Pressure control versus volume control invasive mechanical ventilation in pediatrics: A narrative review

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ABSTRACT

Invasive mechanical ventilation (IMV) is widely used in pediatric intensive care units. Acute lower respiratory infection is its primary indication; it is characterized by increased inspiratory and expiratory resistance, as well as decreased lung compliance. It can progress to acute respiratory distress syndrome, which poses a challenge in optimizing IMV.

Although different ventilatory modes are not presumed to generate significant clinical differences, there is a marked preference for the pressure control mode in pediatrics. In predominantly obstructive conditions, volume control mode ensures ventilation regardless of the degree of inspiratory resistance, allowing for extended expiratory time and preventing hyperinflation. In restrictive conditions, pressure control enables ventilation to be adjusted to protective parameters, albeit with the potential risk of inducing damage due to higher flow rates.

The physiological basis of the different ventilation modes and their clinical application are reviewed.

Keywords: *pediatric intensive care units; bronchiolitis; mechanical ventilation; flow profiles.*

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INTRODUCTION

Invasive mechanical ventilation (IMV) is a common practice in pediatric intensive care units (PICUs), not without complications.[1] To mitigate its adverse impact, training programs in IMV and adherence to recommendations are necessary.[2-5]

In pediatrics, the preferred ventilatory mode is pressure control (PC) over volume control (VC).[6,7] The choice of either mode is interchangeable in practice, as the available data do not suggest any difference in clinical outcomes.[8]

Some assumptions –not always supported by evidence– have been made that attribute specific advantages to certain ventilation modes. Among them, it is postulated that PC mode would be more physiological due to the way it administers inspiratory flow; would limit induced lung damage; in restrictive conditions would promote alveolar recruitment and oxygenation, and would be more comfortable during assisted ventilation.[8,9]. On the other hand, VC mode is usually considered more suitable for assessing lung mechanics and ventilation in obstructive conditions.[8,9]

We set out to write this manuscript to address the physiological principles of the main ventilatory modes and their application in pediatric clinical practice.

CLINICAL PATTERNS IN PEDIATRICS

It is assumed that a specific absolute clinical pattern (obstructive or restrictive) can guide the choice of ventilation mode. This segmentation is not as precise in pediatrics, where the leading causes of admission to IMV are respiratory in origin,[6] including acute lower respiratory infection (ALRI), with respiratory syncytial virus (RSV) being the most common etiology.[10-12]

The pathophysiological changes in bronchiolitis include obstruction of the small airways, resulting in a consequent increase in inspiratory and expiratory resistance, air trapping, decreased lung compliance, and hypoxemia. This is associated with alveolar collapse, increased dead space, and pulmonary shunting.[13] A proportion of these patients will progress to pediatric acute respiratory distress syndrome (PARDS).[4] In a study of 47 PICUs, 44% of 472 infants with ALRI (75% with viral rescue) met criteria for PARDS on day 1 of IMV.[14]

The treatment of bronchiolitis consists of supportive measures and is based on recommendations.[15] In its severe form, it can be classified into phenotypes: apnea in infants younger than 1 month, obstructive and/or restrictive patterns in infants aged 1 to 6 months, and asthma-like expression in infants older than 6 months.[16]

Few studies have evaluated lung mechanics in ALRI with IMV.[17-23] Cruces observed that, in 16 patients, the elastic component of the respiratory system, which is involved in working pressure, is the most compromised.[17] In line with this finding, in another cohort of 31 patients, a linear correlation was found between distensibility and functional residual capacity.[21] However, this association could be reversed with higher programmed positive end-expiratory pressure (PEEP) values, given that these patients tend to overdistention.[20,21]

Andreolio and Burrati described, in cohorts of 64 and 37 infants, respectively (predominantly RSV), high inspiratory and expiratory resistances, accompanied by decreased distensibility.[18,23]

These findings show that ALRI does not follow absolute patterns and that, once IMV is established, the heterogeneity of this syndrome makes ventilation in these patients challenging.

