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Vasopressor Burden and Outcomes in Critically Ill Patients With Aortic Disease

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Backgroun

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Patients with acute and chronic aortic disease frequently require ICU admission due to:

- Hemodynamic instability
- Hemorrhage
- Cardiogenic shock
- Postoperative vasoplegia
- Multiorgan dysfunction

Vasopressors are commonly used to maintain perfusion in these settings.

In general critical care populations, vasopressor dose and duration are strongly associated with mortality.

However, in aortic disease specifically, where hemodynamic management is central to pathophysiology, the relationship between vasopressor exposure, cumulative burden, and outcomes has not been well characterized in large heterogeneous ICU cohorts.

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AORTIC MANAGEMENT GOALS

Goal: Reduce aortic wall stress to prevent rupture/propagation.

Strategy: Strict blood pressure and heart rate control (e.g., β-blockers).

Keep pressure down.

THE REALITY OF SHOCK

Problem: Patients often present with hemodynamic instability (hemorrhage, sepsis, pump failure).

Intervention: Vasopressors are required to maintain organ perfusion.

Drive pressure up.

The Conflict: Vasopressors increase afterload, directly increasing aortic wall tension (Laplace's Law).

Study

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What We Know

- In general ICU populations, higher vasopressor dose/duration predicts mortality.
- Aortic surgeons practice "permissive hypotension," but often require pressors.

The Gap

- The relationship between vasopressor burden and outcomes specifically in aortic disease is unknown.

Study Objective

- The association between vasopressor use and short-term mortality
- The relationship between cumulative vasopressor burden and outcomes
- Whether vasopressor use remains associated with hospital mortality after adjustment for early markers of illness severity

Methods

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Study Design: Retrospective cohort study using the Medical Information Mart for Intensive Care IV (MIMIC-IV) database (2008–2019).

Study Population

- N = 3,111 Adult ICU patients.
- Identified by ICD codes for Aortic Disease (dissection, aneurysm, rupture).
- First ICU admission per hospitalization.

Methods

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Exposure Definitions

Vasopressor exposure defined as receipt of any intravenous infusion of:

- Norepinephrine
- Epinephrine
- Dopamine
- Vasopressin
- Phenylephrine
- Dobutamine

Vasopressor burden quantified as total infusion duration (hours) during ICU stay.

Among exposed patients, burden was categorized by median duration (17.3 hours):

- Low burden
- High burden

Methods

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Primary outcomes

- ICU mortality
- Hospital mortality
- 30-day mortality

Secondary outcomes

- ICU length of stay (hours)
- Hospital length of stay (days)
- Statistical analysis: Mann–Whitney U tests for continuous variables, Chi-square tests for categorical variables, Multivariable logistic regression for hospital mortality

Adjusted covariates: Age, Maximum lactate within first 24 hours, Maximum creatinine within first 24 hours

Result

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Study Cohort

A total of 3,111 ICU patients with aortic disease were included.

- 1,303 (42%) received vasopressors
- 1,808 (58%) did not receive vasopressors

Median vasopressor duration among exposed patients: 17.3 hours [5.1–51.6]

Result

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Table 1. Baseline Characteristics by Vasopressor Use			
Variable	No Vasopressor (n=1808)	Any Vasopressor (n=1303)	p value
Age, years	73.0 [63.0–81.0]	70.0 [61.0–78.0]	<0.001
Male sex	1121 (62.0%)	886 (68.0%)	<0.001
Lactate max (24h), mmol/L	2.9 [1.5–18.7]	3.8 [2.3–9.0]	<0.001
Creatinine max (24h), mg/dL	1.1 [0.8–1.5]	1.1 [0.8–1.6]	0.035
ICU LOS, hours	44.0 [26.0–77.2]	74.0 [36.6–154.6]	<0.001
Hospital LOS, days	6.6 [4.0–10.8]	8.7 [5.7–14.2]	<0.001

Values are median [IQR] or n (%).

Vasopressor patients had higher early lactate.

ICU and hospital stays were significantly longer.

Creatinine differences were small despite statistical significance.

Result

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Table 2. Clinical Outcomes by Vasopressor Exposure			
Outcome	No Vasopressor (n=1808)	Any Vasopressor (n=1303)	p value
ICU mortality	53 (2.9%)	158 (12.1%)	<0.001
Hospital mortality	127 (7.0%)	189 (14.5%)	<0.001
30-day mortality	120 (6.6%)	180 (13.8%)	<0.001
ICU LOS, hours	44.0 [26.0–77.2]	74.0 [36.6–154.6]	<0.001
Hospital LOS, days	6.6 [4.0–10.8]	8.7 [5.7–14.2]	<0.001

Values are median [IQR] or n (%).

ICU mortality was more than fourfold higher in pressor-treated patients.

Hospital and 30-day mortality approximately doubled.

Resource utilization was significantly greater in the vasopressor group.

Result

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Table 3. Outcomes by Vasopressor Burden

Vasopressor Burden	N	ICU Mortality	Hospital Mortality	30-day Mortality	ICU LOS (hours)	Hospital LOS (days)
No pressors	1808	53 (2.9%)	127 (7.0%)	120 (6.6%)	44.0 [26.0–77.2]	6.6 [4.0–10.8]
Low burden	653	45 (6.9%)	52 (8.0%)	51 (7.8%)	48.3 [29.9–86.9]	7.0 [5.2–10.2]
High burden	650	113 (17.4%)	137 (21.1%)	129 (19.8%)	124.3 [65.5–246.9]	11.1 [7.1–19.2]

Mortality increased stepwise across burden categories.

ICU mortality rose from 2.9% (no pressor) to 17.4% (high burden).

