JEVTM

Journal of Endovascular Resuscitation and Trauma Management

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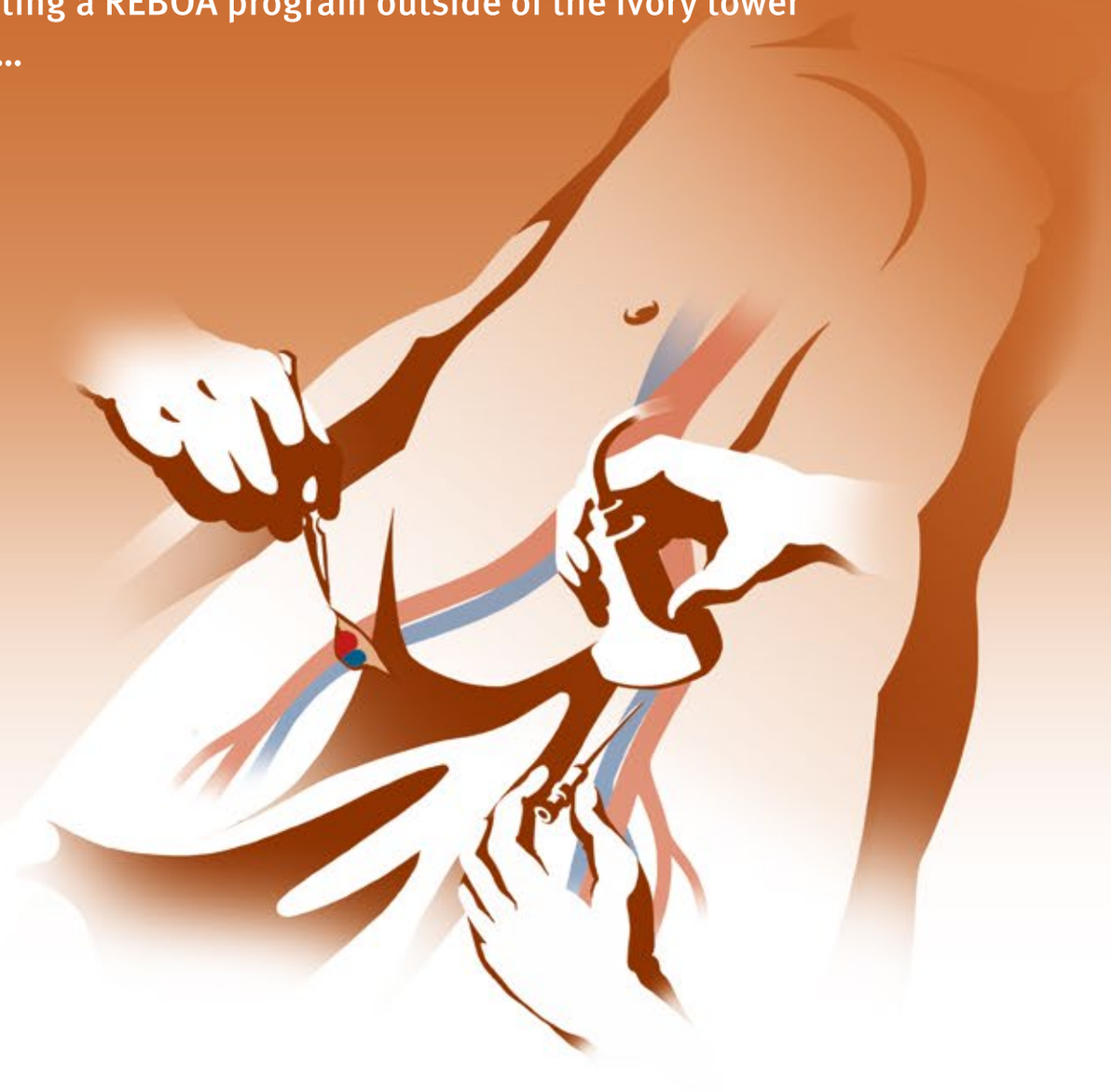
Issue Highlights

Novel interventions for non-compressible trauma hemorrhage
REBOA to facilitate V-V hemodiafiltration in an intoxicated patient
A role of endovascular surgery in severe venous injury
A novel automated endovascular variable aortic control device
Implementing a REBOA program outside of the ivory tower
And more...



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This is the *Journal of Endovascular Resuscitation and Trauma Management*. We want to provide a truly Open Access platform for the dissemination of knowledge and peer-reviewed research in the field of endovascular and hybrid hemorrhage control.

To achieve this, we do not wish to be bound by medical discipline, country, resource or even the conventional rules of medical publishing. To achieve this goal, we have assembled an Editorial Board of clinicians and scientists who are experts within the field. This project is generously supported by a grant from Örebro University Hospital Research Unit.

We are keen to receive manuscript submissions that present new original findings, review important topics or educate our readers on any aspect of hemorrhage control, where an endovascular technique has been employed. This can either be in isolation or in combination with open surgical techniques (hybrid surgery). For further information for authors, please see *www.jevtm.com*.

As the subject of hemorrhage and resuscitation is a common problem across many medical disciplines, we encourage submissions from all specialties: vascular, trauma, acute care, obstetrics, emergency medicine, to mention a few.

The Journal will publish quarterly and will be truly Open Access. There will be no article processing charges or publishing fees. All articles will be published online and indexed using a digital object identifier. The Journal aims to be PubMed cited by 2019.

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In cooperation with Örebro University Hospital and Örebro University, Sweden.

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EVTM Society

Join the Endovascular Resuscitation Platform

The EVTM society is a non profit organization that aims to share information on advanced methods for bleeding control and endovascular resuscitation, exchange of data, and cooperation and education. It is also designed to serve as a professional platform for the multidisciplinary approach.

By joining the EVTM Society you will be part of this global development.

To join, please visit jevtm.com and click on "Join EVTM Society" in the menu.

Membership is free at this stage.

Vision and Mission:
Our mission is to promote optimal treatment and new methods for bleeding control in trauma and non-trauma patients, and state-of-the-art endovascular resuscitation. This will be achieved by a joint international body that will support the following:

- A web-based free platform for EVTM issues (jevtm.com).
- JEVTM – the Journal of Endoascular Resuscitation and Trauma Management, an open access peer-reviewed journal.
- The EVTM round table symposium, a platform for continuous debate and data exchange.
- Educational opportunities in the form of manuals (Top Stent), courses, workshops, and web seminars.
- Promoting open dialogue and cooperation between societies, organizations and the industry.
- Promoting new guidelines and recommendations for EVTM-related issues and protocols.
- Promoting research in EVTM-related areas, both human and animal.
- Promoting PR for EVTM issues, grants, and collaboration with industry.
- Encouraging residents and young colleagues to carry out research on EVTM issues.
- Promoting cooperation and data exchange with other medical instances.

Structure:
The EVTM council, led by the society chair will change membership periodically (i.e., after two years). The council aims to have one or two representatives from each participating country and discipline.

The EVTM society is supported at this stage by Örebro University Hospital in all financial respects (as part of EVTM research group support). This support has been granted for the forthcoming two years.

The main task of the council is to pave the way for the EVTM venture, and promote the JEVTM/EVTM symposium, EVTM-related courses, cooperation, and free exchange of information.

Members will obtain free access to all JEVTM information and discussions as well as regular updates on EVTM-related activities, education, and developments. Members will also be offered a reduced fee for the EVTM round table symposium.

EVTM Society is registered in Sweden, and is managed in collaboration with the EVTM program at Örebro University Hospital, the JEVTM journal and web platform, and other institutes.

Since the society is registered in Sweden, it will follow the rules and guidelines of the Swedish government and the EU. Expansion to other countries is welcome, but should follow our ethical guidelines and the EVTM society should be named in all documents appropriately.

Call for collaboration: We call out to physicians with an interest in endovascular resuscitation, trauma and bleeding management. We need the contributions of the medical professionals who want to be a part of our venture.

Please consider joining by filling out the form at:
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Author Guidelines

A manuscript submitted to the Journal must constitute a unique piece of work that is not under consideration for publication, in part or whole, by another journal.

Original Studies
Manuscripts reporting unique scientific studies should be no longer than 3000 words. They should consist of the following sections:
Introduction: This should concisely present the background to the problem that the study hopes to answer. A hypothesis should be clearly stated.
Methods: This section should be suitably detailed to permit replication of the study. The regulatory permissions for the study should also be detailed, e.g. Institutional Review Board, ethical committee etc... including a protocol/registration number. Where animal research has been undertaken, the institutional animal care and use guidelines that have been followed should be clearly stated.
Results: These should involve the reporting of the salient positive and negative findings of the study in clear language. The use of images, figures and tables are encouraged, of which the data should not be duplicated in the prose. There is no maximum number of figures or tables, but these should be appropriate to the study. should be reported to three decimal places.
Discussion: This should place the reported study findings in the context of the literature. Limitations and future direction should also be discussed. Authors must be careful to ensure that conclusions are not overstated and are supported by data.

Editorials
Short, focused Editorials on an important aspect of endovascular hemorrhage control are welcomed. These should endeavor to bring attention to an important topic, or accompany an article published within the journal. The latter will be invited by the Editor. Submitted manuscripts should be no longer than 1500 words.

Narrative Review Articles
This style of article can afford the author considerable latitude in examining a pertinent topic in endovascular hemorrhage control. The literature should be examined objectively and presented to the reader in the context of current understanding. The author should be able to synthesize a narrative, which leaves the reader with a good understanding of an emerging or controversial topic. The author is welcome (and encouraged) to express an opinion, but where this is the case, it should be clearly stated.

Articles should be a maximum of 5000 words. There is no formal structure; however, the use of logical headings/sub-headings is important to enable readers to follow the article easily. The abstract should also be unstructured and be a maximum of 150 words.

Systematic Reviews and Meta-Analyses
Where there is a topic within the subject area of endovascular hemorrhage control that has a substantial evidence base, a Systematic Review with/without a Meta-Analysis is considered more appropriate than a narrative review article. These articles should follow the methodology established by PRISMA. Submission should be a maximum of 5000 words and authors should include a PRISMA checklist in their submission. The overall aim is to provide a pooled analysis that enables firm conclusions to be drawn on a particular subject.

Tips and Techniques
In the evolving world of endovascular hemorrhage control, the advice and opinion of actively practicing clinicians is of great importance. Both solicited and unsolicited submissions are reviewed, both on major or minor components on endovascular techniuqes . This can be presented in the context of â€œevidenceâ€ or just as an opinion. The use of quality images and diagrams is encouraged. The submission should be a maximum of 1500 words.

Images of Interest
Rather than accept case reports, the Journal accepts images of interest, which include a short commentary. The aim of this section is to demonstrate and illustrate an educational message, rather than just to demonstrate dramatic pathology. Images can be submitted as a multi-panel with a series of scans/photographs in order to support the message presented in the narrative. The submission should be a maximum of 250 words.

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Contents

Original Article

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Implementing a REBOA Program Outside Large Academic Trauma Centers: Initial Case Series and Lessons Learned at a Busy Community Trauma Program

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Background: Resuscitative endovascular balloon occlusion of the aorta (REBOA) has become an established adjunct to hemorrhage control. Prospective data sets are being collected, primarily from large high-volume trauma centers. There are limited data and guidelines, to direct the implementation and use of REBOA outside these highly resourced environments. Smaller centers interested in adopting a REBOA program could benefit from closing this knowledge gap.
Methods: A clinical series of cases utilized REBOA at a busy community trauma center (ACS Level 2) from January 2017 to May 2018. Seven cases are identified and reported, including outcomes. Considerations and 'lessons learned' from this early institutional experience are discussed.
Results: REBOA was performed by trauma and acute care surgeons for hemorrhage and shock (blunt trauma $n = 3$, penetrating trauma $n = 2$, no trauma $n = 2$). All were placed in Zone 1 (one was placed initially in Zone 3 then advanced). The mean (SD) systolic pressure (mmHg) before REBOA was 43 (30); post-REBOA pressure was 104 (19). Four of the patients were placed via an open approach, and three were percutaneous ($n = 2$ with ultrasound). All with arrest before placement expired ($n = 3$) and all others survived. Complications are described.
Conclusions: REBOA can be a feasible adjunct for shock treatment in the community hospital environment, with outcomes comparable to large centers, and can be implemented by acute care and trauma surgeons. A rigorous process of improvement programs and critical appraisal are critical in maximizing the benefit in these centers.

Keywords: *REBOA; Community; Lessons Learned*

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Disclaimer: This research was supported (in whole or in part) by Hospital Corporations of America (HCA) and/or an HCA affiliated entity. The views expressed in this publication represent those of the author(s) and do not necessarily represent the official views of HCA or any of its affiliated entities. The views expressed in this article are those of the author and do not necessarily reflect the official policy or position of the Department of the Navy, Department of Defense, nor the U.S. Government. J Glaser is a military service member. Title 17 U.S.C. §105 provides that 'Copyright protection under this title is not available for any work of the United States Government.' Title 17 U.S.C. §101 defines a U.S. Government work as a work prepared by a military service member or employee of the U.S. Government as part of that person's official duties.

INTRODUCTION

While resuscitative endovascular balloon occlusion of the aorta (REBOA) has been described as a tool for the control of exsanguinating hemorrhage since 1954 [1], there has been an explosion in its use globally within the last decade [2–5]. Although practiced in Japan and Europe for longer, there has been a recent increase in the utilization of REBOA in the trauma systems of the United States. Initial studies utilizing REBOA have been performed at large highly resourced trauma centers with reassuring results. The American Association for the Surgery of Trauma (AAST) prospective Aortic Occlusion for Resuscitation in Trauma and Acute Care Surgery (AORTA) registry continues to collect and analyze data on utilization and trends for REBOA use.

While the defined role of the multi-institutional study is to "capture contemporary methods of aortic occlusion (AO) with the purpose of refining clinical protocols and future data collection", the database fails to capture much of the available data regarding REBOA use at smaller trauma centers not enrolled in the study. Illustrating this point, 103 centers out of the 256 hospitals currently using the newer ER-REBOA catheter®, are Level II or III trauma facilities [6]. The AORTA database includes data from roughly 22 centers, while the National Trauma Data Bank reports over 60 centers using REBOA, therefore, the AORTA database only captures a small portion (approximately 1/3) of the available usage data. Thus, preliminary published results may disproportionally skew findings toward large and robust trauma systems due to the higher use of REBOA at those institutions [5].

In an attempt to increase awareness of REBOA complications and pitfalls, the Basic Endovascular Skills for Trauma (BEST) study group has published consensus guidelines on the implementation, pitfalls and best practices for the application of REBOA – comprised primarily of expert opinion, a compilation of case reports, and anecdotal evidence [7]. Military use has also been carefully utilized, with associated guidelines that could serve as a starting point for institutional implementation [5]. Currently, there is a lack of published data on the implementation and institutional 'lessons learned' from smaller trauma centers in the United States and guidance on best practices for the safe institutional adoption of REBOA for early hemorrhage control.

The following case series represents the early experience of a smaller trauma center serving multiple counties in coastal South Carolina, following the adoption of REBOA technology. When the FDA approved Prytime Medical's ER-REBOA® catheter for medical use in October 2015, the decision was made to include REBOA as a potential alternative to Emergency Department (ED) thoracotomy at this institution in January 2017, while it was still a growing Level II trauma center. We report our early institutional successes, as well as lessons learned, during the initial 16 months after implementation.

PATIENTS AND METHODS

A clinical series of all cases utilizing REBOA, from January 2017 to May 2018, were included. The use of REBOA was considered part of the routine clinical care for traumatic, vascular, and/or acute surgical bleeding. Indications for use were for the emergent management of hemorrhage. In all cases, the ER-REBOA® was placed by acute care and trauma surgeons. Observations and data extraction were performed retrospectively, with an exempt determination from the Edward Via College of Osteopathic Medicine Institutional Review Board.

Case Study 1

A 52-year-old male presented to an outside hospital with shortness of breath and left lower quadrant pain. He was diagnosed with a 7.8 cm × 7.8 cm ruptured infra-renal abdominal aortic aneurysm with extensive retroperitoneal hemorrhage, and transferred to this institution (Table 1). Prior to arrival, the patient had multiple episodes of hypotension and was transiently responding to 2 liters of normal saline and 2 units of packed red blood cells (PRBCs). Upon arrival, the patient was confused, pale, cool and diaphoretic. Blood pressure was unobtainable with non-invasive blood pressure cuffs. Access for REBOA was initially unsuccessful with palpation alone (by the accepting vascular surgeon), but subsequently obtained with ultrasound (US) guidance (US-guided percutaneous, 18 gauge access to the right common femoral artery (CFA), upsized over a .035 wire directly to a 7-Fr sheath) by the on call trauma surgeon. The ER-REBOA balloon was positioned into Zone 1 using anatomic landmarks, placement confirmation was made with portable chest x-ray, and the balloon was inflated with 10 ml of contrast/saline mix (Figure 1). Systolic blood pressure (SBP) became obtainable at 110 mmHg (sustained), massive transfusion protocol (MTP) was initiated and the patient was taken for laparotomy. The aortic rupture was identified, proximal and distal control obtained, and the REBOA balloon was deflated under direct visualization. The aorta was repaired with a tube graft. Post-operative care was uncomplicated (sheath removal by vascular surgery on post-operative day (POD) 1) with the exception of temporary acute kidney injury (creatinine elevation to 2.30 POD 2). The patient was discharged home POD 11 following resolution of his acute kidney injury.

Case Study 2

A 45-year-old male presented after a fall from five stories (Table 1). The patient was alert but confused, with a heart rate of 115 b/m and SBP of 130. The initial physical exam revealed bruising to the abdomen, an unstable pelvis, and an open left elbow deformity. The initial extended focused assessment with sonography

Table 1 Demographics and summary of REBOA use in seven patients. Time to occlusion is calculated from the time that REBOA is decided upon (verbal confirmation or obtaining the access kit) until the balloon is verbalized to be inflated.

	Patient						
	1	*2*	*3*	*4*	*5*	*6*	*7*
Age (yrs)	52	45	82	71	37	19	20
Sex	Male	Male	Female	Female	Female	Male	Male
Indication/mechanism	Ruptured AAA	Fall	MVC	Found down	GSW	GSW	Crush
Injury severity score (ISS)	n/a	22	21	0	25	25	18
Access method	Ultrasound	Ultrasound	Cut down	Cut down	Percutaneous	Cut down	Cut down
SBP before REBOA, mmHg	60	63	0	0	70	60	50
SBP after REBOA, mmHg	110	116	131	70	110	90	100
Cardiac arrest before REBOA	No	No	Yes	Yes	No	Yes	No
Zone of Inflation	1	3, 1	1	1	1	1	1
Admission pH	7.18	7.20	n/a	7.81	7.10	6.54	7.24
Admission base deficit	–8	–11	n/a	>30	–14	<–30	–5
Time to occlusion, min	11	4, 6	7	6	2	n/a	n/a
Time of occlusion, min	28	32	n/a	n/a	19	n/a	9
Surgery after REBOA	Yes	Yes	Yes	No	Yes	Yes	Yes
Embolization after REBOA	No	Yes	No	No	No	No	No
Complication	Temporary acute kidney injury	No	No	No	Transient ischemia, pulmonary embolism	No	Ischemia, amputation, pseudo-aneurysm
Outcome	Alive	Alive	Death	Death	Alive	Death	Alive

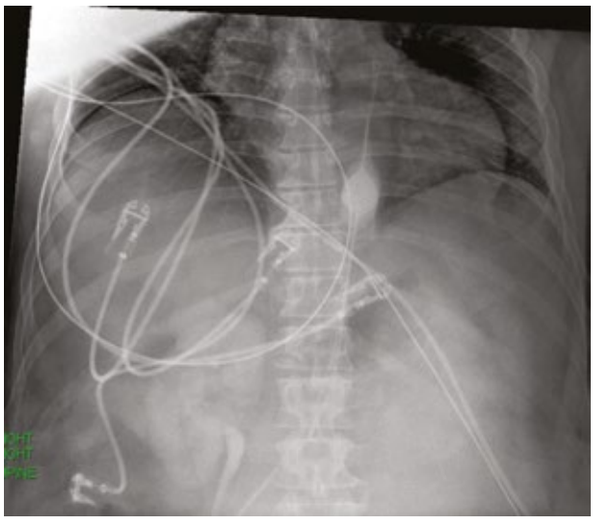

Figure 1 ER-REBOA balloon inflated in Zone 1 via landmarks. Case 1.

for trauma (eFAST) exam was negative for fluid, with the absence of lung sliding on the left chest suggesting a pneumothorax. The patient rapidly declined, becoming obtunded, leading to intubation and tube thoracostomy, which resulted in no rush of air nor evacuation of significant blood. SBP after intubation and insertion of chest tube was 100 mmHg and deteriorated to 63 mmHg despite 1 L of normal saline and 1 unit of PRBCs, and MTP was activated. As the patient was felt to be in a 'peri-arrest' condition, despite initial blood product resuscitation, REBOA was placed (US-guided percutaneous, 18 Gauge access to the right CFA, upsized over .035 wire directly to a 7-Fr sheath) in Zone 3 (using anatomic landmarks) and inflated with 5 ml of saline. The SBP did not respond (via cuff measurement) after 4 minutes of balloon inflation, therefore the balloon was deflated, advanced to Zone 1, and re-inflated with 8 ml of saline. This resulted in a stable SBP of 116 mmHg. The patient was taken to CAT scan in an effort to rapidly rule out neurologic injury and evaluate the extent of the pelvic fracture (and facilitate mobilization of the interventional radiology team), followed immediately by the operating room (OR) for exploration.

Operatively, the ileo-colonic mesentery was found to be avulsed with active bleeding. A non-expanding Zone 2 retroperitoneal hematoma, grade 1 liver and splenic lacerations were identified and packed. The REBOA balloon was deflated after 32 minutes. Devitalized bowel was resected and the patient's abdomen was left open.

He was then taken to the interventional radiology (IR) suite for pelvic angiography and no active extravasation was found. After stabilization in the ICU, the REBOA sheath was removed (approximately 8 hours later). The patient underwent subsequent surgical correction of multiple fractures. On hospital day 6, the patient was neurologically intact and transferred to a hospital closer to his home (at family request) for recovery.

Case Study 3

An 82-year-old female presented after a motor vehicle crash (MVC) from a distant rural county (Table 1). The patient was unresponsive on presentation, cold, pale, and mottled, with a thready femoral pulse. Initial chest x-ray was negative for hemorrhage. Within minutes of arrival, the patient lost a palpable pulse (PEA on monitor). CPR was started, and the patient was intubated. Bilateral finger thoracostomies resulted in no blood or rush of air. An eFAST exam was indeterminate but revealed no pericardial tamponade. The decision was made to utilize REBOA (Cut down, 18 Gauge access to the right CFA, upsized over .035 wire directly to a 7-Fr sheath). The balloon was placed and inflated in Zone 1 (anatomic landmarks with recorded insertion distance of 35 cm measured at the sheath, 8cc saline inflated) and confirmed with x-ray. Continued CPR and MTP resulted in the return of spontaneous circulation (ROSC). Post-ROSC vitals revealed an SBP of 131 mmHg and a heart rate of 121. A diagnostic laparotomy in the trauma bay revealed hemoperitoneum. She again became pulseless, and ROSC was achieved a second time. The patient was taken to the OR, where she was found to have a grade 3 splenic laceration, multiple mesenteric rents, Zone 1 and bilateral Zone 2 retroperitoneal hematomas. The patient lost her pulse again in the OR, further resuscitation was felt to be futile and the patient expired.

Case Study 4

A 71-year-old female presented after a reported fall from standing, 6 hours prior, where she struck her right hip (Table 1). She had no external signs of trauma. Medical history included uncontrolled diabetes and dementia, and was noted by Emergency Medical service personnel to have elevated blood glucose (BG>500 mg/dl). Upon presentation, the patient was alert but confused, with bilateral breath sounds, but hypotensive (SBP 88) and tachycardic (HR 130). Chest x-ray and eFAST exam were unremarkable. Her mental status rapidly declined, requiring intubation, and precipitated subsequent cardiac arrest. CPR was started. Two units of PRBCs were rapidly administered in addition to a liter of normal saline. Rather than proceed directly to thoracotomy in this elderly patient with a questionable history for traumatic arrest, a REBOA catheter was placed (Cut down, 5-Fr sheath access to the right CFA, upsized over .035 wire directly to a 7-Fr sheath) and inflated in Zone 1 (anatomic landmarks, Figure 2), with a return of carotid pulses after AO. No effort at imaging confirmation was made due to the desire for uninterrupted CPR. Arterial blood gas results revealed a profound alkalosis (pH 7.81, pCO_2 >50, HCO_3 63.3, Base Excess >30), lactate of 10.11, and glucose 547, and hemoglobin 5.4 mg/dl. The patient went into persistent PEA and expired.

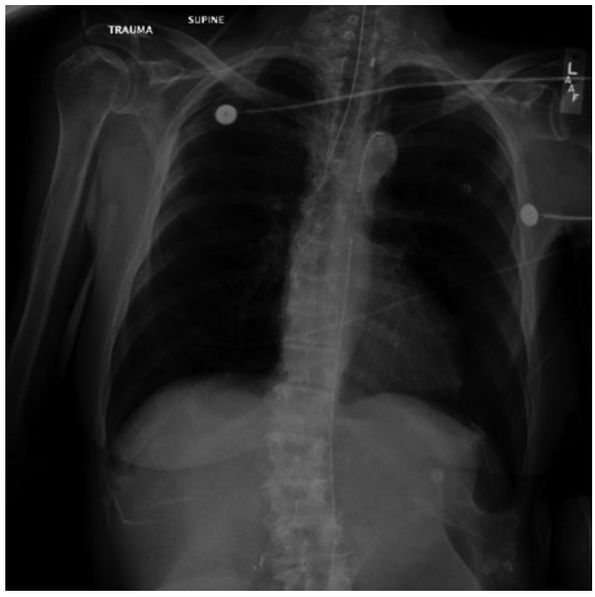

Figure 2 ER-REBOA catheter positioned in Zone 1 via anatomic landmarks. Case 4.

