 20 ml syringes. The quantity present in one of these syringes is often indicated as correct, but once again these are not recognized techniques. The only correct control is that of pressure.

Typically, a 7 or 7 ½ tube requires less than 10ml for proper cuffing.

To check the cap, we need a suitable and accurate pressure gauge.

There are different types, analog or digital.

We can get help from the ventilator to understand whether or not we have cuff leaks. If the inspiratory and expiratory Tidal values were very different, we could think of a leak. This leak may be due to low cuff pressure.

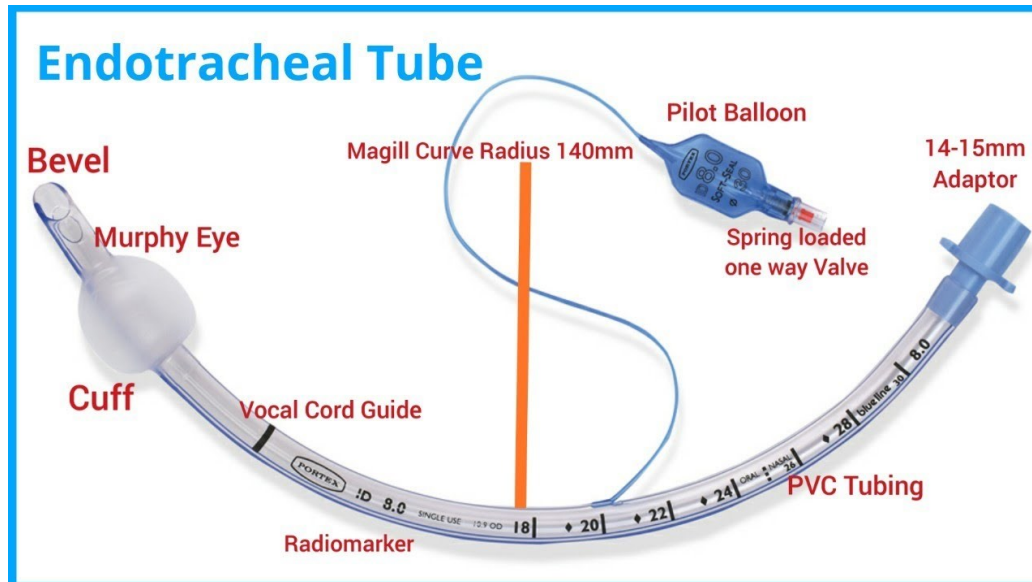
The cuff in these devices performs the most important task, it isolates the airways, but it must do so in a gentle way. The pressures indicated above are calculated to avoid necrotic tissue damage and are adequate for the level of perfusion pressure of the tracheal parenchyma. The trachea for its part does not make the task easy, with a "rigid" part with cartilaginous rings and a "soft" rear part. At that point the pressure of the cuff will have the opportunity to deform the wall and the higher it is, the more there will be the possibility of creating a tracheoesophageal fistula due to the pressure and necrosis. When, in the management of an intubated or tracheostomized patient, we take care of the "cuff" aspect, we are already at a good point!

Let's add the time factor to this recipe. It plays an important role, so much so that the management of a tracheostomy tube is much riskier than a tube. This is because, on average, a pipe can remain in place for 12-14 days up to a maximum of 30 days; a tracheostomy tube even

years! I am not referring here to the cannula itself, but to the effects due to the presence of this device on a continuous basis.

Last curiosity, an endotracheal tube must be positioned at the correct height.

This measurement should be noted and used as a future reference for proper patient management. A tube that slides downwards can easily "get stuck" and ventilate only the right lung. We listen to a patient at each shift to verify this possibility as well.



This in the image is an endotracheal tube, from right to left you can see its components: 15 mm fitting for connection to the catheter mount or circuit.

The tube, i.e. the actual air duct, equipped with an indication of the tube number in this case ID 8.0, of centimeters from the distal part, also has an opaque radio marking for easy viewing with chest x-ray.

About halfway, the sign to use for positioning with reference to the vocal cords.

Further on, the sign to use for positioning with reference to the vocal cords.

The actual headset.

On the flute spout, in some models there is the so-called "Murphy's eye", a hole that helps with ventilation in the event that the flute spout hits the wall.

The endotracheal tube exists in different sizes, to be used based on the patient's body weight and the control laryngoscopy.

There are different types of tubes, with supraglottic suction, armed, pre-formed and for particular uses.

Similar to the tube but shorter, because it is inserted directly into the trachea through surgery, it is the tracheostomy tube.



In it, the diameter indications are engraved in the flange, which has the task of "cannula stop", defining the point at which the cannula can no longer proceed towards the inside of the trachea. The flange also functions as a "holder" or fixing support, having two eyelets through which to pass the strap that will secure it to the neck. Both the cannula and the tube may or may not have a cuff. Some models of tracheostomy tubes have the possibility of replacing the internal part, called "canto-cannula", to be able to clean it adequately. Being a device that easily tends to get dirty with accumulation of secretions, replacing the internal part with a clean one is a feature to take into consideration when choosing the cannula. For obvious reasons, it is a device that presents a different resistance and a different dead space, smaller in the tracheostomy tube than in the tube of the same diameter. Therefore, when the device is inserted into the ventilator, it is not the same thing to put tube 8 or cannula 8! Remember that.

Replacing the cannula is however a delicate maneuver and must be carried out every 30 days or when necessary for other reasons. It is appropriate to make a distinction between the two types of tracheostomy, especially for colleagues with little experience. If the replacement, which the doctor performs anyway, concerns a tracheostomy with the removal of tracheal rings, the hole, once the tracheostomy tube to be replaced has been removed, will remain open, allowing for an easy and peaceful replacement.

If it is a tracheostomy performed in intensive care with only widening of the space between two tracheal rings, once the old cannula has been removed, we have no time to waste before inserting the new one. This is because the rings, as soon as the "intruder!" is removed, will tend to close quickly. Why am I telling you this? Because everything must be ready before the procedure begins, avoiding delays... by ready I mean, new cannula already tested with occluder inserted etc. Look for the correct procedures, it's not like I have to tell you everything!

## The catheter mount



The catheter mount, also called "airway connector", has the characteristic of facilitating the interface between the ventilation device and the circuit coming from the ventilator. Usually, these two components are positioned orthogonally, to make their connection easier the catheter mount is used which has a 90° angle and is also adaptable thanks to the soft central part. Furthermore, some have the connection to the tube detached from the body of the catheter mount, allowing it to rotate without forcing on the tube or cannula.

On the side opposite the tube connection and in line with it, circled in red, there is a cap, often two. The larger cap is used for suctioning secretions and is designed to be opened with one hand. The smaller hole allows the use of a fiberscope while maintaining the airway sealed even during ventilation.

The catheter mount contributes to the increase in dead space, because it is placed in a point where exhaled and inhaled mix. Replacing the catheter mount opens up a discussion that I would rather not have....

But since I'm writing these "two lines", I'll tell you mine:

Since it is a disposable device, it is often replaced every 24 hours to guarantee a certain degree of "sterility" of the system that transports the ventilatory gases. There are different indications online and if it is certainly not dirty it is possible to keep the catheter mount for 48 hours or more.

The reasoning is not so "crazy", if you think about it, he lives with the tube which remains there for much longer. In any case, adapt to the choice of your structure.

## The filters



Filters are essential elements for mechanical ventilation, they protect the patient, the operator and the equipment. We have different types of filters available, which we can arbitrarily distinguish into "machine" filters and "patient" filters.

This quick division describes the characteristics of the filters considering the machine filters only antibacterial and antiviral filters, the patient filters are antibacterial and antiviral filters, but also capable of maintaining an adequate temperature and humidity of the airways.

**HEPA** (High-Efficiency Particulate Air) type filters only have antibacterial and antiviral filtering capacity with particles larger than **3 microns**.

**HME** type filters are (Heat and Moisture Exchanger) filters that are responsible for retaining adequate temperature and humidity. For humidity, they use different systems, such as using a specific salt that has hygroscopic characteristics.

Unless specified, an HME filter has no antibacterial-viral filtering power. To guarantee this characteristic the wording should be **HMEF**. A full-fledged HMEF filter has the characteristics of an HME and a HEPA in a single device.

HEPA (machine) filters also have the ability to retain moisture and heat, but to a much lesser extent, often not adequately. There are different filters capable of guaranteeing their characteristics based on the air flow to which they are subjected. This makes certain filters suitable only for ventilation in pediatric areas. The filter dimensions, in this specific case, are not the correct method for choosing. A small filter does not necessarily mean "pediatric" and vice versa. We must always read the characteristics of the filters available. As I mentioned above, there are different schemes for using filters in the ventilation system setup. We can insert 3 filters or none in particular conditions. The "patient" filters, as they are positioned where the inspiratory and expiratory gases share the same volumes, increase the dead space.

How do you say it? By how much? Read the features please!

Could increase dead space be a problem? Diplomatic response: It depends. The clinician will evaluate it.

Replacement: here the situation is slightly different than before. Manufacturers recommend daily replacement and, if dirty or saturated with condensation, as soon as necessary. A high pressure ventilator alarm could actually be caused by a filter saturated with moisture.

The same manufacturers who recommend 24 hours allow up to 48 hours of life for a filter that is still in good condition.

Here too, follow the guidelines dictated by your structure.

Among the characteristics of the filters, you will find the dead space in mL, the accepted Vt, indicated in mL and the moisture output in mg/VT1000. Moistur output is nothing other than the ability to maintain a certain humidity.

Filters designed to be placed as occluders for tracheostomy tubes or tubes in spontaneously ventilating patients also have a hygroscopic salt to keep the airways humidified and prevent them from drying out with breathing. Some of these have an additional treatment with chlorhexidine to reduce the possibility of contamination of the airways themselves (MEDIFLUX by Medival)

Almost all filters have a luer connection on which it is possible to connect a sensor for capnometry, therefore the measurement of the partial pressure of carbon dioxide in the exhaled air.

## The circuit or "corrugated tube"



The ventilation circuit acts as a link between the ventilator and the patient. It has the characteristic of being flexible and allows freedom of positioning.

The ventilation circuits can be corrugated or smooth on the inside, this makes the smooth ones quieter.

They can be connected directly to an endotracheal tube (emergency only) for this reason they all have a 15 mm female connection in the Y; but they should never be connected directly to the tube but only via a device that allows ergonomic positioning of the tube and circuit, the catheter mount. By law they must be transparent or opaline to allow the operator to notice the presence of any condensation or secretions that have escaped the filter. There are dozens of different models with double tubes like the one in the photo, single tubes, with tubes in an axial configuration, with condensation traps and other types.

The most used in mechanical ventilation in intensive care are the double-ended circuits like the one in the photo. At the end all the tubes have a Y where the gases entering and leaving the circuit find a common space. It is precisely at that point that the so-called "dead space" of the artificial airways begins.

The circuit is disposable and single patient, it must be disposed of according to instructions every 30 days, but some companies have abandoned this procedure, leaving it in use indefinitely.

Follow your department's instructions.

Also in some ventilation circuits there is a connection for capnometry.

### Why is it important to do pre-use testing on ventilators?

Mechanical ventilators are as advanced as they are delicate. Not only that, those "maniacs" of engineers who worked on it decided that every time we have to use it after a break or a different patient, we have to check a couple of things. Over time we have moved from checks that required a bit of effort on the part of the user to machines that perform these checks almost by themselves. So, when Mr. Before this can be used, we must carry out these pre-use checks. The ventilator, in this case, will check that the electrical and medical gas supply is correct, that the measurement systems are calibrated correctly, and that the exhalation valve and the flow sensor work properly. This check also allows us to check the tightness of the circuit and ultimately give us a complete report of what works, and, above all, what doesn't work. A ventilator that does not pass the pre-use test should not be used and deserves greater attention from us. It could be a trivial problem such as a poorly mounted expiratory head or it could require the intervention of a specialized technician.

In any case, the check must be carried out before the patient is in the room and we must have a Mapleson C circuit or an Ambu bag with a mask and an oxygen source at hand.

Doing the pre-use check is a guarantee for the patient and avoids last-minute rushes, which are never a good thing.

Now we just connected the ventilator to the patient, all with lots of fittings between the parts. Add to these the luer ports, the removable catheter mount caps and you have an easy, easy diagram to follow if there was a leak somewhere.

I'll give you a little advice: if the ventilator "sounds" like leaks, and you are close to the patient, and a significant vent is easily perceptible, look in the main connections. If the ventilator "alarms" leaks and you hear nothing or a barely perceptible sound, it could be the luer fittings that have lost the cap, or the bronchoscopy line of the catheter mount. In both cases, start from the ventilator connections and move towards the patient, quickly checking all the connections. Over time, you will find the leak in the blink of an eye, and after just 10,000 hours of intensive care, you will know a leak and where it is before the ventilator starts "screaming" **LEAK!**

We have seen various aspects regarding mechanical ventilation. Now we are able to understand different aspects of "breathing", let's compare the two types of ventilation.

## Difference between normal and mechanical ventilation

Let's take a short break and compare the normal breath and the breath delivered by the ventilator. What we need to understand is that the ventilator, to achieve the same end goal, uses an anti-physiological system. This is why, when possible, we must resolve respiratory failure without resorting to mechanical ventilation and, when this is necessary, terminate it as soon as possible. Don't misunderstand my words, I don't mean to say that mechanical ventilation is bad in itself. What we need to know is that the longer a patient remains on mechanical ventilation, the more possible problems he will encounter. Speaking, in "medical terms", a prolonged time on mechanical ventilation is associated with a worsened outcome.

### Normal ventilation

The diaphragm lowers, changing the pressures involved. The alveolar pressure becomes negative and draws ambient air into the lungs. At the end of inspiration, the elastic force of the chest wall pushes the air out of the lungs with exhalation.

### Mechanical ventilation

The ventilator is responsible for generating a flow of air, pushing a volume of air into the lungs, to do so it generates a positive pressure higher than the alveolar pressure. It must be higher than the alveolar one to allow the flow to go from the ventilator to the lungs. For now, we can consider this mechanism completely passive on the part of the patient... for now! When the ventilator, based on the set parameters, knows that it has reached the correct volume or pressure, it opens the expiratory valve and allows the air to escape, again thanks to the elasticity of the patient's chest wall.

Told in this way, there is apparently no difference between the two ventilations; in truth many things change and believe me, really important things.

Remember when we said that "physiologically" the intrapleural pressure remained negative in the phase between inspiration and expiration? Here, when the lungs are filled with air not by using the negative pressure that is generated inside, but by generating a positive pressure, that pressure also becomes positive!

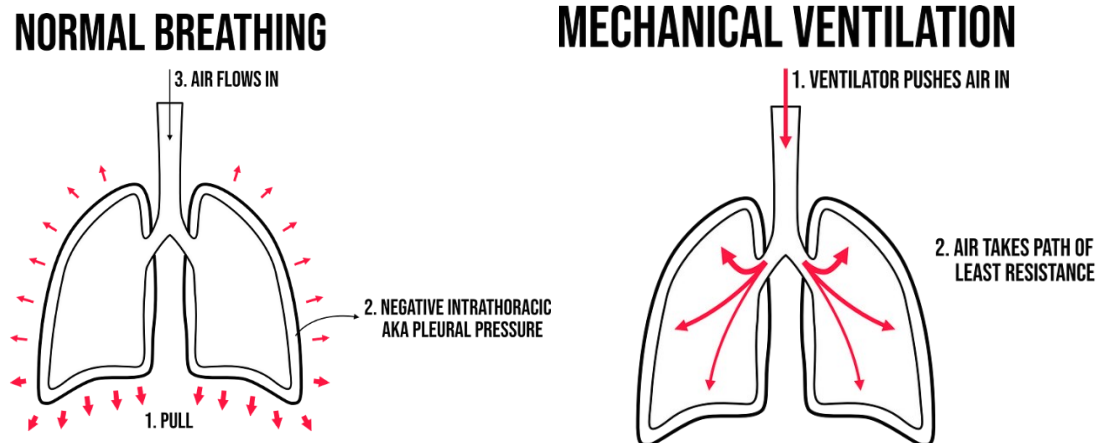
All ventilation modes used with mechanical ventilation are positive pressure types.

Therefore, while we breathe physiologically, the diaphragm lowers and the rib cage expands, generating the negative pressure that will allow an inspiratory flow. Most of this result is due to the excursion of the diaphragm, and it is for this reason that the dorsal and basal lung area is better ventilated. This is why nature has placed a higher density of blood vessels in these areas.

This mechanism is so important that it is called the "**respiratory pump**".

In mechanical ventilation, that positive pressure which is responsible for generating a flow will depend on the resistance it will encounter along its path. Simply put, the air will have an easier time reaching areas with less resistance, coincidentally the apical areas of the lung. These are the areas with the least distribution of blood vessels. A big difference when compared to normal ventilation!

Look at these images.



Now, analyzing this operation we arrive at three results:

The regions with lower perfusion are the ones ventilated better.

The regions with greater perfusion are worse ventilated.

The apical region, which offers less resistance, is the one most exposed to risk of damage from ventilation, receiving greater pressure and volumes during ventilation.

I explain the concept of perfusion and ventilation better where I talk about V/Q mismatch.

### Influence of mechanical ventilation on hemodynamics.

Since the heart and lungs share the same thoracic space, the functioning of one determines variations in the functioning of the other. Thus, during normal breathing, during inspiration there is an increase in venous return and therefore in preload, because we have a compression of the vena cava and therefore a greater dilation of the right atrium.

This does not happen during positive pressure ventilation.

**The exact opposite happens**, we replace a normally negative pressure with a positive pressure; this has repercussions on the entire thoracic region, causing a decrease in venous return and consequently a reduction in cardiac output.

If we add to this positive pressure, as practically always happens, a PEEP (end-expiratory pressure), this phenomenon will be accentuated.

Now, just to have an idea of what happens, if the PEEP remains lower than the patient's CVP value, we can maintain acceptable hemodynamics. If PEEP has a higher value than CVP, the patient may experience hemodynamic instability.

The heart-lung correlation in mechanical ventilation is very interesting.

If you are curious about the topic, search online.

For this job the information I have given you is enough.



## Mechanical ventilation

In this part I mainly used materials from Hamilton Medical.

Mechanical ventilation is nothing other than the act of breathing that humans normally perform alone, carried out by a machine, completely or partially. This device does the work in place of the patient's respiratory muscles and is called a mechanical ventilator or simply ventilator.

Why do we use it?

We use it in cases of respiratory insufficiency of varying degrees and it helps to improve pulmonary gas exchange, therefore oxygenation, reduce acidosis, treat ventilation disorders such as respiratory insufficiency, reduce the oxygen requirement of the respiratory muscles, as a support to tired muscles, improve ventilatory dynamics, to promote alveolar opening, improve compliance, prevent lung lesions, to reduce WOB and increase ventilatory volumes.

With mechanical ventilation, we can bring a hypoxic or hypercapnic condition back to normal, with normalization of blood gas for PaO<sub>2</sub>, PaCO<sub>2</sub> and pH values.

It allows the sedation of a patient, for example in the case of surgery, reducing the need for systemic and cardiac oxygen. It can reduce intracerebral pressure, ensure airway safety and patency

In addition to all this, we have the possibility of using mechanical ventilation with different interfaces: face mask, helmet, endotracheal tube and laryngeal cannula or tracheostomy tube.

The mechanical ventilator manages the two phases of breathing through solenoid valves and various regulators; it can manage pressure, flow speed and how quickly it is delivered.

It also has many other settings in order to finetune and personalize the correct "breathing" of the patient. To these adjustments we add the alarm limits present for almost every setting, limits which if well managed make mechanical ventilation safe and easy to manage.

But does the ventilator do everything by itself?

Certainly not! When we talk about mechanical ventilation, we must consider six main elements, and not just the mechanical ventilator, we call this "**ventilation system**":

- 1 Compressed air and compressed oxygen
- 2 Electrical supply
- 3 Motor
- 4 Respirator circuit
- 5 Artificial airways and ventilation interfaces
- 6 Lungs

In full Gi spirit, I also add the seventh element, that is the operator, without him nothing would work properly.

It doesn't end there, for the mechanical ventilation system to work correctly, certain conditions are necessary:

- All parts must be present and functioning
- The system must be mounted correctly
- The gas supply must be appropriate and stable
- The power supply must be correct and stable
- The system must not have leaks
- There must be no occlusions in the path
- The patient must have ventilatable lungs
- The operator must know and know how to best use the system

Mechanical ventilation is a truly complex aspect of patient care. There are so many things to know and the ventilation system is complicated. By applying everything to different patients with different types and levels of severity of pathologies, dozens and dozens of settings and measures to put into practice emerge, in short to be clear:

**Mechanical ventilation is not like putting nasal cannulas to two liters of oxygen and off you go!  
Just to clarify, whoever sets it up is a dragon!**

To begin our discussion on mechanical ventilation, there are some concepts you need to know. We've already seen some of them, but it's not bad to review them quickly:

Pressure, pressure gradient, volume, flow, compliance, resistance and time constant.

**Pressure:** it is the force applied on a surface

**Pressure gradient:** it is the pressure difference between two different areas. This pressure difference, if we are talking about gas, allows a movement from the higher pressure area towards the lower pressure area. This movement of gas is called flow.

**Volume:** it is the space occupied by a certain quantity of gas at a certain pressure.

**Flow:** Flow is the movement of a certain volume of gas over time. It has two characteristics: direction (from the higher pressure zone towards the lower pressure zone) and speed.

The direction depends only on the pressure gradient, the speed on the pressure and resistances.

The flow has positive values during inhalation and negative values during exhalation.

A low flow generates a uniform flow, called "laminar", a high flow generates disturbances called turbulence.

**Compliance:** Represents the relationship between volume variation and pressure variation. The lungs change their internal volume as internal pressure changes. If to reach a certain volume we have to apply a small pressure, we say that the lung has a high compliance. If to reach the same previous volume we have to apply great pressure, we find ourselves in the presence of a lung with low compliance. Compliance should be measured in the absence of flow, in this case it is called static compliance.

**Resistance:** It is a force that opposes or slows down the movement of a gas.

Resistance depends on three factors:

The flow, the greater the flow the greater the resistance and vice versa.

**If the flow is zero, the resistance will also be zero**

The circuit through which the flow passes, if long it increases the resistance, if it is thin, it increases the resistance, if its surface is not smooth it increases the resistance.

The physical properties of the gas.

**Time constant:** the time constant represents the time necessary for a given volume, with a given flow, to enter the lungs with inspiration or exit with expiration.

If the time for inspiration is shortened, the  $V_t$  is lower. If the time for exhalation is reduced, all the gas will not exit the lung and will remain "trapped" (air trapping) in the lung, thus increasing the pressure present in the lungs at the end of expiration.

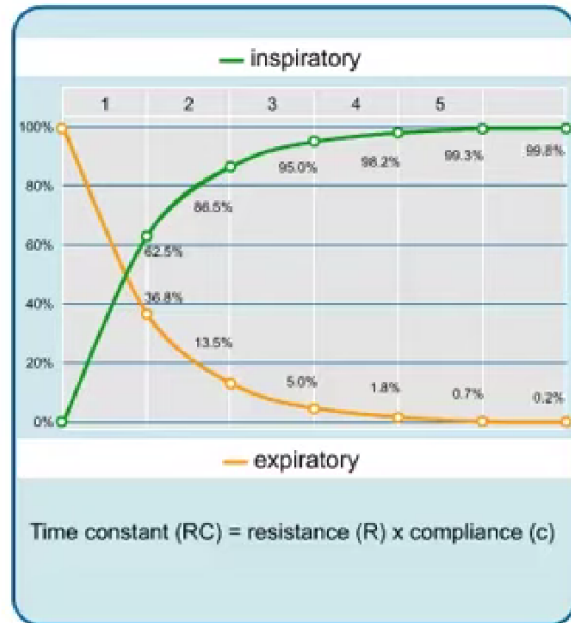
This phenomenon is called Auto PEEP.

We can know the time constant by knowing the characteristics of the respiratory system, compliance and resistance. Thus, inspiratory "filling" time increases when compliance is low or resistance is high; otherwise it decreases. The time constant is exponential, with a maximum filling

or emptying speed in the first part and then reducing (decelerating). The time constant is expressed in seconds.

## Key physical elements

- P: Pressure
  - $\Delta P$ : Pressure gradient
  - V: Gas Volume
  - $\dot{V}$ : Gas Flow
- 
- R: Resistance
  - C: Compliance
  - RC: Time constant



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Here is a representation of the Hamilton Medical time constant

The time constant identified with 1 represents the capacity to inhale or exhale gas in a normal lung, equal to 63% of the total volume.

A normal lung the time constant is 0.5-0.8 seconds.

So, those numbers expressed above do not represent seconds, but the time necessary for 63% of the volume of gas to be moved into or out of the lungs.

An inspiratory and expiratory time constant of 3 is considered normal

It is important to have a ventilator that calculates the time constant to avoid phenomena such as air trapping.

Among the many settings that ventilators have, one of the most important is the ventilation mode. By ventilatory mode we mean a type and sequence of breathing acts suitable for a particular patient's condition.

Ventilators have several modes they can be set to, and can only operate in one mode at a time. Once a mode has been set, the ventilator will only "obey" our requests, doing what it was programmed to do for that mode.

Another thing that can be set on a ventilator are the parameters that can define the characteristics of breathing. Among the parameters that can be set we have cycling, trigger, volumes, pressures, ramp, flow, inspiratory time, and much more.

We can divide these parameters into three groups: ventilation, oxygenation and synchronization.

- Ventilation parameters are:  $V_t$ , pressure, pressure support, respiratory rate,  $T_i$ , I:E.
- Oxygenation parameters:  $FiO_2$ , PEEP

- Synchronization parameters: trigger, rise time/ramp, flow cycle (ETS)

**Grouped control parameters**

Controls for ventilation	Controls for oxygenation	Controls for synchronization
<ul style="list-style-type: none"> <li>• Tidal volume</li> <li>• Pressure control</li> <li>• Pressure support</li> <li>• (Mandatory) rate</li> <li>• Inspiratory time</li> <li>• I:E ratio</li> </ul>	<ul style="list-style-type: none"> <li>• Oxygen (=FiO<sub>2</sub>)</li> <li>• PEEP</li> </ul>	<ul style="list-style-type: none"> <li>• Patient trigger (pressure or flow) and the sensitivity</li> <li>• Rise time / pressure ramp</li> <li>• Flow cycle (ETS)</li> </ul>

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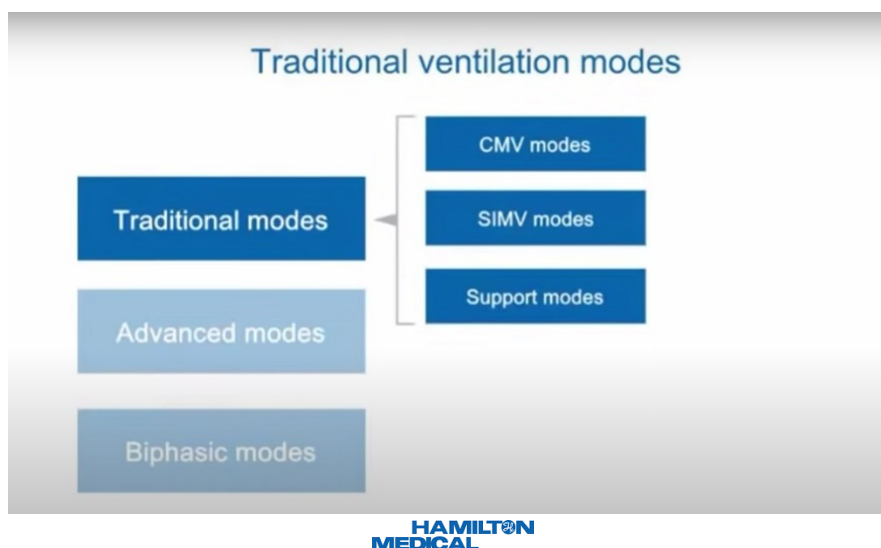
For each patient, modes and parameters must be set individually. According to Robert L. Chatburn's classification, there are hundreds of ventilatory modes. How could this happen? Because there is no standardization of ventilation modes at a global level. These, over time, have evolved, multiplied and twinned with similar methods from different brands. In a few years, each brand has "created" its own ventilation mode, very similar to the others, with very few differences. This has made the available ventilatory modes much fewer than the hundreds we have seen before.

The methods can usually be classified into three groups: Traditional, advanced and biphasic.

**Traditional modes:**

These are the modes found in most ventilators, they are safe and proven modes and are widely used.

We can divide them into three subgroups: CVM mode, SIMV mode and support mode.



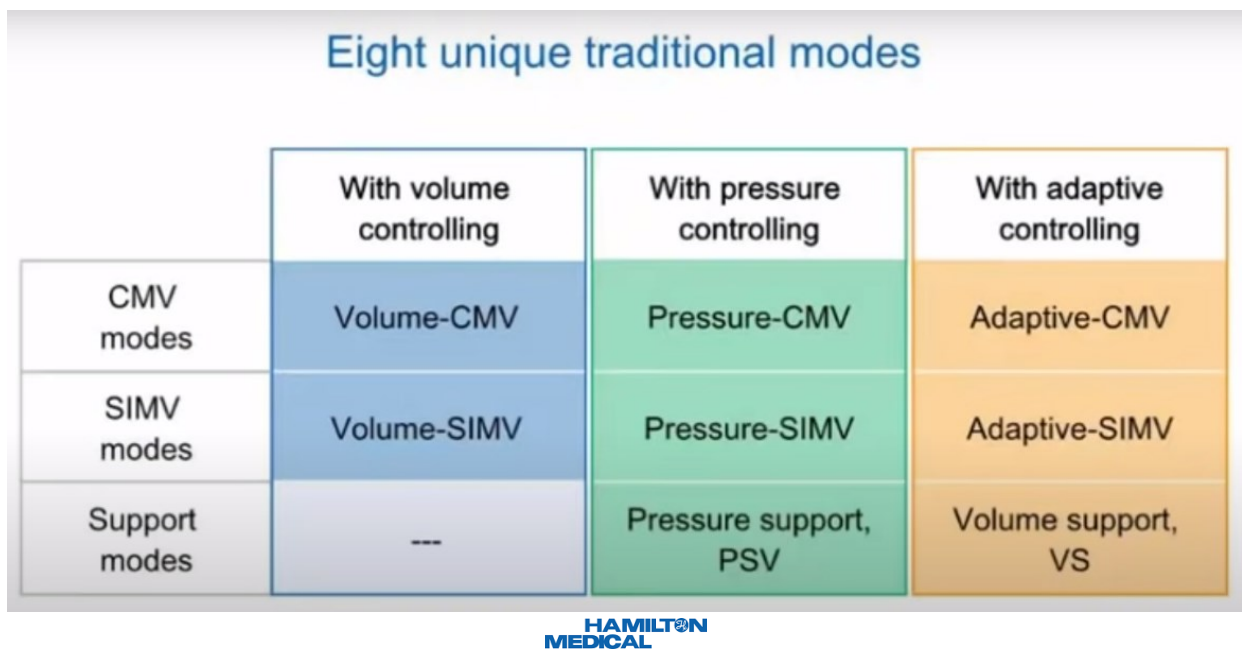
**CMV modes** are divided into 2 groups:

**A/C:** in which the patient can trigger spontaneous breaths, are modes in which the patient can be completely passive or partially active.

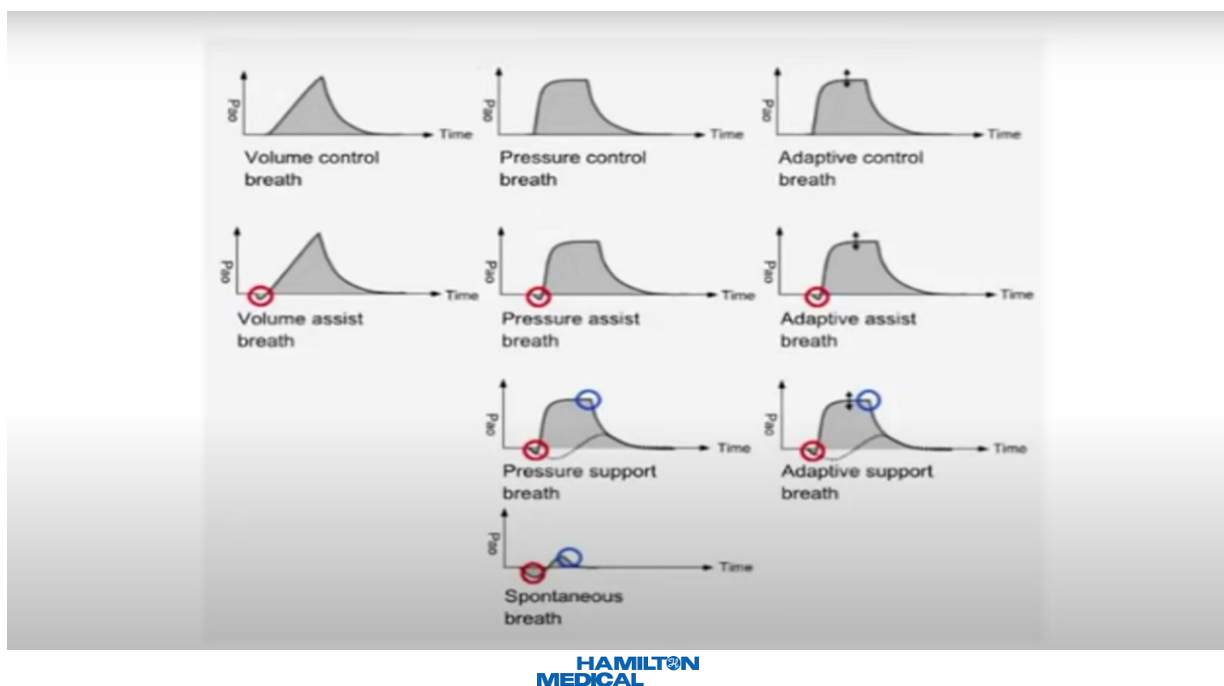
**CMV:** in which the patient can trigger, but spontaneous breaths are not possible between mandatory breaths.

A/C mode with a passive patient works like a CMV.

From this division, taken from a Hamilton Medical video, only eight different traditional ventilatory modes emerge as in the image.



Here are the types of breathing with their curves:

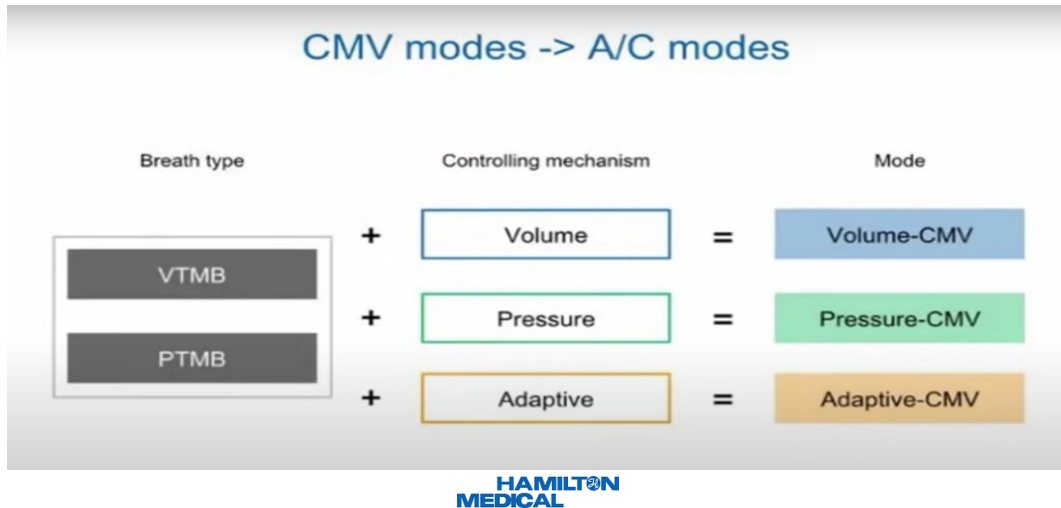


In CMV modes we have VTMB, or ventilator triggered mandatory breath, and PTMB, or patient triggered mandatory breath.

These indicate how the breath is triggered: by the ventilator (VTMB), where the trigger will be time-based, or by the patient (PTMB), where the trigger will be flow or pressure.

