Not ready for prime time: Intermittent versus partial resuscitative endovascular balloon occlusion of the aorta for prolonged hemorrhage control in a highly lethal porcine injury model

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BACKGROUND: Partial resuscitative endovascular balloon occlusion of the aorta (pREBOA) and intermittent REBOA (iREBOA) are techniques to extend the therapeutic duration of REBOA by balloon titration for distal flow or cyclical balloon inflation/deflation to allow transient distal flow, respectively. We hypothesized that manually titrated pREBOA would reduce blood losses and ischemic burden when compared with iREBOA.

METHODS: Following 20% blood volume controlled hemorrhage, 10 anesthetized pigs underwent uncontrolled hemorrhage from the right iliac artery and vein. Once in hemorrhagic shock, animals underwent 15 minutes of complete zone 1 REBOA followed by 75 minutes of either pREBOA or iREBOA (n = 5/group). After 90 minutes, definitive hemorrhage control was obtained, animals were resuscitated with the remaining collected blood, and then received 2 hours of critical care.

RESULTS: There were no differences in mortality. Animals randomized to iREBOA spent a larger portion of the time at full occlusion when compared with pREBOA (median, 70 minutes; interquartile range [IQR], 70–80 vs. median, 20 minutes; IQR, 20–40, respectively; $p = 0.008$). While the average blood pressure during the intervention period was equivalent between groups, this was offset by large fluctuations in blood pressure and significantly more rescue occlusions for hypotension with iREBOA. Despite lower maximum aortic flow rates, the pREBOA group tolerated a greater total amount of distal aortic flow during the intervention period (median, 20.9 L; IQR, 20.1–23.0 vs. median, 9.8 L; IQR, 6.8–10.3; $p = 0.03$) with equivalent abdominal blood losses. Final plasma lactate and creatinine concentrations were equivalent, although iREBOA animals had increased duodenal edema on histology.

CONCLUSION: Compared with iREBOA, pREBOA reduced the time spent at full occlusion and the number of precipitous drops in proximal mean arterial pressure while delivering more distal aortic flow but not increasing total blood loss in this highly lethal injury model. Neither technique demonstrated a survival benefit. Further refinement of these techniques is necessary before clinical guidelines are issued. (*J Trauma Acute Care Surg.* 2020;88: 298–304. Copyright © 2019 Wolters Kluwer Health, Inc. All rights reserved.)

KEY WORDS: REBOA; trauma; hemorrhage; partial REBOA; intermittent REBOA.

Resuscitative endovascular balloon occlusion of the aorta (REBOA) is increasingly utilized as a therapy for noncompressible torso hemorrhage (NCTH).[1–3] This technique prolongs survival prior to definitive surgical hemostasis by decreasing distal hemorrhage below the level of occlusion and augmenting proximal perfusion to the heart, lungs, and brain. Yet, its maximal duration is limited to 30 minutes, as the benefits of REBOA are quickly offset by progressive ischemia below the level of occlusion.[2,4,5]

To address these limitations, other flow strategies have been proposed as an alternative to complete occlusion, including partial REBOA (pREBOA) and intermittent REBOA (iREBOA).[4,6–10] Conflicting studies advocating one approach over another are emerging, however, the optimal balance of aortic flow to support proximal hemodynamics while limiting distal ischemia is still uncertain. Moreover, not all experiments have included a postintervention critical care phase to allow for the ischemia-reperfusion injury to manifest.[6,7] While it is possible to keep a study animal alive during prolonged periods of aortic occlusion, both translational studies and clinical experience indicate that simply surviving the REBOA intervention does not correlate with short-term survival in the intensive care unit, let alone a meaningful clinical outcome.[9,10] The lack of a postocclusion resuscitation period, along with heterogeneity in the hemodynamic data reported in recent translational research publications, confound efforts to understand the effectiveness of these interventions.

Despite these obstacles, the need for "next generation" therapies with longer effective durations is pressing.[11] For the

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The animals involved in this study were procured, maintained, and used in accordance with the Laboratory Animal Welfare Act of 1966, as amended, and NIH 80–23, Guide for the Care and Use of Laboratory Animals, National Research Council.

