Injury, Int. J. Care Injured 50 (2019) 1042–1048

Contents lists available at ScienceDirect

Injury

journal homepage: www.elsevier.com/locate/injury

Examination of hemodynamics in patients in hemorrhagic shock undergoing Resuscitative Endovascular Balloon Occlusion of the Aorta (REBOA)

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ARTICLE INFO

Article history:
Accepted 21 December 2018

Keywords:
Resuscitative Endovascular Balloon Occlusion of the Aorta
REBOA
Aortic occlusion
Hemodynamics
Blood pressure

ABSTRACT

Background: The objective of this study was to investigate the hemodynamic effects of aortic occlusion (AO) during Resuscitative Endovascular Balloon Occlusion of the Aorta (REBOA) using a sophisticated continuous vital sign (CVS) monitoring tool.
Methods: Patients admitted between February 2013 and May 2017 at a tertiary center that received REBOA were included. Patients in cardiac arrest before or at the time of REBOA were excluded. Time of AO was documented by time-stamped videography and correlated with CVS data.
Results: 28 patients were included, mean (standard deviation) ISS was 38 (11). 18 received Zone 1 (distal thoracic aorta) and 10 received Zone 3 (distal abdominal aorta) AO. Among Zone 1 patients the pre-AO systolic blood pressure (SBP) nadir was 64 (19) mmHg, which increased to a mean of 124 (29) mmHg within 5 min after AO ($p < 0.01$). Among Zone 3 patients the pre-AO SBP nadir was 75 (19) mmHg, which increased to a mean of 98 (14) mmHg within 5 min after AO ($p < 0.01$). 72% of Zone 1 patients had episodes during AO where SBP was less than 90 mmHg as compared to 80% of Zone 3 patients ($p = 0.51$). 100% of Zone 1 patients had periods during AO where SBP was greater than 140 mmHg as compared to 70% Zone 3 patients ($p = 0.04$). The overall mean decrease in SBP after balloon deflation was 13 (20) mmHg ($p < 0.01$), with similar decreases among groups (14 (21) mmHg vs 12 (18) mmHg for Zone 1 and 3 patients, respectively ($p = 0.85$)). Patients undergoing Zone 1 AO were more likely to have an acute change (increase or decrease) in their heart rate immediately after AO as compared to Zone 3 AO ($p = 0.048$).
Conclusions: Significant hemodynamic alterations occur before, during, and after AO. The effects of Zone 1 AO on blood pressure and heart rate appear different than Zone 3 AO. This may have important implications for cardiac or cerebral function and perfusion goals, particularly with concomitant injuries such as cardiac contusion or traumatic brain injury.

Introduction

Aortic occlusion (AO) results in increased pressure and blood flow proximal to the level of occlusion, and decreased blood flow distally [1–5]. In the context of trauma care, there has been increasing interest in using Resuscitative Endovascular Balloon Occlusion of the Aorta (REBOA) as a hemorrhage control adjunct for abdominal, pelvic, and junctional hemorrhage. Animal models have shown benefit with isolated injuries [6–8], and initial reports have described utility in the attenuation of hemorrhage [9–11].

AO in the context of normovolemia (such as elective vascular surgery) can result in supranormal hemodynamic indices [1,12–14]. Awareness of this has resulted in enthusiasm for techniques to mitigate changes in blood pressure and perfusion such as partial REBOA [15,16]. However, the hemodynamic changes caused by AO in patients suffering trauma may already be offset due to the reduced preload [14] seen in hemorrhagic shock. Prior clinical reports [9–11,17,18], have simplistically, and without a standardized method, reported singular blood pressure and heart rate values before and during AO. In patients with traumatic hemorrhagic shock, it is unclear how robust the hemodynamic effect from AO is, and if it is sustained.

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https://doi.org/10.1016/j.injury.2018.12.030
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In addition, increased arterial pressures and perfusion may result in exacerbation of concomitant injuries proximal to the level of AO, such as traumatic brain injuries [19,20] or thoracic vascular injuries. The presence and extent of this potentially harmful effect has not been well characterized or examined.

The objective of this study, using a continuous vital sign (CVS) monitoring tool, was to investigate the hemodynamic changes before, during, and after REBOA in a group of patients suffering from traumatic hemorrhagic shock.

Methods

This prospective, observational, single-center study was approved by the Institutional Review Board of the University of Maryland, Baltimore. Demographics and hospital course data was collected on all trauma patients, age ≥18 years old, who underwent REBOA between February 2013 and May 2017 at the University of Maryland Shock Trauma Center. Patients who were in cardiac arrest and/or received chest compressions before AO were excluded. REBOA-specific procedural timing metrics were recorded by available time-stamped videography in the resuscitation areas and operating rooms.