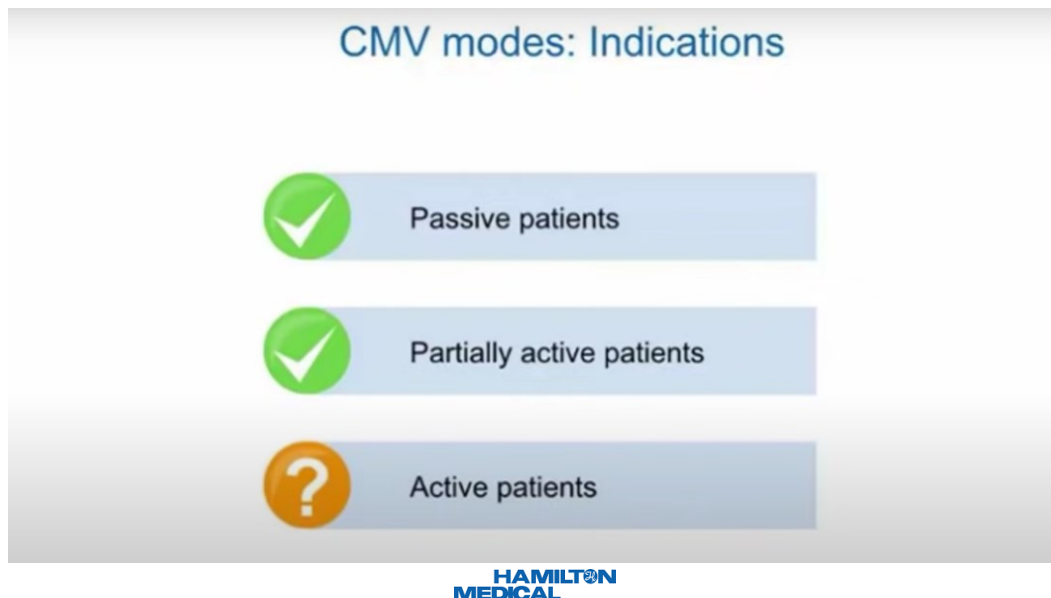
This mode can be used with volume control, pressure control or adaptive, thus obtaining three types of CMV:

VC CMV, PC CMV and Adaptive CMV



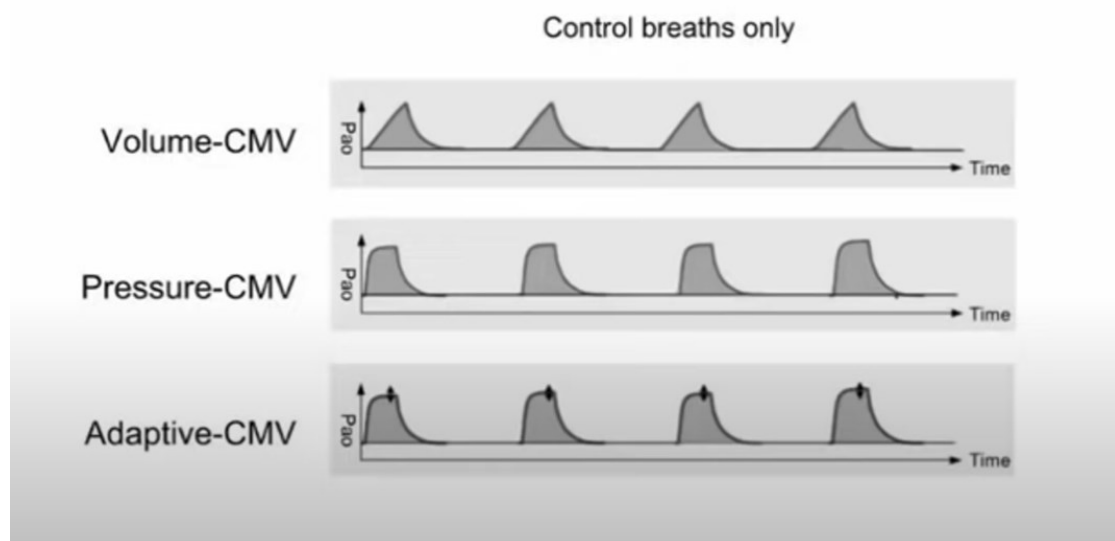
In CMV mode, you need to set a breathing rate. If the patient triggers breaths, these will usually be more frequent than those set on the machine.

CMV mode is indicated in passive, partially active patients, but not in fully active patients from the point of view of spontaneous breathing.



In this mode, the ventilator that does not detect a patient trigger delivers an automatic breath. When it detects a patient request, it delivers a breath with user-defined settings.

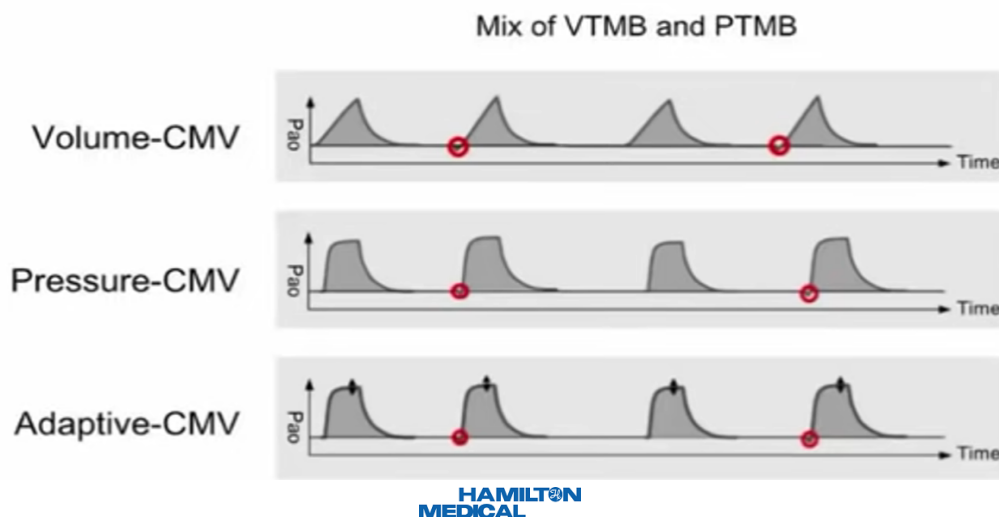
## CMV: If patient is passive, ...



This above is a CMV with a passive patient.

And this one below with a partially active patient.

## CMV: If patient is partially active, ...

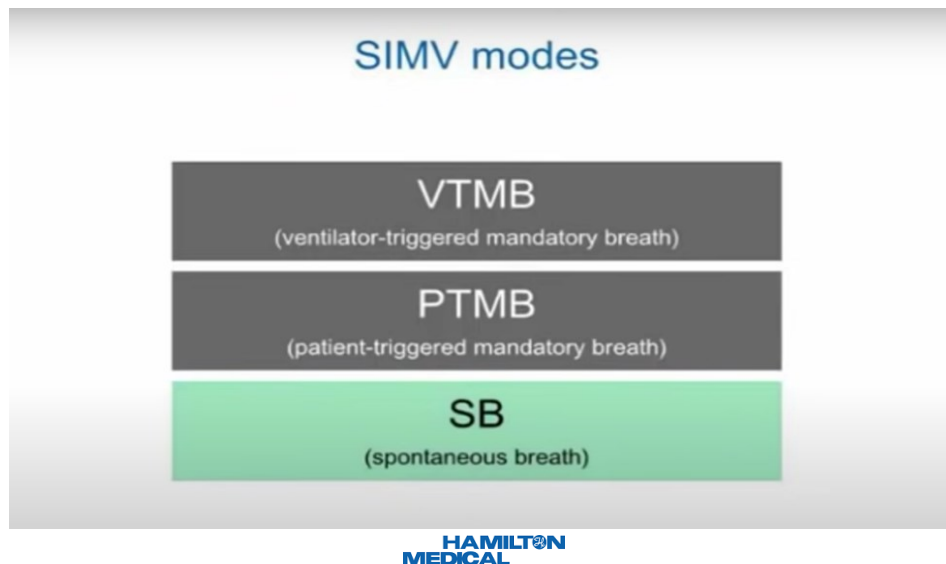


### The SIMV modes

It derives from the IMV mode, now almost completely replaced by SIMV.

SIMV also has three modes: VTMB triggered and cycled by the ventilator,

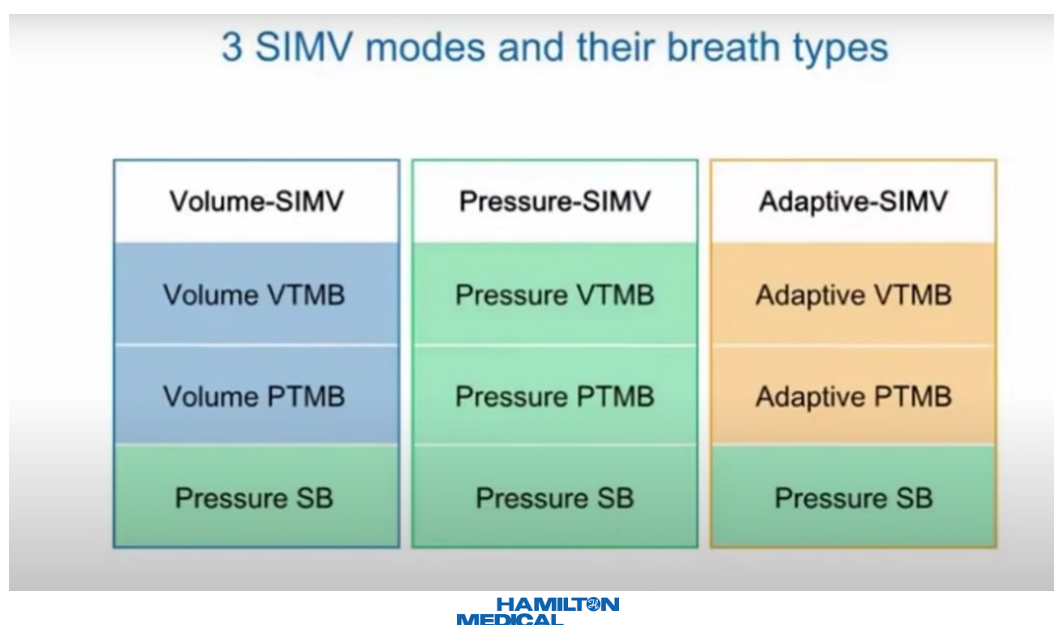
PTMB triggered by the patient with timed cycling and SB (spontaneous breathing) triggered and cycled by the patient.



SIMV can also be volume controlled (VC) or pressure controlled (PC), while SB, on the other hand, is **always** pressure controlled.

It is a ventilatory mode suitable for completely passive, partially active and active patients. In partially active patients, it is necessary to set a relatively normal backup frequency. If the patient is active, we must set a very low rate to allow the patient to breathe spontaneously as comfortably as possible.

In this way, the ventilator does not intervene to "break" the patient's rhythm.



Above obtainable SIMV modes.

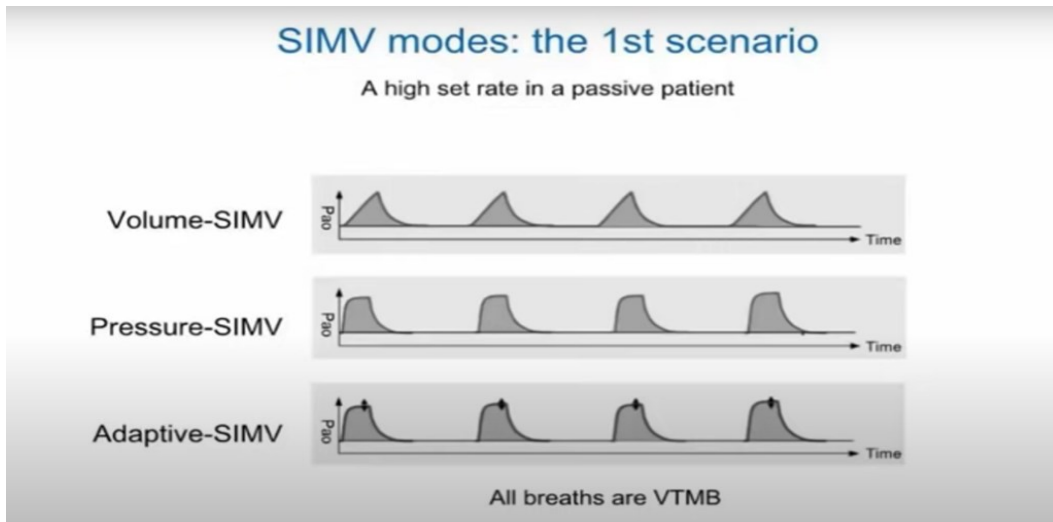
If we set a low respiratory rate (RR), we give the patient time to take spontaneous breaths. Otherwise, the mode may work like a CMV.

In a passive or partially active patient, we need to set up a backup mode.

We are faced with three scenarios:

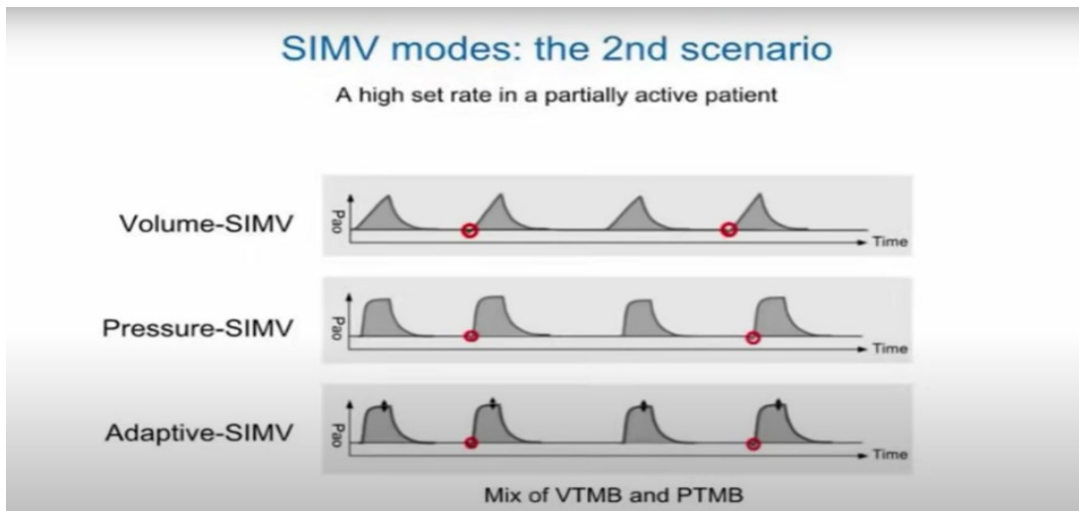
passive patient, all breaths are VTMB





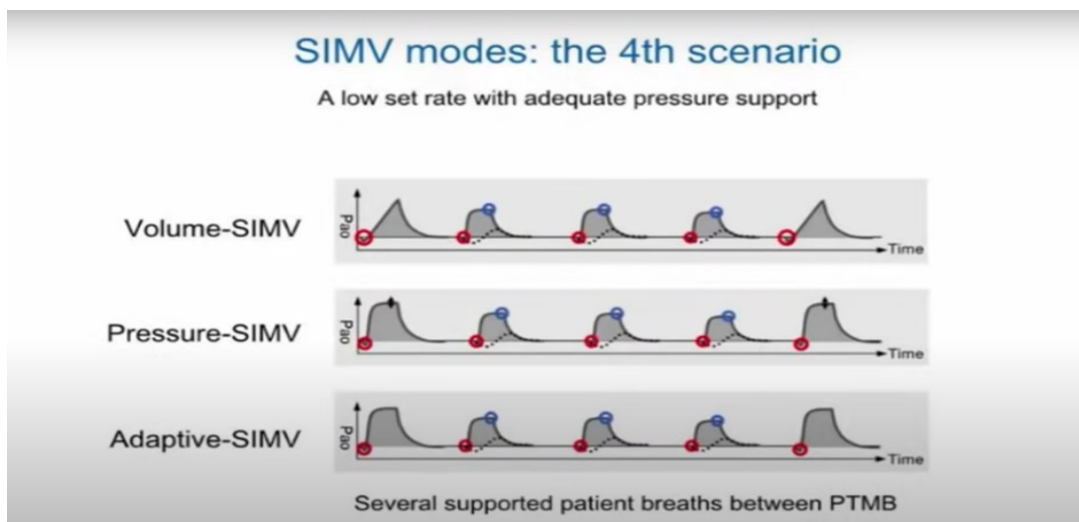
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Partially active patient, we have a mix of VTMB and PTMB:



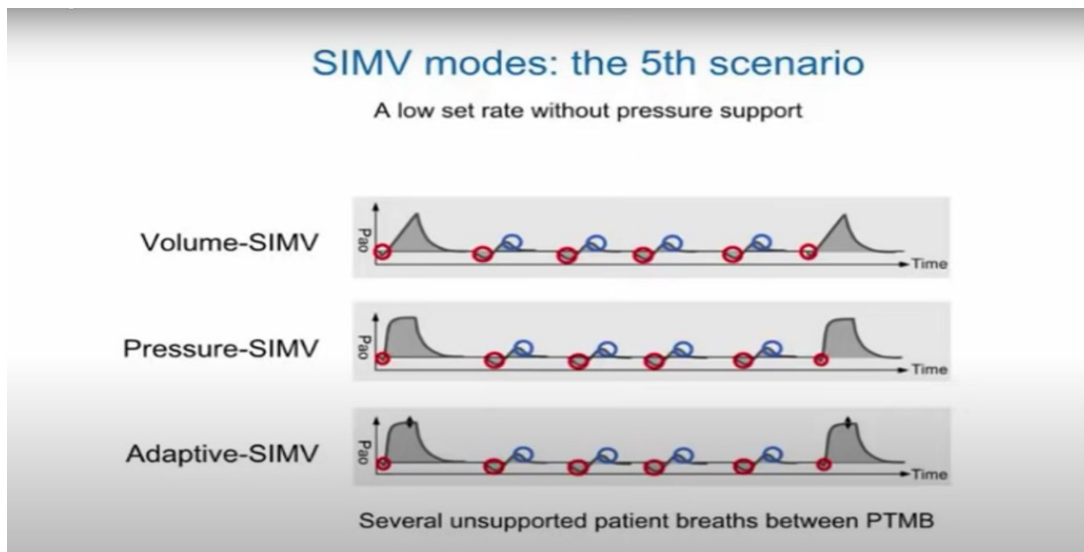
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If the patient is active, all breaths will be PTMB naturally by setting a low RR:



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What happens if we set a SIMV mode without support pressure? This:

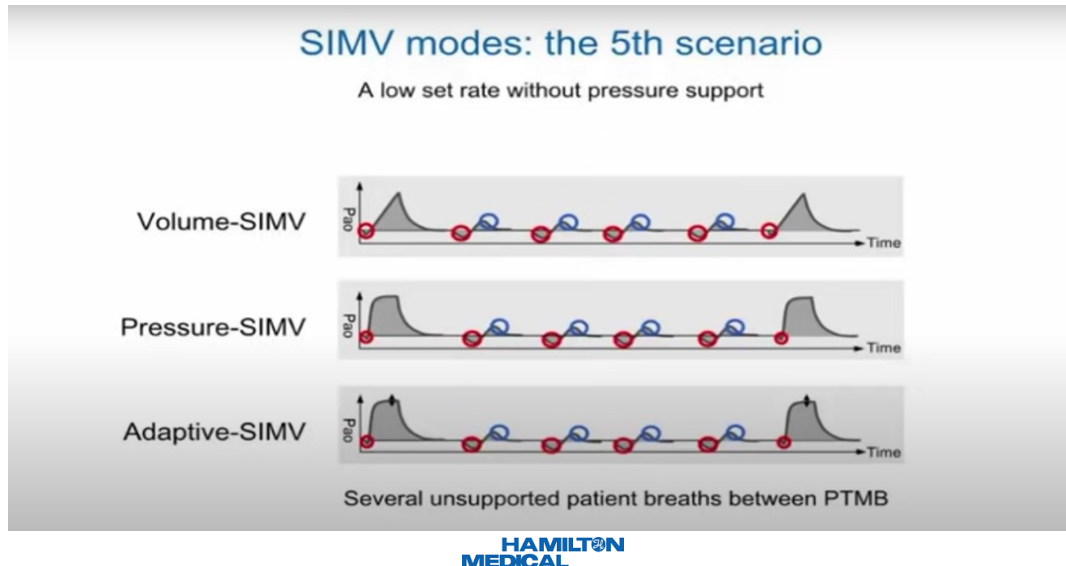


In the SIMV we set: RR, Ti, ETS, PEEP, PS, FiO<sub>2</sub>, vt or P control, rise time.

### The support methods:

It has two actuation modes, pressure support and volume control.

In this mode the ventilator delivers breath as soon as the patient triggers a spontaneous breath. It is valid only for active patients. It is still necessary to activate a backup or apnea ventilation mode in case the patient stops breathing.



It is also known as SPONT, PS or PSV mode. In these modes, if we set the pressure to zero, the patient will breathe completely autonomously, we are talking about "true" spontaneous breathing. It is a method used for weaning.

In volume-supported mode, you set a desired volume and the pressure is adjusted to achieve the set V<sub>t</sub>.

In Pressure Support mode, the correct setting of the support pressure is important, which is the one that correctly supports the spontaneous acts of the patients.

## Pressure support mode

high
P<sub>support</sub>

- Pressure support control is important
- With a high P<sub>support</sub>, patient breaths are sufficiently supported



Recall that this is necessary because the patient may not be able to perform all the necessary WOB.

Volume support mode, relatively young, in which a target V<sub>t</sub> is set.

In this way the ventilator supports the patient by adjusting the pressure with each breath to obtain that famous V<sub>t</sub> target.

In these modes we set: PEEP, FiO<sub>2</sub>, PS or V<sub>t</sub> target, trigger, t<sub>i</sub> max, ETS, rise time.

**Advanced modes:** Use elaborate algorithms to perform personalized ventilation that is finer than traditional modes.

Their aim is to improve the quality of ventilatory therapy and lighten the work of clinicians.

To do this, the ventilator is able to modify settings on its own in accordance with the choices of whoever programmed it for that patient.

These are some of the advanced modes.

## Advanced modes

Mode name		Vendor
Adaptive Support Ventilation	ASV	Hamilton Medical
INTELLIVENT-ASV	---	
Proportional assist ventilation	PAV	Covedien/PB
Proportional pressure support	PPS	Dräger
SmartCare	---	
Neurally adjusted ventilatory assist	NAVA	Maquet
Automatic controller of Oxygen	CLiO2	CareFusion



## Biphasic modes

In normal mechanical ventilation, the inspiratory valve is open when the expiratory valve is closed, allowing gases to reach the lungs in the necessary volume.

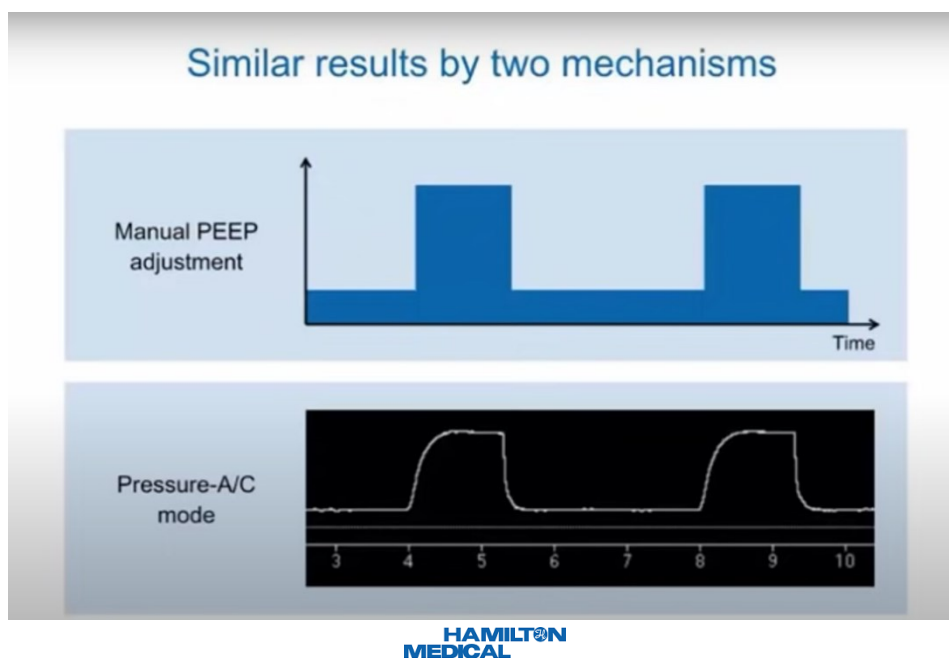
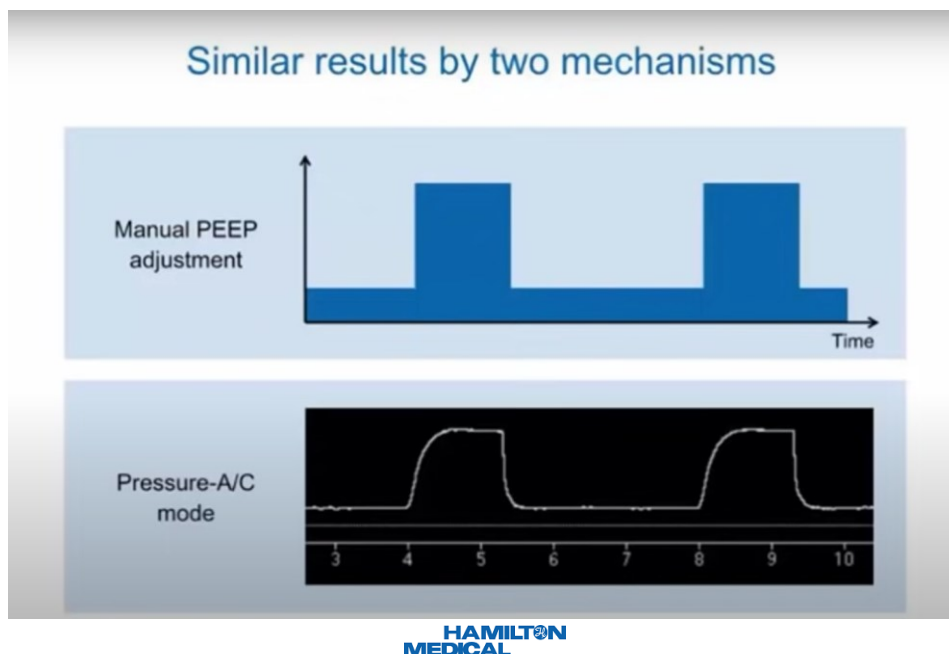
During the exhalation phase, the expiratory valve is open, and the inspiratory valve is closed.

The two valves always work alternately.

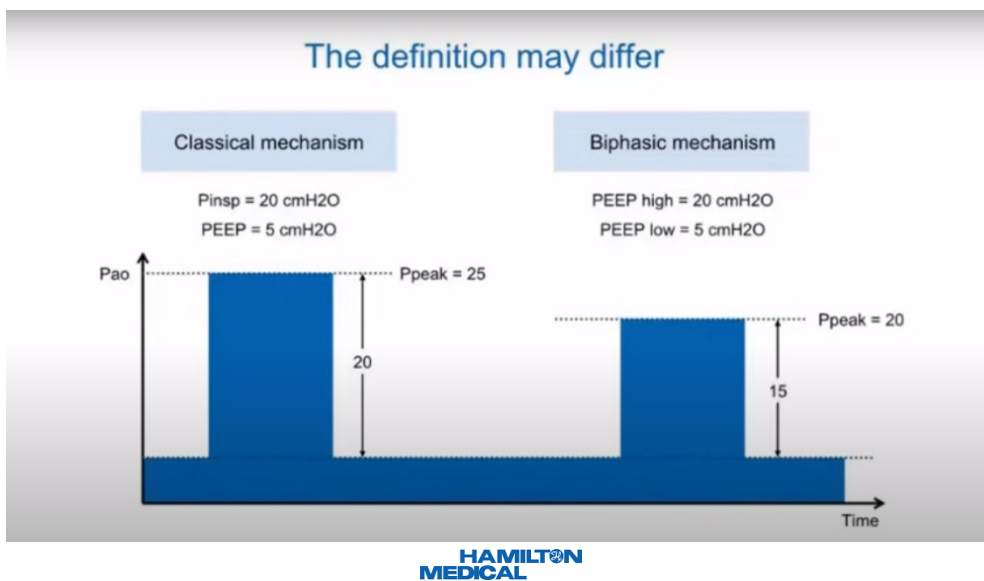
When we apply a control on the exhalation valve during the exhalation phase, we can control the escape of gases. This is useful for maintaining a certain PEEP within the alveoli.

A variation of this mechanism is the basis of biphasic ventilation. By continuously applying baseline and higher PEEP, a pattern very similar to pressure-controlled ventilation is generated.

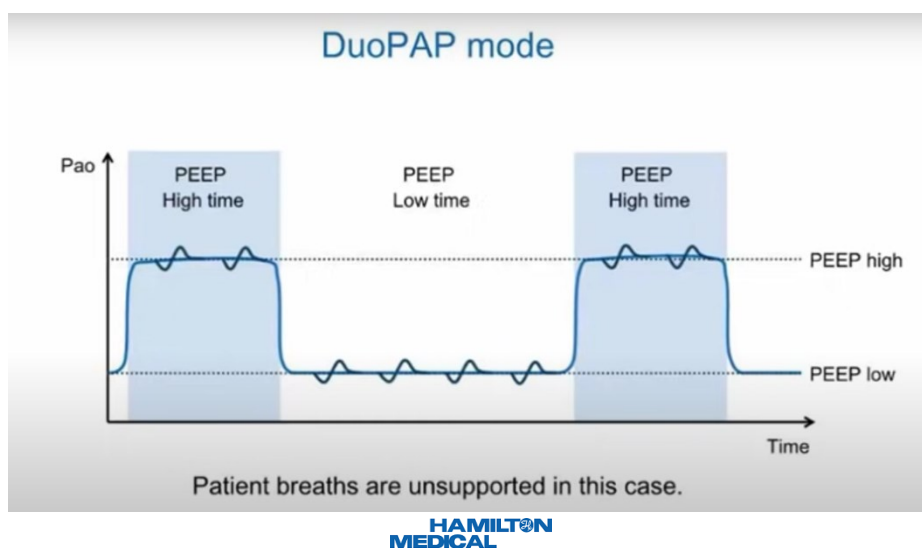
Look at the image below:

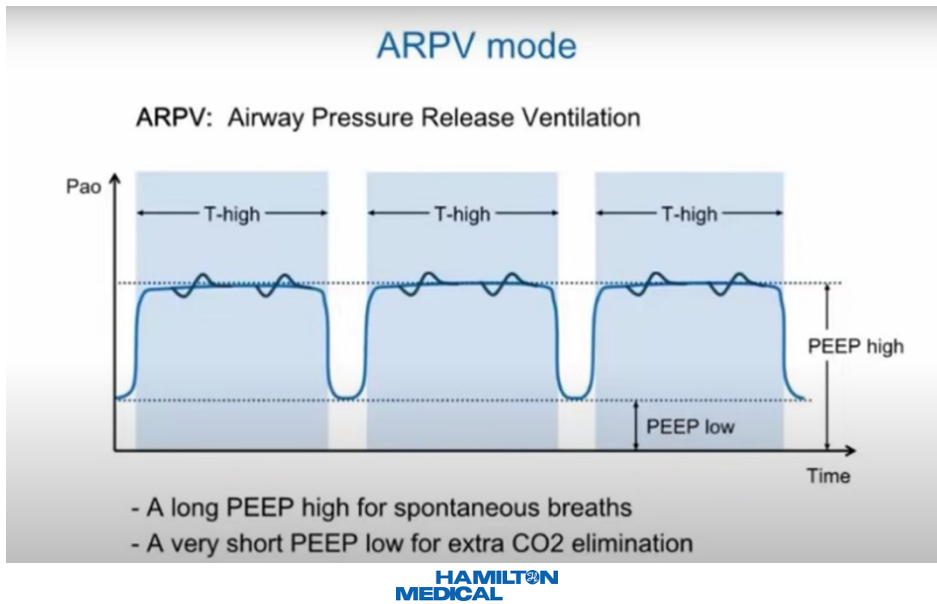


In this image you can see the alternation of two different levels of PEEP. These levels are called PEEP high, PEEP low, and each of them is associated with a certain duration called High time and Low time. The pressure levels are set in this way: the high level is present during the inspiratory phase, and for this reason PEEP high is called IPAP (inspiration), while the low level present during exhalation, PEEP low, is called EPAP (expiration). Therefore, biphasic ventilation has the characteristic of having four basic settings: IPAP, EPAP, High time and Low time. They are, in theory, more comfortable modes for the patient, but they have a downside because they consume more gas than a normal mode. A biphasic mode can be pressure-only or adaptive, **not volume-controlled**. It is also called BiLevel, BiPAP, DuoPAP, Bipap, APRV etc. This is the comparison between a traditional system and a two-phase one:



The patient is free to breathe on those PEEP levels, in this way it is as if he is "riding the wave":





This above is the APRV mode.

As you can see, in the APRV we have a very short expiratory phase and we do not have a low PEEP, the limit is set to zero. What allows you to maintain a certain level of PEEP is precisely the minimum expiratory time.

They are used in patients with ARDS, but can cause volutrauma, increased WOB and asynchronies.

In the image below a comparison of the names of the ventilation modes depending on the brand and finally according to the Chatburn taxonomy:

### Mode name comparison

Vendor	Hamilton Medical	Dräger	CareFusion	Covidien	Maquet	GE Medical	Chatburn
Ventilator	G5, S1	V500	AVEA	PB 840	SERVO-i	Carestation	---
Volume-CMV	(S)CMV	VC-AC	Volume A/C	AC-VC	Volume control*	VCV	VC-CMV <sub>s</sub>
Pressure-CMV	P-CMV	PC-AC	Pressure A/C	AC-PC	Pressure control	PCV	PC-CMV <sub>s</sub>
Adaptive-CMV	APVcmv	VC-AC + AutoFlow	PRVC A/C	AC-VC+	PRVC	PCV-VG	PC-CMV <sub>a</sub>
Volume-SIMV	SIMV	VC-SIMV	Volume SIMV	SIMV-VC	SIMV(VC) + PS	SIMV-VC	VC-IMV <sub>s,s</sub>
Pressure-SIMV	P-SIMV	PC-SIMV	Pressure SIMV	SIMV-PC	SIMV(PC) + PS	SIMV-PC	PC-IMV <sub>s,s</sub>
Adaptive-SIMV	APVsimv	VC-SIMV + AutoFlow	PRVC SIMV	SIMV-VC+	SIMV(PRVC) + PS	SIMV-PCVG	PC-IMV <sub>a,s</sub>
Pressure support	SPONT	SPN-CPAP / PS	CPAP / PSV	SPONT PSV	PS/CPAP	CPAP/PS	PC-CSV <sub>s</sub>
Volume support	VS	SPB-CPAP / VS	---	VS	VS	---	PC-CSV <sub>a</sub>
Biphasic	DuoPAP	PC BiPAP	APRV / BiPhasic	BiLevel	Bi-Vent	BiLevel	PC-IMV <sub>s,s</sub>
APRV	APRV	PC-APRV					PC-IMV <sub>s,s</sub>

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**Experts remind us that: there are no bad ventilation modes but ventilation modes used improperly!**

When you have to choose which mode to use you have to consider several factors:

**First:** Is the patient passive, partially active or active?

Modes: should and should not use				
	Mode	passive patient	partially active patient	active patient
Traditional modes	CMV modes	Yes	Yes	Suboptimal
	SIMV modes	Yes (high rate)	Yes	Yes (low rate)
	Support modes	No	No	Yes
Biphasic	ARPV	No	No	Yes
Advanced modes	ASV	Yes	Yes	Yes
	INTELLiVENT-ASV	Yes	Yes	Yes
	PAV or PPS	No	No	Yes
	SmartCare	No	No	Yes
	NAVA	No	No	Yes



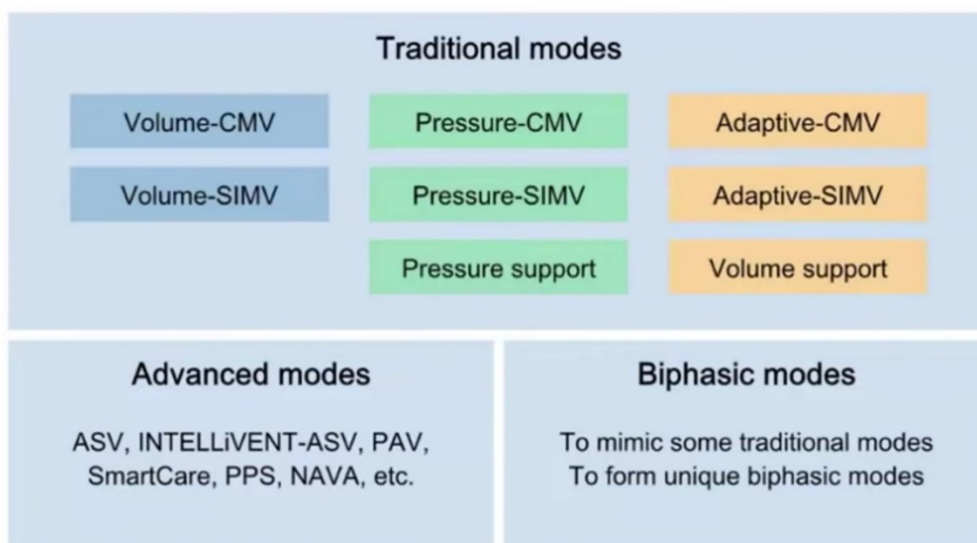
**Second:** which system to use with pressure, volume or adaptive control.