PHYSIOLOGICAL PRINCIPLES OF VENTILATION MODES: PRESSURE CONTROL AND VOLUME CONTROL

For educational purposes, we will refer to downward flow delivery for PC mode and constant flow delivery for VC mode. This form of delivery is a consequence of the pressure gradient between the ventilator and the alveolar pressure.

PC mode is considered a low-pressure mode, and the gradient is the result of the programmed peak inspiratory pressure (PIP) and alveolar pressure. As air enters and alveolar pressure increases, the gradient decreases and, with it, the volume; as a result, delivery is exponential.

VC mode is considered a high-pressure mode, referring to the pressure exerted by the device, in the range of 80 to 5000 cmH_2O. Consequently, as air enters and alveolar pressure increases, the decrease in gradient is negligible, and the volume enters at a constant rate.[24,25]

It follows that, in PC, the ventilator delivers air until the PIP and alveolar pressure are equal (zero flow), which may or may not coincide with the prescribed inspiratory time (Ti). In VC, as the gradient is high, the volume is delivered entirely within the programmed Ti, without the need for pressure equalization (*Figure 1*).

TIME CONSTANT AND LINK BETWEEN VENTILATION MODES

The time constant (TC) is a key concept for understanding how mechanical ventilation works. It is a product of compliance by resistance, being unique, proper to the system. For educational purposes, we will divide the TC into two segments: during inspiration (TCi) and during exhalation (TCe).

A TC describes the characteristics of equilibrium between pressure, flow, and volume between two points in the patient-ventilator circuit, representing the segmentation of inspiratory and expiratory times into variable periods but with fixed percentages.[26]

In the case of TCe, the ventilatory mode is indifferent, as exhalation is passive and airflow depends on the gradient between alveolar pressure and atmospheric pressure or programmed PEEP. It is estimated that with a pressure of 45 cmH_2O, most alveoli are recruited,[27] resulting in expiration behaving as a low-pressure system, where airflow is exponential.

The TCe represents the time it would take for this exponential change to be completed if the rate of change remained at its initial level, and will be 63% of the volume set at each TC. Thus, 63%, 86%, 95%, 98%, and 99% of the volume will be exhaled in successive TC.[26] If the expiratory time (Te) respects the five TCs, the pressures at both ends of the circuit will be equalized, and the total PEEP (PEEPt = intrinsic [PEEPi] + programmed) will be equal to the programmed PEEP. At the same time, if the Te is shorter, exhalation will be incomplete (PEEPt > PEEP)[28] (Appendix: TCe measurements).[29]

In relation to the TCi, in PC mode, there is a phenomenon similar to that which occurs during expiration, given the low pressure gradient (PIP - alveolar), the air delivery will be exponential until the pressures at both ends are equal (zero flow), when the five TC are met. If this does not happen before the programmed Ti, there will be no zero flow, the expected volume will not be delivered in its entirety, and the pressure gradient will remain in effect at the end of inspiration (*Figure 2*).[17,23,28] This premise also applies to the adaptive pressure-regulated volume control mode, which attempts to ensure the preset volume with the lowest possible pressure.[30,31]

In VC mode, as the pressure gradient is broad, only exponential delivery will occur if the lung accepts the volume resulting from that gradient. Since this is incompatible, and only the programmed volume is delivered, it will be done constantly and reliably, regardless of the TCi of the system.

VENTILATION MODES AND THEIR RELATIONSHIP TO CLINICAL PRACTICE

By understanding how gas is delivered in ventilatory modes and their relationship with TCs, we can analyze their impact on clinical practice.

As mentioned, absolute ventilatory patterns are unlikely to occur in routine practice, which presupposes complexity when choosing and programming the mode and parameters of IMV. However, through adequate monitoring of the different components of working pressure, it is possible to identify trends (*Figure 3*).[17,32,33]

In this section, we will examine the implications of different ventilatory modes and their consequences in relation to various pathologies and other considerations.

Restrictive pathology (PARDS)

It has traditionally been argued that, in this type of entity, PC mode would favor greater alveolar recruitment and better oxygenation, due to its form of gas delivery. In *Table 1*, example A, a greater alveolar volume (indicative of recruitment) is available for gas exchange earlier. However, this claim lacks solid support in the available evidence.

