Conversion of the Abdominal Aortic and Junctional Tourniquet (AAJT) to Infrarenal Resuscitative Endovascular Balloon Occlusion of the Aorta (REBOA) Is Practical in a Swine Hemorrhage Model

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ABSTRACT

Background: Two methods of controlling pelvic and inguinal hemorrhage are the Abdominal Aortic and Junctional Tourniquet (AAJT; Compression Works) and resuscitative endovascular balloon occlusion of the aorta (REBOA). The AAJT can be applied quickly, but prolonged use may damage the bowel, inhibit ventilation, and obstruct surgical access. REBOA requires technical proficiency but avoids many of the complications associated with the AAJT. Conversion of the AAJT to REBOA would allow for field hemorrhage control with mitigation of the morbidity associated with prolonged AAJT use. **Methods:** Yorkshire male swine (n = 17; 70–90kg) underwent controlled 40% hemorrhage. Subsequently, AAJT was placed on the abdomen, midline, 2cm superior to the ilium, and inflated. After 1 hour, the animals were allocated to an additional 30 minutes of AAJT inflation (continuous AAJT occlusion [CAO]), REBOA placement with the AAJT inflated (overlapping aortic occlusion [OAO]), or REBOA placement following AAJT removal (sequential aortic occlusion [SAO]). Following removal, animals were observed for 3.5 hours. **Results:** No statistically significant differences in survival, blood pressure, or laboratory values were found following intervention. Conversion to REBOA was successful in all animals but one in the OAO group. REBOA placement time was 4.3 ± 2.9 minutes for OAO and 4.1 ± 1.8 minutes for SAO (p = .909). No animal had observable intestinal injury. **Conclusions:** Conversion of the AAJT to infrarenal REBOA is practical and effective, but access may be difficult while the AAJT is applied.

Keywords: *hemorrhage; Abdominal Aortic and Junctional Tourniquet; resuscitative endovascular balloon occlusion of the aorta; swine*

Introduction

Hemorrhage is responsible for a quarter of all deaths in combat and is the leading cause of potentially survivable battlefield deaths.[1] The liberal use of tourniquets has been successful in preventing exsanguination from extremity injuries, but noncompressible pelvic hemorrhage is particularly difficult to treat because of the inability to rapidly and safely compress the site of injury. Recently, specialized products, including expandable foams, injectable compressed sponges, and various junctional tourniquets, have been developed to manage hemorrhage from such injuries.[2,3] Unfortunately, none of these has been uniformly successful or gained widespread adoption. Two specific therapies capable of treating pelvic and junctional bleeding are the AAJT and REBOA.

The AAJT consists of a belt with a wedge-shaped inflatable bladder that can be applied to occlude blood flow at junctional sites (i.e., axilla and groin) or occlude the distal aorta and iliac vessels when placed in the lower abdomen. The AAJT was chosen for this study because of this ability to occlude blood flow in proximal inguinal and pelvic hemorrhage compared with more commonly used junctional tourniquets, such as the SAM Junctional Tourniquet (SAM Medical).[4–6] When placed around the abdomen, the AAJT applies pressure at the level of the aortic bifurcation. The product has been shown to be effective in limiting femoral artery blood flow in both laboratory and clinical settings.[7–9] One of the main benefits of the AAJT is that it can be applied quickly and accurately by prehospital personnel with minimal training.[9,10] Complications of AAJT application include increased pain, respiratory arrest, obstruction of surgical access sites, and the potential for ischemic bowel damage with extended inflation times.[6,11–13] The AAJT is currently fielded by Special Operations medical personnel for use to prevent exsanguination from severe pelvic and femoral injuries. As a noninvasive, externally applied device, it is suitable for use on the battlefield and during evacuation.

REBOA uses an inflated intravascular balloon that stops blood flow to achieve hemostasis. The balloon catheter is introduced via the femoral artery and can be positioned in the descending aorta between the subclavian and the celiac arteries (Zone 1) or inserted between the renal artery and the aortic bifurcation (Zone 3), depending on the site of hemorrhage.[14] Current clinical practice guidelines from the American College of Surgeons recommend an aortic occlusion time of less than 15 minutes when positioned in Zone 1 and less than 60 minutes when positioned in Zone 3.[15] Initiation of REBOA requires significant training and technical proficiency. Because of this, and in contrast to the AAJT, REBOA has been used in military casualty care no farther forward than Role II facilities with surgical capability on site.[16,17] Several translational studies have demonstrated broad equivalence between REBOA and the AAJT for the control of femoral hemorrhage.[18–20] In simple controlled hemorrhage and polytrauma models, similar hemostatic, hemodynamic, and metabolic profiles were observed.

