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ASSESSMENT OF THE RESPONSE TO FLUID LOADING: THE ROLE OF STROKE VOLUME VARIATION AND PULSE PRESSURE VARIATION

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Relevance of the topic:

Fluid therapy remains one of the key components in the management of patients in intensive care units, especially in cases of shock and acute circulatory failure. At the same time, both inadequate and excessive fluid administration are associated with unfavorable outcomes: hypovolemia maintains tissue hypoperfusion, whereas fluid overload contributes to the development of interstitial edema, deterioration of oxygenation, and increased mortality. In this regard, accurate and timely assessment of the response to fluid loading (fluid responsiveness) is of critical importance for individualizing therapy.

Traditionally used static preload indicators (central venous pressure, pulmonary artery occlusion pressure) have demonstrated limited predictive value in determining the effect of fluid administration, as they do not reflect the functional reserve of the cardiovascular system. This has led to a shift toward the use of dynamic parameters based on cardiopulmonary interactions, such as stroke volume variation (SVV) and pulse pressure variation (PPV), which in several studies have shown higher sensitivity and specificity in predicting fluid responsiveness.

Despite the widespread implementation of SVV and PPV in clinical practice, their use is associated with a number of limitations, including dependence on mechanical ventilation parameters, heart rhythm, and spontaneous breathing. In addition, the extent to which the real-time use of these indicators influences clinical outcomes, including length of stay in the intensive care unit, incidence of organ dysfunction, and mortality, remains insufficiently studied.

Thus, further investigation of the role of stroke volume variation and pulse pressure variation in assessing the response to fluid loading, as well as their comparison with clinical and perfusion parameters, is an important task in modern intensive care and critical care medicine.

Keywords: fluid therapy; fluid responsiveness; stroke volume variation (SVV); pulse pressure variation (PPV); dynamic preload indicators; hemodynamic

monitoring; critical conditions; shock; intensive care; mechanical ventilation; tissue perfusion; cardiac output.

Introduction:

Optimization of fluid therapy is one of the central goals of intensive care medicine, as adequate restoration of intravascular volume directly affects tissue perfusion and clinical outcomes in critically ill patients. In conditions of shock and acute circulatory failure, timely correction of hypovolemia helps prevent the progression of organ dysfunction, whereas excessive fluid administration is associated with the development of tissue edema, impaired gas exchange, and increased mortality. This highlights the need for accurate assessment of an individual patient's response to fluid loading.

In clinical practice, static preload indicators such as central venous pressure and pulmonary artery occlusion pressure have long been used. However, accumulated evidence demonstrates their low predictive value for fluid responsiveness, which is related to their inability to reflect the functional reserves of the cardiovascular system and the complex interactions between the heart and lungs.

In this context, particular attention is paid to dynamic parameters based on variations in hemodynamic variables in response to the respiratory cycle, primarily stroke volume variation (SVV) and pulse pressure variation (PPV). These indices make it possible to assess the dependence of cardiac output on preload and, consequently, the likelihood of an increase in cardiac output in response to fluid administration. Several studies have shown that SVV and PPV have higher diagnostic accuracy compared to static parameters, especially in patients undergoing controlled mechanical ventilation.

However, the use of dynamic indices in real clinical practice is limited by several factors, including the presence of arrhythmias, spontaneous breathing, low tidal volumes, and changes in chest wall compliance. In addition, the impact of using these parameters on clinical outcomes and their relationship with markers of tissue perfusion remains a matter of debate.

In this regard, the aim of the present study is to evaluate the role of stroke volume variation and pulse pressure variation in predicting the response to fluid loading in patients in the intensive care unit, as well as to analyze their relationship with systemic hemodynamic parameters and tissue perfusion indicators.

Materials and Methods

Study design:

A prospective observational study was conducted in the intensive care unit of a

multidisciplinary hospital. The study included patients with signs of acute circulatory failure requiring fluid therapy for hemodynamic optimization.

Inclusion criteria:

Age ≥18 years; presence of clinical signs of hypoperfusion (arterial hypotension, oliguria, elevated lactate levels); indication for fluid loading as determined by the attending physician; and use of invasive hemodynamic monitoring.

