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Effects of propofol on vasopressor use in patients with sepsis and severe sepsis: A pilot study☆

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ABSTRACT

Purpose: Propofol is one of the most commonly used sedatives in the intensive care unit (ICU) despite its undesirable hypotensive effects. The purpose of this study was to determine the effects of continuous intravenous (CIV) propofol on vasopressor requirements in mechanically ventilated patients with sepsis.
Materials and methods: A multicenter, retrospective, propensity-matched pilot study was conducted comparing patients with sepsis or severe sepsis who received CIV propofol for sedation to those who did not. The primary outcome was incidence of vasopressor support. Secondary outcomes included change in mean arterial pressure, mortality, and length of stay.
Results: A total of 279 patients (149 CIV propofol, 130 non-CIV propofol) were evaluated, with 174 patients matched 1:1 based on propensity score. There was no difference in vasopressor support requirements (49.4% vs 54%; $P = .65$) or in those experiencing a greater than 20% decrease in mean arterial pressure from baseline (58.6% vs 63.2%; $P = .53$) in the CIV propofol and non-CIV propofol groups. Furthermore, there were no differences in any secondary outcomes including hospital mortality (32.2% vs 33.3%; $P = .87$).
Conclusions: Continuous intravenous propofol for sedation did not increase vasopressor requirements in this septic population. Furthermore, CIV propofol was not associated with significant differences in the use of multiple vasopressors, change in mean arterial pressure, length of stay, or mortality.

1. Introduction

Propofol is an intravenous γ-amino butyric acid agonist used for continuous sedation in intensive care unit (ICU) patients [1,2]. Advantages of propofol include ease of titration, fast onset and offset of action, and favorable pharmacokinetic properties [2]. The pharmacokinetic properties of propofol are beneficial as they allow for rapid awakenings and a reduced risk of drug accumulation, especially in patients with hepatic and renal dysfunction. Furthermore, recent literature has demonstrated that propofol is associated with a decreased length of mechanical ventilation compared to benzodiazepines, making it a preferred agent for patients requiring continuous sedation [3].

Although commonly used in ICU patients, propofol does have several significant disadvantages, including the development of hypotension secondary to a reduction in vascular sympathetic tone [2,3]. Based on previous studies, the development of hypotension during propofol administration occurs in approximately 25% to 30% of ICU patients receiving the drug [1,4]. As a result of this potential risk, clinicians may prefer alternative sedatives over propofol in patients at risk for developing hypotension [5,6]. It is well known that critically ill patients with sepsis have an increased risk of hypotension and require early resuscitation to maintain hemodynamic stability and perfusion of critical vascular beds [7,8]. Failure to maintain hemodynamic stability can result in

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progression to shock, resulting in potential increases in mortality as high as 46% [9–13]. Therefore, avoiding factors that may amplify hypotension and the progression to septic shock is critical to patient care.

Risk factors for progression to septic shock have been previously identified and include increased age, hyperthermia, increased shock index, pulmonary disease, and site of infection [9,11]. Although some medications have been postulated to increase the risk of death in septic patients, definitive literature confirming this risk is lacking [14,15]. Etomidate has been hypothesized to influence the progression to septic shock by causing adrenal suppression, although data from multiple studies have failed to demonstrate an effect on hemodynamics or other clinical outcomes [16–18]. Although hypotension has been widely reported with propofol administration, it has not been described in the setting of sepsis as a risk factor for progression to shock. As propofol remains the most commonly used sedative in the ICU and has the potential to cause clinically relevant hypotension in septic patients, the purpose of this investigation was to determine the effects of continuous intravenous (CIV) propofol administration on the need for vasopressor support in patients with sepsis and severe sepsis.

