Medicina Intensiva xxx (xxxx) 502201

medicina intensiva

http://www.medintensiva.org/en/

POINT OF VIEW

Is hemodynamic instability an absolute contraindication for prone position?

¿Es la inestabilidad hemodinámica una contraindicación absoluta para la posición prono?

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Received 15 January 2025; accepted 28 February 2025

Acute respiratory distress syndrome (ARDS) has a high mortality rate, ranging from 35% up to 45%.[1] The prone position (PP), an effective strategy for the treatment of the most severe cases of this syndrome, has shown its benefits in terms of gas exchange, optimization of protective lung mechanisms against mechanical ventilation (MV) injury, and its impact on survival.[2] Although some contraindications have been described for placing a patient in PP, most of them are not absolute (except for patients with spinal cord trauma with unstable spine and those with unstable pelvis), so patients can adopt the PP with minimal risks. Hemodynamic failure in these patients is often a limiting factor for PP and is the subject of this article.

The incidence rate of circulatory failure in ARDS varies between 50% and 70% of cases, and its presence is considered an independent variable contributing to mortality. The need for high doses of inotropes and vasoconstrictors should be particularly cautious when changing position, as the deterioration of hemodynamic stability and the risk of cardiac arrest in this context can be particularly difficult to manage in patients in PP.[3] Studies that evaluated the impact of PP in patients with moderate-to-severe ARDS[2] excluded those with a mean arterial pressure (MAP) < 65 mmHg. However, in the PROSEVA study, 72% of the patients were on vasopressors at the time of inclusion and remained hemodynamically stable. In these cases, the hemodynamic status not only did not worsen but tended to improve.

The etiopathogenesis of circulatory failure in ARDS can be attributed to various variables such as systemic inflammatory response syndrome (SIRS) and sepsis, which trigger vasodilatory mechanisms, reducing tissue perfusion and MAP. Additionally, myocardial depression, also associated with sepsis or SIRS, can contribute to circulatory dysfunction. Acute cor pulmonale (ACP) is one of the complications associated with ARDS, with incidence rates ranging from 25% up to 30% in ARDS patients.[4] This right ventricular failure is related to multiple factors, which will be described below:

- *Hypoxic vasoconstriction*, a homeostatic defense mechanism that activates during severe hypoxemia, allows balancing the ventilation-perfusion ratio in the lungs. This mechanism ensures that lung units with less ventilation receive reduced perfusion, thus decreasing both true shunt and the shunt effect (venous admission). The size of this vasoconstriction in the pulmonary circulation

DOI of original article:
https://doi.org/10.1016/j.medin.2025.502201
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https://doi.org/10.1016/j.medine.2025.502201

Please cite this article as: M. Busico and F. Villarejo, Is hemodynamic instability an absolute contraindication for prone position? Medicina Intensiva, https://doi.org/10.1016/j.medine.2025.502201

Table 1 Progression of internal environment, ventilatory and inotropic support.

Day	Hour	Position	pH	$PaCO_2$	PaO_2	HCO_3^-	FiO_2	$PaFiO_2$	LA	RSC	Vent. Volume	NE μg/kg/min	Vaso. IU/min
1	4 pm	S	7.15	41	132	14	0.6	220	86.1	46	16.1	5.06	
1	7 pm	S	7.11	31	90	9.6	0.6	150	97.6		5.06	0.05	
2	6 am	S	7.12	30.5	100	9.8	1	100	129.9	36	17.1	5.3	0.1
2	10 am	S	7.17	31.8	73.1	11.3	1	73	85.6		5.3	0.1	
2	2 pm	P	7.24	33	84	14	1	84	59.4	37	16.4	3.4	0.06
2	9 pm	P	7.36	31	99	17	1	99	36.1		2.4	0.05	
3	6 am	P	7.42	28.9	141	18.4	1	142	34.3	37	14.1	1.06	0.05
3	1 pm	P	7.43	30	117	20	0.7	167	31		1.06	0.05	
4	6 am	P	7.41	33.5	86	20.6	0.5	172	26.2	44	10.3	0.32	0.05
4	2 pm	S	7.46	28	82	19	0.6	137			0.21	0.05	
5	6 am	S	7.38	39	87	23	0.6	145	16.1	49	7.8	0.21	0
6	2 pm	S	7.3	41.5	120	19.7	0.6	200	9	53	8.5	0.26	0
7	6 am	S	7.44	44.9	72	29.8	0.6	120	21.7	58	8.3	0.04	0
8	6 am	S	7.43	41.1	80	26.9	0.5	160	21.3	53	8.4	0.04	0
9	6 am	S	7.45	41.4	91	28.2	0.5	182	20.9	56	9.2	0	0
10	6 am	S	7.46	37	101	25.8	0.4	253	28	55	10	0	0