Case Study 5

A 37-year-old female presented after sustaining a close proximity gunshot wound to the left lower quadrant of the abdomen (Table 1). She arrived neurologically intact. Initial SBP was 143 and was tachycardic (125 bpm). She was taken immediately to the OR for exploratory laparotomy, and upon induction of anesthesia became hypotensive. MTP was activated. Laparotomy revealed an expanding Zone 3 retroperitoneal hematoma with extension to Zone 2 on the left. During exposure, significant bleeding was encountered, and despite packing, the patient's SBP fell below 70 mmHg. A REBOA catheter was inserted into Zone 1 (percutaneous via palpation, 5-Fr sheath access to the right CFA, upsized over .035 wire to a 7-Fr sheath). AO resulted in normalization of SBP and cessation of intra-abdominal hemorrhage, allowing identification and local vascular control of a partially transected (<50% circumference) left external iliac artery. The aortic balloon was deflated, and the arteriotomy was repaired. The femoral 7-Fr sheath with REBOA catheter were left in place post-operatively with the balloon fully deflated, she was left in discontinuity with an open abdomen, and transferred to the ICU for further management.

In the ICU the patient was noted to have loss of pulses distal to the REBOA access site. The catheter and sheath were then removed, resulting in the return of palpable distal pulses. Further operative care was routine, though complicated by respiratory failure (secondary to pulmonary embolus, heparin-induced thrombocytopenia, and IVC filter placement). She was discharged home on POD 16.

Case Study 6

A 19-year-old male was transferred from an outside hospital after suffering a gunshot wound to the right lower abdomen and right buttock (Table 1). During transport via helicopter, the patient arrested twice, was intubated and resuscitated with ROSC. The patient received 5 liters of saline, 10 units of PRBCs, and tranexamic acid prior to arrival. Upon arrival, the Glascow Coma Scale (GSC) was 3. Airway was confirmed, bilateral chest tubes were placed with a minimal return of blood or air. The patient had palpable femoral pulses and was tachycardic. MTP was continued with a focus on recovering a balanced blood product resuscitation. Epinephrine, sodium bicarbonate, and calcium chloride were given. SBP declined to <60 mmHg, with intermittent bradycardia, and so a REBOA was placed (cut down, 5-Fr sheath access to the right CFA, upsized over .035 wire to a 7-Fr sheath), inserted in Zone 1 via anatomic landmarks and inflated. The patient had a transient SBP increase and was taken to the OR for laparotomy. In the OR, multiple injuries were noted including a transected right iliac artery and expanding hematoma in the pelvis. Despite maximal efforts at bleeding control, including the assistance of the on call vascular surgeon, the patient was unable to be recovered and expired in the OR.

Case Study 7

A 20-year-old male presented after sustaining an abdominal crush injury and pinning from the bucket of heavy excavating machinery (Table 1). The patient had a patent airway, normal neurologic exam and with a GCS of 15 and palpable distal pulses on arrival. He was initially hypotensive, however, became normotensive after receiving crystalloid resuscitation. He had diffuse abdominal tenderness and periumbilical ecchymosis. A FAST exam was negative. He was taken to CT, during which his SBP began to drop. MTP was initiated and he was taken to the OR immediately. Significant arterial bleeding was noted near the mesenteric root.

Initial attempts at bleeding control failed and the SBP fell to <50 mmHg. A REBOA balloon was placed (cut down, 5-Fr sheath access to the right CFA, upsized over .035 wire to a 7-Fr sheath), advanced into Zone 1 via anatomic landmarks and inflated. The arterial bleeding at the mesenteric root was then controlled and ligated, after which the REBOA balloon was deflated. No further injuries were noted. Devitalized bowel was removed, continuity was restored and the abdomen was closed. The REBOA sheath was removed and the left femoral arteriotomy was repaired primarily with the assistance of the on call vascular surgeon. While still in the OR, the patient lost distal pulses and Doppler signals on the left side. The repair was reopened, and Fogarty thrombectomy was performed for the removal of a large amount of clot, and return of dopplerable signal. The patient was then heparinized and was therapeutic. A prophylactic four-compartment fasciotomy was performed and the patient was transferred to the ICU for further management.

On POD 3, after extubation, the patient was unable to move his left toes, foot or ankle. He developed worsening ischemia to the Left Lower Extremity (LLE), resulting in myonecrosis at his fasciotomy site. Despite maximal efforts at limb salvage, the patient required LLE amputation on POD 15. On POD 23, he was noted to have a left femoral pseudo-aneurysm, with bleeding, requiring operative repair. He continued to recover and was discharged to rehab on hospital day 48.

DISCUSSION

This clinical series represents the initial experience of using the REBOA device at a busy ACS Level II trauma center, and offers an opportunity to evaluate successes and pitfalls in this environment. Our institution provides 24/7 in-house trauma surgeons, meaning that the trauma and acute care surgeon is likely to be the first surgical line of defense for a range of traumatic and non-traumatic cases of hemorrhage. A priori, a review of institutional practice for controlling hemorrhage was undertaken and, as is paralleled in the literature, hemorrhage *continues* to be a leading cause of trauma-related mortality. After this review (and with the FDA approval of the Fluoroscopy free, 7-Fr delivery ER-REBOA) it was determined that the addition of a 'new technique' to control hemorrhage may be beneficial.

The initial team exposure and introduction to endovascular aortic occlusion devices occurred via training provided at the Center for Advanced Medical Simulation and Learning (CAMLS) Center in Tampa, Florida. All staff, residents and trauma nurses had the opportunity to participate in practice on the REBOA Access Task Trainer (RATT) at the simulation center, or at a similar local training session. This training and exposure were all simulation based. Two staff surgeons had also previously completed the American College of Surgeons BEST course prior to implementation. No additional local credentialing was required for board-certified critical care surgeons to utilize REBOA, as it is considered a core competency at this institution. Residents were allowed to assist, but not perform, REBOA. After every use, a detailed performance review is performed, with critical appraisal of the indication, technique, and outcomes.

The trauma team initially created its own supplemental catheter insertion kit, which has since been updated to the pre-made Prytime convenience kit. The ER-REBOA catheter and convenience kit are kept together in each primary trauma bay, with another stocked in the main trauma OR. Continuous and portable arterial waveform monitoring capabilities were not initially available, but have been added to the trauma resuscitation bays and are utilized with REBOA when required. Ultrasound is available in all resuscitations. Access choice is currently surgeon preference, but with increasing exposure to US-guided access, we feel that this modality will be chosen more frequently.

Institutional indications for use were initially agreed upon within the trauma team, but not formally defined. These have since been formalized and largely parallel algorithms from larger trauma centers [7]. In general, the critical first step is the recognition of a patient in shock, from both traumatic and non-traumatic causes. SBP <90 mmHg, HR >120 b/m, shock Index >1.3, or BD less than −5 have been used to define shock. Contraindications for REBOA include arrest from non-hemorrhagic causes, PEA arrest >10 min, known proximal traumatic aortic dissection, and cardiac tamponade/obstructive shock. However, as is illustrated in case study 4 (very likely to be a medical arrest) it can be difficult to identify the precipitating cause of cardiac arrest in every situation. When a patient arrives in arrest, with an unknown cause (trauma as a possible mechanism), all efforts are made at resuscitation.

Additionally, we do not consider age as a contraindication to REBOA. When a witnessed traumatic arrest occurs in an elderly patient, we consider it reasonable to resuscitate with the adjunct of an intra-aortic balloon, preferring this over the additional morbidity of an invasive open thoracotomy – although this is highly situation and provider dependent.

All deaths in our cases ($n = 3$) were patients that arrested prior to REBOA deployment. All patients that were hypotensive prior to REBOA deployment ($n = 4$) survived. Considering the results from the AORTA database and dismal survival rates of all aortic occlusion when a patient is pulseless (3% survival to discharge [5]), it may be reasonable to include prehospital arrest as a contraindication for using REBOA.

The significance of these observations is, of course, limited due to the small sample size, but in general, we feel that *early* utilization of REBOA (prior to arrest) offers an advantage in our institution. In the case of non-traumatic bleeding, where specialty services may be delayed, REBOA may add a layer of security and 'buy time.' To illustrate this point, the first application of REBOA at this facility was for a hemodynamically unstable ruptured AAA (rAAA) patient in extremis in the ED, bridging the patient to the successful definitive management of the AAA. It also bears mention that this initial success (seen in case 1) resulted from the collaboration of the trauma and vascular surgeon, and highlights that team communication, with the inclusion of vascular surgeons, can be an important aspect of safe implementation at smaller facilities. Also of note is the use of REBOA in a rAAA with a wire-free, non-steerable device in the ED. Careful attention to tactile feedback and the use of x-ray permitted safe insertion is required as there is potential for the catheter to advance out into the aneurysm sac and cause further damage.

This retrospective case series also highlights several pitfalls, especially in regards to vascular complications following REBOA. It is important to point out that there were no balloon specific complications – such as aortic lacerations, balloon ruptures, etc. – which are consistent with national data. However, the patients in cases 5 and 7 suffered sheath and access related complications – including one amputation. In case 5, where the sheath was left in place to the ICU, limb ischemia was only transient and resolved with sheath removal. This may be related to the smaller arterial diameter of this female patient, or possibly vascular spasm, but it resolved nonetheless. In case 7, the sheath was removed in the OR, however early thrombosis of the access site led to a period of ischemia that ultimately required limb amputation, despite salvage efforts by trauma and vascular surgeons. While practice patterns are variable, it *may* be prudent to remove the REBOA sheath prior to leaving the OR. Certainly, a high level of vigilance during and after resuscitation is necessary. If the decision to leave the OR with the sheath in place is made, assessment of distal perfusion with angiography and physical exam is highly recommended.

After review, we have implemented new REBOA placement and post-placement management guidelines. For example, in several cases above there is no recording of insertion distance. It is now protocol to record distances. Though still not always recorded, there is now a mechanism for feedback, proper quality assurance, and review. Documented vascular exams pre-insertion, during use and hourly post-placement are also now mandated, and are tracked by the trauma performance improvement (PI) team. Doppler evaluation for insertion site pseudo-aneurysm within 48–72 hours post-insertion is recommended. An angiogram of the access extremity is ordered if the patient shows signs of lower limb ischemia. Vascular surgery is available to assist with any complications, and mandatory vascular consultation for every REBOA case is being considered. All attempts are made to limit each occlusion time to less than 30 minutes (though it is understood that some guidelines allow 30–60 minutes occlusion for Zone 1, longer for Zone 3 [8]), with inflation/deflation times recorded in every case. Our local guidelines recommend removing the sheath 'as soon as possible once resuscitative goals have been achieved.' This is purposely ambiguous to allow for practice variability, but the use of thromboelastometry, thromboelastography, or other adjuncts may be helpful

REBOA DOCUMENTATION ☐ N/A

(circle responses or fill in the blank)

Prehospital **CPR** required? Y / N

CPR in Trauma Bay? Y / N **CPR** in OR? Y / N

Signs of life present *on arrival*:

- Pupillary Response? Yes / No
- Organized rhythm on monitor? Yes / No
- Spontaneous mvmt? Yes / No

REBOA **"decision to use"** time & MD: _____

Aortic Occlusion (AO) Initiation:

- **Physiology** immediately prior REBOA insertion: SBP_____ HR _____ GCS _____
- CPR ongoing during insertion? Y / N
- **Access site** for REBOA: (1st attempt)
  - R / L femoral art.
  - Palpation / US / Fluro guided
  - Percutaneous / Cut-down
  - Sheath size: 7Fr / Other_____
  - Insertion length: _____ cm
    - ➢ XR confirmation? Y / N
  - Repositioned post XR? Y / N
    - ➢ If yes, to _____ cm
- **Balloon inflation (AO) time:** _____
  - _____ml balloon inflation volume
  - Balloon vol: NS only / contrast mix
  - **5 minutes post AO** physiology: SBP_____ HR _____ GCS _____

AO Discontinuation:

- **Balloon deflation:** (if in stages)

Time	Inflated / Deflated	ml
	Inflated / Deflated	
	Inflated / Deflated	
	Inflated / Deflated	
	Inflated / Deflated	
	Inflated / Deflated	

- **Balloon final down time:** _____
- Catheter / Sheath (circle which) removal time: _____ ← location: OR / ICU

Post REBOA Vascular:

- Pulses in affected extremity post removal?
  DP: Y / N → Palp/Doppler
  PT: Y / N → Palp/Doppler
- Vascular intervention required in REBOA extremity? Y / N

MTP DOCUMENTATION (cont'd from flowsheet):

PRBC #	Admin Time & Location*	FFP #	Admin Time & Location*	Plt 6Pk #	Admin Time & Location*

*Location: ER/OR/IR/ICU

Cryo administration (#/time/location):_____

MTP Termination Time: _____

MTP Totals: (PRBC/FFP/Plt/Cryo)

ER ___:___:___:___

OR ___:___:___:___ (OR totals will also be on anesth. flowsheet)

Other ___:___:___:___

Total ___:___:___:___

Tourniquets:

Tourniquet down time, if applicable: _____

Location: ER / OR

Notes:

_____

_____

_____

_____

_____

_____

_____

_____

_____

_____

_____

Figure 3 REBOA flowsheet used at our institution, included as the final page of the Trauma flowsheet.

to refine these endpoints. This institution utilizes Rotational Thromboelastometry (ROTEM) primarily. We have also created a flow sheet, included in the standard trauma 'packet,' to improve the quality and consistency of data collected in these patients (Figure 3).

These results describe our early experiences in using the ER-REBOA device in the resuscitation of seven hemodynamically unstable patients. We feel that these results are comparable to available national data. While we continue to review local practices, there have already been many lessons learned within our facility and we are considering the utility of expanding to indications described at other institutions [9–11]. Though we have limited experience with other indications at this point, the trauma PI team has identified other cases where REBOA could have potentially been useful. Overall, we feel that any difference in outcomes, particularly complications, may be attributed to the ways in which REBOA was implemented compared to large academic centers with protocols, algorithms, senior mentorship, a rigorous QA process, and a specific credentialing process that requires completion of a standardized REBOA training program. The lack of some or all of the above at our institution may or may not make a difference in outcomes, but data from high-volume institutions suggests these entities are likely to be important. Ideally, this early experience from a smaller trauma center can compress the learning curve of other institutions, and help identify the patients for whom REBOA will confer the most benefit.

REFERENCES

[1] Hughes CW. Use of an intra-aortic balloon catheter tamponade for controlling intra-abdominal hemorrhage in man. Surgery 1954;36:65–8.

[2] Morrison JJ, Rasmussen TE. Noncompressible torso hemorrhage: a review with contemporary definitions and management strategies. Surg Clin North Am 2012; 92:843–58.
[3] Moore L, Brenner M, Kozar R, et al. Implementation of resuscitative endovascular balloon occlusion of the aorta as an alternative to resuscitative thoracotomy for noncompressible truncal hemorrhage. J Trauma Acute Care Surg 2014;79:523–32.
[4] DuBose JJ, Scalea TM, Brenner M, et al. The AAST prospective Aortic Occlusion for Resuscitation in Trauma and Acute Care Surgery (AORTA) registry: Data on contemporary utilization and outcomes of aortic occlusion and resuscitative balloon occlusion of the aorta (REBOA). J Trauma Acute Care Surg 2016;81:409–19.
[5] Brenner M, Inaba K, Aiolfi A, et al. AAST AORTA Study Group. Resuscitative endovascular balloon occlusion of the aorta and resuscitative thoracotomy in select patients with hemorrhagic shock: Early results from the American Association for the Surgery of Trauma's Aortic Occlusion in Resuscitation for Trauma and Acute Care Surgery Registry. J Am Coll Surg. 2018;226:730–40.
[6] Company data, http://prytimemedical.com/ accessed 31 July 2018.
[7] Davidson AJ, Russo RM, Reva VA, et al. BEST Study Group. The pitfalls of REBOA: risk factors and mitigation strategies. J Trauma Acute Care Surg. 2017; *In Press*. doi: 10.1097/TA.0000000000001711
[8] Joint Trauma System Clinical Practice Guideline. Resuscitative Endovascular Balloon Occlusion of the Aorta (REBOA) for Hemorrhagic Shock (CPG ID: 38). JTS CPG. 2017;1–21.
[9] Weltz AS, Harris DG, O'Neill NA, O'Meara LB, Brenner ML, Diaz JJ. The use of resuscitative endovascular balloon occlusion of the aorta to control hemorrhagic shock during video-assisted retroperitoneal debridement or infected necrotizing pancreatitis. Int J Surg Case Rep. 2015;13:15–8.
[10] Okada A, Nakamoto O, Komori M, Arimoto H, Rinka H, Nakamura H. Resuscitative endovascular balloon occlusion of the aorta as an adjunct for hemorrhagic shock due to uterine rupture: a case report. Clin Case Rep. 2017;5(10):1565–8.
[11] Sano H, Tsurukiri J, Hoshiai A, Oomura T, Tanaka Y, Ohta H. Resuscitative endovascular balloon occlusion of the aorta for uncontrollable non-variceal upper gastrointestinal bleeding. World J Emerg Surg. 2016; 11:20.

Original Article

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Stepwise Reperfusion After Zone 1 REBOA: Is Repositioning to Zone 3 a Useful Maneuver?

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Background: One limitation of resuscitative endovascular balloon occlusion of the aorta (REBOA) is hemodynamic instability upon balloon deflation due to distal hyperemia and washout of ischemic metabolites. We sought to determine whether stepwise reperfusion after supraceliac (Zone 1) REBOA by transitioning to infrarenal (Zone 3) occlusion would mitigate the physiologic consequences of balloon deflation and decrease hemodynamic instability.
Methods: Twelve anesthetized swine underwent controlled hemorrhage of 25% blood volume, 45 minutes of Zone 1 REBOA, then resuscitation with shed blood. Standardized critical care began with deflation of the Zone 1 balloon in all animals, and continued for six hours. Half of the animals were randomly assigned to Zone 3 REBOA for an additional 55 minutes following Zone 1 balloon deflation.
Results: There were no differences in physiology at baseline, during the initial 30 minutes of hypotension, or during the 45 minutes of Zone 1 occlusion. After Zone 1 balloon deflation, there was no difference in proximal mean arterial pressure (pMAP) with or without Zone 3 occlusion or percentage of critical care time spent within the target pMAP range between 65 and 75 mm Hg. There were also no significant differences in peak lactate concentration or resuscitation requirements.

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Author contributions: EMT, GLH, AJD, JKG, MAJ, and TKW conceived and designed the study. Acquisition of data: EMT, GLH, MAS, AJD, ESD, ERF, JKG, MAJ, and TKW acquired the data. EMT, GLH, LPN, JKG, MAJ and TKW analyzed and interpreted the data. EMT, GLH, LPN, MAJ, and TKW drafted the manuscript. EMT, ERF, LPN, JKG, MAJ, and TKW undertook the critical revision.

Conflicts of interest: EMT, MAJ, and LPN are founders and stockholders of Certus Critical Care, Inc.

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Disclosure: 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. The views expressed in this material are those of the authors and do not reflect the official policy of the US Government, the Department of Defense, the Department of the Air Force, or the University of California Davis. The work reported herein was performed under United States Air Force Surgeon General approved Clinical Investigation No. FDG20160026A.

Conclusions: In an animal model of hemorrhagic shock and Zone 1 REBOA, subsequent Zone 3 aortic occlusion did not add a significant ischemic burden, but it also did not provide significant hemodynamic support. The effect of this strategy on functional outcomes warrants further study. Continued investigation is necessary to determine optimal resuscitative support strategies during reperfusion following Zone 1 REBOA.

Keywords: *REBOA; Shock; Ischemia-reperfusion*

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INTRODUCTION

Resuscitative endovascular balloon occlusion of the aorta (REBOA) is becoming an acceptable and effective intervention for hemorrhage control and hemodynamic support in the setting of non-compressible torso hemorrhage (NCTH). One limitation of REBOA is the profound hemodynamic lability that occurs upon reinstituting distal aortic flow. The hemodynamic instability following balloon deflation is a product of distal vascular vasodilation from ischemia as well as the ischemia-reperfusion injury that occurs upon reinstitution of blood flow [1–3]. This vasodilation can result in a profound shock state that may require extensive resuscitation efforts regardless of the initial injury. With the ongoing evolution of REBOA, several strategies have been described in an attempt to mitigate the ischemic burden of REBOA and minimize the subsequent ischemia-reperfusion state. Among them, intermittent REBOA (I-REBOA) [4,5], partial REBOA (P-REBOA) [6–8], and endovascular variable aortic control (EVAC) [9] have all demonstrated promise, but the applicability of each is currently limited by technologic challenges [10]. Although REBOA use continues to increase in patients with NCTH, there remains a need for a practical, effective strategy to manage the resultant reperfusion injury.

The 2018 guidelines from the American College of Surgeons Committee on Trauma do not recommend any specific steps for weaning from REBOA, except that the balloon should be deflated as soon as possible, and the patient must be monitored for at least 24 hours thereafter [11]. The Joint Trauma System Clinical Practice Guideline recommends slow deflation in conjunction with ongoing resuscitation and mentions the potential need for intermittent balloon inflation and deflation as hemodynamic stability is restored [12]. Finally, the Basic Endovascular Skills for Trauma (BEST) course recommends stepwise deflation over a 5-minute period to slowly reperfuse distal vasculature [13]. Prior translational work has demonstrated that the timing of aortic flow return upon deflation of a REBOA balloon is inconsistent and unpredictable, with a return to full flow occurring over a very small range of balloon volumes [14,15]. Furthermore, due to the distal vasodilation that occurs during complete aortic occlusion, aortic flow returns at a higher rate than initial baseline flow resulting in rapid washout of ischemic metabolites from distal vascular beds [14]. Based on the current literature, the optimal method of weaning a patient from complete aortic occlusion remains unclear, yet critical for increasing the safety and therapeutic duration of REBOA technologies.

Considering the hemodynamic compromise upon Zone 1 balloon deflation, one potential solution would be simply to reposition the balloon more distally into Zone 3 while resuscitation continues. Even though the degree of hemodynamic support provided by Zone 3 REBOA remains unclear based on the current literature, it is reasonable to infer that Zone 3 REBOA would provide some hemodynamic support, albeit less than that provided by Zone 1 REBOA. We proposed this stepwise reperfusion strategy as an immediately clinically relevant method to address the hemodynamic instability upon REBOA balloon deflation. Our objective for this study was to determine if immediately transitioning from Zone 1 REBOA to Zone 3 REBOA would have physiologic benefits compared to deflation from Zone 1 occlusion alone. We hypothesized a net benefit of hemodynamic support that would outweigh any additional ischemic burden incurred. We used a swine model to investigate this strategy, using proximal MAP following reperfusion as a primary outcome. Secondary outcomes included resuscitation requirements, serum lactate concentration, and markers of renal function.

MATERIALS AND METHODS

Overview

The Institutional Animal Care and Use Committee at David Grant Medical Center, Travis Air Force Base, California approved this study. All animal care and use were in strict compliance with the Guide for the Care and Use of Laboratory Animals in a facility accredited by the Association for the Assessment and Accreditation of Laboratory Animal Care International. Healthy adult castrate male and non-pregnant female Yorkshire-cross swine (*Sus scrofa*) from various vendors were acclimated for a minimum of seven days. Animals were housed in individual cages with 12-hour day-night cycles. They had free access to food and water until 12 hours prior to experimentation. Every experiment was supervised by an attending veterinarian. At the time of experimentation,

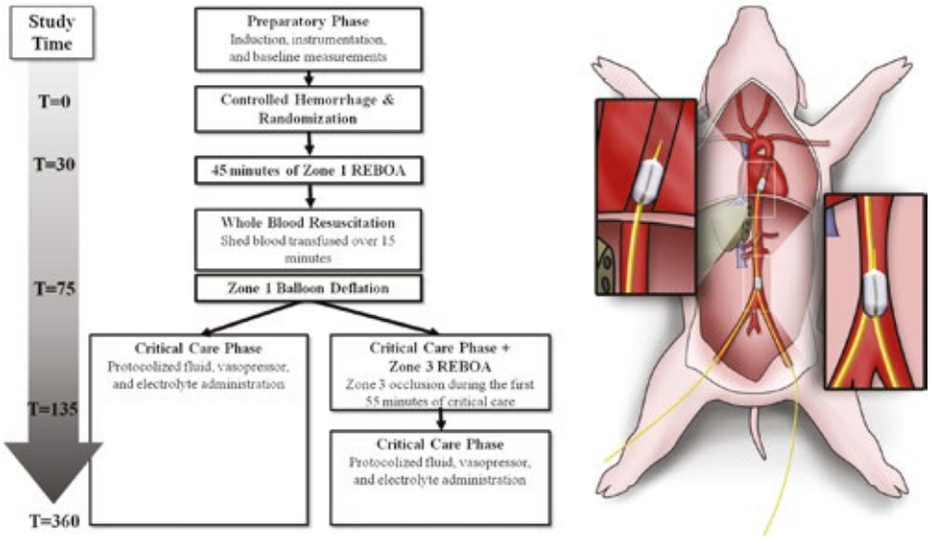

Figure 1 Experimental design.

animals weighed between 70 and 92 kg and were between 4.5 and 5.5 months of age.

Conduct of the protocol, including animal preparation, injury, intervention, and critical care, is illustrated in Figure 1. Animals were subjected to a 25% total blood volume hemorrhage over 30 minutes, followed by 45 minutes of Zone 1 REBOA. During this time, animals were assigned, using a block randomization scheme, to one of two experimental arms: standardized critical care along with an additional 55 minutes of Zone 3 REBOA following deflation of the Zone 1 balloon (Z1Z3 group, $n=6$), or standardized critical care without Zone 3 REBOA (Z1 group, $n=6$). Critical care was continued for a total experimental time of 360 minutes, during which vasopressors were titrated and isotonic fluid boluses were administered based upon predefined physiologic parameters.