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military, this need has led to updated Tactical Combat Casualty Care REBOA guidelines that advocate for the early transition to iREBOA if prolonged occlusion times are anticipated.[12] However, it is unclear if this clinical recommendation can be supported by the available evidence given the lack of data to describe the postocclusion phase. We hypothesize that iREBOA will produce more hemodynamic instability, more bleeding, and more ischemia-reperfusion injury than pREBOA in an established and highly lethal swine model of combined arterial and venous injury.

METHODS

The Institutional Animal Care and Use Committee at Travis Air Force Base, California approved this study. Animal care and use was in strict compliance with the Guide for the Care and Use of Laboratory Animals in a facility accredited by Association for Assessment and Accreditation of Laboratory Animal Care (AAALAC) International. Healthy adult, castrate male Yorkshire-cross swine (*Sus scrofa,* S and S Farms, Ramona, CA) weighing between 60 kg and 95 kg were acclimated for a minimum of 7 days prior to experimentation.

All animals were subjected to a controlled 20% total blood volume hemorrhage over 30 minutes. Animals were then subjected to a free bleed from a combined iliac artery and vein injury until the mean arterial pressure (MAP) reached 40 mm Hg. During hemorrhage, animals were randomized to 75 minutes of either iREBOA or pREBOA in Zone 1 (n = 5 animals per group). After the intervention period, surgical control of the hemorrhage was obtained, and animals were resuscitated with their shed blood. Animals then received protocolized critical care, during which vasopressor and isotonic fluid administration were provided based on predefined physiologic parameters.

Animal Preparation

Animals were fasted for 12 hours prior to study, but had access to water. On the day of surgery, they were premedicated with 6.6 mg/kg intramuscular tiletamine/zolazepam. Following isoflurane induction and endotracheal intubation, general anesthesia was maintained with 1.5% to 3% isoflurane and 100% oxygen which was titrated to 40% oxygen to maintain a pulse oximetry between 92% and 98%. If needed, a 10-mL/kg bolus of balanced isotonic crystalloid solution (Plasmalyte A; Baxter Healthcare Corporation, Deerfield, IL) was delivered intravenously prior to the beginning of the experiment to achieve a target MAP between 65 mm Hg and 75 mm Hg. An infusion of norepinephrine ($0.01\ \mu g \cdot kg^{-1} \cdot min^{-1}$) was instituted to overcome the vasodilatory effects of general anesthesia if the fluid bolus did not increase the MAP within the target range. Animals were mechanically ventilated with 6 mL/kg to 10 mL/kg and 4 cm H_2O positive end expiratory pressure to maintain end-tidal CO_2 at 40 ± 5 mm Hg. An underbody warmer was used to maintain core body temperature between 35°C and 37°C.

A splenectomy was performed to minimize hemodynamic variation from autotransfusion, and the bladder was catheterized via a mini cystotomy. A perivascular flow probe (Transonic Systems Inc, Ithaca, NY) was placed on the supraceliac aorta proximal to ligated intercostal arteries to minimize any flow between the flow probe and the aortic occlusion balloon catheter. A 9-Fr introducer sheath was placed percutaneously into the left common femoral artery, and a balloon catheter (ER-REBOA; Prytime Medical, Boerne, TX) was positioned just distal to the aortic flow probe. Venous catheters were placed for resuscitation and medication infusions.

Creation of Iliac Artery and Vein Injury

This model has been previously described.[6] Briefly, the right common iliac artery and vein were exposed and dissected free from surrounding connective tissues. Vessel loops were placed proximal and distal to the planned injury site for rapid hemorrhage control at the end of the intervention. Using Seldinger technique, 12-Fr and 16-Fr vessel dilators were placed in the iliac artery and vein, respectively. The laparotomy was closed around the dilators and the vessel loops. At time 0, the dilators were removed from the vessels, and the abdominal closure was tightened to ensure free hemorrhage into the abdomen.

