Is cerebral perfusion maintained during full and partial resuscitative endovascular balloon occlusion of the aorta in hemorrhagic shock conditions?

Derek A. Benham, MD, Richard Y. Calvo, PhD, Matthew J. Carr, MD, Lyndsey E. Wessels, MD, Andrew J. Schrader, DVM, Joseph J. Lee, BS, Michael J. Krzyzaniak, MD, *and* **Matthew J. Martin, MD***, San Diego, California*

BACKGROUND: Partial resuscitative endovascular balloon occlusion of the aorta (pREBOA) is a technology that occludes aortic flow and allows for controlled deflation and restoration of varying distal perfusion. Carotid flow rates (CFRs) during partial deflation are unknown. Our aim was to measure CFR with the different pREBOA balloon volumes and correlate those to the proximal mean arterial pressure (PMAP) and a handheld pressure monitoring device (COMPASS; Mirador Biomedical, Seattle, WA).

METHODS: Ten swine underwent a hemorrhagic injury model with carotid and iliac arterial pressures monitored via arterial lines. Carotid and aortic flow rates were monitored with Doppler flow probes. A COMPASS was placed to monitor proximal pressure. The pREBOA was inflated for 15 minutes then partially deflated for an aortic flow rate of 0.7 L/min for 45 minutes. It was then completely deflated. Proximal mean arterial pressures and CFR were measured, and correlation was evaluated. Correlation between CRF and COMPASS measurements was evaluated.

RESULTS: Carotid flow rate increased 240% with full inflation. Carotid flow rate was maintained at 100% to 150% of baseline across a wide range of partial deflation. After full deflation, CFR transiently decreased to 45% to 95% of baseline. There was strong positive correlation ($r > 0.85$) between CFR and PMAP after full inflation, and positive correlation with partial inflation ($r > 0.7$). Carotid flow rate had strong correlation with the COMPASS with full REBOA ($r > 0.85$) and positive correlation with pREBOA ($r > 0.65$).

CONCLUSION: Carotid flow rate is increased in a hemorrhagic model during full and partial inflation of the pREBOA and correlates well with PMAP. Carotid perfusion appears maintained across a wide range of pREBOA deflation and could be readily monitored with a handheld portable COMPASS device instead of a standard arterial line setup. (*J Trauma Acute Care Surg.* 2021;91: 40–46.

KEY WORDS: Noncompressible truncal hemorrhage; resuscitative endovascular occlusion of the aorta; hemorrhagic shock; partial resuscitative endovascular occlusion of the aorta; swine.

Noncompressible truncal hemorrhage (NCTH) is a lethal challenge facing both the civilian and military surgeon,[1,2] and the race to reduce the time to surgical intervention has led to the development of new technologies over the past two decades. Resuscitative endovascular balloon occlusion of the aorta (REBOA) has been used as a relatively new adjunct for hemorrhagic shock[3] and has been studied in both traumatically injured and nontraumatically injured patients.[4–7] With the widespread recognition of preventable battlefield deaths due to NCTH, REBOA has now also been proposed to be used as an en route adjunct in military settings to delay physiologic collapse and temporarily control ongoing NCTH.[8]

Submitted: November 30, 2020, Revised: January 6, 2021, Accepted: January 28, 2021, Published online: February 17, 2021.

From the Department of Surgery (D.A.B., M.J.C., L.E.W., A.J.S., J.J.L., M.J.K.), Naval Medical Center San Diego; and Trauma Service, Department of Surgery (R.Y.C., M.J.M.), Scripps Mercy Hospital, San Diego, California.

This study was presented at the 34th Annual Meeting of the Eastern Association for the Surgery of Trauma Annual Scientific Assembly, January 13–14, 2021, virtual meeting.

Address for reprints: Matthew J. Martin, MD, Trauma Service, Department of Surgery, Scripps Mercy Hospital, 4077 Fifth Ave, MER62, San Diego, CA 92103; email: martin.matthew@scrippshealth.org.

DOI: 10.1097/TA.0000000000003124

The standard REBOA device, also known as the ER-REBOA, provides nontitratable complete occlusion of the aorta.[9] ER-REBOA has been associated with complications related to distal perfusion that included acute kidney injury, abdominal organ ischemia, and distal limb ischemia.[10–13] In addition, there are also concerns about potential proximal complications such as cardiac injury (including cardiac failure and myocardial infarction from increased afterload) and worsening of traumatic brain injury related to increased proximal mean arterial pressures (PMAPs)[10–12] that resemble known proximal complications following aortic clamping during resuscitative thoracotomy.[14]

A miniaturized, handheld pressure monitoring device that attaches in line to intravascular lines was used to measure mean arterial pressures (MAPs) in this study. COMPASS devices (Mirador Biomedical, Seattle, WA) are a Food and Drug Administration–approved device used to measure MAP, intracranial pressures, and central venous pressures. There are currently other ongoing studies by the current authors evaluating the precision and accuracy of the COMPASS devices with the ER-REBOA and partial resuscitative endovascular balloon occlusion of the aorta (pREBOA) devices.