Animal Preparation

Animals were premedicated with 6.6 mg/kg intramuscular tiletamine/zolazepam (Telazol, Fort Dodge Animal Health, Fort Dodge, IA). Following isoflurane induction and endotracheal intubation, general anesthesia was maintained with 2% isoflurane in 100% oxygen. To offset the vasodilatory effects of general anesthesia, an intravenous infusion of norepinephrine (0.01 µg/kg/min) was instituted upon venous access and titrated prior to experimentation to achieve a target mean arterial pressure between 65 and 75 mm Hg. Animals were mechanically ventilated with tidal volumes of 7–10 mL/kg and a respiratory rate of 10–15 breaths per minute, which was sufficient to maintain end-tidal CO_2 at 40 ± 5 mm Hg. Balanced electrolyte solution (Plasma-Lyte A, Baxter Healthcare Corporation, Deerfield, IN) was administered at a rate of 10 mL/kg/h until the abdomen was closed, at which point the rate was decreased to 5 mL/kg/h for the remainder of the study to replace insensible losses. All animals received a bolus of 1 L Plasma-lyte A upon venous access. Intravenous heparin was administered to achieve an activated clotting time (ACT) of 100 seconds, similar to human baseline values. An underbody warmer was used to maintain core body temperature between 35 and 37°C.

Following celiotomy, the spleen was removed to minimize hemodynamic variation from autotransfusion [16]. The supraceliac aorta was exposed by dividing the diaphragm and dissected circumferentially for a length of 5–10 cm. Two adjacent intercostal arteries were ligated to facilitate placement of an 11 mm periaortic flow probe (Transonic Systems Inc, Ithaca, NY). The abdomen was closed with cable ties.

Bilateral external jugular veins were cannulated (7 Fr sheath on the left, 9 Fr dual lumen catheter on the right) to facilitate medication and fluid administration, as well as for central venous pressure monitoring. The right brachial artery was exposed and cannulated with a 7 Fr sheath for controlled hemorrhage. The left axillary artery was exposed and cannulated with a 9 Fr sheath for proximal mean arterial pressure (pMAP) monitoring. The left femoral artery was exposed and cannulated with a 12 Fr sheath for Coda balloon placement (Coda, Cook Medical, Bloomington, IN) and distal mean arterial pressure (dMAP) monitoring. The right femoral artery was exposed and cannulated with a 7 Fr sheath for ER-REBOA placement (ER-REBOA, Prytime Medical, Boerne, TX). The left femoral vein was exposed and cannulated with a dual lumen resuscitation catheter for blood transfusion. The position of the Coda balloon in Zone 1, just proximal to the diaphragmatic hiatus, and the ER-REBOA balloon in Zone 3, just distal to the most distal renal artery, were guided by palpation during

celiotomy and confirmed by fluoroscopy after abdominal closure but prior to the start of experimentation.

Data Collection

Physiologic parameters and aortic flow measurements were collected in real time using a multichannel data acquisition system (MP150, Biopac Systems Inc., Goleta, CA). Measured parameters included heart rate, blood pressure proximal and distal to the intraaortic balloons, central venous pressure, core temperature, and aortic blood flow. Arterial blood was collected at routine intervals throughout the study for blood gas analysis, basic metabolic profile, and blood counts. Urine was collected and quantified at similar intervals.

Hemorrhage, Intervention, and Critical Care

At the start of the experiment, animals underwent controlled hemorrhage of 25% of their estimated total blood volume. Blood volume was estimated at 60 mL per kilogram of body weight. Blood was withdrawn over 30 minutes into citrated blood collection bags. During this time, animals were randomized.

At the end of 30 minutes, the Zone 1 balloon was inflated in all animals. Complete aortic occlusion was confirmed by loss of aortic flow. Occlusion in Zone 1 was maintained for 45 minutes. Transfusion of shed blood began 10 minutes prior to deflation of the Zone 1 balloon. The volume of shed blood was transfused over 30 minutes with a rapid infuser (Belmont Instrument Corporation, Billerica, MA). The Zone 1 balloon was deflated over 5 minutes, starting at T75. At T80, critical care commenced for all animals. The animals randomized to the Z1Z3 group immediately underwent inflation of the Zone 3 balloon. Complete aortic occlusion was again confirmed by the loss of the distal arterial pressure waveform. This balloon remained inflated in Zone 3 for an additional 55 minutes. It was deflated over a 5-minute interval starting at T135. This duration of Zone 3 occlusion was chosen based upon previous experience in our lab as the amount of time that would provide support during the most tenuous period in resuscitation after Zone 1 reperfusion, but would not produce a burden of ischemia from which the animal could not recover.

For all study animals, critical care with isotonic fluid boluses, vasopressor titration, and electrolyte correction proceeded from T80 to T360. Boluses of 500 mL Plasmalyte A were administered for pMAP less than 60 mm Hg with CVP less than 7 mm Hg. The norepinephrine infusion rate was increased by 0.02 mcg/kg/min for pMAP less than 60 with CVP greater than or equal to 7 mm Hg. The norepinephrine infusion rate was decreased by 0.01 mg/kg/min when the pMAP exceeded 70 mm Hg. These interventions were administered automatically in a closed loop algorithm by a custom automated syringe pump for norepinephrine and a Masterflex peristaltic pump (Cole-Parmer, Vernon Hills, IL) for fluid boluses. Serum potassium concentrations greater than 6.0 mmol/L were corrected with insulin and dextrose. Serum glucose concentrations less than 60 mg/dL were corrected with dextrose boluses and continuous infusions. Serum calcium concentrations less than 1.00 mmol/L were corrected with calcium gluconate. Animals were euthanized at T360 without recovering from general anesthesia.

Data Analysis

Experimental data were entered into Excel datasheets (Microsoft Corporation) and transferred to STATA version 14.0 (Stata Corporation, Bryan, TX) for analysis. Continuous variables are presented as means and standard errors of the means (SEMs) if normally distributed, and as medians with interquartile ranges if not normally distributed. Between groups, comparisons were conducted with a *t*-test or Mann–Whitney *U*-test, as appropriate. Statistical significance was set at p less than 0.05. The sample size was determined using an a priori power calculation using G*Power (Heinrich-Heine Universität Düsseldorf, Germany) to detect a difference in MAP of 15 mm Hg with a standard deviation of 9 mm Hg, a power of 80%m and a statistical significance if $p < 0.05$.

RESULTS

There were no differences in baseline hemodynamics or laboratory analysis between groups, except that animals in the Z1Z3 group had a higher starting creatinine (1.7 mg/dL 95% confidence interval (CI) 1.6–1.8 versus 1.3 mg/dl 95% CI 1.2–1.5) (Table 1). After the initial hemorrhage period both groups had a similar minimum pMAP (Z1 32 mm Hg 95% CI 29–36 versus Z1Z3 30 mm Hg 95% CI 23–36) and had similar average pMAP during the initial Zone 1 intervention phase (Z1 129 mm Hg 95% CI 106–152 versus Z1Z3 118 mm Hg 95% CI 103–133) (Figure 2).

During the Zone 3 phase there were no differences in proximal MAP (Z1 57 mm Hg 95% CI 50–64 versus Z1Z3 61 mm Hg 95% CI 56–65), aortic flow (Z1 3,984 mL/min 95% CI 3,247–4,721 versus Z1Z3 3,362 mL/min 95% CI 2,961–3,762), need for IV crystalloids (Z1 1,633 mL 95% CI 1,272–1,995 versus Z1Z3 1,150 mL 95% CI 543–1,757), norepinephrine dose (Z1 7 µg /kg 95% CI 4–10 versus Z1Z3 4 µg/kg 95% CI 1–7), or urine output (Z1 237 mL 95% CI 91–382 versus Z1Z3 265 mL 95% CI 222–307) between groups (Table 2).

By the end of the study there were no differences in final lactate (Z1 5.2 mmol/L 95% CI 3.7–6.8 versus Z1Z3 4.4 mmol/L 95% CI 3.1–5.7), although the creatinine was higher in the Z1Z3 group when compared to the Z1 group (2.3 mg/dl 95% CI 1.9–2.6 versus 1.7 mg/dl 95% CI 1.4-2.0). However, there was no difference in the change in creatinine from baseline to the end of

Table 1 Baseline physiology, labs, and hemodynamic characteristics.

	Z1 (n = 6)	*Z1Z3 (n = 6)*	*p-Value*
Weight kg	78 (69–85)	79 (71–86)	0.76
Sex male:female	1:5	3:3	
Temperature (°C)	35.8 (35.1–36.5)	35.2 (34.7–35.7)	0.11
Labs			
pH	7.43 (7.40–7.47)	7.42 (7.40–7.45)	0.42
P:F ratio	372 (272–473)	381 (299–463)	0.87
Hemoglobin (g/dL)	10.3 (9.5–11.1)	10.7 (9.8–11.6)	0.42
White blood cells (× 10^9/L)	15.2 (12.0–18.4)	15.4 (13.8–17.0)	0.89
Platelets (× 10^9/L)	275 (228–323)	273 (170–375)	0.95
Potassium (mmol/L)	3.7 (3.5–3.9)	3.7 (3.5–3.9)	0.78
Creatinine (mg/dL)	1.3 (1.2–1.5)	1.7 (1.6–1.8)	<0.01
Lactate (mmol/L)	2.4 (2.0–2.9)	2.5 (2.2–2.8)	0.82
Glucose (mg/dL)	93 (84–101)	85 (75–96)	0.18
Hemodynamics			
Proximal MAP (mm Hg)	66 (60–73)	69 (63–74)	0.51
Aortic flow (mL/min)	2,959 (2,492–3,425)	3,027 (2,697–3,359)	0.76

Error presented as standard error of the mean.

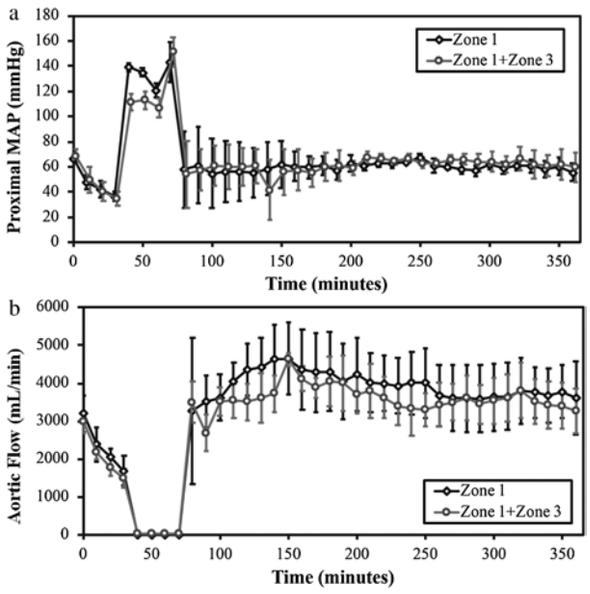

Figure 2 Comparison of mean arterial blood pressure (**a**) and mean aortic flow (**b**) over the time of the entire study between the groups with and without Zone 3 REBOA during the first hour of critical care (T80-T135) following Zone 1 balloon deflation. Neither pressure nor flow was significantly different between the groups at any point during the course of the experiment. Data are presented as the mean and standard error of the mean. $n = 6$ per group.

study between groups (Z1 0.4 mg/dl 95% CI 0.1–0.7 versus Z1Z3 0.6 mg/dl 95% CI 0.3–0.9). There was also no difference in final urine output between the two groups across the timeline of the study (Z1 3,057 mL 95% CI 1,727–4,384 versus Z1Z3 1,872 mL 95% CI 863–2,881, $p = 0.10$).

DISCUSSION

The physiologic consequences of reperfusion after Zone 1 REBOA are a significant barrier to increasing its accepted therapeutic duration and widespread adoption. Balloon deflation results in rapid washout of ischemic metabolites as well as the initiation of reperfusion injury. This combined physiologic insult leads to significant hemodynamic instability. To counter this, we tested an alternate method of REBOA weaning by repositioning the REBOA catheter in the distal aorta after initial Zone 1 occlusion. In this study, our data showed no difference in MAP with or without Zone 3 occlusion, therefore we concluded that this repositioning strategy does not provide any further support to the vital organs when compared to a standardized resuscitation with intravenous crystalloids and vasopressors for this particular pathologic state. We have also demonstrated that Zone 3 occlusion below the level of the renal arteries does not increase the overall ischemic burden, but does result in a trend toward decreased urine output following deflation of a Zone 1 balloon. While the decrease in urine output did not reach statistical significance, it may be clinically significant and is an area of ongoing investigation. Taking these findings into consideration, Zone 3 occlusion does not appear to be a helpful adjunct following prolonged Zone 1 occlusion to counter hemodynamic depression or for the treatment of the ischemia-reperfusion injury.

Current REBOA instructional programs suggest balloon deflation should be performed in a stepwise manner over a 5-minute period to allow for a slow reintroduction of blood flow to distal vascular beds. This time period will allow the vascular beds proximal to the point of occlusion to compensate for the decreased perfusion that can result from the reintroduction of blood flow

Table 2 Physiologic, laboratory, and hemodynamic outcomes.

	Z1 (n = 6)	*Z1Z3 (n = 6)*	*p-Value*
Resuscitation totals			
Crystalloid (mL)	7,400 (6,148–8,652)	5,700 (2,462–8,938)	0.24
Norepinephrine (µg/kg)	50 (38–62)	45 (17–73)	0.67
Urine output (mL/kg)	3,057 (1,727–4,384)	1,872 (863–2,881)	0.10
Resuscitation during T80–T135			
Crystalloid (mL)	1,633 (1,272–1995)	1,150 (543–1,757)	0.11
Norepinephrine (µg/kg)	7 (4–10)	4 (1–7)	0.14
Urine output (mL/kg)	237 (91–382)	265 (222–307)	0.64
Labs			
Peak lactate (mmol/L)	9.6 (8.5–10.7)	10.6 (9.5–11.7)	0.12
Final lactate (mmol/L)	5.24 (3.7–6.8)	4.4 (3.1–5.7)	0.30
Final creatinine (mg/dL)	1.7 (1.4–2.0)	2.3 (1.9–2.6)	<0.01
Delta creatinine (mg/dL)	0.4 (0.1–0.7)	0.6 (–.3–0.9)	0.19
Hemodynamics			
pMAP nadir during hemorrhage (mm Hg)	32 (29–36)	30 (23–36)	0.29
pMAP average during Zone 1 REBOA (mm Hg)	129 (106–152)	118 (104–133)	0.34
pMAP average T80–T135 (mm Hg)	57 (50–64)	61 (56–65)	0.25
pMAP average during critical care (mm Hg)	60 (57–63)	61 (56–66)	0.59
Aortic flow average T80–T135 (mL/min)	3,984 (3,247–4,721)	3,362 (2,961–3,762)	0.09
Aortic flow average during critical care (mL/min)	3,960 (3,176–4,743)	3,604 (3,160–4,048)	0.33

T80–T35 represents the first 55 minutes of the critical care phase, during which the Zone 3 balloon was inflated in the Z1Z3 group. pMAP, proximal mean arterial pressure. Error presented as standard error of the mean.

into a dilated distal vasculature that can occur following even short periods of complete Zone 1 aortic occlusion. Prior work has demonstrated that the reintroduction of distal flow likely occurs in an unpredictable fashion, and that aortic flow after a period of occlusion is hyperemic, with rates over twice the baseline aortic flow rate [14,15]. This can result in profound hemodynamic instability. Recent data from translational animal models have shown that Zone 3 REBOA alone provides a modest degree of proximal hemodynamic support in the setting of hemorrhagic shock, but not enough to rescue a patient on the brink of cardiovascular collapse [17]. In this current study, the Zone 3 occlusion was implemented not as a resuscitative maneuver during hemorrhage, but as a weaning adjunct after resuscitation and deflation of a Zone 1 occlusion balloon. Therefore, this was essentially a Zone 3 occlusive intervention during an ischemia-reperfusion shock state. When comparing the earlier studies with Zone 3 occlusion during hemorrhagic shock, it seems that the lack of significant hemodynamic support by the Zone 3 balloon in this study may be explained by the difference in the pathophysiology of the shock state in these animals [18–21]. Instead of hypovolemia with the possibility of vasoconstriction in vascular beds above the Zone 3 occlusion, these animals had a combination of profound vasodilation from 45 minutes of ischemia combined with the inflammatory mediators released during the reperfusion when the Zone 1 occlusion ended. Prior work has demonstrated that circulating cytokines following reperfusion can lead to systemic vasodilation, inflammatory state, and multi-organ dysfunction [2]. The synergistic hit of maximal vasodilation and release of the ischemic metabolites may be too great for Zone 3 REBOA to be of benefit. Further work is warranted to specifically isolate and study the effects of REBOA and subsequent weaning on cardiac performance.

The most recent Joint Trauma System Clinical Practice Guideline advises no longer than 30–60 minutes of REBOA in Zone 1, but the recommendations for Zone 3 are less specific [12]. In our study, we expected to observe a second hemodynamic insult upon reperfusion after Zone 3 REBOA, but we did not see one that reached statistical significance. The equivalence of final lactate concentrations between the two groups also suggests that any "second hit" was likely to be of no significant consequence to the animal. The animals in our Z1Z3 experimental arm underwent a total of 100 minutes of hindlimb ischemia. The lack of a second hit in terms of physiologic complications suggests that longer occlusion times are possible with Zone 3 REBOA, and further study is warranted to guide maximum duration recommendations for REBOA in Zone 3.

One unexpected finding of this experiment was the trend toward lower urine output in the Z1Z3 group. Our expectation was that diverting blood flow from the hindlimbs to the abdominal viscera with Zone 3 occlusion would improve perfusion of abdominal vascular organs, even in the absence of systemic hemodynamic augmentation. We did find a significant difference in final creatinine, but this was accounted for by a baseline difference in creatinine between the two groups. Specifically, when the change in creatinine over the entire experimental

duration was calculated, there was no difference between groups. There was a trend toward oliguria in the animals randomized to the Z1Z3 group which was surprising and is not yet fully elucidated. However, this trend has been noted in canine studies of Zone 3 occlusion that demonstrated decreased renal blood flow [22,23]. A potential next step will be to determine if the visceral organs did, in fact, receive more flow during the Zone 3 occlusion period. Future analysis will be aimed at understanding the relationship of these trends to renal blood flow.

There are several limitations to this study. First, it was impossible to blind the investigators to the study arm assigned to each animal and there is always the possibility of investigator bias in a non-blinded study. However, with full automation of balloon inflation and deflation, as well as delivery of resuscitative interventions, we attempted to minimize treatment variability among subjects. Another limitation of this study was the small sample size. We did observe some trends in data that did not elaborate into significant differences between the groups, but that may indeed become statistically significant at larger sample sizes. It is unclear based upon this study whether these would amount to clinical significance. Additionally, this study's relatively short time period after initial resuscitation prevented fuller understanding of longer-term benefits or consequences that may have manifested later in a survival study. Also, without the utilization of proteomics or advanced biomarker analysis, it is possible that there were unrecognized physiologic phenomena underlying the trends that we demonstrated. It is unclear from this study if alternative methods of transitioning from Zone 1 to Zone 3 could provide benefit. For this study, we allowed a 5-minute period between Zone 1 deflation and Zone 3 inflation to adhere to the BEST course guidelines. It is possible that the immediate washout of the additional ischemic metabolites in the hindlimbs was enough to cause sufficient instability in the animal such that Zone 3 REBOA was not effective. It is possible that a two-balloon method with slower deflation of Zone 1 while Zone 3 is already inflated would provide a more hemodynamically stable transition, although this maneuver may prove to be too complex with current technology to be clinically applicable. Along the same lines, it is possible that the partial obstruction of the bilateral femoral arteries with the sheaths that we used may have contributed to the ischemic burden in the hindlimbs. We attempted to control for this with uniform sheath placement among all animals regardless of the experimental arm, but different results may have been seen if a different model had been used. Another potential limitation is in the design of our resuscitation paradigm, as it was based upon CVP and MAP. Since there is currently no consensus regarding the best estimator of volume status, we chose CVP due to its ease of measurement and clinical applicability. There is certainly room for future investigation using other resuscitation parameters, which may yield different results. These limitations notwithstanding, this study does provide quantifiable data regarding the relative contribution of an alternate weaning method that is possible with current REBOA technology.

CONCLUSION

In our swine model of ischemia-reperfusion injury induced by hemorrhagic shock with Zone 1 REBOA, the subsequent transition to Zone 3 aortic occlusion as a method of weaning did not provide increased hemodynamic stability but also did not add a significant ischemic burden. Continued investigation is necessary to develop optimal support strategies to wean from Zone 1 REBOA and minimize hemodynamic instability.

REFERENCES

[1] Markov NP, Percival TJ, Morrison JJ, et al. Physiologic tolerance of descending thoracic aortic balloon occlusion in a swine model of hemorrhagic shock. Surgery. 2013;153:848–56.

[2] Morrison JJ, Ross JD, Markov NP, Scott DJ, Spencer JR, Rasmussen TE. The inflammatory sequelae of aortic balloon occlusion in hemorrhagic shock. J Surg Res. 2014; 191:423–31.

[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: 1869–72.

[4] Ogura T, Lefor AT, Nakano M, Izawa Y, Morita H. Nonoperative management of hemodynamically unstable abdominal trauma patients with angioembolization and resuscitative endovascular balloon occlusion of the aorta. J Trauma Acute Care Surg. 2015;78:132–5.

[5] Saito N, Matsumoto H, Yagi T, et al. Evaluation of the safety and feasibility of resuscitative endovascular balloon occlusion of the aorta. J Trauma Acute Care Surg. 2015;78:897–903.

[6] Johnson MA, Neff LP, Williams TK, DuBose JJ. Partial Resuscitative Balloon Occlusion of the AORTA (P-REBOA): Clinical Technique and Rationale. J Trauma Acute Care Surg. 2016;81:S133–7.

[7] Russo RM, Neff LP, Lamb CM, et al. Partial Resuscitative Endovascular Balloon Occlusion of the Aorta in Swine Model of Hemorrhagic Shock. J Am Coll Surg. 2016;223(2):359–68.

[8] Russo RM, Williams TK, Grayson JK, et al. Extending the golden hour: Partial resuscitative endovascular balloon occlusion of the aorta in a highly lethal swine liver injury model. J Trauma Acute Care Surg. 2016;80:372–8.

[9] Williams TK, Neff LP, Johnson MA, et al. Extending REBOA: Endovascular variable aortic control (EVAC) in a lethal model of hemorrhagic shock. J Trauma Acute Care Surg. 2016;81:294–301.

[10] Russo R, Neff LP, Johnson MA, Williams TK. Emerging Endovascular Therapies for Non-Compressible Torso Hemorrhage. Shock (Augusta, Ga). 2016;46:12–19.

[11] Brenner M, Bulger EM, Perina DG, et al. Joint statement from the American College of Surgeons Committee on

Trauma (ACS COT) and the American College of Emergency Physicians (ACEP) regarding the clinical use of Resuscitative Endovascular Balloon Occlusion of the Aorta (REBOA). Trauma Surgery; Acute Care Open. 2018;3(1).
[12] Pasley J, Cannon J, Glaser J, et al. Joint Trauma System Clinical Practice Guideline: Resuscitative Endovascular Balloon Occlusion of the Aorta (REBOA) for Hemorrhagic Shock. Department of Defense. 2017; http://www.usaisr.amedd.army.mil/cpgs/REBOA_%2006Jul2017 CORRECTED.pdf (accessed Jan 2018).
[13] Brenner M, Hoehn M, Teeter W, Stein D, Scalea T. Trading scalpels for sheaths: Catheter-based treatment of vascular injury can be effectively performed by acute care surgeons trained in endovascular techniques. J Trauma Acute Care Surg. 2016;80:783–6.
[14] Davidson AJ, Russo RM, Ferencz SE, et al. 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;83:139–43.
[15] Johnson MA, Davidson AJ, Russo RM, et al. 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;82:1106–11.
[16] Brenner M, Hoehn M, Pasley J, Dubose J, Stein D, Scalea T. Basic endovascular skills for trauma course: bridging the gap between endovascular techniques and the acute care surgeon. J Trauma Acute Care Surg. 2014;77:286–91.
[17] Moore LJ, Brenner M, Kozar RA, et al. Implementation of resuscitative endovascular balloon occlusion of the aorta as an alternative to resuscitative thoracotomy for noncompressible truncal hemorrhage. J Trauma Acute Care Surg. 2015;79:523–30.
[18] Sondeen JL, Dubick MA, Holcomb JB, Wade CE. Uncontrolled hemorrhage differs from volume- or pressure-matched controlled hemorrhage in swine. Shock (Augusta, Ga). 2007;28:426–33.
[19] Tibbits EM, Hoareau GL, Simon MA, et al. Location is everything: The hemodynamic effects of REBOA in Zone 1 versus Zone 3 of the aorta. J Trauma Acute Care Surg. 2018; 85:101–7.
[20] Martinelli T, Thony F, Declety P, et al. Intra-aortic balloon occlusion to salvage patients with life-threatening hemorrhage shocks from pelvic fractures. J Trauma. 2010;68:942–8.
[21] Morrison JJ, Percival TJ, Markov NP, et al. Aortic balloon occlusion is effective in controlling pelvic hemorrhage. J Surg Res. 2012;177:341–7.
[22] Abbot WM, Cooper JD, Austen WG. The effect of aortic clamping and declamping on renal blood flow distribution. J Surg Res. 1973;14:385–92.
[23] Abbot WM, Austen WG. The reversal of renal cortical ischemia during aortic occlusion by mannitol. J Surg Res. 1974;16:482–9.

Narrative Review Article

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Novel Interventions for Non-Compressible Torso Hemorrhage: Secondary Considerations in Consequences of Aortic Occlusion

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Non-compressible torso hemorrhage in trauma remains a leading cause of death in austere environments. Advancements for treatment include resuscitative endovascular balloon occlusion of the aorta (REBOA), selective aortic arch perfusion (SAAP), and external compression approaches (junctional tourniquets and abdominal aortic tourniquets), which have provided several promising avenues. However, the application of these devices carries the risk of distal ischemia and the consequences associated with reperfusion injury. This review aims to look at these novel interventions and the physiologic burden associated with them. Following a review of these new advents, we will evaluate the possible solutions to reverse the physiologic penalties.