Intervention Phase

Animals in both groups received 15 minutes of complete REBOA for hemorrhage control and clot stabilization. For animals randomized to the iREBOA strategy, the balloon was completely deflated over 30 seconds at time 15 minutes using a standard 20-mL syringe. Inflation/deflation criteria for the iREBOA group were based on published studies of iREBOA that indicated that a pressure-based trigger for reocclusion was superior to a time-based schedule.[6] If the proximal MAP *in either group* dropped below 40 mm Hg, the balloon was inflated to complete occlusion for 10 minutes. After 10 minutes, if the proximal MAP was above 40 mm Hg, the balloon was completely deflated over 30 seconds (iREBOA) or partially deflated to achieve the distal pressure goal (pREBOA). For animals randomized to the pREBOA strategy, the balloon was partially deflated using a rotational inflation device (Encore-26; Boston Scientific, Marlborough, MA) until the distal pressure was increased by 7 mm Hg to 10 mm Hg over the observed distal pressure during occlusion.

The 20% shed blood was divided into four equal aliquots. Administration was triggered by the need for rescue occlusions and given up to three times during the intervention phase, leaving at least one fourth of the shed blood available for the resuscitation phase. Death was predefined as a proximal MAP less than 20 mm Hg for 5 minutes or asystole at any time.

Resuscitation and Critical Care Phase

After 90 minutes, the animals received the remaining shed blood. Simultaneously, the abdomen was opened, the injured artery and vein were ligated to obtain hemorrhage control, and blood loss was quantified. The REBOA balloon was deflated over 10 minutes in both groups and the catheters removed. During the critical care phase, animals received Plasmalyte-A boluses or norepinephrine when their MAP was below 60 mm Hg based on our routine critical care algorithms.[8] The critical care phase was concluded 120 minutes after balloon deflation with collection of final blood and urine samples.

Data Collection and Analysis

The primary outcome was mortality. Secondary outcomes of interest included hemodynamics during the intervention period and laboratory measurements of ischemia. Physiologic parameters were continuously collected using an automated

data acquisition system (PowerLab; ADInstruments, Colorado Springs, CO) including heart rate, proximal and distal mean arterial blood pressure, aortic flow, central venous pressure, core temperature, pulse oxygenation, and end tidal carbon dioxide concentration. At necropsy, heart, lung, liver, kidneys, and duodenum specimens were harvested and stored in 10% buffered formalin solution.

Heart sections were scored for myocardial ischemic necrosis (coagulation necrosis with eosinophilia, nuclear pyknosis and karyorrhexis, loss of striations) and subendocardial hemorrhage on a Likert scale (0, not present; 1, focal area; 2, multifocal areas; 3, locally extensive; and 4, diffuse).[13] Subendocardial hemorrhage was also scored on a Likert scale (0, absent; 1, hemorrhage restricted to the subendocardium; 2, small amount of hemorrhage between myocardial cells; 3, locally extensive hemorrhage between myocardial cells; and 4, transmural hemorrhage). Lung sections were evaluated for congestion, edema, and inflammation. Kidney sections were scored for acute tubular necrosis, and duodenums were scored for villous coagulation necrosis all with a Likert scale similar to the heart sections. All histologic analyses were done by a veterinarian blinded to the treatment.

Statistical Analysis

Although this was a pilot study, we performed a power analysis using G*Power (version 3.1.9.3) that demonstrated we would be able to differentiate a 90% difference in mortality with an 80% power and an alpha of 0.05 with five animals in each group. Data analysis was performed with STATA version 14.0 (Stata Corporation, Bryan, TX). Continuous variables are presented as means and standard deviations if normally distributed and as medians with interquartile ranges (IQRs) if not. *t* Tests were used to compare normally distributed continuous data, and χ^2 or Wilcoxon rank-sum tests were used for data that were not normally distributed. Statistical significance was set at *p* values less than 0.05.

RESULTS

Baseline physiologic parameters and laboratory values were similar between groups (Table 1). There was no difference in intra-abdominal hemorrhage volume at damage control laparotomy (Table 2). In both groups, three of five animals survived to the end of the study. Median survival time (min) was 210 minutes (IQR, 108–210) for pREBOA animals and 210 minutes for iREBOA animals (IQR, 180–210; $p = 0.81$). There were no differences in volume of blood administered during the intervention period (Table 2).

