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REVIEW ARTICLES

Comparative Hemodynamic Effects of BiPAP and APRV in Patients with ARDS. A Systematic Review

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Abstract

Introduction: Positive pressure ventilation is key in the treatment of patients with acute respiratory distress syndrome. Bilevel Positive Airway Pressure and Airway Pressure Release Ventilation modalities aim to improve oxygenation and reduce lung injury, but their hemodynamic effects vary.

Objective: To compare the clinical and hemodynamic effects of Bilevel Positive Airway Pressure and Airway Pressure Release Ventilation in acute respiratory distress syndrome patients.

Materials and Methods: Clinical studies published through November 2025 in the PubMed, Scopus, and Web of Science databases were analyzed. Articles on acute respiratory distress syndrome patients treated with Bilevel Positive Airway Pressure or Airway Pressure Release Ventilation that reported hemodynamic parameters were included. Studies without hemodynamic data, those outside acute respiratory distress syndrome, and non-clinical studies were excluded.

Results: Bilevel Positive Airway Pressure improves alveolar ventilation and oxygenation while modestly reducing left ventricular afterload; cardiac output remains stable in normovolemic patients. Airway Pressure Release Ventilation significantly increases oxygenation and ejection fraction, reduces alveolar shunt, and opens atelectatic alveoli, but is associated with more pronounced decreases in mean arterial pressure and preload. Clinical studies report that Airway Pressure Release Ventilation increases cardiac index and reduces central venous pressure, improving oxygenation while reducing the need for hemodynamic support.

Conclusions: Bilevel Positive Airway Pressure is suitable for moderate acute respiratory distress syndrome and hypercapnic respiratory failure, offering favorable hemodynamics and noninvasive support. Airway Pressure Release Ventilation is preferred in severe acute respiratory distress syndrome with refractory hypoxemia, as it provides superior oxygenation and alveolar recruitment but requires careful hemodynamic monitoring. The choice of modality should balance respiratory benefits with cardiovascular risks.

Keywords: Bilevel Positive Airway Pressure, Airway Pressure Release Ventilation, ARDS

Introduction:

Acute respiratory distress syndrome (ARDS) is a clinical syndrome of acute respiratory failure characterized by hypoxemia and bilateral pulmonary infiltrates that cannot be fully explained by cardiac failure or volume overload. [1]

Etiology: ARDS is divided into two groups: systemic causes and primary pulmonary parenchymal injury.

Systemic causes include:

- Sepsis (most common), e.g., secondary to infection, trauma, or peritonitis [2],
- Acute Pancreatitis, Shock, Trauma, Major Burns, Massive Transfusions

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- Drugs: bleomycin, tricyclic antidepressants [2],

Causes of primary pulmonary parenchymal injury:

- Pneumonia, Aspiration, Pulmonary contusions [3][4], Fat embolism, Amniotic embolism, Lung transplantation
- Inhalation injuries, such as hyperbaric oxygen inhalation

Methodology

Study design: This systematic review evaluates the hemodynamic effects of BIPAP vs APRV ventilation modes in patients with ARDS. The review follows established guidelines for systematic research, ensuring comprehensive data collection and methodological rigor.

Search Strategy: A structured search was conducted using electronic databases, including PubMed, Scopus, and Web of Science, supplemented by manual searches of grey literature. The search included studies published before October 2025. Key search terms included "ARDS," "Positive pressure ventilation," "BiPAP," "APRV," and "Hemodynamics effect."

Inclusion Criteria: This systematic review includes peer-reviewed articles covering patients with ARDS who were ventilated using BiPAP or APRV. All clinical studies reporting hemodynamic effects, including changes in preload, afterload, cardiac output, and pulmonary pressures, were included. Articles published in any language were considered.

Data were collected from population-based, matched-cohort, case-control, cross-sectional, cohort, and clinical trials. Studies that did not report hemodynamic parameters or focused outside ARDS were excluded.

This review compares the hemodynamic effects of BiPAP and APRV, focusing on how intrathoracic pressure, duration of pressure application, and ventilation modality affect preload, afterload, cardiac output, and alveolar oxygenation in patients with ARDS.

Exclusion Criteria: Studies were excluded if they: Did not report hemodynamic parameters (preload, afterload, cardiac output, pulmonary pressures). Did not involve ARDS patients or did not use BiPAP or APRV ventilation modalities. They were editorials, commentaries, letters to the editor**,** or animal studies**.** They were non-peer-reviewed publications**.**

Pathophysiology of ARDS [3]:

Damage to lung tissue, whether from direct pulmonary or systemic causes, triggers the release of inflammatory mediators, such as IL-1, thereby initiating an inflammatory response. This response causes neutrophils to migrate into the alveoli, where they release mediators, including proinflammatory cytokines, proteases, and superoxide anions, leading to diffuse alveolar damage (DAD) [5]; consequently, endothelial cells and alveolar capillaries are damaged. This results in several stages:

Phase I – Exudative phase, where exudate accumulates in the pulmonary interstitium and alveolar surface, resulting in noncardiogenic pulmonary edema with normal pulmonary capillary wedge pressure (PCWP). This decreases pulmonary compliance and causes respiratory distress.