2. Materials and methods

This was a multicenter, retrospective, pilot study of patients with sepsis or severe sepsis who required mechanical ventilation. Patients were enrolled from 2 separate urban teaching hospitals. Patients discharged between the years 2011 and 2014 were identified through corporate patient financial services using the *International Classification of Diseases, Ninth Revision, Clinical Modification*, codes for sepsis and mechanical ventilation. Patients were included if they were at least 18 years of age, intubated within 24 hours of hospital admission, and met sepsis or severe sepsis criteria within 2 hours before intubation [7]. We excluded those who were not septic within 2 hours of intubation, immunosuppressed, intubated before arrival, or received vasopressor support within 24 hours before study inclusion or within 15 minutes of a propofol bolus for intubation. The institutional review boards at both institutions approved the project design.

The experimental group consisted of patients who received CIV propofol for at least 30 minutes within 48 hours after intubation, whereas the control group consisted of patients who did not receive CIV propofol during the 48-hour period after intubation. A minimum enrollment of 200 patients was planned with an approximate equal distribution between the CIV propofol and non-CIV propofol groups. A power analysis was not performed to determine an adequate sample size due to a lack of data describing the rate of hypotension with propofol in this population. Therefore, this investigation served as a pilot study in this area. We followed patients for a period of 48 hours after the initial intubation to measure primary and secondary outcomes. We selected the 48-hour evaluation period based upon the hypothesis that any changes in mean arterial pressure (MAP) would occur earlier in treatment instead of after prolonged administration. We abstracted all data included in the study directly from the electronic medical record at both institutions.

The 2012 Surviving Sepsis Guidelines were used to categorize sepsis and severe sepsis, whereas hospital-associated infections were defined using guidelines from the Infectious Disease Society of America [7,19,20]. Vasopressor support was assessed by documented administration of epinephrine, norepinephrine, vasopressin, dopamine, or phenylephrine. Initiation of appropriate empirical antibiotics was defined by the start of antimicrobial regimens for which presumptive or definitive pathogens were susceptible to in vitro. In the case of culture-negative sepsis or severe sepsis, broad-spectrum antibiotics were deemed sufficient based on the presumptive source of infection and in accordance with local practice guidelines [21].

The primary outcome of the study was the need for vasopressor support between the 2 groups. Secondary outcome measures included absolute change in MAP, a greater than 20% decrease in MAP from baseline, maximum vasopressor infusion rates, the requirement for multiple vasopressor agents, and duration of vasopressor use. The duration of ICU and hospital length of stay as well as the incidence of hospital