In passive patients the choice could be similar, in active patients the pressure control mode has an edge, allowing greater control of respiratory dynamics and reducing possible ventilator -patient asynchronies.

Adaptive modes, if the Vt is well set, can be superior to VC and PC modes.

**Third:** choose a modality familiar to all clinical staff! This promotes better management of ventilatory parameters throughout the patient's hospital stay.

Then there are intelligent modes such as ASV which manage the best settings for the patient.



In this image the three macro-groups of ventilation modes.

Let's now do a review with relative depth.

## Let's quickly go back to concepts already seen of FR, Vt, MV

The number of breaths we perform in a minute is called **Respiratory Frequency, RR** (Respiratory Rate) in English. With each breath we move a certain volume of air called **Tidal Volume** or **Vt** expressed in ml or liters.

The volume moved in an entire minute is obtained by multiplying the respiratory rate by tidal volume and is called **Minute Volume**, or **MV** and is expressed in ml or liters/minute.

A normal minute volume is between 5 and 8 liters/minute.

<b>Respiratory Rate x Tidal Volume = Minute Volume</b>
--

<b>FR x VT = MV</b>
---------------------

Let's take an example: a ventilator has been set to deliver 10 breaths per minute, we want to know the duration of a single breath.

We divide 60 seconds by 10 and find that each individual act will last 6 seconds. Consider this act from the beginning of the inhalation to the end of the exhalation.

What can this data be used for? Easy: knowing this data and knowing that we have a relationship between inhalation and exhalation based on time, we can calculate how long the inspiratory and expiratory phases last.

This value is called the I:E ratio, we will understand its importance in subsequent discussions.

Know that a ratio considered normal between inhalation and exhalation is 1:2. Having now this data, we can know that a ventilator that delivers 10 breaths per minute will dedicate 2 seconds to inhalation and 4 seconds to exhalation for each breath. Why this difference?

Because inhalation is usually active, and exhalation is usually passive, due to elastic forces that are not as "strong and fast" as those dedicated to inhalation. In short, to eliminate all that VT that we have introduced, we need more time.

The I:E value is not always 1:2, it can also vary a lot and even become inverted 2:1... we will see it later.

## Tidal volume in depth

I continue the discussion on Vt because sometimes I hear some good ones! The Vt is not the volume of air that I introduce with inhalation and although it can often coincide with the real value, the definition is incorrect. I want to explain it to you the way I like it: if you open the door and close it it's not like you've opened and closed two doors!!! There is always one door!

Vt is the air moved in a single respiratory act, inhalation and exhalation. This doesn't mean that if I inhale 500 ml and expel 500 ml, I have moved a total of 1000 ml! Because I only put 500 ml into play; this is why Vt is often defined as the volume of air inspired, it is not such a serious error, but you need to know the difference. Now hold on tight because in ventilator you will often, if not always, find a VTi and a VTe. They refer precisely to the volume of inhaled and exhaled air, usually



these values are very similar, and in mechanical ventilation, they differ due to leaks or compliance of the circuit.

It is interesting to know that these differences self-level over time and, if they are due to losses, the two values are to be considered identical. If this were not the case and the system was perfectly sealed, a greater VTe would lead, sooner or later, to the lung collapsing and a greater VTi to exploding!

Is the discussion about Vt over here? But whenever!

Vt is a very important parameter for mechanical ventilation. In many cases, in fact, it is a parameter that is set by the doctor to guarantee an adequate Minute Volume for the patient. But how the doctor chooses which Vt to set is more complicated to explain.

...No, don't worry, it doesn't concern the patient's date of birth nor the last three digits of the mobile phone number!!!

We saw in the first part that the normal Vt of a man weighing around 70kg is around 500 mL and I have just answered the previous question; mainly, the Vt is set based on the patient's ideal weight. However, there is a lot of data to consider that the doctor uses to refine the setting of this parameter, to be clear, it is not that he takes a table and ends there. Behind that set value there is a careful clinical evaluation of each individual patient, a setting which must then be re-evaluated and adjusted based on the results obtained.

...To explain it to you, it's not like you buy a ventilator on Aliexpress, you set a Vt and connect the cat to it "to see the effect it has"! I'm telling you, that cat doesn't end well!

If the ventilation mode requires Vt as a parameter to be set, a first step to take is to calculate it based on the ideal body weight.

### Calculation of ideal body weight:

Women	$(\text{Height in cm} - 152.4) \times 0.91 + 45.5$
Men	$(\text{Height in cm} - 152.4) \times 0.91 + 50$

There are different formulas to calculate the ideal body weight, there are also other factors that could be used for a more correct evaluation.

Let's take two examples:

Woman 171 Cm –  $171 - 152.4 = 18.6$  | multiply by 0.91 = 16.92 | add 45.5 = 62.4 Kg

Man 183 Cm –  $183 - 152.4 = 30.6$  | multiply by 0.91 = 27.84 | add 50 = 77.8 Kg

Once the ideal weight has been obtained, we calculate the Vt based on a parameter used precisely for this purpose in mechanical ventilation. Here too, and what am I telling you to do!, the resuscitation doctor intervenes like a laser to choose the most correct setting.

This is the value to set to obtain a correct Vt:

6 – 8 ml/Kg

Putting 8 mL/Kg, the correct Vt value for the two examples given above will be:

Women	$62.4 \text{ Kg} \times 8 \text{ mL} = 499.2 \text{ mL}$
Men	$77.8 \text{ Kg} \times 8 \text{ mL} = 622.4 \text{ mL}$

By setting 6 mL/Kg the Vt will be this:

Women	62.4 Kg x 6 mL = 374.4 mL
Men	77.8 Kg x 6 mL = 466.8 mL

The values obtained will then be rounded. A little curiosity: for patients with ARDS, the values to use are usually lower, between 4 and 8mL/Kg

**In patients with ARDS, a high Vt can cause problems; for this reason, they must always be ventilated with low volumes, always!**

Both Vt and MV are important parameters, but of the two, MV is the more important. By obtaining an adequate MV we can make our patient improve and evaluate a correct setting through the ABG results. Remember that hyperventilation, and therefore an increase in MV, leads to a reduction in CO<sub>2</sub>; on the other hand, hypoventilation, and therefore a reduced MV, leads to an accumulation of CO<sub>2</sub>. In the first case, a picture of alkalosis can develop, in the second of acidosis.

High Minute Volume	Hyperventilation ↓ PCO <sub>2</sub>
Low Minute Volume	Hypoventilation ↑ PCO <sub>2</sub>

Vt is a parameter that we thought we knew well, but in mechanical ventilation it reserved a lot of surprises for us. Let's proceed with other concepts.

### Five very important variables

Continuing the discussion on mechanical ventilation, there is some information that can help to better understand the topic. This information was also taken from Hamilton Medical material, I have tried to summarize it to the best of my ability.

Thus, we must know that there are 5 variables that make mechanical ventilation an efficient system, these are:

**Triggering:** the mechanism that starts the inspiratory cycle.

**Cycling:** the mechanism that ends the inspiratory cycle.

**Control:** The method used to determine the volume or pressure of inspiration.

**Limit or Targeting:** The maximum or minimum value that the ventilator must adhere to.

**PEEP:** positive end-expiratory pressure.

If we take into consideration the types of breathing that can be achieved through mechanical ventilation, we can have different ones depending on whether we use pressure-controlled, volume-controlled or adaptive ventilations. These can also be controlled, assisted or supported. Putting these breath characteristics together we obtain only eight possible breath types.

We can see them in the images below:

	Volume breath	Pressure breath	Adaptive breath
Controlled breath	✓	✓	✓
Assisted breath	✓	✓	✓
Supported breath	—	✓	✓

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MEDICAL

Mechanical breath types	Volume breath	Pressure breath	Adaptive breath
Controlled breath	Volume control breath	Pressure control breath	Adaptive control breath
Assisted breath	Volume assist breath	Pressure assist breath	Adaptive assist breath
Supported breath	—	Pressure support breath	Adaptive support breath

HAMILTON  
MEDICAL

The minus symbol corresponding to volume-controlled supported breathing cannot be managed by the ventilators and therefore simply does not exist.

Here are the eight breath types available in mechanical ventilation, listed together with the targeting, cycling, controlling and targeting values.

Nice scheme from Hamilton Medical, right?

But if you don't know what these terms are, you don't understand anything!

Now I'll explain everything, please be patient for two seconds.

	Triggering	Cycling	Controlling	Targeting
Volume control breath	Time	Time	Volume	Tidal volume
Volume assist breath	Flow or pressure	Time	Volume	Tidal volume
Pressure control breath	Time	Time	Pressure	Inspiratory time
Pressure assist breath	Flow or pressure	Time	Pressure	Inspiratory pressure
Pressure support breath	Flow or pressure	Flow	Pressure	Inspiratory pressure
Adaptive control breath	Time	Time	Pressure	Target volume
Adaptive assist breath	Flow or pressure	Time	Adaptive	Target volume
Adaptive support breath	Flow or pressure	Flow	Adaptive	Target volume

HAMILTON  
MEDICAL

## The Trigger

In mechanical ventilation there is "something" that starts the respiratory act, and to be more precise, inhalation. This "something" has a name, and it is called **trigger**. When a ventilator independently manages breathing acts based on the frequency we have set for it, it starts each inhalation in the way we have seen above. With a breathing rate of 10 breaths/minute, the respirator will begin an inhalation every 6 seconds. This type of trigger is called a **timed trigger**. Whatever happens, the timed trigger will start the inhalation at regular intervals dictated by the set respiratory rate.

There are two other types of triggers (to be more precise, three) which are activated by the patient and in particular by the attempt, whether finalized or not, to carry out a spontaneous breath.

The patient can activate the trigger in two different ways: pressure, flow and, but rarely, volume.

Pressure trigger:

The pressure one responds to a drop in pressure between 2 and 3 cmH<sub>2</sub>O, rarely more than 3; the ventilator recognizes this pressure change and delivers the breath.

In practice, this happens: the ventilator, which is always ready to verify that the pressures delivered are correct, if it notices that there is a depression in the system and if it is enabled to do so, allows the patient to take a spontaneous breath .

The pressure trigger is adjusted based on the basic pressure level, called the baseline.

If we are in the presence of a PEEP of +5 cmH<sub>2</sub>O and we set a sensitivity of 2 cmH<sub>2</sub>O, then the ventilator will support the patient's spontaneous breathing when a pressure of +3 cmH<sub>2</sub>O is reached. This is because the baseline of +5 was added to a negative pressure of 2 that the patient developed.

## Flow Trigger:

To understand the flow trigger, we need to understand how the ventilator performs exhalation. If the exhalation valve is fully open, the outflow will be as rapid as possible.

The ventilator then adjusts the opening of the expiratory valve to maintain the set PEEP pressure without reaching the zero baseline.

This described, is the exact mechanism that allows the ventilator to maintain a PEEP in the pulmonary system. At the same time, the ventilator maintains a minimum inspiratory flow, and it is precisely this that allows the flow trigger to work.

If the patient is not breathing at this time, inflows and outflows between inspiratory and expiratory flow are identical. If the patient inhales, the exhalation flow is reduced, triggering the trigger mechanism.

The flow trigger is more difficult to explain than the pressure trigger. In this case, the ventilator maintains a constant flow between him and the patient. If he sends 5 liters per minute and receives 5, it means that the patient is not trying to breathe. If only 3 liters fit, the patient must have participated in varying this flow. Usually, you set 2 or 3 liters per minute as the flow trigger. The difference between the two types of triggers lies only in how the ventilator notices the patient's request: through a change in pressure or flow.

One would think that 2 or 3 liters/minute is a lot for a patient to trigger this trigger; on the other hand, these are patients in respiratory failure. However, we must think in terms of time, and we must consider the patient's inspiratory request practically instantaneous.

We consider that the patient's request for air has a time of 0.2" - 0.3" and we relate it to the set trigger value, so as to obtain this value in ml.

3 liters/minute is equivalent to 3000 mL/minute, we must divide this value by the time.

We know that in 1 minute there are 60 seconds, let's divide them by 0.3 second and get this value:  $60:0.3 = 200$  we have to modify those 3 liters or 3000 ml with a factor of 200! The value obtained will therefore be 200 times smaller and, if you haven't noticed, the volume obtained is the volume that the patient "moved" to trigger the spontaneous breathing act.

This is the value we must use to divide the volume obtained like this:

$$3000 : 200 = 15\text{ml}$$

At minimum settings it becomes  $2000 \text{ mL} : (60:0.2) = 2000 : 300 = 6.6 \text{ mL}$

What have we learned? That the patient's "trigger" request with a flow setting is between 6.6 and 15 mL! A value that the patient is certainly able to "move" and which gives us a much more realistic image than 2 or 3 liters/minute.

In some ventilators it is possible to choose a flow or pressure trigger regardless of the ventilatory mode used. For example, in a PCV you can use a flow or pressure trigger. Not all ventilators, especially older ones, have this option.

The trigger, therefore, simply put, is the way in which the ventilator starts inspiration, whether the time decides it or the patient.

There is a third way that is activated by the patient and is called the NAVA system.

NAVA = Neurally Adjusted Ventilatory Assist

It is a type of ventilation that is "triggered" by a nervous impulse from the patient, taken at the level of the phrenic nerve via a special nasogastric tube equipped with sensitive electrodes, which capture the nervous stimulus upon inhalation.

Compared to other triggers, this is by far the best system, allowing perfect synchronization with the patient's request for spontaneous breathing.

A high trigger sensitivity can lead to autotriggering, i.e. breaths that are not actually required. This may be due to circuit leaks or condensation in the circuit, and not be caused by patient effort. In

this case, it is necessary to resolve the cause, and if this is not possible, reduce the sensitivity of the trigger.

If the sensitivity is too low, there is a risk of trigger losses, and the patient requiring spontaneous breaths that will not be delivered.

In this case the patient's effort is not sufficient to trigger breathing.

## Cycling:

The term cycling defines the end of inhalation and the transition to exhalation. Remember that if we have a timer, we also have a known full respiratory cycle length. If cycling occurs later, the  $T_i$  becomes larger, and the  $T_e$  is reduced while keeping the total cycle time the same.

Therefore, cycling represents the end of inhalation, that is, as already mentioned, the moment of transition between inhalation and exhalation. In mechanical ventilation, cycling can be performed by the ventilator or by the patient.

Cycling is also timed when we have set a breathing rate or inspiratory time. In other conditions, cycling is initiated when we have reached a certain percentage of flow or pressure. If a patient takes his own spontaneous breaths, he will be the one to command the transition to exhalation.

Cycling can be activated in three different ways:

- With an inspiratory time ( $T_i$ ) that can be set for both volume and pressure: in this case, a time is set at the end of which the ventilator cycles towards exhalation.
- Through the I:E ratio (I:E Ratio): in this case, having set a respiratory frequency and an I:E ratio, we will have that the duration of the respiratory act will be what determines the cycling.

For example, for 10 RR we have a time of 6 seconds per complete act. With an I:E ratio of 1:2, we have a  $T_i$  duration of 2 seconds and a  $T_e$  duration of 4 seconds.

This method can also be applied to volume or pressure ventilation.

- Using the peak flow value (Peak Flow) as a reference value: in this case, the ventilator will cycle when the inspiratory flow drops to a value, usually as a percentage, of the Peak Flow. Taking the Peak Flow value as 100%, when this has dropped to values usually between 25% and 30%, cycling will occur.

## Pressure or volume control

Control is how the ventilator delivers the breath. It can do it by pressure or by volume, the ventilator can control one at a time, never at the same time.

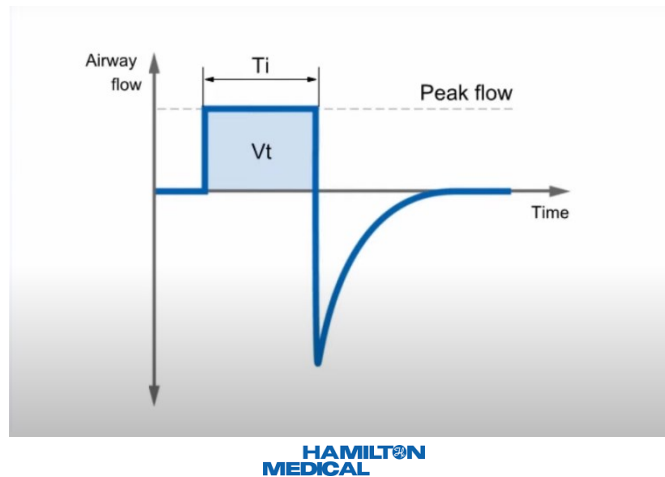
If the ventilator is in volume control, it will vary the pressure to obtain that volume.

If the ventilator is in pressure control, it will vary the volume to obtain that pressure.

The pressure and volume will change based on the compliance and resistance characteristics of the pulmonary system. Compliance and resistance are also variable characteristics.

## Volume control.

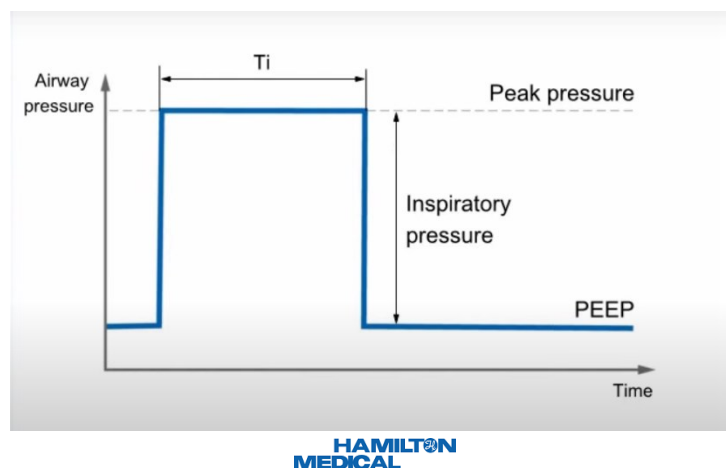
In a volume control the ventilator has as settings a  $V_t$ , the  $T_i$  and the Peak Flow.



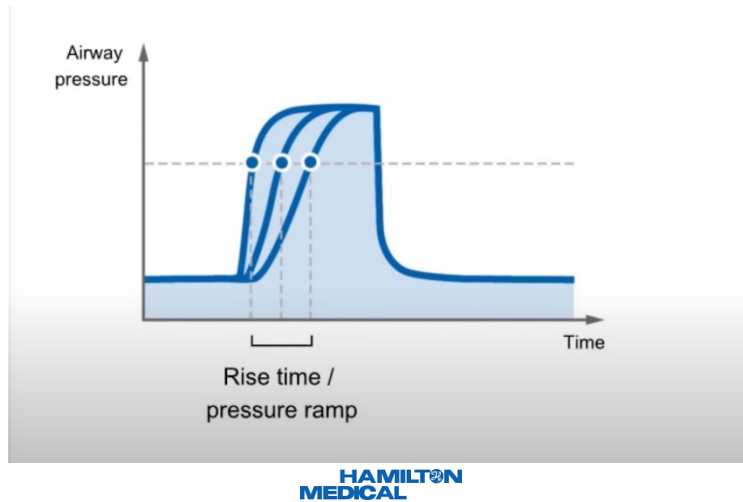
With this mode, in which the curve is square, we have control of the  $V_t$  and MV. The curve in the image is the flow curve, and we are talking about volume control mode. It is the mode where the patient has the most difficulty, if active, in synchronizing with the ventilator. To be clearer, it is the mode where there are greater asynchronies. Due to the compression of the gases in the circuit, the volume delivered may not correspond to the desired one. For example, by setting 500 ml, the patient could actually receive 450 ml. This deviation must be corrected with the circuit test, which calculates its compliance and therefore adjusts the work of the ventilator to obtain the required  $V_t$ . Furthermore, in this mode it is not possible to compensate for circuit losses, because the ventilator always delivers the same  $V_t$ . The peak pressure is variable based on the values of Peak Flow, compliance and resistance. Remember that high airway pressure can ruin your lungs.

Pressure control.

The ventilator delivers the flow at a set pressure, the shape of the pressure in this case will be square:



The one in the image is the pressure curve and, since we are talking about a pressure control mode, the relevant curve will be square. The inspiratory pressure is to be understood as above the PEEP. This pressure, when applied in a support mode, is called support pressure. We can also manage how pressure is delivered via ramp or rise time controls. Here is the ramp:

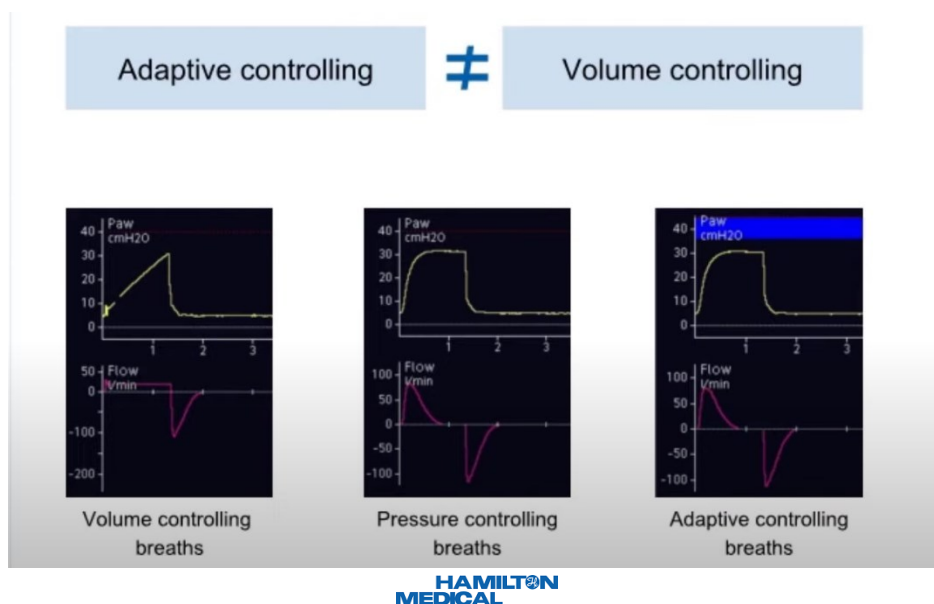


Changing the slope of the ramp corresponds to how “softly” the pressure is delivered into the pulmonary system.

In this mode, we have greater synchrony, and loss compensation.

On the other hand,  $V_t$  is variable. It is therefore necessary to set alarms on the  $V_t$ .

There is a hybrid of the two, called adaptive where the pressure varies from breath to breath to adjust the  $V_t$

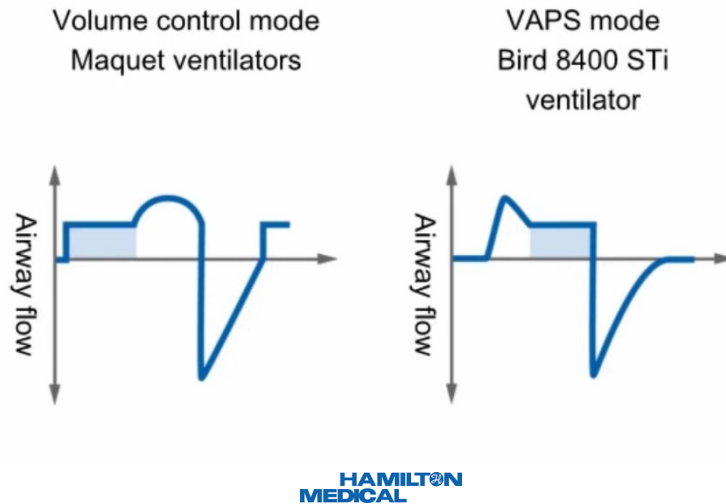


The adaptive mode maintains a more stable  $V_t$ , with the advantages of pressure control.

If we set an inadequate Tidal Volume in an active patient, he may take a greater number of breaths and require higher  $V_t$ . In this case the ventilator will gradually reduce the pressure. This could lead the patient to breathe too much and do all the work himself, at the same time the work done by the ventilator would be reduced.

Then there are dual modes, such as VAPS and Maquet<sub>TM</sub>'s volume control mode, Robert Chatburn defines them as dual control.





## Targeting or limiting

By limit or targeting we mean identifying the objective that the clinician has set on the ventilator, in this case we mean the objective to be achieved.

So, if we set a volume controlled mode (VCV), our limit, targeting or goal to achieve will be the  $V_t$ .

**The target is  $V_t$  in volume controlled mode**

If the clinician decides to use a pressure-controlled mode (PCV), our limit, targeting or goal to achieve will be the inspiratory pressure.

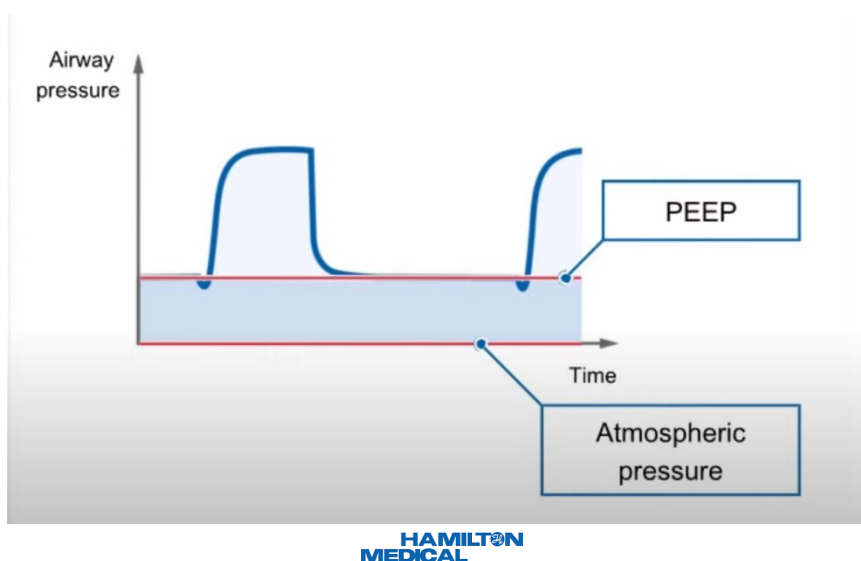
**The target is inspiratory pressure in a pressure-controlled mode**

Using an adaptive type mode, our limit, targeting or objective will still be the  $V_t$ .

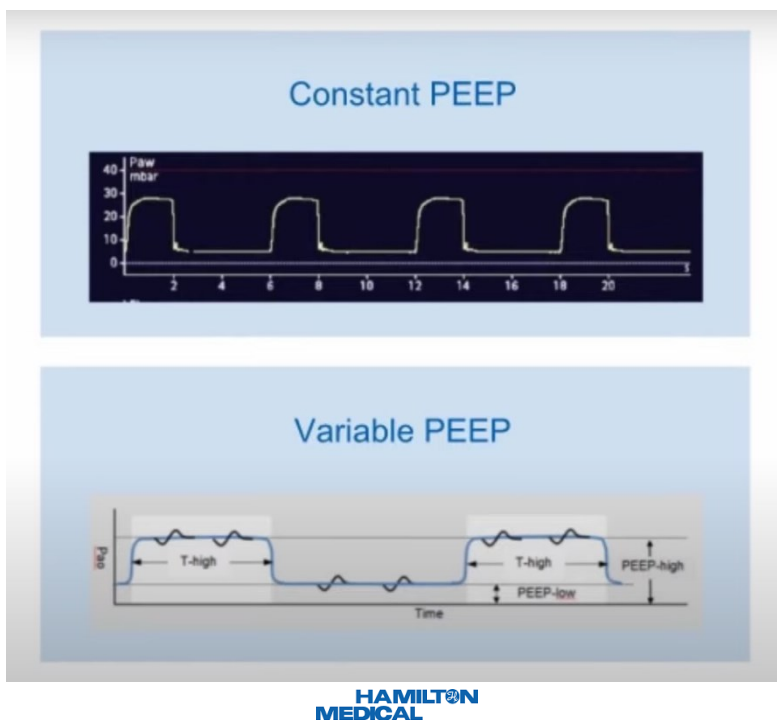
**The target is  $V_t$  in adaptive mode**

When the target or limit is reached, the ventilator blocks the inspiratory flow. This does not mean that you switch from inhaling to exhaling as soon as you reach the limit.

PEEP is variable number 5:

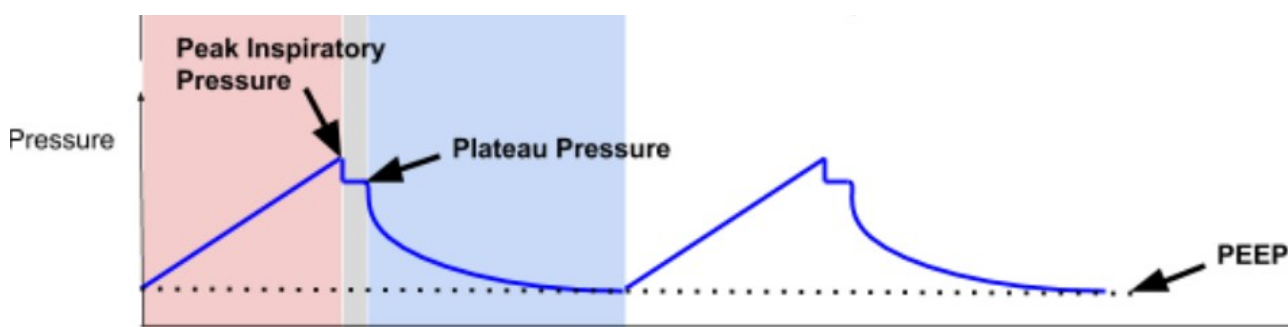


PEEP improves FRC, oxygen exchange, alveolar recruitment and improves lung compliance. Values between 3 and 5 are recommended for all intubated patients. This is considered a minimum or low value. High PEEP is used in patients with ARDS or restrictive lung disease. PEEP is present in all ventilatory modes. As far as possible, it is recommended to avoid setting PEEP to zero. Only the biphasic mode has part of the PEEP at zero, but its mechanism is maintained by the brevity of the exhalation. Furthermore, in biphasic, PEEP varies at two different levels, in all other cases it has a constant value.



### The pressures: PIP, PEEP, Plateau, Media, Driving Pressure and other...

This image will be useful for the following explanations:



This is the pressure-time curve, the first one at the top of the ventilator. Observe the curve, it rises up to a certain point, when it reaches the maximum, we have the **PIP** or **Peak Inspiratory Pressure**. We are in the first part of the respiratory act and precisely in the inhalation. The pink part of the curve represents the pressure rising inside the alveoli.

This is followed by the small gray part which represents the stabilization of pressures at the alveolar level. This level of pressure is called **Plateau Pressure**, in Italian *Pressione di Plateau* or,

amicably called Plateau. Once this short pause at the end of inspiration (not always present or visible) is over, the actual exhalation begins. As you can see the pressure drops gradually and the duration of exhalation is longer than inhalation. Now look at the continuous baseline, its pressure level is 0 (zero) while the dotted line represents the "famous" **PEEP or Positive End Expiratory Pressure**.

Print it well in your head: the colored parts are those in which we have a flow, therefore the movement of a volume of air. The gray part, however, represents a moment in which we have no flow, as in plateau pressure.

You can notice that these two breaths start and end with a pressure that does not reach zero, but the PEEP value.

On with the definitions:

### **PIP: Peak Inspiratory Pressure**— Peak Inspiratory Pressure

**PIP is pronounced like PEEP but they are not the same thing!**

It represents the maximum pressure reached during inspiration in the alveoli and airways. PIP represents the pressure that must be applied to the airways to allow air to reach the alveoli.

Therefore, PIP can be considered a measure of airway resistance and alveolar elasticity.

In this sense, it is correct to talk about lungs, meaning alveoli and airways, when talking about PIP. High peak pressure is harmful to the lungs and can cause barotrauma.

This is why it is a value that must be monitored, to which an appropriate alarm level must be assigned.

As normal PIP values we can consider 20-30 cmH<sub>2</sub>O, the maximum values not to be exceeded are 40-45 cmH<sub>2</sub>O, both for an adult patient.

<b>Normal PIP</b>	<b>20-30 cmH<sub>2</sub>O</b>
<b>Maximum PIP</b>	<b>40-45 cmH<sub>2</sub>O</b>

Peak inspiratory pressure is the maximum pressure reached at the end of inspiration.

**PIP is the pressure inside the lungs, therefore the airways + the alveoli**

It is a value measured during mechanical ventilation and depends on the type of ventilation used, on the resistance and lung compliance, but not only.

Here's what it depends on:

Airway resistance

The elasticity of the lungs

Chest compliance

The current volume

The plateau pressure

Possible causes of increased PIP are:

Airway obstruction, secretions, mucus plug

Bronchospasm

Increased inspiratory flow

Cough

Kink in the endotracheal tube or patient biting the endotracheal tube

PIP is an important parameter to monitor during mechanical ventilation, as it can provide information on the patient's respiratory status.

In general, a high PIP can indicate:

- Airway obstruction
- Pneumothorax
- Pulmonary edema
- Acute respiratory distress syndrome

A PIP that is too low, however, can indicate:

- Respiratory failure
- Pulmonary distension

## **PLATEAU: Plateau Pressure (Pplat)**

It represents the pressure at the end of inspiration which stabilizes at the alveolar level, it does not indicate pressures at the level of the respiratory tract.

Plateau pressure is not affected by airway resistance because **it is measured in the absence of flow**.

It is not always possible to see the plateau pressure in the ventilator curves. To measure it correctly, we need to perform a maneuver. This maneuver (Inspiratory pause) gives us the real plateau pressure and consists of interrupting the ventilatory flow for a pre-established time, usually from 0.5 to 2 seconds. This allows the pressure to stabilize and the ventilator to calculate its value.

Almost all ventilators allow this maneuver easily, just by pressing a button.

Plateau pressure is determined by a number of factors, including:

- The elasticity of the lungs
- Chest compliance
- The current volume
- The PEEP
- The respiratory rate

Higher tidal volume or PEEP will give a higher plateau pressure, while higher static compliance will give a lower plateau pressure.

Plateau pressure is an important parameter to monitor during mechanical ventilation, as it can provide information on the patient's respiratory status.

In general, a high plateau pressure can indicate:

- Pneumothorax
- Pulmonary edema
- Acute respiratory distress syndrome

A plateau pressure that is too low, however, can indicate:

- Pulmonary distension
- Air leak

The normal value of plateau pressure varies depending on the patient and the ventilation mode used. In general, a plateau pressure between 25 and 35 cmH<sub>2</sub>O is considered normal.

<b>Normal plateau pressure</b>	<b>25-35 cmH<sub>2</sub>O</b>
--------------------------------	-------------------------------

The plateau pressure can also be calculated in this way:

$$\text{Plateau pressure} = (\text{tidal volume} / \text{static compliance}) + \text{PEEP}$$

For example, let's say a patient has the following:

Tidal volume: 500 ml

Static compliance: 20 mL/cmH<sub>2</sub>O

PEEP: 5 cmH<sub>2</sub>O

To calculate plateau pressure, you can plug numbers into the formula, which would look like this:

$$\text{Plateau pressure} = (500 \text{ ml} / 20 \text{ ml/cmH}_2\text{O}) + 5 \text{ cmH}_2\text{O} = 30 \text{ cmH}_2\text{O}$$

Two pieces of information to know: the plateau pressure is read in the pressure curve and is almost never visible in the mechanical ventilation curves. Its correct measurement must be carried out through the inspiratory pause. It always has a value lower than the PIP and higher than the PEEP.

### **Mean Airway Pressure: Paw**

The mean airway pressure (Paw) is given by the average of all the pressures present during the complete respiratory act. It is calculated as the sum of plateau pressure and positive end-expiratory pressure (PEEP).

So the Paw is:

### **The average pressure of all pressures present in the pulmonary system throughout the respiratory system**

Mean Airway Pressure (P<sub>aw</sub>)

Mean airway pressure (P<sub>aw</sub>) is determined by three factors: peak inspiratory pressure (PIP), the fraction of time spent inhaling (represented by TI/Ttot, where Ttot is the total duration of a single breath cycle), and positive end-expiratory pressure (PEEP).