Phase II involves the formation of hyaline membranes composed of erythrocytes, fibrin, and cellular debris. These membranes hinder gas exchange, leading to hypoxemia. Damage to type I and type II pneumocytes reduces surfactant synthesis and secretion, leading to alveolar collapse and an intrapulmonary shunt.

Phase III - Organizational phase (late), characterized by proliferation of type II pneumocytes and infiltration of fibroblasts, leading to progressive interstitial fibrosis.

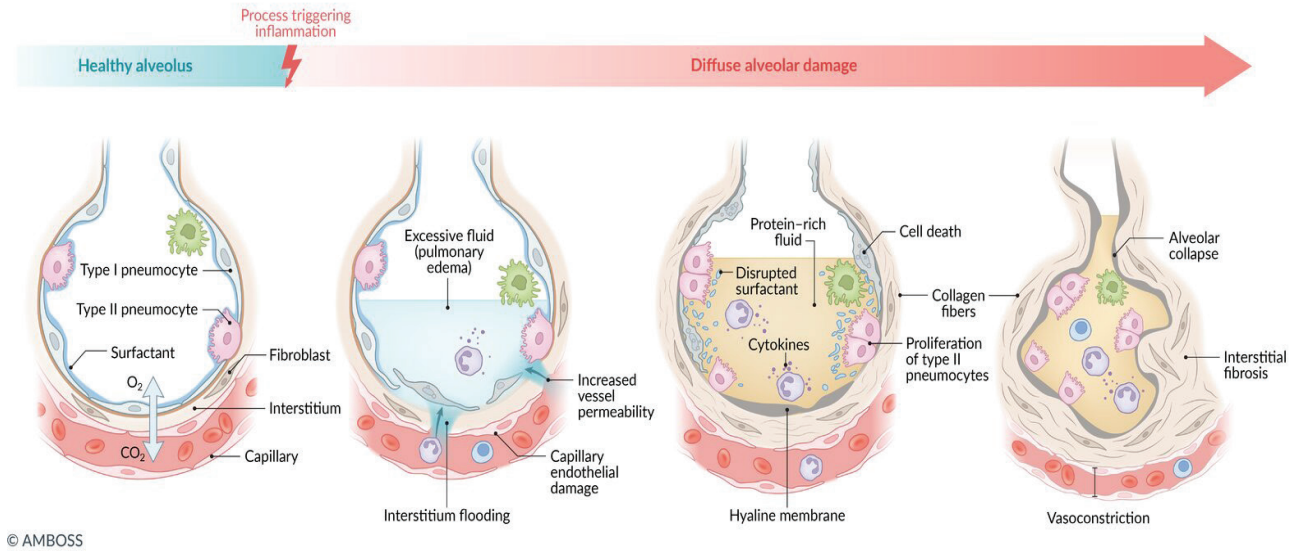

Figure 1. Pathophysiological mechanism in ARDS

Diagnosis

The diagnostic approach to patients with ARDS should begin with excluding differential diagnoses, such as TRALI (transfusion-related acute lung injury), cardiogenic pulmonary edema, TRACO (transfusion-related acute circulatory overload), or hypervolemia, and exacerbation of interstitial lung disease.

It should be suspected when a patient presents with acute pulmonary insufficiency that has a rapid onset and a potential underlying cause. The initial step is to perform a chest X-ray to evaluate for bilateral infiltrates, followed by an ABG analysis and comparison of the PaO2/FiO2 ratio to determine severity and confirm the diagnosis. Additional studies, such as transthoracic echocardiography, chest CT, NT-pro-BNP, BNP, and cardiac biomarkers, should be considered to assess for complications and identify causes.

Global definition – **Berlin criteria** for the diagnosis of ARDS:

1. Respiratory failure with acute onset
2. Bilateral opacity (diffuse shadowing) on imaging
3. Hypoxemia, assessed by evaluating the PaO2/FiO2 ratio, where a value < 300 mmHg is preferred; if ABG is not available, the SpO2/FiO2 ratio < 315 can be used, provided SpO2 is < 97%.
4. Request for respiratory support

Diagnostic methods:

1. ***Chest X-Ray*** [4, 5] – indicated in all patients with suspected ARDS. Findings vary according to the stage of the disease: bilateral, diffuse, symmetric infiltrates are seen at 1–7 days, sometimes with air bronchograms, and, in very severe cases, the lungs may be completely white ("Whitelung").

At 8–14 days and after 15 days, the findings may fade or persist; some patients develop fibrosis with reticular opacities that may be permanent. Features that favor ARDS over congestive heart failure are the absence of cardiomegaly, a regular cardiac silhouette, few or no pleural effusions, and a predominance of peripheral opacities.