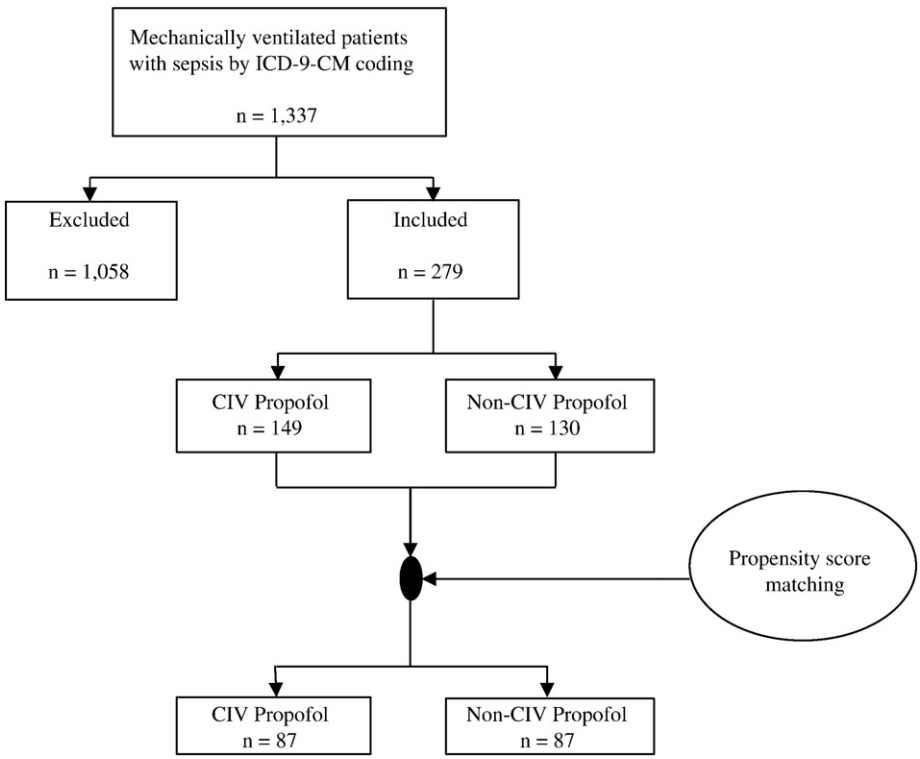

Fig. 1. Patients identified, analyzed, excluded, and propensity score matched.

Table 1
Baseline characteristics

Characteristic	Unmatched cohort (n = 279)			Propensity-matched cohort (n = 174)		
	CIV propofol (n = 149)	Non-CIV propofol (n = 130)	*P*	CIV propofol (n = 87)	Non-CIV propofol (n = 87)	*P*
Age (y)	59 (50-69)	62.5 (53-74)	.04	59 (50.5-69.5)	60 (50.5-70.5)	.74
Weight (kg)	87.6 (68-107)	80 (64-99.3)	.04	82.2 (65.5-104.7)	83.4 (65.8-100.5)	.99
Male, n (%)	64 (49.2)	70 (47)	.71	42 (48.3)	44 (50.6)	.76
Race, n (%)						
Black	86 (57.7)	83 (63.8)	.30	58 (66.7)	51 (58.6)	.27
White	40 (26.8)	23 (17.7)	.07	16 (18.4)	20 (23)	.45
Other	24 (16.1)	24 (18.5)	.60	14 (16.1)	16 (18.4)	.69
APACHE II	22 (18-28)	24 (20-29)	.11	23 (21-28)	23 (22-26)	.76
Primary sepsis source						
Pulmonary, n (%)	88 (59.1)	80 (61.5)	.67	49 (56.3)	52 (59.8)	.65
Gastrointestinal, n (%)	21 (14.1)	25 (19.2)	.25	15 (17.2)	13 (14.9)	.68
Renal/GU, n (%)	31 (20.8)	17 (13.1)	.09	13 (14.9)	14 (16.1)	.83
SSTI, n (%)	15 (10.1)	13 (10)	.99	7 (8)	9 (10.3)	.6
Other, n (%)	18 (12.1)	18 (13.8)	.66	6 (6.9)	10 (11.5)	.29
Unknown, n (%)	7 (4.7)	4 (3.1)	.49	5 (5.7)	4 (4.6)	.73
Multiple, n (%)	32 (21.5)	27 (20.8)	.89	17 (19.5)	17 (19.5)	>.99
Health care-associated infection, n (%)	71 (47.7)	70 (53.8)	.31	38 (43.7)	45 (51.7)	.29
Sufficient empiric antibiotics, n (%)	145 (97.3)	126 (96.9)	.85	83 (95.4)	85 (97.7)	.41
Severe sepsis, n (%)	91 (61.1)	95 (73.1)	.03	60 (69)	56 (64.4)	.52
Baseline MAP (mm Hg)	83 (67-97)	75 (62-90)	.01	81 (65-98)	79 (65.5-91.5)	.67

All data are presented as median (25%-75% interquartile range) unless otherwise noted. SSTI indicates skin/skin structure infection; GU, genitourinary.

mortality were compared between groups. All statistical analysis except propensity score matching was completed using SPSS version 21 (Chicago, IL). The χ^2 test or Fisher exact test was used to compare categorical data, whereas continuous data were evaluated with a *t* test. Statistical significance was established at a *P* value of less than .05.