Vital signs were recorded from a continuous vital sign monitoring system (see Fig. 1 for case example) using a network of GE-Marquette-Solar-7000/8000® (General Electric, Fairfield, CT) patient vital signs monitors as previously described [21]. In brief, all patient vital sign monitors in the trauma resuscitation unit, operating room, post-anesthesia care unit, and intensive care units are networked to provide continuous collection (0.5 Hz) of real time patient vital sign data. Vital sign data collected included invasive and non-invasive blood pressure, heart rate, and electrocardiographic waveforms, among others. Data is transmitted through a secure intranet connection to dedicated BedMaster® servers (Excel Medical Electronics, Jupiter, FL) and archived. Lastly, in order to reduce the impact of individual server collection failure, a triple-redundant system with three BedMaster® servers was implemented.

For each case, it was determined during all time points where the source of blood pressure data should be extracted for analysis (arterial line versus non-invasive blood pressure cuff). The source of arterial line data (femoral, radial, REBOA catheter) was recorded. Of note, during the study period, there was a transition in balloon catheter type from the CODA® catheter (Cook Medical, Bloomington, IN) to the ER-REBOA™ catheter (Prytime Medical, Boerne, TX), which occurred in February 2016. The ER-REBOA™ catheter has an arterial line monitoring port above the balloon whereas the CODA® catheter does not, permitting systemic blood pressure monitoring before, during, and after AO. Non-physiologic vital sign data, such as artifact and times during which the vital sign monitoring devices were recording but not connected to the patient, was excluded. In order to better describe the severity of hemodynamic decompensation before AO, a blood pressure (BP) nadir and the time to AO after the nadir was recorded.

The study population was divided into two groups; patients who underwent REBOA in zone 1 (descending thoracic aorta) or zone 3 (infra-renal aorta). The primary outcomes were the change in heart rate and blood pressure immediately before and after AO. Secondary hemodynamic outcomes included characterizing blood pressure and heart rate values during the 20 min prior to AO, during the first 120 min of AO, and during the 5 min before and after AO. The prevalence and patterns of hemodynamic monitoring (invasive vs. non-invasive) were also compared, especially with regard to catheter type. Additional secondary outcomes included characterizing concomitant proximal injuries (brain and chest) and assessing whether the use of REBOA may have caused or exacerbated these injuries.

Paired one-sample *t*-test was used for mean comparison of blood pressure changes and fisher's exact test for proportion comparison of changes in heart rate and blood pressure. Variables with normal distributions are reported as mean (standard deviation), and those without normal distributions are reported as median and interquartile range. Comparisons of demographics and injury characteristics were made using unpaired two-sided Student's *t*-test, Fisher's exact test, and Mann Whitney U test. Statistical significance was defined as a p-value of 0.05 or less. Statistical analysis was performed using Matlab (R2014b, Natick, MA).

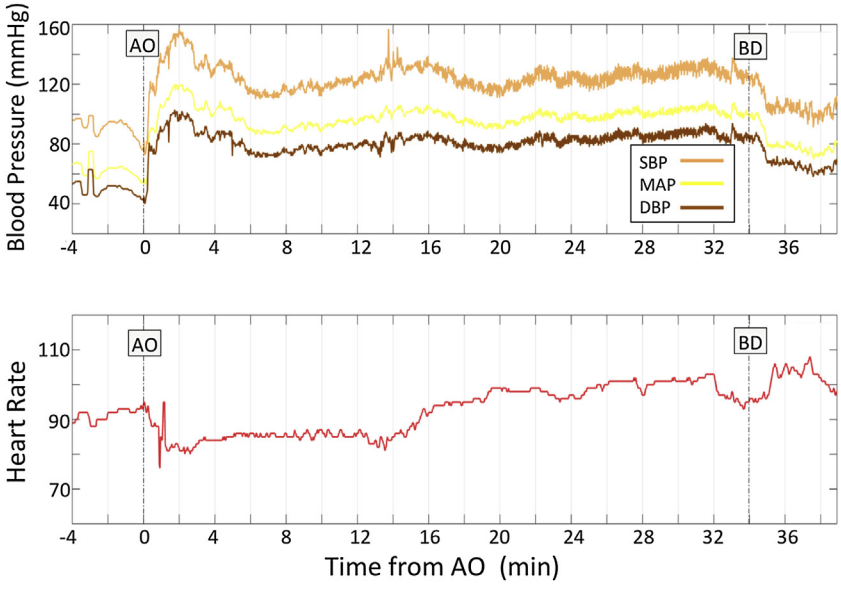


Fig. 1. Vital sign data recorded continuously every 2 s from a patient in hemorrhagic shock undergoing REBOA. A dramatic increase in blood pressure (BP) and decrease in heart rate is identified after AO. The timing of AO is confirmed with videography in the resuscitation area and operating room. Upon balloon deflation, the patient experienced a small decrease in BP and increase in heart rate. SBP, Systolic Blood Pressure; MAP, Mean Arterial Pressure; DBP, Diastolic Blood Pressure; BD, Balloon Deflation.

