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Original Research

Bolus Vasopressor Use for Air Medical Rapid Sequence Intubation: The Vasopressor Intravenous Push to Enhance Resuscitation Trial

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A B S T R A C T

Background: Rapid sequence intubation (RSI) may compromise perfusion because of the use of sympatholytic medications as well as subsequent positive pressure ventilation. The use of bolus vasopressor agents may reverse hypotension and prevent arrest.

Methods: This was a prospective, observational study enrolling air medical patients with critical peri-RSI hypotension (systolic blood pressure [SBP] < 90 mm Hg) to receive either arginine vasopressin (aVP), 2 U intravenously every 5 minutes, for trauma patients or phenylephrine (PE), 200 μg intravenously every 5 minutes, for nontrauma patients. The main outcome measures included an increase in SBP, a reversal of hypotension, and the occurrence of dysrhythmia or hypertension (SBP > 160 mm Hg) within 20 minutes of vasopressor administration.

Results: A total of 523 patients (344 aVP and 179 PE) were enrolled over 2 years. An increase in SBP was observed in 326 aVP patients (95%), with reversal of hypotension in 272 patients (79%). An increase in SBP was observed in 171 PE patients (96%), with reversal of hypotension in 148 patients (83%). A low rate of rebound hypertension was observed for both aVP and PE patients.

Conclusion: Both aVP and PE appear to be safe and effective for treating critical hypotension in the peri-RSI period.

Invasive airway management is a critical component of resuscitation for a variety of disease states, including severe traumatic injury as well as medical conditions such as sepsis and respiratory failure.[1,2] Unfortunately, cardiopulmonary arrest is common during the immediate peri-intubation period, accounting for up to 5% of in-hospital arrests and occurring in almost 3% of air medical rapid sequence intubation (RSI) patients.[3,4] Although the need for emergent intubation is a marker for severe illness and injury, some causes of peri-intubation arrest are potentially preventable through targeted interventions.[5] For example, we observed a dramatic reduction in peri-RSI desaturations and improvements in overall airway management success with the implementation of advanced airway training structured around a novel algorithm emphasizing aggressive preoxygenation strategies.[6]

In addition to desaturation, RSI patients are at risk of hypotension because of the hemodynamic effects of induction agents as well as the reduction in cardiac output with positive pressure ventilation.[3,4,7] Although the choice of induction agent may be a consideration, we did not observe a decrease in the incidence of peri-RSI arrest with the use of ketamine.[8] An alternative strategy is to provide inotropic support and/or arterial vasoconstriction by administering vasopressor agents during the peri-RSI period, potentially as a parenteral bolus to expedite the pharmacologic effects.[9,10] Although the use of push-dose vasopressors to reverse peri-RSI hypotension has increased in popularity, evidence to support this approach as safe and effective is lacking.[11-15]

Although epinephrine has been advocated as a good push-dose vasopressor given its effectiveness at increasing blood pressure through inotropic and vasoconstrictive effects, concerns about ventricular dysrhythmias as well as rebound hypertension exist.[16] The

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use of pure vasoconstrictive agents, such as arginine vasopressin (aVP) and phenylephrine (PE), may offer several advantages. The use of PE to treat perioperative hypotension is well-documented.[17-21] In addition, aVP has potentially desirable effects on cerebral perfusion and hemostasis after traumatic injury.[22-26] The primary objective for this analysis was to evaluate the safety and effectiveness of aVP and PE to reverse peri-RSI hypotension in a population of air medical RSI patients.

Methods

Design

This was a prospective, observational cohort study conducted in a large air medical agency. Patients were enrolled over a 24-month period (September 2018-August 2020). A waiver of informed consent was granted by the Air Methods Institutional Review Board (#2017-1011 [February 21, 2018]). This article adheres to the applicable Strengthening the Reporting of Observational Studies in Epidemiology guidelines.