TABLE 1. Baseline Data

	Intermittent (n = 5)		Partial (n = 5)		*p*
Male (n, %)	5	100%	5	100%	1.0
Weight (med, IQR), kg	68	68–69	72	66–72	0.60
pH (mean, SD)	7.5	0.0	7.5	0.0	0.3
Hematocrit (mean, SD), %	31.4	1.1	30.2	1.1	0.13
P/F (mean, SD), mm Hg	4.6	0.9	5.0	0.7	0.53
Creatinine (mean, SD), mg/dL	1.6	0.1	1.5	0.1	0.16
Baseline aortic flow (mean, SD), L/min	2.7	0.5	2.6	0.8	0.87
Proximal pressure (mean, SD), mm Hg	67.0	8.5	66.8	8.4	0.97
CVP (mean, SD), mm Hg	3.6	1.3	4.0	1.1	0.55
Time to MAP < 40 mm Hg (median, IQR), s	15	10–19	30	11–34	0.53

Biochemical and Histologic Markers of Tissue Ischemia

The lactate levels following resuscitation were equivalent between groups during the intervention phase and the critical care phase (Fig. 1, Table 3). There was no difference in creatinine at the end of the study in the animals that survived (Table 3). On histologic analysis, animals in both groups demonstrated similar degrees of renal tubular necrosis, but animals in the iREBOA had significantly more duodenal mucosal necrosis when compared with animals in the pREBOA group ($p = 0.04$) Edema of the duodenal lamina propria and submucosa was present in four (80%) of five pigs in the iREBOA versus two (40%) of five pigs in the pREBOA group, although the difference was not significant ($p = 0.20$; Table 4).

Hemodynamic Stability

There were no differences between the two groups in the overall average pMAP during the intervention phase (iREBOA, 80 mm Hg [IQR, 65–88 mm Hg]; pREBOA, 71 mm Hg [IQR, 68–73 mm Hg]; $p = 0.46$). Yet, the iREBOA group spent a greater amount of time in a state of profound hypotension with a pMAP less than 35 mm Hg (iREBOA, 73 seconds [IQR, 19–86 seconds]; pREBOA, 0 seconds [IQR, 0–4 seconds]; $p = 0.01$) (Table 2, Fig. 2). The pREBOA group spent a longer duration within an optimal pMAP range of 60 mm Hg to 90 mm Hg, but this did not reach statistical significance (iREBOA, 31 minutes [IQR, 33–38 minutes]; pREBOA, 51 minutes [IQR, 41–58 minutes]; $p = 0.25$; Table 2). The iREBOA group required an average of seven rescue occlusions (IQR, 7–8) for proximal MAP below 40 mm Hg during the intervention phase compared with two rescue occlusions (IQR, 2–4; $p = 0.008$) in the pREBOA group. The time from the reinitiation of aortic flow to need for first rescue occlusion in the iREBOA group was 0.6 minutes (IQR, 0.4–0.7 minutes) and 40.9 minutes in the pREBOA group (IQR, 7.5–46.7 minutes; $p = 0.0003$). There were two premature REBOA catheter balloon ruptures in the iREBOA group that prompted rapid deployment of new REBOA catheters and no ruptures in the pREBOA group.

Distal Perfusion

Total perfusion distal to the balloon was calculated from the aortic flow measurements. The pREBOA animals had significantly higher total amounts of flow over the 90-minute intervention period when compared with iREBOA animals (pREBOA, 20.9 L [IQR, 20.1–23.0 L]; iREBOA, 9.8 L [IQR, 6.8–10.3 L]; $p = 0.03$). During the intervention period, the iREBOA animals had an average of fourfold higher maximum aortic flow during balloon deflation (2.4 L/min [IQR, 1.1–3.3 L/min]) when compared with pREBOA animals (0.6 L/min [IQR, 0.6–0.7 L/min]; $p = 0.03$, Table 2).