Exclusion criteria:

Cardiac rhythm disturbances (atrial fibrillation and other significant arrhythmias); spontaneous breathing; low tidal volume (<6 mL/kg of predicted body weight); severe right ventricular failure; and pregnancy.

All patients were on controlled mechanical ventilation with fixed respiratory parameters (tidal volume 6–8 mL/kg, no spontaneous breathing efforts). Hemodynamic monitoring was performed using an invasive arterial catheter and monitoring systems capable of assessing pulse pressure variation (PPV), stroke volume variation (SVV), and cardiac output.

Fluid loading was administered as a bolus infusion of crystalloid solution in a volume of 250–500 mL over 10–15 minutes. Hemodynamic measurements were recorded before the infusion and immediately after its completion. Additionally, mean arterial pressure (MAP), heart rate, lactate levels, and urine output were recorded.

Patients were classified as "responders" if stroke volume or cardiac output increased by ≥10–15% following fluid loading. Otherwise, patients were classified as "non-responders."

Statistical analysis included assessment of data distribution, group comparisons using parametric and non-parametric methods, and construction of ROC curves to determine the diagnostic accuracy of SVV and PPV in predicting fluid responsiveness. A p-value <0.05 was considered statistically significant.

The study was conducted in accordance with ethical principles, and all patients or their legal representatives provided informed consent for participation.

Results

A total of 82 patients meeting the inclusion criteria were enrolled in the study. Based on the response to fluid loading, 46 patients (56%) were classified as responders, while 36 patients (44%) were classified as non-responders.

Baseline values of stroke volume variation (SVV) and pulse pressure variation (PPV) were significantly higher in the responder group compared to non-responders. The median SVV in responders was 14% (IQR 12–18%) versus 8% (IQR 6–10%) in non-responders ($p<0.001$). Similarly, PPV was 15% (IQR 13–19%) in responders and 9% (IQR 7–11%) in non-responders ($p<0.001$).

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Table 1. Patient characteristics

Parameter	Responders (n=46)	Non-responders (n=36)	p-value
Age, years	62 (54–70)	64 (56–72)	0.41
Sex (M/F)	28/18	20/16	0.83
MAP, mmHg	68 (65–72)	70 (66–74)	0.18
HR, beats/min	96 (88–104)	92 (85–101)	0.22
Lactate, mmol/L	3.1 (2.4–4.2)	2.8 (2.1–3.9)	0.27

Following fluid loading, responders demonstrated a significant increase in stroke volume (by 18% [15–24%], p<0.001) and cardiac output (by 16% [12–21%], p<0.001), whereas changes in the non-responder group were not significant. No significant differences in mean arterial pressure dynamics were observed between the groups.

ROC analysis demonstrated high diagnostic accuracy of dynamic preload indicators. The area under the curve (AUC) for SVV was 0.87 (95% CI 0.79–0.94), and for PPV it was 0.85 (95% CI 0.77–0.93). Optimal cutoff values were ≥12% for SVV (sensitivity 82%, specificity 78%) and ≥13% for PPV (sensitivity 80%, specificity 75%).

Table 2. Baseline hemodynamic parameters

Parameter	Responders	Non-responders	p-value
SVV, %	14 (12–18)	8 (6–10)	<0.001
PPV, %	15 (13–19)	9 (7–11)	<0.001
Cardiac output, L/min	4.2 (3.6–4.9)	4.4 (3.8–5.0)	0.35
CVP, mmHg	8 (6–10)	9 (7–11)	0.29

Table 3. Changes in parameters after fluid infusion

Parameter	Responders	Non-responders	p-value
ΔSV, %	+18 (15–24)	+4 (2–6)	<0.001
ΔCO, %	+16 (12–21)	+3 (1–5)	<0.001
ΔMAP, mmHg	+6 (3–9)	+3 (1–5)	0.07
ΔLactate, mmol/L	−0.8 (−1.2 to −0.4)	−0.2 (−0.5 to 0)	0.01

Correlation analysis revealed a moderate positive correlation between baseline SVV values and changes in lactate levels after fluid administration (r = 0.42; p = 0.003), suggesting a relationship between macrocirculatory response and improvement in tissue perfusion. In contrast, baseline central venous pressure did not demonstrate significant predictive value for fluid responsiveness.