Results

Patient demographics and characteristics

A total of 86 patients were assessed for eligibility, with 58 patients being excluded secondary to suffering cardiac arrest prior to performance of REBOA. 28 patients were included, and all of those included underwent REBOA according to our institutional protocol [22]. The choice to perform REBOA in each case was at the discretion of the attending trauma surgeon, but all patients had a period where the systolic BP was < 90 mmHg in their clinical course prior to REBOA use. The decision to perform REBOA occurred in each case near the time of worst hemodynamic decompensation (5.2 (3.1) minutes before REBOA use). Patient demographics, characteristics, and injury patterns are listed in Tables 1 and 2.

Further examination revealed several differences between both groups. Zone 1 patients were younger ($p < 0.01$), suffered less pelvic fractures ($p < 0.01$), and had a higher abdominal AIS (abbreviated injury score, $p = 0.01$) with a higher incidence of abdominal organ injuries ($p = 0.02$) including multiple abdominal organ injuries ($p = 0.04$). In addition, zone 1 patients had a trend towards a higher prevalence of major abdominal/pelvic vascular injuries (defined as injuries to the aorta, aortic branch vessels, inferior vena cava, portal vein, and common and external iliac arteries and veins) as well as concomitant major abdominal/pelvic vascular and organ injuries (both $p = 0.14$). Zone 3 patients trended towards a higher lower extremity AIS ($p = 0.06$), suffered more blunt injury ($p = 0.054$), and lower admission heart rate ($p = 0.08$).

Examination of concomitant proximal injuries

Concomitant proximal (brain and chest) injuries were examined in greater detail to ascertain if REBOA had potentially caused or exacerbated these injuries. 8 patients (29%) had concomitant brain injuries which were determined to be caused by their mechanism of injury. Of these brain injuries, 4 were ultimately deemed to be non-survivable injuries leading to withdraw of care. It is unknown whether, or to what degree, REBOA exacerbated these injuries. 19 (68%) had concomitant chest injuries, including rib fractures (14), sternal fractures (1), pneumothoraces (8), and lung contusions (2). Of these, two cases had significant thoracic hemorrhage which may have been exacerbated by the use of REBOA. In one case, there was a sternal fracture associated internal mammary artery injury which resulted in a mediastinal hematoma requiring evacuation. In the second case hemorrhage from an intercostal artery occurred requiring operative intervention. The presence and severity of both of these injuries were not evident at the time the decision to perform REBOA was made.

Blood pressure monitoring characteristics

In the emergent clinical settings of a patient requiring AO, the quality and quantity of BP data varied and was heterogeneous. Four patterns of BP measurement were observed before AO by REBOA. The first pattern had systemic arterial line data throughout REBOA (Fig. 1). The second pattern was characterized by a femoral arterial line that was obtained and transduced before AO; however, the femoral arterial line was upsized for the balloon catheter insertion therefore arterial line data immediately around the time of AO is absent. In other cases, there was no arterial line placement before REBOA balloon catheter insertion, with some cases having a non-invasive BP cuff giving data (third pattern) and in others, no quantitatively measured BP but a “palpable pulse” documented (fourth pattern).

The timing and presence of arterial line transduction in relation to AO was examined. Before AO, 94% of patients (16 out of 17) who had REBOA performed using the CODA® catheter had arterial line monitoring, with 35% (6) having only a femoral arterial line and 59% (10) having a radial arterial line in place. Patients with only femoral arterial lines had the arterial lines upsized for REBOA, with only 59% (10) of patients with the CODA® catheter having arterial line monitoring in place at time of AO. In comparison, 100% of patients (11 out of 11) who had REBOA performed using the ER-REBOA™ catheter had arterial line monitoring in place before AO; 73% (8) of those with femoral arterial lines were later upsized, 9% (1) had the ER-REBOA™ placed and used for arterial line monitoring without any other arterial lines, and 18% (2) had radial arterial lines. 82% (9) had the ER-REBOA™ catheter arterial line monitoring port utilized and transduced. The two patients who did not have the catheter arterial line monitoring port utilized already had radial arterial lines in place before REBOA.

As seen in Fig. 2, the cumulative incidence of arterial line monitoring before AO was similar between both catheters.

Table 1
Demographics and Characteristics.