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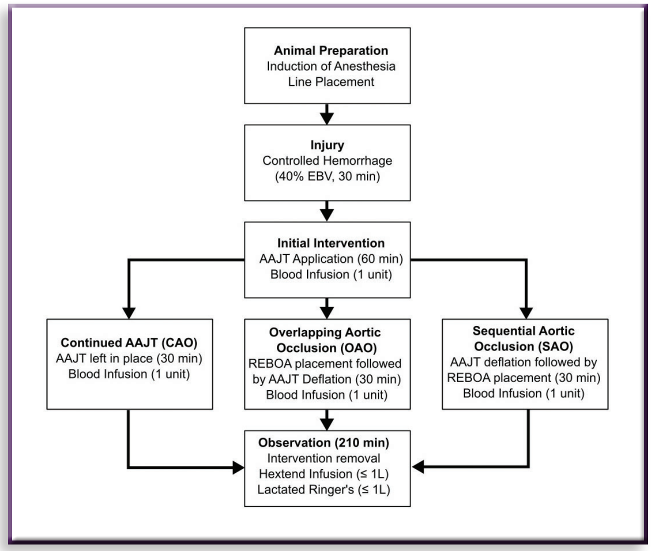
The ability to safely convert an AAJT to a Zone 3 REBOA would allow for rapid control of pelvic and lower extremity junctional hemorrhage in the field while minimizing the morbidity associated with prolonged AAJT application. Transitioning to internal vascular occlusion would also remove the AAJT as an impediment to surgical access to the abdomen, pelvis, and inguinal areas. As the AAJT is a standalone device that can be applied rapidly by prehospital personnel, it is uniquely suited for use by first responders, especially in austere environments. Upon escalation of care, transition to a Zone 3 REBOA would avoid the adverse outcomes seen with prolonged AAJT application and allow for continued hemorrhage control. A recent study examined the transition from AAJT to Zone 3 REBOA but did not include realistic conversion, examination of laboratory parameters, or an extended period of observation.[21] This study was designed to rigorously explore the consequences of conversion of the AAJT to Zone 3 REBOA in a clinically relevant translational model of severe hemorrhagic shock.

FIGURE 1 *Flow chart showing the progression of experimentation.*



EBV, estimated blood volume (66mL/kg).

Methods

This study was approved by the Institutional Animal Care and Use Committee for the Bridge PTS (Preclinical Testing Services) Research Facility (Brooks City-Base, TX). This facility's animal care and use program is accredited by the Association for Assessment and Accreditation of Laboratory Animal Care International. All animals were treated in accordance with the *Guide for the Care and Use of Laboratory Animals*.[22] All products used were commercially purchased.

Animal Preparation

Yorkshire cross castrated male swine were procured from a local USDA-registered vendor. The animals were housed at the facility vivarium for 3 to 5 days prior to their use in the study. Animals weighing 70 to 90 kg were fasted overnight with free access to water. Animals were premedicated with an initial intramuscular (IM) injection of 0.02 to 0.05mg/kg of atropine for 15 to 30 minutes, followed by tiletamine-zolazepam (4–8mg/kg IM). Anesthesia was induced with a facemask and 2-4% isoflurane. Once intubated, isoflurane was adjusted to 1–3% during procedures. The overall experimental design is shown in Figure 1.

Vascular access was accomplished via cutdown except as otherwise noted. The right carotid artery was accessed for blood pressure measurement and blood sampling. The left external jugular vein was accessed for infusion of resuscitation fluids. The right femoral artery was percutaneously accessed for blood withdrawal and monitoring distal pressure. Near-infrared spectroscopy (NIRS) pads were placed over the left pectoralis muscle, the left flank (overlying the kidney), and the medial thigh muscle of both legs. NIRS technology allows for increased noninvasive tissue penetration, thus permitting monitoring of regional tissue oxygenation.[23,24] Finally, the AAJT was pre-positioned under the animal to minimize disturbing the animal during the experiment.