Keywords: *Non-Compressible Torso Hemorrhage; REBOA; Selective Aortic Arch Perfusion; Junctional Tourniquets; Cytosorbants*

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INTRODUCTION

Hemorrhage is the leading cause of preventable death in pre-hospital combat casualty care and civilian trauma [1,2]. The majority of potentially survivable deaths involve hemorrhages in the torso, which are difficult to control in the field [3]. There have been several promising advancements in modalities for management, including resuscitation with balanced blood products, aortic occlusion [e.g. resuscitative endovascular balloon occlusion of the aorta (REBOA) and selective aortic arch perfusion (SAAP)], and abdominal aortic/junctional tourniquets. Interventions involving compression or aortic occlusion give a short-term survival advantage by offering continued perfusion to vital organs; however, the associated morbidity of ischemia distal to the occlusion can result in irreversible injury and potentially exacerbate trauma induced systemic inflammatory response syndrome (SIRS) [4–7].

With advancements in endovascular techniques for aortic occlusion in trauma, the physiologic sequelae are still poorly understood. There is both translational evidence and clinical outcomes, which indicate that our focus must shift to the management of these outcomes. Multiple accounts of distal ischemia and secondary reperfusion injury following recirculation have been reported following occlusion [6]. Ischemia-reperfusion injury (IRI) develops through several signaling pathways involving ischemic-induced cell injury, impaired intracellular calcium hemostasis, depletion of ATP, the formation of toxic metabolites and production of free radicals leading to further oxidative damage [8]. It can also cause a subsequent inflammatory response resulting from the induction of multiple cytokines and chemokines

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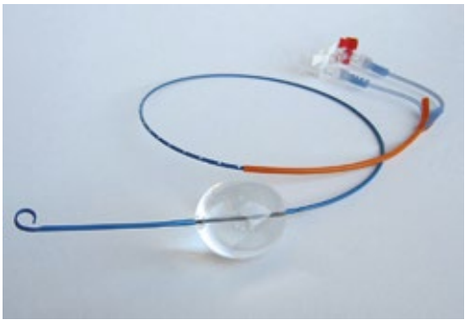

Figure 1 ER-REBOA™ catheter (reprinted with permission from Prytime Medical™).

with worsening of oxidative stress [9]. This, in turn, leads to complications in regulating vascular barrier properties, control of adhesion and extra-vascular trafficking of immune cells, regulation of vascular tone, and control of hemostatic mechanisms [10].

The purpose of this review is to discuss and compare the different modalities of treatment for non-compressible hemorrhage in the context of therapeutic window and technique, physiologic impact, and logistical constraints. In particular, we will discuss the implications of these therapies and the consequences associated with IRI. Finally, we will examine therapeutic targets for managing the physiologic dyshomeostasis or "physiologic penalty" that can be incurred by aortic occlusion during non-compressible hemorrhage control.

REBOA

Despite improvements in resuscitation, non-compressible torso hemorrhage is challenging to manage without control of the bleeding source. Different techniques have been proposed recently including REBOA (Figure 1) [11]. REBOA is an endovascular technique of achieving distal control to treat NCTH that has resulted in near cardiovascular collapse [11,12]. The hopes of this technique are to reduce mortality, along with reperfusion injury, in control of non-compressible torso hemorrhage [13].

Reports of the use of balloon occlusion of the aorta were seen as early as the Korean War [11]. In 1954, Hughes et al. described the use of a novel intra-aortic balloon catheter for the use of moribund patients with uncontrolled hemorrhage (>10 U of blood administered) in the hopes of enhancing perfusion of the coronary arteries [12]. In 1986, Low et al. had demonstrated its use again with a death due to ischemia in one patient and difficulty with placement in others [14]. The application of REBOA over the following years lagged due to technology, lack of skill set for placement, and anticipated ineffectiveness [11,15]. With the recent evolution of technology and endovascular advancements, there has been an increased use and push for further research [11,15].

The current technique of placement is by deploying a balloon occlusion catheter into the aorta via the common femoral artery (CFA) [11]. The CFA is palpated, identified via ultrasound or found under direct vision by cutdown [15]. The artery is then accessed using a sheath which can then be upsized over a wire for use with larger balloon catheters. Following access, the insertion depth can be approximated via external landmarks, x-ray confirmation or under fluoroscopy [11,15,16]. The balloon catheter is then inserted into the pre-determined level and inflated until aortic occlusion is achieved [17].

The aorta is divided into three zones as described by Stannard et al. [11]. Zone 1 is an occlusion site denoted between the left subclavian artery to the celiac artery. Zone 1 is above the xiphoid process. Zone 2 is between the celiac artery and the lowest renal artery and is known as the no-occlusion zone. Zone 3 is designated as the area below the lowest renal artery to the aortic bifurcation and the anatomical landmark for the bifurcation is the umbilicus [11,15,17].

Aortic occlusion and cross clamping in the settings of abdominal exsanguination are usually pursued in the setting of near cardiovascular collapse [15]. The emergent nature of this procedure makes balloon positioning and confirmation more difficult. Given the acuity of the situation, it may not be feasible for positioning confirmation via fluoroscopy, as these may not be available. The majority of Level I trauma centers confirm placement via plain film (52%), followed by blind insertion (26%), and fluoroscopy (13%) [18]. Current techniques for confirmation of placement require imaging that is not readily available in an austere environment, however, promising results of fluoroscopy free deployment of REBOA has been reported by special operations medical forces in recent combat operations [19].

Fluoroscopy free placement of REBOA catheters using anatomical landmarks, however, may be associated with potentially harmful complications ranging from balloon malposition to aortic rupture. Placement proximal to Zone I could theoretically lead to ischemic stroke or dangerously elevated cardiac afterload [20]. Placement in Zone II can lead to occlusion of the celiac trunk, superior mesenteric artery, or inferior mesenteric artery leading to visceral ischemia [20]. Placement distal to Zone III can occur in the iliac artery; injury to which can cause severe pelvic or junctional hemorrhage [20]. Misplacement in a groin access site can cause dissection or ischemia to the lower extremity with the risk of amputation [4]. Scott et al. have reported inadvertent placement in the renal artery in animal studies involving REBOA [21].

While fluoroscopy free deployment represents a technical/mechanical risk factor for REBOA use, there are additional physiological penalties to consider [6,7]. The decision to perform aortic occlusion requires a balance of the need to support pressure and the physiologic consequences of occlusion. Kralovich et al., for example, reported ventricular strain and impaired function

following balloon occlusion in swine [22]. Another study by White et al. demonstrated lactic acidosis and elevated serum lactate levels in swine during aortic occlusion [17]. However, in comparison to resuscitative thoracotomy, lactate levels are decreased and there is less requirement for fluids during resuscitation [17].

Although REBOA appears to have less metabolic acidosis than aortic cross clamping, there are still physiologic consequences associated with placement and duration of occlusion appears to be a factor in the amount of tissue damage [6]. Markov et al. found that in comparison to control animals, REBOA groups had greater increases in serum lactate after balloon deflation subsequent to visceral and lower extremity reperfusion [7]. In another example, Annecke et al. demonstrated remote pulmonary injury following lower body ischemia and reperfusion [25]. Additionally, in retrospective studies, there have been reports of higher hospital mortality with REBOA use in Zone 1 following occlusion times greater than 30 minutes, lower extremity ischemia, and acute kidney injury [4,26].

Markov et al. and several others have demonstrated a negative physiologic effect with increasing periods of occlusion, especially when greater than 40–60 minutes [7,25,26]. Although duration of aortic occlusion resulting in negative physiologic sequelae have not been easily extrapolated from the current literature due to ischemic burden present from hemorrhagic shock, there is a consensus that increases in occlusion time increases adverse effects [28]. Complications that have resulted following prolonged occlusion include irreversible organ injury, supraphysiologic increases in blood pressure causing cardiac failure, worsening traumatic brain injury, and increased mortality [23,24,25]. Lastly, Morrison et al. demonstrated that greater occlusion times were associated with increased release of interleukin (IL)-6, increased acute respiratory distress syndrome (ARDS), and increased lactate burden [6].

Several studies have proposed partial balloon deflation in order to mitigate these physiologic consequences and reperfusion injury associated with increasing occlusion times [27,29,30]. Partial REBOA (P-REBOA) is a continuous, low volume, distal perfusion through partial aortic occlusion [30]. Russo et al. demonstrated that P-REBOA was able to maintain proximal mean arterial pressure (MAP) at normal physiologic levels, avoid hemodynamic extremes, and continue distal perfusion to minimize ischemia and subsequent IRI when compared to complete occlusion [30].

By maintaining even a small amount of perfusion distally, there is the potential to reduce rates of tissue ischemia and rebound hypotension after balloon deflation, which can result in decreased morbidity and mortality [29]. It also has the potential to decrease the effects of supraphysiologic elevations in systolic blood pressure and increased afterload [29]. Not only can partial REBOA be a promising solution to these sequelae, but we can continue to mitigate secondary insults by properly defined strategies in the use of aortic occlusion by limiting overall occlusion times, monitoring responses to ongoing blood transfusions, and prompt hemorrhage control in the operating room. Overcoming the limitations associated with complete aortic occlusion could further impact management of non-compressible torso hemorrhage.

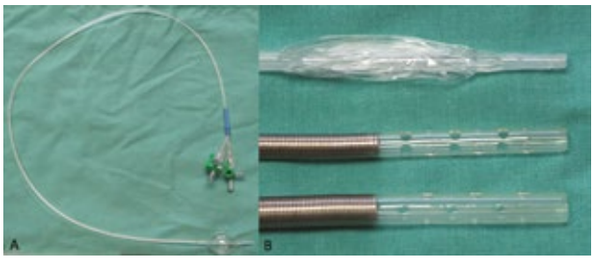

Figure 2 Example of 87 cm selective aortic arch perfusion catheter prototype (**a**) and 11.5 Fr balloon catheter tip compared to extracorporeal membrane oxygenation catheters (**b**).

SAAP

SAAP is a resuscitation technique for medical and traumatic cardiac arrest [32]. SAAP is a catheter that is placed into the descending thoracic aorta that is used to provide perfusion to the heart and brain with an oxygen-carrying perfusate while also limiting further subdiaphragmatic hemorrhage (Figure 2) [33]. Theoretically, this could reverse myocardial ischemia and acidosis, increase myocardial contractility, restore arterial vasomotor tone, and possibly promote the return of spontaneous circulation (ROSC) [32]. The benefit over REBOA would be in those patients with lost intrinsic circulation.

The technique itself is similar to REBOA, in which a large lumen balloon catheter is inserted through the femoral artery into the high thoracic aorta just distal to the left subclavian artery takeoff [32]. The balloon is then inflated preventing distal flow and further hemorrhage. An oxygenated perfusate is then infused into the isolated aortic arch, thus increasing flow to the heart and brain. Different types of perfusate have been tested such as red blood cells (RBCs), fluorocarbon and hemoglobin-based oxygen carriers [34].

As SAAP takes over perfusion to the heart and brain, the blood products transfused through the catheter can cause profound ionized hypocalcemia and ventricular dysrhythmias [34]. Manning et al. demonstrated co-administration of intra-aortic calcium appeared to counteract this complication and allowed for successful ROSC [34]. Blood is presently the only feasible product available for use at this time and would need continuous infusion of calcium during SAAP.

Prior SAAP studies have used other products such as perfluorocarbons and hemoglobin-based oxygen carriers that currently are not approved for use [34]. These perfusates are hypothesized to increase oxygen delivery

to tissues and possibly decrease oxidative stress. This resuscitation fluid would be helpful in the critical management of hypovolemia while simultaneously reducing or preventing end-organ injury due to IRI [33].

Other proposed perfusate additives can include the addition of epinephrine. Limitations are noted on the total infusion volume, rate, and total infusion that can be used [32]. Manning et al. demonstrated that with normal saline in swine studies, despite using substantial flow rates, mid-aortic arch pressures did not reach normal physiologic ranges [32]. With the addition of epinephrine, however, the mid-aortic and coronary perfusion pressures increased well into the normal physiologic range [32].

The temperature of the perfusate, as well, remains unaddressed. A relatively hypothermic solution may be beneficial for facilitating neurologic recovery but may make defibrillation more difficult [32]. Further studies need to be undertaken to establish the best temperature for the perfusate.

Multiple complications have been associated with this technique with some similarities to REBOA. These include pulmonary edema, cerebral edema, aortic dissection, aortic rupture, catheter misplacement, air embolism, femoral artery injury/thrombosis, and wound infection or hematoma [32]. Current research is still ongoing regarding the physiologic consequences of SAAP. These consequences will also need to be compared to those who are revived after cardiac arrest without the use of SAAP, as arrest alone has similar effects on ischemia-reperfusion. At this time, we can only infer that it will have similar complications to REBOA, along with sequelae of distal ischemia and possible reperfusion injury in prolonged occlusion.

External Compression Approaches

Junctional trauma is defined as an injury occurring at the junction of two anatomically distinct zones. Junctional hemorrhage cannot be controlled by standard limb tourniquets, creating an area of uncompressible hemorrhage [35]. To attempt to control this in the field, there have been advancements in specialized tourniquets capable of controlling these areas, including the abdominal aortic and junctional tourniquet (AAJT).

This tourniquet has been suitable in explosive device situations where there is a high level of leg amputation, urogenital, and pelvic injuries [35]. Its clearance has also been expanded for junctional use at the groin level [35]. Application of the tourniquet is aimed at compression of the groin or pelvic area, particularly the iliac vessels. The belt is placed around the lower abdomen and pneumatic bladder is inflated to 300 mmHg. Theoretically, it is able to compress the abdominal wall enough to occlude the underlying abdominal aorta and vena cava [35].

There have been several studies in both human and swine populations demonstrating the effectiveness of AAJT at reducing peripheral blood flow [35,36,37]. Several case reports have also demonstrated its use, including application during an en-route phase of care to control severe hemorrhage in a casualty with traumatic bilateral amputation of the lower extremities [38]. Following application and resuscitation, the patient who was previously in extremis upon arrival, had improvement of end-tidal CO_2, return of a carotid pulse, and survived through surgical intervention following transport [38]. Thus, the AAJT may be a promising intervention in combat trauma during situations of prolonged field care and temporary control of NCTH.

The hemodynamic responses seen with application show a significant increase in blood pressure and a significant reduction in circulatory volume with an increase in peripheral vascular resistance [35]. A notable tachycardia also occurred following application, which may be attributed to the discomfort or pain that is caused by application [35]. This aspect of painful application was also noted on human test subjects [36]. The pressure applied in order to cease blood flow caused moderate pain in the subject, however, pain scores returned to zero upon device deflation [36].

Kheirabadi et al., however, observed several concerning aspects in the application of AAJT including respiratory arrest upon release of the tourniquet, sudden cardiac arrest after reflow, sudden hyperkalemia, elevations in lactate, and metabolic acidosis [35]. Their group also demonstrated marked increases in creatine kinase (CK). Of note, there were no observations of acute renal failure and disseminated intravascular coagulation (DIC), despite a similarity to some of the physiologic changes in crush syndrome. In addition, these complications were not seen in studies where application time was decreased [35].

Given the possible complications associated with AAJT placement, there may be unforeseen and even detrimental consequences during release due to the distal ischemia as seen with other methods of occlusion. Further studies need to be undertaken in swine and human populations in regards to the physiologic effects of AAJT placement and release.

SIRS, IRI, and Future Metabolic Targets

The SIRS response in trauma and burns shares similar features with sepsis but may have mechanisms more intimately tied to tissue oxygenation and perfusion. Severe trauma and sepsis were found to be associated with increased inflammation and also compromised immune systems [39]. Surgery and trauma cause selective suppression of the T helper (Th)-1 lymphocytes with a shift toward Th-2 cytokine patterns for immune suppression, which differs from the immune response associated with sepsis [40]. This increased inflammation is evidenced by elevated IL-6, neutrophilia, increased immature granulocyte counts, anemia, lymphopenia, and tachycardia [41].

Not only do we see an inflammatory response from SIRS in trauma but IRI has the ability to create another induction of inflammation with release of various cytokines, chemokines, and increased oxidative stress [9]. With this cascade of events, there is further microvascular dysfunction in ischemic tissues and organs following reperfusion. IRI produces complications in regulating vascular barrier properties, control of adhesion and extra-vascular trafficking of immune cells, regulation of vascular tone, and control of hemostatic mechanisms [10].

Radical oxygen species (ROS) that are produced after reperfusion following traumatic injury are one of the most potent chemoattractants for polymorphonuclear leukocytes (PMNs) and result in further cell membrane damage by lipid peroxidation [42]. This increase in activated PMNs is the main cause of secondary organ and tissue damage with subsequent development of multi-organ dysfunction (MOD) and ARDS [42]. Several cytokines are also activated, including IL-1, IL-6, thromboxane A2 (TXA2) and tissue necrosis factor (TNF), which provide signals between the responding leucocyte and the vascular endothelial barrier and are believed to be responsible for selective adhesion and transmigration of leukocytes [43].

Aortic occlusion, in particular, has been associated with the greater release of IL-6 and increasing lactate levels with inflammatory sequelae, including increased incidence of ARDS [6]. The increasing lactate reported in multiple studies was associated with a significant ischemia related perfusion injury [6]. There are also significant metabolic derangements seen with the release of tourniquets and deflation of balloon catheters [6,35]. This includes profound hyperkalemia that, as discussed previously, may be similar to a crush injury with the use of AAJT or IRI in aortic occlusion.

Along with IRI and the increasing rates of massive blood transfusion (>100–50 mL/min), we are more commonly seeing severe hyperkalemia and acute kidney injury (AKI). Rates of AKI occurred in 12.5% of the combat population with a 5-fold increased risk of death [44]. Hyperkalemia may occur during massive transfusion secondary to increased concentration in blood with longer durations of storage [45]. This, in turn, has been linked to cardiac arrest in trauma patients and critically ill adults following a massive blood transfusion.

In order to prevent post-transfusion hyperkalemia, a potassium adsorption filter has been developed [46]. This filter aims to function by adsorbing excess potassium ions from red blood cell concentrate. The maximum speed that blood can be transfused through this filter is at a rate of 50 mL/min [46]. Matsurra et al. studied potassium adsorption with rates of transfusion and noted that there is a decreased amount of adsorption with increasing rates of transfusion [46]. Given these results, it may not be feasible for use in massive transfusion protocols, as the rate is not adequate.

More recently, however, newer modifications to potassium filters for use in austere environments have been developed for use as a "bridge to dialysis" concept. The gold standard for renal replacement therapy is difficult in this setting given the logistical footprint of equipment, high volumes of sterile fluid, and the training necessary for operation. There are currently ongoing studies evaluating the feasibility of a simplified hemoperfusion system that can reduce potassium in cases of acute hyperkalemia during pre-hospital combat casualty care.

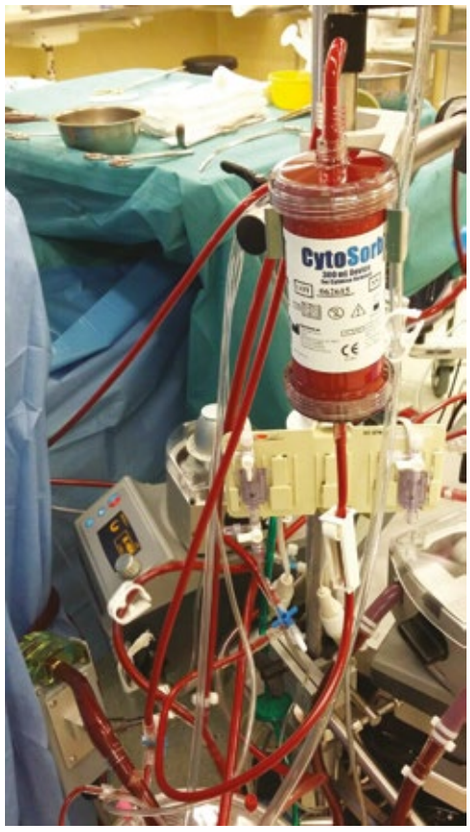

Figure 3 CytoSorb® filter for removal of serum cytokines.

With the complications of IRI and hyperkalemia associated with aortic occlusion, there is also an increase in multiple cytokines that in part create a secondary injury in trauma patients. Removal of these harmful cytokines has been shown to produce an effective result in critically ill patients. Cytokine filtration has been demonstrated in several patient populations including SIRS, sepsis, cardiopulmonary bypass surgery, and even ex vivo lung perfusion [47,48,49]. Given that the cytokines released in IRI are similar to SIRS and sepsis, cytokine filtration may be a promising additional target.

Cytokine adsorber, or Cytosorb (Figure 3), works by non-selectively removing various mediators via hemoadsorption [47]. It contains hemocompatible, porous polymer beads and adsorbs mediators by size exclusion chromatography and hydrophobic interactions [47]. The column is attached to a circuit for filtration such as hemodialysis or continuous renal replacement therapy [47].

Over-abundant cytokines and other proteins can then be removed and a balance of inflammatory mediators is achieved [47].

Cytosorb has been investigated in many different patient populations and utilized in specific case studies. In a small study, patients with ARDS and sepsis underwent filtration with Cytosorb and it was noted that the circulating levels of IL-6 were almost halved vs. the standard of care. However, there has been no research on clinical outcomes to state that this therapy reduces mortality [50].

Another promising avenue for research has been the advent of adenosine deaminase (ADA) inhibitors. Adenosine, in particular, is associated with increased cellular release during SIRS and has functions in vasomotor control, cardiac rhythm, coagulation, and immune function [51]. It is thought to have a protective effect on physiologic responses, including in selective vasodilation, moderate leukocyte activation, and diminish peroxidation of tissues [51].

An inhibitor of ADA, 2′-deoxycoformycin or pentostatin, has been shown to have improved survival in SIRS with a reduction in leukocyte adhesion and vascular damage in the mouse model [51,52]. The elimination half-life is approximately 5–6 hours and the binding to ADA is irreversible, which puts significant variability on the biologic half-life depending on organ/tissue [51,53]. Another study with a mouse model experiencing SIRS from fecal peritonitis also demonstrated significant survivor benefit up to 6 days after insult, with both pretreatment and treatment when clinical signs manifested [51]. Law et al. also demonstrated reduced extravasation of albumin, diminished leukocyte rolling and adhesion and attenuated pro-inflammatory cytokine responses [51].

Following these studies, pentostatin has been evaluated in steroid resistant graft versus host disease, hairy cell leukemia, inflammatory bowel disease, and malignant pleural mesothelioma [54,55,56,57]. It has shown some benefit in leukemia patients, however, further studies are still underway in its use in other populations. The use of pentostatin as a prophylactic agent in SIRS and trauma requires further studies but may have a beneficial effect in reducing the physiologic consequences and rates of mortality.

CONCLUSION

With the advent of several innovative techniques for aortic occlusion in trauma, we must shift our focus to the physiologic penalty associated with distal occlusion. Although these patients benefit in the moment of pre-hospital casualty care, we may be further harming them with a secondary hit of distal ischemia and reperfusion injury. Pentostatin, Cytosorb, and potassium filters are a promising beginning to this avenue of treatment and further studies need to be undertaken to determine feasibility in austere environments.

REFERENCES

[1] Davis JS, Satahoo SS, Butler FK, et al. An analysis of prehospital deaths. J Trauma Acute Care Surg. 2014;77:213–8.

[2] Eastridge BJ, Mabry RL, Seguin P, et al. Death on the battlefield (2001–2011): implications for the future of combat casualty care. J Trauma Acute Care Surg. 2012;73:S431–7.

[3] Eastridge BJ, Hardin M, Cantrell J, et al. Died of wounds on the battlefield: causation and implications for improving combat casualty care. J Trauma Inj Infect Crit Care. 2011;71:S4–8.

[4] Saito N, Matsumoto H, Yagi T, et al. Evaluation of the safety and feasibility of resuscitative endovascular balloon occlusion of the aorta. J Trauma Acute Care Surg. 2015;78:897–904.

[5] Sridhar S, Gumbert SD, Stephens C, Moore LJ, Pivalizza EG. Resuscitative endovascular balloon occlusion of the aorta: principles, initial clinical experience, and considerations for the anesthesiologist. Anesth Analg. 2017;125:884–90.

[6] Morrison JJ, Ross JD, Markov NP, Scott DJ, Spencer JR, Rasmussen TE. The inflammatory sequelae of aortic balloon occlusion in hemorrhagic shock. J Surg Res. 2014;191:423–31.

[7] Markov NP, Percival TJ, Morrison JJ, et al. Physiologic tolerance of descending thoracic aortic balloon occlusion in a swine model of hemorrhagic shock. Surgery. 2013;153:848–56.

[8] Akbari G, Ali Mard S, Veisi A. A comprehensive review on regulatory effects of crocin on ischemia/reperfusion injury in multiple organs. Biomed Pharmacother. 2018;99:664–70.

[9] Maeda K, Ruel M. Prevention of ischemia-reperfusion injury in cardiac surgery: therapeutic strategies targeting signaling pathways. J Thorac Cardiovasc Surg. 2015;149:910–1.

[10] Kalogeris T, Baines CP, Krenz M, Korthuis RJ. Cell biology of ischemia/reperfusion injury. Int Rev Cell Mol Biol. 2012;298:229–317.

[11] Stannard A, Eliason JL, Rasmussen TE. Resuscitative endovascular balloon occlusion of the aorta (REBOA) as an adjunct for hemorrhagic shock. J Trauma Inj Infect Crit Care. 2011;71:1869–72.

[12] Hughes CW. Use of an intra-aortic balloon catheter tamponade for controlling intra-abdominal hemorrhage in man. Surgery. 1954;36:65–8.

[13] Abe T, Uchida M, Nagata I, Saitoh D, Tamiya N. Resuscitative endovascular balloon occlusion of the aorta versus aortic cross clamping among patients with critical trauma: a nationwide cohort study in Japan. Crit Care. 2016;1–10.

[14] Low RB, Longmore W, Rubinstein R, Flores L, Wolvek S. Preliminary report on the use of the Percluder occluding aortic balloon in human beings. Ann Emerg Med. 1986;15:1466–9.

[15] Qasim Z, Brenner M, Menaker J, Scalea T. Resuscitative endovascular balloon occlusion of the aorta. Resuscitation. 2015;96:275–9.