It can also be seen from example A, given the low TCi (low compliance), that at the scheduled time the pressures between the ventilator and the alveoli will have equalized. As a result, the PIP and plateau pressure (Pplat: airway pressure at the end of inspiration) will be identical. This allows direct evaluation of the driving pressure (DP: Pplat - PEEP) and adjustment to protective parameters (<15 cmH_2O).[3-5]

In relation to TCe, an adequate PEEP that prevents alveolar collapse would allow it to be prolonged because of the higher-pressure gradient, enabling an effective emptying.[26]

Obstructive pathology with increased inspiratory resistance

Table 1, example B, illustrates that, in PC, at the end of Ti, a pressure gradient remains in the ventilator-patient circuit. As a result, the PIP will be higher than the Pplat, and the volume administered will be lower than expected. This is evidenced in a recent laboratory study evaluating ventilation with decelerated flow, which demonstrates the overestimation of PIP compared to Pplat when resistance in smaller endotracheal tubes (ETTs) is increased, by measuring tracheal pressure and eliminating the resistance component of the ETT.[28] Clinical studies highlight this particularity. Buratti demonstrated that the PIP is not accurate for estimating the Pplat in infants with ALRI, with a mean difference of 7.3 cmH_2O.[23]

This is relevant because increased inspiratory and expiratory resistance is a common phenomenon in pediatric IMV. Bruno, in a study of 113 patients (median age: 5 months), found high resistance not only in those diagnosed with obstructive disease but also in the cohort with lung injury.[19]

As mentioned, ALRI is heterogeneous in its presentation.[16] Cruces compares a cohort of patients with severe asthma (n = 11) with another with severe bronchiolitis (n = 16) and establishes a PIP-Pplat difference of 8.7 cmH_2O for the asthma group as the discriminating value between the two entities.[34]

The alternatives for ensuring volume delivery in PC mode are to increase the PIP or prolong Ti (*Figure 2*), with the risk of reducing Te and promoting hyperinflation. Another option is to ventilate in VC (*Table 1*, example B), where ventilation is ensured during the programmed Ti, allowing sufficient Te for emptying.

The latter option involves an increase in the PIP. This phenomenon occurs in VC because the resistive friction component remains unchanged and overlaps the viscoelastic component (pulmonary retraction) at the end of inspiration. In contrast, in PC, it occurs in series, with a greater frictional component at the beginning of inspiration (higher flow), followed by the viscoelastic component, without overlapping.[32]

Obstructive pathology with increased expiratory resistance

The segmentation of inspiratory and expiratory resistance is provided for educational purposes only, since both will be affected in obstructive pathologies.

The expiratory phase is a passive phenomenon characterized by a decelerated, exponential flow slope. At the beginning of expiration, the greater

Table 1. Clinical patterns and ventilation modes

Example A: Restrictive pattern. A 2-month-old patient weighing 8 kg, diagnosed with ALRI with a TCi of 0.1 seconds and IMV parameters with a Ti of 0.5 seconds and Vt of 6 ml/kg.

	Pressure control		Volume control	
Time	**TC**	**Volume**	**Time**	**Volume**
0.1	1	30 ml	0.1	9.6 ml
0.2	2	41 ml	0.2	19 ml
0.3	3	45 ml	0.3	29 ml
0.4	4	47 ml	0.4	38 ml
0.5	5	48 ml	0.5	48 ml

In just over half of the programmed Ti, in PC mode, 95% of the target volume (3 TC) will have been delivered due to its exponential delivery. In comparison, only 60% will have been delivered in VC mode, given its constant delivery.