Hemorrhage

Blood was withdrawn from the femoral artery in a multi-rate fashion to more physiologically simulate uncontrolled hemorrhage compared with a single constant rate.[25] Up to 40% (27mL/kg) of estimated blood volume was withdrawn over 30 minutes, divided into two phases: half of this volume was withdrawn over 10 minutes and the remainder over the last 20 minutes. However, hemorrhage was paused whenever the mean arterial pressure (MAP) dropped below 30mmHg and was resumed once it rose above 30mmHg, resulting in less hemorrhage than the goal of 40% estimated blood volume.

Intervention

Immediately after the 30-minute hemorrhage, the AAJT was applied to the animal's waist at the midline, approximately 2cm superior to the ilium, and inflated to 300mmHg according to the manufacturer's instructions. Correct AAJT placement was confirmed by the absence of an arterial pressure waveform from the right femoral artery. Five minutes after application, 500mL of shed blood was administered at 100mL/min. During the application period, the AAJT would be further inflated whenever pulse fluctuations reappeared.

Animals were randomly allocated to one of three experimental groups 55 minutes after AAJT inflation: the first group had the AAJT left in place and inflated (CAO), the second had the AAJT completely deflated prior to REBOA insertion (SAO), and the third group had the REBOA inserted while the AAJT was still inflated, after which the AAJT was deflated (OAO). In both REBOA groups, the left femoral artery was percutaneously catheterized with a 5 French micropuncture set using ultrasound guidance, and a 7 French sheath was inserted. The ER-REBOA (Prytime Medical Devices) was then inserted 25cm into the artery (based on fluoroscopic catheter depth measurements taken during model development) and inflated with 5mL of normal saline, either prior to or following AAJT deflation, depending on group allocation. Once the balloon was fully inflated and no femoral arterial waveform was observed, the time was set as T0, and a 30-minute period of Zone 3 REBOA began. For the CAO group, the 30-minute period began immediately following the initial 60-minute AAJT application, for a total of 90 minutes of AAJT occlusion. In all groups, 5 minutes before the end of the intervention period, a second 500mL of shed blood was infused at 100mL/min.

Intervention Removal and Observation

The REBOA catheter and AAJT were deflated slowly over 3 minutes in all cases. Following deflation of the intervention, up to 1L of Hextend (Cerner Multum) and 1 liter of lactated

Ringers solution were administered as needed to maintain a MAP greater than 60mmHg. Animals were observed for an additional 3.5 hours without further interventions. Arterial blood samples were taken at baseline, following hemorrhage, after the initial AAJT period (T0), then 30, 60, 120, 180, and 240 minutes after randomized intervention. Animals were euthanized using intravenous pentobarbital 100 mg/kg (i.e., euthanasia solution) and in accordance with the 2013 American Veterinary Medical Association *Guidelines for the Euthanasia of Animals*. Immediate laparotomy with inspection of the small and large bowels for signs of compression damage was performed.

Outcomes and Analysis

Primary outcomes of this study were the ability to correctly place the REBOA and time of REBOA deployment. Other outcomes included survival, hemodynamics (e.g., blood pressure measurement, end-tidal CO_2 [$EtCO_2$] level, heart rate [HR]), and markers of tissue damage (e.g., lactate, blood urea nitrogen [BUN], creatinine, pH, potassium, myoglobin levels).

Data are presented as mean ± standard deviation for continuous variables. One-way analysis of variance (ANOVA) was used for baseline comparisons, and two-way repeated measures ANOVA was used for continuous variables over a time course. Survival was analyzed using log-rank analysis. Fisher's exact test was used for categoric variables. Statistical analysis and data management were performed using Excel 2010 (Microsoft) and SigmaPlot 12 (Systat Software).

Results

Baseline Characteristics

A total of 17 animals weighing 82.2 ± 7.4kg were included for analysis: CAO (n = 5), OAO (n = 6), SAO (n = 6). One animal was removed from analysis in the CAO group because of iatrogenic injuries that occurred during surgical preparation. The groups were similar at baseline (Table 1).