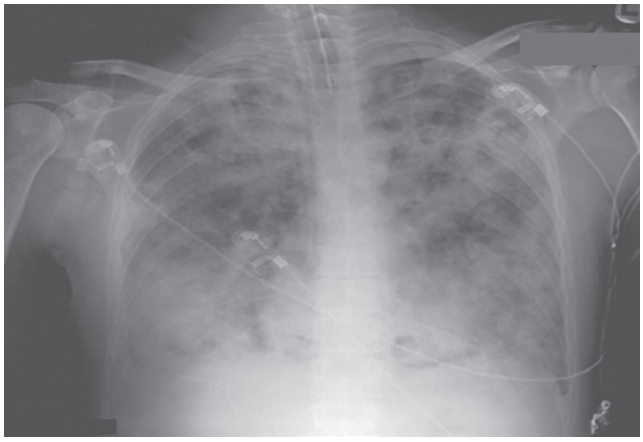

Figure 2. Radiographic image of a patient with ARDS. Bilateral diffuse infiltrates and opacities are seen, with a regular heart silhouette.

2. Chest CT [6]– may be indicated in cases where radiographic findings are insufficient to favor the diagnosis of ARDS, or when it is necessary to evaluate the causes further, initiating factors, or even complications. The findings are the same as in the X-ray,

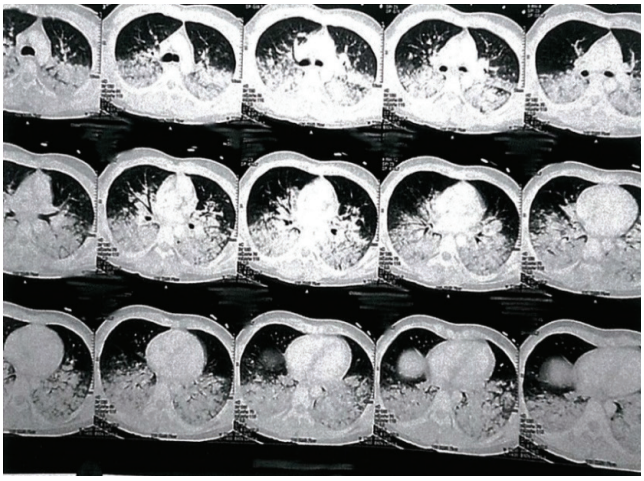

Figure 3. CT of the chest, lung window. Bilateral diffuse opacities are seen in the supine view of the patient. Source: Axial CT thorax with contrast, showing ARDS with severe worsening.

3. Lung ultrasound – especially useful for distinguishing between ARDS and congestive heart failure, with particular emphasis on hospitalized patients who are bedridden and unable to move, such as in the ICU.

4. Investigative studies [7] – assess the clinical condition and possible causes

Arterial blood gas (ABG) analysis, through which the PaO2/FiO2 ratio is assessed, and the alveolar-arterial gradient. CBC: leukocytosis in cases of Pneumonia or Sepsis. Sputum sample and microbiological culture, as well as Hemoculture to assess infectious causes, especially Sepsis as the most common cause, and Pneumonia. Lipase and Amylase are used when Acute Pancreatitis is suspected. BNP, NT-pro-BNP in heart failure (also as a predictor of the disease). Cardiac biomarkers are also required.

Treatment [8]

1. Oxygenation – is done using NIPPV, Non-rebreather mask, or HFNC, as the primary method, can be used in patients with moderate ARDS, who are hemodynamically stable. Oxygenation is also essential before intubation.

2. Corticosteroid therapy – some studies show corticosteroids reduce mortality and morbidity, mainly by decreasing the duration of intubation. Dexamethasone and Methylprednisolone are the most commonly used. They are administered during intubation. They should not be given within 2 weeks after intubation.

3. Supportive therapy – includes the use of PPIs as prophylaxis against peptic ulcers, anticoagulant

prophylaxis for venous thromboembolism from systemic hyperinflammation, adequate nutrition often using Total Parenteral Nutrition, careful management with i.v. Fluids, prudent management of hypervolemia.

4. Endotracheal intubation and mechanical ventilation is the standard of treatment, applied to all patients with rapid clinical disorientation and acute respiratory failure. Intubation is often performed using rapid sequence intubation (RSI).

Lung-Protective Ventilation

All patients undergoing endotracheal intubation should receive lung-protective ventilation to reduce the risk of ventilator-induced lung injury. This modality includes a low tidal volume (Vt 4-8 mL/kg), PEEP > 5 cmH2O, and a pressure plateau (Pplat < 30 cmH2O to prevent barotrauma). [8[1]].

1. Moderate to Severe ARDS [9, 10]

Supine positioning should be initiated as soon as the patient is stabilized, as it enhances oxygenation by reducing the ventilation-perfusion (V/Q) mismatch in the dorsal lung, which becomes atelectatic, and by increasing compliance.