TABLE 2. Intervention Period

	Intermittent (n = 5)		Partial (n = 5)		*p*
Free hemorrhage blood loss (median, IQR), mL	486	452–679	495	434–574	0.92
Total blood loss (median, IQR), mL	1258	1,196–1,303	1,223	1,161–1,518	0.92
No. rescue blood transfusions (median, IQR)	3	3–3	2	2–3	0.053
No. hypotensive episodes triggering rescue occlusions (median, IQR)	7	7–8	2	2–4	0.008
Total occlusion duration (median, IQR), min	70	70–80	20	20–40	0.008
Time from initiation of aortic flow to rescue occlusion (median, IQR), min	0.6	0.4–0.7	40.9	7.5–46.7	0.0003
Maximum aortic flow during intervention (median, IQR), L/min	2.4	1.1–3.3	0.6	0.6–0.7	0.03
Total distal blood flow delivered over the 90-min intervention period (median, IQR), L	9.8	6.8–10.3	20.9	20.1–23.0	0.03
Average pMAP during intervention, mm Hg	80	65–88	71	68–73	0.46
Time at profound hypotension (MAP < 35 mm Hg, seconds) (median, IQR)	73	19–86	0	0–4	0.01
Time within normotensive MAP range (60–90 mm Hg) (median, IQR), min	31	33–38	51	41–58	0.25

Ischemia-Reperfusion

To quantify ischemia-reperfusion injury, all animals entered into a 2-hour critical care period. During the critical care period, two animals in each group died. Both groups required similar amounts of intravenous crystalloids and similar amounts of norepinephrine (Table 3) to maintain a blood pressure of 60 mm Hg. In the animals that survived, there were no differences in average MAP or aortic flow during the critical care phase, and the serum lactate and creatinine concentrations were similar between the two groups at the end of the study (Table 3, Fig. 3, Fig. 4).

DISCUSSION

The present study comparing pREBOA to iREBOA sought to determine which resuscitation strategy provides the optimal outcome in a model of NCTH. While no survival benefit was observed in this small study, multiple findings suggest a stark difference between the two techniques. First, animals randomized to iREBOA spent a larger portion of time at full occlusion when compared with pREBOA animals. While the average blood pressure during the intervention period was equivalent between groups, this finding is misleading because any equivalence between the groups was offset by the large fluctuations in blood pressure and significantly more rescue occlusions for extreme hypotension in the iREBOA group. Despite lower maximal aortic flow rates, the pREBOA group had a greater total amount of distal perfusion during the intervention period when compared with the iREBOA with no differences in blood loss. Despite the different hemodynamic profiles of the two groups, the final plasma lactate and creatinine concentrations were the same by the end of the study.

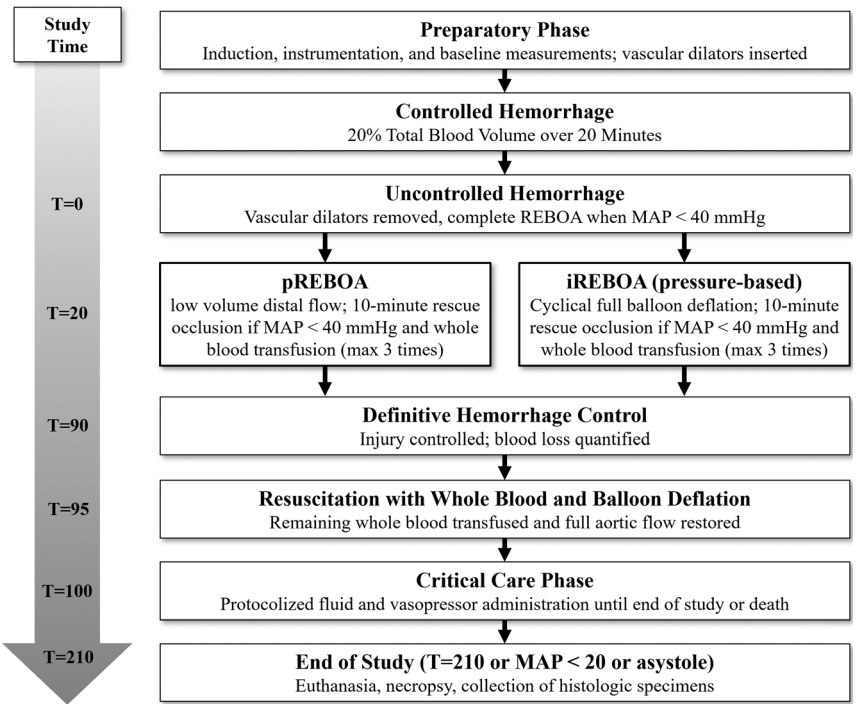

Figure 1. Study flow.