Propensity score matching was used to adjust for observed differences at baseline between the CIV propofol and non-CIV propofol groups. Propensity scores were calculated using a logistic regression model that included the following demographic and clinical variables: age, height, weight, race, severe sepsis, baseline MAP, Acute Physiology and Chronic Health Evaluation (APACHE) II score, source of sepsis, and administration of blood pressure medications during the 48-hour evaluated period. The propensity model was evaluated for evidence of lack of fit based on the Hosmer-Lemeshow goodness-of-fit test. Model performance was assessed using the area under receiver operating characteristic curve to determine the ability of the model to differentiate between patients receiving CIV propofol and patients who did not receive CIV propofol. Each individual in the CIV propofol group was then matched to a patient in the non-CIV propofol group (1:1, nearest neighbor, no replacement) on the logit of the propensity score using calipers equal to 0.20 of the SD of the logit of the propensity score [22,23]. The balance of measured confounders prematching and postmatching was performed through comparison of standardized differences. The logistic regression propensity model demonstrated no evidence of lack of fit based on the nonsignificant Hosmer-Lemeshow statistic ($P = .34$). Model performance was assessed using the area under receiver operating characteristic curve; at 0.70, this value indicates that the model demonstrated the ability to differentiate between patients in the CIV propofol and non-CIV propofol groups. The propensity score logit model and matching procedure was run using STATA 14.1 (Stata Corporation, College Station, TX).

Table 2
Propofol administration (n = 149)

Max dose (μg/kg/min)	Minimum dose (μg/kg/min)	No. of rate changes	Duration (h)
40 (20-50)	10 (5-15)	5 (2-8)	41 (16.5-48)

All data are presented as median (25%-75% interquartile range).

3. Results

3.1. Results of unmatched analysis

Over a 4-year period, 1337 patients were identified by *International Classification of Diseases, Ninth Revision*, codes and screened for enrollment. A total of 1058 patients were excluded, leaving 279 for evaluation. One-hundred forty-nine patients were included in the CIV propofol group; and 130 patients, in the non-CIV propofol group (Fig. 1). Primary reasons for exclusion were that patients were not septic within 2 hours of intubation, immunosuppressed, or intubated before arrival. Baseline characteristics were similar between groups and are summarized in Table 1. Severity of illness at baseline was similar between groups based upon APACHE II score. Notable differences between unmatched groups include the rate of severe sepsis (61.1% vs 73.1%; $P = .03$) and baseline MAP (83 vs 75 mm Hg; $P = .01$). Data regarding propofol administration are provided in Table 2.

The primary outcome of vasopressor support (45% vs 60%; $P = .01$) was significantly higher in the non-CIV propofol group (Table 3). However, the non-CIV propofol group was more likely to have a lower MAP at baseline, and the overall MAP was reduced in both groups throughout the 48-hour study period (Fig. 2A). There were no differences in secondary outcomes with regard to the absolute change in MAP (23 vs 18.5 mm Hg; $P = .32$) and incidence of greater than 20% decrease in MAP (61.7% vs 58.5%; $P = .58$) throughout the study. Norepinephrine was the most commonly administered vasopressor (43.6% vs 56.9%; $P = .03$) with no difference in the maximum infusion rate between groups (Table 4). Patients in the non-CIV propofol group were also more likely to require multiple vasopressors (14.1% vs 23.1%; $P = .05$). Overall, there was no difference between groups in mortality, hospital length of stay, or duration of mechanical ventilation.

Regarding supplemental therapies, etomidate was the most commonly used agent for intubation in both groups (47.7% vs 46.2%; $P = .8$) (Table 5). Notably, 19.2% of patients in the non-CIV propofol group received a propofol bolus for intubation without subsequent CIV propofol. The CIV propofol group was more likely to receive hydromorphone (21.5% vs 10.8%; $P = .02$) but less likely to receive fentanyl (20.1% vs 40.8%; $P = .001$) or midazolam (8.1% vs 20.8%; $P = .002$). More than one third of patients in both groups did not receive any form of analgesia (45% vs 34.6%; $P = .08$).