Setting

This study was conducted in a large air medical agency with over 250 bases throughout the United States. Two-person medical crews, typically a flight nurse and an advanced practice paramedic, respond to both scene calls and interfacility transports. Flight crews can perform advanced procedures, including RSI, under standing orders. Clinical data are entered into an electronic patient care record. In addition, data regarding the RSI procedure are entered into the Air Methods Airway Database for performance improvement applications.

Intervention

The RSI procedure includes the administration of an induction agent (ketamine 1-2 mg/kg intravenous push [IVP] or etomidate 0.3 mg/kg IVP) and a paralytic (rocuronium 0.6-1.2 mg/kg IVP or succinylcholine 1.5-2.0 mg/kg IVP). In addition, fentanyl 1 to 2 μg/kg IVP, midazolam 2.5 to 5.0 mg IVP, or additional ketamine 0.5 to 1.0 mg/kg IVP may be administered as an analgesic before or after the procedure. Fluid and/or blood therapy is recommended for the treatment of hypotension in both medical (normal saline/lactated Ringer solution 250 mL intravenous bolus, repeated to maximum of 30 mL/kg) and trauma patients (normal saline/lactated Ringer solution intravenously or blood transfusion to maintain systolic blood pressure [SBP] of 80-90 mm Hg). In 2018, our patient care guidelines committee approved the administration of push-dose vasopressors for persistent hypotension (SBP < 90 mm Hg) despite other interventions (hemorrhage control, intravenous fluid bolus, existing vasopressor infusions, and blood products) before or within 15 minutes after the RSI procedure.[27] These included aVP for trauma patients (2 U intravenously/intraosseously every 5 minutes) and PE for non-trauma patients (200 μg intravenously/intraosseously every 5 minutes).

Data Analysis

The primary purpose of this analysis was to document the safety and effectiveness of bolus administration of aVP and PE to reverse peri-RSI hypotension. The primary outcome measure was defined as the reversal of hypotension within 20 minutes of the initial aVP or PE administration. In addition, the number of doses required to reverse hypotension and the incidence of "relapse" hypotension were also documented.[28,29] The safety of these agents was defined by the incidence of "rebound" hypertension (SBP > 160 mm Hg) within 20 minutes of aVP or PE administration.[20,30,31] Data were presented descriptively using mean, median, or incidence (with 95% confidence intervals) as appropriate. Medication doses were presented as dose/kg ideal body weight. Odds ratios were calculated to compare patients with and without rebound hypertension. StatsDirect (Leeds, UK) was used for all calculations. Statistical significance was assumed for P values < .05.

Results

A total of 523 patients received push-dose vasopressors during the 2-year study period (Table 1). Study enrollment and major outcome measures are displayed in Figure 1. A total of 344 patients received 680 doses of aVP (398 pre-RSI and 282 post-RSI). An increase in SBP was observed in 326 aVP patients (94.8%) with a mean pre-aVP SBP of 61.0 mm Hg and a mean peak SBP (within 20 minutes) of 109.3 mm Hg; reversal of hypotension was observed in 272 aVP patients (79.1%). A total of 174 aVP patients (50.6%) experienced relapse hypotension. These data are displayed in Table 2. A total of 16 aVP patients (4.7%)

Table 1
Demographic and Clinical Data for Arginine Vasopressin (aVP) and Phenylephrine (PE) Cohorts

