

Original Research

Weight a Minute: assessing the efficacy and safety of weight-based vs. non-weight-based vasopressor dosing in critical shock patients

Heather Howell, PharmD¹, Patrick Leung, PharmD, BCPS, BCCCP, PhC¹, Adlina Chiang, RPh, MBA, MPS, BS-ICT, MHA¹, Ivan da La Rosa, PhD, LSW²

¹ Memorial Medical Center, ² New Mexico State University

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Background

Vasopressors are a commonly used medication class for patients suffering from hypotensive shock and requiring intervention to maintain their goal mean arterial pressure (MAP). With the increase in obesity rates at the United States, there are concerns that traditional non-weight-based (NWB) vasopressor dosing may result in underdosing and thus, reduced therapeutic efficacy. There are limited studies assessing the efficacy and safety of a weight-based (WB) dosing regimen.

Objective

The purpose of this study is to assess the efficacy and safety of implementing a WB vasopressor dosing protocol on critical shock patients according to actual body weight.

Methods

This was a single-center, retrospective cohort study that compared efficacy and safety parameters before and after implementing a WB vasopressor dosing protocol. Patient charts were analyzed for pre-intervention using NWB dosing from January 1, 2024 to June 30, 2024 and post-intervention using WB dosing from October 1, 2024 to March 31, 2025.

Results

This study found no significance in the time to achieve goal mean arterial pressure, number of concurrent vasopressors used, days spent on vasopressors, incidence of acute kidney injury, and incidence of extravasation. However, it showed a significant reduction in the length of time spent in the intensive care unit and hospital.

Conclusion

A WB vasopressor dosing protocol may allow a more individualized approach to treat hypotensive shock, with the potential to reduce length of stay without compromising the safety of using a WB regimen.

INTRODUCTION

Vasopressors are considered first-line medications for the treatment of refractory hypotension when fluid resuscitation fails in critically ill patients suffering from shock.^{1,2} Current strategies utilize either non-weight-based (NWB) dosing (mcg/min) or weight-based (WB) dosing (mcg/kg/min) for hypotensive shock, however no guidelines or consensus statements recommend an optimal dosing strategy, leading to further variability.

Several uncertainties exist for NWB dosing strategies, particularly the risk of underdosing overweight patients and overdosing underweight patients leading to decreased efficacy and safety of vasopressor administration.^{2,3} In con-

trast, WB strategies allow for individualized therapy according to patient's weight and clinical response with the ability to optimize outcomes.⁴ As of 2019, 33% of the United States population was categorized as obese.⁵ In fact, we have seen a gradual trend in the rate of obesity across the 21st century. In New Mexico, this obesity rate is matched with 35% and 34.6% of New Mexicans being overweight (BMI 25-29.9 kg/m²) and obese (BMI ≥30 kg/m²), respectively.⁶ This increasing prevalence of obesity may suggest a potential underdosing of vasopressors and not receiving the maximum therapeutic vasopressor dose.

Few studies have directly compared the efficacy and safety of using one dosing strategy over another. Of the studies conducted,^{3,7} several have focused on NWB versus WB efficacy in select categories, such as one vasopressor,

Table 1. Non-Weight Based versus Weight Based Vasopressor Dosing Protocols

	Non-Weight-Based	Weight-Based
Norepinephrine	- Initiate: 10 mcg/min - Titrate: 1 mcg/min every 5 min - Max: 30 mcg/min	- Initiate: 0.1 mcg/kg/min - Titrate: 0.03 mcg/kg/min every 5 min - Max: 1 mcg/kg/min
Epinephrine	- Initiate at 4 mcg/min - Titrate: 1 mcg/min every 5 min - Max: 20 mcg/min	- Initiate: 0.05 mcg/kg/min - Titrate: 0.02 mcg/kg/min every 5 min - Max: 0.5 mcg/kg/min
Phenylephrine	- Initiate: 40 mcg/min - Titrate: 10 mcg/min every 5 min - Max: 300 mcg/min	- Initiate: 0.5 mcg/kg/min - Titrate: 0.3 mcg/kg/min every 5 min - Max: 4 mcg/kg/min

one shock etiology, or obese patients as a selected subgroup. A recent study by DeMillard et al.⁸ compared a broad patient population across three intensive care units and evaluated norepinephrine, epinephrine, and phenylephrine dosed on an ideal body WB dosing protocol. Consistently, these studies have analyzed difference in time to achieve goal mean arterial pressure (MAP) between dosing strategies and have not found any significant difference. However, these studies have focused only on the efficacy of one protocol over another without evaluating the differences in safety profiles. Our study aims to assess both the efficacy and safety of using a WB dosing strategy on several shock etiologies, including septic, cardiogenic, hypovolemic, and mixed shock etiologies, as well as across several vasopressors.