Hemorrhage and Intervention

At baseline, animals had a mean MAP of 63.2 ± 5.9mmHg, an HR of 94 ± 11 beats per minute (bpm), and an $EtCO_2$ of 40.6 ± 3.2mmHg, with no statistically significant difference among groups (Table 1). All animals except one required temporary suspension of hemorrhage, resulting in an average loss of 35.9 ± 4.2% of the estimated blood volume. Hemorrhage resulted with a MAP of 41.8 ± 11.0mmHg, a HR of 155 ± 31bpm, and an $EtCO_2$ of 36.6 ± 4.5mmHg. Inflation of the AAJT resulted in an absence of waveform in the femoral arteries, with a MAP of 26.2 ± 15.7mmHg and a pulse pressure of 2.5 ± 5.8mmHg after 10 minutes. Most animals required some additional inflation to maintain the required 300mmHg inflation pressure in the air bladder. At the end of the initial 60-minute period of AAJT inflation, MAP was 67.7 ± 17.4mmHg, HR was 174 ± 34bpm, and $EtCO_2$ was 42.2 ± 4.3mmHg.

In the REBOA groups, catheterization was attempted using ultrasound guidance. All femoral arteries in the SAO group were successfully cannulated and the REBOA correctly placed. One artery in the OAO group was improperly cannulated, with the REBOA inserted into the femoral vein. This was noted as a failure of REBOA insertion, and the animal was excluded from further analysis. Time to REBOA inflation (including arterial access, introducer sheath placement, and advancement of the REBOA catheter) was 4.2 ± 2.2 minutes for all REBOA animals and did not differ between the two REBOA groups (Table 2). The two-step conversion of AAJT to REBOA in the SAO group resulted in various acute effects, including a drop in carotid blood pressure, decrease in pectoralis NIRS, increase in both left and right thigh NIRS, and increased $EtCO_2$during the conversion. Additionally, an increase in carotid MAP and $EtCO_2$ was observed after balloon inflation (Table 2, Figure 2).

Posttreatment

Hemodynamic values during the observation period were not different between the groups (Figure 2). All groups experienced a modest increase in MAP of about 15mmHg in response to the administration of 500mL of shed blood prior to intervention deflation. After AAJT or REBOA deflation, all groups had a return of femoral arterial waveform within 5 minutes. All groups also had an acute increase in $EtCO_2$ 5 minutes after removal, with values remaining above baseline for the remainder of the observation period. Biochemical markers of shock and tissue injury were assessed throughout the protocol (Figure 3). No differences were observed among groups in any of the laboratory values assessed at any time point. A sharp drop in blood pH and a corresponding increase in lactate levels were observed in all groups following intervention removal. Potassium, BUN, creatinine, interleukin-6, and myoglobin levels all were elevated throughout the observation period in all groups, with no differences among groups. However, creatinine and myoglobin levels were persistently higher in the CAO group following intervention removal, but these differences did not reach statistical significance. At laparotomy, no gross evidence of intestinal injury was observed in any animal, and no abdominal tissue damage was noted on inspection. However, histologic testing was not performed.

Discussion

We have demonstrated that conversion from the AAJT to Zone 3 REBOA is technically feasible in a swine model of severe controlled hemorrhage and that transition between the two devices can be accomplished with or without prior deflation of the AAJT device. Initiation of REBOA prior to AAJT deflation mitigated BP, HR, and $EtCO_2$ variability compared with advancement and inflation after AAJT deflation but was associated with increased difficulty in obtaining and confirming transfemoral access to the infrarenal aorta.

After induction of class IV shock and placement of the AAJT device, proximal aortic MAP returned to pre-hemorrhage values. while the decrease in femoral artery MAP was consistent with prior studies.[18,19] In each of the three groups (CAO, OAO, SAO), no sustained significant hemodynamic differences were observed with respect to carotid MAP, femoral MAP, $EtCO_2$, and HR (Figure 2). However, SAO was found to yield statistically significantly different hemodynamic effects within the time periods during and shortly after conversion of AAJT to REBOA. These differences included a drop in carotid MAP, decreased pectoralis NIRS, increased left and right leg NIRS, and increased $EtCO_2$ (Table 2). These changes are consistent with hemodynamic effects seen directly following cessation of aortic occlusion. The variations can be attributed to the temporary lapse in aortic occlusion during SAO placement, which allows for transient reperfusion of the bilateral lower extremities, likely resulting in a washout of built-up lactate and carbon dioxide. These effects are seen in