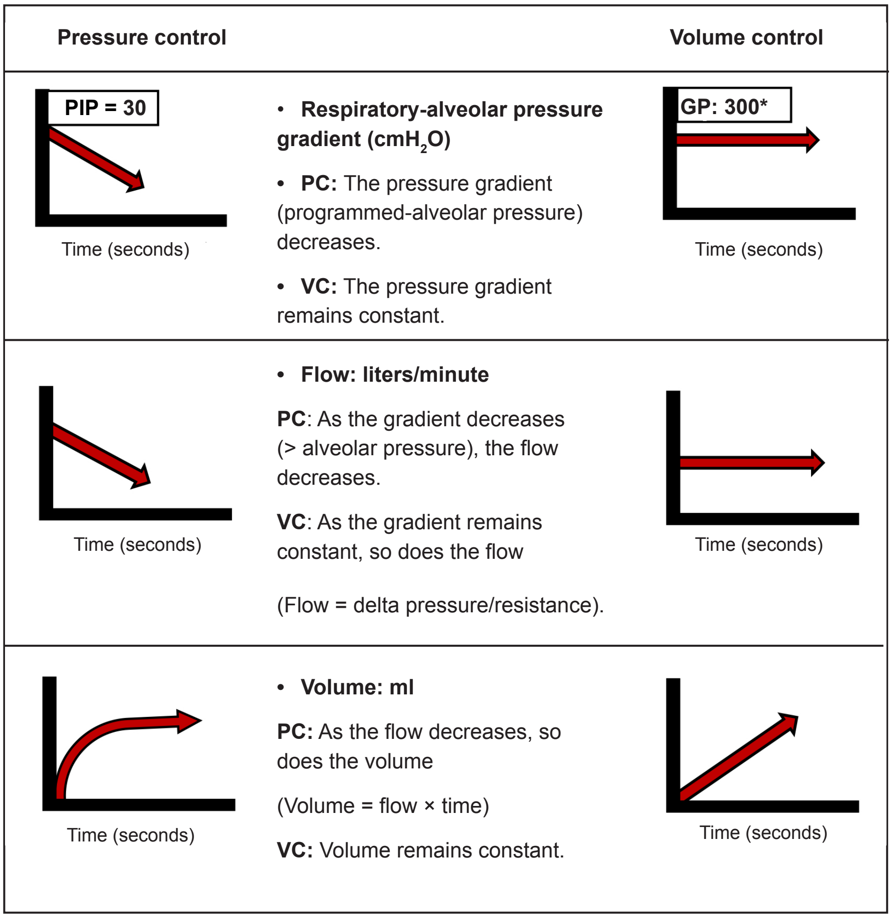
Example B: Obstructive pattern. A 2-month-old patient, weight: 8 kg, diagnosed with ALRI with a TCi of 0.3 seconds and IMV parameters with a Ti of 0.5 and a Vt of 6 ml/kg.

	Pressure control		Volume control	
Time	**TC**	**Volume**	**Time**	**Volume**
0.1	-	-	0.1	9.6 ml
0.2	-	-	0.2	19 ml
0.3	1	30 ml	0.3	29 ml
0.4	-	-	0.4	38 ml
0.5	1.7	1,7	0.5	48 ml

At the end of the programmed time (Ti), in PC mode, only 78% (1.7 TC) of the planned volume will have been administered, whereas in VC mode, the total volume will have been delivered.

ALRI: acute low respiratory infection; TCi: time constant inspiratory; Ti: inspiratory time; IMV: invasive mechanical ventilation; Vt: tidal volume.
Time: in seconds.

Figure 1. Gas delivery pattern for ventilation modes



*PC: pressure control; VC: volume control; PIP: peak inspiratory pressure (cmH_2O); GP: generator pressure (cmH_2O); *300 cmH_2O: arbitrary number used as an example.*
Modified from Manual de Ventilación Mecánica Pediátrica y Neonatal (Manual of Pediatric and Neonatal Mechanical Ventilation).[24]

pressure gradient is responsible for the greater flow. Successive TCs are prolonged, as the flow attenuates (due to a lower pressure delta and greater resistance), which can lead to distal collapse of the small airways. [26]