This position is typically used for patients with a P/F ratio below 150 mmHg or with pulmonary edema. Relative contraindications include hemodynamic instability, severe trauma, increased intracranial pressure, and massive hemoptysis. The position is typically maintained for 12 to 16 hours each day but can cause complications such as endotracheal tube displacement, decreased oxygen saturation or blood pressure, hemoptysis, and pressure injuries.

High PEEP can be adjusted through several methods, such as titration based on oxygenation, plateau pressure, or maximal lung compliance. Increasing PEEP in this way improves oxygenation and is believed to reduce mortality in patients with acute respiratory failure.

Lower PEEP/higher FiO2

FiO_2	0.3	0.4	0.4	0.5	0.5	0.6	0.7	0.7
PEEP	5	5	8	8	10	10	10	12

FiO_2	0.7	0.8	0.9	0.9	0.9	1.0
PEEP	14	14	14	16	18	18-24

Higher PEEP/lower FiO2

FiO_2	0.3	0.3	0.3	0.3	0.3	0.4	0.4	0.5
PEEP	5	8	10	12	14	14	16	16

FiO_2	0.5	0.5-0.8	0.8	0.9	1.0	1.0
PEEP	18	20	22	22	22	24

Table 1. ARDSnet protocol for FiO_2/PEEP titration [11] *(Source: oops — Pulmcast | ICU APP Fellowship | Critical Care Training)*

2. Severe ARDS [12],

In patients with early ARDS (less than 7 days from onset), the use of ECMO should be considered, mainly when one of the following situations occurs: Very severe hypoxemia (PaO_2/FiO_2 < 80 mmHg) or hypercapnia (pH < 7.25 and $PaCO_2 \geq 60$ mmHg) that persists despite optimal management, including high PEEP and prone position and Plateau pressure reaches potentially dangerous values (e.g., > 30 cmH_2O). Monitoring tools, such as the Murray score, can help identify patients at high risk for needing ECMO [13].

Comparison of hemodynamic effects between BiPAP and APRV

Positive pressure mechanical ventilation (PPV) includes modalities such as BIPAP and ARPV. In general, positive-pressure ventilation has hemodynamic effects, which will be discussed in the following paragraphs.

During spontaneous breathing, intrathoracic pressure is negative, creating a pressure gradient that aids venous return.

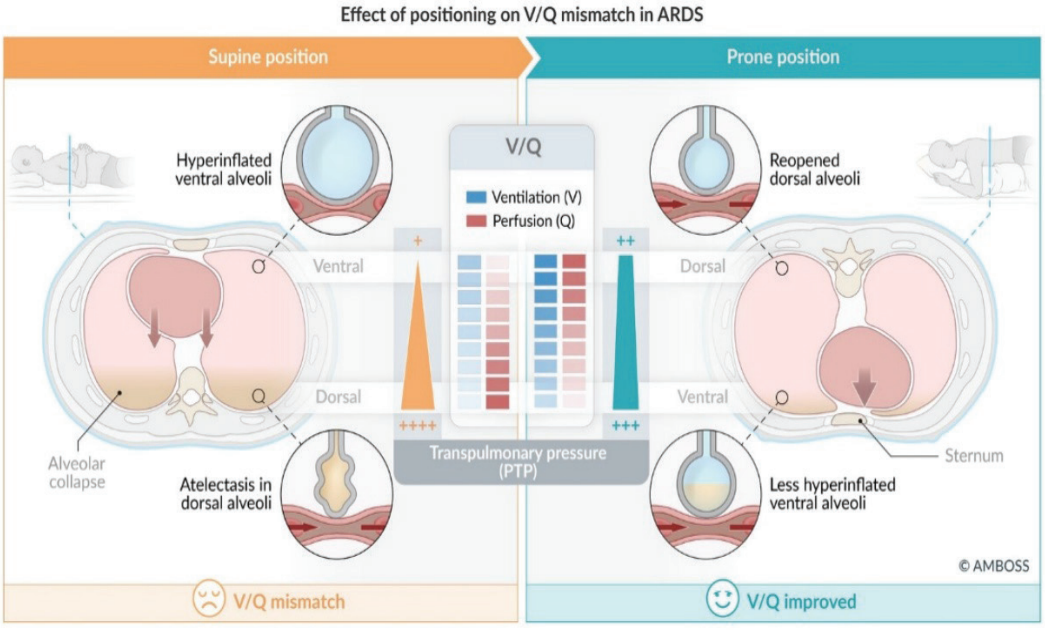

Figure 4. Schematic representation of the effect of position on the V/Q ratio

In positive-pressure ventilation, intrathoracic pressure rises. This increase in thoracic pressure also elevates pulmonary arterial pressure and pulmonary vascular resistance, thereby reducing pulmonary perfusion in some areas.