[16] Teeter WA, Matsumoto J, Idoguchi K, et al. Smaller introducer sheaths for REBOA may be associated with

fewer complications. J Trauma Acute Care Surg. 2016; 81:1039–45.
[17] White JM, Cannon JW, Stannard A, Markov NP, Spencer JR, Rasmussen TE. Endovascular balloon occlusion of the aorta is superior to resuscitative thoracotomy with aortic clamping in a porcine model of hemorrhagic shock. Surgery. 2011;150:400–9.
[18] DuBose JJ, Scalea TM, Brenner M, et al. The AAST prospective Aortic Occlusion for Resuscitation in Trauma and Acute Care Surgery (AORTA) registry: Data on contemporary utilization and outcomes of aortic occlusion and resuscitative balloon occlusion of the aorta (REBOA). J Trauma Acute Care Surg. 2016;81:409–19.
[19] Northern DM, Manley JD, Lyon R, et al. Recent advances in austere combat surgery: use of aortic balloon occlusion as well as blood challenges by special operations medical forces in recent combat operations. J Trauma Acute Care Surg. 2018; *In Press* doi: 10.1097/TA.0000000000001966
[20] Morrison JJ, Stannard A, Midwinter MJ, Sharon DJ, Eliason JL, Rasmussen TE. Prospective evaluation of the correlation between torso height and aortic anatomy in respect of a fluoroscopy free aortic balloon occlusion system. Surg (United States). 2014;155:1044–51.
[21] Scott DJ, Eliason JL, Villamaria C, et al. A novel fluoroscopy-free, resuscitative endovascular aortic balloon occlusion system in a model of hemorrhagic shock. J Trauma Acute Care Surg. 2013;75:122–8.
[22] Kralovich KA, Morris DC, Dereczyk BE, et al. Hemodynamic effects of aortic occlusion during hemorrhagic shock and cardiac arrest. J Trauma. 1997;42: 1023–8.
[23] Long KN, Houston R, Watson JDB, et al. Functional outcome after resuscitative endovascular balloon occlusion of the aorta of the proximal and distal thoracic aorta in a swine model of controlled hemorrhage. Ann Vasc Surg. 2015;29:114–21.
[24] Stokland O, Miller MM, Ilebekk A, Kiil F. Mechanism of hemodynamic responses to occlusion of the descending thoracic aorta. Am J Physiol – Heart Circ Physiol. 1980; 238:H423 LP-H429.
[25] Annecke T, Kubitz JC, Langer K, et al. Lung injury following thoracic aortic occlusion: comparison of sevoflurane and propofol anaesthesia. Acta Anaesthesiol Scand. 2008;52:977–86.
[26] Inoue J, Shiraishi A, Yoshiyuki A, Haruta K, Matsui H, Otomo Y. Resuscitative endovascular balloon occlusion of the aorta might be dangerous in patients with severe torso trauma: A propensity score analysis. J Trauma Acute Care Surg. 2016;80:559–66.
[27] Napolitano LM. Resuscitative endovascular balloon occlusion of the aorta: indications, outcomes, and training. Crit Care Clin. 2017;33:55–70.
[28] Davidson AJ, Russo RM, Reva VA, et al. The pitfalls of resuscitative endovascular balloon occlusion of the aorta: risk factors and mitigation strategies. J Trauma Acute Care Surg. 2018;84:192–202.
[29] Johnson MA, Neff LP, Williams TK, DuBose JJ. Partial resuscitative balloon occlusion of the aorta (P-REBOA). J Trauma Acute Care Surg. 2016;81:S133–7.
[30] Russo RM, Neff LP, Lamb CM, Cannon JW, Galante JM, Clement NF, et al. Partial resuscitative endovascular balloon occlusion of the aorta in swine model of hemorrhagic shock. J Am Coll Surg. 2016;223: 359–68.
[31] DuBose JJ. How I do it: Partial resuscitative endovascular balloon occlusion of the aorta (P-REBOA). J Trauma Acute Care Surg. 2017;83:197–9.
[32] Manning JE, Murphy CA, Hertz CM, Perretta SG, Mueller RA, Norfleet EA. Selective aortic arch perfusion during cardiac arrest: a new resuscitation technique. Ann Emerg Med. 1992;21:1058–65.
[33] Manning JE, Katz LM, Pearce LB, et al. Selective aortic arch perfusion with hemoglobin-based oxygen carrier-201 for resuscitation from exsanguinating cardiac arrest in swine. Crit Care Med. 2001;29:2067–74.
[34] Manning JE, Ross JD, McCurdy SL, True NA. Aortic hemostasis and resuscitation: preliminary experiments using selective aortic arch perfusion with oxygenated blood and intra-aortic calcium coadministration in a model of hemorrhage-induced traumatic cardiac arrest. Acad Emerg Med. 2016;23:208–12.
[35] Kheirabadi BS, Terrazas IB, Miranda N, et al. Physiological consequences of abdominal aortic and junctional tourniquet (AAJT) application to control hemorrhage in a swine model. Shock. 2016;46:160–6.
[36] Lyon M, Shiver S, Greenfield EM, et al. Use of a novel abdominal aortic tourniquet to reduce or eliminate flow in the common femoral artery in human subjects. J Trauma Acute Care Surg. 2012;73:S103–5.
[37] Kragh JF, Kotwal RS, Cap AP, et al. Performance of junctional tourniquets in normal human volunteers. Prehosp Emerg Care. 2015;19:391–8.
[38] Anonymous. Abdominal aortic tourniquet? Use in Afghanistan. J Spec Oper Med. 2013;13:1–2.
[39] MacLean LD, Meakins JL, Taguchi K, Duignan JP, Dhillon KS, Gordon J. Host resistance in sepsis and trauma. Ann Surg. 1975;182:207–17.
[40] Marik PE, Flemmer M. The immune response to surgery and trauma: Implications for treatment. J Trauma Acute Care Surg. 2012;73:801–8.
[41] Gentile LF, Cuenca AG, Efron P, et al. Persistent inflammation and immunosuppression: A common syndrome and new horizon for surgical intensive care. J Trauma Acute Care Surg. 2012;72:1491–501.
[42] Lenz A, Franklin GA, Cheadle WG. Systemic inflammation after trauma. Injury. 2007;38:1336–45.
[43] Gillani S, Cao J, Suzuki T, Hak DJ. The effect of ischemia-reperfusion injury on skeletal muscle. Injury. 2012;43: 670–5.
[44] Stewart IJ, Sosnov JA, Howard JT, Chung KK. Acute kidney injury in critically injured combat veterans: a retrospective cohort study. Am J Kidney Dis. 2016;68(4): 564-570.
[45] Asai T, Inaba S, Ohto H, et al. Guidelines for irradiation of blood and blood components to prevent post-transfusion graft-vs.-host disease in Japan. Transfus Med. 2000;10:315–20.
[46] Matsuura H, Akatsuka Y, Muramatsu C, et al. Evaluation of the potassium adsorption capacity of a potassium adsorption filter during rapid blood transfusion. Vox Sang. 2015;108:428–31.
[47] Linden K, Scaravilli V, Kreyer SFX, et al. Evaluation of the CytoSorb™ hemoadsorptive column in a pig model

of severe smoke and burn injury. Shock. 2015;44: 487–95.
[48] Iskender I, Cosgun T, Arni S, et al. Cytokine filtration modulates pulmonary metabolism and edema formation during ex vivo lung perfusion. J Heart Lung Transplant. 2017;1–9.
[49] Bernardi MH, Rinoesl H, Dragosits K, et al. Effect of hemoadsorption during cardiopulmonary bypass surgery – a blinded, randomized, controlled pilot study using a novel adsorbent. Crit Care. 2016;20:96.
[50] Morris C, Gray L, Giovannelli M. Early report: The use of CytoSorb™ haemabsorption column as an adjunct in managing severe sepsis: Initial experiences, review and recommendations. J Intensive Care Soc. 2015;16:257–64.
[51] Law WR, Valli VE, Conlon BA. Therapeutic potential for transient inhibition of adenosine deaminase in systemic inflammatory response syndrome. Crit Care Med. 2003;31:1475–81.
[52] Cohen ES, Law WR, Easington CR, et al. Adenosine deaminase inhibition attenuates microvascular dysfunction and improves survival in sepsis. Am J Respir Crit Care Med. 2002;166:16–20.
[53] Brogden RN, Sorkin EM. Pentostatin. A review of its pharmacodynamic and pharmacokinetic properties, and therapeutic potential in lymphoproliferative disorders. Drugs. 1993;46:652–77.
[54] Alam N, Atenafu EG, Tse G, et al. Limited benefit of pentostatin salvage therapy for steroid-refractory grade III-IV acute graft-versus-host disease. Clin Transplant. 2013;27:930–7.
[55] Johnston JB. Mechanism of action of pentostatin and cladribine in hairy cell leukemia. Leuk Lymphoma. 2011;52:43–5.
[56] Brown JB, Lee G, Grimm GR, Barrett TA. Therapeutic benefit of pentostatin in severe IL-10-/- colitis. Inflamm Bowel Dis. 2008;14:880–7.
[57] Nakajima Y, Kanno T, Nagaya T, et al. Adenosine deaminase inhibitor EHNA exhibits a potent anticancer effect against malignant pleural mesothelioma. Cell Physiol Biochem. 2015;35:51–60.

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Severe Venous Injury in Acute Trauma Setup – Is There a Role for Endovascular Treatment?

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Background: The role of endovascular treatment of acute traumatic venous injuries (ATVI) remains controversial. Endovascular resuscitation and hybrid trauma management (EVTM) concepts, which constitute the combination of conventional and endovascular capabilities in the treatment of vascular injuries continue to evolve, yet published reports of traumatic venous injuries treated by endovascular means remain confined to sporadic case reports.
Methods: The medical literature from 1990 to 2017 using Pubmed and OVID Medline databases was reviewed to search for reports on the endovascular treatment of ATVI. No publications were excluded due to the small number of publications available. Sixteen reports were found. The manuscripts were analyzed regarding the mechanism, location, and type of injury; endovascular techniques utilized; and both clinical and radiographic outcomes.
Results: Endovascular treatment was reported in only 16 patients with ATVI during the study period. Most cases (10/16; 62.5%) were secondary to blunt trauma, while the rest (6/16; 37.5%) were secondary to penetrating injuries equally divided between gunshot wounds and stab wounds. Endovascular stent or stent graft utilization was employed in 12 of these cases, 2 cases were treated by endovascular embolization and in 2 cases endovascular balloons were used for temporary hemorrhage control to facilitate open surgical exposure and intervention. No mortalities related to the endovascular interventions were reported.
Conclusion: The experience with endovascular treatment of ATVI remains very limited. The results currently available, although very limited in numbers, appear promising. Additional study will prove essential in defining the optimal employment of EVTM concepts in the management of venous injuries, particularly as technology continues to advance and experience with these modalities increases.

Keywords: *EVTM; Venous Injury; Review; Endovascular Treatment*

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INTRODUCTION

Acute traumatic venous injuries (ATVI), caused secondary either to blunt or penetrating mechanisms, are very rare. These injuries are, however, associated with high mortality rates of up to 50% at the scene of injury and 30–50% among those that reach hospital [1,2]. These figures have not changed during the past decades, despite all the advances in prehospital care, damage control resuscitation, and surgery.

ATVI are frequently accompanied by multiple, often severe, organ injuries. Most major veins, which are high

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volume–low pressure systems, are located deep within the neck, mediastinum, and retroperitoneum, and as such are not easily accessible to control with expedient packing. The location of these major injuries often dictates extensive anatomic dissection in order to expose and control the bleeding venous source. Van Rooyen et al. reported a 37% mortality rate among patients with inferior vena cava (IVC) injuries secondary to the penetrating mechanism of injury. These investigators suggested that the consideration of less aggressive treatment for hemodynamically stable patients may improve the high mortality rate [3].

The evolving concepts of endovascular resuscitation and hybrid trauma management (EVTM), which have gained in popularity, represent attractive alternative treatment paradigms for cases which were previously treated with open surgery. Recent publications have demonstrated lower complication and mortality rates among patients treated by such endovascular means [4,5].

The published literature regarding endovascular management of significant ATVI is scarce and remains confined to case reports. The aim of our study was to summarize the cumulative experience of endovascular treatment of ATVI through a review of the available English medical literature.

MATERIALS AND METHODS

A systematic review of the English medical literature was conducted, using the Pubmed (www.pubmed.gov, accessed 1 February 2012) service of the National Library of Medicine/National Institutes of Health and the OVID Medline databases to identify all case reports and case series of endovascular management of ATVI. Only reports regarding acute trauma patients were included while iatrogenic as well as delayed traumatic venous complications were excluded from the search database. Specifically, the search terms "venous", "axillosubclavian veins", "iliac veins", "superior and inferior vena cava", "innominate vein" were combined with "trauma" or "injury" and "endovascular management" and "grafts" to identify articles for review. The following criteria were used to select studies to be included in the analysis: adequate information regarding the mechanism, location, and type of the injury, type of endovascular management, surgical intervention, and follow-up.

RESULTS

The use of endovascular techniques for the treatment of ATVI was described in only 16 cases, constituting 13 males and 3 females, during the 20 years period from 1997–2017. The mean age of patients was 43.1 years old (18–76). Blunt trauma was the leading cause of ATVI, comprising 62.5% (10/16) of cases. Penetrating trauma comprised the remaining 37.5% (6/16), with equal distribution between gunshot wounds (GSW) and stab wounds (3 cases each). Stent or stent grafts were utilized in 12 cases – 5 IVC, 4 iliac, 1 subclavian, 1 axillary, and 1 hepatic vein stents (Table 1). Femoral veins were the preferred access portal for intra-vascular approach to injuries. Stent or stent graft placement achieved 100% angiographic as well as clinical hemorrhage control. Radiographic follow-up images were available for only 7 cases, all of which demonstrated patent veins. The timing of follow-up ranged from 48 hours to 12 months after the injury. Endovascular balloons were used in 2 cases in order to gain temporary hemorrhage control while surgical exposure was conducted to facilitate repair of IVC injuries secondary to GSW [18,19]. Endovascular embolization was used in 2 cases of zone III penetrating neck injuries with isolated jugular vein injury [20,21]. The details of these 4 cases are described in Table 2.

There were no endovascular-related mortalities reported. One patient, successfully treated for IVC bifurcation injury with good clinical and angiographic results, died of a concomitant brain injury [12]. No complications related to the endovascular procedures were reported.

DISCUSSION

The use of venous stents and stent grafts in cases of chronic venous stenosis, as in May Thurner syndrome, has proven a successful and commonly adopted practice for many years. Primary patency rates of 71% and secondary patency of 90% over 24 months following the utilization of this approach, have been reported by Raju and colleagues [22]. The optimal role of endovascular treatment in cases of acute trauma venous injury, however, has not been well elucidated. The main purpose of our present review was to review the accumulated world experience with such treatments in human clinical applications.

ATVI are extremely rare and are associated with high mortality rates – up to 50% in subclavian and axillary vein injuries as described by Demetriades and co-investigators [23]. The traditional treatment of ATVI has been limited to surgical exploration with primary or lateral repair, or ligation of the vein in extreme cases.

EVTM, however, is becoming the preferred mode of treatment among selected hemodynamically stable patients in capable centers, either as a replacement or as an adjunct to traditional open surgical treatment at distinct injury locations [24]. The tendency to use EVTM is gaining more and more popularity with the improved understanding of the ability of this modality to minimize the "secondary hit" associated with major open surgical intervention associated with open surgical repair [25].

Several previous animal studies have demonstrated the potential feasibility and benefit of EVTM technology application in significant venous injury. In addition to human studies outlined in our results, our review identified a number of exploratory studies describing endovascular hemostasis of severe venous injuries in animal hemorrhagic models. In one of these studies, Porta et al.

Table 1 Summary of studies on severe venous trauma patients treated by endovascular stenting.

	Age	*Sex*	*Mechanism*	*Injured Vein*	*Access*	*Treatment*	*Mortality*	*Follow-Up Imaging*
Denton [6]	27	Male	Blunt	Retrohepatic IVC	Trans hepatic right hepatic v.	Two 12 × 40 mm Wallstents (Schneider, Inc., Minneapolis, MN, USA)	No	Venography
Uppot [7]	18	Male	Blunt	Right common iliac	N/A	A 10 × 40 mm Cordis Endovascular SMART nitinol stent (Johnson & Johnson, Miami, FL, USA)	No	N/A
Watardia [8]	62	Male	Blunt	Retrohepatic IVC	RFV	A 24-mm 3 10-cm endoluminal graft was constructed from 2 self-expanding 30-mm 3 5-cm Gianturco Z stents (Cook Diagnostic and Interventional Products, Bloomington, IN, USA) covered with collagen-coated 24-mm Dacron graft (Hemashield, Meadox Medicals, Inc., Oakland, NJ, USA)	No	US+CT
Jeroukhimov [9]	35	Male	Blunt	Left subclavian	LFV	Covered stent-graft (Jostent 12-48, Jomed, Germany)	No	N/A
Kumar [10]	27	Male	Stab wound	Left Axillary	LBV	10 × 50-mm Viabahn endoprosthesis (WL Gore and Associates, Flagstaff, AZ, USA)	No	US
Zieber [11]	44	Male	Blunt	Left EIV	LFV	Two 14 × 50-mm Wallgraft endoprostheses (Boston Scientific, Natick, MA, USA)	No	US
Castelli [12]	65	Female	Blunt	IVC at the ilio-caval bifurcation	RFV & LFV	14 × 31 × 140-mm stent graft (Excluder-WL Gore and Associates)	Yes	N/A
Sam [13]	62	Male	Blunt	Infrarenal IVC	RFV	Two 28.5-mm aortic cuffs (WL Gore and Associates)	No	US
Hommes [14]	29	Female	Stab wound	Retrohepatic IVC	RFV	Two overlapping stent grafts 32 mm diameter and 45 mm length each (GorePXA320400, WL Gore and Associates)	No	CT
Sofue [15]	52	Female	Blunt	Left EIV	LFV	Two 14-mm-diameter, 64-mm-long self-expandable uncovered stent (Easy Wallstent, Boston Scientific, Natick, MA, USA)	No	CT
Beitner [16]	36	Male	Stab wound	Middle hepatic	IJV	Covered stent, Gore Viator Tips endoprosthesis (WL Gore and Associates)	No	N/A
Merchant [17]	47	Male	Blunt	Right EIV	RFV.	16-mm (distal diameter) × 14.5-mm (proximal diameter) Excluder stent-graft (WL Gore and Associates)	No	N/A

BV, left basilic vein; EIV, external iliac vein; IJV, internal jugular vein; IVC, inferior vena cava; LFV, left femoral vein; RFV, right femoral vein; SCV, subclavian vein.

Table 2 Summary of studies on severe venous trauma patients treated by hybrid approach without stenting.

	Age	*Sex*	*Mechanism*	*Description*	*Access*	*Treatment*	*Mortality*	*Follow-Up Imaging*
Angeles [18]	24	Male	GSW	IVC transection	LFV &. RIJV	Proximal and distal control	No	N/A
Bui [19]	50	Male	GSW	IVC transection -	RFV	Proximal and distal balloon control	No	US
Sanabria [20]	35	Male	GSW	Right internal jugular vein transection	LIJV	Embolization	No	CT
Yamanaka [21]	76	Male	Stab wound	Right jugular bulb transection	RIJV & LIJV	Embolization	No	N/A

GSW, gunshot wound; IVC, inferior vena cava; LFV, left femoral vein; LIJV, left internal jugular vein; N/A, not available; RFV, right femoral vein; RIJV, right internal jugular vein.

created angiographic retrohepatic vena cava injury in 20 canines, demonstrating that all animals treated by stent grafts (10 dogs) in their model could achieve survival compared with the control group (10 dogs) in whom mortality was 100% [26]. Additional work conducted by Wang et al. in a swine injury model showed that temporary stent graft insertion through the infrahepatic vena cava could be utilized effectively to facilitate surgical repair of major hepatocaval injury when combined with a Pringle maneuver [27]. Finally, in a study of retrohepatic IVC injuries conducted by Reynolds et al., researchers examined the efficacy of balloon occlusion of the suprahepatic IVC as an adjunctive maneuver. In this swine model, injury was created to the retrohepatic vena cava and hepatic inflow control was achieved by clamping of the hepatoduodenal ligament and infrahepatic vena cava. In their intervention group, suprahepatic IVC control was obtained by resuscitative balloon occlusion. In the intervention group, time to death was significantly prolonged and the blood loss was significantly reduced compared with the control group [28].

Endovascular treatment adjuncts have increasingly proven attractive for the treatment of specific severe vascular injuries due to the complexity and poor results of traditional open treatments [29,30]. The repair of traumatic major venous injuries presents a similar surgical challenge traditionally associated with a myriad of complications. The challenge with the open surgical treatment of these injuries is attributed to the distorted anatomy secondary to ongoing bleeding and associated obscuring hematoma. Exposures can be time-consuming, and iatrogenic injuries to adjacent tissues during harried exposures in obscured hemorrhagic fields represent a significant risk. The exposure itself, by its very nature, disrupts any tamponade effect which may have been temporizing ongoing hemorrhage – often to a disastrous effect.

While attractive options, the present endovascular treatments do possess some limitations. Effective access and imaging are required, in addition to the appropriate expertise. Long-term patency of employed stent-grafts in low flow states remains a matter about which little is known and, accordingly, their use should likely be reserved for high flow large diameter veins, such as the vena cava or iliac veins, among hemodynamically stable patients [31].

Our present review demonstrates that existing literature is devoid of well-organized studies of endovascular treatment of ATVI. At present, they appear encouraging in their reported results, all that has yet been demonstrated in the literature is based on case reports or case series. Reporting bias almost certainly exists – as all published reports describe only the successful treatment of patients who survived the ATVI.

Existing case reports also demonstrate that endovascular adjuncts have been applied across a variety of large venous structures. As these capabilities represent a range of modalities that can be utilized – from balloons to embolization techniques to stent grafts – they also represent a menu of adjuncts that can be effectively considered for utilization in isolation or in conjunction with open repair. In one case reported by Denton et al., primary repair, embolization, and stenting were used in sequence to treat venous injuries in the same patient [6]. Despite the array of potential options, there remains no effective consensus on the ideal anatomic sites for endovascular adjuncts. Regardless, the present consensus among trauma and vascular surgeons suggests that the vessels most likely to prove amenable are those which are difficult to approach surgically and are associated with the greatest risk of iatrogenic injury during the exploration and exposure for open treatment [32].

The use of the femoral vein as the preferred access site was noted from the available review. This finding is easily explained by the maneuverability and versatility this site allows the treating physician and the fact that the upper part of the body is usually much more "crowded" with the staff members who take care of the

airway and breathing systems of the patient during the acute phases of treatment.

Balloon occlusion of major venous injuries likely represent the most expedient and applicable modality for employment in major venous injury. Reynolds and colleagues were the first to describe the successful use of resuscitative balloon occlusion of the IVC (REBOVC) in an experimental setting. In the hands of these researchers, REBOCC demonstrated superior hemorrhage control and prolonged time to death in a swine model of liver hemorrhage as compared to a non-intervention control group [28]. Balloons were utilized by multiple groups in our review. Descriptions of use included the use of two balloons, proximal and distal to the injured vein, in order to control the bleeding and allow a proper definitive solution, either by a surgical or endovascular approach.

Although the majority of injuries employed stent grafts to provide for venous injury repair, Sofue and colleagues used a non-covered stent to facilitate repair. In their described use, this bare metal device was utilized due to the unavailability of a covered one, as part of the treatment of an iliac vein injury [15]. Covered stent grafts, however, have shown superiority in other similar vascular uses [33,34], and should be the device of choice – as they provide one deployment coverage of the injury location from intravascular access. The use of heparinized stents, the degree of the stent oversize (compared to estimated or measured vessel diameter), and types of balloons that should optimally be employed as adjuncts remain to be elucidated more effectively.

Post-intervention management after endovascular repair also requires additional study in the trauma setting – particularly as it relates to optimal surveillance of patency and anti-platelet or anticoagulation regimens. The use of anti-aggregation therapy, specifically via dual antiplatelet (DAP) use, is advised when stents are used in order to treat arterial pathology [35]. The venous system may be even more prone to thrombosis following intimal injury as compared to the arterial system due its basic low flow state. Select reported literature suggests that primary venous repair is associated with a 15% risk for thromboembolic events [36]. It is important to note, however, that there remains no significant meaningful data to guide anti-aggregation or anticoagulation therapy following venous repair by any modality – open or otherwise. [35]. Strong consideration of DAP or anticoagulation therapy is warranted, however, little is known about the ideal dose or duration.

Although our study is the largest review of its kind to date, it has important limitations that must be recognized. Reporting bias among case reports of these types must be considered. While successful in their results, the reports also represent the practices of multiple centers across which standardization of evaluation, treatment, and capabilities are unlikely.

CONCLUSIONS

Our present review, which gathers together existing available case reports, demonstrates that the endovascular treatment of major venous injury is both feasible and potentially effective. Well-organized studies should be conducted regarding the type of endovascular repair modalities that are most likely to be beneficial. Optimal post procedure pharmacological adjuncts and follow-up require additional study.