Table 3
Clinical outcomes

Characteristic	Unmatched cohort (n = 279)			Propensity-matched cohort (n = 174)		
	CIV propofol (n = 149)	Non-CIV propofol (n = 130)	*P*	CIV propofol (n = 87)	Non-CIV propofol (n = 87)	*P*
Vasopressor use						
Any agent, n (%)	67 (45)	78 (60)	.01	43 (49.4)	47 (54)	.65
Multiple agents, n (%)	21 (14.1)	30 (23.1)	.05	16 (18.4)	17 (19.5)	.85
Change in MAP from baseline						
Absolute change (mm Hg)	23 (5-37)	18.5 (5.0-32.0)	.32	23 (6-40)	21 (9-36)	.66
20% or greater decrease, n (%)	92 (61.7)	76 (58.5)	.58	51 (58.6)	55 (63.2)	.53
Postintubation hydrocortisone use, n (%)	5 (3.4)	5 (3.8)	.83	3 (3.4)	2 (2.3)	>.99
Mechanical ventilation duration (d)	4 (2-8)	4.8 (2-11)	.28	3.5 (2-7.5)	5.3 (2-12)	.07
Length of stay (d)						
ICU	5.5 (2.5-10.8)	6 (2.5-12)	.57	5 (2-9)	6 (3-14)	.15
Hospital	12 (7-21)	12 (7-21)	.86	11 (7-20)	13.5 (8-22)	.38
All-cause mortality, n (%)	42 (28.2)	49 (37.7)	.09	28 (32.2)	29 (33.3)	.87

All data are presented as median (25%-75% interquartile range) unless otherwise noted.

3.2. Results of propensity-matched analysis

A total of 174 patients (87 CIV propofol, 87 non-CIV propofol) were matched based on the results of the propensity model. After the matching procedure, the 2 groups were balanced on demographic and clinical characteristics, including age, weight, APACHE II score, severity of sepsis, source of sepsis, and baseline MAP. Most patients were admitted through the emergency department and intubated for respiratory distress secondary to pneumonia. The groups were similar in the rate of severe sepsis (64.4% vs 69%; *P*= .52) and baseline MAP (79 vs 81 mm Hg; *P*= .67) (Table 1).

Upon completion of propensity score matching, no differences remained in the primary outcome of vasopressor support (49.4% vs 54%; *P*= .65) or greater than 20% decrease in MAP from baseline (58.6% vs 63.2%; *P*= .53) (Table 3). Norepinephrine remained the predominant vasopressor in each group (Table 4), and the supplemental sedation and analgesia requirements remained unchanged compared to the unmatched groups (Table 5). Nearly one fourth (23.3%) of