The increase in pressure within the pulmonary artery and circulation, along with higher pulmonary vascular resistance, reduces venous return from extra-thoracic organs and decreases cardiac output. At first, it lowers the filling of the right atrium and decreases the preload of the right ventricle. Additionally, increased right ventricular afterload due to elevated pulmonary vascular resistance can cause right ventricular dysfunction.

These mechanisms, along with increased intrathoracic pressure, reduce left ventricular preload, thereby decreasing stroke volume. As a result, there are noticeable changes in hemodynamic parameters: stroke volume decreases, systolic arterial pressure (SBP) drops, and mean arterial pressure (MAP) declines. Diastolic pressure initially remains stable.

Still, in severe cases of positive pressure also diastolic pressure (DBP), reflex tachycardia appears as a compensatory mechanism, cardiac output (CO) decreases, total systemic vascular resistance increases as a compensatory mechanism, we have an increase in central venous pressure (CVP) from increased intrathoracic pressure, and PCWP (Pulmonary capillary wedge pressure) is increased, in clinical practice, this is important because PCWP can give "false high" in patients who are on PPV even though they are hypovolemic. [14, 15, 16]

During spontaneous breathing, thoracic expansion creates a negative intrathoracic pressure that draws air into the lungs, while expiration occurs primarily passively through elastic recoil of the lungs. In mechanical ventilation, the ventilator forces air into the airway, generating a positive pressure that peaks at the end of the inspiratory phase. The pressure remains constant during the non-flow phase and falls again at the beginning of expiration.

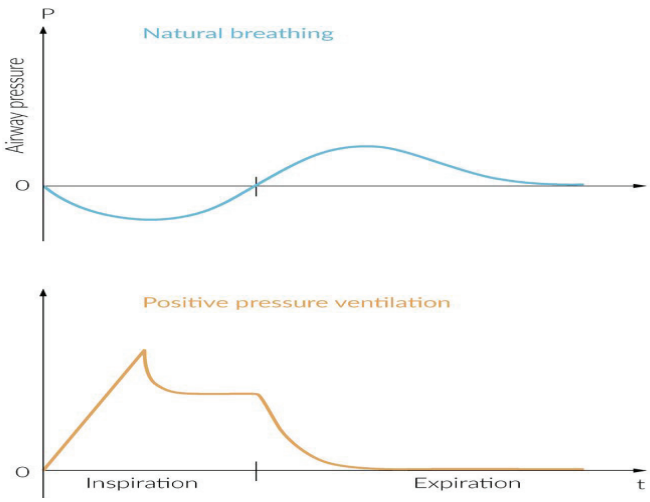

Diagram 1 shows the pressure changes during spontaneous breathing compared with positive-pressure ventilation.

Among positive-pressure ventilation modes, BIPAP and APRV have gained particular attention, both aimed at improving oxygenation and reducing ventilation-induced lung injury (VILI). However, their hemodynamic effects vary significantly with mean airway pressure, inspiratory-to-expiratory ratio, and ventilator–patient interaction.[17]

1. BIPAP (Bilevel Positive Airway Pressure) [18] is a type of non-invasive ventilation that provides positive airway pressure during the respiratory cycle, supporting spontaneous breathing. It is defined by a biphasic switch between two positive pressures.

The frequency of alternation depends on the respiratory rate. It improves the ventilation-to-perfusion

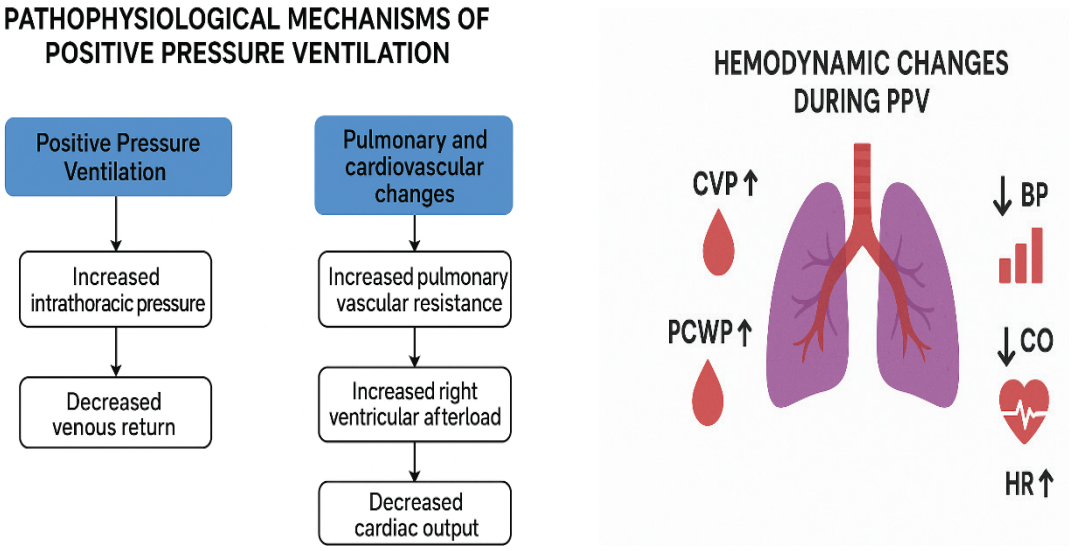

Figure 5. Schematic representation of pathophysiological mechanisms and hemodynamic parameters during positive pressure ventilation (PPV)