Calculation of Mean Airway Pressure:

- Constant flow-volume ventilation: When the airway pressure waveform resembles a triangle (common in this mode), P<sub>aw</sub> can be calculated using this formula:

$$P_{aw} = 0.5 (PIP - PEEP) (TI/T_{tot}) + PEEP$$

- Pressure ventilation: When the airway pressure waveform is more rectangular (typical in this mode), P<sub>aw</sub> can be estimated with this formula:

$$P_{aw} = (PIP - PEEP) (TI/T_{tot}) + PEEP$$

Mean Alveolar Pressure (P<sub>alv</sub>):

It's important to note that mean alveolar pressure (P<sub>alv</sub>) might differ from mean airway pressure (P<sub>aw</sub>), especially when there's a significant difference between inspiratory airway resistance (RI) and expiratory airway resistance (RE). This situation is common in patients with lung diseases.

The formula to estimate the difference between P<sub>alv</sub> and P<sub>aw</sub> is:

$$P_{alv} = P_{aw} - (V'E / 60) (RE - RI)$$

where V'E represents the expiratory flow rate.

We can calculate an **estimate** of the average airway pressure in mechanical ventilation, using the following simplified formula:

Mean Airway Pressure = Airway Resistance x Airflow

We have a high Paw with:

Airway obstruction

Pneumothorax

Pulmonary edema

Acute respiratory distress syndrome

A low PAW, instead for:

Pulmonary distension

Atelectasis

Paw has normal values of 10-15cmH<sub>2</sub>O. In ARDS 15-30 cmH<sub>2</sub>O, in COPD 10-20 cmH<sub>2</sub>O

<b>Normal Paw = 10-15cmH<sub>2</sub>O</b>
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Here are some factors that can influence Paw:

Airway resistance

Elasticity of the lungs

Chest compliance

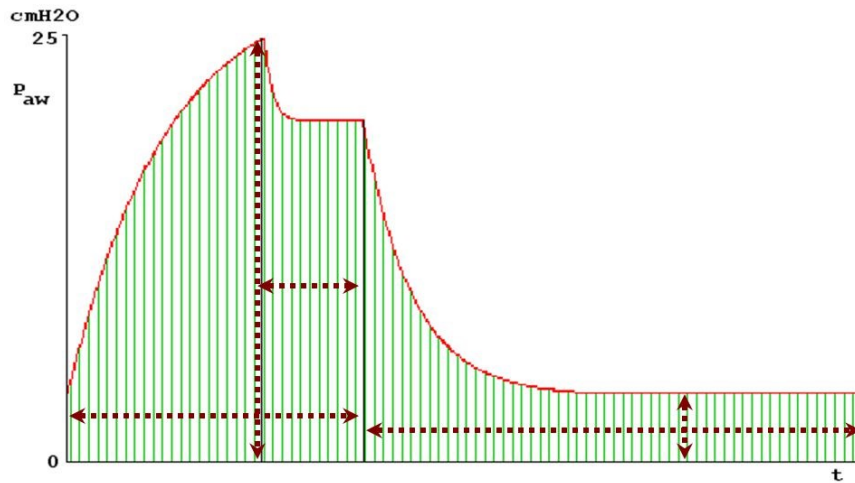
Tidal volume

PEEP

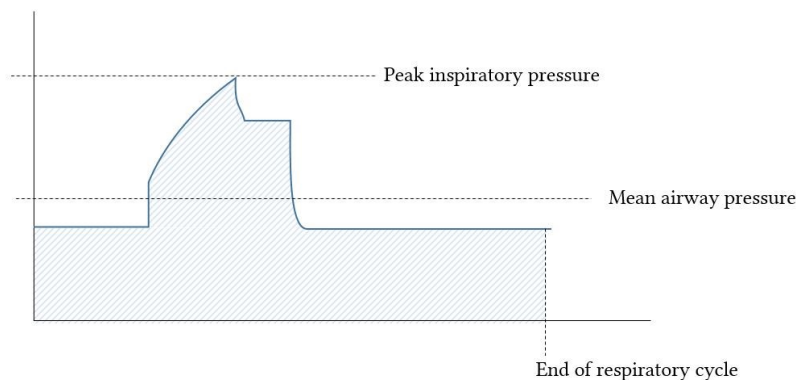
Breath frequency

Inspiratory time

Mean Airway Pressure  $\cong$  14 cmH<sub>2</sub>O



Since it has a value between 0 and PIP, you will often find it shown as a line between the two, like this:



To measure it we can take the entire respiratory act, measure the pressure at regular time intervals and, adding these values, divide by the number of time intervals.

An alternative way is to measure the entire occupied area under the pressure/time curve. The entire area in the image above is the part highlighted in light blue.

Now a little curiosity: since 60-70% of breathing occurs "without flow", and only at the PEEP level, the Paw value is often close to the PEEP value itself.

By "without flow" we mean that part that is below the PEEP line. Of course this is not always the case.

The average pressure is also linked to the PIP and Plateau pressure values, the higher these are, the higher the Paw will be and vice versa.

An increase in mean airway pressure improves oxygenation. In fact, a high Paw increases alveolar pressure, which in turn increases the diffusion of oxygen from the lungs to the blood. This can improve oxygenation, especially in patients with respiratory failure.

A high PAW also improves ventilation. In fact, high Paw also increases tidal volume, which is the amount of gas inhaled and exhaled every minute. This can help remove carbon dioxide from the lungs, improving ventilation.

## PEEP

**PEEP** or Positive End Expiratory Pressure in Italian Positive End Expiratory Pressure is the pressure that remains inside the pulmonary system at the end of exhalation. The function of PEEP is to keep the alveoli open, and, in the initial phase of ventilation, it helps the opening of closed alveoli. Closed alveoli are a big problem because they do not participate in gas exchange, causing and/or worsening a condition of respiratory failure.

Opening the alveoli through pressure that remains even after exhalation is called alveolar recruitment. It is understood that little by little, when the number of "ventilating" or recruited alveoli increases, the patient's respiratory condition will also improve. Furthermore, the use of PEEP helps to reduce the patient's need for oxygen.

The PEEP therefore:

Improves oxygenation

Keeps the alveoli open

Improves cardiac function of the left ventricle

Reduces the work of breathing

The PEEP value is usually not less than 5 Cm H<sub>2</sub>O; the aim, as mentioned before, is to never let the pressure in the respiratory system reach 0 (zero).

If this were to happen, the PEEP value changes name and is called ZEEP or Zero End Expiratory Pressure.

In CPAP mode the pressure value that we enter as the baseline or base value is the PEEP value. In this case therefore there will not be a PEEP value to enter, but the CPAP value will be the PEEP. In this case it will remain, not only after exhalation, but throughout the entire respiratory cycle. We reduce PEEP in case of pneumothorax and to reduce intracranial pressure.

## Driving Pressure

**Driving pressure** is less famous than PEEP, but it is quickly gaining fame in the world of mechanical ventilation. Previously, a correct PEEP setting, especially with patients with ARDS, was the gold standard. Now it is the driving pressure which, if well adjusted, contributes to improving the outcome of patients, reducing mortality by several percentage points.

The driving pressure is obtained by taking the plateau value and subtracting the PEEP. The value thus obtained is more useful for patient monitoring than PEEP alone.

<b>Driving Pressure = Plateau Pressure – PEEP</b>
---

<b>DP = Pplat – PEEP</b>
--------------------------

**Driving pressure is the difference between plateau pressure and peep pressure**

The driving pressure also reflects the relationship between tidal volume (V<sub>t</sub>) and compliance of the respiratory system.

The driving pressure is a predictive data of mortality and is a key point in treatment with protective mechanical ventilation. Studies demonstrate that increasing PEEP or decreasing tidal volume are associated with better survival if, together with them, they lead to a reduction in driving pressure!



<https://rc.rcjournal.com/content/64/8/1017#:~:text=Driving%20pressure%20is%20easily%20calculated,current%20lung%20protective%20ventilation%20strategies.>

Since driving pressure is related to tidal volume, plateau pressure and compliance, reducing tidal volume leads to a reduction in driving pressure. Although the data obtained so far in patients at risk indicate 15 Cm H<sub>2</sub>O as the limit value, we are increasingly moving towards values of 13 - 14 Cm H<sub>2</sub>O.

Driving pressure values should be kept below 14 -15 cm H<sub>2</sub>O.

All these pressures and characteristics of the respiratory system are linked to each other. Just out of curiosity, look at these correlations, you don't have to memorize them:

Driving Pressure = (Pplat – PEEP)

Static compliance = Vt/(Pplat – PEEP)

Static Compliance = Vt/Driving Pressure

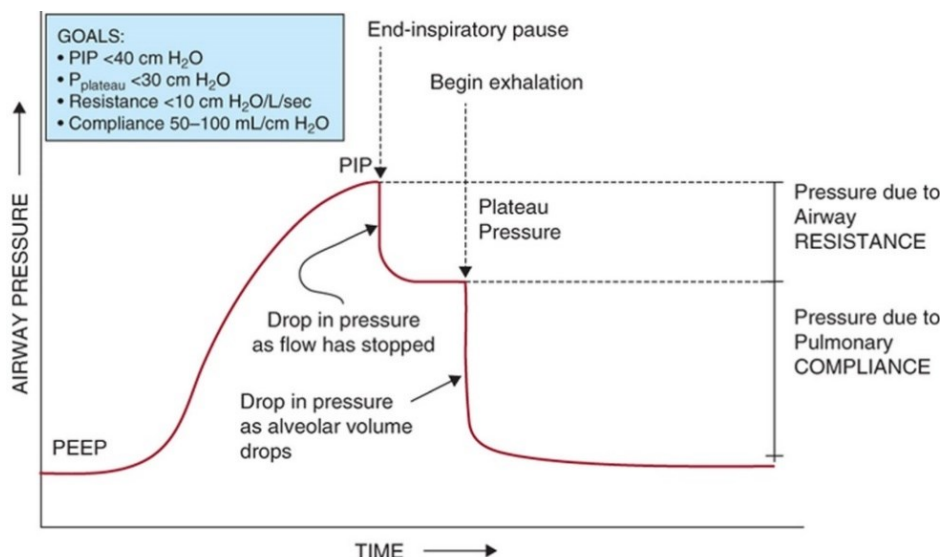
Driving pressure = Vt/Static Compliance

Driving Pressure is the pressure required for alveolar opening.

In other words, driving pressure indicates the pressure that must be applied to overcome airway resistance and intrapleural pressure, in order to open collapsed alveoli.

Now hold on tight, as we try to better understand what these pressures represent and, above all, their correlations.

Looking at the image below, we see that driving pressure is referred to as pressure due to lung compliance. We can therefore, indicatively, say that the driving pressure is an indicator of lung compliance.



If you look further up, the difference between PIP and Plateau Pressure has the characteristic of referring to airway resistance. So:

$$\begin{aligned} P_{\text{plat}} - P_{\text{EEP}} &= \text{value of lung compliance} \\ P_{\text{IP}} - P_{\text{plat}} &= \text{value of upper airway resistance} \end{aligned}$$

I want to repeat myself once again:

All data, speeches and examples in this text of mine are to be taken as indicative.

Thus, a value indicated as “normal” may not be accurate.

The information on what the curves indicate, since it is very simplified, may have some gaps and must be read critically. They have the sole purpose of making it clear in broad terms what they indicate and certainly do not have the presumption of being able to be applied to every clinical condition.

Use these sheets for the sole purpose of creating a starting point!

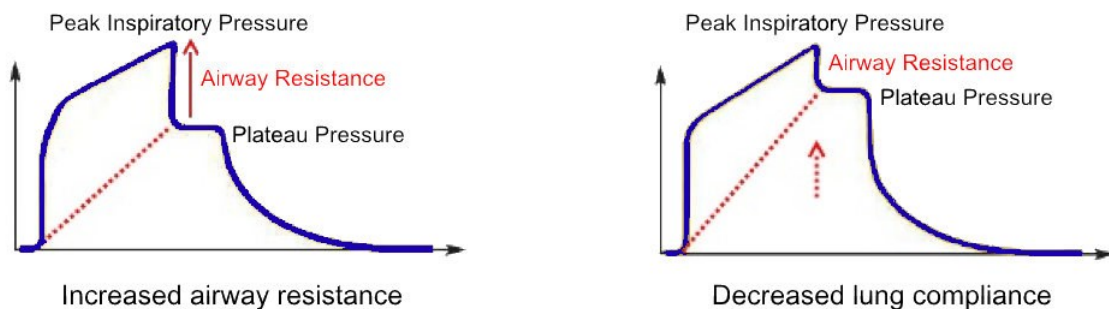
## Correlation between PIP and Plateau Pressure

Usually, the PIP has a value  $<40$  cmH<sub>2</sub>O, the Pplat is less than 30 cmH<sub>2</sub>O.

Their difference indicates the resistance of the upper airways, in this case it is 10 cm H<sub>2</sub>O.

While the sector between Pplat and PEEP is called Driving Pressure, the sector between PIP and Pplat does not have a specific name.

Now following these reasonings, if you look at the image below and the previous one, you can notice that this segment is an indicator of airway resistance.



So the PIP – Pplat represents the resistance, now let's go into more detail.

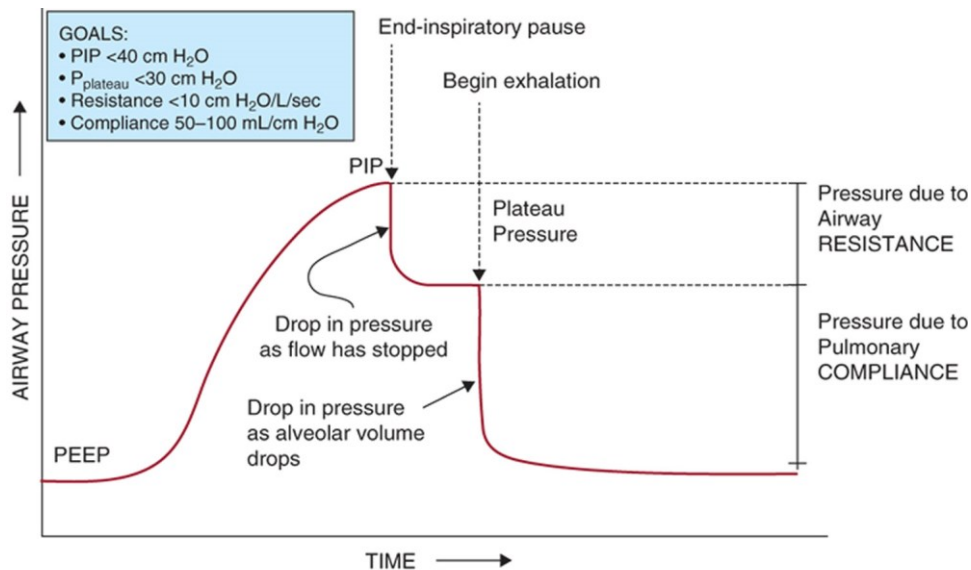
**To be precise PIP – Pplat depends on resistances and flow.**

So, if we have an elevated PIP and a normal Pplat, we must think of a problem in the upper airways or upstream of them. It could be a problem with the tube, the circuit, the ventilator itself or the presence of secretions.

This picture indicates an increase in upper airway resistance.

So remember, the higher the difference between PIP and Pplat, the higher the upper airway resistance will be.

A patient with high PIP and high Pplat with a small difference between the two, however, indicates a patient problem, such as pulmonary edema, ARDS, or pneumothorax.



Basically, you can think of it this way, if the difference between PIP and Pplat is high, there will be high resistance in the upper airway. If you have a large difference between Pplat and PEEP, the problem lies in the terminal airway

In fact, plateau pressures above 35 have been associated with barotrauma.

This indication is as simple as it is not very precise, there are many factors that contribute to making these intervals different. With good approximation, however, we can consider my statement correct:

**PIP - Pplat = resistances**

**Pplat - PEEP = compliance**

Remember that we have two types of compliance which are different from each other, namely:

**The dynamic compliance formula is  $VT/PIP - PEEP$**

**The static compliance formula is  $VT/ Pplat - PEEP$**

Dynamic compliance, therefore, includes alveolar compliance + airway resistance. This is because it is the result of moving air meeting resistance.

Static compliance is alveolar compliance only and is measured in the absence of flow.

From here we understand that peak pressure reflects airway resistance and alveolar compliance.

Plateau pressure reflects only alveolar or static compliance.

Let's see it better:

Peak pressure reflects airway resistance and alveolar compliance. This is because peak pressure is the maximum alveolar pressure during inspiration. Airway resistance is the additional pressure that must be applied to overcome airway resistance and open collapsed alveoli. Alveolar compliance is the measurement of the ability of the lungs to expand when pressure is applied.

Plateau pressure reflects only alveolar or static compliance. This is because the plateau pressure is the alveolar pressure at the end of the inspiratory pause.

At the end of the inspiratory pause, airway resistance is no longer a limiting factor, so plateau pressure reflects only alveolar compliance.

Static compliance is only the responsibility of the alveoli and chest wall and therefore of the parenchyma, and only a problem of the parenchyma can modify it. Therefore, a problem such as pneumonia, pleural effusion or atelectasis or morbid obesity can modify it. In fact, these problems reduce static compliance.

The dynamic one, as already mentioned, includes alveolar compliance and airway resistance. Here, the problems could be asthma, bronchospasm, air obstruction, parenchymal pathologies and chest wall are also included, in this case compliance will naturally drop.

Also remember this concept:

PIP it's always greater than your Pplat! PIP is always greater than Pplat!

This means that:

Dynamic compliance is always higher of static compliance.

## ATC Automatic Tube Compensation

In Italian, ATC stands for automatic tube compensation. This is a setting to be used in different ventilation modes where the patient is able to breathe spontaneously.

It is also called **AAC** (Artificial Airway Compensation) and intervenes only in spontaneous breathing in any mode. Helps the patient overcome the resistance of the artificial airway, tracheostomy tube or cannula.

The latter, in fact, offer resistance to the passage of air, a resistance that a patient may not be able to "overcome" without help.

Endotracheal tubes offer greater resistance than tracheostomy tubes for the same diameter; for this we must enter into the ventilator which interface the patient is ventilated with and its diameter. Therefore, a tube 7 and a cannula 7 will always have a different compensation, always greater for the tube.

You need to be careful about pressure support in these patients because it will add to the ATC; the ideal would be to use one or the other setting like this:

### **PS present ATC off, ATC present PS off.**

Some ventilators do not allow you to use them at the same time, others do.

What happens, if you use them together, is that the support pressure will also cause the PIP to go up and as a result the ATC will be higher. Not only that, you will have to deal with PEEP+PS+ATC and you could get pressures that are really too high.

This will not help the patient who needs to start a weaning procedure.

The airway compensation system will provide pressure support, just as it does when we insert pressure support. It does this based on the parameters we have entered into the ventilator.

This support is calculated by the machine like this:

### **PIP minus PEEP or CPAP**

So, if we have a PIP of 10 and a PEEP or CPAP of 6, the machine will give an ATC of 4 CmH<sub>2</sub>O. It may be interesting and necessary to know this value if you want to have a complete picture of how the patient is ventilating.

The difference between support pressure and ATC is very simple: when you set a support pressure you know that it will remain unchanged over time.

When you set an ATC, it will adapt to the type of interface, but beyond that, it will adapt to variations in spontaneous breathing. In fact, a spontaneous breath has the characteristic of having different respiratory acts, with different PIPs and therefore with different ATCs.

This does not occur with pressure support.

We can say that ATC is more ergonomic for the patient.

Remember that both PS and ATC intervene only in spontaneous breaths.

## Protective ventilation

But what do we mean when we talk about protective ventilation?

Let's take a quick example and take into consideration a patient whose lung is considered "**more delicate**" than others; this is the case of a patient with ARDS.

The lung, in this case, will have low compliance and will be rigid. Furthermore, as I explained before, its "**ventilatable amount**" is reduced. In these cases, introducing mechanical ventilation with normal values will quickly lead to very high pressures, with the possibility of **barotrauma**. Practically we must reduce at all costs damage caused by mechanical ventilation, called **VILI**.

What does VILI mean?

VILI is the acronym for Ventilator Induced Lung Injury, in Italian Lung Injury Induced by Mechanical Ventilation. A patient with ARDS or severe pneumonia is particularly predisposed to VILI.

It can be caused by:

Pulmonary hyperdistention, for example from high tidal volume, high plateau pressure with damage to the alveoli and mechanical stress

Barotrauma, with rupture of the alveoli and formation of pneumothorax, pneumomediastinum, and subcutaneous emphysema.

The continuous collapse and opening of the alveoli can generate or worsen a picture of inflammation.

## Stress and strain

In mechanical ventilation, stress and strain refer respectively to the force applied to the lungs and the deformation experienced by the lungs during artificial respiration.

**Stress** is defined as the force applied to a surface per unit area. In mechanical ventilation, stress is represented by transpulmonary pressure, i.e. the pressure difference between the inside of the lungs and the external environment. (Alveolar pressure – pleural pressure)

**Strain** is defined as the deformation of a material from its original shape.

In mechanical ventilation, strain is represented by the tidal volume over the FRC, i.e. the amount of air that is administered to the lungs with each respiratory cycle.

Stress and strain are directly proportional: an increase in stress leads to an increase in strain. This means that if the transpulmonary pressure increases, the lungs will deform more.

Excessive stress and strain can lead to lung damage, known as ventilator-induced lung injury (VILI). VILI is a serious complication of mechanical ventilation that can lead to respiratory failure, shock and death.

To reduce the risk of VILI, it is important to use reduced levels of stress and strain. This can be done by adjusting transpulmonary pressure and tidal volume.

## What is meant by barotrauma and volutrauma?

**Volutrauma** is caused by pulmonary hyperdistention caused by excessively high tidal volumes; this leads to rupture of the alveoli. If the  $V_t$  is too high, the pulmonary alveoli may be overdistended, causing their walls to rupture. This can lead to a leak of air from the lungs, which can cause pneumothorax, subcutaneous emphysema, or pneumomediastinum.

**Barotrauma** is caused by ventilation pressures that are too high, even in the presence of low volumes. It also leads to rupture of the alveoli with signs of pneumothorax, pneumomediastinum or subcutaneous emphysema.

**Atelectrauma:** it is damage caused by the cyclical closing and reopening of the alveoli during mechanical ventilation. In the long run it causes inflammation with a picture similar to ARDS.

## Mechanical Power

Refers to the total energy transferred to the respiratory system by the ventilator per unit of time as J/min. It quantifies the work done by the ventilator to move air into and out of the lungs and is a critical parameter for assessing the potential for ventilator-induced lung injury (VILI).

Mechanical power integrates several key aspects of ventilation, including:

Tidal Volume ( $V_t$ ): The volume of air delivered to the lungs with each breath.

Respiratory Rate (RR): The number of breaths delivered per minute.

Driving Pressure ( $\Delta P$ ): The difference between the plateau pressure and the positive end-expiratory pressure (PEEP).

Flow Rate: The speed at which air is delivered to the lungs.

Airway Resistance ( $R_{aw}$ ) and Lung Compliance (C): Characteristics of the patient's respiratory system.

Mechanical power helps in understanding the impact of ventilatory settings on the lungs and in making adjustments to minimize the risk of VILI. It provides a comprehensive measure by incorporating both the ventilator settings and the mechanical properties of the lungs.

The formulas for calculating mechanical power (P) can be complex, but simplified versions are available for each of the volume and the pressure controlled modes.

### [Calculators](#)

## What is Ventilator Associated Pneumonia (VAP) ?

VAP is an infection of the lung parenchyma that occurs in patients who have been mechanically ventilated for at least 48 hours.

VAP is caused by a variety of pathogens, including Gram-negative bacteria, Gram-positive bacteria, and fungi.

Symptoms of VAP are similar to those of other forms of pneumonia, including fever, cough, difficulty breathing, and chest pain.

The treatment of VAP involves the administration of antibiotics specific to the pathogen responsible for the infection.

VAPs cause prolonged length of hospital stay, increase healthcare costs, and increased mortality. One of the factors that causes VAP is precisely the presence of a tracheal device positioned with an invasive intervention that acts as an intermediary between the mouth and lungs. This pathway can facilitate the entry of pathogens into the lungs, causing inflammation and subsequent infection. The often poor conditions of patients who undergo mechanical ventilation favor the development of VAP. Maximum attention, in these patients, must be paid to oral hygiene and airway management.

Duration of mechanical ventilation is a risk factor for VAP. The longer mechanical ventilation lasts, the more likely VAP is to appear. Reducing mechanical ventilation time is an effective way to reduce the risk of VAP. VAP is a serious complication of mechanical ventilation that can have significant consequences for the patient's health.

Prevention is key to reducing the risk of VAP.

### The I:E ratio

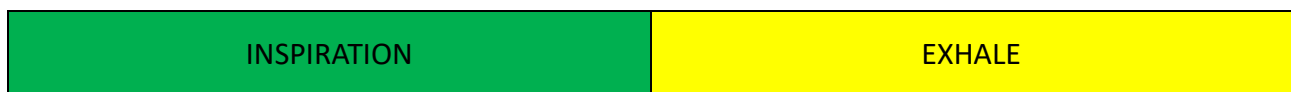
The I:E ratio is the ratio of the duration of inhalation to exhalation.

The I:E ratio cannot be set in all ventilation modes, but only in those with time triggers. If set in other modes, any spontaneous event would make it unusable.

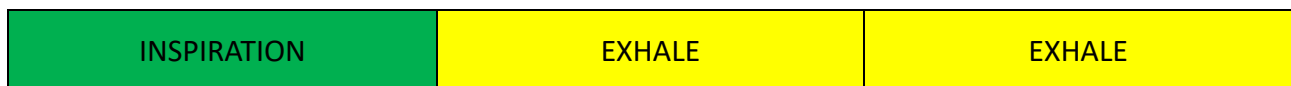
This is under the segment that represents the complete respiratory act or TCT (Total Cycle Time).



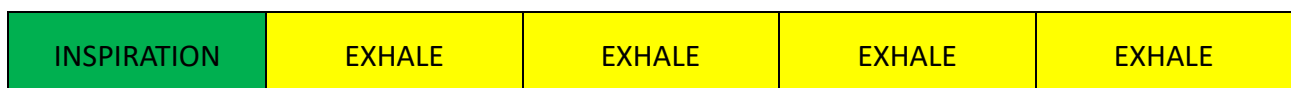
We know that the complete respiratory cycle is given by two main events that follow one another, and we can represent them in this way:



This is a visual way to show you the I:E ratio of 1:2, here it is:



We represent a 1:3, 1:4 and inverse 2:1 ratio like this:





With this visual representation you can more easily imagine the time that is dedicated to the two different phases of the respiratory act.

The complete respiratory act is made up of the inhalation and exhalation times; the I:E ratio expresses the relationship between these two moments in relation to time.

Thus, a "normal" patient has an I:E ratio of 1:2. This means that exhalation, which is a passive phenomenon, is left twice as long as inhalation.

A patient with severe hypoxia or ARDS will have a ratio of 1:1, 1.5:1 or greater.

An I:E ratio with values such as 1:4-1:5 is used in those cases where we can have air trapping; patients with COPD, chronic bronchitis, asthma or emphysema require a very long expiratory time to allow the evacuation of air rich in CO<sub>2</sub>.

Now, let's think for a moment about what happens if we modify some parameters of the ventilator.

Let's assume we have set a frequency of 20 acts per minute; the single act will last 3 seconds and with a ratio of 1:2, we have 1 second of time for inhalation and 2 seconds for exhalation. At this point, if we modify the inspiratory flow, we also modify the I:E ratio. If we increase the flow, the time needed to reach the set V<sub>t</sub> will be shorter. Consequently, also the inspiratory time (T<sub>i</sub>). The opposite is also true, and if we lower the flow the inspiratory time (T<sub>i</sub>) lengthens. All this at the expense of expiratory time (T<sub>e</sub>), which in both cases will occupy the remaining time available.

Returning to the previous example with 20 acts per minute and therefore 3 seconds per act: if I increase the flow, I will have a T<sub>i</sub> of 0.5 seconds and the T<sub>e</sub>, to satisfy those 3 seconds, will become longer, exactly 2.5 seconds. These are numbers put there as an example, but the I:E ratio will become 1:5 !

By reducing the flow, I always modify the T<sub>i</sub> which becomes longer, for example 1.5 seconds, "stealing" time from the T<sub>e</sub> or time dedicated to exhalation; this will only have 1.5 seconds available; thus the I:E ratio became 1:1.

We can apply the same thing to a variation of the set V<sub>t</sub>. Remember that in controlled volume, flow and V<sub>t</sub> determine inspiratory time. The frequency regulates both T<sub>i</sub> and T<sub>e</sub> and the I:E ratio affects the duration of the T<sub>e</sub>.

When we have a greater T<sub>i</sub> (inspiratory time), we give more time to gas exchange and, theoretically, improve the patient's oxygenation.

## I forgot.....these damn alarms!

In my introduction, I talked about nursing students, who are so hungry for knowledge that they immediately identify in mechanical ventilation that something that: **you absolutely must understand!** So, they ask you:

*"..when you have a minute, can you explain the ventilator to me???" !!!*

Your jaw drops to the floor, but you try to explain what that machine is. Incredible but true, the first time the ventilator alarm sounds, whatever it is, the student has already identified the button that silences it; what follows is a continuous "game" between the ventilator that sounds and the student who "silences" it, without worrying at all about the visual warnings and the writings that say what the problem is. In this condition, the function of the alarm itself is lost and places that patient in a dangerous situation, as dangerous as a ventilator that does not have any type of alarm.



But what are alarms for? The ventilator, although it has become increasingly technological and precise, does not have the "eyes" that we users can have. So, when some parameter exceeds the range it should respect, he does the only thing he is capable of doing: "ask for help for the patient!" You understood correctly that little music that plays every time the ventilator recognizes a problem has the task of drawing our attention to the patient and how he is breathing. The problem could concern the patient, perhaps with too many secretions, the position of the tube, a blocked filter, the circuit not connected, leaks, the ventilator having problems, the volumes not reached or exceeded, the pressures not reached or exceeded and everything for which the ventilator is able to warn us with an alarm.

You understand well that a machine, no matter how advanced, is not able to understand what is happening, much less remedy it; it just needs to get our attention.

In this, the task of those who monitor the patient is a task of primary importance and must be performed continuously and scrupulously.

Every now and then, when the ventilator "sings", you hear a phrase like "... yes, I know it's the high frequency"!!!

But this is not true, or rather, it could be the high frequency that perhaps sounds every minute, but it could be something else entirely. Each alarm should be evaluated on a case-by-case basis, no alarm should be left to itself.

Of course, you don't have to sprint like a sprinter as soon as the ventilator starts blowing; often it is a momentary alarm override and after two beeps it stops on its own.

But if it continues you must not avoid intervening.

Managing alarms in this way is very risky for the patient and counterproductive for his ventilatory therapy.

### **But how can I do it if that "parapa pa pa!!!" rings every two seconds?**

You have to understand what the problem is. If the alarms are set well, you have a problem to solve.

If you can't solve it yourself, ask for help. Often the problem could be an alarm that is too tight.

Thus, the patient who has 20 breaths, but the alarm at 21 may cause the ventilator to sound very often. Have the upper respiratory rate limit changed.

Another big mistake is letting an alarm ring for hours without checking it.

Just check your heart rate and saturation and you're calm!

It's alive and breathing, that's all you need! If you really are a genius, congratulations!

Hell, never make that mistake, I always tell students:

**An alarm that sounds continuously is equivalent to having no alarms!**

**A poorly set alarm is like having no alarms!**

In summary, ventilators are "stupid" and only recognize that something is not right.

This is why they use alarms, so that you can fix that "something".

### **How can we divide mechanical ventilation?**

There are different systems for dividing mechanical ventilation based on:

Positive, negative or HFV pressure

Who does what and therefore controlled, assisted, supported or spontaneous  
Operator freedom, open loop or close loop  
Volume controlled, pressure controlled or mixed  
Neurally controlled, adaptive etc  
And based on the color of the ventilator, as well as the brand of course!

## Positive pressure, negative pressure and high frequency ventilation

I'll explain very quickly what it is: it is a way in which ventilation is implemented.

**Positive pressure:** through the generation of a flow of air that is pushed inside the respiratory system, through positive pressure, in fact.

**Negative pressure:** through the generation of a depression in the pulmonary system such that it draws air into it (iron lung).

**HFV:** implementing ventilation by generating a high frequency flow.

As already mentioned, in this text I limit myself to positive pressure ventilation only.

## Types of mechanical ventilation, from controlled to spontaneous

How the ventilator interacts with the patient can be broken down in several ways.

This division describes how breath is delivered. This is a classic subdivision and is based on the role that the ventilator has in the "ventilation" process. Thus, if the ventilator takes care of "doing" all the work, it is called controlled ventilation. By this term, we mean that all phases of ventilation are controlled by the ventilator.

In this context we have four different ways of ventilator-patient interaction:

1. **Controlled ventilation**
2. **Assisted ventilation**
3. **Ventilation supported**
4. **Spontaneous ventilation**

This division is to be considered indicative because it is susceptible to overlaps between one type of ventilation and another. It is easy to have mixed ventilation types that allow supported ventilation to become spontaneous or controlled ventilation to become assisted. As you can see, they have been listed based on the work carried out by the ventilator, ranging from the complete work carried out by the ventilator in controlled mode, up to the work completely carried out by the patient in spontaneous mode.

The reasoning is also invertible, placing the work done by the patient at the center of the discussion, but this does not change what happens.

The definition put in this way is not complete and, to be clearer, I must insert in which phases of ventilation the ventilator and the patient intervene.

We consider three main phases of ventilation: **Trigger, limit and cycling.**

**Triggers:** it is the signal that initiates inhalation

**Limit:** is the pressure or volume target that must be achieved

**Cycling:** it is the transition from inhalation to exhalation

We analyzed them well a few pages earlier, you remember that right?

The type of ventilation is defined based on who controls what. In the diagram below I tried to be more explicit:

Types of ventilation	Triggered	Limited	Cycled
Checked	ventilator	ventilator	ventilator
Assisted	Patient	ventilator	ventilator
Supported	Patient	ventilator	Patient
Spontaneous	Patient	Patient	Patient

I reiterate the importance of not taking this division as clear-cut and absolute. Furthermore, you can find in some texts the union of assisted ventilation with supported ventilation. In recent months, I have had the opportunity to come across many works by other authors, where some ventilatory modes were improperly included in one of these classifications. The level of this work of mine must only allow us to understand how the thing works: who does what and how the WOB is divided between machine and patient. In fact, other factors come into play; thus, to give you an example, the compensation of the resistance of the tracheal tube, present in many ventilators, may or may not be considered ventilatory support. This means that a patient who ventilates spontaneously without any help, other than an adequate FiO<sub>2</sub>, making use of the compensation of the resistance of the endotracheal tube, is ventilating in a supported manner. But, if we follow the scheme above, we need to put this patient on spontaneous ventilation. The truth, as often happens, is somewhere in the middle!

## Open Loop and Close Loop ventilation

**Open loop ventilation** is a ventilation mode in which the ventilator does not monitor the patient's condition and does not regulate the breathing process based on these conditions. The ventilator simply delivers a preset volume or pressure, regardless of the patient's needs. In this type of ventilation, the clinician decides what to do and, if there are variations, he must intervene with appropriate modifications.

**Close loop ventilation** is a ventilation mode in which the ventilator monitors the patient's condition and regulates the breathing process based on these conditions. The ventilator uses sensors to measure pressure, volume, respiratory rate and other respiratory parameters. Using this data, the ventilator calculates the level of ventilation needed to meet the patient's needs. If there are variations, the ventilator can make adjustments to adapt ventilation to the changed conditions.

### Advantages and disadvantages

Open loop ventilation modes are generally less complex and more economical than close loop ventilation modes. However, close loop ventilation modes are more precise and can provide better ventilation for patients.

Here are some examples of open loop ventilation modes:

Volume Controlled Ventilation (VCV)

Pressure Controlled Ventilation (PCV)

Continuous positive pressure ventilation (CPAP)

Here are some examples of close loop ventilation modes:

Adaptive Pressure Controlled Ventilation (ACPV)

Proportional assist ventilation (PAV)

Adaptive Controlled Positive Pressure Ventilation (APAV)

Adaptive Support Ventilation (ASV)

## Types of mechanical ventilation: pressure-controlled and volume-controlled

Also called pressometric and volumetric or flowmetric.

Another important division of mechanical ventilation involves pressure-controlled ventilation and volume-controlled ventilation, perhaps the most significant distinction.

I will try to explain the difference between the two, highlighting the advantages and disadvantages. This topic may seem complicated to you, but don't worry; I collected all the available data and simplified it as much as possible, making the concept accessible even to non-experts. Even my cat now knows the difference!

First of all, the simple concept and then I'll explain how it works:

In the **pressure-controlled ventilation**, the operator has set a pressure to reach; to reach this pressure, **the ventilator will vary the volume** of air delivered based on the patient's effort, compliance values and resistance.

In the **volume-controlled ventilation**, the operator has set a volume to be reached; to reach this volume, **the ventilator will vary the pressure** it uses to deliver that volume, based on the patient's effort, compliance and resistance.

The term "controlled" is a bit counterintuitive and can lead some to get confused, but you can think of the ventilator that "controls" one parameter by varying the other.