A novel device using a bilobed balloon that allows for titration of flow distal to the occlusive balloon has been developed[15] and been termed *partial REBOA*. Previous swine studies have

shown pREBOA to have increased survival and survival times, and decreased ischemia-reperfusion injuries when compared with standard full occlusion using the ER-REBOA device.[16,17] Although traumatic brain injury has been cited as a relative contraindication to REBOA because of concerns about the intervention increasing carotid artery flow rates and intracranial pressures or hemorrhage, there is little available evidence to support this assertion. In addition, with pREBOA, there are concerns about both elevated carotid flow during complete occlusion and decreased or inadequate carotid flow with initiation of partial occlusion and distal reperfusion. However, currently, there is a paucity of reported measurements between changes in the proximal perfusion over time and their correlation with carotid flow rates (CFRs). We hypothesized that pREBOA would maintain adequate CFR at near baseline physiologic levels in a severe hemorrhagic shock swine model. The primary outcome was the measurement of CFR compared with the baseline CFR, and secondary outcomes consisted of correlation of CFR with PMAP and proximal COMPASS measurements.

MATERIALS AND METHODS

This study was approved by the Institutional Animal Care and Use Committee at Naval Medical Center San Diego as protocol S-19-001. Veterinary support was provided by the staff at the Naval Medical Center San Diego Vivarium. Ten Yorkshire swine between weights of 36.3 kg and 45 kg were used for this study. All pigs were male. Pigs received an intramuscular preanesthetic combination of either telazol (5 mg/kg)/xylazine (2.5 mg/kg) or telazol (5 mg/kg)/ketamine (2.5 mg/kg)/buprenorphine (0.03 mg/kg). All animals received a single dose of glycopyrollate (0.005 mg/kg). Anesthetic induction was accomplished using isoflurane (3–4% mean alveolar concentration) delivered via facemask. Animals were intubated and maintained with isoflurane (0.8–2.0% mean alveolar concentration) with mechanical ventilation (18–24 breaths per minute, 25–30 cm H2O peak inspiratory pressure, 3–5 cm H_2O positive end expiratory pressure) titrated to effect. Each pig received intravenous normal saline (5–10 ml/kg per hour) when not undergoing active resuscitation for the duration of the procedure, and active warming was provided. Anesthetic depth was monitored using the following parameters: jaw tone, pulse rate, respiratory rate, rectal temperature, blood oxygen saturation, invasive arterial blood pressure, and electrocardiogram.

The procedure began with obtaining invasive monitoring. The right common carotid and the right internal jugular vessels were exposed via a central neck cut down. The right internal jugular was cannulated with a 9.5-Fr Cordis, and a Swan-Ganz catheter was introduced and positioned to obtain pulmonary capillary wedge pressures. The right common carotid was then directly cannulated with a 5-Fr micropuncture catheter, and an arterial pressure tracing was obtained. This became the proximal arterial monitor measuring the PMAP. A perivascular laser flow probe was placed around the left carotid artery. The left internal jugular vein was accessed with a 9.5-Fr Cordis, to be used at a later point for controlled hemorrhage, as well as rapid infusion following uncontrolled hemorrhage. A laparotomy was then performed, and the distal aorta and aortic bifurcation were exposed. A 7-Fr sheath was introduced into the left external iliac artery. This sheath was used to later place the REBOA catheter and an arterial waveform obtained from the sheath's port, measuring the distal MAP. Following this exposure, the superior mesenteric artery (SMA) was then exposed, and a perivascular laser flow probe was placed around the SMA. A foley catheter was placed directly into the bladder for urine output monitoring. Following the laparotomy, a left anterior thoracotomy was performed through the eighth intercostal space. The descending thoracic aorta was exposed superior to the diaphragm. A perivascular laser flow probe was then placed around the distal descending aorta.

Hemodynamic monitoring consisted of the following: a proximal arterial waveform line through the right common carotid and a distal arterial waveform line through the right iliac artery. A proximally placed COMPASS device to monitor the proximal arterial pressures was attached to the arterial port on the pREBOA catheter, while the distally placed COMPASS device was attached to the side port on the introducer sheath in the left iliac artery. A pulmonary artery catheter was placed and allowed for assessment of central pressures, right and left ventricular filling pressures, and cardiac output/index. Laboratory assays and full hemodynamic recordings were obtained at baseline, at 5 minutes post-REBOA inflation/uncontrolled hemorrhage, at 15 minutes following REBOA inflation/uncontrolled hemorrhage, and then at 15-minute intervals until the conclusion of the study. COMPASS MAPs and MAPs obtained from the arterial line monitoring were obtained at baseline, at initial injury, and at 5-minute intervals following injury.