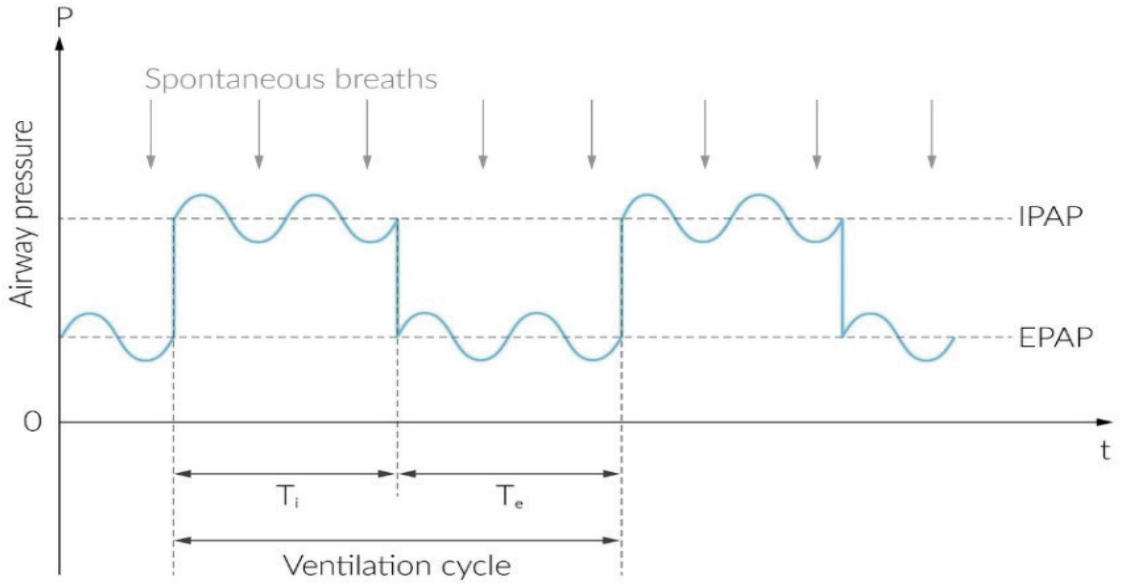

Figure 6. Alveolar pressure and its differences during BIPAP ventilation

(V/Q) ratio, increasing PO2 and decreasing PCO2, thereby improving alveolar ventilation, particularly in patients with hypercapnia, such as those with COPD or obese patients with hypoventilation syndrome.

As a procedure, it creates a pressure gradient between the inspiratory and expiratory phases, which involves two parameters:

1.1) ***IPAP (inspiratory positive airway pressure)*** - which indicates the level of positive pressure applied during the inspiratory phase. The application of positive pressure increases transpulmonary pressure and, in turn, enhances the effectiveness of alveolar ventilation by increasing tidal volume and reducing the mechanical work of breathing. The device assists the respiratory muscles, thereby improving the efficiency of CO2 elimination.[19].

1.2) ***EPAP*** (Expiratory Positive Airway Pressure) – is the positive pressure maintained during the expiratory phase in noninvasive ventilation with BIPAP. This is equivalent to PEEP in invasive mechanical ventilation. The main goal of this method is to prevent alveolar collapse during expiration and keep the alveoli open, because, as such, it maintains a positive basal pressure, increases residual capacity, and increases transpulmonary pressures by reducing the perfusion areas that do not have ventilation and, in this form, maintains arterial oxygenation (PaO2) [20].

Hemodynamic effects of BIPAP*:* BiPAP increases intrathoracic pressure, thereby decreasing preload and venous return. This effect helps patients with congestive heart failure by lowering left ventricular end-diastolic pressure and pulmonary capillary wedge pressure, improving oxygenation, and reducing pulmonary edema. Positive pressure increases alveolar ventilation, residual capacity, and end-expiratory pressure, decreasing left ventricular afterload and improving systemic perfusion. It also reduces respiratory muscle work and O_2 consumption. However, in patients with hypovolemia or left ventricular systolic dysfunction, cardiac output may decrease and cause hypotension, or in severe cases, cardiogenic shock. [21, 22].