Variable	Zone 1 (n = 18)	Zone 3 (n = 10)	P-value
Age (years), mean (SD)	33 (14)	55 (15)	<0.01*
Gender, n (% male)	16 (89%)	10 (100%)	0.81†
Admission Systolic Blood Pressure (mmHg), mean (SD)	115 (29)	110 (42)	0.70*
Admission Heart Rate, mean (SD)	121 (26)	102 (31)	0.08*
CODA® Catheter, n (%)	11 (61)	6 (60)	1.00†
ER-REBOA™ Catheter, n (%)	7 (39)	4 (40)	1.00†
AO Duration (minutes), median [IQR]	76 [25, 131]	76 [48, 117]	0.97‡
In-Hospital Mortality, n (%)	8 (44%)	2 (20%)	0.25†
Peri-REBOA Resuscitation[a]			
PRBC (units), median [IQR]	13 [7, 33]	9 [6,10]	0.18‡
FFP (units), median [IQR]	7.5 [4.25, 23]	7.5 [5.25, 13]	0.82‡
PLT (units), median [IQR]	3.5 [1, 5.75]	2 [1, 3.75]	0.90‡
Cryoprecipitate (units), median [IQR]	0 [0,1]	0 [0, 0.75]	0.51‡
Crystalloid (L), median [IQR]	3.25 [2,4]	3.5 [2.5, 4.9]	0.43‡
Vasopressor Usage Peri-AO, n (%)	14 (78%)	6 (60%)	0.57†
Initial Hemoglobin (g/dL), mean (SD)	11.0 (2.3)	10.9 (2.2)	0.87*
Lowest Hemoglobin in First 24 Hours (g/dL), mean (SD)	8.5 (2.0)	8.6 (2.5)	0.94*
Initial pH, mean (SD)	7.11 (0.16)	7.31 (0.25)	0.03*
Initial Base Excess, mean (SD)	−12.5 (6.6)	−11.0 (4.1)	0.60*
Initial Lactate (mmol/L), mean (SD)	7.3 (3.1)	7.3 (4.3)	0.96*

Student's T-test*, Fisher's exact test†, Mann-Whitney U test‡. AO, aortic occlusion; PRBC, packed red blood cell; FFP, fresh frozen plasma; PLT, platelets; IQR, interquartile range; SD, standard deviation. Peri-REBOA Resuscitation[a] defined as resuscitation given around the time of REBOA, both in the resuscitation area and through the index operation.

Table 2
Injury Characteristics.

Variable	Zone 1 (n = 18)	Zone 3 (n = 10)	P-value
Mechanism, blunt n (%)	11 (61%)	10 (100%)	0.054†
ISS, mean (SD)	39 (11)	36 (12)	0.44*
Brain AIS, median [IQR]	0 [0, 0]	0 [0, 3.5]	0.69‡
Chest AIS, median [IQR]	3 [0,4]	2.5 [0.25, 3.75]	0.99‡
Abdominal AIS, mean (SD)	4.1 (0.9)	3.3 (0.5)	0.01*
Lower Extremity AIS, median [IQR]	2.5 [1.25, 3]	3 [3, 3.75]	0.06‡
Pelvic Fracture, n (%)	5 (28%)	10 (100%)	<0.01†
Abdominal Organ Injury, n (%)	16 (89%)	4 (40%)	0.02†
Abdominal Solid Organ Injury, n (%)	14 (78%)	3 (30%)	0.04†
Abdominal Hollow Organ Injury, n (%)	8 (44%)	1 (10%)	0.14†
Multiple Abdominal Organ Injuries, n (%)	10 (56%)	1 (10%)	0.04†
Major Abdominal/Pelvic Vascular Injury, n (%)	8 (44%)	1 (10%)	0.14†
Concomitant Major Abdominal/Pelvic Vascular and Organ Injury, n (%)	8 (44%)	1 (10%)	0.14†

Student's T-test*, Fisher's exact test†, Mann-Whitney U test‡. AIS, Abbreviated Injury Score; ISS, Injury Severity Score; IQR, interquartile range; SD, standard deviation.

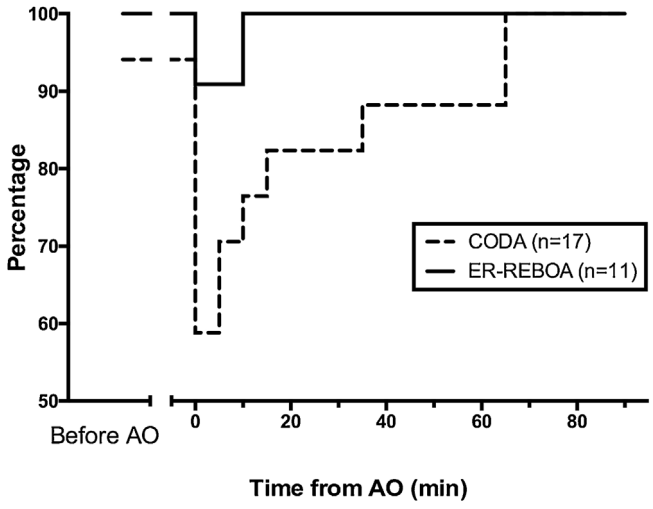


Fig. 2. Cumulative Incidence of Arterial Line Monitoring Around the Time of Aortic Occlusion (AO).