TABLE 1 *Baseline, Post Hemorrhage, and Post AAJT Values*

Value	CAO	OAO	SAO	*p* value
n	5	6	6	—
Weight (kg)	81.0 ± 10.4	84.6 ± 5.6	81.0 ± 6.5	0.523
Baseline				
MAP (mmHg)	61.0 ± 8.2	62.0 ± 5.0	66.3 ± 3.4	0.278
HR (bpm)	97± 9	92 ± 11	95 ± 13	0.728
$EtCO_2$ (mmHg)	41.6 ± 3.2	42.0 ± 3.5	38.5 ± 2.2	0.125
pH	7.508 ± 0.058	7.492 ± 0.053	7.539 ± 0.042	0.374
K (mmol/L)	4.03 ± 0.51	3.85 ± 0.26	3.76 ± 0.23	0.586
Lactate (mmol/L)	1.79 ± 0.21	1.72 ± 0.70	2.07 ± 0.62	0.871
Post Hemorrhage				
MAP (mmHg)	43.0 ± 10.5	41.8 ± 12.2	40.8 ± 12.1	0.955
HR (bpm)	167 ± 36	139 ± 31	161 ± 26	0.303
$EtCO_2$ (mmHg)	37.8 ± 2.7	37.2 ± 5.1	35.0 ± 5.3	0.577
pH	7.499 ± 0.022	7.506 ± 0.033	7.536 ± 0.049	0.178
K (mmol/L)	4.30 ± 0.57	4.00 ± 0.50	4.14 ± 0.25	0.746
Lactate (mmol/L)	2.53 ± 0.58	2.05 ± 0.82	2.46 ± 0.49	0.960
Hemorrhage (%EBV)	34.7 ± 7.1	35.9 ± 4.2	35.3 ± 4.0	0.532
Post 60-min AAJT				
MAP (mmHg)	65.8 ± 21.4	67.1 ± 17.9	69.8 ± 16.4	0.933
HR (bpm)	180 ± 40	161 ± 42	181 ± 17	0.566
$EtCO_2$ (mmHg)	42.8 ± 2.4	42.5 ± 6.2	41.3 ± 4.0	0.852
pH	7.478 ± 0.042	7.450 ± 0.079	7.460 ± 0.059	0.868
K (mmol/L)	4.30 ± 0.48	4.30 ± 0.50	4.08 ± 0.41	0.966
Lactate (mmol/L)	3.29 ± 1.3	4.03 ± 1.1	3.88 ± 1.5	0.688

CAO, continuous AAJT occlusion; $EtCO_2$, end tidal CO_2; HR, heart rate; MAP, mean arterial pressure; OAO, overlapping aortic occlusion; SAO, sequential aortic occlusion.
Data are mean ± SD.

the rise of $EtCO_2$ (Table 2) and indirectly by the trend toward lower lactate levels in the SAO group. Additionally, the lapse of occlusion could allow for the potential for continued hemorrhage during that time.