Before the injury, a controlled hemorrhage of 20% total blood volume (TBV) was removed via the left internal jugular central line, although the rate of removal was titrated to not allow the MAP to fall below 40 mm Hg. No other resuscitation occurred during this hemorrhage. This blood was then stored to be used later as resuscitation fluid. A lethal noncompressible truncal hemorrhage injury was then simulated by isolating the right iliac artery and vein in the retroperitoneal space. This artery was accessed and cannulated with a 12-Fr dilator, and the right iliac vein was cannulated with a 16-Fr dilator. The dilators were then capped and externalized from the midline incision. These dilators were then removed to create the uncontrolled hemorrhage. The dilator removal mimicked a closed retroperitoneal venous and arterial hemorrhage.

Following injury, the pREBOA was fully inflated until there was a loss of distal arterial waveform to confirm complete occlusion. This continued for 15 minutes, at which point the pREBOA was deflated in an attempt to achieve minimal distal flow. An operator titrated the pREBOA balloon using a titrating saline syringe to maintain a flow rate of 0.7 L/min. Previous experimentation by the authors demonstrated a correlation between distal COMPASS pressure of 25 to 35 mm Hg to maintain this flow rate. A second investigator recorded the distal and PMAP, the proximal COMPASS MAP recording, and SMA flow rates and aortic flow rates.

A simulated combat-relevant resuscitation was then given to support the hemodynamics in a way that mimics the existing tactical casualty combat care guidelines for prehospital resuscitation in the absence of the immediate availability of blood products.[18] An initial 250-mL bolus of Hextend was given to the swine when the MAP fell to less than 40 mm Hg, simulating prehospital resuscitation. Once the MAP fell to past 40 mm Hg

and 250 mL of Hextend had been used, the previously saved 20% TBV whole blood was then transfused back to the animal to maintain a MAP of 40 mm Hg, simulating en route care with the availability of whole blood. Following Hextend and 20% TBV resuscitation, minimal maintenance crystalloid fluid was then used only to maintain intravenous line patency and flushes.

The experiment continued until 2 hours following injury or a sustained MAP of <20 mm Hg. Animals were euthanized using a barbiturate-based euthanasia solution, and death was confirmed with direct auscultation and absence of visualized heartbeat for 10 minutes. At the conclusion of the procedures, the distal arterial waveforms and recordings were compared with the distal COMPASS readings, as well as the proximal arterial waveforms and the proximal COMPASS MAP recordings. The flow rates of the aortic recordings were compared with the distal COMPASS MAP readings.

Data were collected and analyzed using Microsoft Excel (Microsoft Corporation, Seattle WA) and Stata MP version 13.0 (StataCorp LP, College Station, TX), respectively. Baseline CFRs were calculated based on the mean flow rates at time 0, before injury, and pREBOA inflation. Changes in CFR were shown as a percentage compared with the baseline. Pearson correlation coefficients (ρ) were calculated to evaluate the association between carotid flow, proximal and distal arterial lines, and proximal and distal COMPASS measurements for every 5-minute interval from the baseline of the study and controlling for repeated measures. This was performed to evaluate for any deviation in measurements over time. Statistical significance was set at a p value of <0.05.

RESULTS

Of the 10 swine in the experiment, survival times ranged from 10 to 120 minutes, with a mean survival time of 50 minutes. Eight pigs survived from full inflation of the pREBOA to the partial inflation stage of the procedure, while four pigs survived to the full deflation stage of the procedure. One pig survived the entire 120-minute duration of the experiment.

The baseline CFR was compared with the CFRs measured at 5-minute intervals. This was reported as a percent change compared with the baseline and is shown in Figure 1. When the pREBOA was fully inflated (with full aortic occlusion, similar to ER-REBOA), carotid flow was noted to be uniformly at supranormal levels, with CFRs of 175% to 240% of baseline measurements. At 15 minutes, with the pREBOA partially deflated to a goal distal aortic flow rate of 0.7 L/min, the CFR was maintained at a level of 100% to 150% of baseline. After 45 minutes of partial deflation and hemorrhagic shock, the pREBOA was fully deflated, and CFRs were noted to decrease to 45% to 95% of baseline.