Parameter	aVP (n = 344)	PE (n = 179)
Demographics		
Mean age (y)	44.9 (42.7-47.0)	63.1 (60.7-65.5)
Male sex (%)	73.8 (69.0-78.2)	56.9 (49.5-64.3)
Scene call (%)	88.7 (84.9-91.6)	50.8 (43.5-58.2)
Rapid sequence intubation medications		
Etomidate (%)	33.4 (28.7-38.6)	34.6 (28.1-41.9)
Mean dose (mg/kg)	0.4 (0.1-0.7)	0.3 (0.2-0.3)
Ketamine (%)	75.0 (70.2-79.3)	49.7 (42.5-57.0)
Mean dose (mg/kg)	2.0 (1.5-2.5)	2.3 (1.6-3.0)
Midazolam (%)	36.3 (31.4-41.6)	16.8 (12.0-22.9)
Mean dose (mg/kg)	0.07 (0.02-0.13)	0.03 (0.02-0.03)
Fentanyl (%)	9.0 (6.4-12.5)	24.6 (18.9-31.4)
Mean dose (μg/kg)	0.8 (0.4-1.2)	0.8 (0.6-1.3)
Succinylcholine (%)	27.3 (22.9-32.3)	41.3 (34.4-48.7)
Mean dose (mg/kg)	1.4 (0.8-2.0)	1.4 (1.1-1.6)
Rocuronium (%)	72.7 (67.7-77.1)	41.9 (34.9-49.2)
Mean dose (mg/kg)	1.1 (0.8-1.4)	1.1 (0.7-1.5)
Fluids and blood products		
Prearrival isotonic fluids (mL)	1,381 (300-2,250)	1,462 (647-2,276)
Postarrival isotonic fluids (mL)	1263 (546-1,980)	1,130 (562-1,698)
Blood administration	26.5 (22.1-31.4)	3.4 (1.5-7.1)
Volume (mL)		
Vasopressor infusion	11.6 (8.7-15.5)	52.5 (45.2-59.7)

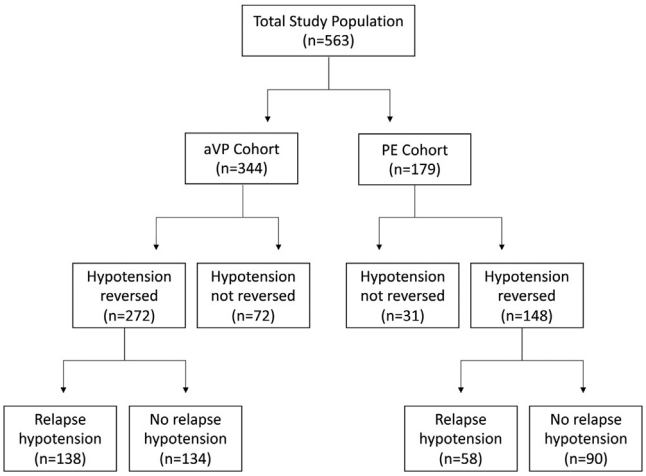

Figure 1. The Study Flowchart.

Table 2
Push-Dose Pressor (PDP) Dosing and Clinical Response for Arginine Vasopressin (aVP) and Phenylephrine (PE) Cohorts

Parameter	aVP (n = 344)	PE (n = 179)
PDP dosing		
Initial PDP dose pre-RSI (%)	76.2 (71.4-80.4)	68.3 (61.5-75.2)
Pre-RSI PDP doses (n)	1.2 (1.1-1.3)	1.2 (1.0-1.4)
Post-RSI PDP doses (n)	0.8 (0.7-0.9)	0.9 (0.7-1.1)
Lowest pre-PDP SBP (mm Hg)	57.2 (54.3-60.2)	60.9 (57.5-64.3)
Last pre-PDP SBP (mm Hg)	61.4 (58.5-64.3)	63.7 (60.2-67.3)
Initial PDP doses (n)	1.5 (1.4-1.6)	1.7 (1.5-1.9)
PDP response		
Increased SBP (%)	94.8 (91.9-96.7)	95.5 (91.1-97.9)
Reversal to SBP 90 mm Hg or greater (%)	79.1 (74.5-83.0)	82.1 (76.6-87.9)
Time to reversal (min)	6.4 (5.8-7.0)	6.8 (6.0-7.6)
Highest post-PDP SBP within 20 min (mm Hg)	109.4 (105.9-112.9)	106.9 (103.0-110.7)
SBP > 160 mm Hg within 20 min (%)	4.7 (2.9-7.4)	4.5 (1.4-7.4)
Cardiopulmonary arrest (%)	15.4 (12.0-19.6)	14.5 (10.1-20.4)
Relapse hypotension		
Relapse within 20 min (%)	50.6 (45.3-55.8)	60.9 (53.6-67.7)
Repeat PDP administration (%)	29.1 (24.5-34.1)	34.1 (27.5-41.6)
Pre-PDP SBP (mm Hg)	61.4 (56.0-66.7)	65.6 (59.7-71.6)
PDP doses (n)	1.5 (1.3-1.6)	1.4 (1.2-1.5)
Reversal to SBP 90 mm Hg or greater (%)	73.5 (64.6-82.4)	68.9 (56.9-80.8)