Immediately at the time of AO, there was a higher prevalence of arterial line monitoring in patients with the ER-REBOA™ catheter compared to the CODA® catheter. Eventually systemic arterial line monitoring was obtained for all patients after AO, but was accomplished significantly sooner in patients with the ER-REBOA™ catheter due to the immediate availability of the arterial line port.

Hemodynamic measurements and observations

The aggregate hemodynamic trends of HR and BP around the time of AO for each group were analyzed in 5-minute blocks (see Fig. 3a and b). More patients (76%, 13 out of 17) undergoing zone 1 AO had an acute change (increase or decrease $\geq$ 10 beats per minute) in their heart rate immediately after AO as compared to patients (30%, 3 out of 10) undergoing zone 3 AO ($p = 0.048$). A significant immediate increase in blood pressure was seen in zone 1 and zone 3 AO. Among zone 1 patients the pre-AO SBP nadir (obtained 4.8 (3.5) minutes before AO) was 64 (19) mmHg, compared to the mean SBP of 124 (29) mmHg within 5 min after AO ($p < 0.01$). Among zone 3 patients the pre-AO SBP nadir was 75 (19) mmHg (obtained 5.0 (2.7) minutes before AO), compared to the mean SBP of 98 (14) mmHg within 5 min after AO ($p < 0.01$).

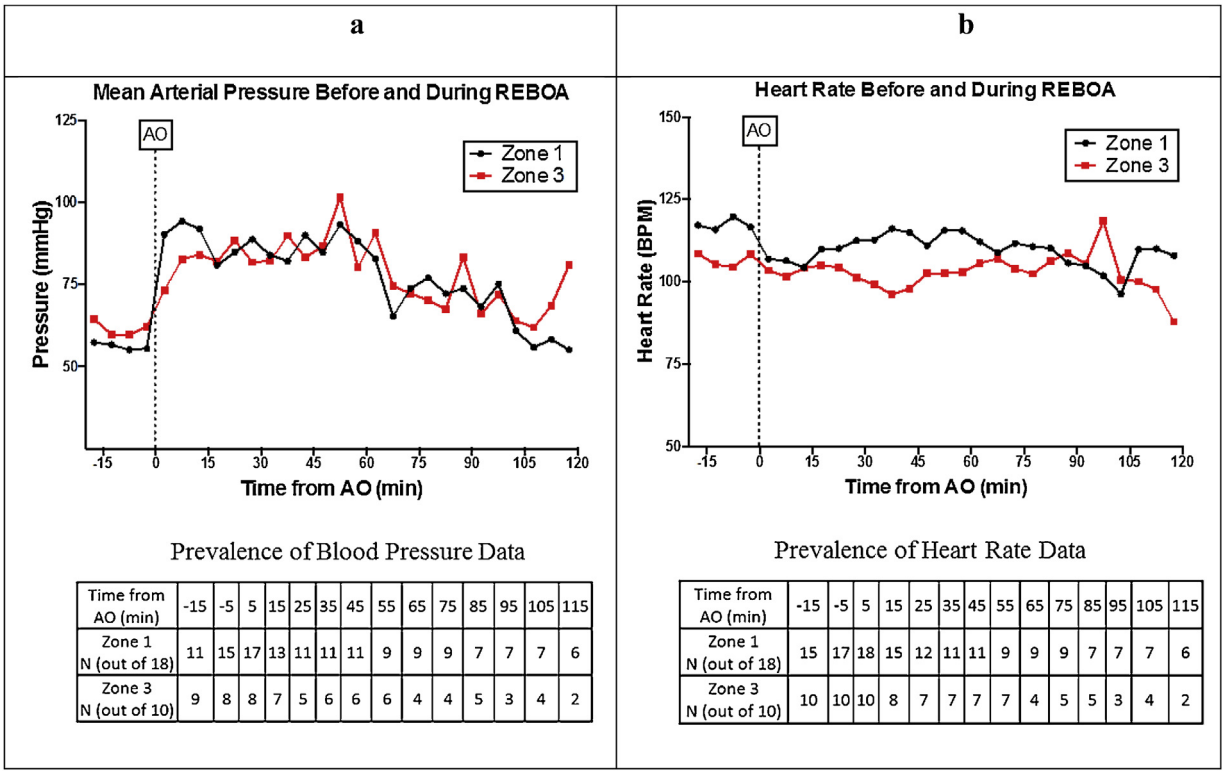
Further analysis of hemodynamic data during AO revealed periods of continued lability. After an initial increase in BP, there was an overall slow downtrend as the duration of AO increased, as seen in Fig. 3a. In some cases there was significant lability of blood pressure with substantial durations of hypotension (SBP < 90 mmHg) or hypertension (SBP > 140 mmHg) during aortic occlusion. These trends appeared to be different depending on aortic occlusion level. In fact, 72% of zone 1 patients had episodes during AO where SBP was less than 90 mmHg as compared to 80% of zone 3 patients ($p = 0.51$). 100% of zone 1 patients had periods during AO where SBP was greater than 140 mmHg as compared to 70% of zone 3 patients ($p = 0.04$). Furthermore, patients undergoing zone 3 AO appear to have a decreased incidence of hypo- or hypertension during AO compared to patients undergoing zone 1 AO, which was further supported when examining the average SBP value thresholds over time (Fig. 4).