Correlations were calculated to compare CFRs to the PMAP (measured by the proximal standard arterial line). There was a strong positive correlation ($r = 0.850$, $p < 0.001$) during full inflation (Table 1). During the first 30 minutes of the partial deflation stage, there was a significant but less robust correlation ($r > 0.7$) between CFR and PMAP. Assessment of the portable handheld arterial pressure monitoring device (COMPASS) provided similarly reliable correlations, and the proximal and distal COMPASS pressures were compared with the simultaneously measured CFR. Similar to the standard arterial pressure monitor, there was a strong positive correlation between the COMPASS-derived MAP and CFRs (all $r > 0.85$) during full inflation. There was also a significant but less robust positive correlation ($r > 0.65$) noted during partial deflation of the pREBOA and restoration of partial distal perfusion. There was no statistically significant correlation between distal arterial pressures and CFRs using either standard arterial pressure monitoring or the COMPASS handheld device. While the balloon was fully inflated, there was an increase in the mean PMAP and a decrease in the mean distal MAP (Table 2). After partial inflation, mean distal MAP measurements demonstrated a gradual increasing trend.

DISCUSSION

This study is among the first to examine the question of the impact of both complete and partial REBOA on carotid

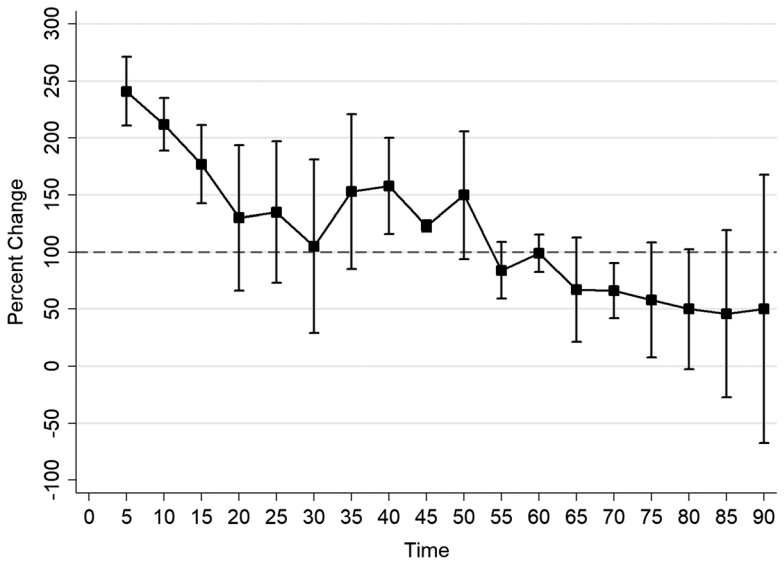

Figure 1. Carotid flow (as a percentage of baseline) compared with time in minutes.

TABLE 1. Correlation Between Proximal COMPASS Measurement and CFR by Experimental Time

Time From Injury, min	No. Surviving Pigs	CFR and Proximal COMPASS	CFR and Proximal MAP	Proximal COMPASS and Proximal MAP
0 (Full inflation)	10	0.320 (0.368)	0.445 (0.197)	0.979 (<0.001)
5	10	0.850 (0.002)	0.919 (<0.001)	0.921 (<0.001)
10	10	0.857 (0.002)	0.861 (0.001)	0.999 (<0.001)
15 (Partial inflation)	9	0.961 (<0.001)	0.965 (<0.001)	0.999 (<0.001)
20	7	0.906 (0.005)	0.898 (0.006)	0.995 (<0.001)
25	6	0.922 (0.009)	0.921 (0.009)	0.999 (<0.001)
30	5	0.929 (0.022)	0.931 (0.021)	0.999 (<0.001)
35	5	0.750 (0.144)	0.714 (0.175)	0.999 (<0.001)
40	4	0.681 (0.319)	0.733 (0.267)	0.997 (0.003)
Overall	—	0.860 (<0.001)	0.871 (<0.001)	0.990 (<0.001)

Values are shown as r (p value).

artery flow rates and their relationship (if any) to systemic MAP measurements in a realistic model of hemorrhagic shock and simulated battlefield resuscitation. This is a particularly relevant question for both military and civilian trauma populations because of the relatively common presence of associated traumatic brain injury among severely injured patients with NCTH. Although the ability of REBOA to provide temporary control of active bleeding from abdominal or pelvic sources by occluding aortic flow is commonly highlighted, the equally important desired physiologic effect is to augment central (cerebral and cardiac) perfusion in the patient with severe hypotension and/or shock. Thus, careful examination of the effect of REBOA on cerebral perfusion and cerebral blood flow, and other measures relevant to shock or the presence of traumatic brain injury (TBI) are sorely needed. These data are also important to critically assess the commonly cited concern about TBI or known/suspected intracranial hemorrhage being a relative contraindication to REBOA and whether REBOA is actually harmful or helpful in this scenario. Finally, the increasing recent interest in partial REBOA and the development of the pREBOA catheter have now raised additional concerns about the effects of partially restoring distal aortic flow on proximal cerebral pressures and blood flow.