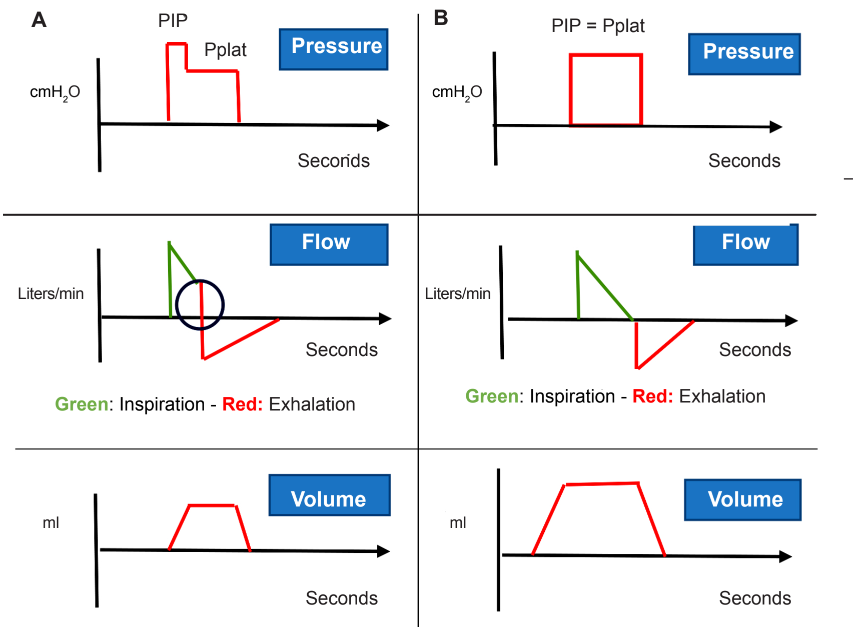
La PEEPt will only be equal to the programmed PEEP when Te has 5 TC and the flow is zero; otherwise, there will be hyperinflation.[26] This may not be observed using the flow-time curve or expiratory pause, because of distal airway collapse; the Pplat is the appropriate marker for assessing the degree of entrapment. The addition of extrinsic PEEP in this circumstance would act as a "mechanical stent" for the obstructed airways, shortening the TCe and allowing them to empty (*Figure 4*).[35]

Ventilation modes and lung mechanics

It is assumed that pulmonary mechanics are more reliable in VC mode. Cruces evaluated this hypothesis in 18 patients with PARDS (median age: 5 months) and observed that, in VC mode, there was a greater PIP, a greater viscoelastic component pressure curve, end of the first decrease from PIP to Pplat, and lower peak inspiratory flow (7 vs. 21 L/min). No differences were found in Pplat, DP, and auto-PEEP.[32]

In conclusion, both modes allow for the assessment of lung mechanics; however, the PC mode underestimates the viscoelastic component, which is clinically significant, as the accurate marker of lung injury, driving pressure, should include this component. Additionally, PC mode generates three times more flow, which may contribute to ventilator-induced lung injury.

Figure 2. Pressure control mode, relationship between inspiratory time constant, inspiratory time and volume



A) If Ti is less than 5 inspiratory TCs, the flow does not reach zero, and a pressure gradient remains at the end of inspiration PIP > Pplat. Less volume will have been delivered.
B) PIP will only be equal to Pplat if Ti accommodates the 5 TCs, reaching zero flow.
PC: pressure control; TC: time constant; PIP: peak inspiratory pressure; Pplat: plateau pressure; Ti: inspiratory time.
Prepared by the authors.

Ventilation modes and lung injury

In 2016, two new concepts emerged: *mechanical power*, which encompasses all factors involved in IMV and together constitute a single physical variable, the energy delivered to the respiratory system over time; and *ergotrauma*, which describes the induced injury of a global and dynamic form, resulting from the degree of deformation (tidal volume/functional residual capacity) and stress (transpulmonary pressure delta) developed by mechanical power.[36]

The flow profiles deliver the same amount of energy at the end of inspiration. Still, the decelerated profiles do so in a higher percentage earlier, and the constant profiles transfer the energy load uniformly.[37] *Table 1*, example A, shows that at the end of Ti, both ventilation modes will have delivered the same volume (same strain). However, in the first TCi, the PC mode will have delivered three times more volume. The consequence is a higher deformation rate of the alveolar units with respect to time (strain rate = functional residual capacity/flow).[38]