TABLE 3. Critical Care Period

	Intermittent (n = 5)		Partial (n = 5)		*p*
IV fluids during critical care (median, IQR), mL	9,831	5,527–10,773	9,267	800–10,350	0.60
Norepinephrine during critical care (median, IQR), μg	89	89–108	89.2	2.8–107	0.75
Proximal MAP during critical care (median, IQR), mm Hg	64	45–67	53	50–54	0.51
Aortic flow during critical care (median, IQR), L/min	2.5	1.0–3.4	3.5	2.7–3.8	0.13
Lactate at end of study (median, IQR), mmol/L	9.3	8.8–10.6	9.9	9.0–10.7	0.56
Creatinine at end of study (median, IQR), mg/dL	1.8	1.6–1.9	1.9	1.6–2.1	0.64
Survival time (median, IQR), min	210	180–210	210	108–210	0.81

These findings raise significant concern about the perceived safety and efficacy of iREBOA and pREBOA to extend the duration of intervention and minimize hemorrhage. This model utilized the same venous and arterial injury performed by Kuckelman et al.[6] who reported 100% survival with a 2-hour intervention period. However, during the model development phase of this pilot study, we were unable to survive the two animals that received 120 minutes of intervention. This mortality difference is likely the result of the lack of a critical care period in the Kuckelman study, as both of our model development animals died early in the critical care period. By terminating the study at the end of the intervention period, the total cumulative effect of the ischemia-reperfusion injury resulting from the intervention was not observed by those authors. In light of the 100% mortality during model development, we truncated the intervention period to 90 minutes as our prior work has shown the possibility of survival through critical care with 90-minute interventions.[9] Despite the shorter intervention period, both groups had a 40% mortality rate. All deaths occurred early in the critical care phase of the study, suggesting that the ischemia-reperfusion injury was profound in both groups. While it is possible that with more aggressive blood transfusion these animals might have survived, funding constraints prevented blood donor animals for extended resuscitation in this study. Nevertheless, our findings suggest that studies reporting 100% survival with any extended intervention phase are likely significantly underestimating the true morbidity and mortality of these interventions.[14]

Animals randomized to the iREBOA group experienced abrupt and severe hypotension immediately following balloon deflation, prompting early and frequent reinflation. The iREBOA group also spent more time in a profoundly hypotensive state below the threshold for reinflation of the balloon and had a trend toward decreased time at normal MAP. The rapid drop in blood pressure with deflation of the REBOA balloon is at odds with results from Kuckelman et al.,[6] who reported prolonged periods of balloon deflation with iREBOA. This discrepancy may be due to the size of animals that were used for their experiments (40–50 kg in the Kuckelman et al. study, 65–75 kg in this study). Small swine have a smaller aortic diameter which may result in the ER-REBOA catheter augmenting proximal blood pressure and minimizing bleeding even when fully deflated. We have observed proximal MAP augmentations of 15 mm Hg solely with insertion of the deflated catheter in small animals (unpublished data). This phenomenon is likely due to aortic vasoconstriction to near occlusion around an uninflated catheter. Together, these factors may account for the surprisingly short 45-minute cumulative amount of complete occlusion in the pressure-based iREBOA animals reported by Kuckelman over a 120-minute intervention and likely contributed to our inability to reproduce their results with larger animals. In either setting, allowing hypotension into the 40-mm Hg range will not translate into favorable clinical outcomes for humans due to the potential for cardiac arrest with profound hypotension, exacerbation of concomitant injuries like traumatic brain injury (TBI), and further hemorrhage with subsequent reintroduction of full aortic flow. This fact highlights the potential for harm when translating findings from hemorrhage control research with small animals to adult humans.