This study has demonstrated that pREBOA is effective in maintaining proximal carotid flow, maintaining perfusion to proximal vital organs to include the brain, in a severe hemorrhagic swine model. This study has shown that, when fully inflated, the pREBOA provides complete aortic occlusion similar to the standard ER-REBOA device, with supraphysiologic increases in CFR but maintaining near physiologic levels of CFRs when titrated to a distal flow rate of 0.7 L/min. The CFR also positively correlated with PMAP during full and partial occlusion. This shows that PMAP may be used as an acceptable surrogate for CFR and guide to the effect of complete and partial REBOA on cerebral perfusion or pressures.

Previous studies have modeled the flow distally past the pREBOA,[16] the diminished ischemic injuries to distal organs,[17,19,20] and the ease of titrating the pREBOA to modify flow when compared with the ER-REBOA.[15] This is the first study that examined the changes to proximal perfusion in a severe hemorrhagic shock model using pREBOA. A previous study, using a swine model with a controlled hemorrhage, used partial aortic clamping to allow for a distal flow rate of 0.3 L/min and compared this with complete REBOA. This showed increased PMAP and CFRs for REBOA when compared with partial occlusion, but no difference in carotid perfusion pressures, although both partial occlusion and REBOA showed no worsening of TBI based on imaging.[20] However, in a rodent model with penetrating ballistic-like brain injury, aortic occlusion was shown to increase intracranial pressure, which could lead to a worsening of TBI.[21]

Previous literature has attempted to use ER-REBOA to act as the bivalve pREBOA acts[22] but has shown difficulty with titration of flow rates and maintenance of proximal pressure. ER-REBOA has a steep inflection point that results in rapid increase in distal aortic flow and subsequent decrease in PMAP,[9] but

TABLE 2. Mean Values Over Time for MAP Measurements and CFR

Time From Injury, min	No. Surviving Pigs	CFR, L/min	PMAP, mm Hg	Distal MAP, mm Hg
0 (Full inflation)	10	0.17 (0.08)	50.3 (9.2)	49.8 (10.0)
5	10	0.46 (0.22)	89.4 (37.5)	12.1 (5.6)
10	10	0.36 (0.19)	77.0 (34.4)	10.1 (4.9)
15 (Partial inflation)	9	0.31 (0.18)	66.3 (34.4)	9.7 (5.2)
20	7	0.21 (0.20)	61.6 (36.5)	23.1 (8.4)
25	6	0.22 (0.19)	54.0 (43.4)	25.0 (7.8)
30	5	0.20 (0.17)	54.4 (49.5)	32.8 (12.4)
35	5	0.29 (0.23)	78.4 (56.2)	29.0 (6.9)
40	4	0.30 (0.13)	79.3 (34.5)	29.5 (3.8)

PMAP has been shown to be a poor marker for distal blood flow when restoring distal aortic perfusion after ER-REBOA; however, there is a positive linear relationship between distal MAP and distal aortic blood flow.[23] When attempting to deflate the ER-REBOA to meet a goal distal aortic flow, the ER-REBOA needed a significant higher number of adjustments to maintain the goal flow rate, compared with pREBOA.[15]

The use of REBOA has been fraught with complications, related to both downstream ischemia and proximal supraphysiologic perfusion. Acute kidney injury,[11,12] distal limb ischemia,[13] and other vascular complications may occur from ER-REBOA placement. Proximal complications include cardiac complications. Prolonged ER-REBOA inflation in zone 1 has induced type 2 myocardial ischemia in a swine model,[24] as well as heart failure in other studies.[10] Previous studies on aortic cross clamping have shown negative effects on myocardial function, as well as increased risk of neurologic dysfunction.[14,25]

Resuscitative endovascular balloon occlusion of the aorta has also been implicated in worse outcomes in patients with severe TBI. A previous study in Japan showed increased mortality in patients with head injury and low Glasgow Coma Scale,[26] while a case report showed significant worsening of a TBI in an elderly patient with pelvic hemorrhage after complete REBOA was placed in zone 3, leading to herniation and death of the patient.[27] However, these data remain retrospective, poorly controlled, and, in the example of the case report where causation cannot be inferred, entirely anecdotal. Further study, in both large animal translational models and clinical case series or trials, will be required to accurately determine the impact or risks of REBOA in the setting of TBI. While there is the obvious concern of elevated cerebrovascular pressures and flow leading to higher intracranial pressures or worsening of intracranial hemorrhage, there is the equally plausible assertion that REBOA could maintain and augment cerebral perfusion in patients with TBI and hemorrhagic shock.

There are some limitations to this study. Because of supply constraints, there was not a true control group evaluating nonhemorrhagic shock swine models and comparing data. A distal flow rate of 0.7 L/min during pREBOA was used because of previous studies by the authors validating ideal flow distal flow rates. There was a concurrent study evaluating pREBOA that showed survival benefit and decreased distal ischemia using a distal flow rate of 0.5 L/min, but this was published after the completion of the study.[17] There was no evaluation of pREBOA placement outside of zone 1, while there have been many studies showing efficacy in using nonfluoroscopic methods of confirming placement of REBOA.[28–31] It would be beneficial for future studies to evaluate proximal perfusion with a more distal placement of the pREBOA. This study was modeling a prolonged field scenario where immediate surgery would not be available. Future studies that use a model with surgical hemorrhagic control before balloon deflation would be expected to have different results.