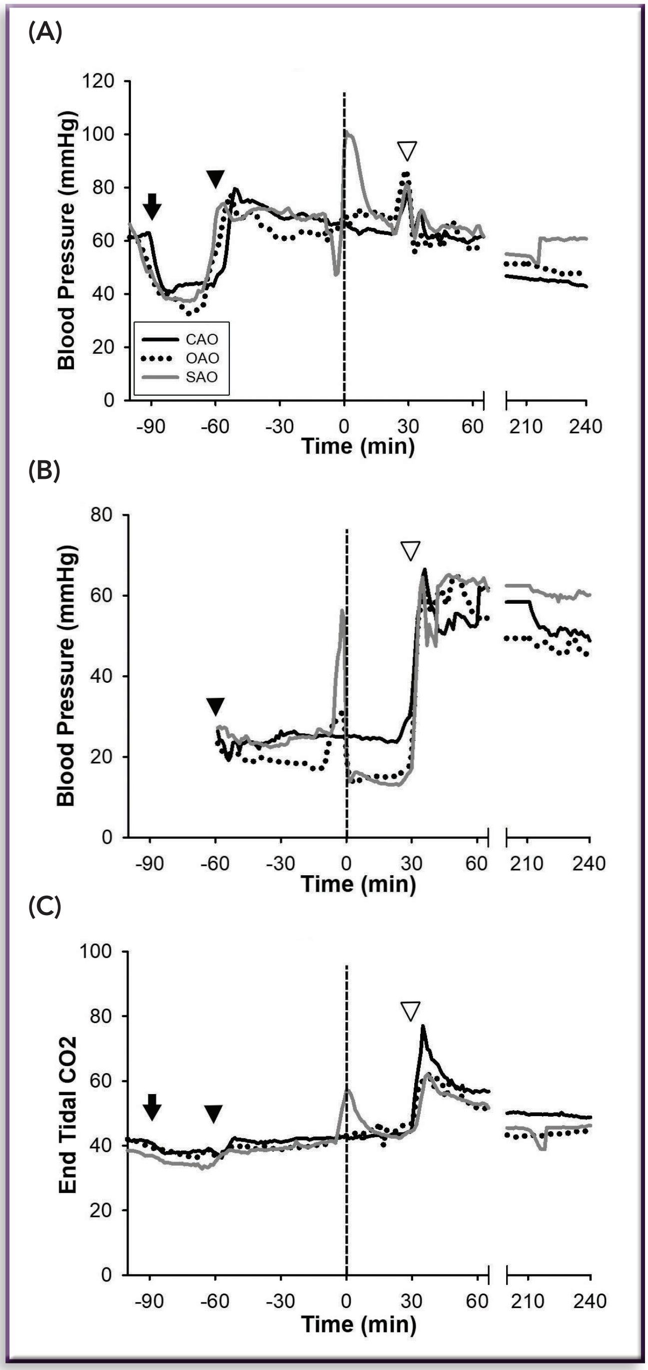
Although both the AAJT and REBOA occlude antegrade major pelvic arterial flow, their methods of occlusion are drastically different. The AAJT produces extrinsic compression across the lower abdomen, resulting in occlusion of both arterial and venous flow, and affects collateral as well as major vessels. In comparison, Zone 3 REBOA specifically occludes the infrarenal aorta. This allows for continued collateral arterial circulation and preserves venous outflow. Despite these major methodologic differences and the transient hemodynamic variation seen with SAO, we observed no significant physiologic differences between groups. The hyperkalemia and lactic acidosis observed in this study are consistent with the metabolic derangements noted in prior studies and are associated with

TABLE 2 *Select Values During and Shortly After Conversion to REBOA*

Value	CAO	OAO	SAO	*p* value
Success n (%)	—	5/6 (83%)	6/6 (100%)	0.999
Time (min)	—	4.3 ± 2.9	4.1 ± 1.8	0.909
Values obtained proximal to occlusion				
Carotid MAP (mmHg) - Nadir	63.4 ± 21.2	60.8 ± 19.1	43.6 ± 6.6	0.172
$EtCO_2$ (mmHg) - Peak	42.8 ± 2.4	44.2 ± 8.3	57.5 ± 6.5	0.003**
Pectoralis StO_2 (%)	67.8 ± 4.6***	71.0 ± 12.9***	53.8 ± 5.3	0.010*
Left Flank StO_2 (%)	53.2 ± 10.5	56.2 ± 13.8	43.3 ± 8.3	0.153
Values obtained distal to occlusion				
Femoral Artery Pressure(mmHg) - Peak	26.2 ± 7.2****	34.5 ± 16.6****	60.7 ± 12.4	0.0014**
Left Thigh StO_2 (%)	16.5 ± 3.0***	22.5 ± 10.9	39.7 ± 18.5	0.0380*
Right Thigh StO_2 (%)	15.6 ± 1.3****	21.8 ± 8.2****	48.3 ± 15.1	0.0002**
Values obtained shortly after conversion				
$EtCO_2$ (mmHg) - Peak	46.2 ± 2.0****	51.0 ± 6.7****	61.5 ± 5.4	0.0008**
Carotid MAP (mmHg) - Peak	66.8 ± 20.7***	77.5 ± 22.0	105.6 ± 21.4	0.0343*

CAO, continuous AAJT occlusion; $EtCO_2$, end title CO_2; MAP, mean arterial pressure; OAO, overlapping aortic occlusion; SAO, sequential aortic occlusion; StO_2, skeletal muscle tissue oxygenation.
*$p < .05$; **$p < .01$; ***$p < .05$ vs SAO; ****$p < .01$ vs SAO.
Data are mean ± SD.

FIGURE 2 *Blood pressure and end-tidal CO_2.*



(A) Carotid artery blood pressure; **(B)** femoral artery blood pressure; **(C)** end-tidal CO_2. Arrow indicates start of hemorrhage. Solid arrowhead indicates application of AAJT in all groups. Dashed line is start of REBOA or continued AAJT. Open arrowhead is removal of occlusion. Error bars not shown for clarity.