METHODS

As of September 1, 2024, our institution began using a WB dosing protocol for norepinephrine, epinephrine, and phenylephrine according to actual body weight. Dosing of the WB protocol was defined by recommendations in the Surviving Sepsis Campaign.¹ Prior to this, our institution followed a NWB protocol (Table 1). The previous NWB protocol was phased out over the course of September whilst the new WB protocol was phased in. Effective October 1, 2024, all IV pumps were set to only allow the WB protocol to ensure 100% compliance to the new dosing regimen. We conducted a single-center, retrospective cohort study to evaluate the efficacy and safety of this change. The study was reviewed and approved by the study facility's Institutional Review Board.

Data were collected for patients admitted from January 1, 2024 to June 30, 2024 for the NWB group, and October 1, 2024 to March 31, 2025 for the WB group until 120 qualifying patients were enrolled in each treatment arm. Inclusion criteria included being at least 18 years of age, receiving at least 1 vasopressor, vasopressor initiation in the ED and/or ICU, admission to the intensive care unit (ICU), and a MAP goal between 60–65 mmHg. Patients were excluded if they were transferred to another institution or post-surgical patients. The study was conducted across our 20-bed critical care and 12-bed step-down critical care units.

The primary outcome was the time to achieve goal mean arterial pressure (MAP), defined by maintaining a MAP goal for at least 30 minutes. At this institution, MAPs are monitored and recorded at least every 15 minutes but may be as frequent as every 5 minutes when titrating vasopressors. MAPs are measured by both cuff and arterial pressure; arterial lines are routinely placed on intubated patients. Arterial pressures are recorded preferentially to cuff pressures, if present. All patients receiving vasopressors require central venous access. Vasopressor concentrations included norepinephrine and epinephrine 4 mg/250 ml, 8 mg/250 ml, and 16 mg/250 ml; phenylephrine 80 mg/250 ml and 160 mg/250 ml; and vasopressin 60 units/60 ml. Secondary efficacy outcomes included total number of vasopressors used, length of time on vasopressors, length of stay in the ICU and hospital, and cumulative average daily dose of vasopressors. Secondary safety outcomes included the incidence of acute kidney injury (AKI) and extravasation. AKI was defined as an increase in serum creatinine of greater than or equal to 0.3 mg/dl from baseline.⁹ AKIs upon admission were not considered in the incidence of AKI. Extravasation was identified by an order and documented administration of phentolamine. Data collected from the facility's electronic health record included blood pressure, MAP, goal MAP target, vasopressor doses, length of stay, sex, age, weight, height, time of vasopressor initiation, shock etiology, comorbidities, and mortality.

Due to non-normal distributions of primary and key secondary outcomes, non-parametric methods were employed where necessary. Effect sizes were calculated to provide a measure of the practical significance of statistically significant findings. The time to achieve goal MAP, number of vasopressor agents used, and length of stay in ICU and hospital were analyzed via Mann Whitney U tests. Cumulative vasopressor doses were analyzed using independent t-tests and one-way multivariate analysis of variance (MANOVA). Extravasation and AKI analyses were conducted via chi-square tests. An analysis was conducted to assess if mortality rates between the study populations are attributed to the difference in ICU length of stay via independent samples t-test. A 2×4 factorial ANOVA was conducted to examine the effects of dosing strategy and shock etiology on time to achieve goal mean arterial pressure. A sample size of 240 was needed to achieve a power of 80% in order to detect a change in MAP by 30 minutes in WB versus NWB

Table 2. Baseline Demographic and Clinical Characteristics by Treatment Group

Characteristic	Non-Weight-Based Group (N = 120)	Weight-Based Group (N = 120)	Test Statistic	P-Value
Demographics				
Age, years				
Mean $\pm$ SD	67.65 $\pm$ 14.74	65.82 $\pm$ 14.15	T = 0.96	.336
Median (IQR)	69.50 (59.75-78.00)	68.00 (58.00-76.00)		
Gender, n (%)				
			$\chi^2 = 1.08$	.299
Male	62 (51.7)	70 (58.3)		
Female	58 (48.3)	50 (41.7)		
Clinical Profile				
Comorbidities Present, n (%)	98 (81.7)	107 (89.2)	$\chi^2 = 2.14$	.143
Shock Etiology, n (%)				
			$\chi^2 = 8.12$	.044
Septic shock	71 (59.2)	55 (45.8)		
Cardiogenic shock	19 (15.8)	32 (26.7)		
Hypovolemic shock	18 (15.0)	26 (21.7)		
Combination	12 (10.0)	7 (5.8)		

Note. SD = standard deviation; IQR = interquartile range. "Combination" refers to patients with two or more shock etiologies.

protocols. All analyses were performed using SPSS version 18.0. All statistical tests were two-tailed with the critical alpha level set at .05.