RSI = rapid sequence intubation; SBP = systolic blood pressure.

experienced rebound hypertension (Table 2) with no statistically significant differences observed compared with nonhypertension patients (Table 3). Only 25% received blood products because of limited availability during the study period.

A total of 53 aVP patients (15.4%) suffered cardiopulmonary arrest (Table 4). Of note, almost one third of these had arrested at least once before the initial aVP administration, and another third suffered hypoxemic arrest during the RSI procedure. In addition, a total of 9 aVP arrest victims (17.0%) were administered aVP during rapid deterioration and arrested shortly afterward, and another 10 (18.9%) arrested more than 15 minutes after the RSI procedure and were ineligible to receive additional aVP doses. Four aVP patients with rebound hypertension experienced subsequent cardiopulmonary arrest. The first was due to airway management difficulties and hypoxemia, the second involved ventricular fibrillation 31 minutes after aVP administration, the third involved rapid deterioration before aVP administration and arrest 4 minutes later, and the fourth suffered relapse hypotension 17 minutes after aVP administration and was ineligible for additional doses. None of the

Table 3
A Comparison of Arginine Vasopressin Cohort Patients With and Without Rebound Hypertension (HTN)

Parameter	HTN (n = 16)	No HTN (n = 328)	Difference or Odds Ratio (95% CI)	*P* Value
Age (y)	53.2	44.6	8.6 (−1.4 to 18.9)	.066
Male (%)	87.5	72.9	2.6 (0.6 to 11.7)	.255
Scene (%)	84.8	93.8	0.3 (0.1 to 1.07)	.082
Pre-RSI PDP (%)	93.8	75.5	4.8 (0.6 to 37.2)	.132
Lowest pre-PDP SBP (mm Hg)	57.6	57.2	0.4 (−13.7 to 14.3)	.967
Last pre-PDP SBP (mm Hg)	58.5	61.5	−3.0 (−16.7 to 10.7)	.713
Doses (n)	1.4	1.5	−0.1 (−0.6 to 0.4)	.630

Hypertension was defined as SBP >160 mm Hg within 20 minutes of the initial arginine vasopressin administration.
CI = confidence interval; PDP = push-dose pressor; RSI = rapid sequence intubation; SBP = systolic blood pressure.

Table 4
A Detailed Analysis of Cardiopulmonary Arrest Victims in the Arginine Vasopressin (aVP) Cohort (n = 53)

Parameter	Mean or % (95% CI)
Pre-PDP SBP	
Lowest pre-PDP SBP (mm Hg)	51.2 (41.7-60.6)
Last pre-PDP SBP (mm Hg)	53.8 (44.0-63.7)
Mechanism of injury (% for each)	
Motor vehicle accident	45.3 (32.7-58.5)
Motorcycle crash	7.6 (3.0-17.9)
Gunshot wound	20.8 (12.0-33.5)
Stab wound	1.9 (0.3-10.0)
Fall	7.6 (3.0-17.9)
Pedestrian vs automobile	11.3 (5.3-22.6)
Other	1.9 (0.3-10.0)
Adjunctive therapy	
Blood product administration (%)	24.5 (14.9-37.6)
Vasopressor infusion (%)	7.6 (3.0-17.9)
Clinical situation (% for each)	
Arrest before PDP administration	30.2 (19.5-43.5)
Hypoxic arrest during airway management	30.2 (19.5-43.5)
aVP administration during rapid deterioration	17.0 (9.2-29.2)
> 15 min after aVP administration	18.9 (10.6-31.4)
Pericardial tamponade (aortic dissection)	1.9 (0.3-10.0)
Tension pneumothorax (relieved/recovery with NT)	1.9 (0.3-10.0)

CI = confidence interval; NT = needle thoracostomy; PDP = push-dose pressor; RSI = rapid sequence intubation; SBP = systolic blood pressure.