The hemodynamic effects of balloon deflation were examined, and as seen in Fig. 5, were more variable than the effects of balloon inflation/AO. In some cases, balloon deflation resulted in acute decreases in SBP greater than 50 mmHg, while in others there was minimal change. The overall mean (SD) decrease in SBP after balloon deflation was 13 (20) mmHg ($p < 0.01$), with similar decreases among groups (14 (21) mmHg vs 12 (18) mmHg for Zone 1 and 3 patients, respectively ($p = 0.85$)).

Discussion

The current study is the first in-depth characterization of the hemodynamic trends before, during, and after REBOA in humans. Despite the heterogeneity and inherent clinical difficulty in obtaining hemodynamic data, as well as differences in the injury patterns and demographics of the patients undergoing zone 1 versus zone 3 REBOA, it is apparent that AO serves to immediately increase BP substantially, with effects of supra-celiac AO being greater than infra-renal AO. There also appears to be significant hemodynamic variability in some cases, with episodes of hypotension and hypertension during AO. In addition, although there were some patients in which REBOA may have exacerbated concomitant proximal injuries, care should be taken to appropriately select patients to avoid potential detrimental hemodynamic effects of REBOA. Lastly, there can be a precipitous decrease in blood pressure following balloon deflation that should be anticipated. These findings have important clinical implications for physicians performing REBOA and directing the resuscitation of these patients.

Previous studies have focused on the effects of increased perfusion and blood pressure proximal to AO, with concerns that AO may potentially exacerbate existing injuries to the brain [19,20], thorax, great vessels, cerebral vasculature, or heart. Eight patients (29%) in this series sustained concomitant brain injuries determined to be caused by their mechanism of injury; however, owing to small sample size and lack of a control group not



Prevalence of Blood Pressure Data

Time from AO (min)	-15	-5	5	15	25	35	45	55	65	75	85	95	105	115
Zone 1 N (out of 18)	11	15	17	13	11	11	11	9	9	9	7	7	7	6
Zone 3 N (out of 10)	9	8	8	7	5	6	6	6	4	4	5	3	4	2

Prevalence of Heart Rate Data

Time from AO (min)	-15	-5	5	15	25	35	45	55	65	75	85	95	105	115
Zone 1 N (out of 18)	15	17	18	15	12	11	11	9	9	9	7	7	7	6
Zone 3 N (out of 10)	10	10	10	8	7	7	7	7	4	5	5	3	4	2

Fig. 3. Aggregate mean arterial blood pressure (Fig. 3a) and heart rate (Fig. 3b) trends before and during AO. The prevalence of blood pressure and heart rate data are also displayed. AO, Aortic Occlusion; REBOA, Resuscitative Endovascular Balloon Occlusion of the Aorta.