RESULTS

A total of 845 adult patients were identified. 605 patients were excluded with post-surgical patients and transfer-outs being the primary reasons for exclusion. There were 120 patients included in each arm of the study. Baseline patient characteristics are shown in [Table 2](#). The age, gender, comorbidities, and mortality rate were not statistically different between treatment arms. The primary shock etiology showed significant differences despite the primary diagnoses being septic and cardiogenic shock in both arms. All measured outcomes are reported in [Table 3](#).

The primary outcome, time to achieve goal MAP, showed no statistically significant difference across treatment arms, with a median of 1.25 hours to reach goal MAP ($p = .632$). A subgroup analysis also showed no difference in time to achieve goal MAP when analyzed across shock etiology ($p = .763$) ([Table 4](#)). The median number of concurrent vasopressors used was identical between treatment arms ($p = .314$), indicating that most patients in both groups required only one vasopressor, with a similar proportion requiring additional agents. Similarly, there were no statistically significant differences in the types of specific vasopressors used between groups, with nearly identical rates of norepinephrine use (98.3% in both groups, $p = 1.000$), and no significant differences in the use of epinephrine (20.2% vs. 17.5%, $p = .598$), phenylephrine (20.8% vs. 16.7%, $p = .408$), or vasopressin (34.2% vs. 31.7%, $p = .545$).

The length of time spent on vasopressors was slightly shorter in the WB group (median 2 days) compared to the

NWB group (median 2.5 days), although the difference was not statistically significant ($p = .160$). The length-of-stay in the ICU and hospital revealed significantly shorter stays in the WB group, with a median ICU stay of 4 days versus 7 days in the NWB group ($p < .001$) and a median hospital stay of 8 days versus 11 days in the NWB group ($p = .001$). Mortality rates of WB and NWB groups were not significantly different ($p = .062$). There was no significant difference in LOS between patients who survived ($M = 6.40$ days) and died ($M = 6.79$ days) ($p = 0.729$) in the WB group and was overall less compared to the NWB group (survived: $M = 10.17$ days; died: $M = 9.79$ days) ([Table 4](#)).

The cumulative average dose of vasopressors per day showed a significant difference across the four vasopressor doses when considered simultaneously ($p < .001$). Follow-up univariate ANOVAs revealed that this multivariate effect was attributable to a significant difference in norepinephrine dosing. The WB group received significantly higher daily norepinephrine doses ($M = 16.43$ mg/day) compared to the NWB group ($M = 8.69$ mg/day), representing an 89% increase in daily exposure ($p < .001$). No statistically significant differences were observed for epinephrine, phenylephrine, or vasopressin when examined within the multivariate framework.

The safety outcomes revealed no significant difference in rate of AKI or extravasation. AKIs were identical in that they occurred in 48.3% of study participants in both treatment arms ($p = 1.00$). Extravasation was slightly higher in the WB group at 5%, versus 3.3% in the NWB group, but it is not statistically significant ($p = 0.518$).

Table 3. Primary and Secondary Outcomes

Measure	Non-Weight-Based Group (N = 120)	Weight-Based Group (N = 120)	Test Statistic	P-Value
Time to reach goal MAP (hours)				
Mean $\pm$ SD	1.95 $\pm$ 1.76	1.87 $\pm$ 1.93		
Median (IQR)	1.25 (0.75-2.31)	1.25 (0.75-2.00)	U = 6686.50	.632
Number of VPs used				
Mean $\pm$ SD	1.86 $\pm$ 1.16	1.72 $\pm$ 1.06		
Median (IQR)	1.00 (1.00-2.00)	1.00 (1.00-2.00)	U = 6717.50	.314
Specific VPs used, n (%)				
Norepinephrine	118 (98.3)	118 (98.3)	$\chi^2 = 0.00$	1.000
Epinephrine	24 (20.2)	21 (17.5)	$\chi^2 = 0.28$	.598
Phenylephrine	25 (20.8)	20 (16.7)	$\chi^2 = 0.68$	.408
Vasopressin	41 (34.2)	38 (31.7)	$\chi^2 = 0.37$	.545
Cumulative dose of vasopressors per day (mg/day)				
Norepinephrine	8.69 $\pm$ 8.85	16.43 $\pm$ 19.34	t = -3.985	<.001
Epinephrine	1.05 $\pm$ 3.28	1.23 $\pm$ 4.17	t = -.388	.698
Phenylephrine	21.94 $\pm$ 58.52	13.58 $\pm$ 38.08	t = 1.307	.192
Vasopressin (units)	17.59 $\pm$ 29.91	16.02 $\pm$ 26.67	t = .428	.669
Days on Vasopressors				
Mean $\pm$ SD	4.80 $\pm$ 5.36	3.98 $\pm$ 5.37		
Median (IQR)	2.50 (1.00-6.00)	2.00 (1.00-5.00)	U = 6459.00	.160
ICU Length of Stay (days)				
Mean $\pm$ SD	10.05 $\pm$ 9.25	6.57 $\pm$ 6.09		
Median (IQR)	7.00 (3.00-10.75)	4.00 (3.00-5.00)	U = 5230.00	<.001
Hospital Length of Stay (days)				
Mean $\pm$ SD	13.92 $\pm$ 12.49	10.01 $\pm$ 7.75		
Median (IQR)	11.00 (6.00-16.00)	8.00 (6.00-8.00)	U = 5461.00	.001