Table 6
A Detailed Analysis of Cardiopulmonary Arrest Victims in the Phenylephrine (PE) Cohort (n = 26)

Parameter	Mean or % (95% CI)
Pre-PDP systolic blood pressure	
Lowest pre-PDP SBP (mm Hg)	46.3 (34.5-58.2)
Last pre-PDP SBP (mm Hg)	47.6 (34.7-60.5)
Chief complaint (% for each)	
Cardiac (STEMI, CHF)	26.9 (13.7-46.1)
Postarrest	23.1 (11.0-42.1)
Sepsis	7.7 (2.1-24.1)
Respiratory distress	7.7 (2.1-24.1)
Cerebrovascular accident	7.7 (2.1-24.1)
Altered mental status	7.7 (2.1-24.1)
AAA	3.9 (0.7-18.9)
Hypothermia	3.9 (0.7-18.9)
Motor vehicle crash	11.5 (4.0-29.0)
Adjunctive therapy	
Blood product administration (%)	3.9 (0.7-18.9)
Vasopressor infusion (%)	38.5 (22.4-57.5)
Clinical situation (% for each)	
Arrest before PDP administration	42.3 (25.6-61.1)
Hypoxic arrest during airway management	3.9 (0.7-18.9)
PE administration during rapid deterioration	15.4 (6.2-33.5)
> 15 min after PE administration	23.1 (11.0-42.1)
AAA rupture after initial SBP rise	3.9 (0.7-18.9)
Bradycardia after PE administration	11.5 (4.0-29.0)

AAA = abdominal aortic aneurysm; CHF = congestive heart failure; PDP = push-dose pressor; SBP = systolic blood pressure; STEMI = ST-segment elevation myocardial infarction.

other arrest victims had rebound hypertension or dysrhythmia within 20 minutes of aVP administration.

A total of 179 patients received 379 doses of PE (212 pre-RSI and 167 post-RSI). An increase in SBP was observed in 171 PE patients (95.5%) with a mean pre-PE SBP of 64.4 mm Hg and a mean peak SBP (within 20 minutes) of 107.0 mm Hg; reversal of hypotension was observed in 147 PE patients (82.1%). A total of 109 PE patients (60.9%) experienced relapse hypotension. These data are displayed in Table 2. A total of 8 PE patients (4.5%) experienced rebound hypertension (Table 2) with no statistically significant differences observed compared with nonhypertension patients (Table 5).

A total of 26 PE patients (14.5%) suffered cardiopulmonary arrest (Table 6). Of note, 11 of these (42.3%) had arrested at least once before the initial PE administration. In addition, a total of 4 PE arrest victims (15.4%) were administered PE during rapid deterioration and arrested shortly afterward, and another 6 (23.1%) arrested more than 15 minutes after the RSI procedure and were ineligible to receive additional PE boluses. Two PE patients had rebound hypertension followed by subsequent arrest. One involved rapid deterioration before PE administration with temporary hypertension followed by arrest within 3 minutes; the other was a patient with ST-segment elevation myocardial infarction with post-PE hypertension followed by relapse hypotension and arrest 9 minutes later despite the addition of a norepinephrine infusion. It is notable that 2 arrest victims in the PE cohort suffered trauma while aVP was unavailable, although both had reversal of hypotension followed by relapse within 15 minutes. In addition, 2 patients suffered bradycardia followed by hypotension and arrest, both within 5 minutes of PE administration but concurrent with the administration of ketamine and rocuronium.