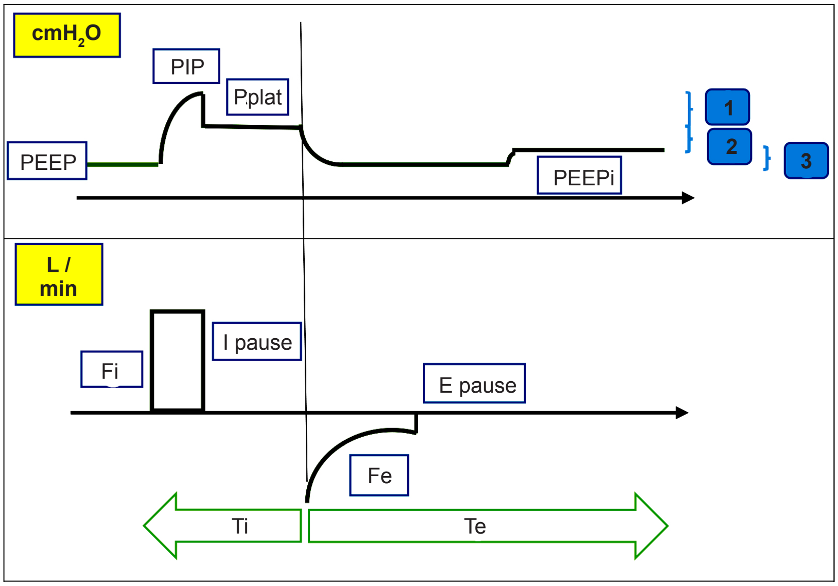
The potential for damage caused by decelerated flow was addressed by Percy, who compared mechanical power with square and decelerated flow in 185 patients with PARDS (median age: 8.3 years), finding greater mechanical power with PC (0.49 vs. 0.46 J/m), with uncertain clinical relevance.[39]

Ventilation modes in patient-triggered (assisted) breathing

Given that PC mode delivers volume until the pressures at both ends of the circuit (PIP and alveolar) are equal, when the patient makes an effort, the pleural (*ergo* alveolar) pressure decreases; this gradient will be greater and, consequently, so will the volume delivered (*Figure 5*), resulting in less respiratory work and less dyssynchrony, due to the administration of a greater volume in the initial phase.[8,40] This may entail a potential risk of patient self-induced lung injury (PSILI), given that the DP is not reflected by the ventilator, but rather by the resulting gradient between the PIP and the alveolar pressure, which tends to become negative.[41] In contrast, in VC, the volume delivered will be fixed, regardless of the increased demand.

This supports the premise that, in patients with

Figure 3. Pulmonary mechanics



The working pressure of the respiratory system is the pressure required to overcome friction forces, elastic forces, and impedance. It can be calculated using the equation of motion: Vt / SC + Ri × Flow + PEEPi.
Three components can be distinguished: resistive: PIP - Pplat (1); elastic: Pplat - total PEEP (2), and load threshold: total PEEP - PEEP (3).

Formulas:
Inspiratory resistance: (PIP - Pplat / inspiratory flow) × 60
Expiratory resistance (Pplat - PEEP / expiratory flow) × 60
Static compliance: Vt / Pplat - PEEP
Time constant: static compliance × resistance (I or E) / 1,000

Vt: tidal volume; SC: static compliance; Ri: inspiratory resistance; PIP: peak inspiratory pressure; PEEP: positive end-expiratory pressure; Pplat: plateau pressure; PEEPi: intrinsic PEEP; Fi and Fe: inspiratory and expiratory flow; I pause and E pause: inspiratory and expiratory pause; Ti and Te: inspiratory and expiratory time (seconds).
Extracted and modified from Cruces[17,32] and Newth.[33]

active ventilation, PC mode is more comfortable and reduces respiratory effort, unless the flow is adjusted to demand in VC mode.