TABLE 4. Histology

Tissue	Intermittent (n = 5)		Partial (n = 5)		*p*
Heart (median, IQR)	3	2–3	2	1–3	0.50
Subendocardial hemorrhage (median, IQR)	2	1–3	2	1–2	0.75
Lung (median, IQR)		0–1	0	0–2	0.82
Liver (median, IQR)	4	2–4	3	2–3	0.33
Kidney (median, IQR)	2.5	2.5–2.5	2.5	1–3	0.91
Duodenum (median, IQR)	2	1–2	1	1–2	0.04

The fundamental methods of these techniques will lead to different total durations of full occlusion. Intermittent REBOA inherently results in longer durations at full occlusion and offsets this more profound distal ischemia with greater tissue perfusion with full balloon deflation. Partial REBOA seeks to maintain a constant, yet small amount of distal perfusion and to minimize full occlusion whenever possible. Significant differences were observed in the total amount of full occlusion between the two groups in this study, and this seems intuitive. However, the total amount of full occlusion in the pREBOA group could have been significantly shortened if not for an effort to keep the rescue occlusion duration of 10 minutes standardized between iREBOA and pREBOA animals. Like the iREBOA groups, the pREBOA rescue occlusion events resulted in a prompt and consistent rebound in the pMAP. Based on this observation, it may have been possible to initiate the gradual weaning of the balloon with pREBOA sooner than 10 minutes with a little downside, further decreasing the ischemic burden in the pREBOA group. To fully elucidate the differences between iREBOA and pREBOA, future studies are needed with more clinically realistic intervention

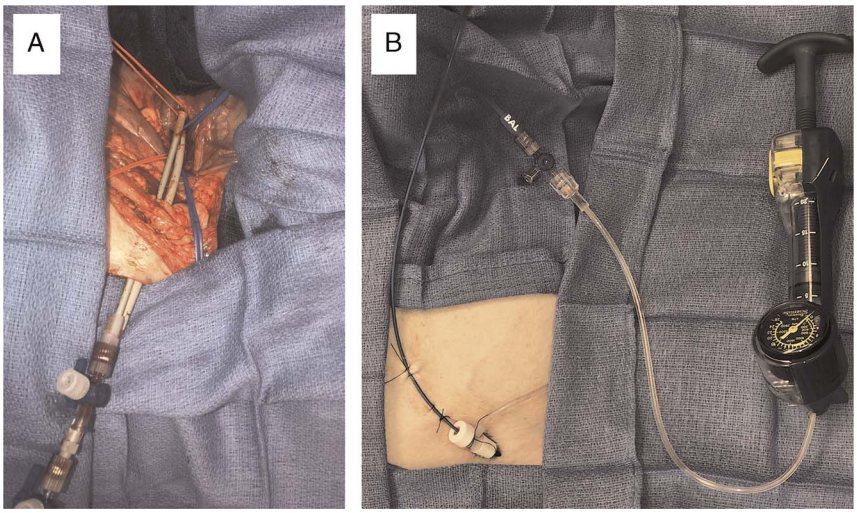

Figure 2. (*A*), Injury setup, (*B*) Rotational inflation device with ER-REBOA catheter.

periods that are unencumbered by attempts to maintain similarities between interventions.

LIMITATIONS

While we were able to achieve carefully titrated pREBOA in this experimental model, it was labor intensive, requiring nearly continuous, real-time balloon volume manipulation in a manual fashion from the researcher in response to changing downstream blood pressures. Not only does this approach require a skilled end-user but can also potentially introduce investigator bias into the results. This study is also limited by the small sample size which resulted from a lack of external funding and limited internal resources. Funding also precluded the use of blood donor animals for more aggressive resuscitation with the possibility of reversing the severe physiologic derangement. The lack of donor animals also ensured that none of the animals could be fully resuscitated to normovolemic states with blood, which may have resulted in higher rates of mortality. Finally, this was a nonsurvival study with only a 2-hour critical care period. It is well recognized that reperfusion injuries can manifest over as long as 24 hours. Therefore, larger limited-survival studies are needed to fully understand the differences between these interventions. These limitations notwithstanding, this study provides a valuable initial insight into the actual differences in hemodynamics and feasibility of iREBOA and pREBOA while creating a standard for minimum experimental design requirements and hemodynamic data reporting for future studies.