Discussion

Critical hypotension is an important mediator of morbidity and mortality for multiple diseases.[3,30,32,33] Although the treatment of hypotension with intravenous fluids, blood products, and vasopressors is a fundamental aspect of critical care, the potential for RSI to uncover or exacerbate perfusion problems is underappreciated.[3-5] This may occur as a result of the sympatholytic effects of medications used for induction, with no single agent emerging as completely safe in this regard, or the change from negative- to positive-pressure ventilation after the insertion of an advanced airway and a reduction in cardiac output.[3-5,8] In this article, we present our experience with a novel protocol for bolus administration of aVP and PE to treat critical hypotension in air medical trauma and nontrauma RSI patients. Both agents were effective at improving SBP and reversing hypotension with a low incidence of rebound hypertension. A substantial percentage of patients experienced relapse hypotension, although many of these occurred outside of the 15-minute treatment window defined by the study protocol. These data support the safety and effectiveness of aVP and PE as bolus-administered vasopressor agents to reverse critical hypotension in the peri-RSI period.

Table 5
A Comparison of Phenylephrine (PE) Cohort Patients With and Without Rebound Hypertension (HTN)

Parameter	HTN (n = 8)	No HTN (n = 171)	Difference or Odds Ratio (95% CI)	*P* Value
Age (y)	70.0	62.7	7.3 (−4.3 to 18.8)	.093
Male (%)	50.0	57.3	0.7 (0.2 to 3.0)	.725
Scene (%)	62.5	49.7	1.6 (0.4 to 7.3)	.720
Pre-RSI PDP (%)	50.0	69.6	0.4 (0.1 to 1.8)	.438
Lowest pre-PDP SBP (mm Hg)	59.3	61.5	−1.7 (−18.3 to 14.8)	.851
Last pre-PDP SBP (mm Hg)	63.5	64.5	−0.2 (−17.6 to 17.1)	.982
Doses (n)	1.6	1.7	−0.1 (−1.1 to 0.9)	.831

Hypertension was defined as SBP >160 mm Hg within 20 minutes of the initial arginine phenylephrine administration.
CI = confidence interval; PDP = push-dose pressor; RSI = rapid sequence intubation; SBP = systolic blood pressure.

The use of bolus-dose vasopressor agents in the emergency department and emergency medical service environments has increased in recent years, with many protocols selecting epinephrine because of familiarity, availability, and ease of administration.[11-13] However, some concerns exist with regard to the cardiac stimulation of epinephrine resulting in an increase in myocardial oxygen demand in a low perfusion state, which may precipitate ventricular dysrhythmias.[14-16] The selection of PE for nontrauma patients reflects the anesthesia and emergency department experience with this agent.[17-20] Several unique pharmacologic features support the use of PE in the peri-RSI period, including peripheral vasoconstriction that limits perfusion to nonessential tissues during this critical procedure as well as the redistribution of splanchnic blood to the central circulation to combat peri-RSI hypotension.[27] Similarly, the use of aVP may be justified in the trauma population based on several unique pharmacologic properties, including retention of potency with acidosis, selective vasoconstriction of the splanchnic circulation to attenuate subdiaphragmatic hemorrhage, and the ability to improve cerebral perfusion with hypovolemia.[22-26] In addition, a randomized trial with a small sample size demonstrated a reduction in blood product administration with traumatic shock, supporting the clinical benefit of these theoretical advantages.[34] We did not directly compare the 2 agents because the patient populations were quite different. However, the safety and effectiveness of aVP and PE were quite similar, supporting the protocol described here.

The ability to reverse hypotension and avoid arrest were the primary measures of clinical effectiveness. Both aVP and PE demonstrated high rates of SBP improvement and reversal of hypotension. It is notable that the mean SBP values before aVP and PE administration were well below a critical prearrest threshold of 80 mm Hg.[35] The reversal of profound hypotension should also be considered significant for patients with conditions involving ischemia-reperfusion injury (acute coronary syndrome, postarrest, stroke, traumatic brain injury, traumatic shock, sepsis, or nontraumatic hemorrhage), which encompass most of the patients in this study. We would anticipate a corresponding reduction in the incidence of peri-RSI arrest as well as improvements in survival and functional outcome, particularly in patients with the conditions described previously.