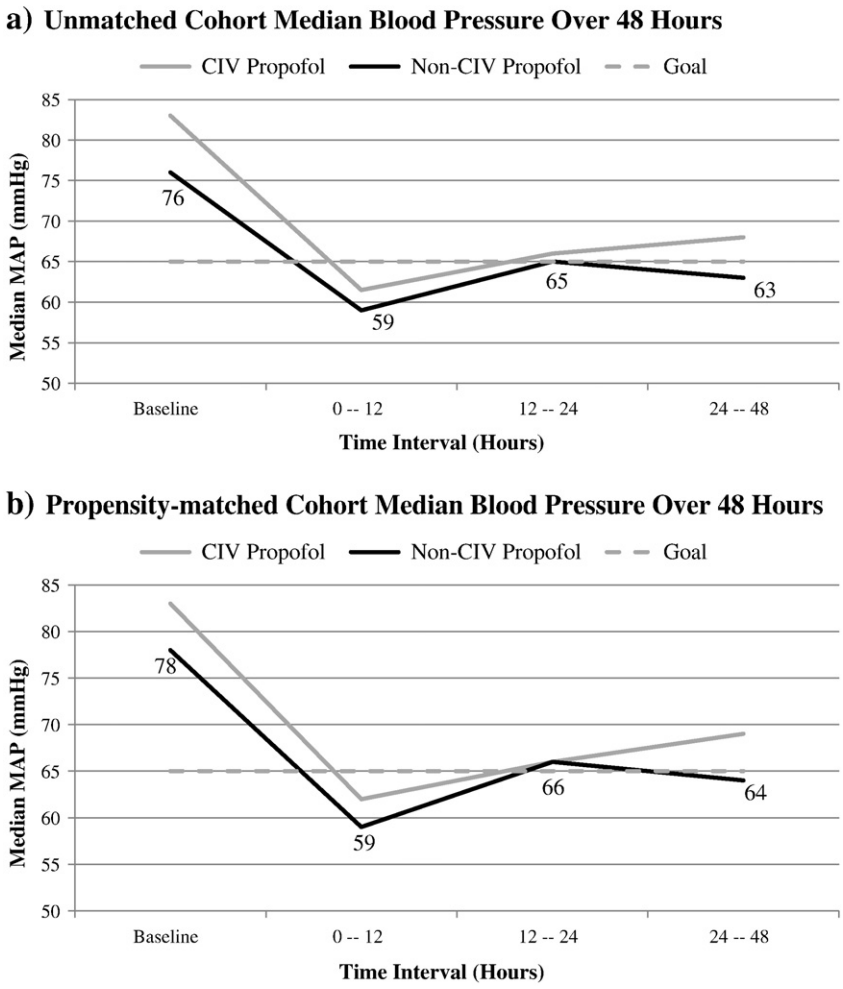

Fig. 2. A, Unmatched cohort median blood pressure over 48 hours. B, Propensity-matched cohort median blood pressure over 48 hours.

Table 4
Vasopressor utilization

Characteristic	Unmatched cohort (n = 279)			Propensity-matched cohort (n = 174)		
	CIV propofol (n = 149)	Non-CIV propofol (n = 130)	*P*	CIV propofol (n = 87)	Non-CIV propofol (n = 87)	*P*
Norepinephrine, n (%)	65 (43.6)	74 (56.9)	.03	41 (47.1)	45 (51.7)	.65
Maximum infusion rate (μg/min)	16 (10-37)	25 (10-42)	.16	20 (10-41)	20 (10-30)	.50
Duration (h)	31.3 (15.5-47)	37 (11.5-48)	.70	32 (14.5-47.5)	35.5 (11.3-48)	.78
Epinephrine, n (%)	2 (1.3)	7 (5.4)	.09	1 (1.1)	6 (6.9)	.05
Maximum infusion rate (μg/min)	10 (8-10)	38 (2-75)	>.99	75[a]	10 (6-10)	.29
Duration (h)	6.75 (6.5-7)	48 (42-48)	.06	6.5[a]	48 (36-48)	.29
Dopamine, n (%)	9 (6.1)	10 (7.7)	.60	6 (7)	5 (5.7)	.74
Maximum infusion rate (μg/kg/min)	8 (5-10)	20 (12-20)	.02	7 (5-20)	20 (20-20)	.13
Duration (h)	12.5 (5.5-16.5)	9.5 (3-23)	.84	14.3 (9-16.5)	6 (3-21.5)	.54
Phenylephrine, n (%)	7 (4.7)	15 (11.5)	.03	6 (6.9)	8 (9.2)	.58
Maximum infusion rate (μg/min)	100 (70-210)	150 (50-300)	.87	110 (50-300)	150 (30-250)	.53
Duration (h)	30 (14-47)	6 (4-14)	.08	37.5 (19-48)	12 (5-32.5)	.28
Vasopressin,[b] n (%)	10 (6.7)	20 (15.4)	.02	7 (8)	10 (11.5)	.44
Duration (h)	21.5 (14-44)	35 (8.5-48)	.85	19 (15.5-40.5)	38.5 (3.5-48)	.74

All data are presented as median (25%-75% interquartile range) unless otherwise noted.
[a] Data for only 1 patient.
[b] 0.04 U/min used in all patients.

patients in the matched non-CIV propofol group received a propofol bolus for intubation without receiving subsequent CIV propofol. After receiving the propofol bolus, these patients had higher absolute decreases in MAP (28.5 mm Hg) compared to the entire cohort, and 80% experienced a greater than 20% decrease in MAP. There were no differences between any other secondary outcomes in the propensity score–matched groups.