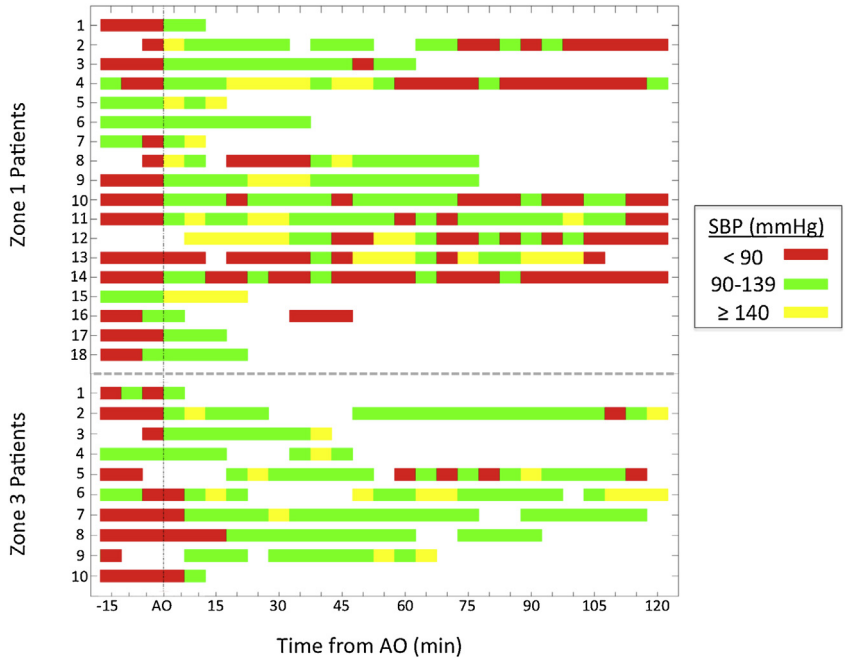


Fig. 4. Heat map assessment of hemodynamics before and during AO. For each patient the median systolic blood pressure value for each 5-minute segment are represented. SBP, Systolic Blood Pressure; AO, Aortic Occlusion.

undergoing REBOA, we could not precisely assess whether, or to what degree, REBOA may have exacerbated these injuries. Notably, although the follow-up was limited, a swine animal model of traumatic brain injury (TBI) and REBOA performed by Johnson et al. [20] did not show differences in TBI progression. Interestingly and unexpectedly, they found that the most significant increases in intracranial pressures occurred during whole blood resuscitation, and were higher in the control group compared to the REBOA group [20]. In the absence of severe cerebral vascular injuries, any deleterious impact with the use of REBOA on TBI progression may be more multifactorial and nuanced than previously thought, and deserves further investigation.

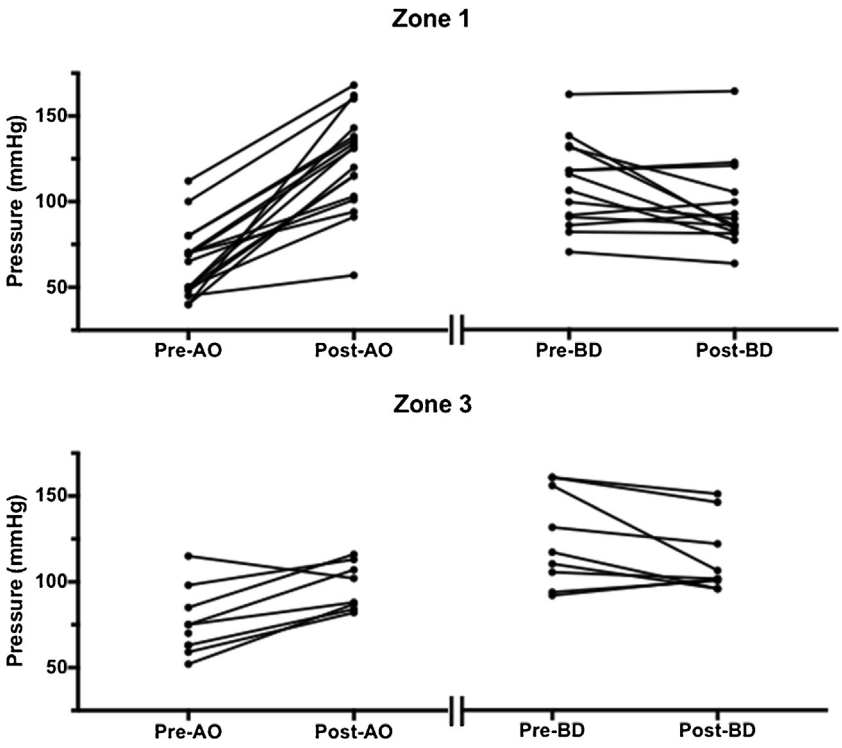


Fig. 5. Systolic blood pressure immediately before and after aortic occlusion and balloon deflation for zone 1 and zone 3 patients. AO, Aortic Occlusion; BD, Balloon Deflation.

There were only 2 instances of thoracic injuries in this series in which REBOA may have exacerbated proximal injuries; this may be indicative of adherence to our institutional protocol [22] and attempting to avoid the use of REBOA in patients who have severe proximal vascular injuries (suggested by physical exam or initial imaging) that could be exacerbated through the use of REBOA. At least one case report [23] has described pre-hospital use of REBOA in a patient with significant aortic injury unknown at the time of placement. This is a difficult injury to rule out, but emphasizes the importance of patient selection in the use of REBOA.