Evolution of laboratory values, ventilatory and inotropic support from day 1 up to day 10 at different times. The patient remained in the supine position (S) on day 1, in the prone position (PP) from day 2 at 1 pm until day 4 at 1 pm, at which point they returned to the supine position. The progression of internal environment, programmed respiratory minute volume on the ventilator, RSC, and daily doses of inotropic drugs used in continuous infusion are presented.
LA: Lactic acid in mg/dL; RSC: Respiratory system compliance esimated as expired tidal volume/plateau pressure - total PEEP; FiO_2: Fraction of inspired oxygen; HCO_3: Bicarbonate; NE: Norepinephrine; $PaFiO_2$: Arterial oxygen pressure in relation to the inspired oxygen fraction; PaO_2: Arterial oxygen pressure; $PaCO_2$: Arterial carbon dioxide pressure; Vaso.: Vasopressin; Vent. Volume: Ventilator minute volume.

can significantly increase the right ventricular (RV), which increases the risk of acute right ventricular failure.
- MV-induced *positive intrathoracic pressure* reduces preload and increases the RV afterload. This effect is more pronounced as the levels of mean airway pressure increase (such as in the use of high levels of PEEP, plateau pressure $\geq$ 30 cmH_2O, and driving pressure > 18 cmH_2O). This phenomenon can significantly contribute to the development of ACP.
- ACP can cause *interventricular septum* deviation to the left, thus reducing the compliance of the left ventricle (LV) and elevating the LV end-diastolic pressure. This increases pulmonary capillary wedge pressure, favoring the passage of fluid into the alveolus, inducing hypoxemia and increasing pulmonary vascular resistance (PVR), which increases the RV afterload and creates a harmful vicious cycle. In ARDS, alveolar and capillary damage, along with pulmonary inflammation, promotes vascular remodeling and muscular hypertrophy, contributing to the higher prevalence of ACP.
- Right ventricular overload can reopen the patent foramen ovale, creating intracardiac shunt and worsening hypoxemia. This mechanism is less frequent.
- Areas with *atelectasis* induce right ventricular overload as they decrease the diameter of extraalveolar pulmonary vessels.
- *Alveolar overdistention due to elevated PEEP settings* can damage healthy lung units, increase the ventilation-perfusion ratio (dead space effect) through capillary compression, and raise the risk of ACP. Mekontso et al.[5] propose that the effect of PEEP on hemodynamics and RV function depends on the balance achieved between alveolar recruitment and unwanted overdistention.
- Finally, *hypercapnia*, induced either by lung protective protocols or by the dead space effect may contribute to increased PVR, as CO2 acts as a potent pulmonary vasoconstrictor.

When the patient adopts the PP, dependent alveolar units experience greater recruitment, with no significant change in perfusion, which remains unchanged in the dependent zone due to the fractal distribution of perfusion.[6] This improves oxygenation, CO2 elimination, and reduces airway pressures. In patients with moderate ARDS and ACP in the supine position, Jozwiak et al.[7] demonstrated that PP reduced PVR in all cases, decreased heart rate, and increased cardiac index by 50%. Other studies on position change showed no significant hemodynamic variations,[8] although some observed improvements in the RV function.[9,10]

PP reduces thoraco-abdomino-pelvic compliance, decreasing venous return and, consequently, the RV preload. Although the increase in intra-abdominal pressure (IAP) has been considered a cause of hypotension when placing a patient in PP, several studies[11] indicate that IAP rarely exceeds 15 mmHg.

As an example, we present data from a severe ARDS case (PaO2/FiO2 < 100) with high inotropic support requirements (Table 1) due to a near-fatal anaphylactic shock. The use of prone positioning, in this case, was associated with improvement in both gas exchange and hemodynamic status (Fig. 1) without complications.