The optimal dose of these agents remains unclear and was beyond the scope of this analysis. Although the literature supports PE in 100- to 200-μg boluses, aVP bolus doses have been reported from as low as 0.4 U to as high as 20 U in nonarrest patients.[36,37] Although different dosing regimens were not incorporated in this study, the high rate of clinical response and the low incidence of complications suggest that the selected doses were reasonable. A substantial number of patients required repeat doses, which was allowed at 5-minute intervals as a strategy to allow “titration” of effect and avoid rebound hypertension. More than half of the patients required aVP or PE administration after RSI, approximately half of whom received a pre-RSI aVP or PE dose that reversed hypotension, underscoring the hemodynamic impact of the procedure. A quarter of the arrests occurred more than 15 minutes after the initial aVP or PE dose, which is outside of the protocol window to receive additional aVP or PE doses. This may suggest a wider time window for bolus vasopressor dosing and supports the aggressive use of vasopressor infusions as adjunct therapy. Of note, fewer arrest patients received blood or vasopressor infusions compared with nonarrest patients. However, this may also reflect the occurrence of arrest before adjunctive therapies could be initiated.

The incidence of rebound hypertension was low for both agents, which is of particular concern for traumatic injury in which hypertension may lead to clot disruption and exacerbation of hemorrhage.[20,30,31] No clear risk factors could be identified to predict rebound hypertension in either the aVP or PE cohorts.

The majority of study patients received aVP or PE before the RSI procedure. This would support the use of SBP as an indicator for bolus vasopressor administration. Other strategies include administration based on the presence of various clinical parameters to predict peri-RSI hypotension.[38-40] Although more accurate predictors may further reduce hypoperfusion, it is also possible that the incidence of rebound hypertension may increase in the absence of hypotension at the time of vasopressor administration.

These data must be considered within the context of study limitations. This was an observational analysis, with most data presented descriptively. Thus, we cannot determine whether the reversal of hypotension occurred as a result of aVP or PE administration versus the impact of other interventions or the natural course of each patient. In addition, the study database did not include the rate or volume of intravenous fluids, the volume of blood products, or the administration rate for vasopressor infusions. Each of these therapies would be expected to have substantially influenced the hemodynamic response of patients to bolus vasopressor doses as well as the RSI procedure itself. Similarly, the occurrence of rebound hypertension or relapse hypotension may be unrelated to the pharmacologic effects of either agent. However, the timing of hypotension reversal and relapse was consistent with the known pharmacology of these agents.[29]

A variety of diseases were included in the study population. Although this may be appropriate when evaluated, the overall effectiveness of treatment guidelines, the influence of multiple variables affecting volume status, cardiovascular state, the presence of catecholamines and other humoral factors, physiological reserve, and the underlying pathophysiology could not be determined. Finally, we did not attempt to correlate these data with either short- or long-term outcomes. We selected hemodynamic variables, including the occurrence of cardiopulmonary arrest, as primary outcome measures. This is appropriate with a focus on improving the safety of the RSI procedure. However, additional benefits with regard to the neurologic outcome or long-term survival may be more appropriate when considering the ultimate role for bolus vasopressors in critical care and resuscitation.

Conclusions

The bolus administration of aVP for trauma patients and PE for nontrauma patients appears to be both safe and effective at reversing peri-RSI hypotension and preventing cardiopulmonary arrest. Nearly all patients had improvements in SBP with a low incidence of rebound hypertension. Most patients had relapse hypotension after their initial response, which may support repeat dosing beyond the peri-RSI period as well as the more aggressive use of vasopressor infusions as adjunctive therapy.

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