The finding of a majority of patients experiencing episodes of hypertension and hypotension during AO was an unexpected finding and highlights the observation that these patients require constant management of their hemodynamics and resuscitation before, during, and after AO. Studies [15,16,24] have sought to mitigate proximal hypertension and distal ischemia by only partially occluding the aorta in order to allow more blood flow distally. However, the findings of hypotension and lability in some patients during AO deserves consideration and further investigation, especially as it relates to performing other adjunct procedures such as partial REBOA. It is unclear, and beyond the scope of this observational study, what additional factors may have contributed to the fluctuations in pressure. Resuscitative adjuncts, particularly vasoactives and pressurized infusions of blood products, may have contributed to these findings. Given the lack of control groups without REBOA, it is also unclear whether this finding of significant hemodynamic lability is worsened or ameliorated by the use of REBOA and requires further investigation.

Successful balloon deflation is a critical time point in the care of these patients. The findings that most patients in this series had a relatively minor decrease in blood pressure after balloon deflation may be related to adequacy of resuscitation and hemostasis, adequate communication between surgeon, anesthesiologist, and support staff, and slow balloon deflation. However, a potentially precipitous drop in blood pressure (>50 mmHg) upon balloon deflation should be anticipated. Even in the setting of a patient with normotensive or hypertensive BP values with AO, they may, in fact, be under-resuscitated and may not be able to tolerate the normalization of blood volume distribution without further resuscitation. Similar to ruptured aortic aneurysm endovascular repairs, it may take up to several attempts at balloon deflation with continued resuscitation before the patient is able to tolerate sustained balloon deflation.

The ability to transduce blood pressure through an arterial line monitoring port above the level of the REBOA catheter results in an increased ability to monitor systemic blood pressure data around the time of AO (Fig. 2). The transition to the ER-REBOA™ catheter that allows for arterial line transduction in our clinical practice has eliminated the need for an additional procedure for blood pressure monitoring in the immediate resuscitation period, and gives critical data required for patient treatment, including the decision to inflate the balloon for AO. No patient in this series had the device placed without AO; however, in cases where hemodynamic collapse seems probable, and/or definitive hemostasis is not readily available, an ER-REBOA™ catheter with arterial line transduction capabilities can provide accurate data to determine whether AO is in fact required, and immediately achieve AO. The ER-REBOA™ catheter is FDA approved for systemic arterial blood pressure monitoring, and may be used solely for this purpose in patients who are nearing or at high-risk of hemodynamic collapse.

The arterial line monitoring present in all patients who received REBOA with the newer device reflects our institutional adoption of early common femoral artery (CFA) access for potential REBOA candidates. The first step in our clinical algorithm advocates for placement of an arterial line in any transient or non-responder [22]. Our experience [25] has demonstrated that CFA access is the rate limiting step of REBOA and should be done at the first sign of hemodynamic instability. Waiting for loss of a palpable pulse or worsened hypotension only makes an already challenging procedure potentially more problematic. This data demonstrates our awareness of the importance of gathering high-fidelity blood pressure data to make critical decisions in patient care, the relative

ease which AO can be performed once CFA access is established, and is perhaps reflective of the increasing comfort level of faculty in placing CFA lines.

This study has several limitations that are important to discuss. This is an observational study with a relatively small sample size and is limited by the diagnostic tests and interventions being performed during patient care, including the prevalence of hemodynamic monitoring devices. Additionally, the hemodynamic data is confounded by an inability to precisely determine the intra-vascular volume status of the patients before and during interventions, the contributions of vasoactive medications, crystalloid, and blood product administration, and the heterogeneity of patient demographics, injury characteristics, and durations of AO. Lastly, the patient populations undergoing zone 1 vs. zone 3 occlusion appear to be different in several ways including demographics and injury patterns. While this is perhaps expected given that the indications for zone 1 vs zone 3 occlusion are different, it also limits the ability to directly attribute the hemodynamic differences between the two groups. These limitations, combined with a lack of control groups not undergoing REBOA, makes it difficult to ascertain the hemodynamic changes caused exclusively by REBOA.

Despite these limitations, the current study demonstrates that REBOA serves to immediately increase BP substantially, with effects of supra-celiac AO being greater than infra-renal AO; however, this effect may not be robustly sustained with continued AO as there were significant hemodynamic fluctuations during AO observed in the majority of patients.

Conclusions

Significant hemodynamic alterations occur before, during, and after AO. The effects of Zone 1 AO on blood pressure and heart rate appear different than Zone 3 AO. This may have important implications for cardiac or cerebral function and perfusion goals, particularly with concomitant proximal injuries.

Funding

This study was funded in part by a grant from the United States Department of Defense, grant number W81XWH-15-1-0025.

Conflict of interest

Dr. Megan Brenner is a clinical advisory board member for Prytime Medical Inc. The remaining authors have no conflicts of interest.

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