Note. MAP = Mean Arterial Pressure; SD = standard deviation; IQR = interquartile range

Table 4. Sub-group analyses.

	Mean ± SD (hr)	Test Statistic	P-Value
Primary outcome shock etiology			
Sepsis (n=126)	1.94 ± 1.81	F = 0.386	.763
Cardiogenic (n=50)	2.03 ± 2.38		
Hypovolemic (n=44)	1.82 ± 1.51		
Combination (n=19)	1.59 ± 1.10		
Mortality x ICU LOS in WB group			
Survived (n=68)	6.40 ± 5.286	t = -.348	.729
Died (n=52)	6.79 ± 7.055		

Note. SD = standard deviation; LOS = length-of-stay; WB = weight-based

DISCUSSION

Similar to previous studies conducted, there was no difference in the time to achieve goal MAP upon switching to a WB vasopressor dosing protocol overall and by shock etiology. This may, in part, be attributable to the inherent limitations of retrospective data collection from nursing doc-

umentation in assessing attainment of the target MAP. In addition, our power was set to detect a difference of 30 minutes in achieving MAP between treatment groups. Therefore, a larger patient population is warranted to detect a smaller difference in attaining goal MAP. Several secondary efficacy outcomes were not significant, including the num-

ber of concurrent vasopressors used and agents used most commonly.

Norepinephrine doses were found to be significantly higher in the WB arm, likely due to a high rate of obesity in our patient population, and norepinephrine being a first-line vasopressor across shock etiology. Furthermore, there was no difference between treatment arms in dosing across the remaining vasopressors (epinephrine, phenylephrine, and vasopressin). This may suggest a decreased medication burden on study participants, where participants were able to achieve goal MAPs by maximizing norepinephrine resulting in fewer dosages of remaining vasopressors overall. While not statistically significant, this study also shows a trend in fewer days spent on vasopressors in the WB treatment arm, which may be attributed to vasopressor optimization.

A median 3-day decrease in ICU and hospital LOS in the WB group may be attributed to several of the above-mentioned factors including more rapid vasopressor titration and fewer agents required to achieve target MAPs, which allow patients to transfer out of the ICU sooner and discharge home sooner. In addition, our sub-analysis proved that the shorter ICU stay was not due to a higher mortality rate in the WB treatment arm, further suggesting efficacy of a WB protocol. From a safety perspective, there was no evidence of increased kidney injury or extravasation when changing to a WB protocol.

Limitations of the study include a retrospective study design which complicates the ability to truly assess time to achieve goal MAP depending on nursing documentation as well as the potential for bias. Furthermore, the baseline characteristics of patients were not similar by shock etiology, which may be an overall confounder of statistical significance. This study only examined AKI and extravasation

incidence, and did not consider the impact of other adverse drug events, including tissue ischemia or cardiac arrhythmia.

Further research would benefit from prospective analysis of true goal MAP attainment as well as analyzing an array of potential side effects due to potentially higher doses of vasopressors used.

CONCLUSION

This study confirms previous literature that suggests no difference in time to achieve goal MAP when using a WB versus NWB dosing protocol. However, this study assessed several additional efficacy and safety outcomes that suggest a potential benefit to using a WB dosing protocol in critical shock patients in decreasing lengths-of-stay. Due to limitations in using a retrospective, single-center design, prospective studies further analyzing safety and efficacy would be of value to confirm findings.

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CONFLICTS OF INTEREST

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

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