**Control volume by varying pressure or control pressure by varying volume**

Volume-controlled ventilation is called VCV, pressure-controlled ventilation PCV; the abbreviation VC or PC applies to both.

When you find yourself in front of these acronyms you will immediately understand what we are talking about:

<b>Volume-controlled ventilation</b>	<b>VCV</b>	<b>VC</b>
<b>Pressure-controlled ventilation</b>	<b>PCV</b>	<b>PC</b>

In a generic way we can say that:

There **VCV** promotes the control of **ventilation**.

There **PCV** promotes the control of **oxygenation**.

VCV ensures a stable and precise ventilation volume, but a higher peak pressure. This can lead to barotrauma and uneven gas distribution. On the other hand, PCV improves arterial oxygenation and has a rapidly decelerating flow pattern but is also associated with lung injury due to tensile forces on the lung and alveoli.

To memorize you will need: VC ventilation will have the square wave in the flow, a PC ventilation will have the square wave in the pressure!

It's not hard to remember but I added a beautiful table!

VC	"Square" flow curve
PC	"Square" pressure curve

However, things are not always as they seem! If we are using a VCV but we have set the autoflow PRVC, APV, VC+ the ventilator, the square curve will be that of the pressure and not that of the flow... You will find out later why, look for "autoflow" at the bottom of the little job. However, we are well beyond the notions that are of interest for a basic "course".

### Differences between VC and PC

In volume-controlled ventilation mode, you set a  $V_t$  and a flow or  $T_i$ ; these are linked to each other and if you have a slow  $T_i$ , the flow also drops and vice versa.

In this mode the pressure will vary. Remember that VCV is the best way to have control over the ABG, because you decide the minute volume with  $V_t$  and RR.

In pressure-controlled ventilation, you set the pressure and a  $T_i$ , i.e. how long that pressure must remain; here it is the volume that varies. Naturally, if the volume varies, the minute volume will also vary and therefore the ABG values will also be variable, therefore less controllable.

Here you can better control the barotrauma, but not the ABG.

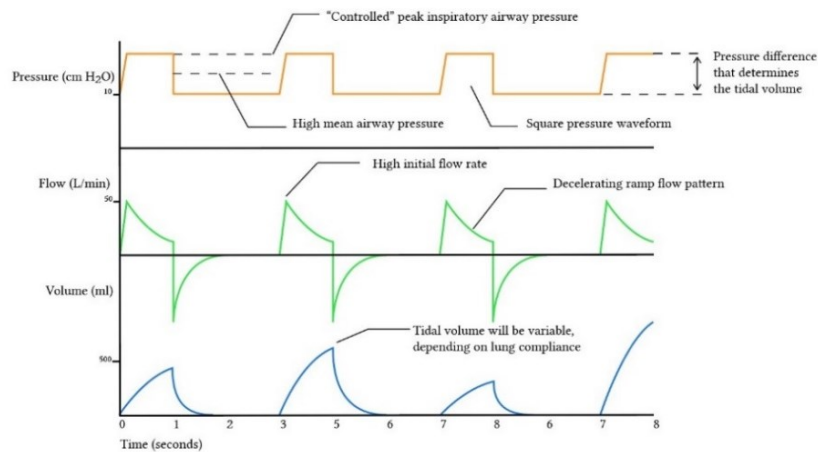
#### Pressure-controlled ventilation

In pressure-controlled ventilation, the ventilator must reach a certain pressure that is set. In this mode, pressure is the target to be achieved and, depending on the condition of the airways, the volume administered will be modified to achieve it. By airway conditions we mean compliance and resistance. It is easy to understand that changing one of these can make it easier or more complicated to reach the set pressure goal.

The person who sets this pressure is the doctor, who decides based on multiple pieces of information which method to use and, in this case, what pressure can be reached. In this type of ventilation, it is very important to set adequate alarm limits. Once the desired pressure has been reached, the ventilator will consider the inspiration as finished and will cycle.

In pressure-controlled ventilation, the flow will always be decelerated, look for its shape in the curves.

In PC mode the set pressure is maintained throughout the inspiratory phase, resulting in a "square" waveform. This means that the pressure always remains at the maximum set value throughout inspiration, causing the average pressure value to increase.



At the beginning of inspiration, the ventilator provides a high flow to quickly reach the pressure limit. Once this limit is reached, the flow can decrease, assuming a downward trend. When the flow has reached zero, and therefore we have no flow, the pressure value stabilizes exactly at the plateau pressure. Remember that plateau pressure is that moment, at the end of inspiration, in which we have no air flow.

#### Advantages of PCV

In a patient with respiratory failure where hypoxia and low compliance are critical aspects, PCV presents significant advantages such as:

##### **Increased mean airway pressure:**

The average pressure, in fact, has an important role in improving oxygenation through the use of positive pressure. Remember that an important contribution to average blood pressure is PEEP. Furthermore, it also improves the FRC.

##### **Increased duration of alveolar recruitment:**

The square shape of the wave allows the alveoli to open earlier and stay open longer, improving gas exchange.

##### **Pressure-limited ventilation can protect against barotrauma:**

The stable pressure value prevents alveolar injuries.

In other words, because the pressure level is controlled, there should never be a time when the patient “experiences” extremely high pressures.

##### **Work of breathing and patient comfort “can” be better:**

The high initial pressure prevents patient-ventilator asynchronies caused by “lack of flow”.

#### Disadvantages of PCV

##### **The tidal volume depends on respiratory compliance, resistance and patient effort:**

Tidal volume may vary during mechanical ventilation. In this mode it may be difficult to achieve tight control of PaCO<sub>2</sub>.

**Uncontrolled volume can cause volutrauma:** that is, if lung compliance suddenly improves, the ventilator could deliver volumes that stretch the most compliant lung units beyond their elastic limit.

**High early inspiratory flow can exceed the pressure limit** if airway resistance is high. The initial high flow rate will create high pressure due to airway resistance, which may be high enough to exceed the pressure alarm limit. If airway resistance is a problem, a gentler inspiratory flow may be beneficial.

not having a "volume" target, if the pressure remains low, it is easy to deliver very large volumes. It is to these that we owe the overdistension that can occur before the target "pressure" is reached and lead to volutrauma.

## Volume-controlled ventilation

In this mode, the goal to be achieved will be determined by the current volume. The doctor, based on the patient's condition, decides which tidal volume to set. Once this volume is reached, the ventilator switches to the exhalation phase.

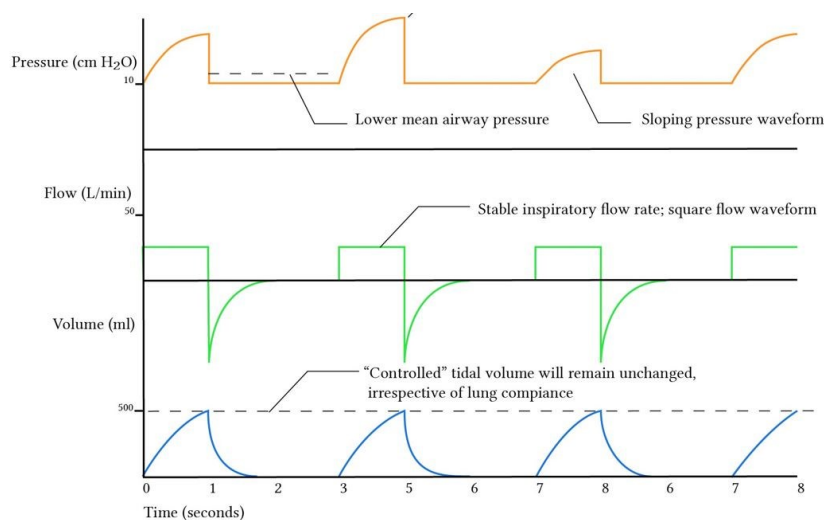
Even in this mode, taking into account compliance and resistance, the ventilator will vary the pressure to reach its goal, i.e. the set volume.

The volume-controlled mode can also be called **flow-controlled**, a less used but not incorrect term.

With the volume-controlled mode it is possible to have accurate control over the patient's blood gas analysis. This is because, by controlling the tidal volume, I control the very important parameter, that is, the minute volume; the minute volume directly influences the pH and  $PCO_2$  value and allows you to quickly correct disorders relating to the patient's ABG.

It is important to note that to achieve the desired volume, the ventilator may have to deliver significantly high pressures. This could increase the risk of ventilation trauma, making the volume-controlled mode potentially riskier in terms of lung damage from excessive pressure.

In volume-controlled ventilation, a tidal volume is set as a goal. Since volume and flow are inextricably linked, volume-controlled modes are generally constant-flow modes, meaning the ventilator delivers a flow that is constant and stops this flow when the desired volume is reached. Because the pressure is not controlled or regulated in any way, the pressure waveform takes on a tilted parabolic shape as the lungs relax during the inspiratory phase.



The pressure waveform is highly variable during volume-controlled ventilation, taking on a tilted parabolic shape as the lungs distend. The waveform is influenced by lung compliance and airway resistance.

### Advantages of VCV

**Guaranteed tidal volumes produce a more stable minute volume:**

The stability of the minute volume makes this ventilation mode more appropriate in situations where careful control of PaCO<sub>2</sub> is important. A stable minute volume helps maintain a balance between ventilation and perfusion, reducing the risk of hypercapnia and hypoxia.

**The minute volume remains stable even if lung characteristics vary:**

If airway resistance varies significantly, for example from status asthmaticus, this mode has the advantage of maintaining a reliable minute volume. This is because the ventilator delivers a constant flow until the desired tidal volume is reached.

**The initial pressure is lower than the controlled pressure:**

This is an advantage when airway resistance is high. “Blowing” more slowly into the narrow bronchi does not produce an early pressure peak related to high resistance and potentially prevents premature termination of the inspiratory phase.

Disadvantages of VCV

**The mean airway pressure is lower:**

With volume-controlled ventilation, the mean pressure value is reduced due to the pressure waveform. This may theoretically be a disadvantage in patients with severe hypoxia; in such situations you may want to use a pressure-controlled mode instead.

**Recruitment may be poorer in alveoli with poor compliance:**

Units with low compliance may remain unrecruited until very late in the inspiratory phase, when pressure approaches its maximum value. This may allow little time for gas exchange. In the presence of atelectasis, VCV may not be the appropriate modality.

**In the presence of leaks, the average pressure may be unstable:**

The constant flow used during VCV may not be sufficient to compensate for a leak. If the leak rate is equal to the inspiratory rate, there will be no delivered volume!

In the presence of leaks, the average pressure may be unstable, with peaks and dips.

**Insufficient flow can cause patient-ventilator asynchronies:**

If there is an increase in inspiratory demand during the course of a breath, the ventilator may not meet the patient's need to increase flow. This would lead to asynchrony between patient and ventilator, with the patient experiencing a feeling of chest tightness or difficulty breathing.

## Mixed pressure-volume ventilations

Found in the ventilation modes, a part dedicated to PRVC.

There are also mixed modes that combine the advantages of the two previous systems and are called adaptive ventilation control modes such as PRVC: this mode ensures a prescribed volume, maintaining a square pressure waveform and therefore a high average airway pressure .

However, in PRVC, mean airway pressure can vary significantly, depending on patient compliance and effort.

Adaptive modalities, such as PRVC, are a fusion of the two strategies that combine the advantages of each without amplifying their disadvantages. For a PRVC mode, the user sets a target tidal volume, as in a volume control mode.

For example, in the Maquet<sup>TM</sup> SERVO-i, a dynamic lung compliance assessment is performed over the course of three initial calibration breaths, which are **volume control** breaths. Using this lung compliance data, the ventilator determines what pressure level is appropriate and necessary to achieve the prescribed tidal volume.



This mode has some disadvantages: variable patient effort can lead to a highly variable tidal volume, since the pressure level depends on the respiratory characteristics compared to previous breaths. However, it retains several advantages of PCV and VCV and none of their disadvantages, in fact:

The square pressure waveform promotes early and sustained recruitment of lung units.

The mean airway pressure is high as with PCV.

Pressure is minimized for any given prescribed tidal volume.

A minute volume is guaranteed, while maintaining some control over the PaCO<sub>2</sub>.

The deceleration of the flow waveform appears to be the primary determinant here. By using this flow model, volume-controlled modes lose many of their disadvantages.

## Bonus part, “Mr.” Robert Chatburn

Almost all the information in this bonus part, text, data and images, is taken from the works of Robert L. Chatburn. Of which he holds copyright and intellectual property.

“Mr.” Robert L. Chatburn, MHHS, RRT-NPS, FAARC  
Research Manager-Respiratory Institute  
Cleveland Clinic  
Professor - Department of Medicine  
Lerner College of Medicine of Case Western Reserve University

### Is it possible to put the ventilation modes in order?

Searching far and wide to understand something and make the discussion easier, I came across some data not to be underestimated:

Currently, more than 40 companies exist and produce mechanical ventilators in the world, each of them has different types of ventilator in their catalogue; this only if we want to photograph the current situation. If we consider that positive pressure ventilation has been used since around 1952, this makes us understand how much progress the technology has made.

With these advances, the amount of ventilation modalities available has also grown, some lost over time, some arriving today with the necessary improvements.

Today we have at our disposal, if we wanted to do a search, more than 500 different names for the ventilatory modes. This does not mean that there are 500 ventilation modes, but that they are called with different names by the different production brands and, over the years, in the same brand some modes have had different names.

You understand that it is impossible to include them all, let alone describe them.

Someone comes to our aid who has done the work for us!

So, while I had reached about a hundred acronyms, I came across a video that gave me some oxygen, to stay on topic! ...and it got me back on track.

I was watching a video from the Society of Mechanical Ventilation describing the grading work done by this “gentleman” whose name I couldn't quite make out; this is because, for me, interpreting a surname in English is difficult, also because I'm becoming a bit deaf!

So, I'm forced to contact them and, these very kind people, they don't even let me press send and they reply giving me their name, surname and their job!

### **But look, I think, overseas they realize that I exist...!**

I thus discover that this "gentleman" is called Robert L. Chatburn, he is a researcher at the Cleveland Clinic, you can find his name in more than 300 publications, 9 books; he is a university professor and an absolute reference, defined as an authority, in the field of mechanical ventilation. He has dedicated 40 years of his career to this topic and has developed a classification system for mechanical ventilation to which he has dedicated more than 25 years.

I found myself faced with a maze of very similar ventilation modes.

A chaos so intricate that it will test our patience and that curiosity alone will not be enough to unravel. You already know that there are countless ventilation modes with different acronyms, many of them are used by specific brands but are similar to others. As much as I have searched far

and wide, there is no "easy" guide that divides and describes them all. There are more or less short lists with standard modes and some mention of particular modes.  
Here I will try to put all this mess in order!

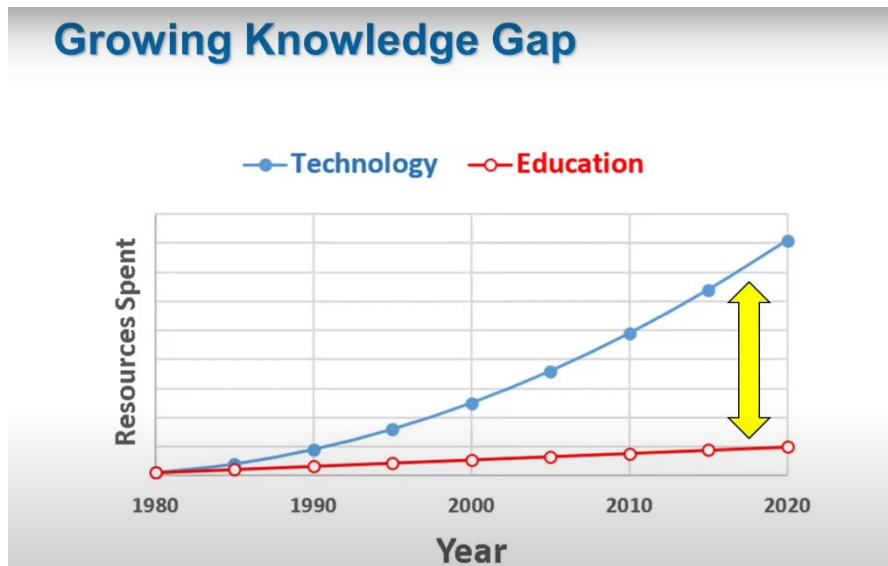
...Spoiler, don't waste time searching online for a list that associates the different ventilatory modes, it simply doesn't exist. Read the following pages, they will be enlightening!...

To make you understand what I'm talking about, I've put some together, about 220!

**All, but really all the ventilation modes?.....yes, yes, believe it!!!!**

AC FC	nCPAP	SIMV Plus	SIMV FC+PL
AC MV PC	nIPPV	SIMV Pressure	SIMV MV PC+PS
AC MV PRVC	NIV	SIMV PT	SIMV MV PRVC+PS
ACPC	NIV AC MV PC	SIMV VC+APF	SIMV MV VC+PS
AC PC+TC	NIV APRV	SIMV Volume	SIMVPC
AC PC+Ti termination	NIV CPAP+PS	SIMV VT	SIMV PC+VL
AC PC+VL	NIV NAVA	SIMV+TTV	SHIP
AC Pressure Target	NIV PC	S-IMV+VG	nCPAP
AC VC	NIV PPV	S-IPPV	Infant Nasal IMV+CT
<b>AC VC+APF</b>	<b>NIV PPV S/T</b>	<b>S-IPPV+VG</b>	<b>MMV+PL</b>
AC VC+PL	NIV PPV Spontaneous	SmartCare	MMV+PL+TC
AC VCPlus	NIV S/T	SPONT	NASAL CPAP
AC VT	NIV Spont CPAP	Spontaneous CPAP	SBTI+TC
APRV	NIV Spont PS	Spontaneous PS	S-IMV
APRV SIMV+TC	NPPV AC	Spont VS	SIMV FC
APRV/Biphasic	NPPV CPAP+PS	Spontaneous	IM+VG
APV CMV	NPPV S/T	Spontaneous+AVAPS	IMV+CPAP
APV CMV+TC	NPPV SIMV	Sync CMVplus	Infant Nasal IMV
APV imv	NPPV Spontaneous	Sync CVM AC	IRV
<b>APV imv+TC</b>	<b>P AC+Flow Term</b>	<b>T</b>	<b>iVaps</b>
APV SIMV	P SIMV	TCPL AC+FC	IVP
Assist/Control Pressure	PAC	TCPL AC+VG	MMV
Assist/Control-Volume	Patient Trig Vent	TCPL SIMV	MMV+AF
ASV	Patient Trig Vent+TTV	TCPL SIMV+VG	MMV+AutoFlow+TC
ASV car	PAVf	Timed	S/T
ASV+TC	PC AC	Timed+AVAPS	S/T+TC
Auto BiLevel	PC AC+Assured Volume	V AC	S/T+TV
Auto CPAP	PC AC+FC	V SIMV	S/T+TV+TC
Auto SV	PC IMV	Vauto	SBT
<b>Automode (PC to PS)</b>	<b>PC IMV Plus</b>	<b>VC</b>	<b>DV (PSV to PA/C)+TC</b>
Automode (VC Flow Adapt to VS)	PC plus Assisted	VC AC	DV (VC SIMV to VA/C)
Automode /PRVC to Vs)	PC SIMV+IntelliSync	VC AC +Flow Trak	DV(CPAP to PA/C)+TC
AutoSet	PC SIMVplus	VC AC+AF	HFCWO
AVAPS	PC Ventilation Plus	VC AC+Demand Flow	HFJV
AVAPS SE	PC+AVAPS	VC AC+Vsync	HFO
AVM	PC-CMV	VC AC+Vsync+FC	HFO+CMV
AVM+TC	PC-IMV	VC IMV	IPPV CMV
Bi-Level	PCplus	VC IMV+Flow Trak	IPPV+VG
<b>BiLevel +VG</b>	PC-SIMV	VC SIMV+TC	IPV
BiLevel+PS	<b>PCV</b>	<b>VC+PL</b>	<b>PSV VG</b>
BiPhasic	PCV+TC	VC-AC+FC	BiLevel pulsatile
BiPhasic tr	PCV+TV	VC-AC+Vsync	CPAP button
BiVent	PCV+TV+TC	VC-SIMV	S
BiVent+PS	Proportional PS	VC-SIMV + Vsync	DV (CPAP to PA/C)
CMV+Active PEEP	Proportional PS	VCV	DV (CPAP to PC SIMV)
CMV+FC+PL	PRVC	VCV+TC	DV (CPAP to PC SIMV)+TC
CMV+PEEP	PRVC AC	VDR4	DV (PSV to PA/C)
CMV+PL	PRVC AC+FC	VG PS	INTELLIVENT ASV
<b>CNEP</b>	PRVC SIMV	VG PS+TC	VS+TC
CPAP+PC+VL	<b>PRVC SIMV+FC</b>	<b>Vol.Targeted PC</b>	<b>PS-SIPPV+VG</b>
CPAP+PS	PS	Vol.Targeted PS	Duo PAP
CPAP+PS+TC	PS HFO	Vol.Targeted SIMV	REV
PS+TTV	PS+TC	VS	HVLP
PS+TV	VC SIMV	PSV	AVPC
Ramp to VS)	VC SIMV+AF	PS-SIPPV	Automode (Vc Square or

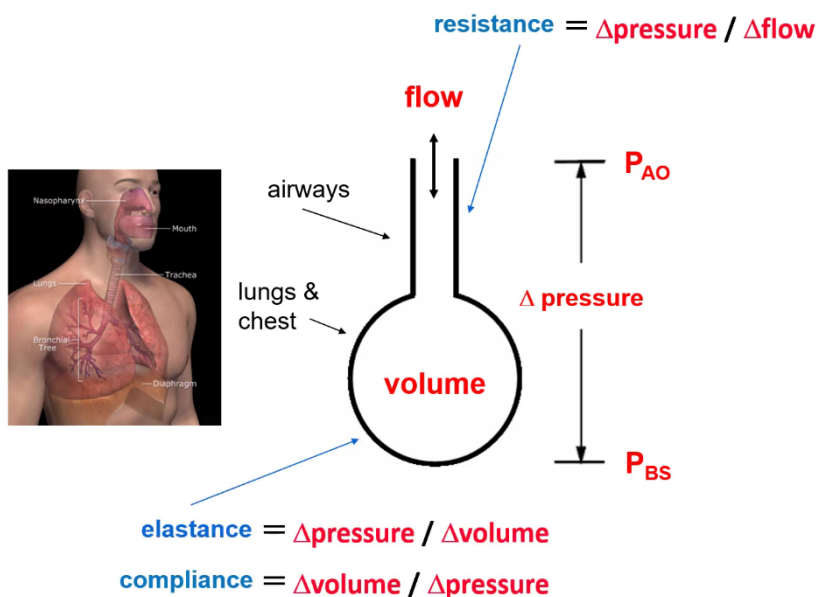
Robert L. Chatburn still dedicates himself to training and research, and also collaborates with brands of mechanical ventilators to improve the technique of mechanical ventilation. Notice the graph below:



From here we understand that the ability to use ventilators appropriately has not gone hand in hand with the evolution of the technique and technological growth of ventilators.

### The equation of motion

Before describing Chatburn's taxonomy in broad terms, it is necessary to describe the equation of motion of the respiratory system. I will do so by borrowing his words regarding this very topic. We can simply schematize the respiratory system with a single air duct representing the airways and a single respiratory unit representing the lungs. In this way, we can talk about the three main factors you need to know to "understand something" about mechanical ventilation. These are pressure, flow and volume.



We then have the resistance, elastance and compliance expressed as:

$$\text{Resistance} = \Delta \text{Pressure} / \Delta \text{Flow}$$

$$\text{Elastance} = \Delta \text{Pressure} / \Delta \text{Volume}$$

$$\text{Compliance} = \Delta \text{Volume} / \Delta \text{Pressure}$$

We know that: a Pressure that causes an action corresponds to a Pressure that generates an equal and opposite reaction. This also happens in the Equation of Motion.

This means that if on the left of the image we have the ventilator which exerts a certain pressure, on the right we have the respiratory system (lungs + airways + rib cage) which will exert an identical pressure. Look at the picture:

**The Equation of Motion**

$$P_{action} = P_{reaction}$$
$$P = P_{elastance} + P_{resistance}$$

elastic load      resistive load

The right side can be broken down into two components, the elastic load and the resistive load.

The elastic load is given by Elastance x Volume (or volume/compliance)

The resistive load is given by Resistance x Flow.

In this equation, volume and flow are variable over time, elastance and resistance are constant.

An image will clarify the concept better:

**The Equation of Motion**

$$P = P_{elastance} + P_{resistance}$$

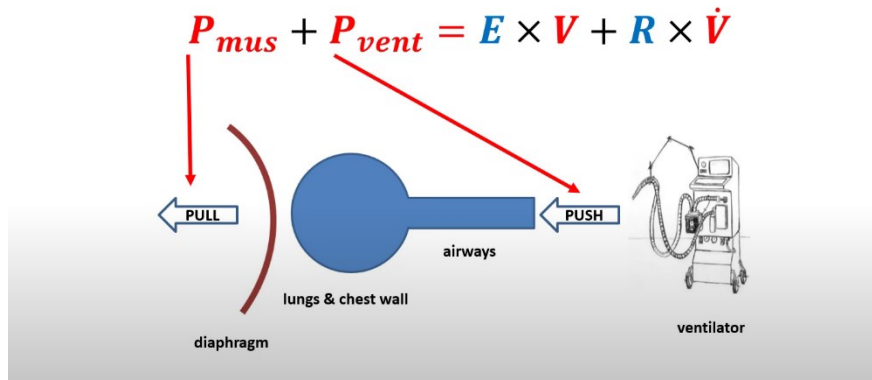
elastic load      resistive load

$$= E \times V + R \times \dot{V}$$

elastance (constant)      resistance (constant)

In a condition in which the patient breathes together with the ventilator to generate a pressure capable of overcoming the elastic and resistive load, the equation becomes like this:

## Equation of Motion



So:  $P_{mus} + P_{vent} = E \times V + R \times \dot{V}$

If we have no patient effort, the formula simplifies to:

$$\text{Equation of Motion}$$

$$P_{vent} = E \times V + R \times \dot{V}$$

We will soon discover that pressure-controlled ventilation controls the left side of the equation, that  $P_{vent}$ , so to speak.

A volume-controlled ventilation will affect the part with volume and flow, and therefore the right part, this:  $E \times V + R \times \dot{V}$

## Chatburn's taxonomy

By taxonomy we mean a classification system that allows identical or similar methods, named by brands in different ways, to be ordered with generic names.

In this system we end up with a TAG that represents the mode used: the TAG represents the control variable, the breathing sequence and the targeting patterns, precisely defining the ventilatory mode.

The **control variable** is pressure or volume (flow)

The **breathing sequence** indicates whether it is spontaneous, controlled or assisted

**Targeting schemes** represent an algorithm that the ventilator uses to achieve the objectives (targets) you set.

**Control  
Variable**

**Breath  
Sequence**

**Targeting  
Scheme**

By objective we mean any value set on the ventilator, such as a  $V_t$  in the VCV or the inspiratory pressure in the PCV.

The trigger represents the triggering of inspiration and can be determined by the patient or by the machine.

Cycling is the end of inspiration and can be triggered by the patient or the machine.

We must then define what is meant by spontaneous and mechanical breathing (mandatory or controlled)

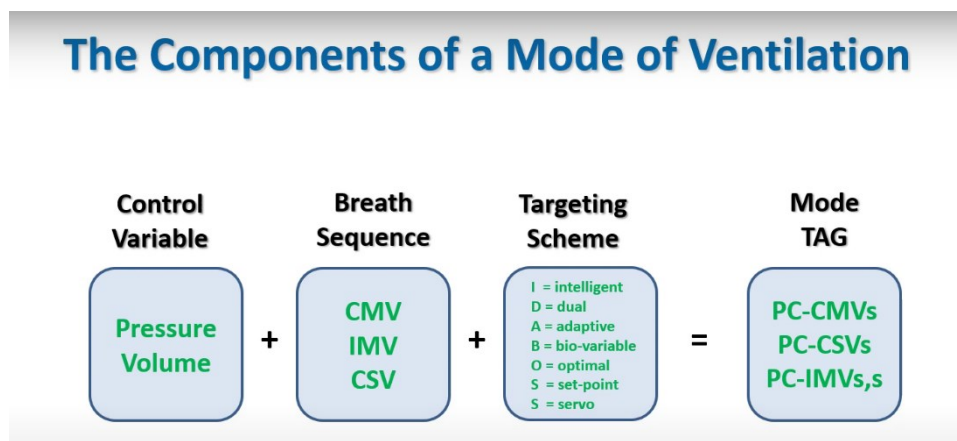
Spontaneous breathing is breathing completely controlled by the patient, initiated (triggered) and ended (cycled) by the patient, regardless of the inspiratory time settings of the machine.

Mechanical breathing (mandatory, controlled or obligatory) is a breath completely controlled by the machine in both triggering and cycling.

Each ventilatory mode according to the taxonomy is composed of three components, which we have seen above:

- Control variable
- Breathing sequence
- Objective (target)

Here is an image that clarifies this whole discussion:



These three elements together form the TAG, i.e. they unequivocally define the generic modality. This method is valid for any present or future mode available.

Let's see the first component. This is the **control variable**, which can be volume or pressure. A ventilator can only use one of these at a time and can only use them together in mixed modes, but not at the same time; it can only do so alternately.

The volume control mode actually controls the flow, but it has now become commonplace to call it volume control.

Any ventilator can only control part of the motion equation.

**Pressure-controlled ventilation** controls the left side like this:

## Pressure Control Modes

$$P_{vent} = E \times V + R \times \dot{V}$$

$P_{vent}$   
 pressure  
 control

In this control mode, the ventilator maintains the set pressure or varies it depending on the patient's inspiratory effort.

In fact, variations in elastance and resistance (blue) generate changes in volume and flow (red). These are modified because the operator has entered inspiratory pressure values as a target which will remain constant and an inspiratory time.

**Volume-controlled ventilation** controls the right side like this:

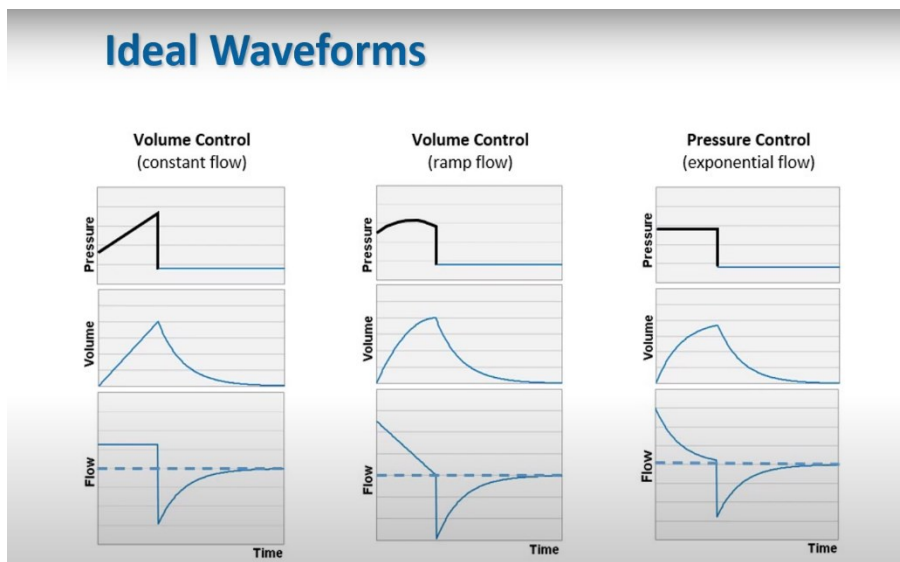
## Volume Control Modes

$$P_{vent} = E \times V + R \times \dot{V}$$

$P_{vent}$   
 volume  
 control

In this ventilation, both the  $V_t$  and the inspiratory flow and its delivery are set by the operator. In this case, elastance and resistance cause changes in pressure.

Here we have the representation of the ideal waveforms for the different control modes:





You can see how in VC mode the flow, if constant, takes on a square shape.

In PC mode the pressure curve has a square shape. If you remember we have already seen these concepts.

A square shape indicates that flow or pressure is achieved quickly and that the same is maintained for the entire duration of inspiration.

Let's now analyze the **breathing sequence**. There are three different types of breathing:

CMV  
IMV  
CSV

Fully machine-controlled or mandatory breaths are called CMV or Controlled Mandatory Ventilation.

If there are spontaneous breaths among the forced ones, we are faced with the IMV or Intermittent Mandatory Ventilation type.

If all the respiratory acts are spontaneous, we have CSV or Continuous Spontaneous Ventilation.

Do not treat the acronyms CMV and IMV in this as pure modes. The acronyms here indicate a “group” not a single modality.

The IMV type deserves a separate discussion, which we divide into four parts:

IMV1: delivers forced breaths without caring what the patient "asks".

IMV2: allows the patient to breathe spontaneously, abolishing forced breaths if the spontaneous ones have a higher frequency than the forced ones. This mode could be “tricked” by rapid shallow breaths which can lead to hypoventilation.

IMV3: here the previous problem is solved by allowing the patient to activate spontaneous breaths while keeping an eye on a set minimum ventilation per minute.

IMV4: Allows the patient to transform a controlled mode to spontaneous with pressure support if the patient's effort is adequate. This ignores the Vt or Inspiratory Pressure targets set.

The fact that there are two control modes, and three types of breathing allows these types of ventilation to be categorized into just five groups, as follows:

## Five Basic Ventilatory Patterns

Control Variable	Breath Sequence	Symbol
Volume	Continuous Mandatory Ventilation	VC-CMV
	Intermittent Mandatory Ventilation	VC-IMV
Pressure	Continuous Mandatory Ventilation	PC-CMV
	Intermittent Mandatory Ventilation	PC-IMV
	Continuous Spontaneous Ventilation	PC-CSV

It is not possible to have a CSV type breath in volume mode, because the volume is controlled by the patient. If you remember it appeared in another image with a nice red minus sign.

**Targeting schemes** represent systems that allow the ventilator to reach set targets. They can be as simple as entering a parameter, or more complex. There are currently seven targeting schemes, each identified with a letter. Different targeting schemes can also be used in a single mode.

These are the 7 targeting points:

1 Set-point (**s**): all targets are set by the operator.

Volume Assist/Control

2 Dual (**d**): The ventilator can switch between volume and pressure control.

“flow adaptation” Servo-U

3 Bio-variable (**b**): The ventilator randomly changes inspiratory pressure to mimic spontaneous breathing.

Variable pressure support Drager V500

4 Servo (**r**): increases the pressure proportionally to the inspiratory effort.

NAVA Servo-U, PAV PB980

5 Adaptive (**a**): The ventilator changes settings on its own to reach its targets.

Pressure Regulated Volume Control Servo U

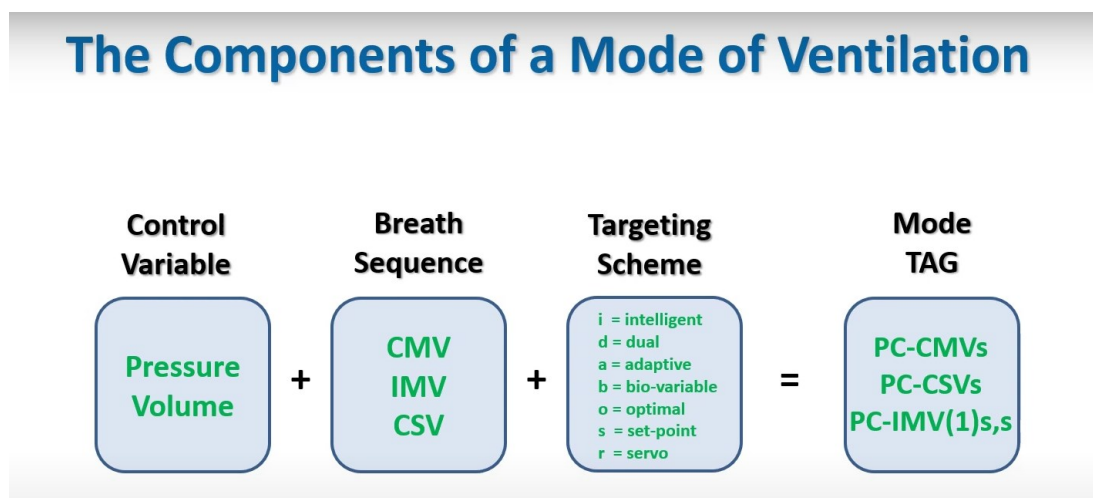
6 Optimal (**o**): there are only two ventilatory modes capable of doing this and here we have tried to reduce the work of the ventilator on the lung to a minimum, also obtaining fewer risks.