There has been recent research into using REBOA in prehospital settings in both the civilian[32] and military[8,33] settings. With diminished distal ischemia and a more normal proximal perfusion state, pREBOA may be the technology that is able to safely expand the use of REBOA outside of the hospital, to lengthen the window of time to transport an injured patient to an environment with definitive surgical care. Further studies to identify safety parameters, as well as ideal patient selection, are needed before REBOA becoming a viable tool in the prehospital setting.

The COMPASS device, was used with ease in our study and showed correlation with the CFR, similar to the standard arterial line set up measuring PMAP. There are currently further studies by the authors of this study validating the accuracy and precision of the COMPASS device with ER-REBOA and pREBOA. If validated, this device may prove useful in both civilian and military prehospital settings where REBOA technologies may be implemented.

With the increasing use of REBOA in traumatically injured patients in both the military and civilian environments, it is critical to understand both the downstream and upstream physiologic effects of balloon inflation and partial deflation. Complete aortic occlusion is associated with a marked rise in CFRs that are approximately double that of baseline. Partial REBOA is an important new adjunct for treating hemorrhagic shock and is effective at maintaining proximal CFR at near-normal levels in a relevant severe hemorrhagic shock model. There is a strong correlation between MAP and CFR, and changes in MAP may be a reliable surrogate for corresponding changes in CFR.

AUTHORSHIP

D.A.B. and L.E.W. conducted the literature search. D.A.B., M.J.C., L.E.W., M.J.M., and M.J.K. designed the study. D.A.B., M.J.C., and J.J.L. performed all procedures. A.J.S. performed veterinary care. D.A.B., M.J.C., J.J.L., M.J.M., M.J.K. and R.Y.C., acquired and analyzed the data. D.A.B., M.J.C., J.J.L., M.J.M., M.J.K., R.Y.C., A.J.S., and L.E.W. participated in drafting the article and critically revising it. All authors approved the final version of the article.

DISCLOSURE

The authors declare no conflicts of interest. This work was funded by the Telemedicine and Advanced Technology Research Center at the United States Army Medical Research and Materiel Command through the AMEDD Advanced Medical Technology Initiative. M.J.M. is an EAST member.

REFERENCES

1. Stannard A, Morrison JJ, Scott DJ, Ivatury RA, Ross JD, Rasmussen TE. The epidemiology of noncompressible torso hemorrhage in the wars in Iraq and Afghanistan. *J Trauma Acute Care Surg*. 2013;74(3):830–834.
2. Kisat M, Morrison JJ, Hashmi ZG, Efron DT, Rasmussen TE, Haider AH. Epidemiology and outcomes of non-compressible torso hemorrhage. *J Surg Res*. 2013;184(1):414–421.
3. Stannard A, Eliason JL, Rasmussen TE. Resuscitative endovascular balloon occlusion of the aorta (REBOA) as an adjunct for hemorrhagic shock. *J Trauma*. 2011;71(6):1869–1872.
4. Lallemand MS, Moe DM, McClellan JM, Smith JP, Daab L, Marko S, Tran N, Starnes B, Martin MJ. Resuscitative endovascular balloon occlusion of the aorta for major abdominal venous injury in a porcine hemorrhagic shock model. *J Trauma Acute Care Surg*. 2017;83(2):230–236.
5. Joseph B, Zeeshan M, Sakran JV, Hamidi M, Kulvatunyou N, Khan M, O'Keeffe T, Rhee P. Nationwide analysis of resuscitative endovascular balloon occlusion of the aorta in civilian trauma. *JAMA Surg*. 2019;154(6):500–508.
6. Hoehn MR, Hansraj NZ, Pasley AM, Brenner M, Cox SR, Pasley JD, Diaz JJ, Scalea T. Resuscitative endovascular balloon occlusion of the aorta for non-traumatic intra-abdominal hemorrhage. *Eur J Trauma Emerg Surg*. 2019;45(4):713–718.
7. Kauvar DS, Schechtman DW, Thomas SB, Prince MD, De Guzman R, Polykratis IA, Kheirabadi BS, Dubick MA. Effect of partial and complete aortic balloon occlusion on survival and shock in a swine model of uncontrolled splenic hemorrhage with delayed resuscitation. *J Trauma Acute Care Surg*. 2019;87(5):1026–1034.