High-burden patients had nearly threefold longer ICU stays.

Result

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Table 4. Multivariable Logistic Regression for Hospital Mortality (N = 2,241 complete cases)		
Variable	Odds Ratio (95% CI)	p value
Any vasopressor use	1.90 (1.43–2.52)	<0.001
Age (per year)	1.04 (1.03–1.06)	<0.001
Lactate max (24h)	1.00 (1.00–1.00)	<0.001
Creatinine max (24h)	1.00 (1.00–1.01)	0.074

Vasopressor use remained significantly associated with hospital mortality.

Age was independently associated with mortality.

Creatinine did not reach statistical significance.

Discussion

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Vasopressor use likely reflects greater illness severity, as patients requiring vasopressors are more likely to have shock, bleeding, postoperative complications, or malperfusion.

Second, prolonged vasopressor dependence may indicate persistent hemodynamic instability, which itself is associated with worse outcomes.

Finally, from a physiologic standpoint, increased vascular tone and afterload may theoretically contribute to increased aortic wall stress, although our observational design cannot establish causality.

Therefore, vasopressor exposure in this population should likely be viewed primarily as a marker of severe hemodynamic compromise rather than a direct cause of mortality

Limitations

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This study has several limitations.

First, as a retrospective observational study, causality cannot be established.

Second, the cohort included heterogeneous aortic pathologies, which may have different hemodynamic profiles.

Third, vasopressor burden was measured using duration of exposure rather than dose, which may not fully capture vasopressor intensity.

Finally, residual confounding related to illness severity may remain despite adjustment

Way Forward

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Future research should aim to better characterize vasopressor exposure in aortic ICU patients by incorporating:

- dose-based vasopressor metrics
- disease-specific subgroups
- and more detailed severity-of-illness adjustment.

Prospective studies may help determine optimal hemodynamic management strategies in this unique population.

Vasopressor Burden and Outcomes in Critically Ill Patients with Aortic Disease: A MIMIC-IV Analysis

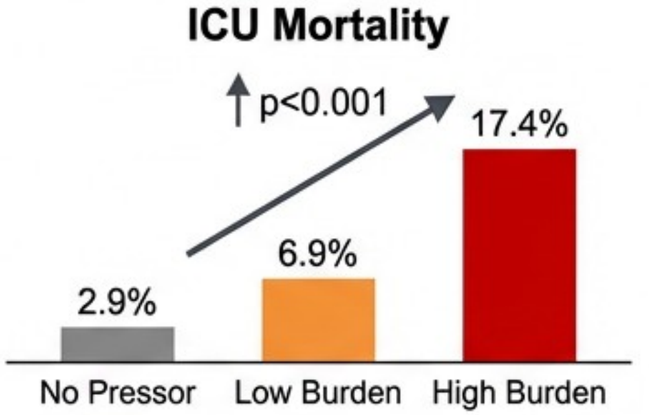
Study Cohort & Exposure

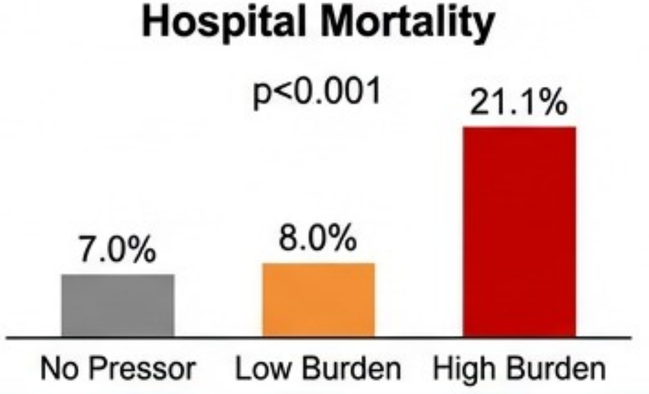
N=3,111 Critically Ill Aortic Disease Patients

Vasopressor Use (42%)

- Low Burden (<Median Duration)
- High Burden (>Median Duration)
- No Vasopressor (58%)

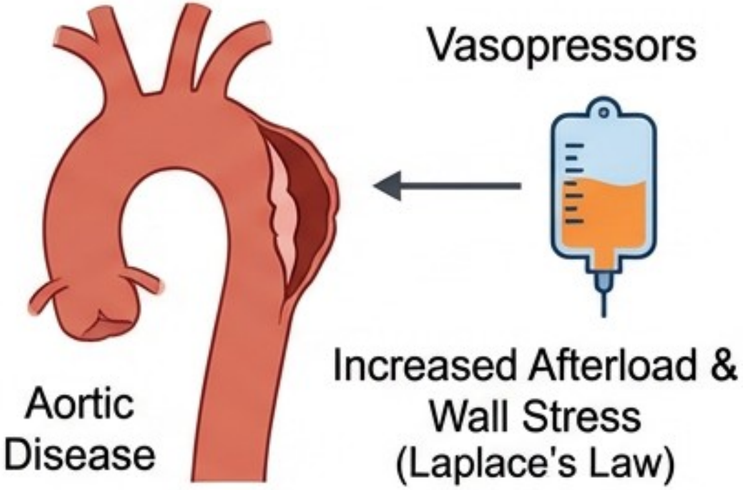
Key Outcomes: Graded Association with Mortality





Adjusted OR for Hospital Mortality (Any vs. None):
1.90 (95% CI 1.43–2.52), $p<0.001$

Mechanistic Insight & Conclusions



- Marker of Disease Severity & Potential Mediator of Harm
- Identify High-Risk Patients for Targeted Management & Closer Monitoring

The Houston Aortic Symposium 2026
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Thank You