REFERENCES

[1] Asensio JA, Chahwan S, Hanpeter D, et al. Operative management and outcome of 302 abdominal vascular injuries. Am J Surg. 2000;180:528–34.
[2] Davis TP, Feliciano DV, Rozycki GS, et al. Results with abdominal vascular trauma in the modern era. Am Surg. 2001;67:565–70.
[3] Van Rooyen PL, Karusseit VO, Mokoena T. Inferior vena cava injuries: a case series and review of the South African experience. Injury. 2015;46:71–5.
[4] Branco BC, DuBose JJ, Zhan LX, et al. Trends and outcomes of endovascular therapy in the management of civilian vascular injuries. J Vasc Surg. 2014;60:1297–307.
[5] Branco BC, Boutrous ML, DuBose JJ, et al. Outcome comparison between open and endovascular management of axillosubclavian arterial injuries. J Vasc Surg. 2016;63:702–9.
[6] Denton JR, Moore EE, Coldwell DM. Multimodality treatment for grade V hepatic injuries: perihepatic packing, arterial embolization, and venous stenting. J Trauma. 1997;42:964–7.
[7] Uppot RN, Garcia M, Nguyen H, Wills JS. Traumatic common iliac vein disruption treated with an endovascular stent. AJR Am J Roentgenol. 2001;177:606.
[8] Watarida S, Nishi T, Furukawa A, et al. Fenestrated stent-graft for traumatic juxtahepatic inferior vena cava injury. J Endovasc Ther. 2002;9:134–7.
[9] Jeroukhimov I, Altshuler A, Peer A, Bass A, Halevy A. Endovascular stent-graft is a good alternative to traditional management of subclavian vein injury. J Trauma. 2004;57:1329–30.
[10] Kumar V. Endovascular treatment of penetrating injury of axillary vein with Viabahn endoprosthesis. J Vasc Surg. 2004;40:1243–4.
[11] Zieber SR, Mustert BR, Knox MF, Fedeson BC. Endovascular repair of spontaneous or traumatic iliac vein rupture. J Vasc Interv Radiol. 2004;15:853–6.
[12] Castelli P, Caronno R, Piffaretti G, Tozzi M. Emergency endovascular repair for traumatic injury of the inferior vena cava. Eur J Cardiothorac Surg. 2005;28:906–8.
[13] Sam AD 2nd, Frusha JD, McNeil JW, Olinde AJ. Repair of a blunt traumatic inferior vena cava laceration with commercially available endografts. J Vasc Surg. 2006;43:841–3.
[14] Hommes M, Kazemier G, van Dijk LC, et al. Complex liver trauma with bilhemia treated with perihepatic packing and endovascular stent in the vena cava. J Trauma. 2009;67:E51–3.

[15] Sofue K, Sugimoto K, Mori T, Nakayama S, Yamaguchi M, Sugimora K. Endovascular uncovered Wallstent placement for life threatening isolated iliac vein injury caused by blunt pelvic trauma. Jpn Radiol. 2012;30:680–83.
[16] Beitner MM, Suh N, Dowling R, Miller JA. Penetrating liver injury managed with a combination of balloon tamponade and venous stenting. A case report and literature review. Injury. 2012;43:119–22.
[17] Merchant M, Pallan P, Prabhakar N, Saker M, Resnick SA. Treatment of traumatic thoracic and iliac venous injury with endovascular stent-grafts. J Vasc Interv Radiol. 2013;24:1920–3.
[18] Angeles AP, Agarwal N, Lynd C Jr. Repair of a juxtahepatic inferior vena cava injury using a simple endovascular technique. J Trauma. 2004;56:918–21.
[19] Bui TD, Mills JL. Control of inferior vena cava injury using percutaneous balloon catheter occlusion. Vasc Endovascular Surg. 2009;43:490–3.
[20] Sanabria A, Jiménez CM. Endovascular management of an exsanguinating wound of the right internal jugular vein in zone III of the neck: case report. J Trauma. 2003; 55:158–61.
[21] Yamanaka K, Yamamoto A, Ishida K, et al. Successful endovascular therapy of a penetrating zone III jugular bulb injury. A case report. Interv Neuroradiol. 2012;18: 195–9.
[22] Raju S, Owen S Jr, Neglan P. The clinical impact of iliac venous stents in the management of chronic venous insufficiency. J Vasc Surg. 2002;35:8–15.
[23] Demetriades D, Chahwan S, Gomez H, et al. Penetrating injury to the subclavian and axillary vessels. J Am Coll Surg. 1999;188:290–95.
[24] Sise M, Wall MJ. Endovascular management of arterial injury. In: JL Cameron JL, Cameron AM, editors, Current Surgical Therapy Baltimore, Maryland: Elsevier; 2017. pp. 1279–85.
[25] Dobson GP. Addressing the global burden of trauma in major surgery. Front Surg. 2015;2:43.
[26] Porta RM, Poggetti RS, Pereira O, et al. An experimental model for the treatment of lethal bleeding injury to the juxtahepatic vena cava with stent graft. J Trauma. 2006; 60:1211–20.
[27] Wang TG, Liu RH, Li FS, Yu SJ, Sun RR, Dong JH. A temporary stent graft as a new shunt for juxtahepatic vena cava injury. J Trauma Acute Care Surg. 2014;77: 351–5.
[28] Reynolds CL, Celio AC, Bridges LC, et al. REBOA for the IVC? Resuscitative balloon occlusion of the inferior vena cava (REBOVC) to abate massive hemorrhage in retrohepatic vena cava injuries. J Trauma Acute Care Surg. 2017;83:1041–46.
[29] Branco BC, DuBose JJ. Endovascular solutions for the management of penetrating trauma: an update on REBOA and axillo-subclavian injuries. Eur J Trauma Emerg Surg. 2016;42:687–94.
[30] DuBose JJ, Scalea TM, Brenner M, et al.; AAST AORTA Study Group. The AAST prospective Aortic Occlusion for Resuscitation in Trauma and Acute Care Surgery (AORTA) registry: Data on contemporary utilization and outcomes of aortic occlusion and resuscitative balloon occlusion of the aorta (REBOA). J Trauma Acute Care Surg. 2016;81:409–19.
[31] Anaya-Ayala JE, Charlton-Ouw KM, Kaiser CL, Peden EK. Successful emergency endovascular treatment for superior vena cava injury. Ann Vasc Surg. 2009;23: 139–41.
[32] Galili O, Hebron D, Dubose J, et al. Use of endovascular balloons may simplify the proximal and distal control in complicated vascular trauma. JEVTM. 2017;1:39–41.
[33] Hajibandeh S, Hajibandeh S, Antoniou SA, Torella F, Antoniou GA. Covered vs uncovered stents for aortoiliac and femoropopliteal arterial disease: A systematic review and meta-analysis. J Endovasc Ther. 2016;23:442–52.
[34] Katsanos K, Spiliopoulos S, Karunanithy N, Krokidis M, Sabharwal T, Taylor P. Bayesian network meta-analysis of nitinol stents, covered stents, drug-eluting stents, and drug-coated balloons in the femoropopliteal artery. J Vasc Surg. 2014;59:1123–33.
[35] Ullery BW, Tran K, Itoga N, Casey K, Dalman RL, Lee JT. Safety and efficacy of antiplatelet/anticoagulation regimens after Viabahn stent graft treatment for femoropopliteal occlusive disease. J Vasc Surg. 2015;61:1479–88.
[36] Quan RW, Gillespie DL, Stuart RP, Chang AS, Whittaker DR, Fox CJ. The effect of vein repair on the risk of venous thromboembolic events: a review of more than 100 traumatic military venous injuries. J Vasc Surg. 2008;47:571–7.

Case Report

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Resuscitative Endovascular Balloon Occlusion of the Aorta to Augment Afterload in Non-Traumatic Cardiac Arrest: A Case Report

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We describe the first case report of using a REBOA catheter to augment cardiac afterload in a non-traumatic cardiac arrest patient.

Keywords: *REBOA; Non-traumatic Cardiac Arrest; Cardiac Afterload; Resuscitation; Resuscitative Endovascular Balloon Occlusion of the Aorta*

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INTRODUCTION

Resuscitative endovascular occlusion of the aorta (REBOA) has a deep history, with recent advances in technology resulting in a resurgence of interest [1,2]. This innovation was driven by an interest in advancing truncal hemorrhage control for trauma patients, and the subsequent increase in the use of REBOA has resulted in documented use predominantly in trauma patients including case series [3,4] and registry studies [5–7]. In addition, several authors have identified the potential utility of REBOA in non-traumatic hemorrhage including postpartum hemorrhage [8–10], retroperitoneal debridement [11], post-surgical and gastrointestinal bleeding [12]. As the use of this resuscitative adjunct has expanded, so too has interest in its potential application to normovolemic hypotension. In particular, the application of REBOA as an adjunct closed chest cardiopulmonary resuscitation has received significant attention, including the presentation of preliminary animal data (Pan-Am EVTM, 2018) and publication of a recent review summarizing animal research and case reports [13]. As was the case for hypovolemic hypotension prior to the development of the ER-REBOA catheter, available devices, such as intra-aortic balloon pump devices, are constrained by the need for specialized clinical environments which are generally not available in Emergency Department trauma bays. Herein we describe the use of the ER-REBOA catheter deployed in a case of non-traumatic hypovolemic shock and cardiac arrest.

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Conflicts of interest: None.

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Case Description

A 26-year-old man was found lying on the ground in an abandoned building known for illicit intravenous drug use. The patient was in cardiac arrest and paramedics began cardiopulmonary resuscitation. The patient had the return of spontaneous circulation after a short course of CPR prior to transport. Paramedics noted the man had lower-abdominal bruising as well as ligature marks across his bilateral lower extremities. Although initially treated as a cardiac arrest secondary to illicit drug use, the patient was transported to an ACS verified Level 1 Trauma Center for treatment.

Upon arrival to the Emergency Department, the patient was nonresponsive with a Glasgow Coma Scale (GCS) of 3 but breathing spontaneously. The patient was intubated for airway protection after naloxone was administered with no response. The patient's pulse rate was 130 bpm and blood pressure 50/palp. Since the patient was unresponsive with extensive bruising and unclear history of events, the patient's status was escalated to a Level I trauma alert by the emergency physician who evaluated him. The trauma team arrived with the patient still in extremis and the massive transfusion protocol was activated. Given the concern for possible intraabdominal or pelvic hemorrhage as a cause for the patient's shock, the decision was made to insert an ER-REBOA™ catheter (Prytime Medical, Boerne, TX) [13–15].

The right femoral artery was cannulated percutaneously using a 7 French arterial sheath and the catheter balloon was advanced and inflated in Zone 1 at 45 cm. Based on available military guidelines at the time, Zone 1 is approximately at 45–47 cm and Zone 3 at 22–25 cm. The balloon was inflated until resistance was encountered and the patient's blood pressure responded (approximately 12 mL of saline). Immediate blood pressure improvement followed to 115/65. A FAST examination, chest x-ray, and pelvic x-ray were performed in the trauma bay at this time which showed no injuries consistent with hemorrhagic shock as well as ER-REBOA™ catheter placement in Zone 1 (Figure 1). The patient was then taken immediately to undergo a CT scan of the head, chest, abdomen, and pelvis, which also were negative for injuries, to explain his hypotension (See balloon above the diaphragm in Figure 2). A small-volume pneumomediastinum was discovered which was attributed to the patient receiving chest compressions and right middle-lobe opacities likely due to aspiration.

As the patient's state of shock was cardiogenic in nature, the catheter balloon was deflated after 30 minutes to allow perfusion of the abdominal viscera. This was an early experience with ER-REBOA™ at our institution and we did not deflate the balloon for the CT scans nor did we attempt partial inflation. We have since changed our practice to deflate the balloon at least partially just before obtaining the abdomen/pelvis portion of our scans to allow contrast flow and better identify injuries. After balloon deflation, systolic blood pressure dropped again to the 70s but was successfully augmented with vasopressors. In total, the patient received 5 units of packed red blood cells, 7 units of fresh-frozen plasma, 2 units of platelets, and 10 units of cryoprecipitate. The balloon catheter and introducer sheath were removed about two hours later at the bedside and pressure held until hemostasis was assured. We now aim to remove the ER-REBOA™ catheter within six hours of placement and the introducer sheath within 24 hours of placement.

The next morning, acute compartment syndrome of the right lower extremity was noted and the patient underwent an emergent four-compartment fasciotomy.

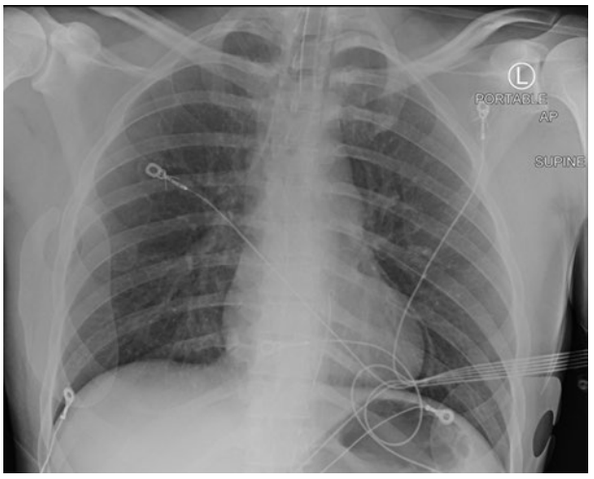

Figure 1 The ER-REBOA™ catheter can be seen left of the spine above the diaphragm.

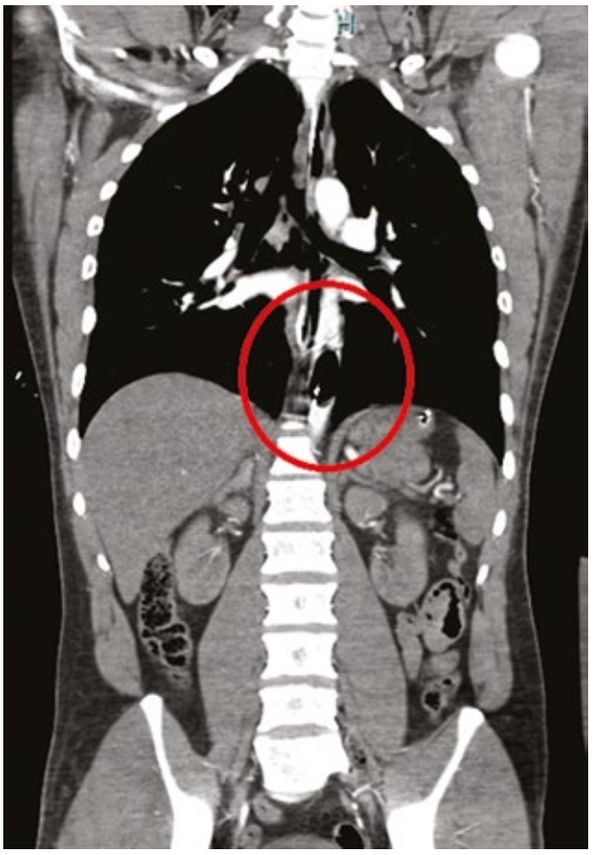

Figure 2 Trauma CT chest/abdomen/pelvis with ER-REBOA™ catheter balloon seen above the diaphragm.

An intra-operative angiogram of the right femoral artery performed at this time revealed no arterial injury or vascular abnormality to suggest catheter-related complications. Thus, the patient compartment syndrome, and

subsequent rhabdomyolysis, was attributed to a prolonged compression position after being down for an unknown period of time and low-flow state during his cardiac arrest.

Patient Outcome

The patient ultimately required an above-the-knee amputation for necrotic and non-functional tissues of his right lower extremity which failed to recover despite fasciotomies. He was discharged on hospital day 52 and was seen doing well in an outpatient clinic two weeks later.

CONCLUSIONS

Resuscitative endovascular balloon occlusion of the aorta (ER-REBOA™) has been used to temporarily occlude the aorta in Zone 1 or 3 in order to control intraabdominal or pelvic hemorrhage and to provide proactive management of life-threatening, refractory, hemorrhagic shock in addition to maximal conventional therapy [10,16]. This is the first published case report to date of ER-REBOA™ use in a non-traumatic cardiac arrest setting leading to a patient surviving. Much like an intra-aortic balloon pump inflating during diastole, the ER-REBOA™ balloon catheter augmented the patient's perfusion of the heart and brain. This allowed time to fluid resuscitate the patient, evaluate for possible traumatic injuries, and ultimately transition blood pressure support to the use of vasopressor medications. We strongly believe the use of ER-REBOA™ to augment cardiac afterload ultimately contributed to the patient surviving this incident. The ER-REBOA™ was readily available and could be placed quickly at the bedside without the need for angiography. Future uses of ER-REBOA™ in the setting of non-traumatic cardiac arrest patients should also consider partial balloon inflation to allow some distal perfusion while augmenting proximal cardiac and cerebral perfusion. Further research into the feasibility as well as outcomes for patients in non-traumatic cardiac arrest is required to better utilize this emerging technology.

REFERENCES

[1] Rasmussen TE, Eliason JL. Military-civilian partnership in device innovation: Development, commercialization and application of resuscitative endovascular balloon occlusion of the aorta. J Trauma Acute Care Surg. 2017; 83:732–5.

[2] Rasmussen TE, Franklin CJ, Eliason JL. Resuscitative endovascular balloon occlusion of the aorta for hemorrhagic shock. JAMA Surg. 2017;152:1072–3.

[3] Manley JD, Mitchell BJ, DuBose JJ, Rasmussen TE. A modern case series of resuscitative endovascular balloon occlusion of the aorta (REBOA) in an out-of-hospital, combat casualty care setting. J Spec Oper Med. 2017; 17:1–8.

[4] Northern DM, Manley JD, Lyon R, et al. Recent Advances in Austere Combat Surgery: Use of aortic balloon occlusion as well as blood challenges by special operations medical forces in recent combat operations. J Trauma Acute Care Surg. 2018;85:S98–S103.

[5] Costantini TW, Coimbra R, Holcomb JB, et al. Pelvic fracture pattern predicts the need for hemorrhage control intervention-Results of an AAST multi-institutional study. J Trauma Acute Care Surg. 2017;82:1030–8.

[6] Brenner M, Inaba K, Aiolfi A, et al. Resuscitative endovascular balloon occlusion of the aorta and resuscitative thoracotomy in select patients with hemorrhagic shock: Early results from the American Association for the Surgery of Trauma's Aortic Occlusion in Resuscitation for Trauma and Acute Care Surgery Registry. J Am Coll Surg. 2018;226:730–40.

[7] DuBose J, Fabian T, Bee T, et al. Ontemporary utilization of resuscitative thoracotomy: Results from the AAST aortic occlusion for resuscitation in trauma and acute care surgery (AORTA) multicenter registry. Shock. 2017; *In Press*. doi: 10.1097/SHK.0000000000001091.

[8] Russo RM, Girda E, Kennedy V, Humphries MD. Two lives, one REBOA: Hemorrhage control for urgent cesarean hysterectomy in a Jehovah's Witness with placenta percreta. J Trauma Acute Care Surg. 2017;83: 551–3.

[9] Ordonez CA, Manzano-Nunez R, Parra MW, et al. Prophylactic use of resuscitative endovascular balloon occlusion of the aorta in women with abnormal placentation: A systematic review, meta-analysis, and case series. J Trauma Acute Care Surg. 2018;84:809–18.

[10] Stensaeth KH, Sovik E, Haig IN, Skomedal E, Jorgensen A. Fluoroscopy-free resuscitative endovascular balloon occlusion of the aorta (REBOA) for controlling life threatening postpartum hemorrhage. PLoS One. 2017;12:e0174520.

[11] Weltz AS, Harris DG, O'Neill NA, O'Meara LB, Brenner ML, Diaz JJ. The use of resuscitative endovascular balloon occlusion of the aorta to control hemorrhagic shock during video-assisted retroperitoneal debridement or infected necrotizing pancreatitis. Int J Surg Case Rep. 2015;13:15–8.

[12] McGreevy D, Toivola A, Bilos L, Pirouzram A , Nilsson KF, Hörer TM. Endovascular resuscitation with aortic balloon occlusion in non-trauma cases: First use of ER-REBOA in Europe. J Endovasc Resus and Trauma Mgmt. 2017;1:42–9.

[13] Daley J, Morrison JJ, Sather J, Hile L. The role of resuscitative endovascular balloon occlusion of the aorta (REBOA) as an adjunct to ACLS in non-traumatic cardiac arrest. Am J Emerg Med. 2017;35:731–6.

[14] DuBose JJ, Scalea TM, Brenner M, et al. The AAST prospective Aortic Occlusion for Resuscitation in Trauma and Acute Care Surgery (AORTA) registry: Data on contemporary utilization and outcomes of aortic occlusion and resuscitative balloon occlusion of the aorta (REBOA). J Trauma Acute Care Surg. 2016;81:409–19.

[15] Napolitano LM. Resuscitative Endovascular Balloon Occlusion of the Aorta: Indications, Outcomes, and Training. Crit Care Clin. 2017;33:55–70.

[16] Pezy P, Flaris AN, Prat NJ, et al. Fixed-distance model for balloon placement during fluoroscopy-free resuscitative endovascular balloon occlusion of the aorta in a civilian population. JAMA Surg. 2017;152:351–8.

Case Report

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Resuscitative Endovascular Balloon Occlusion of the Aorta to Facilitate Continuous Venovenous Hemodiafiltration in a Patient with Methanol Intoxication

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Keywords: *REBOA; Extracorporeal Membrane Oxygenation; Methanol Intoxication; Emergency Medicine*

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BACKGROUND

Resuscitative endovascular balloon occlusion of the aorta (REBOA) was first described by Hughes in the Korean War [1] and regained popularity in the late 2000s. It is successfully used in patients with non-compressible torso hemorrhage due to trauma [2] as well as situations associated with non-traumatic bleeding such as placenta percreta [3] and uncontrollable gastrointestinal bleeding [4]. As REBOA use evolves with time, the life-saving potential of the procedure, especially in bleeding patients, has made itself a useful tool in Emergency Departments (EDs) and also in prehospital environments [5].

CASE REPORT

A 61-year-old male presented to the ED with progressive loss of vision and impaired consciousness for 3 hours. According to his relatives, the patient was a chronic cologne abuser and had ingested an unknown amount of cologne one day prior. He had no medical history and was not on any medication. His initial vital signs were a blood pressure of 80/60 mmHg, a pulse of 98 beats/min, oxygen saturation of 94% and a temperature of 36.2 °C. Due to the patients Glasgow Coma Scale (GCS) of 6 (E4M1V1), he was intubated with rapid sequence intubation. Laboratory studies showed profound lactic acidosis with a calculated level of methanol of 319 mg/dl (pH: 6.76, HCO_3: 6.3 mEq/L, PO_2: 115 mmHg, PCO_2: 37 mmHg, lactate: 6 mmol/L, anion gap: 44). The patient was catheterized for urgent hemodialysis (HD) through his right internal jugular vein and transferred to the intensive care unit (ICU). While awaiting preparations for HD, fluid resuscitation, bicarbonate and norepinephrine infusion was initiated along with 10 cc/kg of intravenous ethanol bolus over 30 minutes followed by a continuous ethanol infusion of 2 cc/kg/h iv infusion. A right radial artery catheter was also placed for monitoring blood pressure. A continuous veno-venous hemodiafiltration (CVVHDF) was initiated due to persistent hypotension and profound acidosis. The blood ethanol level was measured at 6-hour intervals and the infusion was regulated to keep serum ethanol level over 150 mg/dl. As the patient became more hypotensive during CVVHDF, dopamine and epinephrine infusions were added and doses were escalated to reach a norepinephrine infusion of 40 mcg/min, dopamine infusion of 50 mcg/min, and epinephrine infusion of 40 mcg/min. After 24 hours, CVVHDF began to fail because the patient had persistent profound hypotension despite triple inotropic support and this yielded a loss of efficacy in the performance of the HD device.

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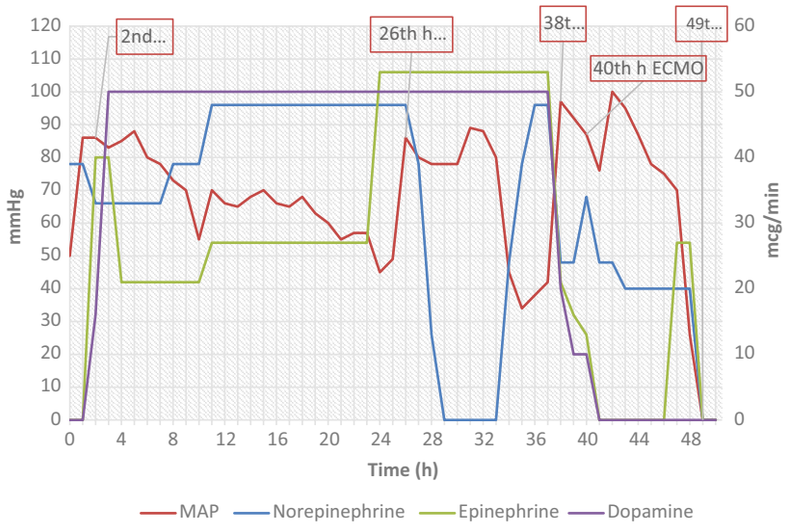

Figure 1 The clinical and interventional course of the patient.

To facilitate CVVHDF, we decided to perform a zone 3 partial REBOA (pREBOA) to augment systolic blood pressure. A 6 Fr introducer sheath was placed into the right common femoral artery (CFA) with ultrasound (US) guidance followed by a 6 Fr Fogarty catheter. After confirmation that the tip of the catheter was at zone 3 by US, the balloon was fully inflated. Another 6 Fr introducer sheath was placed with the same method into the contralateral CFA to monitor blood pressure below the balloon so that pREBOA could be titrated to maintain a distal systolic blood pressure of 80 mmHg. With pREBOA, a blood pressure of 130/80 mmHg was achieved proximal to the balloon and the systolic blood pressure distal to the balloon was 45 mmHg. In the meantime, an extracorporeal membrane oxygenation (ECMO) protocol was initiated and 118 minutes after balloon inflation veno-arterial ECMO was commenced following the balloon deflation and withdrawal of the occlusion catheter. Systolic blood pressure distal to the balloon just before the deflation was 80 mmHg. Concomitantly to ECMO, inotrope support was weaned as tolerated until only a 26 mcg/min of norepinephrine infusion was required (Figure 1). Once a proximal mean arterial pressure greater than 90 mmHg was achieved, CVVHDF resumed with ethanol and bicarbonate infusion. Despite all efforts, lactate levels did not decrease less than 12.8 mmol/L and on the 9th hour of ECMO, the patient developed bradycardia and then cardiac arrest. After 50 minutes of CPR, the patient was declared dead.

DISCUSSION

Although REBOA is a promising new therapy for the management of non-compressible torso hemorrhage even in austere combat environments [6], we presume that it may be an evolving bridging therapy for management of various critically ill patients.