CAO, continuous AAJT occlusion; OAO, overlapping aortic occlusion; SAO, sequential aortic occlusion.

the severity of hemorrhage and the length of ischemic time produced by the intervention.[7,9]

Transfemoral placement of a REBOA device in the presence of an inflated AAJT presents a unique challenge in accessing the artery and inflating the balloon. Although no difference was noted in the time to REBOA deployment with the AAJT inflated or deflated, one REBOA in the OAO group was inaccurately placed into the femoral vein. This error can be attributed to the lack of arterial pulsatility on examination and lack of Doppler flow visualized on ultrasound secondary to AAJT-mediated arterial occlusion. Additionally, confirmation of intra-arterial needle placement is difficult because of the lack of pulsatile pressure and the appearance of deoxygenated distal arterial blood. Inflation of a REBOA must be done "blindly" while the AAJT is in place because the typical signs of complete aortic occlusion are not present (i.e., rise in proximal MAP and/or loss of contralateral pulse). The volume of REBOA inflation must therefore be based on the recommended volume for Zone 3 deployment and adjusted if needed once the AAJT is removed. Finally, introduction and inflation of the REBOA catheter into the infrarenal aorta was met with little or no resistance from the inflated AAJT, likely because of the AAJT's occlusion occurring more cranial than the placement of the REBOA catheter.

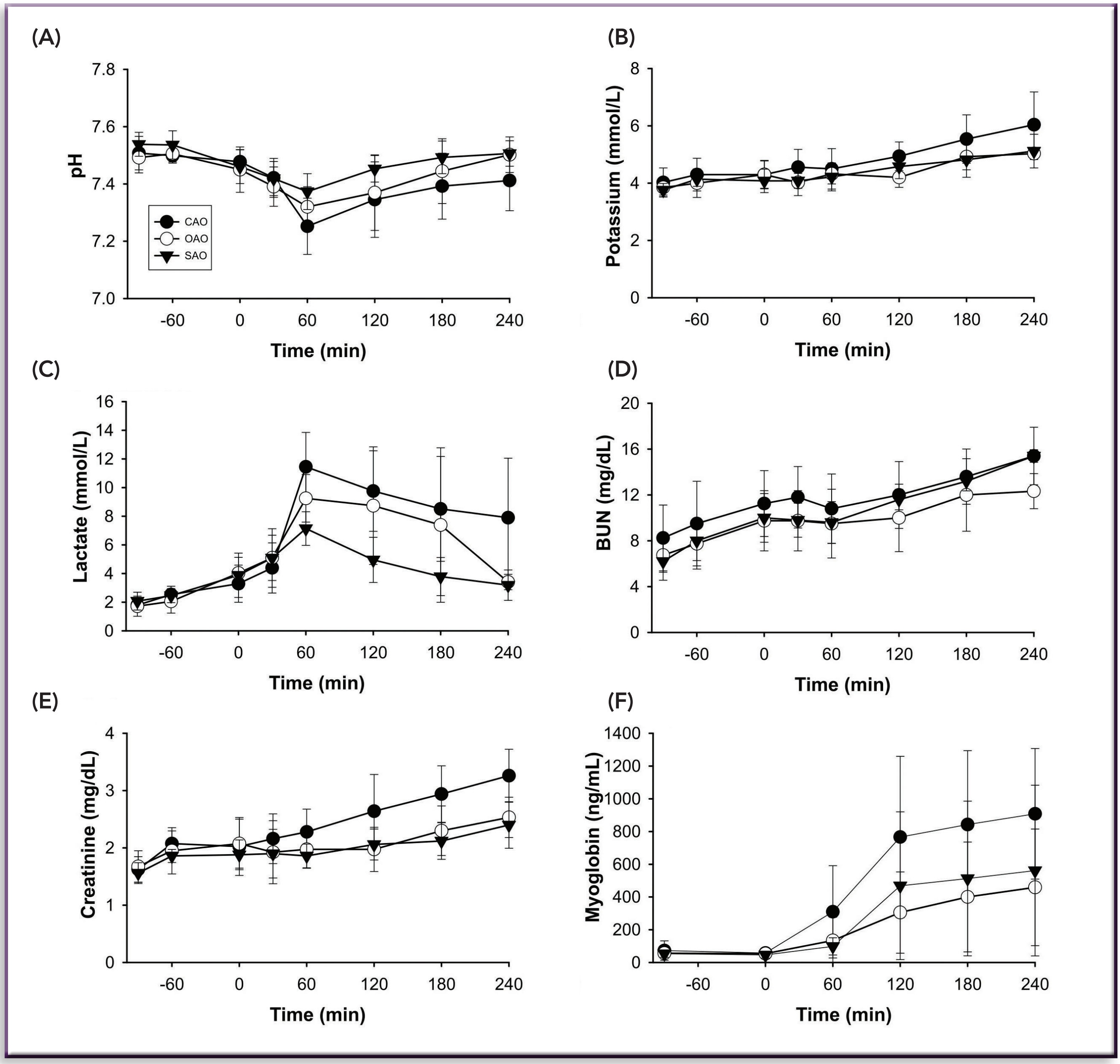
The increased hemodynamic variability within the SAO group and the overall feasibility of AAJT to REBOA transition we observed is concordant with the findings of a similar study by Brännström et al.[21] We expanded upon their findings by assessing 60 minutes of AAJT prior to intervention (compared with 30 minutes) and by investigating the practicality of AAJT conversion to REBOA without deflation of the AAJT bladder and temporary loss of aortic occlusion.