*2. Airway Pressure Release Ventilation (****APRV****)* – is a positive pressure ventilation modality characterized by a high pressure for a long time, throughout the inspiratory phase that increases functional residual capacity, improves oxygenation, opens partial or complete atelectasis alveoli, and a short fraction during the respiratory cycle with low pressure that aims to exhale CO2 to avoid alveolar collapse, all this occurs when the patient is breathing spontaneously. [23]

Prolonged exposure to high pressure keeps the alveoli open, improves oxygenation, decreases intrapulmonary shunting, and reduces hypoxemia. The patient's spontaneous breathing during the procedure and the opportunity it provides to take a breath reduce mechanical respiratory fatigue. Also, CO2 expiration for a short time prevents possible barotrauma. [24].

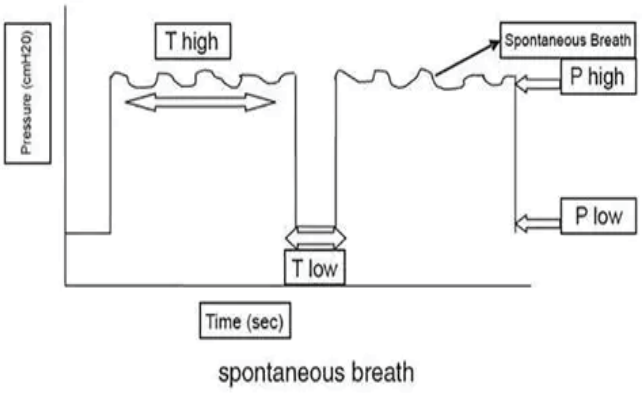

Figure 7. APRV
(source: https://www.researchgate.net/figure/An-airway-pressure-release-ventilation-waveform-showing-the-pressure-and-time_fig2_339048990)

***Hemodynamic effects of APRV**:* Like BIPAP, APRV also affects hemodynamics, but unfortunately, the effects are significantly more severe than with ARPV. This is due to the sustained, high positive pressure during most of the respiratory cycle. This persistent positive intrathoracic pressure significantly reduces venous return compared to BIPAP. In the case of a failing heart or in cases of shock, this reduces stroke volume and CO, giving severe hypotension that can result in cardiogenic shock. With ARPV, the CVP is significantly elevated. [22, 23]

The positive hemodynamic effect of ARPV may be beneficial in acute pulmonary edema from congestive heart failure, improving stroke volume more than BiPAP, because a more prolonged intrathoracic pressure reduces transmural pressure (gradient) more, thereby reducing afterload and improving ejection fraction.[22] Before choosing this ventilation modality, normovolemia and the hemodynamic effects of the anesthetics used must be carefully analyzed. [20, 21, 22, 23]

Discussions

BiPAP and APRV are two positive-pressure ventilation modalities that aim to improve gas exchange and reduce the work of breathing. However, they differ significantly in mechanism, mode of application, and clinical use (see Table 1).

BiPAP is a noninvasive ventilation for hypercapnic respiratory failure (e.g., moderate ARDS or cardiogenic pulmonary edema). It improves respiratory mechanics, reduces oxygen consumption, and improves alveolar ventilation, avoiding intubation. Its efficacy is limited to severe hypoxemia. Hemodynamically, BiPAP reduces left ventricular afterload and pulmonary capillary wedge pressure, increases intrathoracic pressure, reduces preload, and improves EF.

In patients with hypovolemia or ventricular dysfunction, it may cause decreased cardiac output and hypotension, but the risk is lower than with APRV. CVP may increase

Variable	BiPAP	APRV
Mean intrathoracic pressure	Moderate, increases only during the inspiratory phase (IPAP)	High and sustained during most of the respiratory cycle
Preload	Moderately reduced	Markedly reduced due to prolonged high pressure
Left ventricle afterload	Decreased (improves ejection)	Further decreased due to sustained thoracic pressure (marked improvement in ventricular ejection)
Cardiac output (CO)	Slightly reduced or stable in normovolemic patients	Significant drop in CO, especially in hypovolemia or cardiac dysfunction
Mean arterial pressure (MAP)	Slight to moderate decrease	Pronounced decrease, with risk of hypotension
Central venous pressure (CVP)	Slight increase	Marked increase (“false high”) from intrathoracic pressure
PCWP (Pulmonary Capillary Wedge Pressure)	Slightly elevated or normal	Significantly elevated (due to thoracic pressure, not volume)
Pulmonary vascular resistance (PV**R)**	Slightly increased in early phases, stabilizes with oxygenation	Initially markedly increased, then decreases as atelectatic alveoli open
Effect on oxygenation	Significantly improves oxygenation and alveolar ventilation	Maximizes oxygenation, reduces alveolar shunt
Risk of hypotension	Low-moderate	High
Clinical suitability	Favorable for patients with CHF and moderate ARDS	Suitable for severe ARDS with refractory hypoxemia
Effect on spontaneous breathing	Preserved throughout the respiratory cycle	Allowed, but with restrictions from sustained high pressure
Risk of right ventricle disfunction	Minimal in normovolemic patients	High due to increased RV afterload and reduced preload

Table 1. Comparison of hemodynamic effects between BiPAP and APRV [25, 26]

slightly; The risk of right ventricular dysfunction is minimal in normovolemic patients.