To the best of our knowledge, our case is the first example of REBOA in the literature utilized to facilitate CVVHDF in an intoxicated patient. In a recent case report, REBOA was unconventionally used to improve hemodynamic support in a patient with proximal aortic aneurysm rupture [7]. Similarly, we only aimed to slightly augment mean arterial pressure (MAP) without occluding the renal arteries, thus we preferred a zone 3 REBOA. We also did not want to bear the consequences of limb ischemia and reperfusion injury in an already critically ill patient with profound lactic acidosis. This was the main reason for us to perform a pREBOA rather than a complete one (cREBOA). Unfortunately, we were obliged to use a 6 Fr Fogarty balloon catheter with a 13 mm balloon diameter because we could not find a larger catheter at our institution. However, this lack of resources served our purposes well. We inflated the balloon with volume increments of 0.5 ml until we reached a volume of 2 ml which is the maximum balloon volume allowed and left it fully inflated via a stopcock. Thus, we aimed to protect the balloon from blowing.

Reva et al. demonstrated that with a 40–49% partial occlusion the aorta yields a significant increase in MAP in a bleeding sheep model [8]. As we experienced, placing a contralateral CFA sheath might be useful while monitoring the blood pressure distal to the balloon of the catheter. While providing a distal systolic blood pressure over 80 mmHg to avoid limb ischemia, it may also provide valuable information about the efficacy of the aortic occlusion. Along with the concept of Endovascular Perfusion Augmentation for Critical

Care (EPACC) by Williams et al. [9], the main reason for us to perform an aortic occlusion was to augment the blood flow proximal to the occlusion balloon, in particular, renal arteries at the cost of possible lower limb ischemia. However, this possibility constitutes the main reason for monitoring the blood pressure distal to the balloon to predict any potential lower limb hypoperfusion. In a brief report [10], DuBose recommended using an 8 Fr ER-REBOA catheter to measure the distal arterial pressure below the occlusion balloon and deflating the balloon gradually until the distal arterial waveform below the balloon was observed. However, with the lack of an 8 Fr catheter in our institution, we were not capable of performing a cREBOA. Therefore, there was not an interrupted but only slightly diminished blood flow distal to the balloon. In addition, we presume that using a smaller catheter to achieve pREBOA might be practical unlike DuBose's method. In this case, we tried to focus on the balance of the augmentation of the proximal blood pressure versus introducing enough blood flow below the balloon while avoiding limb ischemia.

REBOA may serve as a bridge to numerous life-saving interventions such as ECMO, selective aortic branch occlusion, coiling, and embolization. In our case, the catheter balloon was inflated in order to 'step up' the ECMO. pREBOA acted as the 'fourth inopressor' along with dopamine, norepinephrine, and epinephrine and provided time for ECMO setup and priming.

CONCLUSION

REBOA may be a useful tool in the management of critically ill patients with a variety of etiologies, even for intoxicated patients. In addition to serving as a bridge to advanced life-saving interventions, REBOA may act as a substitute inotrope when combinations of other inotropic agents fail to preserve the mean arterial pressure.

REFERENCES

[1] Hughes CW. Use of an intra-aortic balloon catheter tamponade for controlling intra-abdominal hemorrhage in man. Surgery. 1954;36:65–8.

[2] Moore LJ, Martin CD, Harvin JA, Wade CE, Holcomb JB. Resuscitative endovascular balloon occlusion of the aorta for control of noncompressible truncal hemorrhage in the abdomen and pelvis. Am J Surg. 2016;212:1222–30.

[3] Manzano-Nunez R, Escobar-Vidarte MF, Naranjo MP, et al. Expanding the field of acute care surgery: a systematic review of the use of resuscitative endovascular balloon occlusion of the aorta (REBOA) in cases of morbidly adherent placenta. Eur J Trauma Emerg Surg. 2018; 44: 519–26.

[4] Sano H, Tsurukiri J, Hoshiai A, Oomura T, Tanaka Y, Ohta S. Resuscitative endovascular balloon occlusion of the aorta for uncontrollable nonvariceal upper gastrointestinal bleeding. World J Emerg Surg, 2016;20. doi: 10.1186/s13017-016-0076-3.

[5] Qasim Z, Brenner M, Menaker J, Scalea T. Resuscitative endovascular balloon occlusion of the aorta. Resuscitation. 2015;96:275–9.

[6] Reva V, Hörer TM, Makhnovsky 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;83:170–6.

[7] Strauss S, Engels P, Harlock J. Distal placement of resuscitative endovascular balloon occlusion of the aorta (REBOA) to restore hemodynamic stability in a patient with proximal aortic rupture. J Endovasc Ther. 2018;25: 257–60.

[8] Reva V, Matsumura Y, Samokhvalov IM, et al. Defining degree of aortic occlusion for partial-REBOA: A computed tomography study on large animals. Injury. 2018; 49:1058–63.

[9] Williams TK, Johnson A, Neff L, et al. "What's in a name?" A consensus proposal for a common nomenclature in the endovascular resuscitative management and REBOA literature. JEVTM. 2017;1:9–12.

[10] DuBose JJ. How I do it: partial resuscitative endovascular balloon occlusion of the aorta (P-REBOA). J Trauma Acute Care Surg. 2017;83:197–9.

Case Report

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Isolated Abdominal Injury of Blunt Aortic Trauma: Two Case Reports and Review of the Literature

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Background: Injury to the abdominal aorta as part of a blunt injury is a rare event and is normally associated with other abdominal injuries. The management of these injuries can be non-operative, open repair or endovascular repair.
Methods: We present two cases of blunt abdominal aortic injury (BAAI) in which the aorta was the only abdominal injury. This is followed by a review of the current literature.
Results: Both these cases were treated endovascularly with good results.
Conclusions: BAAIs are rare and can occur in isolation, i.e. without any accompanying abdominal injuries. Despite historically being treated mostly by open repair, endovascular repair offers many advantages and can be safely managed.

Keywords: *Aorta; Abdominal; Trauma; Endovascular Procedure*

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INTRODUCTION

Injury to the abdominal aorta as part of a blunt injury is a rare event. A Western Trauma Association study reviewed 392,315 blunt injury cases and found blunt abdominal aortic injury (BAAI) in only 113 (0.3%) cases [1]. In many of these cases, the aortic injury is associated with other abdominal injuries, such as solid organ injury, small bowel injury, mesenteric hematoma, colon injury, etc. [2]. The increasing use of multi-detector computed tomography (CT) scans has led to an increase in the detection of injuries to the abdominal aorta [3].

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Blunt injuries to the aorta are classified by type and by location. The types of aortic injury include free rupture and pseudoaneurysm, in which the external contour of the aorta is abnormal, as well as intimal tears and large intimal flaps, in which the external contour of the aorta is unchanged. The classification of the zones of injury are based on possible endovascular approaches: Zone I injuries range from the diaphragmatic hiatus to the origin of the superior mesenteric artery (SMA); Zone II encompasses the SMA, inferior mesenteric artery (IMA), and renal arteries; and Zone III includes injuries below the renal arteries [1].

The management of blunt abdominal injury ranges from non-operative management to open aortic repair. In the past few decades, endovascular techniques have emerged. As more experience is gained with aortic aneurysm repair in elective and urgent settings, the endovascular management of thoracic and abdominal aortic repair in trauma is rapidly becoming more common.

We present two cases where injury to the abdominal aorta was the sole abdominal injury. Details on the management and outcome are described and a review of the available literature is presented.

Case Report 1

A 52-year-old cyclist presented to the trauma bay after falling from his bicycle at a high speed of 80 kph (approx.

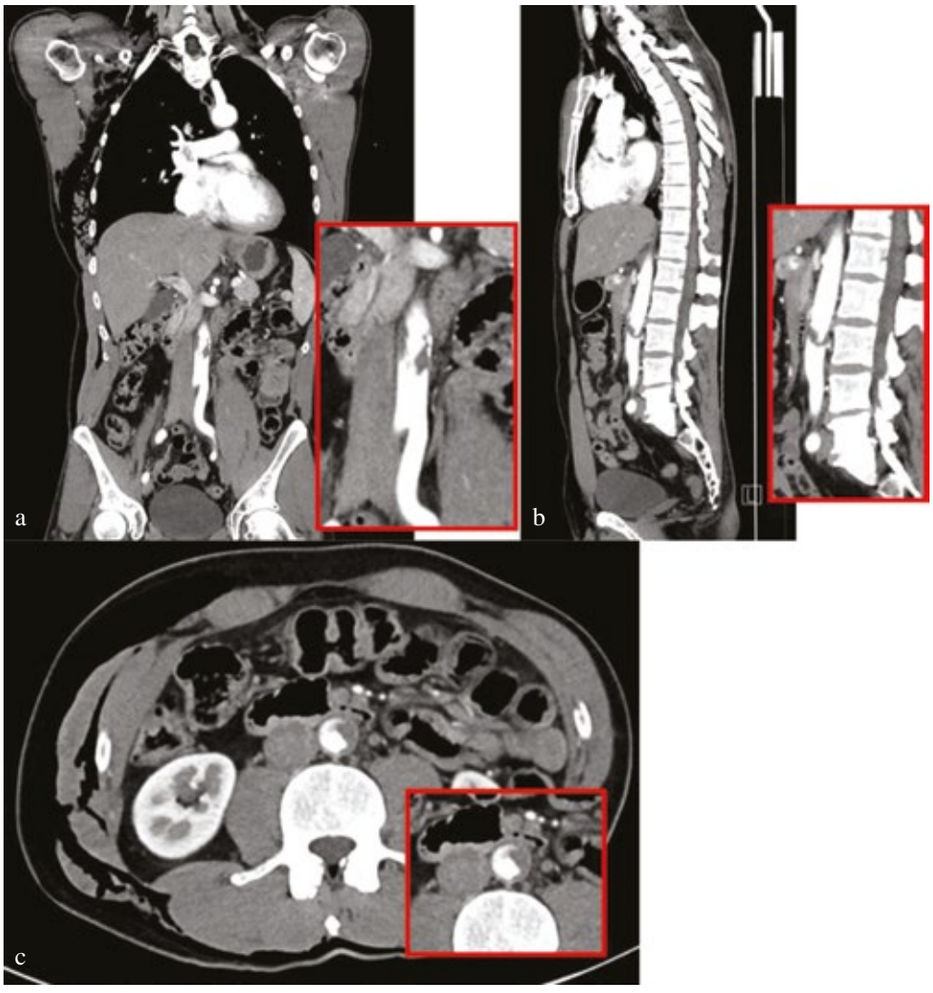

Figure 1 Computed tomography images of the patient in case 1. (**a**) Coronal, (**b**) sagittal, and (**c**) axial views with enlarged images in the inserts.

50 mph) during a cross-country ride. He was alert, conscious and ambulating at the scene, though his helmet had fractured. His vital signs were normal and stable at the scene and during transfer. Primary survey in the trauma bay was normal, and secondary survey revealed substantial subcutaneous emphysema in his right chest and torso, as well as a deformity of the right clavicle. Pedal pulses were normal. A chest X-ray in the trauma bay revealed subcutaneous emphysema with no pneumothorax, and FAST examination was negative.

The patient proceeded to the CT scanner for a "whole-body" scan (see Figure 1). The CT revealed substantial subcutaneous emphysema, extending from the cranium to the scrotum, as well as minimal right-sided pneumothorax, minor pneumomediastinum, and air in the spinal column. There was a right-sided clavicular fracture and a fracture to his right 6th rib. Additionally, there was a large intimal flap in the infra-renal aorta, with partial thrombosis. No other head, chest or abdominal injury was noted.

The patient continued to be stable and with normal vital signs. He was admitted to the intensive care unit, and gastro-esophagoscopy and bronchoscopy were performed to rule out airway and/or esophageal leak. Both studies were normal. After other injuries were ruled out, the patient was taken to the operating room for angiography and endovascular repair of the aortic injury.

A Bentley 16×59 mm^2 covered stent graft was deployed in the infra-renal aorta through a 6 Fr sheath in the right groin, and a second (16×59 mm^2) stent-graft was deployed with some overlap to provide an extension for the stent (see Figure 2). During deployment, thrombus material lodged in the left leg resulting in acute leg ischemia. Immediate thrombectomy was successfully performed. The patient had no history of hypertension, and his blood pressure was in the normal range during his hospital stay, so no effort was made to lower his blood pressure further. The patient was discharged 5 days later after the subcutaneous emphysema had resolved and remained well at follow-up 6 months

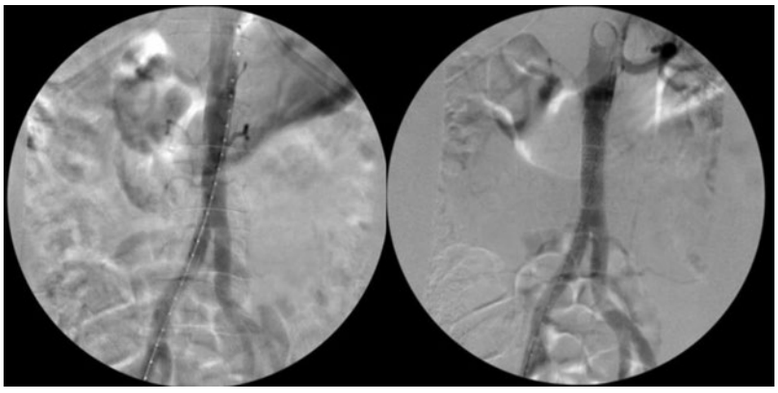

Figure 2 Intra-operative images of the patient in case 1, before (left) and after (right) deployment of the stent-graft.

later. He was treated with enoxaparin (LMWH) for 3 months after surgery, as well as aspirin which we have recommended that he continue to take indefinitely.

Case Report 2

A 57-year-old patient was referred to the ER 15 weeks after sustaining an open fracture of his right tibia and fibula. While repairing his minivan, the vehicle rolled onto him, compressing his leg and abdomen, resulting in an open leg fracture. The patient initially downplayed the severity of his abdominal pain due to his leg injury, thus, no work-up on the abdomen was performed. His leg was repaired, and the patient was referred to a rehabilitation center.

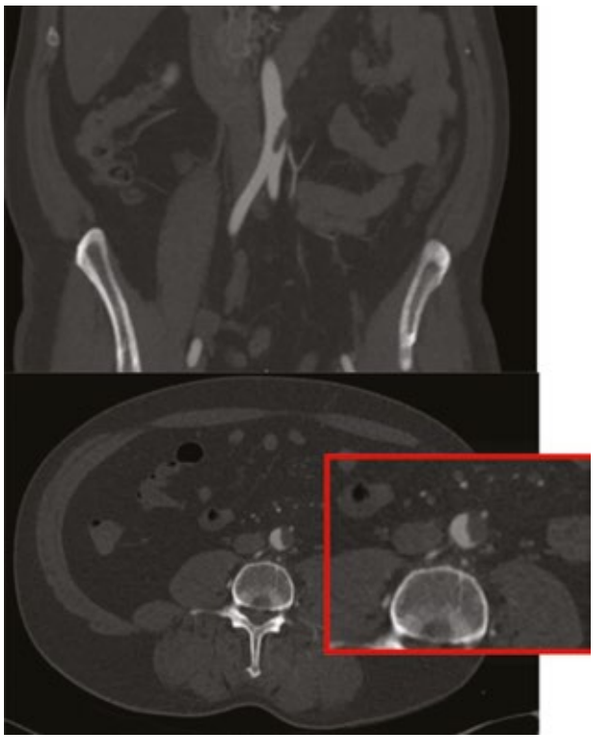

Figure 3 CT angiography images of the patient in case 2.

Three months after his injury, the patient was referred to the ER due to acute ischemia of his left leg. Duplex ultrasonography was performed and revealed thrombosis of the left popliteal artery. An additional CT angiography of the chest and abdomen revealed a large mural and "floating" thrombus in the abdominal aorta (see Figure 3). After further questioning, it seems he had sustained a significant abdominal blunt trauma at the time of the accident. We speculated that the findings in the aorta were a result of an intimal tear which occurred at the time of the initial trauma.

The patient underwent endovascular repair with a (Bentley 16×59 mm^2) covered stent graft with a satisfying technical result (see Figure 4). Some of the fresh thrombus dislodged distally and a femoral thrombectomy was successfully performed, with the pallor resolving and flow restored on the doppler.

The patient had no history of hypertension, and his blood pressure was in a normal range during his hospital stay, therefore no effort was made to lower his blood pressure further. The patient experienced an uneventful post-operative course and was alive and well on follow-up 6 months later. We recommended lifelong aspirin.

DISCUSSION

Isolated blunt injury to the abdominal aorta is rare. We present two such cases, as well as details of the management and outcomes. In patients where abdominal exploration is unwarranted, the advantages of endovascular approaches are even more significant. Suspicion of blunt abdominal trauma should be raised in patients presenting with a seat belt sign, abdominal wall disruption, lumbar spine fracture, hollow viscus injury, or abnormal peripheral pulses [4]. However, both patients we have presented demonstrated none of these signs. There should be a high index of suspicion for blunt abdominal

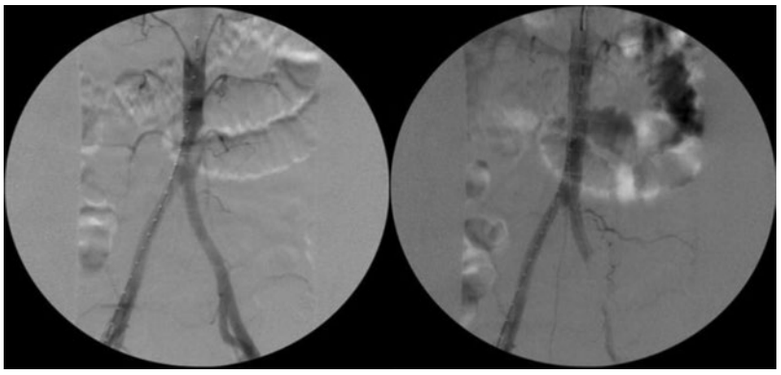

Figure 4 Intra-operative images of the patient in case 2, before (left) and after (right) deployment of the stent.

Table 1 Management of BAAI according to the zone of injury.

Zone of Injury	*Management*
Zone 1	Non-operative, endovascular, or open repair
Zone 2	Non-operative or open repair
Zone 3	Non-operative, endovascular, or open repair

injury in all patients who present no other abdominal or vascular injuries. As the frequency of CT scans in the evaluation of trauma patients continues to increase, more such asymptomatic injuries to the abdominal aorta are likely to be detected [3].

The Western Trauma Association conducted a review of over 392,315 blunt trauma patients from 1996–2011, 113 of which had a BAAI. The leading cause of injury was motor vehicle injury (60%). The review does not specify if patients presented with no other abdominal injury, as was the case in our two patients. However, the number of associated injuries: 50 cases of spine fractures, 45 of hemoperitoneum, 43 of solid organ injury, 39 of small bowel injury, 32 of colon injury, etc. suggests that the majority of cases involve injury to another abdominal organ. In a separate study of 414 patients from 180 centers, 119 patients (27%) sustained blunt injuries to the abdominal aorta without any other major injuries [2].

In the Western Trauma Association cohort, 17.7% of cases presented as intimal tears, 36% as large intimal flaps, 15.9% as pseudoaneurysms, and 31.9% as free ruptures. Forty cases (35.4%) were managed non-operatively, 49 cases (43.4%), including all of the aortic free ruptures, were managed with open surgical repair, and only 17 cases (15%) were managed mainly with endovascular therapy. The choice of the treatment was affected by the patient's condition, the type of injury and the zone of injury (see Table 1). Injuries without contour abnormalities were treated mostly with a non-operative or endovascular approach, whereas free rupture was exclusively treated with open repair. While injuries to zones 1 and 3 were treated with all three approaches, injuries to zone 2 were never treated with an endovascular approach [1]. In a separate review of 436 patients with BAAI, only 42 patients (10%) underwent repair, and the majority of them (29 patient, 69%) underwent endovascular repair [2].

An endovascular approach to a BAAI offers several advantages. In cases where open abdominal exploration is not necessary, endovascular repair avoids the morbidity of an open abdominal or retroperitoneal approach. In patients in which exploration reveals gross contamination of the peritoneal cavity, an endovascular approach avoids contamination of major vessels and graft material [5].

CONCLUSION

BAAIs are rare and can occur in isolation, i.e. without any accompanying abdominal injuries. Despite historically being treated mostly by open repair, endovascular repair offers many advantages and can be safely managed. We expect endovascular treatment to become the mainstay of treatment in the near future.

REFERENCES

[1] Shalub S, Starnes BW, Brenner ML, et al. Blunt abdominal aortic injury: A Western Trauma Association multicenter study. J. Trauma Acute Care Surg. 2014;77:879–85.
[2] de Mestral C, Dueck AD, Gomez D, Haas B, Nathens AB. Associated injuries, management, and outcomes of blunt abdominal aortic injury. J. Vasc. Surg. 2012;56: 656–60.
[3] Mellnick VM, McDowell C, Lubner M, Bhalla S, Menias CO. CT features of blunt abdominal aortic injury. Emerg. Radiol. 2012;19:301–7.
[4] Wicks RF, Raines AR, Enix JL, Irvan J, Albrecht R. Abdominal aortic and iliac injury in blunt trauma: a single center's experience. Am. Surg. 2015;81:E174.
[5] Halkos ME, Nicholas J, Kong LS, Burke JR, Milner R. Endovascular management of blunt abdominal aortic injury. Vascular 2006;14:223–26.

Case Report

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Endovascular Resuscitation for Ruptured Abdominal Aortic Aneurysms with Main Stent-Graft and REBOA via Single-Sided Access

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Background: Aortic balloon occlusion or resuscitative endovascular balloon occlusion of the aorta (REBOA) for hemorrhage control during endovascular aortic repair (EVAR) is a technique that has been used for decades for ruptured abdominal aortic aneurysms (rAAA). This usually requires bilateral femoral access, however, when only single-sided vascular access can be obtained this complicates the procedure if these techniques are to be used. We present two cases of single-sided vascular access, recently performed at our institution, using simultaneous REBOA and aortic stent-graft placement during EVAR in rAAA.
Method: This is a description of two clinical cases where REBOA and EVAR were performed through single-sided vascular access for the treatment of rAAA at Örebro University Hospital between March 2018 and June 2018.
Conclusion: This case report demonstrates that despite the limitation of single-sided access, an aortic stent-graft can be placed for treatment of a rAAA during continuous aortic occlusion with REBOA, facilitated by using a multidisciplinary EndoVascular resuscitation and Trauma Management (EVTM) team approach.

Keywords: *rAAA; EVAR; REBOA; Hemorrhagic Shock; Endovascular Resuscitation*

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INTRODUCTION

The incidence of ruptured abdominal aortic aneurysms (rAAA) has in recent years declined, at least in Scandinavia, however, the mortality rate remains high [1]. Some studies have suggested lower mortality rates with endovascular aortic repair (EVAR) of rAAA but a complete consensus is yet to be established [2–6]. Previously, EVAR of rAAA has been viewed as unsuitable for those presenting with profound hypotension due to the increased time involved for preparation. However, some centers treat all rAAA using EVAR and hybrid techniques with excellent results [2]. Intraluminal aortic balloon occlusion is a technique that has been used for decades to gain proximal aortic control of hemorrhage in rAAA [7–9]. Resuscitative endovascular balloon occlusion of the aorta (REBOA) or aortic balloon occlusion (ABO) may potentially help to stabilize the profoundly hypotensive patient and allow time for transport, diagnosis and appropriate preparation and execution of an endovascular repair [10–12]. This usually requires the aortic stent-graft to be inserted from the contralateral groin and a second occlusion balloon to be inflated inside the main body of the stent-graft in order to continue aortic occlusion and simultaneous modulation of the graft while the more proximal balloon is deflated and withdrawn. However, when only single-sided vascular access can be obtained this complicates the procedure if these techniques are to be used. We present two cases of single-sided vascular access,

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recently performed at our institution, using simultaneous REBOA and aortic stent-graft placement during EVAR in rAAA. This illustrates how endovascular techniques may be useful in extreme situations and how access difficulties can be solved to facilitate endovascular treatment.

PATIENTS AND METHODS

This is a description of two clinical cases where REBOA and EVAR were performed through a single-sided vascular access for the treatment of rAAA at Örebro University Hospital. Ethical committee approval was obtained for all REBOA and/or EVAR procedure patients. Both patients were treated with REBOA procedures performed by the on-call vascular surgeon and EVAR was performed in the hybrid operating room.

RESULTS

Case 1

A 76-year-old male who 8 years previously had been operated with EVAR for an infrarenal abdominal aortic aneurysm, and later received a cross-femoral venous bypass because of a unilateral limb graft occlusion, presented to the emergency department with a distended abdomen after a sudden debut of abdominal pain radiating to the back. An acute computed tomography (CT) scan revealed a rAAA with ongoing extravasation and massive retroperitoneal hematoma caused by a type Ib leakage and dislocated graft limb into the aneurysmal sack. The patient was initially hemodynamically stable and was transported to the hybrid operating room where he quickly deteriorated with a systolic blood pressure (SBP) of around 40 mm Hg. A 7 Fr sheath was placed in the right femoral artery proximal to the cross-femoral bypass anastomosis using ultrasound guidance. Zone 3 REBOA with total occlusion was performed and the patients SBP immediately stabilized around 90 mm Hg. A 4 Fr sheath was placed proximally to the 7 Fr sheath using ultrasound guidance and was later upgraded to a 16 Fr DrySeal sheath. The REBOA was partially deflated to let a guidewire pass and the dislocated graft limb was extended with a GORE™ 16 mm bridging-graft and a 20 mm iliac-graft after mapping of the internal iliac artery. The REBOA was then deflated and removed after a total occlusion time of 25 minutes with the patient remaining hemodynamically stable. The whole procedure was done under local anesthesia and the patient received 2 units of packed red blood cells (PRBC). The patient was then taken to the intensive care unit where 3 hours later he developed abdominal compartment syndrome and anuria. A decompressive laparotomy was subsequently performed by the on-call vascular surgeon and the abdomen was left open with a Bogota Bag in place. Within 30–40 minutes the patient started producing urine, a second-look laparotomy the following day showed no signs of ischemic bowel and the abdomen was closed. Because of elevated infectious and liver values, a CT scan was performed showing signs of acute pancreatitis, treated conservatively with intravenous fluid administration, analgesics and avoidance of oral fluid or food intake. The remainder of the patient's in-hospital care was uneventful and at 30 days post-intervention, he was recovering well, with no signs of endoleak seen on CT and ultrasound examinations, and a functional cross-femoral venous bypass.