8. Reva VA, Hörer TM, Makhnovskiy AI, Sokhranov MV, Samokhvalov IM, DuBose JJ. Field and en route resuscitative endovascular occlusion of the aorta: a feasible military reality? *J Trauma Acute Care Surg*. 2017;83(1 Suppl 1): S170–S176.
9. Davidson AJ, Russo RM, Ferencz SE, Cannon JW, Rasmussen TE, Neff LP, Johnson MA, Williams TK. Incremental balloon deflation following complete resuscitative endovascular balloon occlusion of the aorta results in steep inflection of flow and rapid reperfusion in a large animal model of hemorrhagic shock. *J Trauma Acute Care Surg*. 2017;83(1):139–143.
10. Long B, Hafen L, Koyfman A, Gottlieb M. Resuscitative endovascular balloon occlusion of the aorta: a review for emergency clinicians. *J Emerg Med*. 2019;56(6):687–697.
11. Davidson AJ, Russo RM, Reva VA, et al, BEST Study Group. The pitfalls of resuscitative endovascular balloon occlusion of the aorta: risk factors and mitigation strategies. *J Trauma Acute Care Surg*. 2018;84(1):192–202. Erratum in: *J Trauma Acute Care Surg*. 2018 Mar;84(3):544.
12. Ribeiro Junior MAF, Feng CYD, Nguyen ATM, Rodrigues VC, Bechara GEK, de-Moura RR, Brenner M. The complications associated with resuscitative endovascular balloon occlusion of the aorta (REBOA). *World J Emerg Surg*. 2018;13:20.
13. Taylor JR 3rd, Harvin JA, Martin C, Holcomb JB, Moore LJ. Vascular complications from resuscitative endovascular balloon occlusion of the aorta: life over limb? *J Trauma Acute Care Surg*. 2017;83(1 Suppl 1):S120–S123.
14. Marini CP, Levison J, Caliendo F, Nathan IM, Cohen JR. Control of proximal hypertension during aortic cross-clamping: its effect on cerebrospinal fluid dynamics and spinal cord perfusion pressure. *Semin Thorac Cardiovasc Surg*. 1998;10(1):51–56.
15. Zilberman-Rudenko J, Behrens B, McCully B, et al. Use of bilobed partial resuscitative endovascular balloon occlusion of the aorta is logistically superior in prolonged management of a highly lethal aortic injury. *J Trauma Acute Care Surg*. 2020;89(3):464–473.
16. Matsumura Y, Higashi A, Izawa Y, Hishikawa S, Kondo H, Reva V, Oda S, Matsumoto J. Organ ischemia during partial resuscitative endovascular balloon occlusion of the aorta: dynamic 4D computed tomography in swine. *Sci Rep*. 2020;10(1):5680.
17. Forte DM, Do WS, Weiss JB, Sheldon RR, Kuckelman JP, Cook BA, Levine TC, Eckert MJ, Martin MJ. Validation of a novel partial resuscitative endovascular balloon occlusion of the aorta device in a swine hemorrhagic shock model: fine tuning flow to optimize bleeding control and reperfusion injury. *J Trauma Acute Care Surg*. 2020;89(1):58–67.
18. Butler FK Jr., Holcomb JB, Shackelford S, et al. Advanced resuscitative care in tactical combat casualty care: TCCC guidelines change 18-01:14 October 2018. *J Spec Oper Med*. 2018 Winter;18(4):37–55.
19. Sadeghi M, Hörer TM, Forsman D, Dogan EM, Jansson K, Kindler C, Skoog P, Nilsson KF. Blood pressure targeting by partial REBOA is possible in severe hemorrhagic shock in pigs and produces less circulatory, metabolic and inflammatory sequelae than total REBOA. *Injury*. 2018;49(12):2132–2141.
20. Williams TK, Neff LP, Johnson MA, Russo RM, Ferencz SA, Davidson AJ, Clement NF, Grayson JK, Rasmussen TE. Automated variable aortic control versus complete aortic occlusion in a swine model of hemorrhage. *J Trauma Acute Care Surg*. 2017;82(4):694–703.
21. Bailey ZS, Cardiff K, Yang X, Gilsdorf J, Shear D, Rasmussen TE, Leung LY. The effects of balloon occlusion of the aorta on cerebral blood flow, intracranial pressure, and brain tissue oxygen tension in a rodent model of penetrating ballistic-like brain injury. *Front Neurol*. 2019;10:1309.
22. DuBose JJ. How I do it: partial resuscitative endovascular balloon occlusion of the aorta (P-REBOA). *J Trauma Acute Care Surg*. 2017;83(1):197–199.
23. Johnson MA, Davidson AJ, Russo RM, Ferencz SE, Gotlib O, Rasmussen TE, Neff LP, Williams TK. Small changes, big effects: the hemodynamics of partial and complete aortic occlusion to inform next generation resuscitation techniques and technologies. *J Trauma Acute Care Surg*. 2017; 82(6):1106–1111.
24. Wasicek PJ, Teeter WA, Yang S, Banchs H, Galvagno SM, Hu P, Gamble WB, Hoehn MR, Scalea TM, Morrison JJ. Extended resuscitative endovascular balloon occlusion of the aorta (REBOA)–induced type 2 myocardial ischemia: a time-dependent penalty. *Trauma Surg Acute Care Open*. 2019;4(1): e000194.
25. Dunn E, Prager RL, Fry W, Kirsh MM. The effect of abdominal aortic cross-clamping on myocardial function. *J Surg Res*. 1977;22(5):463–468.
26. Norii T, Crandall C, Terasaka Y. Survival of severe blunt trauma patients treated with resuscitative endovascular balloon occlusion of the aorta compared with propensity score-adjusted untreated patients. *J Trauma Acute Care Surg*. 2015;78(4):721–728.
27. Hayaki A, Nobuichiro UA, Ab T, et al. "REBOA" — is it really safe? A case with massive intracranial hemorrhage possibly due to endovascular balloon occlusion of the aorta (REBOA). *Am J Case Rep*. 2016;17: 810–813.
28. Wessels LE, Wallace JD, Bowie J, Butler WJ, Spalding C, Krzyzaniak M. Radiofrequency identification of the ER-REBOA: confirmation of placement without fluoroscopy. *Mil Med*. 2019;184(3–4):e285–e289.
29. Scott DJ, Eliason JL, Villamaria C, Morrison JJ, Houston R4th, Spencer JR, Rasmussen TE. A novel fluoroscopy-free, resuscitative endovascular aortic balloon occlusion system in a model of hemorrhagic shock. *J Trauma Acute Care Surg*. 2013;75(1):122–128.
30. Linnebur M, Inaba K, Haltmeier T, Rasmussen TE, Smith J, Mendelsberg R, Grabo D, Demetriades D. Emergent non-image-guided resuscitative endovascular balloon occlusion of the aorta (REBOA) catheter placement: a cadaver-based study. *J Trauma Acute Care Surg*. 2016;81(3):453–457.
31. Ogura T, Lefor AK, Nakamura M, Fujizuka K, Shiroto K, Nakano M. Ultrasound-guided resuscitative endovascular balloon occlusion of the aorta in the resuscitation area. *J Emerg Med*. 2017;52(5):715–722.
32. Lendrum R, Perkins Z, Chana M, Marsden M, Davenport R, Grier G, Sadek S, Davies G. Pre-hospital resuscitative endovascular balloon occlusion of the aorta (REBOA) for exsanguinating pelvic haemorrhage. *Resuscitation*. 2019;135:6–13.
33. Teeter W, Romagnoli A, Glaser J, Fisher AD, Pasley J, Scheele B, Hoehn M, Brenner M. Resuscitative endovascular balloon occlusion of the aorta: pushing care forward. *J Spec Oper Med*. 2017 Spring;17:17–21.