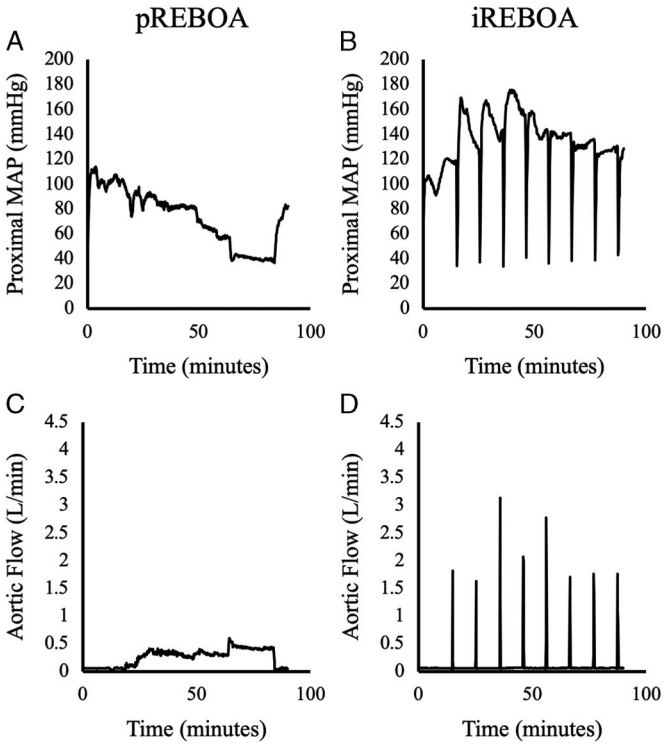

Figure 3. Representative continuous data from the intervention phase. (*A*), proximal MAP with pREBOA, (*B*) proximal MAP with iREBOA, (*C*) aortic flow with pREBOA, (*D*) aortic flow with iREBOA.

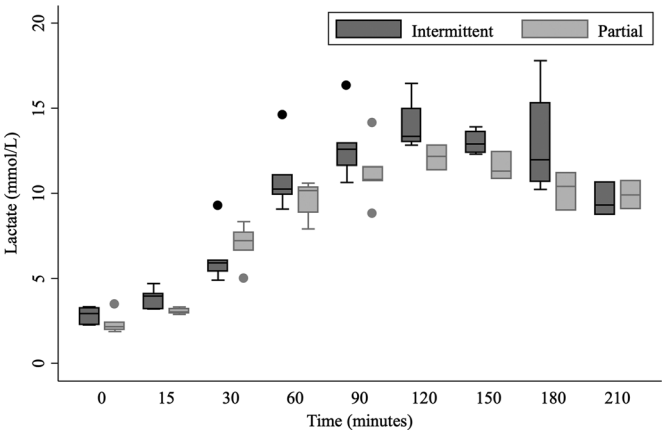

Figure 4. Arterial lactate concentrations over the experimental time.

CONCLUSION

In conclusion, we observed that iREBOA led to more episodes of severe hypotension, more frequent rescue occlusions, more time at complete aortic occlusion, and less total distal perfusion during the intervention period than did pREBOA. Moreover, these data suggest that the survivability following iREBOA use at 90 minutes—much less 120 minutes—may not be as robust as previously reported and described within current guidelines. Neither the iREBOA nor pREBOA technique demonstrated a survival benefit, and larger studies are desperately needed. Further refinement of these REBOA techniques is necessary before clinical guidelines on partial flow strategies for REBOA are issued.

AUTHORSHIP

All authors conducted and contributed to the literature search. All authors contributed to study design. A.J., G.H., C.B., C.C., M.S., and K.G. collected the data. A.J., G.H., K.G., L.N., and T.W. interpreted the data. A.J., G.H., K.G., L.N., and T.W. wrote the article. All authors critically revised the final article.

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DISCLOSURE

M.A.J., T.K.W., and L.P.N. are cofounders of Certus Critical Care, Inc. The remaining authors have no conflicts of interest.
The views expressed in this material are those of the authors and do not reflect the official policy or position of the US Government, the Department of Defense, the Department of the Air Force, or the University of Utah or Wake Forest Baptist Health Medical Center.

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