Adaptive Support Ventilation Hamilton C6

7 intelligent (**i**): where neural systems and an available database allow machines to independently manage ventilation is a field that is entirely a work in progress.

SmartCare Drager V500, IntelliVent Hamilton C6

## The Components of a Mode of Ventilation



In the image, we see how the three components lead to the generic TAG mode.

Ventilations in IMV that have different breathing patterns, spontaneous or forced, will have different names.

What I have tried to explain in these pages is Professor Chatburn's taxonomy, a system that some ventilator manufacturers are already trying to adopt.

Although still little known, it seems to me to be a very useful approach to understanding ventilation more effectively. For the purpose of this work, I believe that these explanations are sufficient.

There are so many things to know and discuss about this system: I invite you to check online, there is so much material available.

# Part five: The ventilation modes you need to know

In the table, you can see how I have grouped, "by my choice", the ventilation modes.

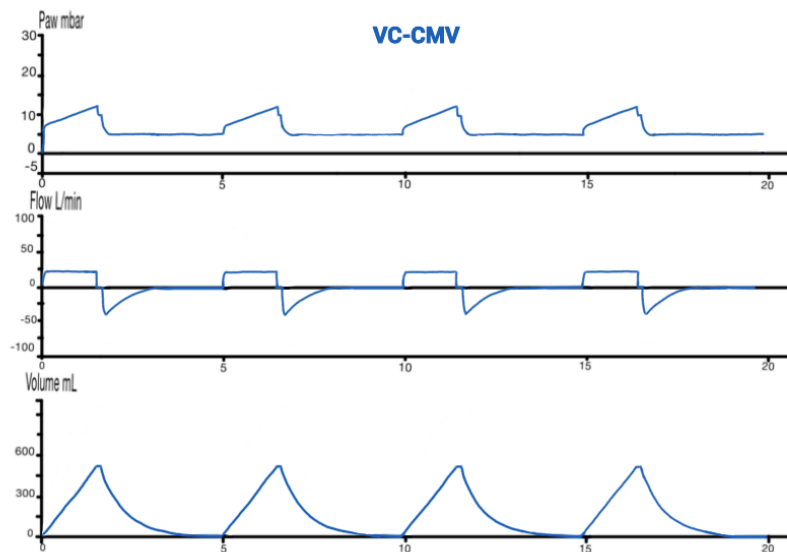
<b>1</b>	<a href="#"><u>CMV</u></a>	<a href="#"><u>A/C</u></a>	<a href="#"><u>IMV</u></a>	<a href="#"><u>SIMV</u></a>	<a href="#"><u>CMV-A/C-IMV-SIMV</u></a>
<b>2</b>	<a href="#"><u>CPAP</u></a>	<a href="#"><u>BiPAP</u></a>	<a href="#"><u>APRV</u></a>	<a href="#"><u>PSV</u></a>	<a href="#"><u>CPAP-BiPAP-APRV-PSV</u></a>
<b>3</b>	<a href="#"><u>PRVC(APV)</u></a>	<a href="#"><u>VAPS</u></a>	<a href="#"><u>ASV</u></a>	<a href="#"><u>PRVC-ASV</u></a>	<a href="#"><u>PRVC-ASV-VAPS-APV</u></a>
<b>4</b>	<a href="#"><u>PAV</u></a>	<a href="#"><u>IRV</u></a>	<a href="#"><u>MMV</u></a>	<a href="#"><u>SHIP</u></a>	

## CMV

CMV, commonly known as assist control, is a breath sequence for which spontaneous breaths are not possible between mandatory breaths because every patient-triggered signal in the trigger window produces a ventilator-cycled inspiration (i.e., a mandatory breath).

The mandatory breathing frequency for CMV may be higher than the set frequency but never below it (ie, the set frequency is a minimum value).

The defining characteristic of CMV is that spontaneous breaths are not permitted between mandatory breaths.



### Advantages

When the patient does not have his own respiratory drive, the CMV does everything expected, following the settings given by the clinician.

### Disadvantages

Widely used before the advent of A/C, but if the patient tries to take a breath, he works against the machine which does not assist him. This can lead to asynchronies and increased patient effort with a sensation of lack of air.

Since the patient must be heavily sedated for correct ventilation, significant muscle atrophy occurs in a short time (about 48 hours) which delays the timing of a possible extubation.

## Monitoring

In this mode the variation in compliance and resistance of the airways will cause the peak inspiratory pressure to vary, this can be dangerous if not carefully monitored and with well-set alarms.

In this mode, we set a  $V_t$  or inspiratory pressure, flow,  $T_i$ , FR, trigger which is always timed, cycling also timed. Both the  $V_t$ , the flow and  $T_i$  are limits here, a maximum pressure alarm is set: if it reaches the pressure limit before having given the set  $V_t$ , the ventilator stops inspiration. We also set the  $FiO_2$  and PEEP which are present in practically all ventilation modes. If you set a  $V_t$ , the control will be by volume and the ventilator will vary the pressure to obtain that volume.

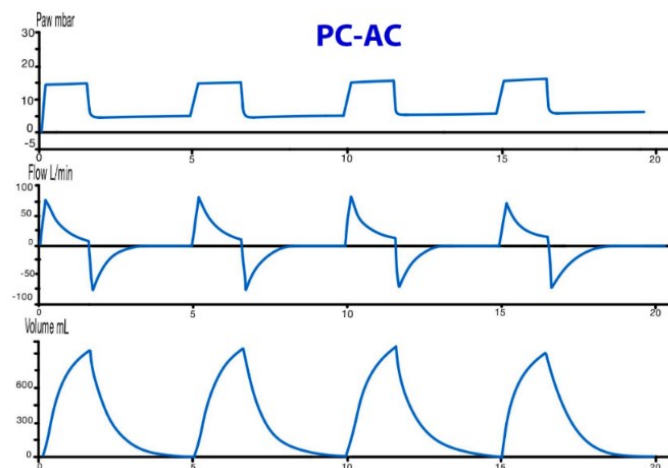
With a patient who is sedated and curarized, and therefore we have eliminated any attempt at spontaneous breathing, then the CMV, A/C and SIMV modes will behave the same. CMV can be volume-controlled or pressure-controlled.

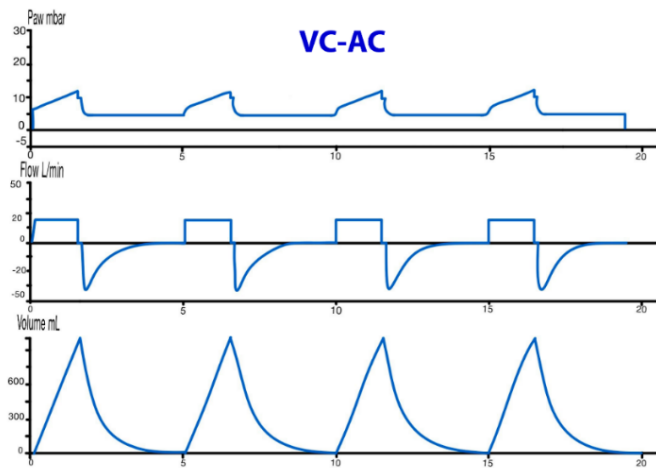
Some ventilators no longer have pure CMV mode, but similar ventilations. I also include the pure PCV and VCV modes among the CMVs which are real CMVs but allow the patient to breathe spontaneously if adequate in volume or pressure.

## A/C

### Assisted Controlled Ventilation

This is an assisted controlled ventilatory mode.





By GLF

Two examples of AC ventilation. Curves vary from patient to patient, these are just examples.

In this mode we set:  $FiO_2$ , RR,  $V_t$ , PEEP, I:E, Ramp,  $T_i$ ,  $T_e$ , limit pressure, trigger.

Once I have set RR and  $V_t$ , the minimum minute volume is also determined, this allows the control of  $CO_2$  and pH; minimal because the patient can intervene with spontaneous breaths.

We set the  $V_t$  as always between 6-8 mL/Kg. By setting the flow, i.e. how quickly the  $V_t$  is delivered, we also decide the  $T_i$ . If a patient is air hungry, you can reduce the  $T_i$ , thereby increasing the flow and therefore reducing the time the  $V_t$  is delivered. Alternatively, you can increase the flow directly. Of course, having set a breathing rate tells us that we have a timed trigger regulated by the ventilator. If the patient tries to breathe, they can do so with a pressure or flow trigger. In this case, the ventilator will allow him to breathe.

Cycling occurs in time. In this mode, the flow, in addition to the  $V_t$ , is also limited by the settings and even if the patient requests a greater flow with his spontaneous breathing, it will not be delivered.

Finally, we set the pressure limit as an alarm.

The patient's spontaneous breathing is assisted-controlled breathing, because it is limited in  $V_t$  and flow.

It is not a **true spontaneous breathing**, but it is limited by the ventilator according to the set parameters.

To give an example, if we have set an RR of 10 breaths/minute and the patient starts a breath before 6 seconds have passed ( $60'' : 10 = 6''$ ), the ventilator will deliver a breath 6 seconds after the spontaneous one, not interfering with the patient. This is because it delivered the volumes pre-established by us as in the automatic acts.

Remember that a spontaneous breath is usually smaller than that of the machine.

The risk in this mode is that the patient will hyperventilate because the respirator will deliver a full breath every time it is required, this can lead to alkalosis.

This modality is very similar to CMV, but here the patient has some spontaneous trigger activity.

When the patient tries to breathe, it generates a negative pressure that the ventilator "feels" via its trigger settings. In this case, the ventilator gives a set amount of air to the patient, therefore the set  $V_t$  or the set pressure.

Every time the patient triggers the ventilator it will give the specific set  $V_t$ .

If the patient does not ventilate, the ventilator will act according to the set frequency.

For this reason, it is an assisted mode that assists with a known  $V_t$  spontaneous breaths or triggers, but controlled when the patient is not breathing.

If you have set the volume control mode, the pressures can become very high, if the high pressure alarm sounds, you need to check what is happening. It may be secretions blocking the tube, there may be condensation forming or the patient biting the tube. Another cause could be bronchospasm.

## IMV

### Intermittent Mandatory Ventilation

It is a now disused ventilation modality, applied to a spontaneously breathing patient to support ventilation, improve the quality and quantity of ventilation. It is a combination of spontaneous breathing with mechanical ventilation.

This mode was originally developed as part of an effort to create a mode in which the patient could interact with the ventilator, simultaneously using the respiratory muscles, so that it could be useful in rapidly weaning the patient from ventilatory support.

In IMV, the ventilator has a set RR and Vt. The breathing acts are guaranteed by the machine based on the settings given by the clinician. If the patient can initiate spontaneous breaths, these will not be supported. Thus, these breaths may be higher in frequency and are usually lower in volume achieved. The volumes obtained are therefore dependent on the patient's effort.

If the set RR is low, the patient is more likely to take spontaneous breaths.

Here, the volume is not guaranteed, unlike the A/C mode, where the breath is delivered at the set volume.

Pressure support is usually added to the spontaneous breaths.

It is used in patients with normal spontaneous ventilation who are unable to perform the entire WOB.

Used as an initial form of weaning, to allow the patient to achieve a certain respiratory independence.

### Advantages

Less likely to hyperventilate, as the patient changes frequency and adjusts tidal volume. This allows you to maintain normal CO<sub>2</sub> values.

This mode reduces the risk of muscle atrophy by using muscles more than CMV or A/C. With spontaneous breathing, hemodynamic alterations are also reduced.

### Disadvantages

Not least, the mandatory breath can be delivered at any moment of the patient's spontaneous breathing, leading to even serious asynchronies.

It can lead to significant increases in Vt if the forced breath falls during the patient's inspiration, especially in the final phase; this can generate barotrauma. Volumes and pressures can become so high that they create problems.

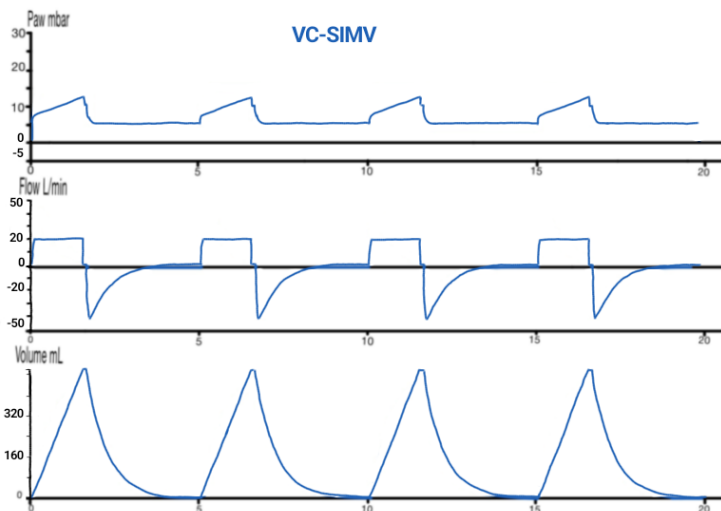
Furthermore, the patient can easily experience fatigue, with a reduction in Vt and an increase in RR. Check for any asynchronies and patient comfort

IMV mode has been completely replaced by SIMV.

## SIMV

SIMV or Synchronized Intermittent Mandatory Ventilation.

In SIMV not all spontaneous breaths are terminated, but with this modality, compared to CMV and A/C, the patient is given more freedom; Cycling is the same as in the previous modes, but here the patient can also cycle.



You set,  $FiO_2$ , PEEP, PS; we do not set a  $V_T$  or RR because they are managed by the patient.

In SIMV if the patient tries to breathe the ventilator does not deliver the full volume, for example 500ml, as in A/C. While in the A/C we do not have a PS, in the SIMV a PS is set which intervenes only in spontaneous acts. The PS is set to ensure the patient takes in the required amount of air (volume), thus ensuring correct MV.

In SIMV, therefore, it is the patient who determines the breath volume  $V_t$ .

Please note that if the patient does not perform any spontaneous acts A/C and SIMV are identical. SIMV is a suitable modality in weaning the patient.

SIMV and A/C are indistinguishable in a sedated patient without spontaneous activity.

If we are ventilating a patient in SIMV for a possible extubation we set PS values of 10  $CmH_2O$  and a PEEP of 5  $CmH_2O$ , and then go up to 5 and 5 for a very short time before extubation.

When the patient wants to take a spontaneous breath, he is able to take a **real spontaneous breath** in this mode. In fact, here the patient decides the cycling, not the ventilator. I remember that in timed cycling, the ventilator senses when the flow is decaying and allows exhalation. Furthermore, the patient is free to go beyond the settings.

In this condition the  $V_t$  and flow can vary, but when the breaths are from the ventilator  $V_t$  and flow will be the ones set.

As for the limits, the flow and  $V_t$  are not limited, only the pressure alarm limit remains. In addition to sounding the alarm, the pressure is also limited by interrupting inhalation. We must also point out that in a spontaneous breath we are not affected by the pressure because we have the movement of the diaphragm which compensates for this pressure.

SIMV covers ventilations from controlled and assisted to pure spontaneous.

SIMV occupies a good 30% of ventilations in intensive care.



In SIMV the ventilator delivers breaths and if the patient has spontaneous breaths it supports him only if they are synchronized. Since the ventilator allows the patient to take spontaneous breaths, muscle atrophy is avoided.

Each machine has different algorithms, but the operation is more or less as follows.

In SIMV, if the patient starts breathing at 3 seconds and the programmed one was at 7 seconds, the ventilator will allow the patient to breathe without assisting him; this breath will be "patient self-made", pressures and volume depend on the patient's effort. But if the patient starts a breath at 6 seconds (or close to the automatic act) the synchronization present in the name intervenes which prevents the two breaths from overlapping. In this mode, if the patient triggers the breath shortly before the ventilator delivers the forced one, the ventilator will assist the patient's breathing with the values that he would have used for an automatic breath, synchronizing with the patient's effort. Remember that spontaneous breath usually has a smaller flow and volume than that of the machine.

To overcome this in the SIMV we can add a support pressure which will allow for adequate  $V_t$  and flows. You already know that pressure support only occurs in spontaneous breaths.

When inspiration ends, the ventilator notices the patient's drop in inspiratory flow and allows him to exhale as a "true spontaneous breath".

SIMV was created because in mechanical ventilation the main problem is the asynchrony between patient and ventilator (see IMV). To prevent this asynchrony, an innovation was designed, in which the ventilator detects the onset of the patient's spontaneous inspiratory effort and provides mandatory breathing by synchronizing it with the effort.

If the patient does not make an inspiratory attempt within the time window, mandatory breath is provided at the scheduled time.

#### Monitoring

In this mode it is necessary to monitor the patient's RR, which could become too high. A patient with shallow, rapid breathing may develop atelectasis, with worsening respiratory failure, decreased compliance, and increased WOB.

In volume-controlled SIMV it is advisable to adjust the pressure alarms of the peak inspiratory pressure (PIP), which can easily be overcome in the event of a change in compliance or resistance. It is necessary to check that the patient is in a comfortable condition.

#### Directions

In patients with normal respiratory stimulus, but with respiratory muscles unable to support the respiratory load (WOB), up to patients in weaning.

#### Advantages

Since the mandatory breaths are synchronized with the patient's inspiratory efforts, greater comfort is ensured for the patient.

Lower risk of respiratory muscle atrophy, as the patient uses the muscles to a greater extent.

Less hemodynamic compromise, as the patient ventilates at a lower mean airway pressure ( $P_{aw}$ ).

#### Disadvantages

If not well monitored and with properly set alarms, we can have high rates and potential alkalosis.

Has been shown to prolong the weaning phase compared to PSV or T-piece modes.

## CMV, A/C and SIMV in short

In the mode **CMV**, all respiratory acts are forced breaths of uniform duration and pressure.

In the mode **A/C**, all respirations are mechanical breaths as in CMV, but spontaneous breaths are allowed; these will be assisted by the machine with the volume or pressure values set. Leaving little freedom to the patient, they are not in fact true spontaneous breaths.

In the mode **SIMV**, the ventilator delivers forced breaths, but allows the patient to take true spontaneous breaths, synchronizing with the patient.

At the end of the fair:

**CMV** does not allow spontaneous breaths

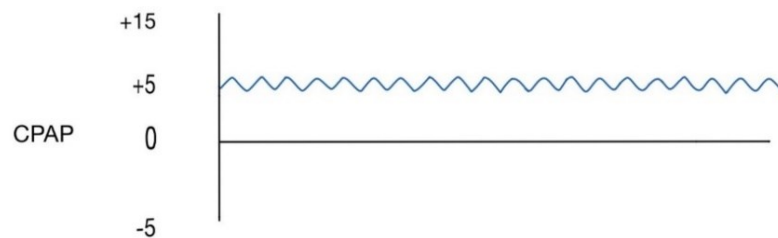
**A/C** allows spontaneous breaths but only with the set volumes.

**SIMV** it allows true spontaneous breaths that have their own characteristics.

## CPAP

Continuous Positive Airway Pressure

Spontaneous, assisted mode



This curve shows how a patient ventilates above the PEEP or CPAP level.

CPAP finds its best use in hypoxemic respiratory failure, in congestive heart failure where it reduces preload by improving CO.

Recruits alveoli, reducing WOB, helps increase FRC.

CPAP is a spontaneous ventilation mode (SV or Spontaneous ventilation)

CPAP is a ventilation mode applicable via a mechanical ventilator or via different devices capable of producing an adequate flow.

Without the ventilator, a continuous flow of air approximately 2-3 times greater than the minute volume, approximately 25 liters/minute, and a PEEP valve are used.

Remember that in this mode we do not have control of the patient's breathing and therefore of any apnea.

The CPAP mode improves the FRC (Functional Residual Capacity), improves oxygenation but not ventilation, therefore it does not change PCO<sub>2</sub> values.

Particular attention must be paid to the PEEP level set which, if it is very high, does not allow the patient to exhale and breathe easily and will have the sensation of expelling air "against a wall".

A person breathing spontaneously starts from a zero airway pressure value to reach a maximum during inhalation, the reverse process occurs during exhalation which will bring the pressure back to zero.

A person who ventilates spontaneously through a PEEP will find at the moment of exhalation a moment in which he will not descend beyond a certain limit; in this sense, the higher the PEEP, the more difficult it will be for the patient to breathe.

This CPAP or PEEP value is applied throughout the respiratory cycle, naturally it is necessary that the patient is able to breathe spontaneously.

In this context, talking about CPAP or PEEP is the same, furthermore in CPAP the patient does all the breathing work and must be able to develop an adequate  $V_t$  since the ventilator does not provide any support and no mandatory breaths are given. However, we can insert a PS into the CPAP in order to help the patient reach adequate  $V_t$ .

#### Advantages

CPAP recruits the alveoli, helps in pulmonary edema of cardiac origin, congestive heart failure and sleep apnea. It is used as NIV in pneumonia, COPD, can prevent intubation and can be used as a weaning modality.

The CPAP has the main task of providing a positive pressure throughout the respiratory act, this pressure behaves like PEEP in the expiratory phase, never causing the pressure level to drop below the set CPAP (PEEP) value.

We usually use settings between 5 and 15  $\text{cmH}_2\text{O}$ , we reach a maximum of 25-30  $\text{cmH}_2\text{O}$ , but above 20 the risk of stomach insufflation will increase with possible vomiting and hemodynamic worsening.

We use it in patients with adequate ventilation but low oxygenation due to reduced FRC, or the presence of atelectasis or accumulation of secretions.

#### Disadvantages

The use of positive pressure reduces cardiac output due to reduced venous return, increases intracranial pressure and exposes to barotrauma.

#### Monitoring

We must monitor the respiratory rate which should be  $<25$  breaths/minute. If too high,  $V_t$  will drop and the patient may become fatigued. In this case further support must be provided.

The comfort of the patient is also important, and he must not be tired, stressed or anxious. This is true both in NIV and non-NIV.

You must pay close attention to the alarm limits set in the ventilator. If the ventilatory rate drops too low, the ventilator must enter a backup mode (called apnea ventilation).

CPAP can be used to wean the patient a few hours before extubation.

#### CPAP vs CPAP with pressure support.

If a patient is connected to the ventilator in CPAP without pressure support, we get fully patient-controlled ventilation. If we have inserted a PEEP the only thing we see in moments of breathing pause is the PEEP line in the part relating to the pressure.

In the flow we will notice a very limited flow and in the volumes what the patient breathes as tidal volume. Typically, the patient will have a high ventilatory rate associated with low  $V_t$  to reach his target minute volume.

Target not set by us but intended as necessary for the patient.

By giving a support pressure (5 $\text{cmH}_2\text{O}$ ), the patient generates much higher  $V_t$  with the same effort as before, because the ventilator will help by increasing the pressure with each inhalation. That pressure support creates steps corresponding to the inspiratory act that exceed the PEEP pressure,

and for this reason in many cases you will hear about Support Pressure as **Over-PEEP** pressure. The Vt easily goes from values of 150 mL to 250 mL.

By raising the support pressure to 10, the increase in pressure and volumes is even more visible. Arriving at normal values, therefore around 500 mL, remember however that each Vt will depend on the patient's effort and therefore they will **all be different**.

We can therefore say that pressure support provides extra pressure to the patient to obtain a higher tidal volume. This allows the patient to reduce the respiratory rate to obtain the necessary Minute Volume.

A nice boost for the patient, don't you think?

This continuous positive pressure constitutes a difficulty in exhaling, as we have already seen, but we can overcome this problem by starting with low pressures, giving the patient time to adapt, and then moving on to the "cruise" pressure, which will allow us to obtain the desired results.

Once the PEEP is set, we can enter a support pressure that will add to the baseline or PEEP pressure. Therefore, by setting 10 PEEP and adding 15 support pressure, we will arrive at 25 pressures, this will increase the Vt and MV. The support pressure must be adjusted until we reach the value of 6-8 ml/kg or the desired one.

Also in CPAP, it is not possible to set an I:E ratio, remember why?

Because the patient decides the I:E ratio which is not fixed.

Care must be taken not to increase the pressure too much, because otherwise we could lead the patient to vomit due to the increase in chest pressure. For this reason, it is necessary to always place a nasogastric tube and empty the gastric contents before starting CPAP. Furthermore, too high blood pressure can cause hemodynamic problems. Even if evidence shows that up to 25 cmH<sub>2</sub>O of pressure is necessary to open the gastroesophageal sphincter, the conditions we may find ourselves in with these patients (CPR for example) must lead us to be cautious. In any case, we refer to the instructions of the structure.

Some differences between PSV and CPAP:

Mode	CPAP	PSV
Set	CPAP or PEEP pressure	Support pressure
Triggers	Patient	Patient
Cycling	Patient	Flow reduction
Limits	No pressure limits	Pressure no more than PS

The set CPAP value does not modify the Vt or MV, it only modifies the pressure baseline, therefore its increase does not increase the removal of CO<sub>2</sub>; changing the Support Pressure changes the Vt and Vm, leading to a change in CO<sub>2</sub> removal. Remember that since it is spontaneous ventilation, each breath will have a different and patient-dependent Vt.

We can also say that CPAP is like SV (Spontaneous Ventilation) + PEEP.

What we understood about this ventilation mode: that CPAP finds its best use in improving oxygenation.

One of its "evolutions" is BiPAP which, in addition to improving oxygenation, also improves ventilation.

Let's briefly remember what we mean by Ventilation and Oxygenation

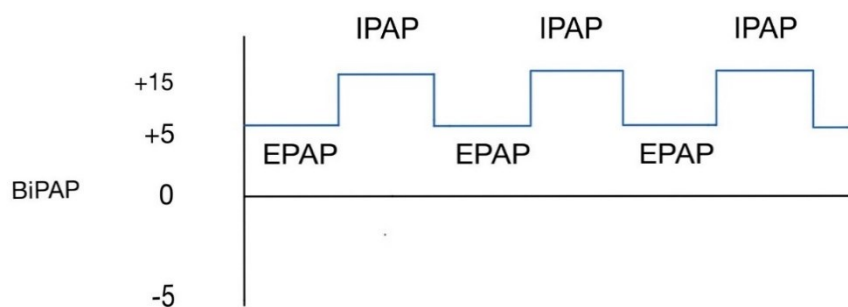
**Oxygenation is the supply of oxygen to the tissues and therefore its exchange.**

**Ventilation is the possible control of CO<sub>2</sub> and therefore of the PH that can be assessed by the EGA.**

## BiLevel, BPAP or BiPAP

Bilevel Positive Airway Pressure

Assisted ventilation mode



*By GLF*

The typical BiPAP curve with the two pressure levels.

Modality used mainly in hypercapnic or mixed respiratory failure. Used in COPD and heart failure, it can prevent intubation and is useful in patients who are difficult to wean. In BiLevel the patient can breathe spontaneously at any moment of the forced respiratory act.

In the Bilevel we need to set IPAP, ePAP, iTime, FiO<sub>2</sub>.

“Bilevel” and “BiPAP” are often used interchangeably to refer to the same ventilation mode. Both refer to biphasic positive airway pressure ventilation. This ventilation mode provides two pressure levels:

one high during inspiration (IPAP - Inspiratory Positive Airway Pressure)

a low one during exhalation (EPAP - Expiratory Positive Airway Pressure)

Saying EPAP or ePAP and iPAP or IPAP is the same thing.

The term "BiPAP" is a registered trademark of the Respironics™ company (Philips) but has come into common use to refer to any device that provides biphasic pressure ventilation.

The term "Bilevel" refers to the fact that two levels of pressure are used.

Do you remember the limit that the CPAP ventilation mode had? I mean that famous wall against which it is difficult for the patient to breathe. Here the ventilation engineers looked for a solution.

They succeeded with BiPAP by making the act of exhaling easier for the patient. Bilevel in fact indicates two different levels of pressure during the respiratory cycle, in particular **that during exhalation is kept lower** than that present during inhalation. In this way "that wall" in a certain sense disappears or at least is reduced.

ePAP keeps the alveoli open and is similar to PEEP or CPAP.

**In fact, CPAP is nothing more than a BiPAP without iPAP!**

If we don't have oxygenation problems ePAP can remain at zero.

We can define the difference between iPAP and ePAP as the support pressure. If the PS is increased it causes the Vt to rise. It is therefore the true creator of the improvement in ventilation. Increasing the Vt will in fact increase the MV and therefore decrease the CO<sub>2</sub>. Let us consider here the level of CPAP = ePAP, and since the basis of CPAP is none other than PEEP it becomes that:

**ePAP-CPAP and PEEP are the same thing associated with different ventilation modes.**

Remember that in this ventilatory mode the difference between ePAP and iPAP corresponds to pressure support.

<b>iPAP - ePAP = Pressure Support</b>
---------------------------------------

This ventilatory mode is used in case of dyspnea, ARDS, worsening or exacerbation of COPD, congestive heart failure, pulmonary edema, prevention of intubation, post extubation to avoid re-intubation, obstructive sleep apnea.

It can also be used as NIV, like CPAP.

BiPAP is a modality that prevents machine-patient asynchronies, certainly more comfortable than CPAP alone.

Here too there is a safety mode in case the patient does not perform respiratory acts. The pressures can be adapted to the patient's improvement, gradually leading him to completely spontaneous breathing.

**Advantages**

Improves alveolar ventilation without sacrificing oxygenation.

Minimizes intrathoracic pressure and reduces the need for sedation while maintaining CO<sub>2</sub> elimination.

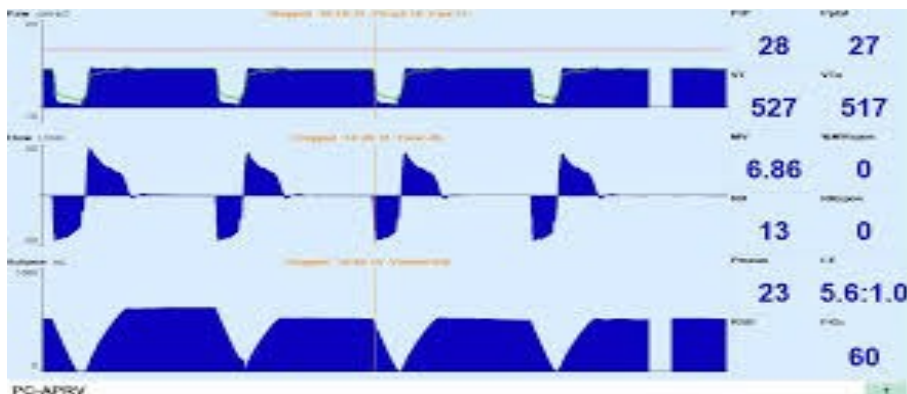
Now I will introduce you to a ventilation mode that is very similar to BiPAP, although different.

**APRV**

Airway Pressure Release Ventilation

Airway pressure relief ventilation

Controlled-assisted ventilation mode



Example of curves in APRV mode.

It is classified as PCV-IMV with inverse ratio

It is a ventilatory mode similar to BiLevel, used especially with patients with ARDS; it is able to increase alveolar recruitment and improve oxygenation.

Used in acute respiratory failure and in cases of severe atelectasis.

It is a pressure ventilation mode, so the  $V_t$  depends on the patient's resistance and compliance.

In this mode you set a  $T_{high}$ , a  $T_{low}$ , a  $P_{high}$  and a  $P_{low}$ .

$P_{high}$  is the first setting you need to set and represents the pressure level in the inhalation phase.

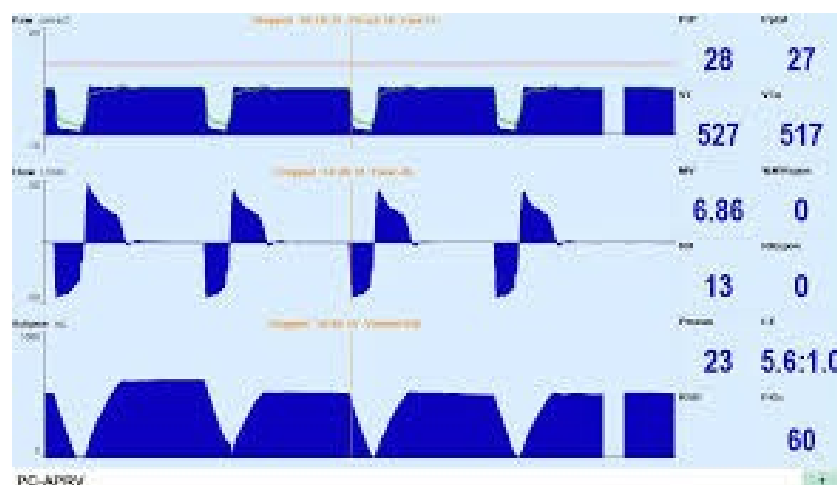
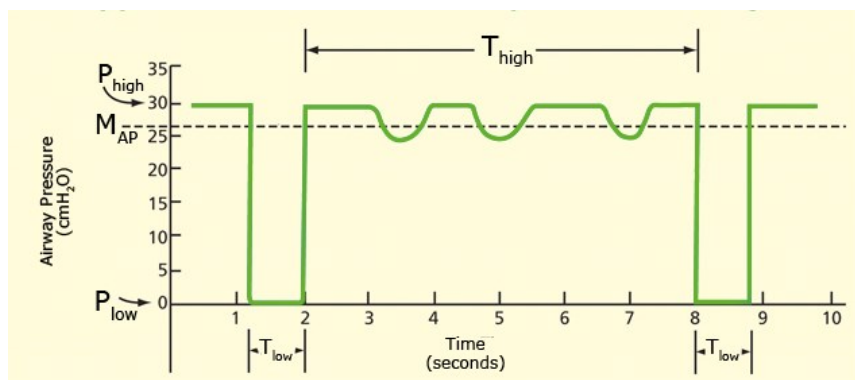
The second is  $P_{low}$ , where you will indicate how far the pressure will drop on exhalation. Basically, we have to tell the ventilator how long to maintain these pressures by setting a  $T_{high}$  and a  $T_{low}$ .

$T_{high}$  is the time referred to  $P_{high}$  and  $T_{low}$  is the time that the  $P_{low}$  must last.

Some ventilators do not allow you to set a  $T_{high}$  and a  $T_{low}$ , but a breathing rate. It doesn't change much: by also setting an I:E ratio we get the same result. We just have to get used to it.

The choice of  $P_{high}$  is usually given by the  $P_{plat}$  value that the patient has with a volume-controlled mode. It can also be set as a percentage of the desired peak inspiratory pressure value or tidal volume; the clinician will decide which path to choose. The most correct method remains the first.  $P_{low}$  is often set to zero. Usually the  $T_{high}$  is around 4-6 seconds and the  $T_{low}$  0.5 seconds if not less, this time is calculated on values between 50% and 75% of the peak expiratory flow.

In a nutshell, once the expiratory peak is reached, let's say 100L/minute it begins to decelerate and once 75% of its value has been reached, the ventilator immediately triggers inspiration.



We do not see a set breathing rate because it is obtained from the Thigh and Tlow settings, based on the total breath time. Let's imagine we have a patient with a Thigh setting of 5.5 seconds and Tlow of 0.5 seconds: the breath will last 6 seconds. In one minute therefore, the respiratory rate will be 10 breaths per minute. Doing the reverse reasoning and wanting to choose a setting, we must consider that in APRV Thigh and Tlow often have a ratio of 10:1. Considering this and wanting a frequency of 10 acts per minute, we consider a Tlow of 0.6 seconds: it will be easy to predict a Thigh of 5.4 seconds for an act that lasts 6 seconds.

#### Advantages

The APRV, maintaining an equal pressure throughout the inspiratory phase, manages to keep the average airway pressure high. Remember that mean airway pressure helps recruit atelectatic areas and closed alveoli, increasing FRC; this will improve the patient's oxygenation.

The shape with which exhalation begins indicates a large flow expelled almost instantly, therefore a large quantity of CO<sub>2</sub>, improving the PCO<sub>2</sub> and pH values.

Since we have set the start of inspiration before 75% of the expiratory flow is expelled we obtain an autoPEEP, which is why we can afford to set Plow to zero; we still have an intentional autoPEEP, in fact the baseline never reaches zero; if this were to happen it would be counterproductive for the purposes of alveolar recruitment.

It allows the patient to take **real spontaneous breaths** during all respiratory phases, naturally it will be "percentage" less likely during the expiratory phase. This is important in a context of mechanical ventilation, because being able to ventilate spontaneously for the patient has the great advantage of reducing any diaphragmatic atrophy that may be present, therefore leading to extubation more quickly.

.... Yes, because, even if it seems counterintuitive, we put patients on mechanical ventilation with the aim of weaning them as soon as possible!....

The patient can take spontaneous breaths throughout the inspiratory phase, his Vt will become higher by adding to that already delivered by the machine.

Let's see some characteristics of the APRV compared to other Volume Controlled ventilations:

- 1 maintains a lower peak pressure
- 2 maintains a higher mean airway pressure
- 3 has an average higher P/F index
- 4 the average arterial pressure rises
- 5 time spent in intensive care reduced
- 6 possible reduction in mortality

The APRV mode allows you to ventilate curarized or paralyzed patients. In this case we do not need a modality that allows spontaneous breathing.