Bowel ischemia is a known complication after prolonged AAJT application, specifically after 240 minutes, as noted by Brännström et al.[13] Current guidelines call for AAJT placement for no more than 60 minutes. Authors of another study who used the AAJT to control pelvic bleeding observed small bowel injury in half the animals subjected to AAJT treatment.[6] In the current study, no evidence of gross intestinal ischemia was noted at the time of necropsy for all groups, including the CAO group, which underwent 90 minutes of AAJT placement. The source of the discrepancy between the two studies is not known but may be the result of the larger swine used in our experiments (70–90kg) compared with the swine in the previous study, which had a mean weight of 44kg. This disparity in animal size likely results in differing pressure distribution generated by the AAJT on the abdomen and underlying organs.

Transition from the AAJT to Zone 3 REBOA is feasible. Although no metabolic advantages are evident, early transition to REBOA would allow for avoidance of prolonged AAJT morbidities, such as bowel ischemia,[6] difficulty ventilating,[11] and poor access to abdominopelvic surgical sites.[21] Furthermore, Zone 3 REBOA can be placed immediately, without the need to deflate the AAJT device. This technique may increase the difficulty of femoral artery access, but it avoids the hemodynamic fluctuation seen with REBOA placement after deflation of an AAJT device.

This study has several limitations worth noting. First, although the animals were observed for 4 hours, true long-term consequences of the interventions may have arisen had the observation period been prolonged. Second, a controlled hemorrhage model was used in this study as opposed to an uncontrolled model, and we therefore not cannot comment on blood loss differences between the groups. Finally, there are notable anatomic differences between swine and humans (primarily vis-à-vis AAJT application) that may affect the applicability of

FIGURE 3 *Laboratory values.*



(A) pH; **(B)** potassium; **(C)** lactate; **(D)** urea nitrogen; **(E)** creatinine; **(F)** myoglobin. T0 is start of randomized aorta occlusion.
CAO, continuous AAJT occlusion; OAO, overlapping aortic occlusion; SAO, sequential aortic occlusion.

these findings during human care. Swine have a more developed collateral system compared with humans, including the well-developed subclavian-to-iliac mammary collateral system that likely supplies the abdominal wall and perfusion below the REBOA.[26,27] These collaterals are likely occluded with the AAJT, thus making a direct comparison with responses in a human patient not ideal.

Conclusion

Conversion of the AAJT to infrarenal REBOA is effective, with each technique having physiologic advantages and disadvantages. Placement of a REBOA catheter in the presence of an inflated AAJT can make accessing the femoral artery difficult, potentially resulting in improper cannulation. Further studies are needed to define clear guidelines for managing noncompressible torso hemorrhage in the austere environment.

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Authorship and Contributorship Statement

JMR, DSK, and PEB conceived of the study concept. JMR obtained funding. KSS, PEB, and JMR coordinated and collected data. JMR, PEB, and DSK analyzed data. KSS, DSK, and PEB wrote the first draft. All authors read and approved the final manuscript.

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Disclaimer

The views expressed are those of the authors and do not reflect the official views of the Department of Defense or its Components. The experiments reported herein were conducted according to the principles set forth in the National Research Council *Guide for the Care and Use of Laboratory Animals*, and the Animal Welfare Act of 1996, as amended.

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