In conclusion, for a patient with severe ARDS and refractory hypoxemia, there is a range of available interventions[12]; those described as traditional, such as protective MV, individualized PEEP titration, PP, and conservative fluid treatment,

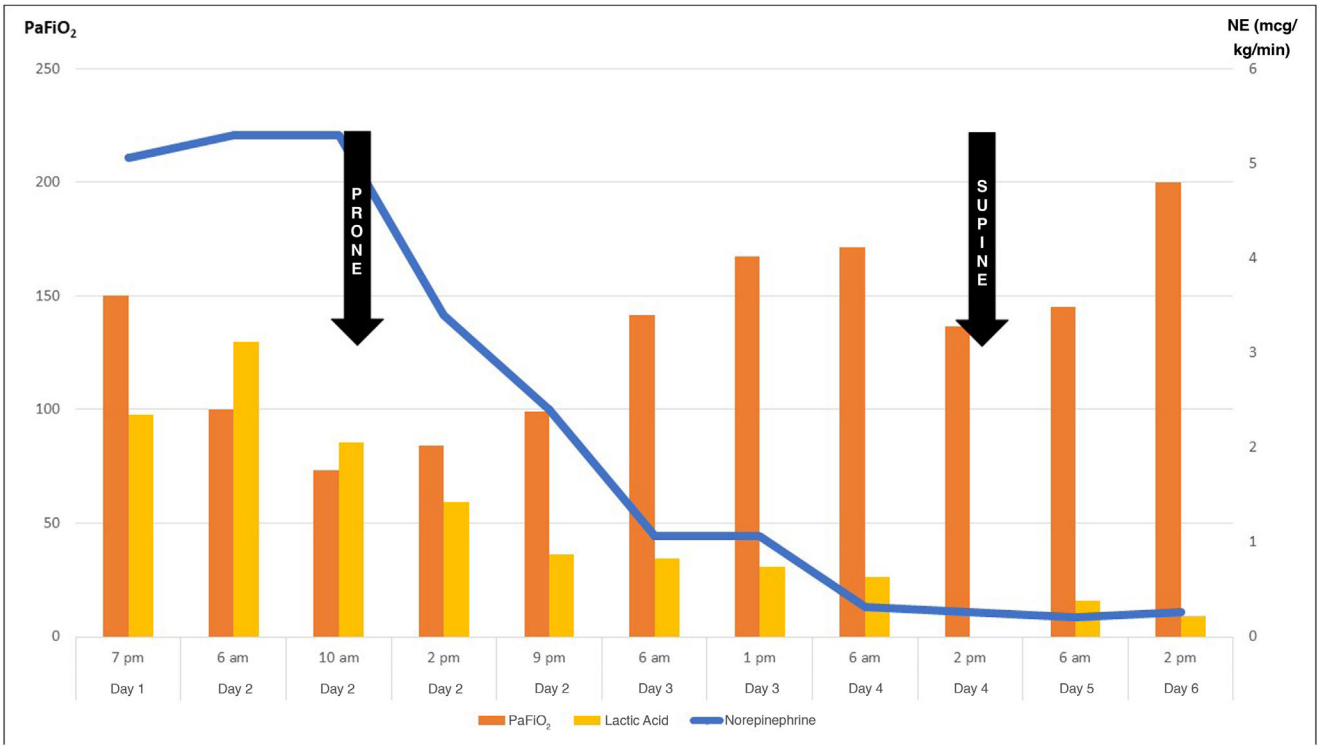

Figure 1 Relationship between PaFiO2 and norepinephrine requirement in the prone position.
NE: norepinephrine dose in μg/kg/min.

and ädjuvantẗherapies, such as pulmonary vasodilators, corticosteroids, and, in selected cases, extracorporeal membrane oxygenation. Specifically, PP improves oxygenation, increases CO2 elimination for the same minute ventilation, and reduces respiratory system pressures by homogenizing transpulmonary pressures. These effects are beneficial in hemodynamic terms, particularly in cases of RV overload. The association of effects on the respiratory and cardiovascular systems would be responsible for the beneficial impact of PP. As stated, hemodynamic status is not considered an absolute contraindication for placing patients in PP. However, it is essential to evaluate whether the benefits of the maneuver outweigh the associated risks, considering the balance between advantages and potential complications. The experience of the medical team and close monitoring are crucial due to the risk of serious complications that threaten life.

CRediT authorship contribution statement

All authors have contributed substantially to the study conception and design, data acquisition, data analysis and interpretation, article drafting, and approved its final version.

Ethical considerations

The research was conducted ethically in full compliance with the Declaration of Helsinki of the World Medical Association. The patient gave his informed consent for the publication of data.

Declaration of Generative AI and AI-assisted technologies in the writing process

No artificial intelligence was used for the design, development, or writing of the study.

Funding

None declared.

Declaration of competing interest

None declared.

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