Thus, the obtained results confirm the high diagnostic value of stroke volume variation and pulse pressure variation as predictors of fluid responsiveness in patients undergoing controlled mechanical ventilation.

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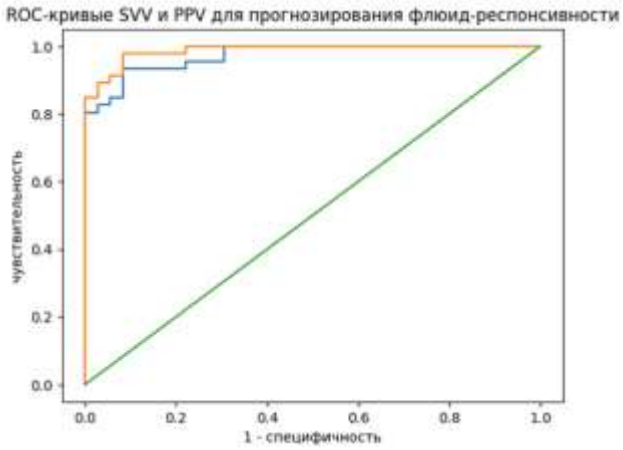

Figure 1. ROC curves of stroke volume variation (SVV) and pulse pressure variation (PPV) in predicting fluid responsiveness

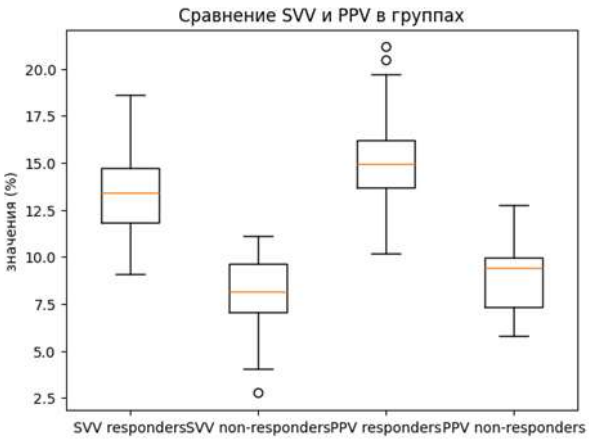

Figure 2. Comparison of SVV and PPV values in responders and non-responders

Figure 1 presents the ROC curves for SVV and PPV, reflecting their diagnostic value in predicting the response to fluid loading in critically ill patients. Both parameters demonstrated high predictive accuracy. The area under the curve (AUC) was 0.87 for SVV and 0.85 for PPV, indicating comparable and high diagnostic performance. These findings confirm the clinical relevance of dynamic preload indicators in the assessment of fluid responsiveness.

Figure 2 shows the distribution of SVV and PPV values in the responder and non-responder groups. Patients classified as responders demonstrated significantly higher SVV and PPV values compared to non-responders ($p<0.001$ for both parameters). These differences support the association between elevated dynamic preload indices and a favorable hemodynamic response to fluid therapy.

Conclusions

1. Dynamic preload parameters, including stroke volume variation (SVV) and pulse pressure variation (PPV), demonstrate high diagnostic value in predicting fluid responsiveness in critically ill patients undergoing controlled mechanical ventilation.
2. Baseline SVV and PPV values are significantly higher in patients who show a meaningful increase in cardiac output after fluid loading, confirming their functional relevance in assessing the dependence of cardiac output on preload.
3. The diagnostic accuracy of SVV and PPV is comparable, with both parameters showing high areas under the ROC curve, supporting their applicability in intensive care practice.
4. Static preload indicators (e.g., central venous pressure) do not reliably predict fluid responsiveness and are less informative than dynamic indices.

5. The use of dynamic preload parameters may contribute to more precise individualization of fluid therapy, reducing the risk of both hypovolemia and hypervolemia in intensive care patients.

6. Further studies are needed to evaluate the impact of SVV- and PPV-guided fluid therapy strategies on clinical outcomes, including organ dysfunction and mortality.

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