COMMENTS

In obstructive pathologies, it seems logical to ventilate in VC, as it guarantees delivery at the programmed Ti and allows adjustment of frequency and TE to avoid hyperinflation. It is crucial to monitor the Pplat and compare it with the PEEP we are programming, which allows detection and treatment of hidden auto-PEEP.[42]

In restrictive pathologies, ventilation in VC with the addition of an inspiratory pause allows for the evaluation of lung mechanics and adjustment of protective parameters.

In this context, it may be attractive to ventilate in PC mode, where the PIP will be equal to the Pplat (if the flow is equal to 0), allowing direct evaluation of DP. Furthermore, by maintaining this constant, it is possible to titrate PEEP for better compliance (greater tidal volume).[43,44] Although this could imply a risk of volutrauma, the real problem lies in poor compliance. Tidal volume is a consequence of this alteration. It can be adjusted within a safe range (6 to 8 ml/kg) and even exceeded, provided that protective parameters, such as adequate DP, are maintained.[45,46]

CONCLUSION

Its physiological basis and the possibility of monitoring justify the use of VC mode. Although PC mode could be helpful in restrictive diseases, its use must be weighed against the potential risk associated with strain rate. ■

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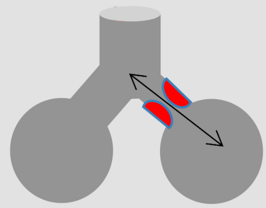
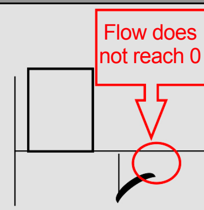
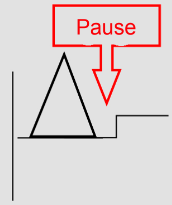
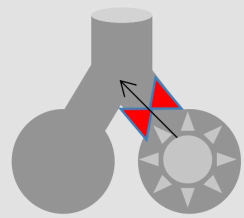
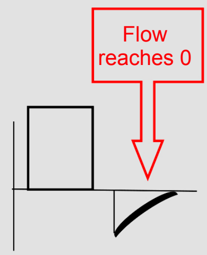
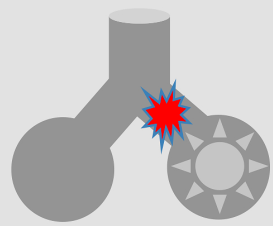
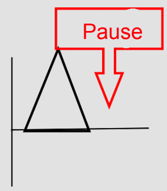
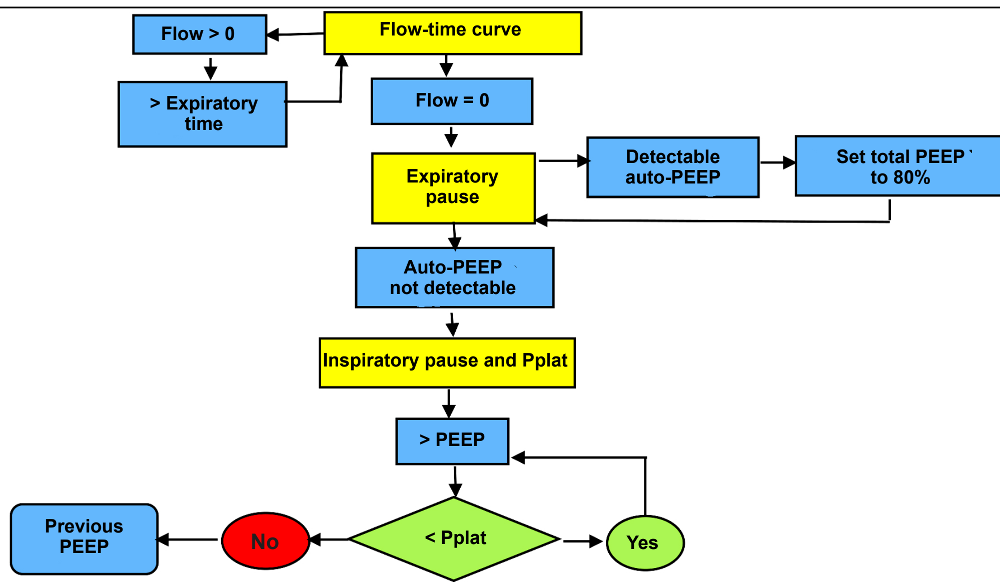
Figure 4. Pathophysiology, diagnosis, and treatment of hyperinflation