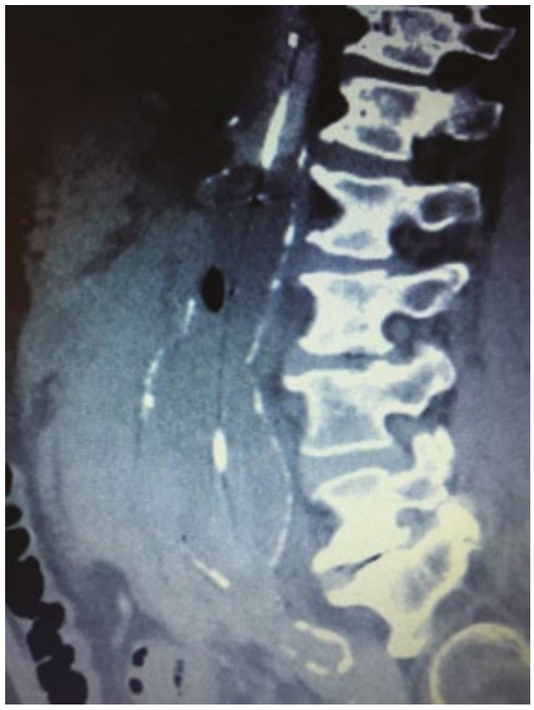

Figure 1 CT scan showing the use of REBOA in a ruptured abdominal aortic aneurysm for hemodynamic stabilization.

Case 2

A 63-year-old male was seen to swerve his car to the side of the road by a passerby. He was found in an unconscious state and later transported by ambulance to the emergency department. During transport, the patient was tachycardic (125 bmp) with a SBP of 90/55 and a faint palpable carotid pulse. Upon arrival at the emergency room, 57 minutes after the emergency call was received, he was hemodynamically unstable with a SBP of 40 mm Hg and GCS 3. The patient was intubated, an 8 Fr sheath was placed by a blind puncture in the right femoral artery on the first attempt and Zone I REBOA was performed (Figures 1 and 2), stabilizing the SBP between 60 and 70 mm Hg. A CT scan revealed a previously unknown infrarenal rAAA with ongoing extravasation and massive retroperitoneal hematoma, and the

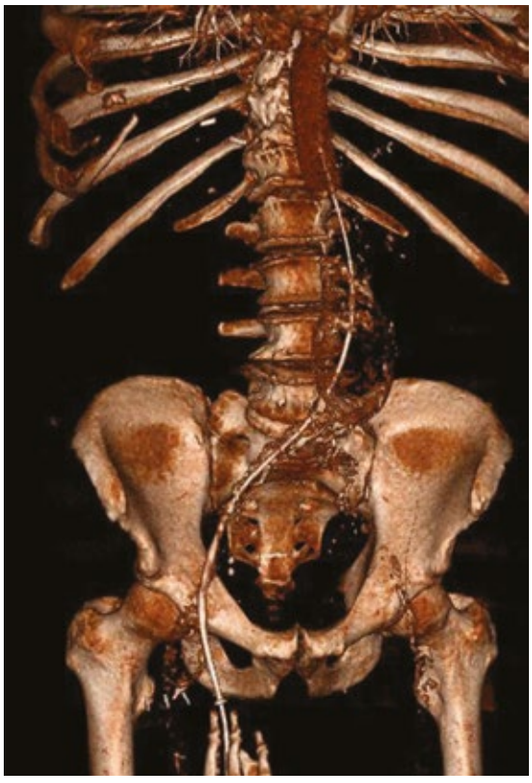

Figure 2 3D image of inflated REBOA in Zone 1.

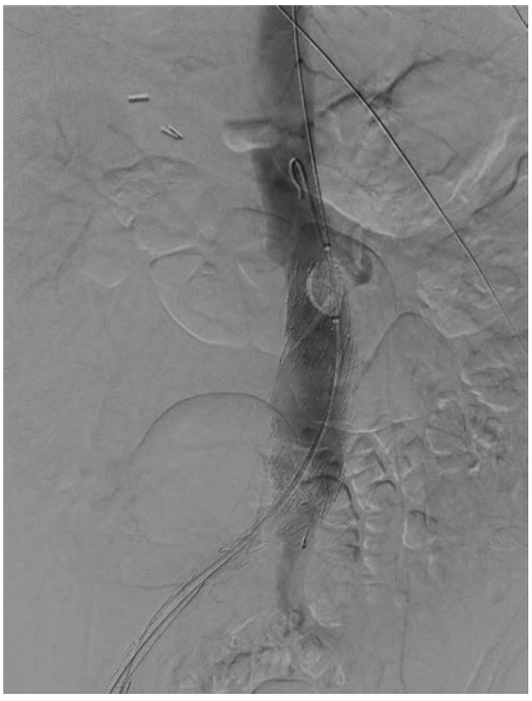

Figure 3 Angiography showing Zone 3 partial ABO and parallel guide wire through a single-sided access.

patient was transported to the hybrid operating room where a massive transfusion of PRBC, fresh frozen plasma (FFP) and platelets (PLT) was initiated. Blood gas analysis showed severe metabolic acidosis with increasing levels of lactate. Ultrasound of the left femoral artery showed signs of significant stenosis and no blood flow, with attempts to puncture not resulting in any backflow. With the REBOA in place, the decision was made to work through a single-sided access and a 36 mm COOK Zenith Alpha™ bifurcated main body graft was introduced proximally to the 8 Fr sheath through a separate puncture in the right femoral artery and deployed in the aorta. The REBOA was deflated to allow angiographic visualization of the renal arteries and graft placement, with the patients SBP immediately decreasing to 50 mm Hg. Stent-graft migration was noted and a 36 mm aortic cuff was placed above the bifurcated graft below the level of the renal arteries. The REBOA was removed and a new aortic occlusion balloon was placed in the main body of the aortic stent-graft below the renal arteries to allow visceral perfusion (Figure 3). Due to the occlusion of the left femoral artery, ultrasound-guided puncture of the left external iliac artery was performed in order to catheterize and extend the main stent-graft with an iliac limb extension to the left common iliac artery. A new angiography was performed revealing no signs of endoleakage. The aortic occlusion balloon was removed and the vascular accesses sealed, with the patient remaining stable with a SBP of 70 mm Hg. During the procedure, a total of 14 units PRBC, 14 units FFP and 3 units of PLT had been administered. By this time, the patient had developed an extended abdomen with obvious signs of abdominal compartment syndrome, acidosis, and increasing lactate levels. During preparation for a decompressive laparotomy, the patients SBP slowly started to decrease despite high levels of inotropic therapy and he later died.

DISCUSSION

Many patients who reach hospital with a rAAA are usually hemodynamically stable enough to allow proper diagnostics and preparation for surgery, however, some are hemodynamically unstable with profound hypotension and impending cardiac arrest [13]. This has previously been a strict indication for open aortic repair, but with the introduction of compliant occlusion balloons for hemodynamic stabilization endovascular definitive treatment has become a tempting option. When simultaneous REBOA and EVAR is performed for rAAA, this usually requires bilateral femoral access, with an aortic occlusion balloon inserted on one side and the aortic stent-graft inserted from the contralateral groin [2]. When faced with access complications in a hemodynamically unstable patient, many would choose the traditional open approach to aortic repair as alternative techniques have previously been sparse [14]. EVAR has in recent systematic reviews been considered a safe method of treating rAAA and should be preferred when technically

feasible [15]. In this report, we present the technique of single-sided simultaneous deployment of both REBOA and an aortic stent-graft with continuous aortic occlusion in two recent cases of rAAA with hemodynamic instability, impending cardiac arrest and unilateral iliac or femoral artery occlusion. This allowed the patients to stabilize, gaining time for proper EVAR execution. Non-compressible massive hemorrhage with hemodynamic instability is one of the most challenging conditions for physicians to deal with. This requires a cooperative relationship between a multidisciplinary team to ensure timely resuscitation, diagnosis, preoperative and perioperative support, definitive operative treatment, and excellent postoperative care. Both cases presented were facilitated thanks to the EndoVascular resuscitation and Trauma Management (EVTM) concept practiced at our institution [16–21]. Resuscitation by early vascular access and REBOA placement by the vascular surgeon, simultaneous circulatory management by the anesthesiologist in addition to rapid diagnostic imaging and working through single-sided accesses allowed EVAR to remain the optimal treatment option in both cases.

CONCLUSION

This case report demonstrates that despite the limitation of single-sided access, it is technically feasible for an aortic stent-graft to be placed for treatment of a rAAA during continuous aortic occlusion with REBOA, facilitated by using a multidisciplinary EVTM team approach.

REFERENCES

[1] Moll FL, Powell JT, Fraedrich G, et al. Management of abdominal aortic aneurysms clinical practice guidelines of the European society for vascular surgery. Eur J Vasc Endovasc Surg. 2011;41 Suppl 1:S1–S58.

[2] Mayer D, Aeschbacher S, Pfammatter T, et al. Complete replacement of open repair for ruptured abdominal aortic aneurysms by endovascular aneurysm repair: a two-center 14-year experience. Ann Surg. 2012;256:688–95.

[3] Nedeau AE, Pomposelli FB, Hamdan AD, et al. Endovascular vs open repair for ruptured abdominal aortic aneurysm. J Vasc Surg. 2012;56:15–20.

[4] Powell JT, Sweeting MJ, Thompson MM, et al. IMPROVE trial investigators. Endovascular or open repair strategy for ruptured abdominal aortic aneurysm: 30 day outcomes from IMPROVE randomised trial. BMJ. 2014;348:f7661.

[5] Desgranges P, Kobeiter H, Katsahian S, et al. Editor's Choice – ECAR (Endovasculaire ou Chirurgie dans les Anévrysmes aorto-iliaques Rompus): A French randomized controlled trial of endovascular versus open surgical repair of ruptured aorto-iliac aneurysms. Eur J Vasc Endovasc Surg. 2015;50:303–10.

[6] Zhang S, Feng J, Li H, Zhang Y, Lu Q, Jing Z. Open surgery (OS) versus endovascular aneurysm repair (EVAR) for hemodynamically stable and unstable ruptured abdominal aortic aneurysm (rAAA). Heart Vessels. 2016;31:1291–302.

[7] Smith FG. Emergency control of ruptured abdominal aortic aneurysm by transaxillary balloon catheter. Vasc Surg. 1972;6:79–84.

[8] Hyde GL, Sullivan DM. Fogarty catheter tamponade of ruptured abdominal aortic aneurysms. Surg Gynecol Obstet. 1982;154:197–9.

[9] Ng AC, Ochsner EC. Use of Fogarty catheter tamponade for ruptured abdominal aortic aneurysms. AJR Am J Roentgenol. 1977;128:31–3.

[10] Malina M, Holst J. Balloon control for ruptured AAAs: when and when not to use? J Cardiovasc Surg. 2014;55:161–7.

[11] Malina M, Veith F, Ivancev K, Sonesson B. Balloon occlusion of the aorta during endovascular repair of ruptured abdominal aortic aneurysm. J Endovasc Ther. 2005;12:556–9.

[12] Philipsen TE, Hendriks JM, Lauwers P, et al. The use of rapid endovascular balloon occlusion in unstable patients with ruptured abdominal aortic aneurysm. Innovations (Phila). 2009;4:74–9.

[13] Mehta M, Paty PSK, Byrne J, et al. The impact of hemodynamic status on outcomes of endovascular abdominal aortic aneurysm repair for rupture. J Vasc Surg. 2013;57:1255–60.

[14] van Beeka SC, Conijna A P, Koelemay M J, Balm R. Endovascular aneurysm repair versus open repair for patients with a ruptured abdominal aortic aneurysm: A systematic review and meta-analysis of short-term survival. Eur J Vasc Endovasc Surg. 2014;47:593–602.

[15] Amato B, Fugetto F, Compagna R, et al. Endovascular repair versus open repair in the treatment of ruptured aortic aneurysms. A systematic review. Minerva Chir. 2018; *In Press*. doi: 10.23736/S0026-4733.18.07768-4.

[16] Bilos L, Pirouzram A, Toivola A, Vidlund M, Cha SO, Horer T. EndoVascular and hybrid trauma management (EVTM) for blunt innominate artery injury with ongoing extravasation. Cardiovasc Intervent Radiol. 2017;40:130–4.

[17] Horer T. Resuscitative endovascular balloon occlusion of the aorta (REBOA) and endovascular resuscitation and trauma management (EVTM): a paradigm shift regarding hemodynamic instability. Eur J Vasc Endovasc Surg. 2018;44:487–9.

[18] Horer TM, Hebron D, Swaid F, et al. Aorta balloon occlusion in trauma: Three cases demonstrating multidisciplinary approach already on patient's arrival to the emergency room. Cardiovasc Intervent Radiol. 2016;39:284–9.

[19] Horer TM, Skoog P, Pirouzram A, Nilsson KF, Larzon T. A small case series of aortic balloon occlusion in trauma: lessons learned from its use in ruptured abdominal aortic aneurysms and a brief review. Eur J Trauma Emerg Surg. 2016;42:585–92.

[20] McGreevy D, Dogan E, Toivola A, et al. Endovascular resuscitation with aortic balloon occlusion in non-trauma cases: First use of ER-REBOA in Europe. Journal of Endovascular Resuscitation and Trauma Management. 2017;1:42–9.

[21] Nilsson C, Bilos L, Horer T, Pirouzram A. Use of resuscitative endovascular balloon occlusion of the aorta in a multidisciplinary approach. Innovations (Phila). 2017;12:e1–e2.

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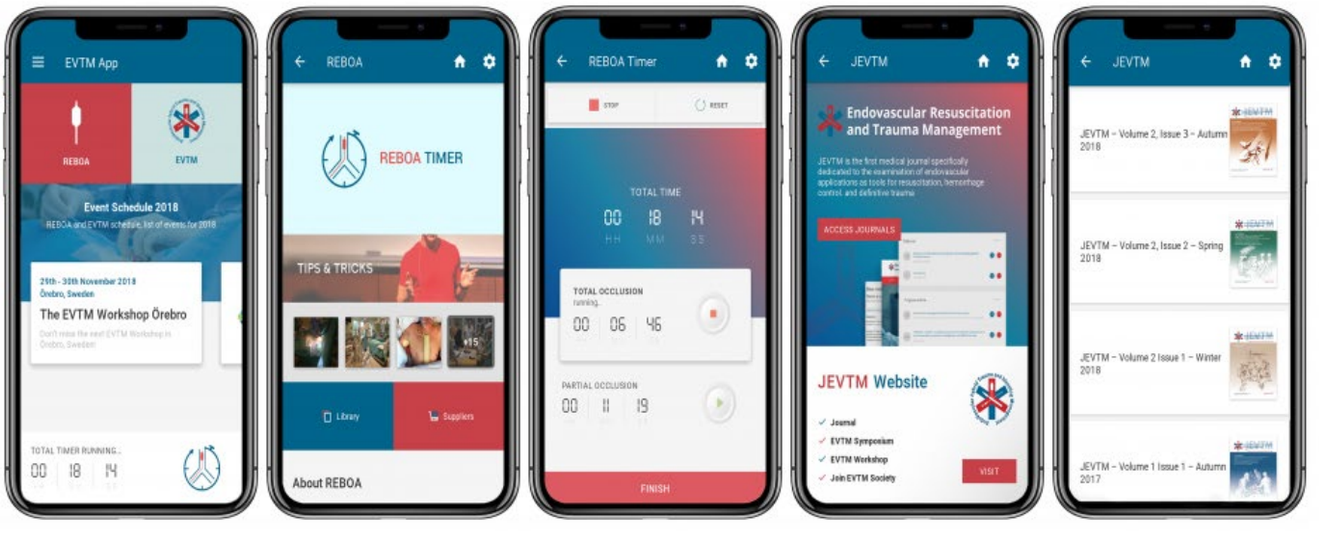

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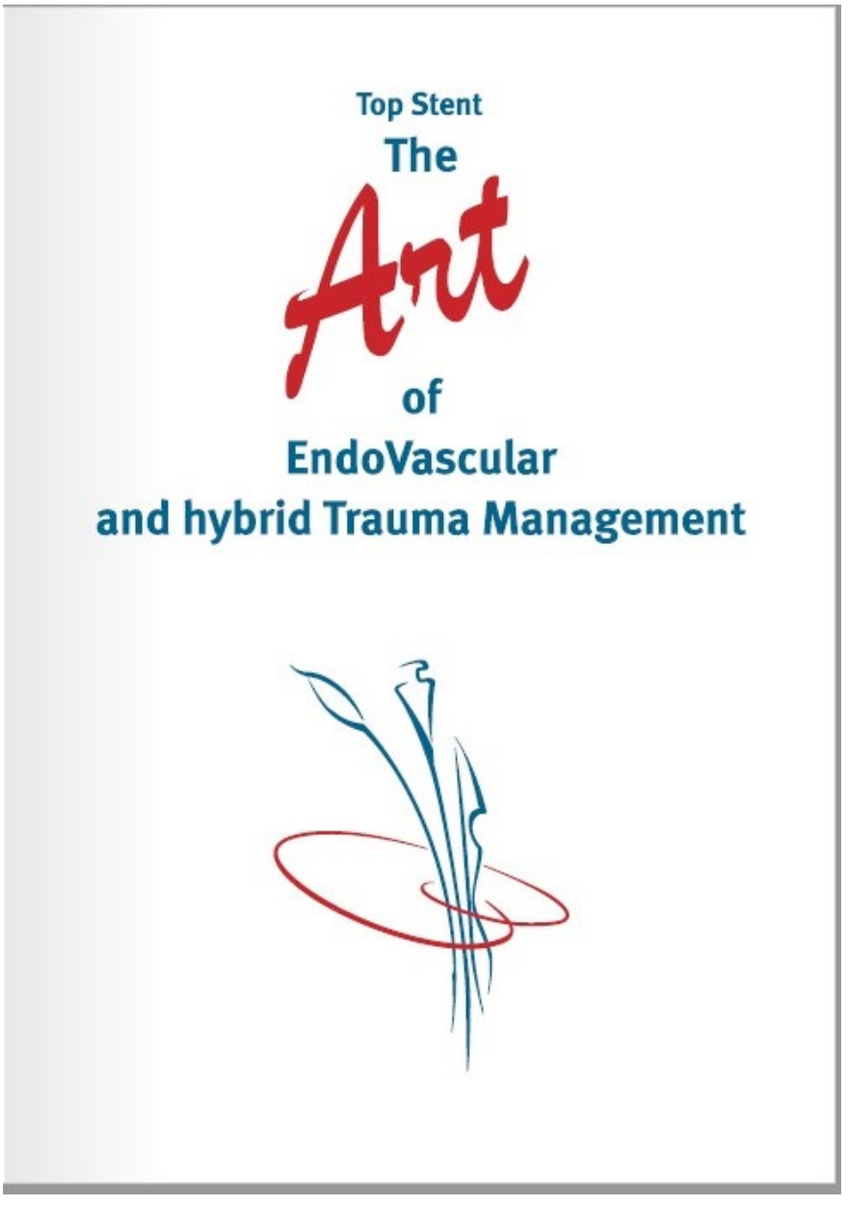

The Top Stent project is a multidisciplinary work manual for EVTM. 28 experts from around the world give tips and tricks for EVTM and REBOA.

The manual was released during the EVTM 2017 Symposium and is now distributed as a non-profit project at production costs of 302 SEK with addition to delivery by post. If interested, send an e-mail to: asa.strandberg@regionorebrolan.se and state: number of books, address of shipment and address for invoice.

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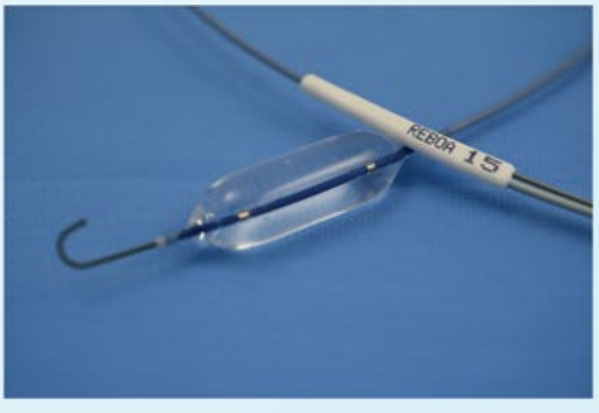

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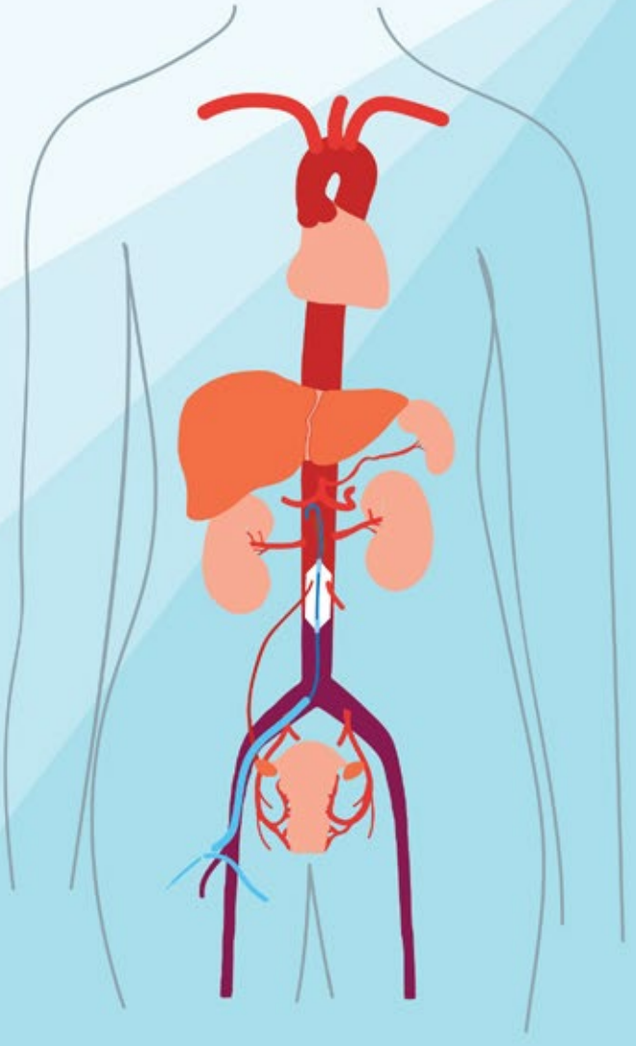

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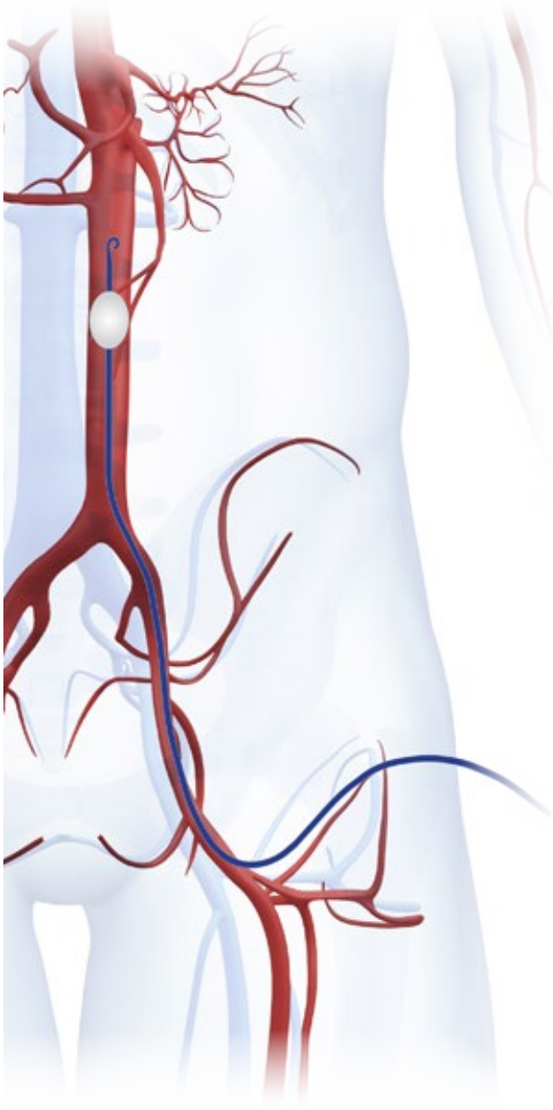

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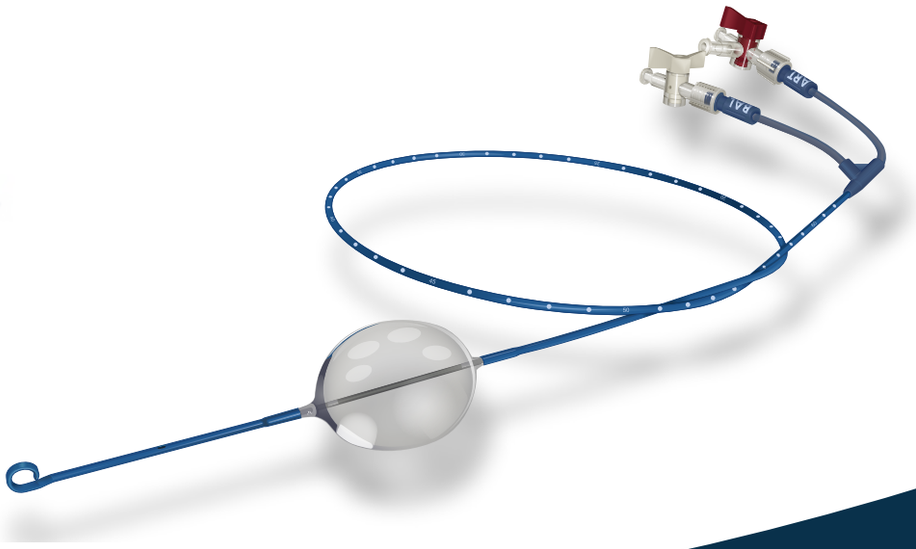

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