It is right to know that there are ventilation modes that are more suitable for this type of patient and, probably, easier to set.

In APRV, the patient can breathe spontaneously above the P<sub>high</sub> level. As soon as we obtain an improvement in compliance and P/F, we can think about reducing P<sub>high</sub> to reach a lower level of assistance; this is useful for the beginning of weaning. Furthermore, you can eliminate the expiratory portion and, since the patient is taking spontaneous breaths, the APRV practically transforms "almost" into a CPAP. Regarding the driving pressure in APRV mode, according to the P<sub>plat</sub>-PEEP calculation we must establish what values to take to perform this calculation, like this:



One might think that  $P_{high} - P_{low}$  is the driving pressure, but this is not the case. So essentially it is not even necessary to evaluate a driving pressure value in APRV mode. It doesn't make sense because it can't be measured correctly.

#### Advantages

The increase in alveolar ventilation with low peak airway pressure without overdistension of the lung is the main advantage.

Low intrathoracic pressure with better matching between ventilation and perfusion.

#### Disadvantages

Complicated to set up

#### Small comparison between APRV and BiLevel

APRV and BiLevel are not the same thing.

They are both modes in which you can set two levels of pressure.

Both modes tend to increase the average pressure.

But one has a long inspiratory time and a very short expiratory time with a pressure level that reaches zero, the APRV. BiLevel starts from a base of PEEP and has more similar inspiratory and expiratory times.

Both modes allow spontaneous breathing.

In APRV you put  $P_{high}$ ,  $P_{low}$  and the times  $T_{high}$  and  $T_{low}$ . You **don't enter your breathing rate** here.

In BiLevel you set PEEP high and PEEP low then you have a  $T_{high}$  and a RR.

Both improve FRC, oxygenation and p/f ratio, maintaining a low  $FiO_2$  level.

In BiLevel you are looking for a 1:1 or 2:1 ratio

For example, for 10 acts put 3 seconds and 3 seconds.

They are not the same ventilation mode, but by changing the settings you can get basically the same ventilation.

These are the best ways to improve oxygenation. APRV would reduce patient mortality, according to encouraging preliminary evidence.

## PSV

#### Pressure Support Ventilation

It is a continuous spontaneous mode

It is a real ventilation mode, but it is also a setting to be added to different ventilation modes so don't get confused. PS is the pressure support, the pressure that the ventilator adds to the patient's respiratory effort; it only does it with spontaneous breaths.

When a support pressure is set, it is delivered by the ventilator **only in the presence of truly developed spontaneous acts**.

If a patient fails to trigger a spontaneous breath, pressure support is not delivered; if the ventilator performs an automatic act, the support pressure is not delivered.

If there are no true spontaneous acts, any support pressure setting has no effect.

This is an important concept to know.

Thus, ASB, IPS, IFA refer more to the additional setting than to the actual ventilation mode.

These acronyms all refer to the PSV ventilatory mode.

**ASB** stands for Assisted Spontaneous Breathing.

**IPS** stands for Intermittent Positive Pressure Support.

**IFA** stands for Inspiratory Flow Assist.

In practice, these settings define how PSV mechanical ventilation helps the patient breathe.

**ASB** it is the simplest mode of PSV. The patient initiates his or her breathing and mechanical ventilation provides the pressure support necessary to complete inspiration.

**IPS** it is similar to ASB, but the support pressure is applied for a shorter period of time, for example for a quarter or half of the inhalation. This can be useful in helping patients develop their breathing strength.

**IFA** is a more complex PSV mode that provides assistance to inspiratory flow.

This mode can be useful for patients who have difficulty breathing.

In PSV each breath is activated by the patient. It can be used in invasive ventilation and NIV and is used for weaning patients. The flow delivered depends on the set pressure, airway resistance, lung compliance and the patient's inspiratory effort. Cycling occurs when there is a decrease in inspiratory flow (25%-30%).

The tidal volume depends on the duration of inspiration.

In this mode we set the support pressure, the PEEP, the FiO<sub>2</sub>, an inspiratory trigger, an expiratory trigger and a limit pressure.

Minute ventilation depends on the RR and the tidal volume of each individual breath.

The ventilator does not deliver any forced actions, but there is a backup system if the patient goes into apnea. For this reason, correct alarm setting, and continuous surveillance are necessary.

We use PSV to provide oxygen and support ventilation in patients with hypoxemic, hypercapnic or mixed respiratory failure. It is also chosen to check whether a patient can be extubated. The support pressure helps to increase the V<sub>t</sub> and MV, favoring the elimination of carbon dioxide. PEEP contributes to alveolar recruitment, improving oxygenation.

PSV promotes a reduction in oxygen consumption and work of breathing.

The pressure support also helps overcome the resistance of the endotracheal tube, making it easier for the patient to "breathe" through it.

It can only be used with patients who have spontaneous respiratory activity.

The minute volume may not be achieved, as it is a patient-dependent variable.

A patient who ventilates in PSV for 30-120 minutes, with good tidal volumes and normal ABG, is a patient considered "extuable".

A patient may have hypoventilation and subsequent hypoxemia if not adequately monitored.

In PSV the patient decides the respiratory rate. Once the inspiratory act has begun, the support pressure is delivered by the ventilator throughout the entire inhalation. The breathing act is started if the patient is able to activate the set trigger. The end of inspiration is triggered by the decrease in inspiratory flow, as mentioned before. In Pressure Support the ventilator must help the patient overcome the pressures of the artificial airway, such as the tube.

Based on the support we apply, we obtain different effects, up to 5-8 cmH<sub>2</sub>O helps to overcome the resistance of the artificial airways; from 10-15 cmH<sub>2</sub>O we have a boost in the patient's V<sub>t</sub>.

Therefore the support pressure has two important tasks: it overcomes the resistances and increases the V<sub>t</sub>.

In some ventilators the support pressure is called pressure above PEEP.

In this type of ventilation, the flow will rise rapidly and then take on the characteristic decelerating shape. This is because, as the lungs fill, the flow slows and is reduced.

The pressure curve is square, the flow curve is decreasing.

Once the lungs have filled, the flow slows. The ventilator cycles when a specific percentage of that flow is reached. Typically, this is a user-adjustable setting.

In the PSV, if we set a PEEP we will have a PIP equal to the sum of the two:

PEEP 6 + PS 10 = PIP 16 cmH<sub>2</sub>O.

The level of support should ease the WOB of the respiratory muscles. Later, under improved conditions, we can reduce this support. When we reach a PS of 6 cmH<sub>2</sub>O, mechanical ventilation can be stopped.

#### Directions

Since through PS we can gradually modify the work of the respiratory muscles, it is an excellent modality for weaning.

#### Advantages

It reduces the WOB, helps to overcome the resistance of the artificial airways, the patient comes away from the respirator without being exhausted, the patient controls inhalation, exhalation and respiratory rhythm, with better comfort and reduction of asynchronies.

The ability to adjust the support level makes it very adaptable.

#### Disadvantages

Having variable V<sub>t</sub> and MV, there may be inadequate alveolar ventilation.

Secretions, bronchospasm and changes in pressure or compliance will produce changes in V<sub>t</sub> which may be inadequate.

If a leak occurs, the set support level may not be reached, and the ventilator may be tricked into not cycling exhalation.

#### Monitoring

An increased RR and reduced V<sub>t</sub> may indicate patient fatigue. In this case it will be necessary to increase the pressure support.

### CPAP, BiPAP, APRV and PSV in short

In **CPAP** the patient ventilates spontaneously, throughout the respiratory act he has a pressure (PEEP-CPAP) above zero. This improves alveolar recruitment and oxygenation.

In **BiPAP** the patient ventilates spontaneously, but between inspiration and expiration there are 2 different pressure levels. This makes ventilation easier and more comfortable. Furthermore, it is useful in hypercapnia.

In the **APRV** spontaneous breathing is allowed, we do not impose a PEEP. APRV and BiPAP improve FRC, oxygenation and p/f ratio, maintaining a low FiO<sub>2</sub> level.

In **PSV** the patient breathes spontaneously and the ventilator supports him with pressure support. It is used with problems of hypoxemia, hypercapnia, reduces the patient's WOB.

## VCV and PCV

In general, VCV is more effective at ensuring a constant inspiratory volume, while PCV is more effective at avoiding overloading the lungs. The choice of the most suitable ventilation mode depends on the patient's condition and the objectives of therapy.

Remember that many different modes can work with pressure and volume control, although there are dedicated modes such as VCV and PCV.

Another thing to remember: the VCV controls the volume and therefore the pressure will vary based on compliance and resistance of the airways, and patient efforts', while the PCV controls the pressures; here the volumes will vary, always based on compliance and resistance, and patient efforts.

I described these modes in a previous part of this text, where I talk about how to divide ventilation.

## PRVC Pressure-Regulated Volume Control

It is a hybrid type adaptive ventilation with the advantages of a PC and a VC.

Ventilation mode is the same as APV or Automatic Pressure-Controlled Ventilation. Both modes use pressure to control the tidal volume delivered to the patient.

The main difference between the two modes is how the plateau pressure is determined.

In APV, we set the VT and the plateau pressure is automatically adjusted exactly like PRVC, VC+, auto-flow. In PRVC, plateau pressure is automatically determined by the ventilator based on the target tidal volume set by the operator.

It is a form of adaptive ventilation

It works like a controlled pressure to obtain a controlled volume, but the pressure is calculated and modified breath by breath, we set a  $T_i$ , the flow is dependent on the patient's request or the characteristics of the lung. Working like a VC it delivers a desired  $V_t$  with the **minimum usable pressure**.

In this mode, if the patient performs spontaneous acts, and therefore exceeds the pre-established volume, the ventilator will reduce the pressure delivered, leading to an increase in the patient's work of breathing. This could lead to fatigue and hypoxia, this is one of the contraindications for using PRVC.

Small comparison between PCV, VCV and PRVC.

PC	VC	PRVC
stable pressure Vt variable The flow changes with the request Greater synchrony Potential hypoventilation or hyperventilation	Vt stable MV stable Variable peak pressure Greater asynchronies Possible barotrauma	Stable vt Stable MV Flow varies with demand Greater synchrony

So PRVC has all the advantages of the two main modes.

It works through a ventilator feedback mechanism that analyzes breath by breath what it must deliver to the patient, based on variations in compliance and resistance.

The ventilator starts breathing like a normal VCV, automatically calculates the plateau pressure of the inspiratory act. When it takes this first breath, behaving like a VCV, it reaches the set Vt. Given the plateau, compliance and resistance of the pulmonary system, switch to a pressure-controlled mode, setting this pressure right at the level of the plateau pressure!

It then regulates the pressure very slowly and gradually for all breaths until the set Vt is obtained. When the Vt deviates from the correct values, the ventilator always proportionally "corrects the shot" by regulating the pressure supplied.

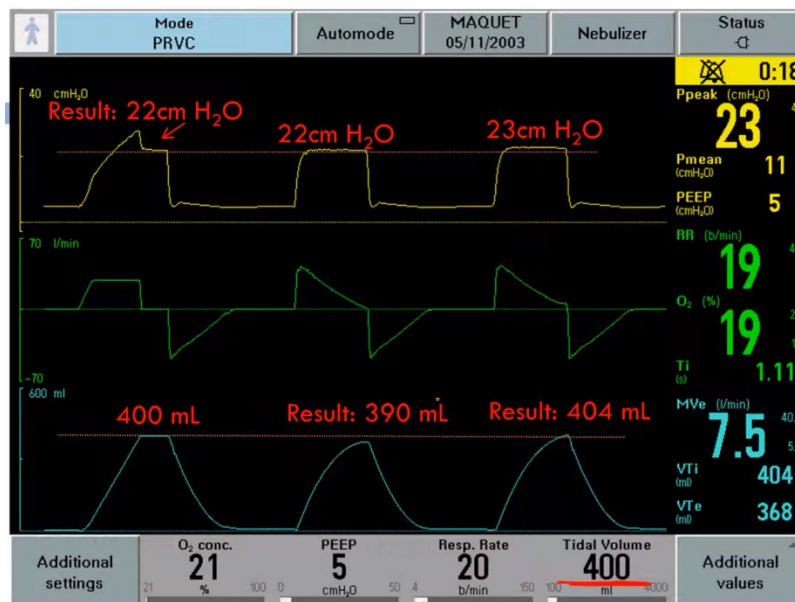


Image permission required

Usually, this mode tries to reach the correct Vt with the minimum possible pressure, and in any case, it will reach a maximum of 5 cmH2O from the maximum pressure set as the limit.

If compliance increases or resistance decreases, the ventilator reduces pressure to bring the Vt back to the set value.

The advantages of this mode include a guaranteed Vt at all times and a much more physiological decelerated flow. In non-sedated patients, this modality is less tolerated and can generate autoPEEP, it is not ideal in patients with obstructive pathologies.

In the Drager™, when you set the autoflow, the ventilator takes the volume controlled mode and turns it into a kind of PRVC.

## VAPS

Volume Assured Pressure Support  
 Ventilation with guaranteed volume pressure support  
 Volume Support

This mode is a modification of PSV.

VAPS is a non-invasive ventilation modality that combines pressure support with volume-controlled ventilation. In this way, the ventilator guarantees a minimum tidal volume, even if the patient does not cooperate with breathing.

The patient can breathe spontaneously using a set pressure support.

It is a modality that can also be used invasively but is usually used as NIV.

In VAPS mode, the patient is allowed to generate a tidal volume greater than the set minimum tidal volume if he or she is able to do so. If he cannot generate the minimum tidal volume, the constant flow ensures delivery of the preset tidal volume.

In VAPS it can be useful to maintain a minimum minute volume, in patients with variable levels of respiratory stimulus and variable lung mechanics.

In this mode a target  $V_t$  is set, and the ventilator reaches it by changing the support pressure.

VAPS is a non-invasive ventilation modality that can be used with:

- Acute respiratory failure
- Chronic respiratory failure
- Obstructive sleep apnea
- Pneumonia
- Chest trauma

Requires careful supervision by a doctor or trained nurse.

## ASV

### Adaptive Support Ventilation

This ventilation mode works both with the patient being passive and in spontaneous ventilation. When passive, the ventilator operates under pressure control, regulating the pressure to obtain the desired  $V_t$ , then calculates the optimal RR to obtain a correct MV, usually 100ml/kg minute if set at 100% minute ventilation.

If the patient begins spontaneous breaths, the ventilator will automatically switch to pressure support mode, adjusting the pressure to obtain the desired volumes. In this way, however, the RR will be decided by the patient.

In this mode, we can wean the patient by reducing the set MV percentage: first 130%, then decreasing by 20% at a time, and then reducing the support pressure.

We can extubate the patient both in ASV and in the PS. In ASV, we lower the  $V_m$  percentage to the minimum, 25%, and reduce the PEEP to 5. After an hour with the patient under control, we can, if all went well, extubate the patient.

Hamilton ventilators use a proximal flow sensor to measure the patient's respiratory mechanics. It is a closed loop ventilatory mode, you set a target minute volume and the ventilator combines RR and  $V_t$  to achieve it based on the patient's ventilatory mechanics. This is measured via a flow sensor proximal to the patient, and measures the patient's expiratory constant, static compliance and respiratory resistance. First, we set the patient's gender and height, and the ventilator calculates the ideal weight. A setting of 100% indicates a normal value of 100ml/kg/minute. PEEP and  $FiO_2$  are set manually. We then set the maximum pressure limit, inspiratory and expiratory trigger and the pressure ramp. If the patient is passive, ventilate in pressure control mode to reach the target minute volume. If the patient is breathing, the pressure mode adjusted to reach the desired MV will remain active and in this case the RR will depend on the patient. We can do a trial for extubation simply by bringing the PEEP to 5 and reducing the minute volume target value.

Thus, ASV is a mechanical ventilation mode that automatically adjusts the pressure support according to the patient's needs.

ASV is a complex mechanical ventilation mode that can be useful in a variety of conditions, including:

- Acute respiratory failure
- Acute respiratory distress syndrome (ARDS)
- Pneumonia
- Congestive heart failure

ASV may be more effective than other mechanical ventilation modalities in reducing complications in these patients.

IntelliVent ASV is a further modification of ASV where the ventilator can adjust the  $\text{FiO}_2$ , PEEP according to oxygen and capnometer sensors.

#### Differences between ASV and PRVC

The correlation between ASV and PRVC is that both modalities use pressure to control the tidal volume delivered to the patient. However, ASV is more complex than PRVC and adjusts pressure support more accurately based on patient needs.

In ASV, the ventilator uses an algorithm to calculate the optimal support pressure for the patient. The algorithm takes into account respiratory rate, tidal volume, work of breathing and  $\text{FiO}_2$ . The support pressure is then automatically adjusted by the ventilator based on the results of the algorithm.

In PRVC, the support pressure is set manually by the operator. The ventilator then automatically adjusts pressure during inspiration to ensure that the target tidal volume is achieved.

In general, ASV is considered a more advanced mechanical ventilation modality than PRVC. It is more effective in reducing mortality and complications in patients with acute respiratory failure. ASV is a relatively new mechanical ventilation modality but has been the subject of numerous clinical studies. The results of these studies demonstrated that ASV may be effective in improving ventilation and lung perfusion, while reducing the risk of complications related to mechanical ventilation, such as acute respiratory distress syndrome (ARDS).

#### Differences between PRVC, ASV, APV and VAPS

PRVC, ASV, APV and VAPS are all dual mode mechanical ventilation modes. In a dual mode, the ventilator controls both pressure and tidal volume.

PRVC is a mode in which pressure support is set manually by the operator and the ventilator automatically adjusts the inspiratory time to ensure the target tidal volume is achieved.

ASV is a mode in which the support pressure is automatically adjusted by the ventilator according to the patient's needs. The ventilator takes into account respiratory rate, tidal volume, work of breathing and  $\text{FiO}_2$ .

APV is a mode in which pressure support is automatically adjusted by the ventilator based on the tidal volume delivered to the patient. The ventilator also takes into account the respiratory rate.

VAPS is a mode in which pressure support is automatically adjusted by the ventilator based on the respiratory rate and tidal volume delivered to the patient.

All these modes are useful in a variety of conditions, but require careful operator supervision, although some are close loop.

## PAV

Proportional Assist Ventilation

Proportional Assistance Ventilation

In PAV, a microprocessor was developed to detect patient activity and produce an appropriate ventilator response.

This improves patient-ventilator synchrony.

PAV is a form of continuous spontaneous ventilatory support in which the ventilator generates pressure proportionally to the patient's effort.

It is an advanced mode of mechanical ventilation used to provide respiratory support to patients in a more adaptable and personalized way than traditional modes.

In PAV, the ventilator measures and responds in real time to the force and speed with which the patient tries to inhale. This type of ventilation is designed to more naturally mimic the physiological breathing process, in which the patient can determine the onset, duration and intensity of inspiration. The ventilator responds proportionally to the patient's inspiratory effort, delivering an air flow that follows its rhythm and intensity.

In practice, PAV provides respiratory support that is directly proportional to the activity of the patient's respiratory muscles. This can help reduce the sensation of working against the ventilator and improve the patient's breathing comfort. Additionally, PAV can dynamically adapt to changes in the patient's respiratory force and lung compliance, contributing to more personalized and optimized support.

PAV has been shown to be effective in treating a variety of conditions, including:

Acute respiratory distress syndrome (ARDS)

Chronic respiratory failure (CKD)

Obstructive sleep apnea syndrome (OSAS)

Pneumonia

Heart disease

However, PAV also has some disadvantages. First, it is a more complex ventilation mode to set up and monitor than other modes. Secondly, it is not suitable for all patients, for example those with severe breathing impairment or heart problems.



The ventilator measures the patient's inspiratory effort via an inspiratory effort transducer, then calculates the inspiratory support pressure needed to assist the patient in reaching his or her tidal volume goal.

The supportive inspiratory pressure is added to the alveolar pressure generated by the patient's respiratory muscles.

PAV can be used invasively or non-invasively. PAV is a complex mechanical ventilation modality that requires specific training to use safely and effectively.

PAV is particularly useful in patients with restrictive lung diseases, such as idiopathic pulmonary fibrosis, and in patients with obstructive lung diseases, such as asthma and chronic obstructive pulmonary disease (COPD).

## IRV

Inverse Ratio Ventilation

Reverse Ratio Ventilation

Reverse ratio ventilation is a mechanical ventilation mode in which the exhalation phase is shorter than the inhalation phase. The ratio between the duration of the inhalation phase and the duration of the exhalation phase is typically 1:2 or 1:3.

Reverse ratio ventilation is used to improve ventilation of the pulmonary alveoli, especially in patients with restrictive lung disease.

Commonly used in pressure controlled mode.

This refers to mechanical ventilation where the I:E ratio is greater than 1.

By increasing this ratio to more than one, the inspiratory time is prolonged and consequently the exhalation time is shortened.

This improves the distribution of gases in the lung and increases the average airway pressure for improved oxygenation.

As the ventilatory pattern is changed, sedation and paralysis using a muscle relaxant is usually required to maintain synchrony between patient and ventilator.

The basic requirements for reverse ratio ventilation are the use of a critical breathing rate to promote the formation of auto-PEEP and sufficient inspiratory pressure to overcome the lung opening pressure. Both are present with pressure-controlled ventilation (PC).

Directions

In surfactant-deficient diseases, such as acute respiratory distress syndrome (ARDS), in which the abnormality is widespread and nonuniform throughout the lung.

This is one of the methods to provide full ventilation support to patients with non-elastic lung who have high airway pressure and poor oxygenation when standard modes are used.

This system recruits the alveoli to "slowly open", which is not possible with a normal I:E ratio.

In ARDS, alveoli with relatively more severe disease have longer time constants and take longer to fill. In normal ventilation, there is not enough time to fill these alveoli, which therefore remain in a collapsed state, causing persistent pulmonary shunting and hypoxemia.

IRV will provide adequate time to fill diseased alveoli and improve overall gas distribution in the lung.

By reducing the expiratory time, we achieve the formation of an auto-PEEP, since the tidal volume cannot be completely exhaled before the next inspiration begins

Auto-PEEP prevents slow compartments from collapsing, increasing the final tidal volume. This improves oxygenation, reducing the shunt, i.e. unventilated alveoli.

In this modality, we also use normal PEEP, which has the task of keeping the alveoli open which are "quick" to close.

Typically, an I:E ratio of 2:1 is used, which is more than sufficient to improve oxygenation.

As soon as the positive effects begin to appear and oxygenation improves, you can think about changing the I:E ratio.

## MMV

Minute Mandatory Ventilation

Mandatory minute volume ventilation

It is a historic ventilatory modality that allows us to manage a large number of patients with different needs well.

It is a ventilatory mode that allows the patient to breathe until a **minimum** minute volume is reached. If he fails to achieve it, the ventilator intervenes with forced or assisted actions. It will do this by helping him with the set support pressure. If the patient goes into apnea and is unable to guarantee that minimum volume, the ventilator intervenes. It is therefore a particularly safe ventilation mode.

If the patient is breathing and achieving the minimum minute volume, the ventilator will cause the patient to breathe spontaneously via pressure support. Easy explanation: if the patient is able to ventilate 4 liters per minute, but we have set the minimum minute volume to be 6 liters, the ventilator will intervene to reach this quota.

The minimum minute ventilation setting is given by the frequency multiplied by the Vt.

We put a patient on MMV ventilation who we are not sure is capable of achieving effective ventilation on his own.

According to Chatburn, it is closed-circuit adaptive ventilation that ensures minimal minute volume. It is a modality that behaves in three different ways depending on whether the patient intervenes or not. Thus, if the patient does not have spontaneous breaths, it behaves like a volume-controlled ventilation. If the patient takes spontaneous breaths but is not able to reach the minimum minute volume quota, the mode works like a SIMV. If the patient alone is able to reach the set minute volume, it will behave as a PSV.

We have a problem when a patient hyperventilates in this case, he could easily go into respiratory alkalosis.

It is a modality that can improve the patient's lung function, helps reduce VAP and is also useful as a weaning modality.

## NAVA

Neurally Adjusted Ventilatory Assist

Neurally regulated ventilatory assistance

NAVA mode is a mechanical ventilation mode in which the tidal volume delivered by the ventilator is controlled based on the patient's breathing efforts.

In this mode, the ventilator detects respiratory efforts via a nasogastric tube equipped with sensor electrodes. These electrodes, introduced with a transesophageal probe, capture the signal of the phrenic nerve (electrical activity of the diaphragm (Edi) which initiates spontaneous inspiration. This system allows perfect synchronization with the patient, synchronization that is the most effective possible.

This signal starts from the patient's brain, arrives via the phrenic nerve and causes the diaphragm to contract. Being able to capture this moment guarantees perfect synchrony.

NAVA mode was developed to improve synchrony between the patient and the ventilator.

In particular, NAVA mode can help reduce respiratory fatigue and improve patient comfort.

Greater synchrony means a better level of ventilation, less time on mechanical ventilation and better ABG.

However, NAVA mode can be more complex to set up and monitor than other mechanical ventilation modes.

If the ventilator is unable to have an adequate electrical signal from the sensor catheter, the backup mode intervenes and will ventilate the patient in pressure support mode; in this case the activation will be given to a flow or pressure trigger.

Naturally, patients with nerve lesions, apnea or disorders that compromise normal phrenic nerve activity cannot be ventilated in NAVA mode.

## Part Six: Two more concepts!

### Check that blood gas!

#### Arterial oxygen content CaO<sub>2</sub>

When we look at a blood gas we think that everything is ok if we have good pH, PCO<sub>2</sub>, PO<sub>2</sub>, HCO<sub>3</sub> values. We need to check this leaflet better.

One of the parameters we care less about is hemoglobin.

.... and what do you care about hemoglobin, you have to watch how that patient breathes, not if you have to transfuse that patient!!!... Ehh not really!

Hemoglobin, on the other hand, plays a very important role in patient ventilation.

A correct hemoglobin value allows adequate exchanges and has a primary role in tissue oxygenation.

Regularly looking at hemoglobin helps us understand whether the patient has an adequate perfusion index.

From here you must evaluate the CaO<sub>2</sub>, i.e. the arterial oxygen content, and therefore the total quantity of oxygen present in the blood, both bound to hemoglobin and dissolved in the plasma. This is the formula to calculate CaO<sub>2</sub>:

$$\text{CaO}_2 = (1.34 \times \text{Hb} \times \text{SaO}_2) + (0.003 \times \text{PaO}_2)$$

Where:

**1.34** is the amount of oxygen that can be bound to each gram of hemoglobin.

**Hb** is the concentration of hemoglobin in arterial blood, expressed in grams per deciliter (g/dL.)

**SaO<sub>2</sub>** is the saturation of arterial hemoglobin, expressed as a decimal fraction or percentage.

**PaO<sub>2</sub>** is the partial pressure of oxygen in arterial blood, expressed in millimeters of mercury (mmHg).

The first term of the formula (1.34 x Hb x SaO<sub>2</sub>) represents the amount of oxygen bound to hemoglobin in arterial blood.

The second term (0.003 x PaO<sub>2</sub>) represents the amount of oxygen dissolved in the blood plasma.

The sum of these two terms gives the total CaO<sub>2</sub>.

The normal value of CaO<sub>2</sub> is between 16 and 22. The first term represents in an "important" way the quantity of oxygen bound to hemoglobin, which will represent the quantity of oxygen available for tissue oxygenation. The second term, however, has a marginal value.

We see it clearly from these numbers:

$$1.34 \times \text{Hb} \times \text{SaO}_2 = 15 \times 1.34 \times 0.98 = 19.7$$

$$0.003 \times \text{PaO}_2 = 90 \times 0.003 = 0.27$$

The amount of oxygen in the first term is 19.7, in the second only 0.27!

A huge difference, the first term contributes to oxygenation in **73** times higher quantities in this example.

When the CaO<sub>2</sub> value is low the body will activate an anaerobic metabolism with the production of lactic acid and metabolic acidosis; still showing good oxygenation. That metabolic acidosis is the result of tissue hypoxia.

At the lung level everything is fine, but we need to improve the quantity of hemoglobin to increase tissue oxygenation. We don't have to give this patient bicarbonate because he is low because all the bicarbonate is all bound up with lactic acid.

In patients with fibrosis or emphysema who have chronic hypoxemia there is an increase in Hb to compensate for the hypoxemia and therefore you could see a  $PO_2$  of even 60. So they will have chronic hypoxemia but not chronic hypoxia, this is because they compensate with high hemoglobin!

$CaO_2 = 18 \times 1.34 \times .87 + 57 \times 0.003 = 20.9 + 0.17 = 21.07$  So a Normal  $CaO_2$ ! Interesting right?

In these patients you don't have to increase the oxygen, they don't need it!

Remember not all patients who are hypoxemic are hypoxic.

## Hyperoxia

By **hyperoxia** we mean excessive tissue exposure to oxygen. This can cause problems with the alveolar endothelial cells, and with the alveolar capillaries, leading to problems such as in ARDS, with increased capillary permeability and surfactant problems due to damage to the surfactant of type 2 pneumocytes.

All this can lead to a pathological lung condition or ARDS.

These damages are directly related to the amount of partial pressure of oxygen administered and the time of exposure. Hyperoxia leads to developing signs of oxygen toxicity, this compromises the balance between oxidants and antioxidants within our body, creating damage to tissues and cells. We observe the increase in free radicals, which are nothing more than chemical species with one or more unpaired electrons, which have the possibility of interacting with compounds present in the proteins of the lipid membranes and acid nuclei, generating serious damage. Unfortunately, alveolar endothelial cells and alveolar capillaries are very susceptible to this insult. Oxygen toxicity can cause damage to many parts of the body. For example, a high concentration of oxygen with increased atmospheric pressure, as in hyperbaric therapy, can cause oxygen toxicity problems in the central nervous system.

We can divide this phenomenon into phases:

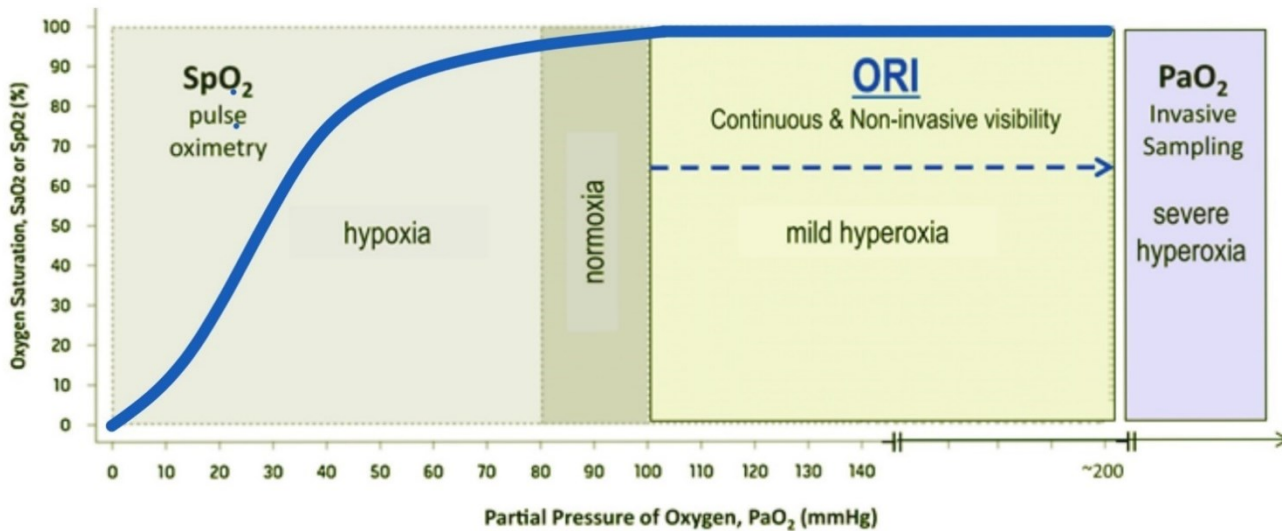
- In the first phase we have a depletion of antioxidants.
- In the second phase, inflammation of the lung tissue begins.
- In the third phase there is proliferation of monocytes, leukocytes and inflammatory markers with cellular hypertrophy.
- In the fourth phase we reach fibrosis, therefore a point in which we cannot have a return to normality of the lung tissue; the injury has become permanent.

The result of this inflammation leads to impaired gas exchange and pulmonary edema. The inflammatory response attracts macrophages and monocytes which will worsen the overall picture. We can also have oxygen absorption atelectasis, caused by high percentages of oxygen administered. All this can cause a pulmonary shunt, this picture often does not respond even to high percentages of oxygen delivered.

But at what level can oxygen be considered toxic?

With administration of more than 60% oxygen for more than 24 hours at normal atmospheric pressure, oxygen may become toxic.

Even in just 12 hours, pulmonary edema, atelectasis and pulmonary congestion can occur. Naturally, we are talking about a risk related to the total dose of exposure to oxygen. For total dose we consider the three factors together, namely: percentage of oxygen, atmospheric pressure and time of exposure to this percentage. Increasing the percentage of oxygen to 80% or prolonging exposure with 60% oxygen or administering it at pressures greater than one atmosphere increase the risk of toxicity.



As you can see in the image, after 100 PaO<sub>2</sub> the straight line begins and the problem of hyperoxia begins.

At 80 PaO<sub>2</sub> the saturation is approximately 96%, we consider this a normal value. However, when you have saturations of 97%, 98%, 99% and 100%, you have no way of knowing what PaO<sub>2</sub> that patient has, you could have really high values!

Hyperoxia is associated with increased lung damage and increased mortality. Reducing saturation levels allows the patient to be extubated sooner and reduces the risks associated with hyperoxia. A 2018 work shows that 70% of the time in mechanical ventilation patients have between 96% and 100% saturation with an average PaO<sub>2</sub> of 144. Half of the time in intensive care patients have more than 98% saturation and 50% of them have a hyperoxic state. These data should make us reflect.

An unwritten "rule" sees in a patient with 100% saturation an almost certain condition of hyperoxia, certainly to be investigated with arterial gas analysis (ABG).

This does not mean that in patients who need oxygen we should not give it to them.

It seemed interesting to me to point out the presence of this often little-considered problem.

## Ventilation/Perfusion Ratio

The ventilation/perfusion ratio or V/Q ratio or V/Q mismatch is the relationship that exists between gas ventilation and the relative blood perfusion, referring to the same alveolar area.

We can define ventilation as the amount of air that enters and exits the alveoli to obtain oxygen and eliminate carbon dioxide.

Perfusion is the amount of blood that circulates in the capillaries of those alveoli.

In order for oxygen to reach the rest of the body there must be adequate alveolar ventilation supported by adequate alveolar perfusion.

In this collaborative relationship we indicate the ventilation rate with V and the perfusion rate with Q. We call this ratio V/Q

We can define this relationship as the ratio between the amount of air that reaches the alveoli in one minute and the amount of blood that perfuses the alveoli in one minute.

### V = Ventilation and Q = Quantity of blood

In an ideal condition, the V/Q should be equal to 1, indicating that ventilation and perfusion are equal and perfectly balanced.

The term mismatch or misalignment indicates that this correspondence between V and Q does not exist. Normally, the V/Q ratio of a normal person is 0.8.

This is due to two main factors: gravity and air.

Thus, in orthostatism, the upper part of the lungs will be more ventilated and the lower part, due to gravity, will be more perfused.

This leads to a condition in which the upper part of the lungs cannot acquire all the available oxygen because perfusion is lower ( $Q < V$ ). The lower part of the lungs does not have enough oxygen for perfusion present ( $Q > V$ ).

There you have it, the simple explanation of why the V/Q ratio is mismatched.

The normal value of alveolar ventilation is 4 L/minute.

The normal alveolar perfusion value is 5 L/minute.

So we have a V/Q of  $4/5 = 0.8$

We can have two altered V/Q conditions:

$V > Q$  ( $V/Q > 0.8$ ), in this case ventilation exceeds perfusion, for example due to a capillary problem, due to a clot, embolism or pulmonary emphysema

$Q > V$  ( $V/Q < 0.8$ ), in this other case the perfusion exceeds the alveolar ventilation. Airway obstruction, asthma, COPD, and pulmonary edema lead to a ratio less than 0.8.

The difference in the size of the alveoli is due to the intrapleural pressure: in the upright position those in the other will be more expanded.

The increased perfusion from gravity.

Therefore, a standing person will have more relaxed and ventilated lung apices, with a higher V/Q ratio than normal.

The lung bases, due to gravity, will have greater perfusion and, again due to gravity, they will have the alveoli compressed and less ventilated by the greater quantity of blood present at the bottom.

Now, quickly, let's see what happens at the extremes of this relationship, that is, if one of the two components of this relationship were to be completely missing.

If we have ventilation but no perfusion, we define this as a condition **Dead space**.

If we have perfusion but no ventilation, we define this as a condition **Shunt**.