4. Discussion

To our knowledge, this is the first investigation evaluating the effects of CIV propofol on vasopressor requirements in septic patients. We compared CIV propofol administration in mechanically ventilated patients with sepsis and severe sepsis and found no differences in vasopressor support or change in MAP within 48 hours after intubation in a matched cohort. Despite its well-known ability to lower blood pressure, our results demonstrated that CIV propofol may be safe in patient populations presenting with sepsis or severe sepsis and does not increase the progression to septic shock. Although a substantial number of patients experienced hypotension and severe sepsis, the overall study mortality was similar to results reported in the literature [24]. Patients received appropriate antibiotic therapy and vasopressor support with norepinephrine in accord with current guidelines for the management of severe sepsis and septic shock [7,24,25]. Steroid utilization for vasopressor supplementation was limited, likely due to the small amount of refractory shock seen in our patient population.

Multiple other factors were present in our study that could potentially influence hemodynamics including use of renal replacement therapy and administration of antihypertensive, sedative, and analgesic medications. Only a minority of patients received hemodialysis or antihypertensive medications during the study period; therefore, it is unlikely to have had a significant effect on the study outcomes. In addition, these factors were equally balanced between groups after propensity score matching. It should be noted that there was a variety of sedative and analgesic therapies administered to patients in both groups of this study.

Although there was no difference in the primary outcome between the matched groups, we believe that propofol use should be discouraged in those who deteriorate hemodynamically soon after receiving a bolus dose for intubation. In a study by Ray et al [26], when intubation was performed with propofol as the sedating agent, it resulted in more vasopressor therapy and higher doses than etomidate. In a similar fashion, Baird et al [15] found propofol administered for intubation was associated with more vasopressor use and hypotension than both etomidate and thiopental. In our study, despite excluding patients started on vasopressor therapy immediately after a propofol bolus for

Table 5
Supplemental therapies received

Characteristic	Unmatched cohort (n = 279)			Propensity-matched cohort (n = 174)		
	CIV propofol (n = 149)	Non-CIV propofol (n = 130)	*P*	CIV propofol (n = 87)	Non-CIV propofol (n = 87)	*P*
Renal replacement therapy, n (%)	21 (14.1)	24 (18.5)	.32	16 (18.4)	18 (20.7)	.70
Antihypertensive agents, n (%)	32 (21.5)	27 (20.8)	.89	24 (27.6)	22 (25.3)	.73
Intubation induction agents, n (%)						
Propofol	49 (32.9)	25 (19.2)	.01	29 (33.3)	20 (23.3)	.13
Etomidate	71 (47.7)	60 (46.2)	.80	42 (48.3)	37 (42.5)	.45
Midazolam	33 (22.1)	35 (26.9)	.35	21 (24.1)	20 (23)	.86
Ketamine	1 (0.7)	4 (3.1)	.19	1 (1.1)	2 (2.3)	>.99
Fentanyl	17 (11.4)	11 (8.5)	.41	9 (10.3)	6 (6.9)	.42
Continuous infusion analgosedation agents, n (%)						
Midazolam	14 (9.4)	52 (40)	<.001	8 (9.2)	36 (41.4)	<.001
Dexmedetomidine	7 (4.7)	7 (5.4)	.79	4 (4.6)	6 (6.9)	.52
Lorazepam	1 (0.7)	7 (5.4)	.91	0 (0)	5 (5.7)	.06
Fentanyl	30 (20.1)	53 (40.8)	<.001	17 (19.5)	36 (41.4)	.002
Bolus analgosedation agents, n (%)						
Midazolam	12 (8.1)	27 (20.8)	.002	5 (5.7)	22 (25.3)	<.001
Lorazepam	38 (25.5)	34 (26.2)	.91	23 (26.4)	22 (25.3)	.86
Morphine	27 (18.1)	20 (15.4)	.54	13 (14.9)	15 (17.2)	.68
Hydromorphone	32 (21.5)	14 (10.8)	.02	17 (19.5)	9 (10.3)	.09
Other pain agent	14 (9.4)	18 (13.8)	.25	10 (11.5)	15 (17.2)	.28