DISCUSSION

The management of hemorrhagic shock has evolved dramatically improved over the last two decades. Still, injuries such as noncompressible truncal hemorrhage pose a continued challenge in management. Resuscitative endovascular balloon occlusion of the aorta (REBOA) is a relatively new technology whose indication has been hotly debated as of recent. Proponents state that with practice the technology can be placed quickly, maintain cerebral and cardiac perfusion, and prevent massive exsanguination. Critics highlight the complications associated with the technology, which include supraphysiologic cerebral and cardiac perfusion exacerbating traumatic brain injury and heart failure, distal ischemia to the abdominal organs, and potential limb loss due to technical issues. Partial REBOA (pREBOA) is the newest form of the technology which could mitigate some of these complications by allowing continued distal flow. The authors performed an animal study to demonstrate that partial REBOA provides adequate brain perfusion in the setting of uncontrolled hemorrhage and can potentially be titrated in a clinical setting.

The authors were successful in demonstrating that pREBOA when fully inflated provided perfusion that was supraphysiologic to baseline and when partially inflated provided perfusion that was similar to baseline during uncontrolled hemorrhage. This data demonstrates the potential benefit to the use of pREBOA when prolonged ischemia is of concern such as in the battlefield, the pre-hospital setting or in transfer between facilities. One question that remains is how do complete or partial occlusion cerebral perfusion values compare to a non-injured (or prior to injury) measurements? The answer to this question may be very telling as the effects of pREBOA are difficult to assess without a comparison to a true baseline.

A second and equally valid issue is whether carotid flow rates correlate with cerebral perfusion, especially in the setting of low flow states where autoregulation may or may not be in effect. This evidence may help answer if traumatic brain injury is a true contraindication to REBOA. If future studies can answer this question, we may be much closer to a viable indication for pREBOA in the management of non-compressible torso trauma.

—Madhu Subramanian, MD
Baltimore, MD