When the patient breathes in the supine position we have a crushing by the heart and the abdominal organs through the diaphragm of the lower lung districts. This, combined with the action of the force of gravity, makes the lower parts more perfused but less ventilated precisely because the alveoli are compressed. By placing the patient in a prone position, we balance this condition by placing the lower and upper parts of the chest in a very similar condition.

Now, in pronostatism, the upper alveolar part located near the spine will be well perfused and well ventilated; the same thing happens to the lower part because in this position the weight of the abdominal organs and the heart is not so "disturbing" for ventilation.

In fact, in the supine position the parts positioned upwards (ventral) of the lung have a higher transpulmonary pressure, this favors the expansion of the alveoli of this district. On the other hand, the lower (dorsal) areas will have a lower transpulmonary pressure, this makes the alveoli in that area less ventilated.

This condition in a patient with ARDS, due to edema, sees this difference become even more distinct. This results in hyperinflation of the ventral alveoli with atelectasis and therefore absence of gas exchange in the dorsal ones. In this condition, the patient's pronation causes the alveoli that are too open to compress because they are under a greater influence of gravity and the alveoli that were atelectatic slowly open up. In this way, rebalancing the V/Q ratio and giving a clear improvement to the patient's ventilation. Furthermore, during pronation the amount of dorsal perfusion remains unchanged precisely because it is a characteristic of that area. In fact, the dorsal areas of the lung are indifferent to changes in position and maintain excellent perfusion without being influenced by the force of gravity, but this does not happen for ventilation.

## Autoflow

I would like to tell you briefly about Autoflow and what its function is.

Autoflow is a setting found on Drager™ ventilator and can be applied to volume-controlled ventilator modes. In autoflow mode, a decreasing flow is delivered to obtain a set Vt, avoiding pressure peaks. The final objective is to obtain that Vt with the minimum necessary pressure, adapting to the patient's resistance and compliance values and at the same time allowing spontaneous ventilation.

To obtain these results, three breaths are delivered in this way:

- 1 A breath with a plateau is delivered.
- 2 The second act is released at 80% of the plateau pressure of the first. The tidal volume delivered during this act is measured.
- 3 The third act will vary the pressure by +/- 3 cm H<sub>2</sub>O to evaluate the Vt.

The pressure variation is gradual and therefore has a "soft" approach to patient ventilation. The continuous measurement, referring to the previous Vt, allows you to adjust the pressure to keep that Vt as stable as possible.

With this setting, volume-controlled ventilation modes acquire significant advantages such as: A constant pressure during inspiration which promotes alveolar recruitment, reduces pressure peaks, improved gas exchange. All this results in less stress for the patient and improved ventilation.

So, when you find a patient who ventilates in VC-AC, and therefore in controlled volume, but has a square-type pressure wave, autoflow has probably been inserted.

In VCV mode, autoflow operates similarly to a PRVC (pressure regulated volume controlled) mode. In a PRVC mode, pressure is automatically determined by the ventilator based on the target tidal volume set by the operator.

## etCO<sub>2</sub>

The acronym etCO<sub>2</sub> indicates the measurement of carbon dioxide present in the air breathed by the patient, exactly at the end of exhalation. In fact, that "et" stands for End Tidal.

We can measure it using a capnograph connected in different ways, but always in the expiratory path of the respiratory circuit. The devices also differ in shape and type of connection. The etCO<sub>2</sub>



measurement is a continuous measurement and gives us information on the patient's metabolism and above all on his ventilation. We already know that in the inhaled air we have 21% oxygen and 0.04% carbon dioxide; once the internal metabolism is finished we exhale the air we have introduced with different percentages of these gases.

Thus, the first exhaled air will have a percentage of 15% in its "oxygen" component and 5.6% in the "carbon dioxide" component; remember that the nitrogen remains unchanged at approximately 78%.

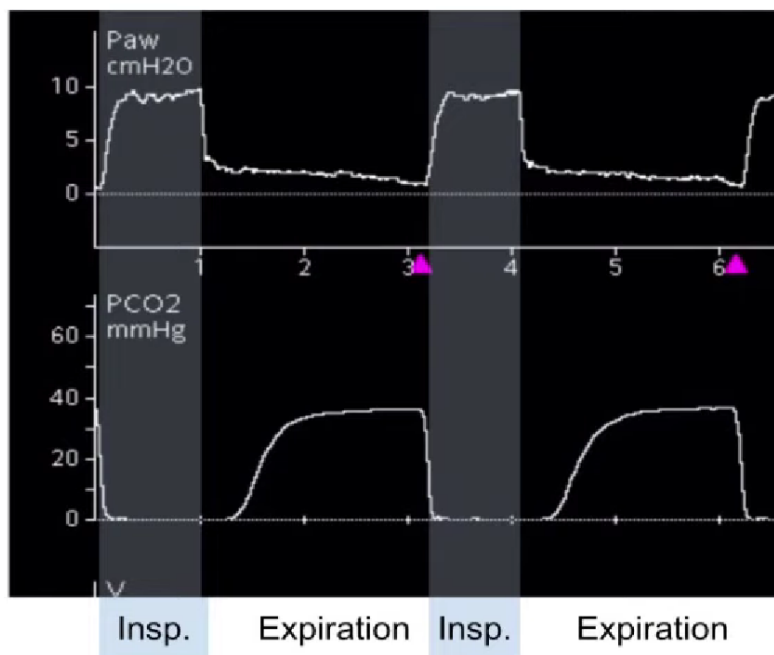
The capnograph works by emitting an infrared light capable of measuring the amount of carbon dioxide in the gas.

You will hear about two types of capnographs: mainstream and sidestream.

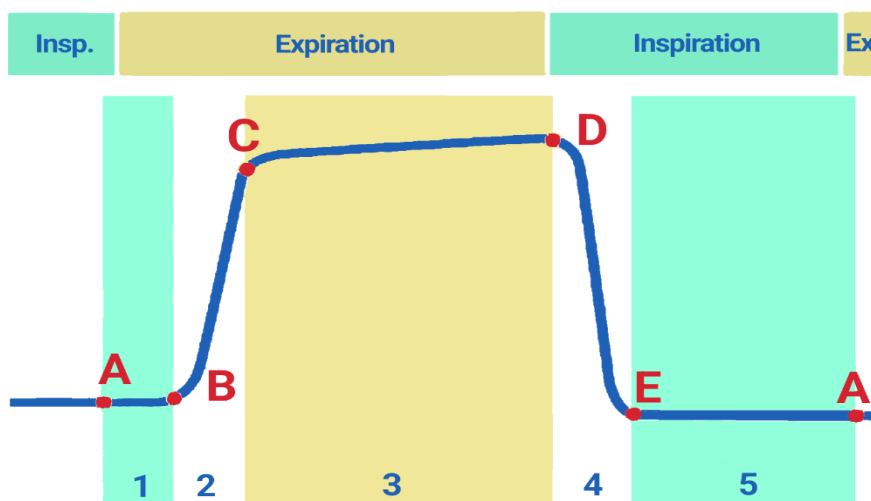
The only difference between the two concerns the positioning of the meter relative to the breathing gas line. Thus, a mainstream capnograph is placed exactly where it needs to take the measurement; a sidestream capnograph is located far from the measurement connection and is usually connected to it via a connection tube. The measurement of mainstream systems is accurate and fast, in sidestream systems it is accurate but slightly delayed.

Capnography can be used in intubated patients, during surgical procedures and therefore under anesthesia, during emergency transport, post-extubation and whenever monitoring is desired.

This in the image is a capnogram:



A normal capnogram presents 5 points like this:



In the AB section the CO<sub>2</sub> is very low, we are at the beginning of exhalation.

In the BC section we have a rapid increase in the CO<sub>2</sub> value because the dead space gases are replaced by alveolar air.

The CD tract represents all the gas coming from the alveoli up to the plateau.

The DE segment represents the inspiratory air which rapidly causes the CO<sub>2</sub> value to drop.

The EA segment represents the inspiration where, if you have been careful, we have very low CO<sub>2</sub> values (0.04%).

We measure etCO<sub>2</sub> precisely at point D of the capnogram, that is, the point where we have the greatest quantity of carbon dioxide in the exhaled gases.

The normal value of etCO<sub>2</sub> is 35-45 mmHg.

Instead of monitoring a single respiratory act and therefore an instantaneous condition, it is useful to evaluate the trend of the etCO<sub>2</sub> values. In this way we could better evaluate the patient's ventilatory progress.

Continuous etCO<sub>2</sub> monitoring can help in case of CPR to evaluate the quality of chest compressions, to evaluate the correct position of the endotracheal tube, correct intubation and therefore, in the diagnosis of ROSC. It can also help evaluate any changes in the ventilatory system, such as possible pulmonary embolisms.

There are conditions that can make the measurement less precise, such as a high ventilatory frequency, presence of condensation or secretions, administration of bicarbonate or low CO (Cardiac Output).

What we should really be interested in is the correlation between PaCO<sub>2</sub> and etCO<sub>2</sub>.

The reasoning is this: if I have an etCO<sub>2</sub> of 40 and a PaCO<sub>2</sub> of 45, I know that in that patient I have a difference of 5 mmHg between one value and the other. I can consider that this difference will remain similar, so when there is a decrease in the patient's etCO<sub>2</sub> I will be able, with good approximation, to also evaluate the PaCO<sub>2</sub> value. This has the advantage of reducing the number of ABG performed on the patient. Naturally, confirmation of this can only occur by continuing to perform the control ABG, even if over a longer period of time.

Therefore, it is more important to use the gradient between the two pressures.

As said before, it is not the specific value that indicates something, but its variation over time that interests us. Why is arterial CO<sub>2</sub> so closely related to expired CO<sub>2</sub>? Because it will be the one that is removed through external respiration!

If PaCO<sub>2</sub> rises, etCO<sub>2</sub> also rises and vice versa, they do so proportionally, keeping that difference or gradient always identical.

If, over time, we see an increase in PaCO<sub>2</sub> and a decrease in etCO<sub>2</sub>, therefore with an increased gradient, we are probably faced with a patient with pulmonary embolism. Even if CO (Cardiac Output) drops significantly, we could have a similar picture, the amount of arterial CO<sub>2</sub> is not eliminated due to a decrease in alveolar perfusion.

Having PaCO<sub>2</sub> and etCO<sub>2</sub> available, we can also calculate the dead space present.

To do this, you perform this calculation  $\text{PaCO}_2 - \text{etCO}_2 / \text{PaCO}_2$

So, if we have 40 – 35/40, we get 5/40 therefore 0.125; we just discovered that 12.5% of your Vt does not participate in gas exchange. If you think about it, as well as interesting, it is also very useful. If you have 40 and 15 you will have 62.5% dead space! Cabbage

With a tidal of 450, they don't ventilate 281 ml of gas! Interesting right?

An increase in CO<sub>2</sub> may be due to hypoventilation or fever which increases metabolism and therefore the formation of CO<sub>2</sub>.

It can drop in case of hyperventilation, reduction of CO, or due to a reduction in metabolism or pulmonary embolism.

## Volumetric capnometry

The tidal volume is plotted against the etCO<sub>2</sub> in a curve, Gives more information about the value of the anatomical dead space, alveolar tidal volume and VCO<sub>2</sub> (amount of carbon dioxide produced per minute)

## Everything you wanted to know about aspiration

If you are an intensive care nurse or are about to become one, you can be sure of one thing: you will become an expert in suctioning. The aspiration of secretions is one of those tasks that falls to us as operators, and it is one of those procedures that we perform for the patient as he is not able to eliminate the secretions on his own.

Normally, in fact, we eliminate the secretions that our respiratory system produces without realizing it. A patient who is intubated or with a tracheostomy tube is unable to do this and the secretions accumulate, causing many problems.

They are the main cause of obstruction of the airways, of increased resistance of the ventilatory system, they can cause real blockages and can cause pneumonia if not **adequately removed**.

But what is meant by adequately removed?

It's one of those catchphrases that "those who know and don't do" use, those who have read a procedure and who explain it to others perhaps without ever having set foot in an intensive care unit.

For those who work in the field, "properly removed" means only one thing: you must aspirate the patient when necessary. You don't have to do it routinely, you don't have to do it on schedule or with patterns, but you have to know when it's necessary and, above all, you have to do it well. As soon as you take care of your patient, immediately check the height of the tube, the tightness of

the cuff and assess whether your patient needs to be aspirated. Do this by looking at what the ventilator tells you, listen to the noises coming from the tube, listen to the patient's chest, evaluate the saturation. In short, check that everything is in order, if not, remove those secretions. When doing this, be careful not to take too long. 15 seconds is the recommended time, if it is 10 even better; remember not to go into suction and not to "stay" inside the tube all the time during suction!

This causes lesions to the mucosa and that annoying bleeding in the secretions that you will probably recognize as the "signature" of some of your less-than-attentive colleagues! I don't have to tell you how to aspirate a patient, but the rule says to insert the tube gently until you find resistance, at this point stop! You're not looking for oil, that's enough! Go back about a centimeter and, from there, come out on suction. During this phase, it is better if the suction is not continuous, but performed alternately. You can also rotate the tube, which seems more effective to some. Exit the tube and as soon as possible, or rather **immediately**, reconnect the patient to the ventilator.

Now it may seem trivial, but if you have not removed all the secretions, it is preferable to repeat the procedure rather than keep the patient in an uncomfortable condition for a long time. In fact, when you are suctioning a patient, you are removing not only the secretions but also the air from his lungs, a not very nice condition for him. Of course, remember to carry out the procedure in a sterile manner, and if the tube becomes contaminated, do not aspirate the patient; reconnect it and prepare everything with a sterile glove and new tube. Don't worry, it's happened to everyone more times than you can imagine.

Let's dedicate a few lines to the way in which the patient is aspirated, or rather, how you prepare him for suction using the ventilator.

You have two possibilities, but only one is correct!

First possibility: increase the percentage of oxygen.

Second possibility: use the intended method for aspiration.

**The first choice:** Almost all ventilators increase the percentage of oxygen for a time preset by the machine (usually 2 minutes). This gives the patient a certain degree of autonomy before desaturation.

Patients desaturate during aspiration for two reasons:

The first is due to the severity of the respiratory failure, in this case you have to be clean, good and fast.

The second reason is because, while you are inhaling it, you are replying to a text message from a friend of yours! Hurry up!!!

**The second choice** it's the one that says to the ventilator:

"Hey dear, look, I have to aspirate the patient. Could you send me more oxygen, so I don't "kill" him? Well, while you're at it, since I informed you, when I remove the patient, could you avoid sending all the patient's secretions onto me as happened when I used the wrong method called **first choice**?"

The ventilator, in this case, pre-oxygenates the patient and stops ventilation as soon as we disconnect it. Then, he resumes ventilation as soon as we connect him to the patient again and give him a post-oxygenation.

The two methods are similar, but the first helps the patient from the desaturation side, the second also protects the operator from contamination.

We could discuss aspiration for pages, but I'll just bore you with a couple of concepts and/or suggestions.

Pre-oxygenation is not always essential, but if you press the button and aspirate the patient after 5 seconds, pre-oxygenation does not serve its purpose. So even when you get help from someone who happens to be passing by and you are ready with the feeding tube, you don't have to imitate the Ferrari pit stop! Wait for the pre-oxygenation time, the patient thanks you!

When suctioning a patient, you should first disconnect the hose from the filter and then the catheter mount from the tube. When reconnecting the patient, carry out the procedure in reverse: first connect the catheter mount to the tube and then the filter to the corrugated tube.

By doing this, the last blow of air from the ventilator will be "sterile" air and you are happy; in practice you limit the risk of polluting yourself. However, refer to the company procedures.

If you also work with a fellow **bodybuilder** and have prepared everything for suction, but that catheter mount doesn't want to come off the tube, open the cap on the front and insert the tube from there. You will later have the opportunity to detach the catheter mount from the tube and say hello to your colleague, as long as he is not much bigger than you!

Now a little word about closed circuit suction, as much loved and useful as it is hated and little accepted in many situations.

This system has advantages and disadvantages like almost everything else.

On the one hand it is a very practical system, it protects the operator by making the suction maneuver much "cleaner" and easier to perform. Over the years, the systems have been improved and, albeit with some defects, the "covid" period that has just passed has made them come back into vogue. You do not need to disconnect the patient from ventilation, but suctioning is performed simultaneously with ventilation. , in some cases, makes the maneuver more difficult and less effective. The tube can be kept for 24-48-72 hours if not visibly dirty, provided it is washed every time it is repositioned in its protective sheath.

Warning: it must **not remain in place for 7 days**. There are 7 stickers in the kit, one for each day of the week, so you can indicate the day you placed it!

I've heard a few times that it's because it can stay in situ for 7 days, forget it!

In some cases, incorrect maneuvers are committed.

Thus, a patient could involuntarily hook up the system left "around" and become extubated or disconnected from the ventilator (this is an iatrogenic damage).

Some leave the suction always connected to the system and this can, due to gravity, cause accidental extubation (this is an iatrogenic damage)

The sterile liquid supplied is often mistaken for bronchial lavage liquid, but instead has the sole purpose of being used to wash the tube after use.

Some facilities have "arbitrarily" decided that keeping the tube for several days, considering it "not a big problem", is instead a big mistake!

Some "enlightened" people consider the performance of culture tests from the closed circuit to be correct even if it has been present for days. **Manufacturing companies do not recommend this procedure.**

Having said this, correct management of the closed circuit, costs aside, would seem to be the gold standard but this is not the case. According to several studies, the level of VAP and mortality is unchanged with one method or the other. Of course, in terms of operator protection it has no rivals. To date there is no study that puts an end to this eternal dilemma. And, searching online, I was able to find studies that were subsequently invalidated due to "the interests of the manufacturing companies"!

For my part, I can only tell you not to look for a winner in every way, **both systems have pros and cons, and both should be used in different cases.**

However, I invite you to inform yourself and practice with one system and the other.

I also invite you to follow the instructions given by your operations center and the manufacturer.

## ECMO, just a nod

ECMO, an acronym for Extracorporeal Membrane Oxygenation, is an extracorporeal circulation technique that is used to provide oxygenation and cardiac support to patients with severe acute respiratory or cardiac failure.

We are interested in its functionality regarding the patient's oxygenation.

ECMO, in fact, works by taking blood from the patient, oxygenating and heating it and then introducing it back into the bloodstream. This process is performed through an extracorporeal circuit, which is composed of a pump, an oxygenator, a heater and a filter.

The oxygenation that ECMO performs is through an oxygenator, which is a device that replaces the function of the lungs. The oxygenator uses a semipermeable membrane to separate blood from air. Air, rich in oxygen, is then passed through the membrane, which allows oxygen to pass into the blood.

The oxygenated blood is then heated and reintroduced into the bloodstream. This process helps provide the patient with the oxygen she needs to survive. Contrary to popular belief, ECMO support can last from a few hours to a few days to weeks, it all depends on the patient's condition; to date the record of a patient on ECMO is approximately 180 days! Of course, this method has nothing to do with positive pressure ventilation but, since I wrote the text, I felt like including it!

## The weaning process

When you have a patient on a ventilator, the first thing you want to do is release them from sedation and the second thing is start a weaning trial period. In fact, every day less mechanical ventilation reduces the risk of pneumonia and other related problems.

There are several ways to carry out adequate weaning, differing in duration and time.

Each facility adapts its own way of carrying out the procedure according to the type of patient and experience gained. We must not consider one method better than the other or wrong, again every patient and every condition is different. In this context, the clinician knows how to extricate himself and adapt to the patient's conditions to achieve extubation in the best possible way.

If a patient has an  $\text{FiO}_2$  above 45% then there is no point in attempting weaning.

We need good blood gases, the ability to ventilate on your own without support, a good mental state, that there are reduced secretions and that you are able to remove them actively.

If all these conditions are present, we can do a spontaneous breathing test, in English it is called **SBT** or Spontaneous Breathing Trial.

This is a test to see if the patient can be weaned and therefore extubated and should be performed in this way:

If he is ventilating on A/C or SIMV, I take him to a ventilation that provides him with PEEP, pressure support, or tube equalization.

Tube clearing reduces the resistance a patient encounters in breathing into the endotracheal tube unaided.

After some time, usually 30-120 minutes, do a follow-up blood gas, and if the patient seems to be breathing well and is not fatigued or hyperventilating, the blood gas will usually be good.

If the blood gas is good, extubation can be performed.

During the trial we monitor an index called rapid shallow breathing (RSBI).

The **Rapid Shallow Breathing Index**, known as the Tobin index. This index is calculated by dividing the respiratory rate (RR) by the tidal volume (Vt). So, if we have a respiratory rate of 20 breaths/min and a tidal volume of 0.5 liters, we get an RSBI of 40.

Now if the RR increases or the Vt is low the RSBI will increase, and this is not a good sign.

Values below 100 are acceptable, while values above 105 are not a good sign for extubation. Even ventilation of less than 15 liters per minute is a good sign, in fact it tells us that the patient has no problems at a metabolic level.

Since we are conducting the SBT with a spontaneously breathing patient and we have learned that the volumes are different for each individual breath, to correctly calculate the RSBI value, I have to take the MV and divide it by the respiratory rate. The value obtained will give the average Vt.

If we want to proceed with the extubation of the patient, we must seriously evaluate these 4 variables:

Good ventilatory mechanics verified with the trial.

A good mental state that allows you to protect the airways.

Not excessive secretions and the perception that the patient can manage them.

The loss of air when the tube cuff deflates (cuff leak).

In particular, the last variable is generally little practiced and will tell us if the patient has a narrowing or edema of the airways, which will not allow good breathing.

The test is performed by suctioning the upper part of the tube cuff and then deflating it. Not hearing any noise or air leaks indicates that we are in the presence of edema or shrinkage. In this case, cortisone treatment would be necessary and extubation should be delayed. This is the empirical method, if we want to be more "scientific" we do it this way: once the cap has been capsized, the ventilator is set to give 500ml of Vt. If the losses exceed 15% of the Vt, therefore on the ventilator we have less than 425 ml of exhaled volume, then it means that the airways are not edematous and is another sign that the patient is executable.

Remember that, on average, about 15% of extubations may fail.

To start an SBT you need a good P/F, a low FiO<sub>2</sub> value, a low PEEP, good blood gases, a good chest x-ray, stable hemodynamics and a patient capable of taking spontaneous breaths. The goal is to move from a controlled or assisted mode to a spontaneous mode.

It can be done very well in CPAP with a PEEP of 5 and a PS of 5.

Once left in these conditions for at least 30 minutes and a blood gas has been assessed, extubation can be performed. If the patient has hemodynamic instability or respiratory difficulty, then it is appropriate to interrupt the SBT.

Even an increase in respiratory rate or an agitated patient should cause the SBT to be suspended.

We have seen here that a spontaneous breathing test is carried out in order to proceed with extubation, knowing that the patient is able to breathe on his own. To do this, we also calculate an RSBI or Tobin index which will help us in the choice. Furthermore, so that there are no further problems, we can also evaluate the air leaking or leak test of the headset, as seen above.

There are also other ways to properly assess whether a patient is a candidate for successful extubation. One of these is the so-called airway occlusion test known as P0.1

The P0.1 test is performed with the trial patient and is a measurement permitted by some ventilators. In this test, while the patient begins to inhale, the ventilator occludes the circuit for 0.1

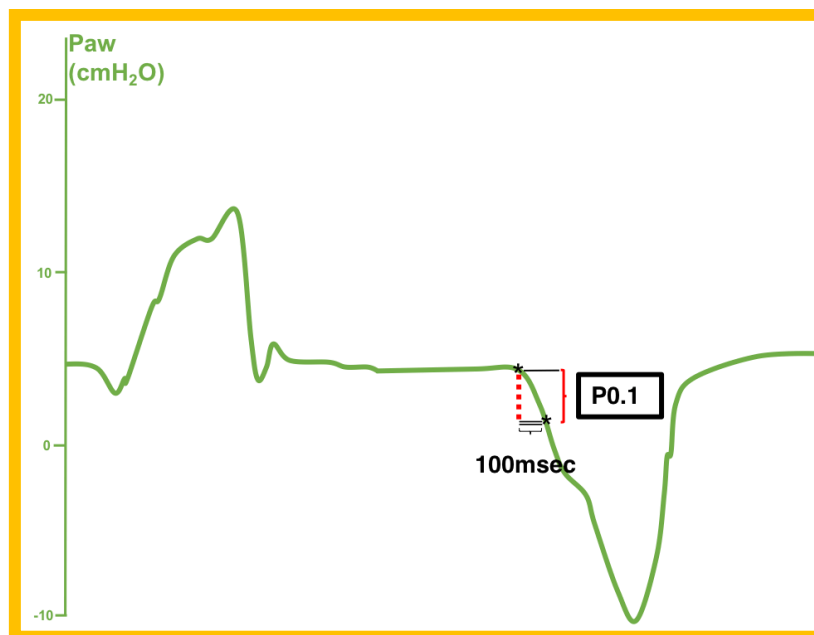
seconds (P0.1). 0.1 seconds or, if you prefer, 100 milliseconds, is such a short time that it is not perceived by the patient, who cannot even alter the result.

In this time the negative pressure that the patient is able to apply is measured, based on the test result and its numerical values, we can consider extubation possible.

The negative pressure exerted is related to the patient's WOB (work of breathing).

These are the test values and what they tell us:

<b>P0.1</b>		
<b>&gt;3.5 cmH<sub>2</sub>O</b>	<b>1.5-3.5 cmH<sub>2</sub>O</b>	<b>&lt;1.5cmH<sub>2</sub>O</b>
High respiratory drive Insufficient level of support <b>Under-assistance</b> High rate of weaning failure	Appropriate sedation Appropriate level of support  High rate of weaning success	Low respiratory drive High level of support <b>Over-assistance</b> High rate of weaning failure
High respiratory drive Insufficient level of support <b>Low assistance</b> High weaning failure rate	Appropriate sedation Adequate level of assistance  High weaning success rate	Low respiratory drive High level of support <b>Excessive assistance</b> High weaning failure rate



Another way to evaluate a patient's ability to breathe independently and therefore be extubated is to evaluate their **vital capacity**, or **VC**. For this test, we remove the patient from the ventilator and ask him to perform a deep inhalation and a forced exhalation.

We use a spirometer connected to the tube as a measuring device. VC is a measure of respiratory muscle endurance and reserve. We expect a double or triple Vt value in the patient's spontaneous response. If able to reach these numbers, the patient can be extubated.

There is also another system to evaluate the muscular strength of a patient who needs to be extubated, and it is the measurement of **NIF**, or **Negative Inspiratory Force**, or Negative Inspiratory Pressure.



With this test, we measure the ability of the respiratory muscles to generate a negative pressure within the airways such that it can be considered adequate for extubation.

This measurement can be carried out either with an instrument with an unpronounceable name (vacuum gauge) or with the ventilator that has the capabilities.

A NIF  $\leq -25$  cmH<sub>2</sub>O predicts successful spontaneous breathing and a NIF  $\leq -26$  cmH<sub>2</sub>O predicts successful extubation. You will find different values in some works, among which the most quoted are -20 cmH<sub>2</sub>O non-executable and -30 cmH<sub>2</sub>O executable.

I'll insert all four, damn greed!

<b>NIF or Negative Inspiratory Force</b>			
<b><math>\leq -20</math> cmH<sub>2</sub>O</b> Not extubable	<b><math>\leq -25</math> cmH<sub>2</sub>O</b> SBT successful	<b><math>\leq -26</math> cmH<sub>2</sub>O</b> Extubation possible	<b><math>\geq -30</math> cmH<sub>2</sub>O</b> Extubable

We have understood that the weaning methods are different and can be chosen based on the type of patient. It is not mandatory to follow a standard weaning process, but it is possible that the clinician decides on an empirical approach. In any case, what must not be missing throughout the SBT phase is careful monitoring of the patient, who must be in a comfortable condition.

### What are the ventilator settings for?

Vt, PEEP, Rise Time etc... But what are they really for? I'll make a very short list of the control parameters that we can modify on the ventilator and "what they are for".

I hope that, arranged visually, they can be clearer.

Checks on ventilation	Checks on oxygenation	Synchronization controls
Tidal volume or Vt Pressure control Pressure support or PS Installments or FR Inspiratory time or Ti I:E ratio	FiO2 PEEP	Patient trigger Rise time/pressure ramp Flow cycle

### Slope, rise time and ramp

In the context of mechanical ventilation, you will often hear about slope, rise time and ramp. These are not exactly the same thing but are related terms that refer to different aspects of how air or gas is delivered in mechanical ventilators.

A very brief description:

**Slope:** Slope refers to the rate of change in pressure or gas flow delivered by the ventilator. In other words, it is the slope of the curve that represents how pressure or flow varies over time during ventilation. A steeper slope indicates a more rapid increase in pressure or flow, while a less steep slope indicates a more gradual increase.

Rise Time: Rise time refers to the time required to reach the maximum pressure or flow value during the inhalation phase. A shorter rise time indicates a faster increase in pressure or flow during inspiration.

Ramp: Ramp is often used as a synonym for "rise time". Refers to the period of time during which pressure or flow increases from the current initial value to the maximum value during inspiration. A longer ramp means that the increase in pressure or flow is more gradual, while a shorter ramp indicates a more rapid increase.

In summary, while these terms are interrelated and all refer to the dynamics of air or gas delivery during mechanical ventilation, modifying them varies the manner, harsh or gentle, in which these gases are delivered.

## What does VG mean?

Sometimes you will find the acronym VG written next to the ventilation mode.

VG stands for **guaranteed volume**, found in pressure control modes. Here the clinician wants the target to be  $V_t$ . In these modes the ventilator will continue its ventilation with target pressure, but trying to reach that famous  $V_t$ .

Such ventilation helps maintain a better ventilation/perfusion ratio.

On the other hand, having to reach that guaranteed volume can lead to an increase in pressure, and this can generate barotrauma.

## Humidification and heating

Under normal conditions, the upper airways are responsible for humidifying and heating the inspired gases. In normal breathing, the gases have already reached the ideal temperature and humidity at the keel level. Correct humidification of the gases is necessary for the motility of the cilia present in the mucosa and promotes adequate fluidization of the secretions.

When we bypass these pathways via a tracheostomy tube or tube, inspired gases are not warmed or humidified.

Furthermore, in invasive ventilation we use oxygen which is free of humidity and is delivered at room temperature, therefore cold compared to the needs of the pulmonary system.

Ventilation in these conditions does not cause problems for short and limited periods of time but can be a big problem with prolonged ventilation.

Tracheitis, bronchitis, pneumonia, alteration of ciliary motility and thick secretions are the possible consequences of inadequate humidification and heating of respiratory gases. You will often find the humidification and heating of respiratory gases referred to as respiratory gas conditioning.

When a patient is subjected to mechanical ventilation, it is advisable to also take care of the aspect of **conditioning** the inspired gases; It doesn't matter whether it is done via special devices or via HME filters. Remember that a patient receiving only oxygen support with devices such as masks or similar requires adequate humidification of the inspired gases when the quantity of oxygen delivered exceeds 4-6 liters per minute.

Patients with an endotracheal tube who are carrying out a trial or with a tracheostomy tube in spontaneous breathing and not connected to a mechanical ventilator must use the so-called "artificial noses". These devices are not only HME filters capable of maintaining an adequate humidity value and heating of the airways, there are different types with different hygroscopic

capacities. What does hygroscopic mean? Simple, which absorbs or retains the humidity present in the air.

## Synchrony, asynchrony, dyssynchrony and more

- **Synchrony**

- $P_{vent}$  and  $P_{mus}$  start and end at the **same** time
- $P_{mus}$  is the reference signal

- **Asynchrony**

- Absence of one signal ( $P_{vent}$  or  $P_{mus}$ )
  - Failed trigger:  $P_{mus}$  without  $P_{vent}$
  - False trigger:  $P_{vent}$  without  $P_{mus}$

### Dyssynchrony

- $P_{vent}$  and  $P_{mus}$  start and end at **different** times
  - Early trigger or cycle:  $P_{vent}$  before  $P_{mus}$
  - Late trigger or cycle:  $P_{vent}$  after  $P_{mus}$

In mechanical ventilation it is preferable to have a good balance between the work done by the ventilator and that done by the patient. When we achieve this balance, we can talk about **synchrony**, that is, the ability of the two protagonists not to disturb each other.

We have **asynchrony** when there is no synchrony between the ventilator and the patient, for example due to a failed trigger or a false trigger. In this case, there may be a patient trigger but no response from the ventilator, or vice versa. We are talking about **dyssynchrony**, when the ventilator responds at the wrong times to the patient's trigger. We can therefore have a too rapid or early response of the ventilator and a late response. In both cases, the ventilator and patient will try to move the air at different times, disturbing each other.

You will often hear people talking about **deventilation** in these terms: "Doc, the patient is deventilating...!".

In reality, it is not a "manual" term, but it has entered the common phrasebook. However, I found in a 2022 work by Critical Care Medicine\* that the word ventilation failure has these meanings: Inadequacy of ventilation: The ventilator is unable to provide sufficient tidal volume or pressure to ensure adequate ventilation.

Lack of synchrony between ventilator and patient: the patient is unable to breathe in a coordinated manner with the ventilator.

Airway obstruction: The airways are blocked by secretions, foreign bodies or edema.

Lung conditions: Lung conditions can make the lungs less able to accept air.

Mechanical ventilator problems: The ventilator is damaged or malfunctioning.

\*Sadek SH, El-kholy MM, Abdulmoez MS, El-Morshedy RM. Patient-ventilator asynchrony as a predictor of weaning failure in mechanically ventilated COPD patients. Egypt J Bronchol. 2021;15(1):30. doi: 10.1186/s43168-021-00076-9. Epub 2021 Jun 3. PMID: PMC8267513.

When you hear about "fight to ventilator", you have to think of a lack of synchrony between ventilator and patient. In this case the term "struggle" fits perfectly, imagine a patient trying to exhale while the ventilator delivers its inspiratory flow!

## The answers

If you were paying attention, I only asked a couple of questions in this long text, here are the answers:

We can reduce the amount of  $FiO_2$  if we ventilate a patient with a different gas, for example nitric oxide. But, more simply, by increasing the dead space we can go below 0.21  $FiO_2$ .

The first thing we do when we are born, referring to air, is to inhale.

The last thing, thanks to spring back, is to exhale.

## Greetings and thanks

It wasn't **easy** to talk about ventilation and it wasn't **easy** to do it in an "**easy**" way!

Some parts are, despite the title, a little complicated, but it was impossible to make them more understandable; I wouldn't say really complicated, they require a moment of extra attention. You need a pinch of curiosity and the desire to review these lines a couple of times, this is the most appropriate approach to this reading. Thanks to the fact that brevity does not belong to it!, I advise you to read it slowly, one part at a time. Use what you read as basic help, nothing more, and compare yourself with those who "know" more than you, don't stop being curious. Remember that it is aimed at people who are completely unfamiliar with the topic.

**Carefully consider who you ask for clarification on these topics, not everyone wants to share information or simply doesn't have information to share.**

In the first case you will waste time, in the second you will waste a lot of time.

I absolutely have to thank:

My family for their help and infinite patience. Thank you.

**Ehab Daoud** MD, FACP, FCCP, Critical Care Physician Founder and president of the Society and Journal of Mechanical Ventilation  
Kaneohe, Hawaii, USA

**Salvatore Sardo** MD, Department of Medical Sciences and Public Health, University of Cagliari, Monserrato, Italy.

The **Society of Mechanical Ventilation** (Ehab Daoud) made me believe I could do a good job!  
Thank you.

**Robert L. Chatburn** who showed extraordinary availability, thank you.

**Hamilton Medical** who offered me all the help possible as well as permission to use his "infinite" materials, so thank you very much.

**Dimar**, who was also available to give me a hand without asking for anything in return.

To **Joe Lewis**, respiratory therapist whose videos I devoured on YouTube where you can find him as **Respiratory Coach**, Thank you.

But I owe the biggest thanks to those who didn't help me.

That's right, I sent numerous requests for help to industry experts, respiratory therapists, well-known brands. How many requests? 42 emails without any response, not 2!

I understand well that a "nobody" is not even worth a NO! But to be honest, it was also those "non-answers" that made me extend my journey a little and managed, for better or worse, to make me reach my goal; this wasn't a bad thing, in fact it allowed me to acquire more knowledge. So to them too, thank you.

My greatest desire is to be able to help someone, even a single person, this would make me proud. I apologize for the length of the "job", believe me I didn't foresee it.

I also apologize for the mistakes I made, after all I'm just a nurse!

I thank everyone who wants to read these lines and who wants to give me their opinion on these 5 months spent "trying" to understand and explain ventilation.

I provide this work completely free of charge in PDF format without any restrictions on distribution.

If you need to modify it to adapt it to your needs, contact me and I will provide you with the work in Word format, easily editable.

[drago.me@tiscali.it](mailto:drago.me@tiscali.it)

[sdrakon@tiscali.it](mailto:sdrakon@tiscali.it)

Greetings, Gigi

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For any questions, please contact the author

Society of Mechanical Ventilation

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