intubation, we found that these patients were more likely to experience a greater than 20% decrease in MAP and also had higher absolute decreases in MAP compared to the entire cohort. Their increased responsiveness to propofol may have influenced the judgment of the clinician and reduced the likelihood of continuing CIV propofol. Further research is needed to substantiate this observation, but we believe that alternative sedation should be considered for patients who quickly deteriorate hemodynamically after a dose of propofol.

Our retrospective, observational study is not without several limitations. We were unable to document the quantity or types of fluid used for initial resuscitation, and the use of sedatives and vasopressors administered was at the judgment of the clinician. We also used *International Classification of Diseases, Ninth Revision*, diagnosis codes as a method to identify patients for study inclusion; therefore, some potentially eligible patients may have not been identified. Furthermore, we evaluated the change in MAP over a 12-hour period after induction bolus doses. The lowest MAP observed during this period was used in data collection, which may have limited our ability to detect blood pressure changes immediately after intubation. Finally, as with any matched analysis, the propensity score matching process resulted in the exclusion of individuals whose propensity scores did not fall in the shared range between the 2 treatment groups. Although we were able to balance the cohort on observable characteristics, the propensity score matching approach does not balance unmeasured characteristics and confounders. Although this approach caused the loss of real-world patient data, it provided an analytical environment closer to a randomized clinical trial where we were able to more accurately assess the efficacy and safety of CIV propofol.

Our study has several strengths, and given it is the first study to our knowledge evaluating CIV propofol in septic patients, it contributes to the growing body of literature surrounding the treatment of sepsis. This study was implemented over multiple sites and included a heterogeneous critically ill patient population in various medical, surgical, and neurological ICUs. We only included patients with sepsis upon presentation who then subsequently required sedation. Our inclusion process permitted hemodynamic data to be captured in the early stages of sepsis before progression to shock and excluded patients who might have had factors confounding the development of hypotension such as adrenal insufficiency and gastrointestinal bleeding. Finally, we used propensity score matching to adjust for observed confounders and reduce bias between patient groups.

We identified those with sepsis based upon the diagnostic criteria published in the 2012 Surviving Sepsis Guidelines, which differs from the newly proposed Sepsis-3 definitions [27]. The definitions used in our pilot study were in accord with sepsis guidelines at the onset and completion of our study. The impact of the proposed Sepsis-3 criteria on our results remains unknown, as the authors of the new definitions do recognize that portions of the SIRS criteria remain useful for identification of infection. Our primary intent was to determine if the administration of propofol to those with sepsis or severe sepsis increased the progression to septic shock. Given that the Sepsis-3 criteria require a serum lactate greater than 2 mmol/L to diagnose shock, the number of patients in our study with shock may have changed because this was not part of the previous guideline. Although general agreement on implementation of the new definitions is still evolving [28], we feel that our evaluation still provides valuable information to critical care clinicians on any potential impact propofol administration may play in the progression of sepsis.

5. Conclusion

In this multicenter, retrospective propensity-matched pilot study, CIV propofol did not increase vasopressor requirements within 48 hours after intubation. Furthermore, CIV propofol was not associated with significant differences in duration of mechanical ventilation, ICU or hospital length of stay, or all-cause mortality. Because this was a pilot study, further prospective studies would be useful to evaluate the hemodynamic effects associated with CIV propofol in septic patients.

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