APRV is an invasive modality that maintains a continuous high pressure with brief releases to a low pressure, opening atelectasis and maximizing oxygenation. It is beneficial in severe ARDS with refractory hypoxemia. Allowing spontaneous breathing improves the ventilation-perfusion ratio and reduces the need for deep sedation.

Hemodynamically, APRV increases left ventricular transmural pressures, decreases afterload and capillary pressures, increases EF, and helps treat acute pulmonary edema. There is a more pronounced decrease in MAP and a greater risk of hypotension, especially in ventricular dysfunction and hypovolemia. Preload is decreased, CVP may be increased by intrathoracic pressure, and PCWP may be falsely elevated.

In the study by *Taha et al. (2014) [27],* the use of APRV ventilation mode was evaluated in patients with ARDS and cardiogenic shock. Main results:

- Cardiac index increased from 2.1 ± 0.3 L/min/m² to 3.0 ± 0.4 L/min/m² after switching to APRV
- Central venous pressure decreased from 15.2 ± 3.4 mmHg to 11.3 ± 2.8 mmHg ($p < 0.05$).
- Middle airway pressure (Paw): Increased from 13.4 ± 2.1 cmH_2O to 18.7 ± 2.5 cmH_2O ($p < 0.01$).
- Reduction in the use of hemodynamic support medications after switching to APRV.
- Improvement in arterial oxygenation after switching to APRV, FiO2/PaO2 ratio with values of 180±30mmHgAlthough mean airway pressure increased, CVP decreased due to improved cardiac output.

In the study by *Kaplan et al. (2001) [28]* on the hemodynamic effects of APRV ventilation (after switching to APRV) in patients with acute lung injury (ALI) or ARDS, results show that:

- Cardiac index (CI): Increase from 3.2 ± 0.4 L/min/m² to 4.6 ± 0.3 L/min/m²
- Central venous pressure (CVP): Decrease from 18 ± 4 mmHg to 12 ± 4 mmHg.
- Mean airway pressure (Paw): Decrease from 38 ± 3 cmH_2O to 25 ± 3 cmH_2O.
- Mean airway pressure (Paw): Decrease from 18 ± 3 cmH_2O to 12 ± 2 cmH_2O.
- Use of hemodynamic support medications: Reduction in the use of hemodynamic support medications after switching to APRV.

This study suggests that switching to APRV mode may improve hemodynamic stability in patients with ALI/ARDS by increasing cardiac index, decreasing central venous pressure and mean airway pressure, and reducing the need for hemodynamic support medications.

Conclusions:

Bilevel Positive Airway Pressure is suitable for moderate acute respiratory distress syndrome and hypercapnic respiratory failure, offering favorable hemodynamics and noninvasive support. Airway Pressure Release Ventilation is preferred in severe acute respiratory distress syndrome with refractory hypoxemia, as it provides superior oxygenation and alveolar recruitment but requires careful hemodynamic monitoring. The choice of modality should balance respiratory benefits with cardiovascular risks.

In conclusion, the choice between BiPAP and APRV should be individualized based on the type of respiratory failure, the degree of hypoxemia, and the clinical team's monitoring capacity.

Abbreviations

BiPAP - Bilevel Positive Airway Pressure; APRV - Airway Pressure Release Ventilation; ARDS - Acute Respiratory Distress Syndrome; DAD - Diffuse Alveolar Damage; PCWP - Pulmonary Capillary Wedge Pressure; TRALI - Transfusion-Related Acute Lung Injury; TRACO - Transfusion-Related Acute Circulatory Overload; ABG - Arterial Blood Gas; RSI - Rapid Sequence Intubation; V/Q - Ventilation-Perfusion; PPV - Positive Pressure Mechanical Ventilation; SBP - Systolic Arterial Pressure; MAP - Mean Arterial Pressure; DBP - Diastolic Pressure; CO - Cardiac Output; CVP - Central Venous Pressure; VILI - Ventilation-Induced Lung Injury; MAP - Mean Arterial Pressure; PVR - Pulmonary vascular resistance; CI - Cardiac Index; Paw - Mean airway pressure; PEEP - Positive End-Expiratory Pressure; CBC - Complete Blood Count.

Ethical Considerations*:* Since this study is a systematic review, no direct human or animal subjects were involved, and ethical approval was not required**.** However, the review was conducted following PRISMA guidelines to ensure transparency and reliability in reporting the hemodynamic effects of BiPAP and APRV in patients with ARDS.

COI Statement: This paper has not yet been submitted in parallel, presented fully or partially at a meeting, podium, or congress, published, or submitted for consideration elsewhere. This research received no specific funding from public, commercial, or non-profit sectors. The authors declare that they, their relatives, or next of kin have no financial relationships with external companies that could be considered relevant or minor.

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