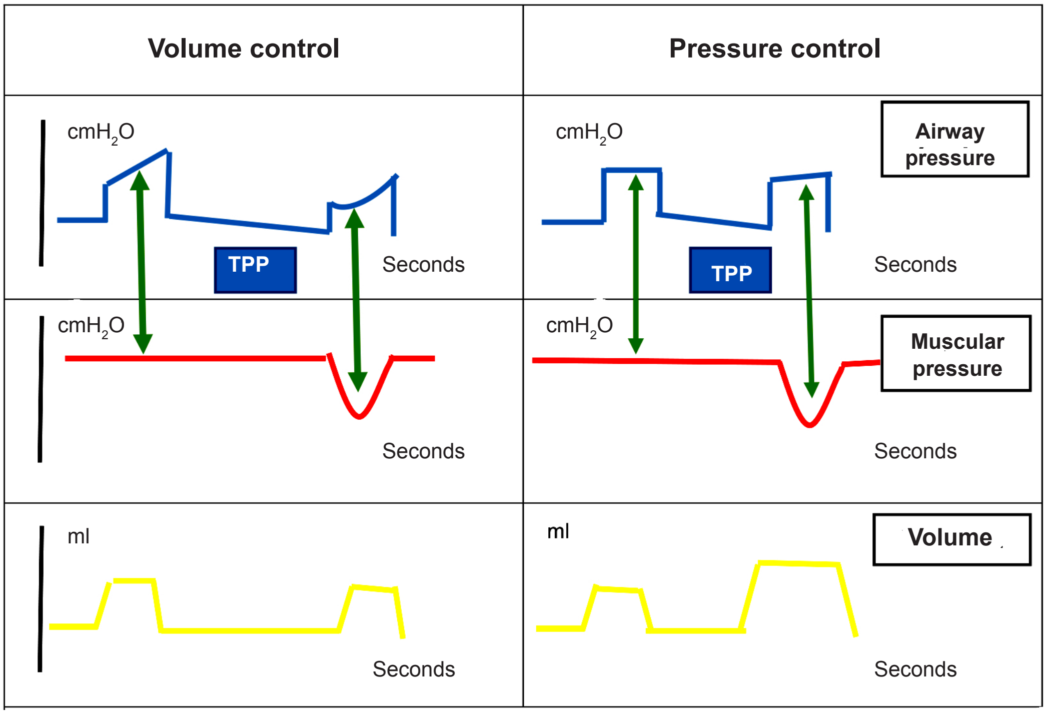
Name		Mechanism		Flow-time curve	Expiratory pause	Treatment
Dynamic hyperinflation		Time-dependent dynamic air entrapment				Increase expiratory time
Non-dynamic hyper-inflation	Measurable	PEEP-dependent air trapping due to distal collapse (Venturi effect)				Increase PEEP
	NON-measurable	PEEP-dependent air trapping due to the unidirectional valve mechanism caused by secretions				

Treatment algorithm for hyperinflation



PEEP: positive end-expiratory pressure; Pplat: plateau pressure.
Extracted and modified from Manual de Ventilación Mecánica Pediátrica y Neonatal.[35]

Figure 5. Ventilation modes in assisted breathing



In VC, airway pressure decreases when the muscular pressure increases and the TPP is maintained. In PC, active breathing causes an increase in the TPP when the airway pressure is constant, providing greater volume.
TPP: transpulmonary pressure; VC: volume control; PC: pressure control.
Modified from Rittayamai.[8]

The supplementary material provided with this article is presented as submitted by the authors. It is available at: https://www.sap.org.ar/docs/publicaciones/archivosarg/2025/10730_ACT_Taffarel_Anexo.pdf

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